Electrophysiological and Clinical Observations in Patients with Alternating Bundle Branch Block

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SUMMARY Electrophysiological studies (His bundle recordings and atrial stimulation) were performed in nine patients who manifested periods of both right and left bundle branch block (RBBB and LBBB).

In seven of the patients, alternating bundle branch block appeared to reflect intermittent or chronic bundle branch block superimposed on incomplete (but electrocardiographically complete) block of the contralateral bundle branch. In three of these seven, shift from one bundle branch block pattern to the other was associated with reproducible change in H-V (mean change 30 msec), and could be induced by alteration of cardiac rate with carotid massage, coupled atrial stimulation, and rapid atrial pacing. In one of the seven, RBBB with a P-R of 0.20 seconds preceded chronic LBBB with a P-R of 0.24 seconds, implying that RBBB had been incomplete. In three of the seven, although a definite mechanism of alternation could not be demonstrated, transient contralateral bundle branch block occurred superimposed on chronic ipsilateral bundle branch block, implying that the ipsilateral block was incomplete.

Two patients manifested periods of narrow QRS, LBBB, RBBB, and paroxysmal A-V block. Based upon pathological data (one case), this pattern appeared to reflect a lesion involving the distal His bundle and proximal bundle branches.

In the total group of patients, clinical course was primarily determined by the severity of heart disease and not by occurrence of A-V block. The conduction defect in the majority of patients was surprisingly benign.

ALTERNATING RIGHT AND LEFT bundle branch block is an electrocardiographic diagnosis suggesting the presence of bilateral bundle branch disease. Previous clinical observations in selected cases have suggested a poor prognosis with occurrence of heart block, syncope, and sudden death. Pathological studies in patients with alternating bundle branch block have revealed destructive lesions involving the His bundle and both bundle branches.

Although Rosenbaum and Lepeschkin suggested varying incomplete delays in the right and left bundle branches as the basis of alternation, there have been no systematic studies of the electrophysiological mechanisms of alternation. In the present study, we attempted to delineate the electrophysiological mechanisms of alternating bundle branch block, and to correlate these with clinical manifestations and prognosis.

Material and Methods

Definitions

Right bundle branch block: Criteria for right bundle branch block included a QRS duration of 0.12 sec or greater with a rSR or qR configuration of the QRS complex in V1. Left bundle branch block: Criteria for left bundle branch block included a QRS duration of 0.12 sec or greater, the presence of a broad notched monophasic R wave in V6, and ST depression and T wave inversion in V6. Alternating bundle branch block: Alternating bundle branch block was diagnosed when conducted periods of right bundle branch block and left bundle branch block were noted in a patient on the same or different electrocardiograms.

Patient Selection

The patients in this study included all patients with alternating bundle branch block seen by the Cardiology Section in the Chicago West Side Medical Center between February 1970 and June 1975, who agreed to electrophysiological studies.

In each patient, retrospective analysis of electrocardiograms and clinical features were performed from the time of first detection. The date of the first electrocardiographic documentation of alternating bundle branch block was noted. Clinical diagnoses were established utilizing history, physical examination, laboratory data, serial electrocardiograms, and radiologic examinations.

Electrophysiological Studies

Informed consent was obtained. His bundle (H) electrograms were recorded utilizing previously described catheter techniques. Incremental atrial pacing and measurement of refractory periods with atrial extrastimulus were performed in some of the patients. The following were measured in msec: 1) A-H interval (normal 54 to 130 msec), from the first high-frequency potential of the low right atrial electrogram to the first high-frequency potential of the His bundle electrogram. 2) H-V interval (normal 31 to 55 msec), from the first high-frequency potential of the His bundle electrogram to the earliest deflection of the QRS complex detected from multiple surface leads. 3) Effective refractory period of the ventricular specialized conducting system (VSCS), defined as the longest H1-H2 (H1 was the His bundle response of the sinus or driven beats; H2 was the His bundle response of the test beat) at which H2 failed to conduct to the ventricles.
Follow-up

After initial detection, each patient was prospectively followed in a conduction disease clinic at one to two monthly intervals, or at a shorter interval when clinically indicated. Each clinic visit consisted of a history, physical examination and electrocardiogram. If syncope occurred during the follow-up period, the patient was rehospitalized in an attempt to define the causative mechanisms. Prolonged periods of electrocardiographic monitoring with the Holter Dynamic Electrocardiographic System were performed to detect transient bradyarrhythmias. If death occurred, the cause of death was analyzed and determined. For this study, the period of observation was defined as the period from the date of first electrocardiographic documentation of alternating bundle branch block to June 30, 1975 or to the date of death.

