Incidence and Site of Atrioventricular Block in Patients with Chronic Bifascicular Block

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SUMMARY Four hundred fifty-two patients with chronic bifascicular block and initially intact atrioventricular (AV) conduction were studied, detected, and prospectively followed between January 1970 and March 1978. There were 360 males and 92 females, ages 18–93 years (mean ± SD, 62 ± 15 years). Follow-up ranged from 29–2804 days (mean 1066 ± 97 days). AV block (2° or 3°) developed in 29 patients, nine with apparent cause and spontaneous in 20. Cumulative annual incidence of all heart block for 1–5 years was, respectively, 4%, 5.9%, 8.7%, 10.1% and 11.3%, and for spontaneous block was 2%, 3.1%, 5.2%, 6.7%, and 7.1%. Sites of spontaneous block were probably or definitely AV nodal in ten, His bundle in one, and trifascicular in nine.

Cumulative incidence of AV block in surviving bifascicular block patients is 11% at 5 years, with 7% reflecting spontaneous block. The probable or definite site of AV block varies and is trifascicular in less than half of the patients. The small incidence of trifascicular block probably explains the difficulty in predicting this complication with electrophysiologic studies.

THE PROGRESSION of chronic bifascicular block to advanced or complete heart block has been previously demonstrated. Schloff et al.,1 Lasser et al.,2 Rosenbaum et al.,3 Scanlon et al.,4 and Kulbertus et al.5 reported a 2–62% incidence of heart block in patients with chronic bifascicular block, over periods ranging from several months to 10 years. Most of the previously reported studies concerning progression of bifascicular block to AV block, in addition to being retrospective, do not specify a definite or presumptive site of block, implying that the AV block developing in these patients is trifascicular.

We report here the results of the prospective follow-up of a large group of patients with chronic bifascicular block, the incidence of AV block, and observations concerning clinical presentation and probable or definite site of block. We also present electrophysiological findings from the time of initial study of bifascicular block.

Materials and Methods

Definitions

The criteria for electrocardiographic diagnosis of right bundle branch block included a QRS duration of 0.12 second or greater with a rsR or qR configuration of the QRS complex in lead V₆.6 Left bundle branch block was diagnosed if the QRS duration was 0.12 second or greater with the presence of a broad mono-

phasic R wave or Rs in lead V₆ with ST depression and T-wave inversion in leads V₂ and V₆.6 The criteria for the electrocardiographic diagnosis of left anterior and posterior hemiblocks were identical to those used in many previous studies.2,4-16 Left anterior hemiblock was diagnosed in the presence of a mean frontal QRS axis more negative than −30°, a small q and tall R wave in lead I, and a small r and deep S wave in lead III.2,4,7-11 Left posterior hemiblock was diagnosed in the presence of a mean frontal QRS axis more positive than +90°, a small r and deep s wave in lead I, and a small q and tall R wave in lead III.4,7,8,10,12,16,16

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. Second and third degree AV block were also referred to us for electrophysiologic study and follow-up from physicians in the greater Chicago area.

Criteria for inclusion in this study were: 1) presence of chronic bifascicular block as defined by the presence of bifascicular block on all (at least two) available resting ECGs, 2) age 18 years or over, 3) giving informed consent for electrophysiologic studies, and 4) voluntary agreement to periodic follow-up in a conduction disease clinic. Patients with a prior second or third degree AV block and patients with acute myocardial infarction were excluded.

