Bundle Branch and Ventricular Activation in Man
A Study Using Catheter Recordings of Left and Right Bundle Branch Potentials

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SUMMARY
His (H), right bundle branch (RB), and left bundle branch (LB) potentials were recorded by electrode catheters passed into the left and right ventricles in seven patients without conduction defects. In another three patients, H and LB were recorded. Initial ventricular activation was considered to coincide with the onset of the QRS (Q). The following intervals were measured in milliseconds: H-Q, LB-Q, RB-Q, H-LB, H-RB, and LB-RB. The average and range of these was as follows: H-Q 46 (39-54), LB-Q 27 (24-30), RB-Q 25 (20-27), H-LB 20 (14-25), H-RB 21 (15-29), and LB-RB 2 (1-10). LB-RB was 3 msec or less in six of seven patients. Coupled atrial pacing produced delays in H-RB without effect on H-LB and LB-Q. The production of incomplete and complete right bundle branch block was related to increase in the LB-RB interval. One patient developed left bundle branch block (LBBB) during coupled pacing with delay of LB while RB-Q remained unchanged. H-Q intervals were measured in three additional patients with rate related LBBB during both control and block. Prolongation of H-Q was less than 3 msec during LBBB. It is concluded that the proximal left and right bundle branches are usually activated simultaneously in man, and that block of either bundle without contralateral bundle delay does not significantly delay the onset of ventricular activation.

Additional Indexing Words:
Ventricular septum QRS PR interval Bundle branch block
Electrophysiology of the heart

RIGHT HEART catheterization with electrode catheter for recording of His bundle electrograms is a useful technic for the study of human cardiac conduction in health and disease.1-6 With slight modification, the technic has also allowed the recording of right bundle branch potentials.7 At present, catheter recordings of left bundle branch activity have been reported only in dogs.8, 9

In this study, we are reporting the simultaneous recording of both right and left bundle branch potentials in patients during cardiac catheterization. This has allowed us to study normal intraventricular conduction, as well as the functional conduction defects produced by coupled atrial pacing. Conclusions have been reached regarding the normal sequences of bundle branch and right and left ventricular activation.

Methods
Patients Without Conduction Defects
The study group consisted of ten patients with suspected organic heart disease without electro-
Clinical and Electrocardiographic Data

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>PR (sec)</th>
<th>QRS (sec)</th>
<th>Electrocardiogram</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 W.R.</td>
<td>30</td>
<td>M</td>
<td>PMD</td>
<td>0.18</td>
<td>0.08</td>
<td>Nonspecific ST and T-wave changes, clockwise rotation</td>
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</tr>
<tr>
<td>2 J.B.</td>
<td>59</td>
<td>M</td>
<td>Pericarditis</td>
<td>0.16</td>
<td>0.08</td>
<td>ST elevations in I, II, III, aV_{L}, aV_{F}, V_{2}-V_{4}</td>
<td></td>
</tr>
<tr>
<td>3 A.M.</td>
<td>56</td>
<td>F</td>
<td>RHD, mitral stenosis and insufficiency</td>
<td>-</td>
<td>0.06</td>
<td>Atrial fibrillation, nonspecific S-T and T-wave changes</td>
<td></td>
</tr>
<tr>
<td>4 J.C.</td>
<td>41</td>
<td>F</td>
<td>No organic heart disease found</td>
<td>0.14</td>
<td>0.08</td>
<td>Nonspecific S-T and T-wave changes</td>
<td></td>
</tr>
<tr>
<td>5 K.R.</td>
<td>20</td>
<td>M</td>
<td>Functional murmur</td>
<td>0.12</td>
<td>0.08</td>
<td>Normal ECG</td>
<td></td>
</tr>
<tr>
<td>6 L.T.</td>
<td>16</td>
<td>M</td>
<td>Functional murmur</td>
<td>0.20</td>
<td>0.08</td>
<td>ST elevations due to early repolarization</td>
<td></td>
</tr>
<tr>
<td>7 O.B.</td>
<td>42</td>
<td>F</td>
<td>RHD, mitral insufficiency</td>
<td>0.12</td>
<td>0.08</td>
<td>Clockwise rotation</td>
<td></td>
</tr>
<tr>
<td>8 E.H.</td>
<td>47</td>
<td>F</td>
<td>Aortic insufficiency</td>
<td>0.16</td>
<td>0.06</td>
<td>Left ventricular hypertrophy</td>
<td></td>
</tr>
<tr>
<td>9 L.S.</td>
<td>73</td>
<td>M</td>
<td>Ejection systolic murmur, HCVD</td>
<td>0.18</td>
<td>0.08</td>
<td>Left ventricular hypertrophy</td>
<td></td>
</tr>
<tr>
<td>10 G.R.</td>
<td>47</td>
<td>M</td>
<td>RHD, post-commissurotomy, mitral restenosis</td>
<td>-</td>
<td>0.08</td>
<td>Atrial fibrillation, right ventricular hypertrophy</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: PMD = Primary myocardial disease; RHD = Rheumatic heart disease; HCVD = Hypertensive cardiovascular disease; M = Male; F = Female.