Results

Nine patients were studied. Clinical features and electrocardiographic findings are summarized in table 1. The results of electrophysiological studies are summarized in table 2.

Case 1

This was a 42-year-old asymptomatic female with primary conduction disease (no demonstrable organic heart disease). Spontaneous intermittent alternation of left and right bundle branch block pattern had been documented since 6-4-73. Left bundle branch block pattern was associated with a P-R interval of 0.20 sec, while right bundle branch block pattern was associated with left axis deviation (−60°) and a P-R interval of 0.24 sec.

Electrophysiological studies performed on 9-20-73 revealed sinus rhythm with a cycle length of 600 msec, right bundle branch block and left axis deviation. A-H interval was 110 msec and H-V interval was 100 msec. Second degree block distal to the His bundle was observed during atrial pacing at a rate of 120/min or greater (fig. 1). Shift of QRS morphology from right bundle branch block pattern to left bundle branch block pattern was induced by carotid massage with slowing of sinus rhythm to cycle lengths (H-H intervals) of 940 msec or greater (fig. 2). Return to right bundle branch block occurred when cycle length returned to 760 msec or less. A similar shift to left bundle branch block was also noted following sudden cessation of rapid atrial pacing when the escape sinus beat achieved an H-H interval greater than 950 msec. Shift of right to left bundle branch block pattern was also noted in the first conducted beat following two consecutive blocked beats (one blocked distal to, and the other proximal to, the His bundle) during pacing induced second degree A-V block (fig. 1). When QRS morphology shifted from right to left bundle branch block pattern, A-H interval remained unchanged and H-V interval shortened to 60 msec (figs. 1 and 2). These findings suggested that right bundle branch block was rate-dependent (tachycardic) and that left bundle branch block was incomplete at sinus rates, with enough delay to produce electrocardiographically complete left bundle branch block.

Table 1. Clinical Feature and Electrocardiographic Findings

<table>
<thead>
<tr>
<th>Case number</th>
<th>Age/Sex</th>
<th>Cardiovascular diagnosis</th>
<th>Type of alteration</th>
<th>P-R interval (sec)</th>
<th>QRS morphology</th>
<th>QRS morphology alteration</th>
<th>Date of first ECG documentation</th>
<th>Clinical course and duration of observation</th>
<th>Specimen</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>42 F</td>
<td>Primary conduction disease</td>
<td>Int</td>
<td>0.20</td>
<td>LBBB + LAD</td>
<td>LBBB + LAD</td>
<td>6-4-73</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>67 M</td>
<td>Primary myocardial disease</td>
<td>Int</td>
<td>0.20</td>
<td>LBBB + LAD</td>
<td>LBBB + LAD</td>
<td>12-6-74</td>
<td>Alive (960 days)</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>63 F</td>
<td>Neurologic aortic stenosis</td>
<td>Int</td>
<td>0.18</td>
<td>LBBB + LAD</td>
<td>LBBB + LAD</td>
<td>12-6-74</td>
<td>Alive (240 days)</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>82 M</td>
<td>Aortic stenosis</td>
<td>Tran</td>
<td>0.22</td>
<td>LBBB + LAD</td>
<td>LBBB + LAD</td>
<td>8-6-70</td>
<td>Alive (1023 days)</td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>81 F</td>
<td>Primary myocardial disease</td>
<td>Tran</td>
<td>0.18</td>
<td>LBBB + LAD</td>
<td>LBBB + LAD</td>
<td>1-10-71</td>
<td>Alive (130 days)</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>81 M</td>
<td>Primary myocardial disease</td>
<td>Tran</td>
<td>0.18</td>
<td>LBBB + LAD</td>
<td>LBBB + LAD</td>
<td>1-10-71</td>
<td>Alive (130 days)</td>
<td>No</td>
</tr>
<tr>
<td>7</td>
<td>50 M</td>
<td>Carotid sinus</td>
<td>Tran</td>
<td>0.20</td>
<td>LBBB + LAD</td>
<td>LBBB + LAD</td>
<td>10-16-74</td>
<td>Recurrent (120 days)</td>
<td>No</td>
</tr>
<tr>
<td>8</td>
<td>52 M</td>
<td>Aortic stenosis</td>
<td>Tran</td>
<td>0.20</td>
<td>LBBB + LAD</td>
<td>LBBB + LAD</td>
<td>1-28-74</td>
<td>Alive (80 days)</td>
<td>No</td>
</tr>
<tr>
<td>9</td>
<td>81 F</td>
<td>Acute myocardial infarction</td>
<td>Tran</td>
<td>0.22</td>
<td>LBBB + LAD</td>
<td>LBBB + LAD</td>
<td>1-25-74</td>
<td>Temporary PMR on day 1-20</td>
<td>No</td>
</tr>
<tr>
<td>10</td>
<td>52 F</td>
<td>Female</td>
<td>Tran</td>
<td>0.24</td>
<td>LBBB + LAD</td>
<td>LBBB + LAD</td>
<td>1-28-74</td>
<td>Alive (80 days)</td>
<td>No</td>
</tr>
</tbody>
</table>