Initial evaluation of patients included history,
TABLE 1. Electrocardiographic and Electrophysiologic Data in 452 Patients with Bifascicular Block

| Electrocardiographic and Electrophysiologic Data in 452 Patients with Bifascicular Block |
|-----------------------------------------|-----------------------------------------|-----------------------------------------|-----------------------------------------|
| QRS morphology                          | No. of pts | Percent | Range | Mean ± sd |
| RBBB + LPH                               | 297       | 66      | 100   | 0.06 ± 0.05 |
| RBBB + LAH                               | 42        | 9       | 100   | 0.03 ± 0.02 |
| LBBB                                     | 113       | 25      | 100   | 0.03 ± 0.02 |
| Rhythm                                   | No. of pts | Percent | Range | Mean ± sd |
| Sinus                                    | 439       | 97      |        | 0.04 ± 0.01 |
| Atrial fibr.                             | 13        | 3       |        | 0.01 ± 0.01 |
| P-R interval (sec)                       | 439       | 97      | 0.11-0.46 | 0.19 ± 0.04 |
| QRS duration (sec)                       | 452       | 100     | 0.12-0.20 | 0.14 ± 0.01 |
| A-H interval (msec)                      | 439       | 97      | 45-402 | 108 ± 42.4 |
| A-H interval (> 130 msec)                | 60        | 14      |        |            |
| H-V interval (msec)                      | 452       | 100     | 24-125 | 54 ± 14.3 |
| H-V interval (> 55 msec)                 | 168       | 37      |        |            |

Abbreviations: RBBB = right bundle branch block; LAH = left anterior hemiblock; LPH = left posterior hemiblock; LBBB = left bundle branch block; Atrial fibr. = atrial fibrillation.

Physical examination, serial ECGs, chest roentgenograms and routine laboratory tests. Based on this evaluation, a clinical diagnosis was established for each patient. Previously described criteria were used to diagnose organic heart disease. Normal values were recorded, and there were no clinical evidence of organic heart disease other than intraventricular conduction defect.

Electrophysiologic Studies

Informed written consent was obtained before electrophysiologic study. His bundle (HB) electrogroms were recorded, using previously described catheter techniques, in all patients at the beginning of the study. Cardiac drugs were withheld for at least 48-72 hours before the study. Measurements of AV nodal conduction (A-H) and His-Purkinje conduction (H-V) intervals were made at paper speeds of 200 mm/sec and reflected the mean of 10 consecutive sinus beats. Normal values for the AH interval was 54-130 msec, and for the H-V interval, 31-55 msec. In patients with atrial fibrillation at the time of study, only H-V intervals could be measured. HB pacing was not routinely performed in our patients; therefore, we cannot comment upon the possibility of lesions within the HB responsible for bifascicular block.

Patient Follow-Up

Patient follow-up was similar to that previously described by our laboratory. After initial study, all patients were prospectively followed in research conduction disease clinics at 1-3-month intervals. Upon each clinic visit, history, physical examination and ECGs were performed. Portable tape recorder monitoring or prolonged inpatient electrocardiographic monitoring, or both, were used when history suggested transient bradyarrhythmias. In patients developing AV block, a presumptive site of block was diagnosed using clinical and surface electrocardiographic data. Second degree AV nodal block was suspected when type I block was present in any combination with type II block or 2:1 block, or if type I block was observed during acute diaphragmatic wall infarction or drug therapy. Third degree AV nodal block was thought to be present when the escape rhythm was relatively faster (> 40/min) and was identical to previously conducted QRS complexes. Second degree trifascicular block was diagnosed when type II block or 2:1 block alone was present. Third degree trifascicular block was thought to be present when the escape rhythm was slower without QRS complexes different from previously conducted complexes. The site of block was confirmed by repeat HB recording, if possible, during episodes of AV block. Permanent pacemakers were implanted in symptomatic patients with documented bradyarrhythmias or in patients with recurrent syncope due to unknown cause. Asymptomatic patients manifesting second and third degree AV block were followed closely, without pacemaker implantation, unless the site of block was distal to the HB.

All information from the initial evaluation and subsequent clinic visits was keypunched and stored on IRS data base discs. Specifically designed programs were used for data recall and statistical analysis. Previously reported life table methods were used for actuarial analysis of incidence of AV block. No patient was lost to follow-up.

