Syncope in Patients with Chronic Bifascicular Block

Significance, Causative Mechanisms, and Clinical Implications

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Syncope was prospectively evaluated in 186 patients with chronic bifascicular block. Syncope occurred in 21 of 124 patients with right bundle-branch block and left anterior hemiblock, 3 of 24 patients with right bundle-branch block and left posterior hemiblock, and 6 of 38 patients with left bundle-branch block. Each case was evaluated by using prolonged electrocardiographic monitoring, His bundle recordings, and other indicated studies. Probable and possible causes of syncope included intermittent heart block in five patients, sinus exit block in one patient, orthostatic hypotension in two patients, seizure disorders in three patients, ventricular arrhythmia in nine patients, and acute blood loss in one patient. No cause could be identified in nine patients. Comparison of patients with and without syncope did not show significant differences. Syncope was recurrent in only five patients (four with heart block and one with seizure disorder). The incidence of late sudden death in patients with and without syncope was identical. Syncope in patients with bifascicular block reflected various cardiac and noncardiac causes and tended not to recur. Permanent pacing seemed indicated in only those patients with documented serious bradyarrhythmia.

The intraventricular conduction system consists of three functioning fascicles: the right bundle branch and the anterior and posterior fascicles of the left bundle branch (1). Bifascicular block (block of two fascicles) may thus be defined as right bundle-branch block with left anterior hemiblock, right bundle-branch block with left posterior hemiblock, or left bundle-branch block.

Patients with bifascicular block may have disease in the remaining functioning fascicle and be at risk for development of complete (trifascicular) heart block (1-3). Syncope, in patients with bifascicular block, could suggest intermittent failure of the remaining functioning fascicle and is considered by some to be an indication for permanent, demand pacemaker implantation (2-7).

We examined the significance of syncope in patients with bifascicular block, using clinical, electrocardiographic, and electrophysiologic evaluation. Patients with bifascicular block, but without syncope, are contrasted to those with syncope. Prospective observations are presented on the clinical significance of syncope.

Patients and Methods

Definitions

All definitions are based on the recommendations of the Criteria Committee of the New York Heart Association (8).

Right Bundle-Branch Block: The criteria for electrocardiographic diagnosis of right bundle-branch block included a QRS duration of 0.12 seconds or greater and the presence of a rSR configuration of the QRS in lead V6.

Left Anterior Hemiblock: When the mean frontal QRS axis is more negative than −30°, left anterior hemiblock was diagnosed.

Left Posterior Hemiblock: Criteria for diagnosis of left posterior hemiblock with right bundle-branch block included a mean frontal QRS axis more positive than +110° and absence of right ventricular hypertrophy.

Left Bundle-Branch Block: Diagnosis of left bundle-branch block used the following criteria: [1] QRS duration of 0.12 seconds or greater; [2] the presence of a broad monophasic R wave in lead V6; and [3] ST depression and T-wave inversion in V6.

Syncope: Syncope was defined as a history of transient total loss of consciousness.

Patients

We studied 186 patients with bifascicular block. These patients form part of a group of patients with intraventricular conduction disease prospectively studied in the West Side Medical Center (Cook County Hospital, the West Side Veterans Administration Hospital, and the University of Illinois Hospital), Chicago, Illinois. Criteria for inclusion in the study were that the patient had chronic bifascicular block, signed an informed consent form for electrophysiologic study, and agreed to periodic examination in a conduction disease clinic. Patients with a history of heart block before the study and patients with acute myocardial infarction were excluded from this report. The total group consisted of 142 men and 44 women, with ages ranging from 19 to 93 years, who were studied between January 1970 and September 1973.

Initial Evaluation

All patients were hospitalized when they entered the study. Complete history, physical examination, serial electrocardiograms, and chest X rays were obtained. Clinical diagnoses were established for all the patients.
His bundle (H) electrograms were recorded, by using previously described catheter techniques (9), in all patients when they entered the study. The following intervals were measured in milliseconds: [1] P-A, a measure of intra-atrial conduction (normal, 9 to 45 msec), from the beginning of the P wave to the beginning of the low right atrial electrogram; [2] A-H, a measure of A-V nodal conduction (normal, 54 to 130 msec), measured from the beginning of the atrial electrogram to the first high potential of the His bundle electrogram; and [3] H-V, a measure of intraventricular conduction in the distal His bundle and remaining functioning fascicle (normal, 31 to 55 msec), measured from the beginning of the His bundle electrogram to the initiation of QRS complex at surface electrocardiogram. Atrial pacing at rapid heart rates was used to validate His bundle potentials and to show latent conduction defects (10). Sinus node recovery time was measured with sudden cessation of atrial pacing at a rate of 130/min (normal, 1680 msec or less) (9).