Abbreviations: LBBB = Left Bundle Branch Block; RBBB = Right Bundle Branch Block; PMR = Permanent PMR; T = Transient
pattern.1, 2, 13-16 The presence of left axis deviation with right bundle branch block suggested that there may have been total failure of conduction in the left anterior division, and that left bundle branch block reflected superimposed critical delay in the left posterior division or main stem. The changes in P-R interval with shift in QRS morphology reflected change in H-V interval.16-18

A prophylactic permanent demand pacemaker (set at escape rate of 60/min) was inserted after electrophysiological study because of the demonstration of block distal to the His bundle with right atrial pacing. During the subsequent period of observation, advanced A-V block and/or syncope were not noted for a follow-up period of 649 days.

Case 2

This was a 67-year-old male with hypertensive heart disease and severe congestive heart failure. Spontaneous intermittent alternation of both left and right bundle branch block pattern was noted on 12-9-74 when he was admitted to the hospital. Left bundle branch block pattern was associated with a P-R interval of 0.20 sec. Right bundle branch block pattern was associated with a QRS axis of -60° and a P-R interval of 0.24 sec.

Electrophysiological study performed on 12-16-74 revealed sinus rhythm with a cycle length of 620 msec. Both right and left bundle branch block patterns were observed during study. A-H interval was 85 msec and H-V interval was 105 msec with right bundle branch block pattern. Shift to left bundle branch block pattern occurred following cessation of rapid atrial pacing when the first sinus escape beat occurred at H-H intervals of 815 msec or greater. Once left bundle branch block pattern was initiated, all the subsequent sinus beats were conducted with left bundle branch block pattern (fig. 3A). With left bundle branch block pattern, A-H interval remained 85 msec and H-V interval shortened to 80 msec. Shift from left to right bundle branch block could be induced with atrial extrastimuli with H₁-Hₑ coupling intervals equal to or less than 550 msec (fig. 3B).

Once right bundle branch block pattern was initiated, all subsequent sinus beats were conducted with right bundle branch block pattern. These electrophysiological findings suggested incomplete left bundle and tachycardia rate-dependent right bundle branch block.1, 2, 13-16 The change in P-R interval associated with the change in bundle branch block pattern was due to change in H-V interval.16-18 The ability to manifest both right and left bundle branch block pattern during sinus rhythm at identical cycle lengths could be explained by the long refractory period of the right bundle relative to sinus cycle length. The maintenance of right bundle branch block pattern was probably due to repetitive retrograde concealed conduction to the right bundle.15, 16 Left axis deviation during right bundle branch block has the same implication as in case 1.