Results

Characteristics of the Study Group (tables 1 and 2)

Four hundred fifty-two patients with chronic bifascicular block were studied and followed between January 1970 and March 1978. There were 360 (80%) males and 92 (20%) females, ages 18-93 years (mean ± sd, 62 ± 15 years).
Table 2. Selected Clinical Data in 452 Patients with Bifascicular Block

<table>
<thead>
<tr>
<th>Clinical findings</th>
<th>Number of patients</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angina</td>
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<td>22</td>
</tr>
<tr>
<td>Congestive failure</td>
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<td>33</td>
</tr>
<tr>
<td>Cardiomegaly</td>
<td>252</td>
<td>57</td>
</tr>
<tr>
<td>NYHA class I</td>
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<td>57</td>
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<td>NYHA class II</td>
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<td>26</td>
</tr>
<tr>
<td>NYHA classes III and IV</td>
<td>76</td>
<td>17</td>
</tr>
</tbody>
</table>

Clinical diagnosis

- Hypertensive: 146 (32%)
- Arteriosclerotic: 140 (31%)
- Primary myocardial: 34 (8%)
- Valvular: 25 (6%)
- Other: 21 (4%)
- Primary conduction disease: 86 (19%)

Abbreviations: NYHA = New York Heart Association.

Of the 452 patients, 297 (66%) had right bundle branch block and left anterior hemiblock, 42 (9%) had right bundle branch block and left posterior hemiblock, and 113 (25%) had left bundle branch block. Four hundred thirty-nine (97%) patients were in sinus rhythm at the time of study and 13 (3%) in atrial fibrillation. PR intervals ranged from 0.11–0.46 sec (mean ± SD 0.19 ± 0.04 sec). The QRS duration ranged from 0.12–0.20 sec (mean 0.14 ± 0.01 sec). Initial A–H intervals ranged from 45–402 msec with a mean of 108 ± 42.4 msec and were prolonged in 60 (14%) patients. H-V intervals ranged from 24–125 msec (mean 54 ± 14.3 msec), and were prolonged in 168 (38%) patients.

Clinically diagnosed congestive heart failure was present in 150 (33%) patients and cardiomegaly diagnosed by chest roentgenogram in 252 (57%) patients. There were 258 (57%) patients in cardiac functional class I (New York Heart Association criteria), and 194 (43%) were in classes II–IV. Organic heart disease was diagnosed in 366 (81%) of the patients; hypertensive and arteriosclerotic heart diseases were present in the majority of cases.

Incidence of AV Block

The follow-up period ranged from 29–2804 days (mean 1066 ± 97 days). Twenty-nine of 452 (6.4%) patients developed second or third degree AV block during follow-up. Of these, AV block occurred spontaneously in 20 (4.4%) and secondary to an apparent cause in nine (2%) patients (see below). Annual total cumulative incidence of heart block as well as cumulative incidence of spontaneous block for the first 5 years, using actuarial life table methods, is presented in fig. 1. We considered patients who had died not at risk for development of AV block. The cumulative yearly incidence of heart block during 5 years is shown in table 3. For all heart block, the incidence varied from 4% in the first year to 11.3% in the fifth year, and for spontaneous heart block from 2–7.1%, respectively.

The incidence of AV block with specific varieties of bifascicular block was as follows: for all AV block, the incidence was 8% (24 of 297 patients) with right bundle branch block and left anterior hemiblock, 5% (two of 42 patients) with right bundle branch block and left posterior hemiblock, and 3% (three of 113 patients) with left bundle branch block. For spontaneous block, the incidences were 5% (16 of 297 patients) with right bundle branch block and left anterior hemiblock, 5% (two of 42 patients) with right bundle branch block and left posterior hemiblock, and 2% (two of 113 patients) with left bundle branch block. These differences were not statistically significant.