Figure 1

A P radiograph of chest showing position of catheters for recording of H, LB, and RB. A steptech electrode catheter is positioned along the lateral wall of the right atrium for pacing.

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attempt to record His bundle (H) and right bundle branch (RB) potentials simultaneously (fig. 1). However, this was frequently impossible, necessitating separate recordings of these potentials.

Left bundle branch (LB) potentials were recorded by a technic similar to that reported in canines.\(^8\)\(^,\)\(^9\) A tripolar catheter was passed through the right brachial artery retrograde across the aortic valve. The electrodes were positioned 1–2 cm below the valve along the ventricular septum (fig. 1), and the proximal terminals were plugged into the switch box. Slight adjustments of catheter positions were made until a high frequency bi- or triphasic spike was noted, discrete from and occurring later than the His bundle spike. Recordings were obtained of simultaneous H and LB potentials, and if RB was recordable, of simultaneous LB and RB potentials.

An additional bipolar pacing catheter was positioned against the lateral wall of the right atrium (fig. 1) and pacing was accomplished with an R-wave coupled pulse generator powered by battery (Medtronic 5837, Minneapolis, Minn.). Single and coupled atrial pacing were utilized for further study of conduction.

The following intervals were measured in milliseconds at a paper speed of 200 mm/sec:

1. P-H = The interval from the P wave to the first high frequency component of H.

2. H-Q = The interval from the first high frequency component of H to the onset of the ECG QRS complex.

(3) LB-Q = The interval from the first high frequency component of the LB potential to the onset of the QRS complex. If the QRS preceded the LB potential, LB-Q was recorded as a negative value.

(4) RB-Q = The interval from the first high frequency component of the RB potential to the onset of the QRS complex. If the QRS preceded the RB potential, RB-Q was recorded as a negative value.

(5) H-LB = The interval from the first high frequency component of H to the first high frequency component of LB.

(6) H-RB = The interval from the first high frequency component of H to the first high frequency component of RB.

(7) LR-RB = The interval from the first high frequency component of LB to that of RB. If RB preceded LB, the interval was recorded as a negative value.

**Patients with Rate Related Left Bundle Branch Block**

To clarify the effect of left bundle branch block (LBBB) on ventricular activation, we obtained His bundle recordings in three patients with rate related LBBB, during control state and during LBBB. Two of the patients were conducting normally in the control state, and we used atrial pacing at a rate above critical rate\(^10\) to produce block. One patient was in LBBB at the time of study, with normal conduction being restored upon slowing of the heart rate with coupled atrial pacing. All patients had typical patterns of

![Figure 2](image_url)

*Figure 2*

Record from patient 2 showing simultaneous ECG lead V1, His bundle electrogram (HBE), left bundle branch electrogram (LBE), and right bundle branch electrogram (RBE). P wave is labeled P and QRS is labeled R. His bundle spike (H), left bundle potential (LB), and right bundle potential (RB) are shown on the second, third, and fourth lines of the recording. Intervals are listed at the bottom. Note that LB and RB are almost simultaneous. Paper speed is 200 mm/sec and time lines are at 1 sec on this and all subsequent figures.
complete LBBB, with QRS widening greater than 0.12 sec, notching of the QRS in V6, ST depression and T-wave inversion in the lateral precordium, and absence of both S and Q waves in V6.

**Results**

**Conduction Tissue Potentials and Intervals**

H, LB, and RB were recorded as rapid bi- or triphasic spikes similar in morphology and timing to potentials previously recorded by direct technics. Typical patient recordings are shown in figures 2–5. With the catheter positioned at the tricuspid valve, the H spike was noted between the atrial and ventricular electrograms. When the more distal electrodes were used or when the catheter was moved 1–2 cm distally into the right ventricle, the RB potential was recordable as a sharp discrete spike occurring later than H but earlier than the ventricular electrogram. With the catheter in the left ventricle just below the aortic valve, the LB potential was recordable, also as a sharp discrete potential occurring between H and the ventricular electrogram. With catheters positioned for recording of bundle branch potentials, the intracardiac electrograms showed predominantly specialized tissue spikes followed by ventricular electrogram, with minimal or absent atrial electrogram. H and LB potentials were recorded in all ten patients, while RB was recordable in only seven of these.

