His Bundle Electrogram in Bundle-Branch Block

By N. Ranganathan, M.D., R. Dhurandhar, M.D., J. H. Phillips, M.D., and E. D. Wigle, M.D.

SUMMARY

Because a great majority of patients with chronic complete heart block have bilateral bundle-branch lesions, it becomes important to recognize earlier degrees of bilateral bundle-branch block. The H-V interval in the His bundle electrogram during unilateral bundle-branch block reflects the conduction primarily through the contralateral bundle branch, and thus the His bundle electrogram in bundle-branch block (BBB) provides information regarding the functional status of the contralateral bundle branch in addition to helping in the localization of defects elsewhere in the conduction system.

His bundle electrograms were obtained in 20 patients with BBB and 13 patients without BBB. The following results were obtained from this study: (1) Prolonged P-R interval in the absence of BBB indicated delayed conduction through the A-V node. (2) Prolonged P-R interval in the presence of BBB indicated delayed conduction through the contralateral bundle branch or through the A-V node, or both. (3) Delayed conduction through the contralateral bundle branch in BBB occurred in the presence of a normal P-R interval and could only be detected by demonstrating a prolonged H-V time in the His recording. (4) In alternating BBB the His recording clearly demonstrated that the changing P-R interval was related to varying conduction through the bundle branches. (5) Finally, it has been demonstrated that the Wenckebach (Mobitz type I) type of decremental conduction can occur in the bundle branches or Purkinje system as well as in the A-V node.

It is concluded that His bundle electrograms provide valuable information concerning the nature and management of conduction disturbances in patients with bundle-branch disease.

Additional Indexing Words:
Alternating bundle-branch block
Bundle-branch block with long P-R interval
First-degree block in the bundle branches
Second-degree block (Mobitz type I or Wenckebach and Mobitz type II) in the bundle branches

SINCE the majority of patients with chronic complete heart block have bilateral bundle-branch lesions,1-9 the ability to recognize disorders of conduction in the bundle branches at an earlier stage would help identify patients who would be at high risk for subsequent development of complete heart block and Stokes-Adams attacks. It is known that bilateral bundle-branch block may present as complex combinations of first-, second-, or third-degree conduction abnormal-

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ties involving the bundle branches intermittently, permanently, synchronously, or asynchronously.\(^5\)\(^,\)\(^6\) When the two divisions of the left bundle are considered separately, as in the trifascicular concept popularized recently by Rosenbaum and co-workers,\(^8\)\(^,\)\(^9\) the resulting combinations of conduction disturbances become even greater. Since any given electrocardiographic pattern may be the result of several different types of block, it is difficult to be certain from the surface electrocardiogram (ECG) alone about the character or degree of block in the respective bundle branches or the individual fascicles. The recently introduced catheter technic of recording the electrical activity of the bundle of His in man\(^10\)\(^,\)\(^11\) provides a new approach to the analysis of conduction disturbances affecting one or both bundle branches and allows for considerably more detail in analysis.

In the His bundle electrogram the A-H interval from the onset of atrial activation (A) to the onset of bundle of His potential (H) represents the conduction time through the atrioventricular (A-V) node, whereas the H-V interval, between H and the onset of ventricular activation (V), primarily represents conduction distal to the His bundle. Conduction defects in the His bundle itself are considered to be rare. Thus, during unilateral bundle-branch block (BBB) the H-V interval reflects conduction through the contralateral bundle branch. It was considered worthwhile, therefore, to study patients with BBB utilizing the His bundle electrogram in an attempt to document any defective conduction through the contralateral bundle branch as well as to correlate the A-V and intraventricular (I-V) conduction time as obtained by His recordings with those of the conventional ECG.

Methods

His bundle electrograms were obtained by using a bipolar-electrode catheter in 20 patients with BBB. There were 13 male and seven female patients; their ages ranged from 43 to 93 years. In three patients the BBB developed in association with acute myocardial infarction. In the remaining 17 it was chronic. The symptoms related to the conduction disturbance in 19 patients are indicated in tables 1 and 2, along with the electrophysiologic data. None of the three patients with acute BBB had any symptom related to the conduction disturbance, but in all three temporary venous pacemakers were inserted prophylactically. Of the 17 patients with chronic BBB, 12 had syncope episodes, two had light-headed episodes, two had congestive heart failure, and one developed complete heart block during cardiac catheterization. Seven of these patients had intermittent complete heart block demonstrated in the ECG taken prior to the time of the His bundle recording. At the time of the His recording, 14 patients had 1:1 conduction (seven with long P-R, i.e., \(>\) 200 msec) and six had second-degree block. Of the latter, four had Mobitz type-II block and two had Wenckebach or Mobitz type-I block. Mobitz type-II block was considered to be present when second-degree block occurred with stable P-R interval (sudden A-V transmission failure).