AV Block Due to Apparent Cause (table 4)

AV block was related to hyperkalemia (potassium levels greater than 7.0 mEq/L) in three patients (oc-
curing on days 5, 6 and 1059 of follow-up), acute myocardial infarction in two (on days 351 and 1453), digitalis or propranolol therapy in three (on days 136, 567, and 925), and cardiac surgery in one (on day 58). In all patients except those who had acute myocardial infarction and cardiac surgery, AV block was reversible. The site of block was distal to the HB in five and proximal to the HB in four patients (three with digitalis or propranolol therapy and one with hyperkalemia). Initial electrophysiologic studies had revealed HV prolongation in four of five patients with block distal to HB, and AH prolongation in three of four patients with block proximal to HB.

**Spontaneous AV Block**

In the 20 patients with spontaneous AV block, the site of block (as determined by clinical and electrocardiographic data with or without HB recordings) was AV nodal in 10 (50%), within the HB in one (5%), and trifascicular in nine (45%).

**Spontaneous AV Nodal Block (table 5)**

We had 10 patients with AV nodal block (nine with second degree block and one with third degree block). In four patients, the site of block was determined by HB studies (fig. 2); in five patients, we inferred probable site of block by the presence of electrocardiographic type I second degree AV block (all with marked PR prolongation of conducted beats); and in one patient, by the occurrence of complete heart block with a relatively fast escape rhythm identical to previously conducted QRS complexes. None of these patients were challenged with atropine. The ventricular rates at the time of AV block ranged from 44–74/min, with a mean of 59 ± 10/min. The ages of the patients with spontaneous AV nodal block ranged

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**TABLE 4. Follow-up Data on Nine Patients with AV Block Due to an Apparent Cause**

<table>
<thead>
<tr>
<th>Case</th>
<th>EKG</th>
<th>A-H interval (msec)</th>
<th>H-V interval (msec)</th>
<th>Degree (and type) of AV block</th>
<th>Day of occurrence</th>
<th>Site of block</th>
<th>Probable cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>RBBB + LAH</td>
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<td>60</td>
<td>2:1 (I &amp; II)</td>
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<td>Hyperkalemia</td>
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<tr>
<td>2</td>
<td>RBBB + LAH</td>
<td>91</td>
<td>42</td>
<td>2° (II) 2:1-4:1</td>
<td>6</td>
<td>Distal*</td>
<td>Hyperkalemia</td>
</tr>
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<td>3</td>
<td>RBBB + LAH</td>
<td>210</td>
<td>30</td>
<td>2° (I)</td>
<td>5</td>
<td>Proximal†</td>
<td>Hyperkalemia</td>
</tr>
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<td>4</td>
<td>RBBB + LAH</td>
<td>89</td>
<td>79</td>
<td>2° (II)</td>
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<td>Distal†</td>
<td>Acute ASMI</td>
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<tr>
<td>5</td>
<td>RBBB + LAH</td>
<td>122</td>
<td>100</td>
<td>2:1 &amp; 3°</td>
<td>1453</td>
<td>Distal*</td>
<td>Acute AMI</td>
</tr>
<tr>
<td>6</td>
<td>LBBB</td>
<td>190</td>
<td>60</td>
<td>2° (I)</td>
<td>925</td>
<td>Proximal*</td>
<td>Digitalis</td>
</tr>
<tr>
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<td>RBBB + LAH</td>
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<td>65</td>
<td>2° (I)</td>
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<td>Digitalis</td>
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<td>45</td>
<td>3°</td>
<td>567</td>
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<td>Propranolol</td>
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<tr>
<td>9</td>
<td>RBBB + LAH</td>
<td>300</td>
<td>60</td>
<td>2:1</td>
<td>58</td>
<td>Distal*</td>
<td>Post-surgery</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: ASMI = anteroseptal myocardial infarction; AMI = anterior myocardial infarction; AV = atrioventricular.