This patient died of congestive heart failure 14 days after

<table>
<thead>
<tr>
<th>Case number</th>
<th>Sinus cycle length (msec)</th>
<th>QRS morphology</th>
<th>H-V (msec)</th>
<th>Type II block distal to H during incremental AP (rate/min)</th>
<th>ERP-VSCS (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>600</td>
<td>RBBB + LAD</td>
<td>100</td>
<td>Yes (≥120/min)</td>
<td>(—)</td>
</tr>
<tr>
<td>2</td>
<td>≥940*</td>
<td>LBBB</td>
<td>60</td>
<td>No (1:1 A-V conduction up to paced heart rate of 150/min)</td>
<td>&lt;445</td>
</tr>
<tr>
<td>3</td>
<td>620</td>
<td>LBBB</td>
<td>60</td>
<td>No (1:1 A-V conduction up to paced heart rate of 140/min)</td>
<td>(—)</td>
</tr>
<tr>
<td>4</td>
<td>≥1800*</td>
<td>LBBB</td>
<td>55</td>
<td>Yes (spontaneous)</td>
<td>(—)</td>
</tr>
<tr>
<td>5</td>
<td>740</td>
<td>LBBB</td>
<td>54</td>
<td>No (1:1 A-V conduction up to paced heart rate of 150/min)</td>
<td>(—)</td>
</tr>
<tr>
<td>6</td>
<td>923</td>
<td>LBBB</td>
<td>65</td>
<td>No (1:1 A-V conduction up to paced heart rate of 170/min)</td>
<td>&lt;370</td>
</tr>
<tr>
<td>7</td>
<td>952</td>
<td>LBBB</td>
<td>59</td>
<td>No (1:1 A-V conduction up to paced heart rate of 170/min)</td>
<td>(—)</td>
</tr>
<tr>
<td>8</td>
<td>777</td>
<td>Narrow</td>
<td>36</td>
<td>No (1:1 A-V conduction up to paced heart rate of 210/min)</td>
<td>(—)</td>
</tr>
<tr>
<td>9</td>
<td>760 (1st study)</td>
<td>LBBB</td>
<td>39</td>
<td>(—)</td>
<td>(—)</td>
</tr>
<tr>
<td>2</td>
<td>780 (2nd study)</td>
<td>RBBB</td>
<td>48</td>
<td>(—)</td>
<td>(—)</td>
</tr>
</tbody>
</table>

*Slowing of sinus rhythm with carotid massage or following cessation of rapid atrial pacing.

Abbreviations: AP = atrial pacing; ERP-VSCS = effective refractory period of ventricular specialized conducting system; RBBB = right bundle branch block; LBBB = left bundle branch block; Atrial Fib. = atrial fibrillation.
admission. A-V block was not noted during the period of observation.

Case 3

This was a 63-year-old female with adenocarcinoma of lung and necrotizing arteritis. Electrocardiogram on 5-14-74 revealed anteroseptal myocardial infarction and left axis deviation (−60°). On 6-14-74, she suffered an episode of prolonged chest pain with enzyme elevation consistent with acute myocardial infarction. Left bundle branch block and type II second degree A-V block were observed (fig. 4). His bundle recordings at the time of left bundle branch block revealed an A-H interval of 75 msec, an H-V interval of 80 msec, and spontaneous type II block distal to the His bundle. A temporary demand ventricular pacemaker was inserted. On 6-16-74, alternating bundle branch block was noted (fig. 4). P-R interval was 0.18 sec during left bundle branch block and 0.22 sec during right bundle branch block. This was followed by spontaneous intermittent alternations of both right and left bundle branch block pattern. Subsequently, type II A-V block disappeared and right bundle branch block was seen with occasional periods of left bundle branch block.

Electrophysiological study was again performed on 7-2-74 and revealed sinus rhythm with a cycle length of 780 msec, right bundle branch block and left axis deviation. A-H interval was 124 msec and H-V interval was 80 msec (fig. 5A and B). 2:1 block distal to the His bundle was observed during rapid atrial pacing at rates equal to or greater than 160/min (fig. 5B). The effective refractory period of the ventricular specialized conduction system was 560 msec during sinus rhythm. Shift from right to left bundle branch block pattern was noted in the first escape sinus beat following cessation of rapid atrial pacing when H-H intervals were 1080 msec or greater (fig. 5A). Shift from right to left bundle branch block pattern was associated with shortening of H-V interval from 80 to 55 msec. These findings again suggested incomplete left bundle branch block and a tachycardiac rate-dependent right bundle branch block.1, 5, 13-16

The temporary pacemaker was withdrawn after the second electrophysiological study. The patient has been asymptomatic without A-V block for a period of 364 days.

Case 4

This was a 92-year-old male with arteriosclerotic heart disease. He had documented atrial fibrillation with left axis deviation (−80°) and prolonged periods of right and left bundle branch block pattern since 8-17-70 (fig. 6).