PATIENT FOLLOW-UP

After the initial study, all patients were prospectively studied in a conduction-disease clinic at 4- to 8-week intervals. Each clinic visit consisted of a history, a physical examination, and a 12-lead electrocardiogram. A history of syncope in any patient warranted rehospitalization. Impaired and outpatients were monitored through long periods with the Holler Dynamic Electrocardiographic System* and other indicated studies, such as electroencephalogram and blood urea nitrogen and blood sugar measurements. With this information we attempted to find the cause of syncope. It was impossible with most of the patients to record an electrocardiogram during a syncopal episode. In many of the patients, probable or possible causative mechanisms for syncope could be defined; if not, the patients were classified “syncope-cause unknown.”

Permanent pacemakers were implanted in only those patients with documented A-V block or S-A block. Other probable or possible mechanisms of syncope were treated with appropriate medical therapy. If no cause of syncope was shown, no definitive therapy was instituted. Recurrent syncope without demonstrable cause would have been treated with permanent demand pacemaking, but this never occurred.

There were no patients lost to follow-up during the study. Deaths occurring outside the hospital were thoroughly investigated, and the particular details as well as copies of death certificates were obtained. Deaths occurring instantaneously or within 3 hours of the onset of acute symptoms were classified “sudden.”

ANALYSIS OF DATA

The information obtained during initial hospitalization and subsequent clinic visits was keypunched and stored on database system discs. Specifically designed programs were used for data recall and statistical analysis. A standard t test was used to test the significance of differences in means, and the 2 x 2 chi-square method was used for frequency data analysis.

RESULTS

The 186 patients included in this study were separated into three groups. Group 1 included 124 patients with right bundle-branch block and left anterior hemiblock, group 2 included 24 patients with right bundle-branch block and left posterior hemiblock, and group 3 consisted of 38 patients with left bundle-branch block. The age and sex distributions of these groups are presented in Table 1.

CLINICAL AND ELECTROCARDIOGRAPHIC DATA

Of the 124 patients with right bundle-branch block and left anterior hemiblock, 21 had a history of syncope either before the initial admission (13 patients) or during follow-up (8 patients). Among the 24 patients with right bundle-branch block and left posterior hemiblock, 3 had episodes of syncope that were present at initial admission. Six of the 38 patients with left bundle-branch block had a history of syncope in 4 as an initial event and in 2 as a late event.

The causes of heart disease in the 186 patients are presented in Table 1. There was no significant difference in the incidence of specific causes between patients with and without syncope. Cardiovascular symptoms (other than syncope), physical findings, and electrocardiographic measurements, including P-R interval, QRS duration, and frontal QRS axis, did not differ significantly between those patients with syncope and those without.

ELECTROPHYSIOLOGICAL FINDINGS

Conduction intervals and other electrophysiologic data are presented in Table 2. There was no significant difference in the mean values of electrophysiologic measurements (P-A, A-H, H-V intervals [see Methods section] and sinus node recovery times) between patients with and without syncope in all three groups.

MECHANISMS OF SYNCOPE

Probable and possible mechanisms for syncope are outlined in Table 3.

In group 1 (right bundle-branch block and left anterior hemiblock), for 8 of 21 patients with syncope a probable cause for their symptom was found, whereas in 5 patients a possible cause was shown.

Interruption of heart block occurred in three patients. In Patient 1, syncope occurred on the 21st day. Holler recordings in this patient showed type II 2° A-V block. Electrophysiologic studies showed prolonged A-H (155 msec) and normal H-V intervals. Second degree block distal to His bundle was noted at a paced rate of 142/min. The patient was advised to have a permanent pacemaker inserted. In Patient 2, syncope occurred 2 months before and 3 days after electrophysiologic study; results were within normal limits. Intermittent complete heart block was documented during the episode of syncope, and a permanent pacemaker was implanted. In Patient 3, a 2° A-V block occurred on the 72nd day of follow-up during hospitalization for recurrent syncope. Initial electrophysiologic studies showed an A-H of 180 msec and H-V interval of 65 msec. Repeated electrophysiologic studies showed second-degree block proximal to His bundle. The patient was treated with a permanent pacemaker.