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**TABLE 5. Follow-up Data on Patients with Spontaneous AV Nodal Block**

<table>
<thead>
<tr>
<th>Case</th>
<th>EKG</th>
<th>A-H interval (msec)</th>
<th>H-V interval (msec)</th>
<th>Degree (and type) of AV block</th>
<th>Day of occurrence</th>
<th>Presenting symptom with AV block</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>RBBB + LAH</td>
<td>125</td>
<td>65</td>
<td>2° (I &amp; II)</td>
<td>71†</td>
<td>CHF</td>
</tr>
<tr>
<td>2</td>
<td>RBBB + LAH</td>
<td>140</td>
<td>37</td>
<td>2° (I) &amp; 2:1</td>
<td>1518*</td>
<td>CHF</td>
</tr>
<tr>
<td>3</td>
<td>RBBB + LAH</td>
<td>160</td>
<td>45</td>
<td>2° (I)</td>
<td>2122†</td>
<td>CHF</td>
</tr>
<tr>
<td>4</td>
<td>RBBB + LPH</td>
<td>138</td>
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<td>2°(I)</td>
<td>398†</td>
<td>CHF</td>
</tr>
<tr>
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<td>85*</td>
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<tr>
<td>6</td>
<td>RBBB + LAH</td>
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<td>10</td>
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<td>160</td>
<td>50</td>
<td>2° (I)</td>
<td>1972†</td>
<td>—</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: LBBB = left bundle branch block; RBBB = right bundle branch block; LAH = left anterior hemiblock; LPH = left posterior hemiblock; CHF = congestive heart failure; AV = atrioventricular.
from 49–84 years (mean 69 ± 12 years). AV block in these patients occurred on days ranging from 17–2122 of follow-up. In six of 10 patients with AV nodal block, congestive heart failure either appeared (four patients) as the presenting symptom or worsened (two patients) with the development of AV block. Two had syncope with block and two were asymptomatic.

Previous electrophysiologic studies in the 10 patients with AV nodal block revealed A-H intervals ranging from 85–400 msec, with A-H prolongation (> 130 msec) in seven (70%). H-V intervals ranged from 37–93 msec, with H-V prolongation (> 55 msec) in three (30%).

**HB Block**

In the one patient with HB block, a 79-year-old man, the site of block was determined by HB studies (fig. 3). This patient developed AV block on day 55 of follow-up. The ventricular rate at the time of block was 40/min. The presenting symptom associated with AV block was congestive heart failure. Initial electrophysiologic studies in this patient were normal (A-H 122 msec, and H-V 42 msec).

**Trifascicular Block (table 6)**

Of the nine patients with block distal to the HB (five with second degree block and four with third degree block), the site of block was determined by HB studies in four (fig. 4). A probable site of block was determined in three by the presence of electrocardiographic type II second degree block, and in the other two by occurrence of complete heart block with relatively slow QRS escape, different from previously conducted QRS complexes. The ventricular rates at the time of AV block ranged from 34–62/min, with a mean of 46 ± 9/min. The ages of these patients, six males and three females, ranged from 58–78 years (mean 67 ± 7 years). AV block occurred on days ranging from 5–1727 of follow-up. The presenting symptom associated with development of AV block was syncope in five patients and the appearance (three patients) or worsening (one patient) of congestive cardiac failure in four patients. None of the nine patients was asymptomatic at the time of AV block.

Initial electrophysiologic studies in the nine patients with trifascicular block revealed A-H intervals ranging from 68–182 msec, with A-H prolongation in one (11%). H-V intervals ranged from 41–100 msec, and were prolonged in seven (78%).

**Pacemakers and Sudden Death**

Permanent pacemakers were implanted in 32 patients for the following indications: symptomatic AV block (18 patients), sinus node disease (five patients), and recurrent syncope of unknown cause (four patients). In five other patients, pacemakers

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**Figure 2.** His bundle (H) recording in a patient developing AV nodal block (type I, second degree block). Shown are ECG leads II, III and V1, a bipolar intracardiac high right atrial electrogram (HRA) and His bundle electrogram (HBE). Paper speed is 100 mm/sec and time lines are at 1-second intervals. P waves are labeled as P, atrial electrogram as A, His bundle potential as H, and ventricular electrogram as V. A-H intervals are listed. Note progressive prolongation of A-H intervals with fourth P wave blocked proximal to H.