The technic used for recording His bundle electrograms was essentially the same as described by Damato and co-workers.\(^11\) All recordings were made at a frequency setting between 40 and 500 Hz at a paper speed of 100 mm/sec. The following intervals were then measured in the His bundle electrogram in milliseconds (msec): 1) A-H from the onset of atrial activation A to the first rapid deflection of the His

<table>
<thead>
<tr>
<th>Patient</th>
<th>Symptoms*</th>
<th>P-R</th>
<th>A-H</th>
<th>H-V</th>
<th>25-R</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A.A.</td>
<td>None</td>
<td>150</td>
<td>85</td>
<td>60</td>
<td>690</td>
</tr>
<tr>
<td>R.M.</td>
<td>None</td>
<td>150</td>
<td>85</td>
<td>60</td>
<td>800</td>
</tr>
<tr>
<td>Chronic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F.L.†</td>
<td>Syncope</td>
<td>160</td>
<td>68</td>
<td>85</td>
<td>750</td>
</tr>
<tr>
<td>N.F.†</td>
<td>Syncope</td>
<td>170</td>
<td>95</td>
<td>65</td>
<td>1000</td>
</tr>
<tr>
<td>C.W.</td>
<td>Light-headed episodes</td>
<td>180</td>
<td>130</td>
<td>44</td>
<td>780</td>
</tr>
<tr>
<td>W.H.</td>
<td>Light-headed episodes</td>
<td>180</td>
<td>115</td>
<td>45</td>
<td>1180</td>
</tr>
<tr>
<td>S.G.</td>
<td>Syncope</td>
<td>210</td>
<td>150</td>
<td>50</td>
<td>820</td>
</tr>
<tr>
<td>H.N.</td>
<td>Syncope</td>
<td>240</td>
<td>182</td>
<td>47</td>
<td>1125</td>
</tr>
<tr>
<td>A.T.</td>
<td>Syncope</td>
<td>250</td>
<td>185</td>
<td>45</td>
<td>1000</td>
</tr>
<tr>
<td>Mobitz II block</td>
<td>S.R.†</td>
<td>CHF</td>
<td>260</td>
<td>170</td>
<td>70</td>
</tr>
</tbody>
</table>

*Symptoms related to the conduction disorder.
†Transient complete heart block demonstrated in the ECG at other times.

Abbreviations: CHF = congestive heart failure; RBBB with LAD = right bundle-branch block with left-axis deviation.

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bundle potential H; (2) H-V from the onset of H to the earliest inscription of ventricular activation whether this was the V in the His bundle electrogram or the onset of QRS in the simultaneously recorded surface ECC lead I. The interval A-H, rather than the interval P-H (from the onset of the P wave in standard ECC), was considered to represent A-V-nodal conduction. The interval P-H reflects intraatrial conduction as well as A-V-nodal conduction and is longer than the A-H interval.

Although multiple surface leads were not recorded, the A-H and H-V intervals in normal patients studied in our laboratories were essentially similar to those reported by others. In normal patients A-H varied from 65 to 150 msec at heart rates of 70 to 90/min, and H-V varied from 38 to 50 msec (average, 45 msec). Patients were divided into three groups:

Group I consisted of 10 patients with complete right bundle-branch block and left-axis deviation of the mean QRS axis in the frontal plane of −30° or more (RBBB with LAD). LAD is considered to represent block in the anterior division of the left bundle branch since several clinical and pathologic studies support this concept.

Group II consisted of nine patients with complete left bundle-branch block (LBBB).

Group III consisted of one patient with alternating bundle-branch block.

For comparison 13 additional patients with long P-R intervals in the absence of BBB were studied (group IV).

In an attempt to assess the risk of development of complete heart block in patients with bundle-branch disease, observations made by two of us (N.R. and J.H.P.) are included on 36 of 38 patients who developed chronic complete heart block following documentation of bundle-branch conduction defect some years previously (group V).