Two patients had syncopal episodes related to postural (orthostatic) hypotension (greater than 50 mm Hg) and induced by alpha methyldopa (Aldomet®†) and phenobarbital. The syncope was recurrent in one patient despite therapy.

† Merck, Sharp and Dohme, West Point, Pennsylvania.
‡ Parke, Davis and Company, Detroit, Michigan.

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Table 1. Clinical Features of 186 Patients with Bifascicular Block

<table>
<thead>
<tr>
<th></th>
<th>Right Bundle-Branch Block and Left Anterior Hemiblock</th>
<th>Right Bundle-Branch Block and Left Posterior Hemiblock</th>
<th>Left Bundle-Branch Block</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Patients with Syncope</td>
<td>Patients Without Syncope</td>
<td>Patients with Syncope</td>
</tr>
<tr>
<td>Age (mean ± SEM), yr</td>
<td>61 ± 3.3</td>
<td>61.2 ± 1.7</td>
<td>56 ± 4.2</td>
</tr>
<tr>
<td>Number of patients</td>
<td>21</td>
<td>103</td>
<td>3</td>
</tr>
<tr>
<td>Men</td>
<td>13</td>
<td>80</td>
<td>3</td>
</tr>
<tr>
<td>Women</td>
<td>8</td>
<td>23</td>
<td>0</td>
</tr>
<tr>
<td>Hypertensive cardiovascular disease</td>
<td>7</td>
<td>37</td>
<td>1</td>
</tr>
<tr>
<td>Arteriosclerotic heart disease</td>
<td>9</td>
<td>25</td>
<td>2</td>
</tr>
<tr>
<td>Primary conduction disease†</td>
<td>2</td>
<td>26</td>
<td>0</td>
</tr>
<tr>
<td>Primary myocardial disease</td>
<td>1</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Other disease</td>
<td>2</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>Syncope</td>
<td>18</td>
<td>103</td>
<td>3</td>
</tr>
</tbody>
</table>

* The P values of all three groups are not significant.
† Bifascicular block with no other demonstrable cardiac disease.

Paroxysmal ventricular tachycardia was considered a possible cause of syncope in five patients who had multiple premature contractions on electrocardiographic monitoring. None of the five has had recurrence of syncope.

In the remaining eight patients with syncope, no causative mechanism for the symptom could be documented. None of these patients has had a recurrence of syncope.

In group 2 (right bundle-branch block and left posterior hemiblock), for two of three patients with syncope, a probable cause of syncope was found. Second degree A-V block (types I and II) was shown in one patient on the 118th day of follow-up during hospitalization for recurrent syncope. Initial electrophysiologic study results were within normal limits. This patient received permanent pacemaker implantation. Another patient had S-A block on the third day of hospitalization for syncope. Electrophysiologic studies showed prolonged sinus node recovery time (2100 msec). A permanent pacemaker was implanted. For the third patient, Holter monitoring suggested ventricular arrhythmia as a cause for syncope, which has not recurred in this patient.

In group 3 (left bundle-branch block), probable causes of syncope were shown for two of six patients with syncope. A 2:1 A-V block was found in one patient on the 130th day of follow-up during hospitalization for recurrent syncope. Initial electrophysiologic studies showed normal A-H and prolonged H-V (74 msec) interval and block distal to the His bundle with atrial pacing at a rate of 127/min. The patient received a permanent pacemaker. Another patient had syncope secondary to acute lower gastrointestinal bleeding during the follow-up period. For three patients, Holter recordings suggested ventricular arrhythmia as a cause of syncope, which has not recurred in any of them. In the sixth patient with syncope, no causative mechanism for the symptom was found. Syncope has not recurred in this patient.

Summary of Follow-Up Data

Group 1: The mean follow-up periods for patients with and without syncope were, respectively, 293 ± 55 and 321 ± 26 (mean ± SEM) days. Syncope was recurrent in three patients (two with A-V block and one with seizure disorder). In the remaining 18 patients syncope was not recurrent. Sudden death occurred in 1 of the 21 patients with syncope (5%) and in 6 of 103 (5%) patients without syncope (NS). The patient with syncope who died suddenly had had "syncope-cause unknown."