**Figure 3.** Recordings demonstrating third degree AV block within His bundle with "split" H potentials (H1 and H2). Atrial rate (AR), ventricular rate (VR), A-H1, and H2-V intervals are listed.
were implanted by referring physicians at other medical centers, prophylactically in two, after cardiac surgery in two, and because of dizziness with prolonged HV interval in one. Thus, 14 patients received pacemakers for reasons other than documented heart block, and conceivably could have subsequently developed heart block which was undetected.

Sixty-four (14%) patients died suddenly during follow-up. The annual cumulative mortality for sudden death for the first five years was (mean ± SEM) 7.6 ± 1.3%, 12.0 ± 1.6%, 15.4 ± 1.9%, 20.0 ± 2.3%, and 20.9 ± 2.4% respectively.

Discussion

Although the anatomy of the intraventricular conduction system is controversial, it is clinically useful to consider that the intraventricular conduction system comprises three functioning fascicles: the right bundle branch, the left anterior fascicle and the left posterior fascicle. Involvement of two fascicles has been termed bifascicular block. Electrocardiographic criteria for bifascicular block have varied. Patients with bifascicular block may have subclinical disease in the remaining functioning fascicle and may progress to trifascicular block. Many reports have demonstrated the progression of bifascicular block to advanced or complete heart block, but they give little information concerning the site of block. The reported incidence of the development of heart block has varied considerably in these studies.

Rotman and co-workers reported on 229 Air Force personnel, 125 with left bundle branch block and 104 with right bundle branch block. Two of these patients progressed to complete heart block over a mean follow-up period of 10.8 ± 4.7 years, with an incidence of 1%. In studies of hospitalized patients with bifascicular block, Lasser et al., Rosenbaum et al., Scanlon et al., DePasquale et al., and Wiberg et al., reported an incidence of heart block ranging from 6–62% over periods from a few months to about 10 years. However, all these studies have been retrospective. In addition, no information was

<table>
<thead>
<tr>
<th>Case</th>
<th>EKG</th>
<th>A-H interval (msec)</th>
<th>H-V interval (msec)</th>
<th>Degree (and type) of AV block</th>
<th>Day of occurrence</th>
<th>Presenting symptom with AV block</th>
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<tbody>
<tr>
<td>1</td>
<td>LBBB</td>
<td>120</td>
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<tr>
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<td>91</td>
<td>45</td>
<td>2° (II) &amp; 2:1</td>
<td>1727*</td>
<td>Syncope</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: LBBB = left bundle branch block; RBBB = right bundle branch block; LAH = left anterior hemiblock; CHF = congestive heart failure; AV = atrioventricular.

FIGURE 4. Recordings demonstrating third degree trifascicular block. Pacemaker spikes are marked with small arrows. Note complete heart block distal to H on transient cessation of temporary pacing.
provided regarding either a presumptive or definite site of block.

Information on the natural history of patients with chronic bifascicular block, using prospective follow-up methods, is limited. In a preliminary study, McAnulty et al. reported a 12% mortality (24 of 214 patients) over a mean follow-up period of 10.5 months, with sudden deaths occurring in 4% of the cases. No information regarding the incidence of heart block was provided. Scheinman and co-workers reported 88 patients with bifascicular block (42 with left bundle branch block and 46 with right bundle branch block with either left anterior or posterior hemiblock). The incidence of AV block in these patients was 9% (eight of 88) over a mean follow-up period of 18 months. The block was thought to be trifascicular in all patients, although the site of block was documented by HB studies in only two of eight patients. In a preliminary report, Kulbertus and co-workers reported a 2% incidence of heart block (two of 106) in patients with right bundle branch block and left anterior hemiblock in a mass screening clinic, over a mean follow-up period of 34.7 months. The latter two studies did not provide information regarding the annual cumulative incidence of heart block calculated using actuarial techniques.