**Results**

**Group I**

The A-V and I-V conduction time in patients with RBBB and LAD are presented in table 1. Because RBBB and LAD is believed to indicate block in the right bundle and the anterior division of the left bundle branch, the H-V interval in group I patients was considered to represent conduction time primarily through the posterior division of the left bundle branch. Of the nine patients with 1:1 conduction (table 1) H-V was normal (less than 50 msec) in five patients, slightly prolonged (60 to 65 msec) in three patients (A.A., R.M., and N.F.), and definitely prolonged (85 msec) in one (F.L., fig. 1), indicating delayed conduction through the posterior division of the left bundle branch. Of importance is the fact that all four patients with prolongation of H-V interval had a normal P-R interval (fig. 1). In two patients (H.N. and A.T.) distinct prolongation of the

**Table 2**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Symptoms*</th>
<th>P-R</th>
<th>A-H</th>
<th>H-V</th>
<th>R-R</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.Y.</td>
<td>None</td>
<td>230</td>
<td>170</td>
<td>60</td>
<td>710</td>
</tr>
<tr>
<td>A.J.†</td>
<td>Syncope</td>
<td>175</td>
<td>110</td>
<td>65</td>
<td>700</td>
</tr>
<tr>
<td>W.M.†</td>
<td>Asystole</td>
<td>260</td>
<td>170</td>
<td>85</td>
<td>700</td>
</tr>
<tr>
<td>A.D.</td>
<td>Syncope</td>
<td>280</td>
<td>186</td>
<td>53</td>
<td>692</td>
</tr>
<tr>
<td>G.Y.</td>
<td>Syncope</td>
<td>420</td>
<td>360</td>
<td>60</td>
<td>750</td>
</tr>
<tr>
<td>Mobitz II block</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M.S.</td>
<td>Syncope</td>
<td>190</td>
<td>100</td>
<td>80</td>
<td>720</td>
</tr>
<tr>
<td>R.M.†</td>
<td>Complete heart block during heart catheterization</td>
<td>360</td>
<td>138</td>
<td>220</td>
<td>1000</td>
</tr>
<tr>
<td>Wenckebach block</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A.F.</td>
<td>CHF</td>
<td>240–380</td>
<td>150–285</td>
<td>60</td>
<td>800–1180</td>
</tr>
<tr>
<td>L.D.</td>
<td>Syncope</td>
<td>230–430</td>
<td>110</td>
<td>90–260</td>
<td>1100–1650</td>
</tr>
</tbody>
</table>

*Symptoms related to the conduction disorder.
†Transient complete heart block demonstrated in the ECG at other times.
Abbreviations: CHF = congestive heart failure; LBBB = left bundle-branch block.
Figure 1

Patient with RBBB and LAD with normal PR. (Top panel) Surface ECG. (Middle and bottom panels) His bundle recording (BE) with simultaneous surface lead I at paper speeds of 50 mm/sec and 200 mm/sec, respectively. Note HV interval (85 msec) is significantly prolonged (normal <50 msec), in spite of a normal PR, indicating delayed conduction through the posterior division of the left bundle branch. In this and all subsequent figures A = atrial electrogram; H = His bundle electrogram; V = ventricular electrogram; MSC = milliseconds.
C.W. RBBB WITH LAD NORMAL AH, HV AND PR
ATRIAL PREMATURE BEAT BLOCKED DISTAL TO H

Figure 2

Patient with RBBB and LAD and normal PR. (Top panel) Surface ECG. Nonconducted atrial premature beats are noted in lead V1. (Middle and bottom panels) His bundle recordings (BE) obtained at paper speeds of 50 mm/sec and 100 mm/sec, respectively, with simultaneous surface lead I are shown. Spontaneous atrial premature beat (aP) is blocked distal to His bundle potential H, in spite of normal AH and HV intervals in conducted beats. The aP seems to have unmasked defective conduction through the posterior division of the left bundle branch.
His bundle recording and ECG from a patient with RBBB and LAD with second-degree block of Mobitz type II. (Lower panel) His recording (BE; continuous strip) demonstrates a block distal to H in the dropped beats. The long PR in the conducted beats is seen to be related to lengthening of both AH (170 msec) and HV (70 msec) indicating combined first-degree delay in the A-V node and alternating or variable first- and second-degree block in the posterior division of the left bundle branch.