Intervals are listed in table 2, and examples are shown in figures 2–5. H-Q interval, a measure of conduction time from the His bundle to the onset of ventricular activation, ranged from 39–52 msec (mean 46 msec). LB-Q, a measurement of conduction time from the left bundle to the onset of ventricular activation, ranged from 24–30 msec (mean 27 msec), while RB-Q ranged from 20–27 msec (mean 25 msec). H-LB and H-RB, measures of conduction time from the His bundle to the respective proximal bundle branches, ranged from 15–28 msec (mean 20 msec) and 15–29 msec (mean 21 msec). The ranges of H-LB and H-RB were greater than those of LB-Q and RB-Q and accounted for most of the variation in H-Q intervals.

The LB and RB potentials occurred almost simultaneously in six out of seven patients.

![Figure 3](circulation.org/doi/10.1161/01.CIR.43.2.196)
with LB-RB varying from -1 to 3 msec (figs. 2-4). In one patient, LB preceded RB by 10 msec (fig. 5).

Effects of Single and Coupled Atrial Pacing on Intervals and QRS Morphology

Single atrial pacing at rates of 80-160 beats/min was utilized in patients 2, 4, and 7 for further study of conduction. In all patients, P-H intervals were prolonged as expected with increasing heart rates, while H-Q and its subintervals (H-LB, H-RB, LB-Q, and RB-Q) remained unchanged. QRS morphology remained constant with increasing rate.
Table 2
Conduction Intervals (msec) in Ten Patients with Normal Intraventricular Conduction

<table>
<thead>
<tr>
<th>Patient</th>
<th>H-Q</th>
<th>LB-Q</th>
<th>RB-Q</th>
<th>H-LB</th>
<th>H-RB</th>
<th>LB-RB</th>
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<td>54</td>
<td>26</td>
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<tr>
<td>2</td>
<td>46</td>
<td>28</td>
<td>27</td>
<td>18</td>
<td>19</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>44</td>
<td>28</td>
<td>25</td>
<td>19</td>
<td>19</td>
<td>3</td>
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<td>4</td>
<td>45</td>
<td>30</td>
<td>20</td>
<td>15</td>
<td>25</td>
<td>10</td>
</tr>
<tr>
<td>5</td>
<td>40</td>
<td>24</td>
<td>25</td>
<td>16</td>
<td>13</td>
<td>(−1)</td>
</tr>
<tr>
<td>6</td>
<td>45</td>
<td>28</td>
<td>25</td>
<td>17</td>
<td>20</td>
<td>3</td>
</tr>
<tr>
<td>7</td>
<td>48</td>
<td>26</td>
<td>27</td>
<td>22</td>
<td>21</td>
<td>(−1)</td>
</tr>
<tr>
<td>8</td>
<td>39</td>
<td>25</td>
<td>−</td>
<td>14</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>9</td>
<td>52</td>
<td>26</td>
<td>−</td>
<td>26</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>10</td>
<td>48</td>
<td>27</td>
<td>−</td>
<td>21</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Mean</td>
<td>46</td>
<td>27</td>
<td>25</td>
<td>20</td>
<td>21</td>
<td>2</td>
</tr>
</tbody>
</table>

We used coupled atrial pacing to produce various patterns of aberrant conduction. This enabled us to validate the LB and RB potentials and also to examine ventricular activation during bundle branch block. Patient 2 developed functional right bundle branch block (RBBB) with coupled atrial pacing at close coupling intervals. As the coupling intervals became closer, progressive delay in H-RB developed while H-Q, H-LB, and LB-Q remained constant (fig. 6A–D). Thus, as H-RB lengthened LB-RB also increased, indicating asynchrony of the bundle branches. Changes of QRS contour in V1 correlated with LB-RB intervals. With an LB-RB of 30 msec slight change in QRS was noted (fig. 6B), with LB-RB of 30 msec incomplete RBBB was seen (fig. 6C), and with LB-RB of 35 msec complete RBBB was present (fig. 6D).