The present series reports the incidence of heart block in a large, prospectively followed group of patients with chronic bifascicular block. Documented progression of conduction disease was relatively uncommon, occurring in only 11% of patients, with 7% reflecting spontaneous block and with a yearly risk of only 1–2%. Surprisingly, the site of AV block was not always distal to the HB. Bilateral bundle branch (or trifascicular) block accounted for only 45% (nine of 20) of cases with spontaneous progression. This agrees with our previously reported series of patients with complete heart block, in whom trifascicular block was demonstrated in only 49% of cases.

The sudden death of 14% of the patients in this series suggests that some of the sudden deaths could be secondary to sudden development of trifascicular block. We recently reported the significance and causative factors of sudden death in patients with chronic bifascicular block. The most common cause of sudden death in these patients was ventricular fibrillation rather than trifascicular block.

The incidence of AV block in our series of patients is consistent with Lasser and co-workers and inconsistent with others. There are several possible explanations for these differences:

1) The true incidence of progression in patients with bifascicular block is not yet known, and in previous studies the risk may be overestimated. Thus, there are conflicting reports about frequency of progression of conduction disease in bifascicular block patients — Rotman et al. (1%), Wiberg et al. (4.6%), Lasser et al. (9%), Scanlon et al. (14.4%), Schloff et al. (42%), and Rosenbaum et al. (6–62%).

2) The present series may have patients with different clinical and pathological substrates compared with the previously reported series. For example, in Rosenbaum’s study, a high proportion of patients had either chagasic myocarditis or acute myocardial infarction. Both of these disease processes have a high incidence of progressive conduction disease, once bifascicular block has developed. In our series there was a high incidence of hypertensive cardiovascular disease and primary conduction disease, which may have a more benign clinical course. The prognosis of bifascicular block may reflect the clinical circumstances in which the conduction disease occurred.

3) In our series, we excluded patients with second or third degree AV block, thus excluding those with manifest bilateral bundle branch disease. In contrast, Rosenbaum’s series included such patients.

4) Most of the previous series were retrospective. The present series was prospective, and designed to investigate the natural history of patients with chronic bifascicular block.

5) Differing results could relate to the different criteria used for patient selection. For example, electrocardiographic criteria for diagnosis of bifascicular block have varied in the previous studies. Rosenbaum’s criteria for diagnosing left anterior hemiblock with axis directed to the left of $-45^\circ$ and left posterior hemiblock with axis directed to the right of $+110^\circ$, based upon observations during acute myocardial infarction, are different from numerous previous studies. We have used definitions of left anterior and posterior hemiblock identical to those used in a large number of previous clinical studies which are somewhat less restrictive than Rosenbaum’s. Our data are comparable to the data of most of the previous studies on the natural history of patients with chronic bifascicular block.

6) Prospective studies by Scheinman et al. and Kulbertus et al. are different from ours, since the former study was biased towards preselection of patients with transient neurologic symptoms, and the latter study included outpatient subjects from a mass screening clinic.

It has been suggested that patients with right bundle branch block and left posterior hemiblock are at a higher risk of developing AV block than patients with right bundle branch block and left anterior hemiblock. Rosenbaum and co-workers reported a 62% incidence of heart block in the former group of patients, compared with only 6% in the latter. Similarly, Scanlon and associates described a 21% risk of AV block in patients with right bundle branch block and left posterior hemiblock, compared with 13.6% in patients with right bundle branch block and left anterior hemiblock. We found no such differences in our patients; the risk of heart block in both groups of patients is identical, about 5% over a mean follow-up period of about 3.5 years. The risk of AV block in all three varieties of bifascicular block was not statistically different.