Electrophysiological study performed on 12-5-72 revealed atrial fibrillation with left bundle branch block pattern and an H-V interval of 54 msec. Right bundle branch block pattern was not observed during the study.

Figure 2. Recordings from Case 1, showing incomplete block of left bundle and rate-dependent block of the right bundle. H-H and H-V intervals were 105 msec. Progression lengthening of cycle (H-H interval) was induced with carotid massage. Note that the first three beats with H-H intervals less than 900 msec were conducted with RBBB and an H-V of 100 msec. The subsequent three beats with H-H intervals longer than 940 msec were conducted with LBBB and an H-V of 60 msec.

Figure 3. Recordings from Case 2, showing incomplete block of left bundle and tachycardia dependent block of right bundle. HRA represents high right atrial electrogram. In panel A, the first four beats were atrial paced beats at a rate of 120/min and were conducted with RBBB pattern with an H-V of 105 msec. The fifth beat was the first sinus return beat following cessation of atrial pacing with an H-H interval of 815 msec. This beat and subsequent beats were conducted with LBBB pattern with an H-V of 80 msec. In panel B, the first two beats were sinus beats with LBBB pattern; the third beat was a coupled paced premature beat with an H-H interval of 500 msec and was conducted with RBBB pattern. Note that all the subsequent sinus beats were conducted with RBBB pattern.
workup, including prolonged electrocardiographic monitoring, failed to reveal a causal mechanism of syncope. She was subsequently followed closely in the clinic without insertion of a permanent pacemaker. Syncope did not recur. A-V block was not observed over a total observation period of 1623 days.

Case 6

This was an 81-year-old male with primary myocardial disease and chronic left bundle branch block with a P-R interval of 0.20 sec. On 2-21-75, a transient episode of right bundle branch block and left axis deviation (−60°) was documented. P-R interval during right bundle branch block was 0.20 sec.

Electrophysiological studies on 2-28-75 revealed sinus rhythm with a cycle length of 923 msec, left bundle branch block and left axis deviation. A-H interval was 85 msec and H-V interval was 65 msec. 1:1 A-V conduction was noted up to the atrial paced rate of 150/min. Right bundle branch block pattern was not observed.

Syncope and/or A-V block were not observed during the observation period of 130 days.

Case 7

This was a 50-year-old male with coarctation of the aorta surgically corrected in 1956. Right bundle branch block, left axis deviation (−45°), and a P-R interval of 0.20 sec had been noted between 1969 and 1972. On 6-15-73, left bundle branch block and a P-R interval of 0.24 sec was noted and has persisted ever since (fig. 7).

Electrophysiological studies on 8-15-73 revealed sinus rhythm with a cycle length of 952 msec and left bundle branch block. A-H interval was 124 msec and H-V interval was 59 msec. 1:1 A-V conduction was noted up to an atrial paced rate of 170/min. Right bundle branch block pattern was not noted.

Although the mechanism of alternating bundle branch block was not demonstrated during electrophysiological study, the observation of right bundle branch block with a P-R of 0.20 sec preceding chronic left bundle branch block with a P-R of 0.24 sec suggested that right bundle branch block was incomplete.

This patient died of acute myocardial infarction on 2-12-73. Advanced A-V block and syncope were not noted during the period of observation (910 days).

Case 5

This was an 81-year-old female with primary conduction disease and chronic right bundle branch block, left axis deviation (−80°) with a P-R interval of 0.18 sec. On 1-19-71, a transient episode of left bundle branch block pattern was documented with a P-R interval of 0.18 sec without significant change in heart rate. All subsequent electrocardiograms revealed right bundle branch block pattern.

Electrophysiological study on 2-9-71 revealed sinus rhythm with a cycle length of 740 msec, right bundle branch block and left axis deviation. A-H interval was 80 msec and H-V interval was 47 msec. 1:1 A-V conduction was noted up to an atrial pacing rate of 140/min. Left bundle branch block pattern was not observed.

She had a single syncopal episode on 6-14-74. Extensive
Syncope and A-V block were not noted during the observation period of 746 days.