Figure 3

P-R interval was related to lengthening of A-H, indicating that the delay in conduction occurred primarily in the A-V node.

Three patients within this group (C.W., W.H., and H.N.) had spontaneous atrial premature beats. In two (W.H. and H.N.), these were conducted with a long P-R and a long A-H, indicating delayed A-V-nodal conduction. In one (C.W.), the atrial premature beats were blocked distal to the His bundle potential, presumably in the posterior division of the left bundle branch (fig. 2). In these three patients the atrial premature beats appeared to reveal latent conduction disturbances, presumably related to relative refractoriness and in one (C.W.), to have indicated the presence of trifascicular rather than bifascicular disease. Wit and associates, however, have suggested that conduction disturbances revealed by atrial premature
beats may be a normal phenomenon based on studies in atrially paced patients in whom atrial premature beats were electrically stimulated.

In the patient (S.R.) with second-degree block of Mobitz type II, the His recording demonstrated a block distal to H in the dropped beats (fig. 3). In addition, the long P-R in the conducted beats was seen to be related to lengthening of both A-H and H-V intervals. Thus there was a first-degree block in A-V node (mild) and alternating or variable first- and second-degree block in the posterior division of LBB (table 1; fig. 3).

Group II

The A-V and I-V conduction time in patients with LBBB is presented in table 2. Five patients had 1:1 conduction and four had second-degree block. The H-V interval during LBBB is considered primarily to reflect conduction through the right bundle branch and the Purkinje system of the right side. Of the five patients with LBBB and 1:1 conduction one (W.M.) had a markedly prolonged H-V interval (85 msec) indicating delayed conduction through the right bundle branch. The rest had slightly prolonged H-V intervals (greater than 50 msec) varying from 53 to 65 msec (table 2). Such slight prolongation of H-V in patients with LBBB has been described by others as well.\textsuperscript{14, 21}

Of the four patients with long P-R and 1:1 conduction (table 2), two (A.D. and G.Y.) had definite lengthening and one (R.Y.) had slight lengthening in A-H indicating A-V nodal delay. In the fourth patient (W.M.), however, the long P-R was related mainly to prolonged H-V, indicating delayed conduction through the right bundle branch.

Two patients (M.S. and R.M.) had Mobitz type-II form of second-degree block (table 2). In both, block occurred distal to the H spike in the dropped beats as is usually the case in Mobitz type-II block.\textsuperscript{12, 13, 22–24} Both of these patients had normal A-H and long H-V intervals in the conducted beats, indicating delayed conduction through the RBB (table 2). In one (M.S.) the P-R interval in the conducted beats was normal, and in the other (R.M.) it was long.

Two patients had a Mobitz type-I or Wenckebach type of second-degree block. In one (A.F.) the progressive lengthening in P-R was related to a progressive lengthening in A-H followed by dropped H and V, the H-V

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure4.png}
\caption{His recording from patient L.D. with LBBB and Wenckebach phenomenon with simultaneous lead II. Paper speed at 100 mm/sec. PR increases from 230 to 430 msec followed by the dropped beat. The dropped beat is due to block distal to H. AH remains constant and normal (110 msec). HV interval is prolonged and it increases from 90 to 260 msec in the two conducted beats, causing the progressive lengthening in PR. Thus the Wenckebach type conduction is occurring in the right bundle branch.}
\end{figure}
interval remaining stable (table 2). In the other patient (L.D.), the progressive lengthening in P-R was related not to any change in A-H, which remained stable, but rather to a progressive lengthening in H-V, indicating that the Wenckebach type block was occurring distal to the His bundle, presumably in the right bundle branch or the Purkinje system of the right side (fig. 4).

**Group III**

This group consisted of one patient who had an alternating bundle-branch block with

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**Figure 5**

*Patient with alternating BBB and Mobitz type-II form of second-degree block. (Top panel) Surface ECG demonstrates the changing PR interval (normal during LBBB and prolonged during RBBB). (Lower panel) His recording (BE) with simultaneous surface lead I at 100 mm/sec paper speed demonstrates a block distal to H in the dropped beats. AH is constant (stable A-V-nodal conduction time) whereas HV is unequal and prolonged (70 msec during LBBB with normal PR and 120 msec during RBBB with a long PR). The changing PR is thus seen to be related entirely to changing HV intervals (varying conduction time through the bundle branches).*