Several recent reports have suggested that H-V prolongation in patients with chronic bifascicular
block is associated with a high risk of AV block. Since H-V prolongation in these patients suggests the presence of bilateral bundle branch disease, future occurrence of heart block could thus relate to the presence of prolonged H-V interval. The presence of H-V prolongation in seven of our nine patients who developed spontaneous trifascicular block appears to support this hypothesis. However, in the present study, 168 of 452 patients (38%) had H-V prolongation, yet trifascicular block occurred in only seven patients, with an incidence of 4%. The small percentage of patients with spontaneous trifascicular block prohibits predicting for this group by means of electrophysiologic studies. In our previously reported study of bifascicular block, we examined the value of H-V interval in predicting future trifascicular block by comparing patients with normal and prolonged H-V interval. The risk of progression of conduction disease was similar in both the prolonged and normal H-V groups. In a subsequent series from our laboratory, we demonstrated that even markedly prolonged H-V interval (80 msec or greater) in patients with bifascicular block was associated with a low incidence (6%) of subsequent AV block. These latter results differed from those of Scheinman et al., who demonstrated that marked H-V prolongation predicted risk of AV block.

AV nodal block occurred in 10 of our patients; of these, seven had prolonged A-H intervals at the time of initial study. Sixty of 452 (14%) patients in this series had A-H prolongation, and AV nodal block developed in seven, with an incidence of 12%. Although the incidence of AV nodal block associated with prolonged A-H was not significantly different from trifascicular block associated with H-V prolongation, it appears that A-H prolongation is a better predictor of AV nodal block than H-V prolongation for trifascicular block.

We delineated a definite site of block in only half of the patients with AV block. Therefore, our conclusions regarding the frequency and clinical implications of site of AV block are tentative. However, the prior occurrence of prolonged H-V interval in seven of our nine patients who developed trifascicular block, and prolonged A-H interval in seven of 10 patients who developed AV nodal block, suggest that our presumptions about the site of block were probably correct.

The clinical profile of our patients developing AV block revealed several interesting features. There were more males than females with AV nodal block; the ratio was 9:1. This correlates well with our recent report on patients with chronic AV nodal block. The predominant presenting features of these patients was congestive heart failure, reflecting a high incidence of myocardial dysfunction in this group. In the group with trifascicular block, the ratio between males and females was 2:1. The predominant presenting feature in these patients was syncope. This is in agreement with previously reported studies of patients with second and third degree AV block distal to the HB, in whom syncope was the most frequent symptom.

Our experience on the natural history of bifascicular block does not allow us to detect a subset of patients at higher risk for progression to AV block. The surface electrocardiographic patterns of bifascicular block were found to be of no prognostic help, since the incidence of heart block was similar with all three varieties of bifascicular conduction defects. A high risk subgroup could not be selected by the presence of neurological symptoms either. Syncope, the most common neurological symptom in patients with bifascicular block, was prospectively evaluated in a recent report. Comparison of patients with and without syncope did not reveal any clinical, electrocardiographic, or electrophysiologic differences. Since only a small percentage of patients manifested syncope secondary to progression of conduction disease, we could not predict the high-risk group. The effect of associated cardiovascular disease on progression of conduction disease was also of little prognostic value.

Conclusions

The cumulative annual incidence of AV block in surviving patients is relatively low, about 11% at 5 years, with 7% reflecting spontaneous block. The incidence of progression to AV block is identical with various forms of bifascicular block. The site of spontaneous AV block varies, and is trifascicular in less than half of the patients. The predominant presenting symptom is usually congestive cardiac failure in patients developing AV nodal block, and syncope in those with trifascicular block. The small total number of patients developing spontaneous trifascicular block prohibits predicting this group with electrophysiologic studies. Additional long-term follow-up is necessary to further delineate the natural history of chronic bifascicular block.

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