Case 8

This was a 52-year-old male with calcific aortic stenosis. He developed recurrent syncope on 1-29-74. Electrocardiographic monitoring revealed the following patterns: 1) narrow QRS complexes with P-R interval of 0.18 sec, 2) right bundle branch block with a P-R of 0.20 sec, 3) left bundle branch block with a P-R of 0.20 sec, 4) paroxysmal heart block with prolonged ventricular asystole. These events occurred over a period of two days. A temporary ventricular demand pacemaker was inserted. Subsequently, he resumed 1:1 A-V conduction with narrow QRS complex.

Electrophysiological study performed on 2-19-74 revealed sinus rhythm with a cycle length of 777 msec and narrow QRS complex. A-H interval was 88 msec and H-V interval was 36 msec. 1:1 A-V conduction was noted up to an atrial paced rate of 210/min. Measurement of refractory periods at several cycle lengths failed to reveal abnormalities.

This patient died on 2-27-74 due to cerebral embolism. Postmortem examination revealed calcific impingement involving the distal His bundle and proximal bundle branches. Detailed clinical, electrophysiological, and pathological data concerning this patient were recently reported.19

Case 9

This was a 61-year-old female with acute myocardial infarction on 5-25-74. Admission electrocardiogram revealed intact A-V conduction with a P-R interval of 0.22 sec, narrow QRS and acute anteroseptal myocardial infarction (fig. 8A). Four hours after admission, she developed left bundle branch block pattern with a P-R interval of 0.22 sec (fig. 8B). Electrophysiological studies revealed sinus rhythm with a cycle length of 760 msec and left bundle branch block. A-H interval was 136 msec and H-V interval was 39 msec. Left bundle branch block pattern lasted for 2-3 hours with subsequent resumption of narrow QRS. During the period between 5-25 and 5-27, QRS complexes were predominantly narrow (fig. 8C) with several transient episodes of left bundle branch block. On 5-28-74, right bundle branch block with a P-R interval of 0.24 sec was noted (fig. 8D). His bundle recordings revealed an A-H interval of 135 msec and an H-V interval of 48 msec. Split H potentials were not noted during either electrophysiological study. Right bundle branch block pattern lasted for several hours with return to narrow QRS. She subsequently manifested several episodes of both left and right bundle branch block pattern from 5-28 to 5-30. On 5-31, during sinus rhythm with narrow QRS, she suddenly developed paroxysmal heart block with a slow ventricular escape rhythm (fig. 8E). A temporary demand ventricular pacemaker was inserted. Her subsequent hospital course was uneventful. The pacemaker was removed two weeks later. Subsequent follow-up (381 days) revealed no syncope or A-V block.

Discussion

Electrophysiological Mechanisms

Alternating bundle branch block is an unusual manifestation of intraventricular conduction disease. In some previously reported cases, the alternation of one bundle branch block pattern with another was associated with prolongation of P-R interval and increase in heart rate.1 3 5 6 7 In other cases, changes in P-R interval and factors initiating alternation of bundle branch block pattern were not demonstrated.1 3 5 6 7 Rosenbaum and Lepschkin postulated that alternating bundle branch block depended upon incomplete block with enough delay to produce an electrophysiographically complete bundle branch block pattern in
one bundle and intermittent block (of greater severity) in the other bundle.\(^2\) Thus, ipsilateral bundle branch block pattern occurred with incomplete block in one bundle and normal or nearly normal conduction in the contralateral bundle. The contralateral bundle branch block pattern was seen when intermittent block of the other bundles became superimposed on the previous ipsilateral incomplete block.

Recently, utilizing His bundle recording and atrial extrastimulus technique, it has been demonstrated that incomplete bundle branch block could produce an electrocardiographic pattern of complete block.\(^3,\)\(^4\) This demonstration depended upon the incompletely blocked bundle branch having a shorter refractory period than the more normally conducting bundle. When these conditions were fulfilled, coupled stimulation provoked the contralateral bundle branch block pattern.\(^5\) A similar phenomenon could occur in a patient with incomplete block in one bundle branch, and intermittent block of the contralateral bundle. This intermittent block could be sporadic or rate-dependent. Since many patients with rate-dependent bundle branch block ultimately develop permanent bundle branch block, shift in bundle branch block pattern could become permanent.\(^6\)