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Mobitz type-II block (fig. 5). The P-R during LBBB was normal and was long during RBBB. The His bundle recording in this patient (fig. 5) demonstrated a block distal to H in the dropped beats, indicating a second-degree block in both bundle branches. In all beats (conducted and nonconducted) the A-H interval remained normal (115 to 120 msec). The changing P-R interval when the BBB alternated was primarily related to the changing H-V. During LBBB the P-R was normal, although the H-V was prolonged (70 msec). During RBBB the prolonged P-R was related to marked prolongation of H-V (120 msec). Thus these recordings reveal first-degree block in both bundle branches (more marked on left) as well as an asynchronous second-degree block in both bundle branches.

**Group IV**

In contrast to the 20 patients with BBB, all 13 patients who had long P-R without BBB invariably had lengthening in AH with normal H-V.

**Group V**

The duration in years that elapsed between the first observed conduction defect and the development of complete heart block in 38 of 38 patients with chronic complete heart block is shown in figure 6. It gives a rough estimate of the risk of development of complete heart block in patients with bundle-branch block. Left anterior hemiblock preceded complete heart block from 4 to 14 years (average, 9 years). LBBB preceded complete heart block from 1 to 6½ years (average, 2 2/3 years), while RBBB and LAD preceded complete heart block anywhere from 1 month to 23 years (average, 7 years). When BBB was complicated by P-R prolongation or second-degree A-V block, or both, the onset of complete heart block was much sooner (usually less than 2 years). It is apparent that the functional status of the contralateral bundle branch as well as the presence or absence of conduction defects elsewhere in the conduction system are foremost among the factors that determine the risk of complete heart block in any given instance of bundle-branch block.

**Discussion**

In the His bundle electrogram the interval A-H represents A-V-nodal conduction whereas the interval H-V reflects conduction distal to the His bundle. During unilateral bundle-branch block the H-V interval becomes a measure of conduction time through the contralateral bundle branch and in cases of bifascicular

![Diagram](http://circ.ahajournals.org/)

**Figure 6**

ECG findings observed before the development of complete heart block in 38 patients with chronic complete heart block. Isolated left anterior hemiblock preceded onset of complete heart block from 4 to 14 years; isolated LBBB preceded complete heart block from 1 to 6½ years; and isolated RBBB and LAD preceded it from 1 month to 23 years. However, when BBB was complicated by additional delay such as a long P-R interval or second-degree A-V block, complete heart block developed sooner (usually in less than 2 years).
block the H-V interval reflects conduction time through the remaining fascicle. Using these principles, we studied 20 patients with BBB by recording His bundle electrograms. In contrast to previously reported studies, all 17 patients with chronic BBB (unassociated with acute myocardial infarction) in our study were symptomatic. There was a high incidence of bilateral bundle-branch or trifascicular disease among these patients as shown by definite prolongation of H-V time (eight of 17 patients) (tables 1 and 2; fig. 5). The present study as well as those of others clearly demonstrates that a His bundle electrogram in BBB can detect conduction defects in the contralateral bundle branch and elsewhere in the conduction system when these are not evident or defined in the surface ECG. Thus, when the P-R interval was prolonged in the presence of BBB, the conduction delay occurred either in the contralateral bundle branch or the A-V node, or both. Of 12 patients with long P-R and BBB in our study (seven with 1:1 conduction and five with second-degree block with a long P-R in the conducted beats) (tables 1 and 2; fig. 5), seven had conduction delay at the A-V node, three had it distal to the His bundle, and two at both levels (fig. 3). This is in contrast to patients with a long P-R in the absence of BBB who invariably had prolonged A-H, indicating A-V-nodal delay (13 of 13 patients).

Similarly, first-degree conduction delay in the functioning bundle branch as shown by prolonged H-V time occurred in a number of instances (four patients with RBBB and LAD and patient M.S. with LBBB and Mobitz type-II block) in spite of a normal P-R in the ECG. In addition our study has also demonstrated normal conduction time (H-V less than 50 msec) through the only functioning fascicle in five patients with chronic RBBB and LAD who were symptomatic (table 1). These five patients, although manifesting no block at the time of study, must, when symptomatic, develop second-degree block at the A-V node, His bundle, or in the remaining fascicle. This is partly supported by the fact that two of the patients (H.N. and A.T.) had prolonged A-H, indicating A-V-nodal delay, and one (C.W.) had block of spontaneous atrial premature beats distal to the His bundle (fig. 2). Two of these five patients (W.H. and H.N.), however, did have sinus bradycardia (R-R > 1,000 msec) and, in them, sinus-node disease cannot be entirely excluded.