Group 2: The mean follow-up periods for patients with and without syncope were, respectively, 392 ± 95 and 295 ± 53 days. Syncope recurred in two patients (in one

Table 2. Conduction Times (Mean ± SEM) in Patients With and Without Syncope

<table>
<thead>
<tr>
<th></th>
<th>Right Bundle-Branch Block and Left Anterior Hemiblock</th>
<th>Right Bundle-Branch Block and Left Posterior Hemiblock</th>
<th>Left Bundle-Branch Block</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conduction times, msec</td>
<td>Patients With Syncope (n = 21)</td>
<td>Patients Without Syncope (n = 103)</td>
<td>Patients With Syncope (n = 3)</td>
</tr>
<tr>
<td>P-A</td>
<td>29 ± 2.7</td>
<td>30 ± 1.0</td>
<td>42 ± 6.4</td>
</tr>
<tr>
<td>A-H</td>
<td>104 ± 5.6</td>
<td>105 ± 3.4</td>
<td>108 ± 9.7</td>
</tr>
<tr>
<td>H-V (&gt;130)</td>
<td>49 ± 2.8</td>
<td>51 ± 1.2</td>
<td>54 ± 11.0</td>
</tr>
<tr>
<td>Sinus node recovery time, msec</td>
<td>941 ± 69.1</td>
<td>1010 ± 26.3</td>
<td>1316 ± 349</td>
</tr>
<tr>
<td>Patients, no. (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A-H (&gt;130)</td>
<td>2 (10)</td>
<td>11 (1)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>H-V (&gt;55)</td>
<td>4 (20)</td>
<td>29 (28)</td>
<td>2 (67)</td>
</tr>
<tr>
<td>Sinus node recovery time (&gt;1680)</td>
<td>1 (5)</td>
<td>2 (2)</td>
<td>1 (33)</td>
</tr>
</tbody>
</table>

* The P values of all three groups are not significant.
with development of advanced A-V block, and in the other because of S-A exit block); both needed permanent pacemakers. In the third patient syncope did not recur. There were no sudden deaths.

**Group 3:** The mean follow-up periods for patients with and without syncope were, respectively, 303 ± 69 and 383 ± 58 days. Syncope recurred in only one patient because of development of advanced A-V block that necessitated permanent pacemaker implantation. Sudden death occurred in one of six patients (in whom the possible cause of syncope was ventricular arrhythmia) with syncope (16%) and in 7 of 32 patients (21%) without syncope (NS).

**Discussion**

It has been suggested that bifascicular block is a form of bilateral bundle-branch disease that frequently precedes complete A-V block. The progression of bifascicular block to advanced or complete heart block has been shown in previous studies (1-3, 11, 12). Lasser, Haft, and Friedberg (2) reported a 10% incidence of complete heart block in patients with right bundle-branch block and left anterior hemiblock. Kulbertus and Collignon (3) reported 16 patients with bifascicular block, all of whom progressed to complete heart block over 8 years. Studies reported by Rosenbaum, Elizari, and Lazzari (1) suggest that 5% to 10% of patients with bifascicular block (right bundle-branch block and left anterior hemiblock) develop complete heart block over many years. In 209 patients with bifascicular block reported by Scanlon, Pryor, and Blount (11), the incidence of complete heart block was 14.4% (30 of 209) over 11 years. DePasquale and Bruno (12) observed a 6% incidence of progression of conduction disease in patients with bifascicular block over a cumulative observation period of 262 patient years. All the previously reported studies concerning progression of conduction disease in patients with bifascicular block have been retrospective.

Syncope in patients with chronic bifascicular block suggests progression of conduction disease to advanced or complete heart block. Several investigators have reported syncopal episodes in these patients secondary to development of intermittent high-grade A-V block. All 16 patients with bifascicular block progressing to complete heart block reported by Kulbertus and Collignon (3) had syncope. Spurrell, Smithen, and Sowton (5) showed syncope in six patients with bifascicular block who progressed to complete heart block. Touboul and Ibrahim (6) reported eight patients with recurrent syncope and bifascicular block, all of them having permanent pacemakers implanted, although progression of conduction disease was proved in only two patients.

There is very little data available concerning causative mechanisms of syncope in patients with bifascicular block. Eleven of 83 patients (13%) reported by DePasquale and Bruno (12) had syncopal episodes. Probable causes for syncope included sinus bradycardia in one patient, tachyarrhythmias in three, high-grade 2° A-V block in two, and complete heart block in two. No cause was found in three patients. Scheinman, Weiss, and Kunkel (7) documented complete heart block as a cause of syncope in 7 of 19 patients with bundle-branch block and Stokes-Adams attacks.

In our series, 30 of 186 patients (16%) had syncopal episodes. Of these 30 patients, only 6 (20%) had bradyarrhythmia that would be amenable to permanent pacing (A-V block in 5 patients and S-A block in 1 patient). In the remaining 24 patients, either no cause of syncope could be shown or causes not amenable to permanent pacing were found.