These electrophysiological mechanisms are diagrammatically represented in figure 9, in which incomplete block in the left bundle is postulated. In figure 9A, an electrocardiographic pattern of left bundle branch block occurs and H-V interval reflects conduction time in distal His bundle and right bundle branch. In figure 9B, rate-dependent right bundle branch block occurs. H-V interval reflects conduction time of the distal His bundle and left bundle, and is lengthened equivalent to delay in the left bundle. Cases 1, 2, and 3 of the present study appear to reflect this type of mechanism. Maintenance of right bundle branch block, once initiated, depended upon repetitive retrograde concealed conduction to the right bundle branch. If right bundle branch recovery was rapid despite concealed conduction, right and left bundle branch block could alternate from beat to beat (fig. 4, middle panel). If the cycle length of spon-

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**Figure 8.** Electrocardiograms from Case 9, showing acute anteroseptal myocardial infarction with alternation of QRS morphology and paroxysmal heart block. In panel A, at 12:00 a.m. on 5-25-74, electrocardiogram showed sinus rhythm and narrow QRS complex with P-R interval of 0.22 sec. In panel B, four hours later, electrocardiogram showed sinus rhythm and LBBB with P-R interval of 0.22 sec. In panel C, at 9:00 a.m. on 5-27-74, electrocardiogram showed sinus rhythm and narrow QRS complex with P-R of 0.22 sec. In panel D, at 8:30 a.m. on 5-28-74, electrocardiogram showed sinus rhythm and RBBB with P-R of 0.24 sec. In panel E, at 8:50 a.m. on 5-29-74, rhythm strip from monitor lead showed sudden development of paroxysmal heart block with ventricular escape rhythm. Note also ventriculo-phasic sinus arrhythmia during the block.

**Figure 9.** Diagrammatic representation of the mechanisms of alternating BBB, in which incomplete block of the left bundle and rate dependent or non rate dependent block of the right bundle are postulated (see text for discussion). RB = right bundle; LB = left bundle.
taneous sinus rhythm was close to the refractory period of the right bundle, prolonged periods of both left and right bundle branch block pattern could be seen. Shift from one bundle branch block pattern to the other could be induced by premature atrial beats or with minimal change in heart rate (fig. 3). Figure 9C demonstrates incomplete left bundle branch block with intermittent non-rate-dependent right bundle branch block. H-V interval again reflects conduction time in the distal His bundle and the left bundle and is lengthened. With this mechanism, electrophysiological study would not delineate the underlying mechanism of alternation, unless intermittent right bundle branch block occurred during the study. Cases 4, 5, and 6 of the present study may reflect this type of mechanism. In both figures 9B and C, if permanent right bundle branch block developed, a fixed right bundle branch block pattern would result. H-V interval would reflect conduction time of the distal His bundle and left bundle, and would be lengthened. Case 7 of the present study could reflect this mechanism. In summary, the occurrence of alternating bundle branch block in these patients appears to depend upon incomplete block in one bundle and intermittent block in the contralateral bundle. It is curious that the pattern of alternation observed in these seven cases was always that of right bundle branch block with left axis deviation and left bundle branch block. This suggests that all patients had fixed left anterior divisional block, and that alternation reflected disease in the right bundle and left posterior division, with incomplete block of one and intermittent (or chronic) block of the other.

The above mechanisms do not readily explain the spontaneous alternation of narrow QRS complexes, right bundle branch block, and left bundle branch block observed in Cases 8 and 9 of the present study. In both patients, alternation was unpredictable and not related to change of heart rate, P-R interval or H-V interval. Both patients manifested paroxysmal heart block. The postulated mechanisms of alternation in these two patients are diagrammatically represented in figure 10. A destructive lesion involving the distal His bundle and proximal bundle branches is postulated. This lesion could cause intermittent block in both left and right bundles. In figure 10A, both bundles are conducting and the QRS complex is narrow. In figure 10B, intermittent left bundle branch block occurs, while in 10C, intermittent right bundle branch block occurs. In figure 10D, intermittent block in both bundles occurs and paroxysmal heart block is manifested. Postmortem examination in Case 8 revealed calcific aortic stenosis with calcific impingement upon the pars membranacea resulting in compression of the distal His bundle and marked disruption of the proximal portions of both bundle branches. These pathologic findings are consistent with the postulated mechanisms. A similar mechanism could explain the alternations in Case 9 with acute anteroseptal myocardial infarction. El-Sherif et al. have reported various degrees of block in the distal His bundle and proximal bundle branches following ligation of the canine anterior septal artery. Case 9 resembles El-Sherif’s dog model.