Of particular interest in our study, however, were the recordings in the two patients with LBBB and Wenckebach phenomenon (table 2; fig. 4), and the patient who had alternating BBB with Mobitz type-II block (fig. 5). Of the two patients with the Wenckebach type (Mobitz type-I) second-degree block with LBBB, one had A-V-nodal delay as shown by progressive lengthening in A-H (table 2) whereas the other had delayed conduction through the right bundle branch as shown by progressive lengthening in H-V (fig. 4). This demonstrates that the Wenckebach type block can occur in the bundle branches as well as in the common site, the A-V node. In addition to our findings there is considerable experimental and clinical evidence that Wenckebach type of decremental conduction can occur in the bundle branches. Thus, the level of block, whether proximal or distal to the His bundle, cannot be used as a distinguishing point between the two types of second-degree block as has previously been suggested.

The recording from the patient with alternating BBB is of unusual interest with respect to ECG interpretation of bilateral BBB. Applying the traditional nomenclature of first-, second-, and third-degree blocks to each of the bundle branches, Lepeschkin has pointed out the following: (1) The side that has the greater degree of block determines the QRS morphology whereas the side that has the lesser degree of block determines the P-R interval. (2) With second-degree block in the bundle branches, alternation and type of QRS morphology would depend on whether the block is synchronous or not. (3) Any change in the P-R interval when one BBB pattern changes to another is much better explained by bilateral disturbance of bundle-branch...
conduction than by changing A-V-nodal conduction time. The recording from the patient with alternating BBB (fig. 5) confirms that these theoretical principles are in fact true to a large extent. In this patient unequal amounts of first-degree delay, as shown by varying H-V time in the bundle branches, occurred. The coexisting asynchronous second-degree block in the bundle branches allowed the alternation of BBB to be manifested in the ECG. The His recording clearly demonstrated that the changing P-R, when BBB alternated, was in fact related to the changing H-V (varying conduction time through the bundle branches) and not to any change in A-H (stable A-V-nodal conduction).

Two important modifications of these theoretical principles, however, need to be made in view of the findings in the present study as well as those of others. 12, 14, 15, 21, 25 (1) A long P-R interval in the presence of BBB does not invariably mean bilateral bundle-branch disease and A-V-nodal delay may be present coexisting or isolated (fig. 3; tables 1 and 2). (2) The side that has the lesser degree of block determines only the H-V time, and this may or may not be manifested in the P-R interval (fig. 1). Thus, the earlier degrees of bilateral BBB can only be recognized by the use of His bundle electrograms.

These findings are of importance with respect to the assessment of risk of development of complete heart block in any given instance of BBB. The exact magnitude of risk of development of complete heart block in patients with BBB is not clearly known. The risk as quoted by previous workers for patients with RBBB and LAD varies from 10%7 to 13.6%.52 However, the risk when plotted with increasing time may in fact be higher. Isolated BBB in our study preceded the onset of complete heart block by a number of years, as long as 23 years in some patients with RBBB and LAD, whereas when BBB was complicated either by long P-R or second-degree A-V block, or both, the onset of complete heart block was much sooner, usually less than 2 years (fig. 6).

The present study has demonstrated the value of His bundle electrograms in defining complicated conduction disturbances as well as in recognizing earlier degrees of bilateral BBB. Thus, only prospective studies of patients with BBB by means of His bundle electrography would help in establishing the actual risk of complete heart block in patients with BBB.

Acknowledgment

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Appendix

Applying the traditional nomenclature of first-, second-, and third-degree block, a system for the interpretation of bilateral bundle-branch block can be developed. The various possible combinations are presented in table A. The principles can be equally applied to the two divisions of the left bundle branch and the resulting combinations would become even greater. It is evident that any given electrocardiographic pattern may be the result of several different types of block, and that from surface ECG alone it is difficult to be certain about the degree of block in the respective bundle-branch system.