Similar findings were noted in patient 4 who developed complete RBBB pattern with LB-RB greater than 25 msec. H-Q, H-LB, and LB-Q all remained constant during development of RBBB.

It is concluded that in RBBB, there is no delay in the onset of ventricular activation, as measured by constant H-Q interval.

In patient 7, patterns of both incomplete and complete left and right bundle branch block were noted at short coupling intervals (fig. 7A–D). Only LB and RB potentials were recorded during coupled pacing in this patient. Again, changes in LB-RB interval correlated with contour changes of QRS in lead V1. When LB-RB was 25 msec, incomplete RBBB was noted (fig. 7A). With LB-RB of 30 msec, complete RBBB pattern was seen (fig. 7B). When the LB potential was delayed, another type of conduction disturbance was seen. With LB-RB of −25 msec the pattern of incomplete LBBB was noted (fig. 7C), and with an LB-RB interval of −30 msec complete LBBB pattern was noted (fig. 7D). During RBBB, LB-Q remained unchanged (fig. 7A and B), and during LBBB, RB-Q remained unchanged (fig. 7C and D). These observations suggested that neither RBBB nor LBBB delayed the onset of ventricular activation.

Rate Related Left Bundle Branch Block

To further study the presence or absence of delay in ventricular activation during LBBB, we studied three patients with rate related LBBB. H-Q intervals, recorded during normal conduction and during LBBB, increased by less than 3 msec during LBBB (table 3 and figs. 8 and 9). Thus, the conduction time from the His bundle to the onset of ventricular activation was not prolonged during LBBB.

Discussion

Previous workers have recorded bundle branch potentials with electrodes implanted on the conduction system in dogs. Both Scherlag et al. and Ettinger et al. have recently reported the recording of LB potentials in dogs, utilizing catheter technics. Damato and coworkers have recorded RB potentials with electrode catheters in man.

In the present study, utilizing multipolar catheters placed in both ventricles contiguous to the upper ventricular septum, we have recorded potentials that we believe to represent depolarizations of the proximal bundle branches.

The reasons for accepting these as representing bundle branch activation are:

1. The electrograms were recorded from within the ventricles with catheters positioned close to the proximal bundle branches on the ventricular septum. The ventricular location was proven by...
Production of functional right bundle branch block with coupled atrial pacing in patient 2. The first QRS (R) in each box is a normally conducted beat which is followed by a coupled atrial pacing spike (Pi) and coupled QRS (R). P-Pi intervals are at the top and LB-RB intervals are listed at the bottom of each box. (A.) The coupled QRS is conducted normally with LB-RB of 3 msec. (B.) At P-Pi of 310 msec, slight aberration of the coupled QRS is noted. H-RB is prolonged and LB-RB is 22 msec. (C.) At P-Pi of 280 msec, incomplete RBBB is produced with LB-RB of 30 msec. (D.) At P-Pi of 260 msec, the pattern of complete RBBB is produced. This is due to delay of RB with LB-RB of 35 msec. Note in all examples that H-LB and LB-Q remain fixed despite delays in H-RB. Time scale is 500 msec.

pacing from the electrodes with production of ventricular pacing. (RBBB pattern was produced from the LB recording site, and LBBB pattern was produced from the RB recording site.)

(2) The RB and LB electrograms were
Production of incomplete and complete left and right bundle branch block with coupled atrial pacing in patient 7. (1) ECG lead V1. (2) RBE. (3) LBE. The first QRS is normally conducted and the second QRS in each box is coupled. (A.) The pattern of incomplete RBBB is noted with LB-RB of 25 msec. (B.) Complete RBBB is noted with LB-RB of 30 msec. Note that in A and B, LB-Q remains constant at 25 msec. (C.) The pattern of incomplete LBBB is noted with delay of LB so that LB-RB is now —25 msec. (D.) The pattern of complete LBBB is noted with LB-RB of —30 msec. Note that in C and D, RB-Q remains unchanged at 25 msec.