Clinical Significance

This study does not allow final conclusions in regard to indications for prophylactic permanent pacing in patients with alternating bundle branch block. However, several guidelines are suggested. In all patients with alternating bundle branch block, permanent pacing is indicated if recurrent syncope or A-V block are observed. In patients with asymptomatic alternating bundle branch block without narrow QRS complexes, close follow-up without insertion of permanent pacemakers appears justified. Since the block in one bundle is incomplete, the long term prognosis in these patients probably resembles those in patients with bifascicular block and prolonged H-V interval. In patients with alternation of narrow QRS, right and left bundle branch block, insertion of permanent pacemakers may be advisable. In patients with alternating bundle branch block complicating acute myocardial infarction, temporary pacemakers should be inserted regardless of mechanism and H-V interval. In these patients, permanent pacemakers may not be necessary since the block can improve. These preliminary conclusions should be further clarified with additional prospective follow-up study in a larger group of patients followed for longer periods.

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References


![Diagram](http://circ.ahajournals.org)
Mitral Valve Prolapse in One Hundred Presumably Healthy Young Females

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SUMMARY Clinical, electrocardiographic, phonocardiographic, and echocardiographic examinations were performed in 100 presumably healthy young females. Treadmill testing and ambulatory electrocardiographic monitoring were performed in a selected group of these subjects. Phonocardiograms, recorded with the subjects supine at rest, after inhalation of amyl nitrite, and in the upright position, revealed a 17% incidence of nonejection clicks and/or late or mid- to late systolic murmurs (PHONO-MSCLSM). Echocardiographic studies were performed in the second, third, fourth, and fifth intercostal space with emphasis on the importance of transducer angulation on the chest. Studies obtained with the transducer perpendicular to the chest in the sagittal plane, or pointing cephalad at a time when both mitral leaflets and left atrium are recorded, are optimal to study the mitral systolic motion. With the transducer in this position, 21 subjects were found to have pansystolic or late systolic prolapse, as previously defined on the echocardiogram. The presence of these echocardiographic findings was statistically related to the presence of PHONO-MSCLSM. Other echocardiographic patterns were identified and their relation to PHONO-MSCLSM and transducer position is discussed. Ten subjects with both echocardiographic evidence of mitral valve prolapse and PHONO-MSCLSM were identified (group EP), while 18 other subjects had either echocardiographic or phonocardiographic findings suggestive of mitral valve abnormality (group EorP). Seventy-two subjects had no abnormality (group noEP). The incidence of various clinical, electrocardiographic, and echocardiographic findings in these three groups was determined. Some findings said to be common in patients with proven mitral valve prolapse were seen more frequently in group EP subjects.

Echocardiographic and phonocardiographic findings suggesting mitral valve abnormalities were found more commonly than expected in a population of presumably healthy young females.

NURBEO REPORTS have documented the association of abnormalities of the mitral valve with nonejection clicks and late systolic murmurs.1-3 Recent studies correlating echocardiographic findings suggesting mitral valve prolapse and left ventricular angiography have demonstrated the high sensitivity of the ultrasound technique.10-13 Also, it has been shown that an abnormal echocardiographic systolic motion of the mitral valve could be demonstrated in patients who did not present with the typical auscultatory findings of mid- to late systolic click or murmur.12-14 We have been impressed recently by the frequency with which echocardiographic patterns reported previously to occur in patients with angiographically proven mitral valve prolapse were recorded in apparently healthy subjects. This study was undertaken in order to define more accurately the normal echocardiographic pattern of the mitral valve during systole, to observe the prevalence of the echocardiographic patterns suggesting mitral valve prolapse in a population of presumably healthy young females, and to correlate these patterns with specific clinical, phonocardiographic, and electrocardiographic features.

Methods

Patient Selection

Studies were performed in 101 paid volunteers recruited through advertisements placed in campus newspapers and on posters. Females aged 17-35 years making a statement of presumed good health were accepted for study. After ob-
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D Wu, P Denes, R C Dhingra, F Amat-Y-Leon, C R Wyndham, R Chuquimia and K M Rosen

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