References

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## Appendix Table A

Various Possible Combinations of Bilateral Bundle-Branch Block and the Resultant Electrocardiographic Patterns

<table>
<thead>
<tr>
<th>Serial no.</th>
<th>Degree of block</th>
<th>In rt. bundle</th>
<th>In lt. bundle</th>
<th>Resultant ECG pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3°</td>
<td>3°</td>
<td>Complete heart block with a ventricular focus</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>3°</td>
<td>2°</td>
<td>RBBB in conducted beats with normal P-R, 2:1 A-V block</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>3°</td>
<td>1°</td>
<td>RBBB with P-R prolongation</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>3°</td>
<td>1° + 2°</td>
<td>Like 2 except P-R prolonged in conducted beats</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>2°</td>
<td>3°</td>
<td>LBBB in conducted beats with normal P-R, 2:1 A-V block</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>1°</td>
<td>3°</td>
<td>LBBB with P-R prolongation</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>1° + 2°</td>
<td>3°</td>
<td>Like 5 except P-R prolonged</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>2°</td>
<td>2°</td>
<td>2:1 A-V block (Mobitz II), P-R normal</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>2°</td>
<td>2° + 1°</td>
<td>Like 8 except with LBBB in conducted beats</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>2° + 1°</td>
<td>2°</td>
<td>Like 8 except with RBBB in conducted beats</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>2°</td>
<td>Asynchronous</td>
<td>2°</td>
<td>Alternating BBB</td>
</tr>
<tr>
<td>12</td>
<td>2°</td>
<td>Asynchronous</td>
<td>2° + 1°</td>
<td>Alternating BBB with P-R prolongation when RBBB is present</td>
</tr>
<tr>
<td>13</td>
<td>2° + 1°</td>
<td>Asynchronous</td>
<td>2°</td>
<td>Alternating BBB with P-R prolongation when LBBB is present</td>
</tr>
<tr>
<td>14</td>
<td>2° + 1°</td>
<td>Asynchronous</td>
<td>2° + 1°</td>
<td>Alternating BBB with P-R prolongation but P-R may vary</td>
</tr>
<tr>
<td>15</td>
<td>2°</td>
<td>Asynchronous</td>
<td>1°</td>
<td>LBBB with normal P-R alternating with RBBB with long P-R</td>
</tr>
<tr>
<td>16</td>
<td>2° + 1°</td>
<td>1°</td>
<td>LBBB with long P-R alternating with RBBB with long P-R</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>1°</td>
<td>2°</td>
<td>RBBB with normal P-R alternating with LBBB with long P-R</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>1°</td>
<td>2° + 1°</td>
<td>Like 16. Alternating BBB with long P-R</td>
<td></td>
</tr>
<tr>
<td>19*</td>
<td>1° (minimum)</td>
<td>1° (maximum)</td>
<td>Like 6</td>
<td></td>
</tr>
<tr>
<td>20*</td>
<td>1° (maximum)</td>
<td>1° (minimum)</td>
<td>Like 3</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>3°</td>
<td>0°</td>
<td>RBBB</td>
<td></td>
</tr>
<tr>
<td>22</td>
<td>2°</td>
<td>0°</td>
<td>RBBB alternating with normal beats</td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>1°</td>
<td>0°</td>
<td>RBBB</td>
<td></td>
</tr>
<tr>
<td>24</td>
<td>0°</td>
<td>3°</td>
<td>LBBB</td>
<td></td>
</tr>
<tr>
<td>25</td>
<td>0°</td>
<td>2°</td>
<td>LBBB alternating with normal beats</td>
<td></td>
</tr>
<tr>
<td>26</td>
<td>0°</td>
<td>1°</td>
<td>LBBB</td>
<td></td>
</tr>
</tbody>
</table>

*Nos. 19 and 20. These may vary from one to the other, and then P-R interval will vary depending on conduction time in respective bundles.

Abbreviations: RBBB = right bundle-branch block; LBBB = left bundle-branch block; P-R = P-R interval; BBB = bundle-branch block.

2° = Under this, only 2:1 type conduction is considered here. However, it must be realized that 3:1, 4:1, etc. may occur, but the analysis would be similar. However, in case of 2° plus 1° blocks, from the surface ECG, one cannot be sure whether one is dealing with a Mobitz I or Mobitz II block when the ratio is 2:1.

*Circulation, Volume XLV, February 1972*
7. Lasser RP, Haft JI, Friedberg CK: Relationship of right bundle-branch block and marked left axis deviation (with left perietal or peri-infarction block) to complete heart block and syncope. Circulation 37: 429, 1968

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