The clinical course of patients with bifascicular block and syncope was surprisingly benign. In the few patients with recurrent syncope, intermittent A-V block was found, and relief of symptoms was achieved with permanent pacing. In a few of the others, the cause of syncope could be eliminated easily, such as by cessation of alpha methyl dopa therapy because of postural hypotension and by blood transfusion for acute blood loss. In patients with syncope possibly caused by ventricular arrhythmia and patients in whom the cause was unknown recurrent syncope was not noted. No antiarrhythmic drugs were administered in the former group of patients because of the potentially depressing effect of such agents on intraventricular conduction.

Sudden death occurred in 5% of patients with syncope who had right bundle-branch block and left anterior hemiblock, 0% of patients with syncope who had right bundle-branch block with left posterior hemiblock, and 16% of patients with syncope who had left bundle-branch block. The incidence of sudden death in the equivalent groups without syncope was 5%, 0%, and 21% respectively. Thus, the risk of sudden death in the syncope patients was relatively small (except for those with left bundle-branch block) and not greater than in equivalent patients without syncope. The causes of sudden death in the patients with and without syncope could not be determined but might include both brady- and tachy-arrhythmias.

**Table 3.** Probable and Possible Causative Mechanisms of Syncope in Three Groups

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Right Bundle-Branch Block</th>
<th>Right Bundle-Branch Block</th>
<th>Left Bundle-Branch Block</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sino-atrial exit block</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Sinoatrial exit block</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Orthostatic hypotension</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Seizure disorders</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Acute gastrointestinal bleeding</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Ventricular arrhythmia</td>
<td>5</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Unknown</td>
<td>8</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>21</td>
<td>3</td>
<td>6</td>
</tr>
</tbody>
</table>

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ELECTROPHYSIOLOGICAL STUDIES

It has been suggested that recording of His bundle electrograms in patients with bifascicular block might be of value in identifying a group having greater risk for development of high-grade A-V block or sudden death (4, 10, 13-15). In previous series, prolonged H-V interval in patients with bifascicular block and syncope was considered an indication for permanent pacemakers (5-7). In such patients, relief of symptoms resulted from pacemaker insertion. Our results suggest that syncope often does not recur. Relief of symptoms may thus occur without pacemaker insertion.

Electrophysiologic studies were not particularly helpful in this study because findings were similar in patients with and without syncope. This is partially explained by the small percentage of patients manifesting syncope secondary to progression of conduction disease. In selected patients, the electrophysiologic studies were helpful, showing development of 2nd block distal to the His bundle with atrial pacing in two patients, who subsequently progressed to advanced A-V block, and demonstrating prolonged sinus node recovery time in a patient with sinoatrial exit block. It should be emphasized that normal electrophysiologic studies do not preclude subsequent progression of conduction disease.

Conclusions

We conclude that syncope in patients with bifascicular block is common and reflects various cardiac and non-cardiac causes that are not amenable to pacemaker therapy. Syncope in patients with bifascicular block warrants prompt investigation. In patients with bifascicular block, syncope is often relatively benign, tends not to recur, and is associated with an incidence of sudden death similar to that in patients without syncope. It is important to point out that these conclusions are based on observations during an average follow-up period of approximately 1 year. Permanent pacing is indicated in only those patients with syncope and proved heart block or other serious bradyarrhythmia.

Our suggested management of the bifascicular block with syncope would include the following procedures: [1] prompt hospitalization, careful history, and physical examination; [2] serial electrocardiograms with prolonged in-patient and, if necessary, outpatient monitoring; [3] electrophysiologic studies, including atrial pacing, since these studies will be occasionally valuable; and [4] other tests as indicated (such as blood sugar and electroencephalogram). Pacemaker therapy should be used for patients with proved bradyarrhythmia. Striking abnormal electrophysiologic findings, such as repetitive block distal to the His bundle with atrial pacing and markedly prolonged sinus node recovery time, may be indications for pacemaker therapy.

Addendum

Since submission of this manuscript, we had another patient with syncope and bifascicular block in whom electrophysiologic studies were helpful. The patient had right bundle-branch block, left anterior hemiblock, an H-V interval of 40 msec, a prolonged left posterior division effective refractory period (720 msec), and type 2 block distal to the His bundle, with atrial pacing at 80/min.

These findings suggested severe trifascicular disease, which was supported by subsequent development of spontaneous second-degree A-V block (Mobitz type 2). The patient was treated with a permanent demand pacemaker.

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