Table 3

<table>
<thead>
<tr>
<th>Patient</th>
<th>Heart rate (beats/min)</th>
<th>ECG</th>
<th>H-Q (msec)</th>
<th>Δ H-Q (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C.K.</td>
<td>70</td>
<td>Normal QRS</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td></td>
<td>90 (paced)</td>
<td>Normal QRS</td>
<td>42</td>
<td>+2</td>
</tr>
<tr>
<td></td>
<td>100 (paced)</td>
<td>Complete LBBB</td>
<td>42</td>
<td>+2</td>
</tr>
<tr>
<td>P.S.</td>
<td>88</td>
<td>Normal QRS</td>
<td>62</td>
<td></td>
</tr>
<tr>
<td></td>
<td>90</td>
<td>Complete LBBB</td>
<td>65</td>
<td>+3</td>
</tr>
<tr>
<td>P.O.</td>
<td>84</td>
<td>Normal QRS</td>
<td>55</td>
<td></td>
</tr>
<tr>
<td></td>
<td>90 (paced)</td>
<td>Complete LBBB</td>
<td>55</td>
<td>0</td>
</tr>
</tbody>
</table>

Δ = Change.
ventricular activation. This initial ECG deflection (Q) is generally very close to the onset of depolarization as recorded from an intraventricular electrogram. With normal conduction, Q approximates initial left midseptal depolarization, and possibly also anterior and posterior paraseptal areas of the left ventricle. In the presence of RBBB, Q reflects early depolarization of these same areas.

In LBBB, Q reflects the onset of right ventricular depolarization.

The H-Q interval approximates conduction times from the His bundle to the onset of ventricular activation. This interval may be broken down into subintervals: H-LB and H-RB, measures of conduction from the His bundle to the proximal bundle branches, and LB-Q and RB-Q, approximating conduction...
from the proximal bundle branches to the onset of ventricular activation. In the present series, there is some variation in H-LB and H-RB, the range of H-LB being 14-28 msec (mean 20) and that of H-RB being 15-29 msec (mean 21). This variation appears to account for much of the variation in H-Q intervals. Patients with the longer H-Q intervals, had longer H-LB and H-RB times. This wider range of H-LB and H-RB intervals may reflect variation in the length of the His bundle, as found in dissection studies, or variation in conduction velocity.

The relationship of LB and RB activation has been of great interest. Our recordings show these to occur almost simultaneously in man, with six of seven patients revealing less than 3-msec variation in LB-RB. One patient had an LB-RB interval of 10 msec. Previous direct recording of bundle branch activation in dogs and bovines suggested that LB and RB activation occurred simultaneously. However, most workers found that left septal depolarization preceded right. It was also demonstrated that when RBBB was surgically induced, no delay in the onset of left ventricular activation occurred. However, when LBBB was experimentally induced, ventricular activation was delayed, proceeding from the right septum. This produced slight PR prolongation.

We attempted to verify these observations in man by utilizing the production of functional bundle branch blocks induced by coupled atrial pacing and by measuring conduction intervals. The most common pattern of aberrant conduction observed was that of RBBB. The QRS pattern noted was related to delay in H-RB and thus, to the LB-RB interval. Complete RBBB pattern was seen in two patients at LB-RB intervals of 30 and 35 msec, respectively. Lesser prolongations of LB-RB were associated with incomplete RBBB pattern. During both incomplete and complete RBBB, H-LB and LB-Q remained unchanged from the normally conducted beats. It is concluded that RBBB in man does not delay the onset of septal activation. It is also of interest that the site of block producing aberrant conduction, was in the proximal RB, probably at the H-RB junction.

One patient also developed functional LBBB during coupled pacing. In this patient, RB-Q remained constant, while the LB potential was delayed. When LB followed RB by 20 msec, incomplete LBBB was seen. When LB followed RB by 25 msec, complete LBBB pattern was noted. Since RB-Q was unchanged during LBBB, it appeared that no delay in the onset of right ventricular activation occurred.

The lack of delay during RBBB was expected from previous work. The lack of delay during LBBB was unexpected, and because of this further studies were undertaken in patients with rate related LBBB. This group was chosen because of the relative ease of recording of H-Q intervals during both normal conduction and complete LBBB produced by atrial pacing. During LBBB, insignificant H-Q prolongation was noted suggesting the absence of delay in the onset of right ventricular activation. It is inferred that P-R prolongations occurring in patients with LBBB do not represent delay in the onset of ventricular activation, but must represent delay at the A-V node, His bundle, or in the right bundle branch system.

In conclusion, proximal left and right bundle branch activation are almost simultaneous in man. Initial left and right ventricular activations also appear to be simultaneous as manifested by almost no delay in the onset of QRS during both left and right bundle branch block. Therefore, H-Q prolongations, which are noted frequently in patients with established bundle branch block, probably represent delay in the unaffected bundle suggesting bilateral bundle branch disease.

Acknowledgment

We would like to thank Miss Barbara Lake for her secretarial help and Mrs. Mary Ellen Rosen for her help in preparation of the illustrations.

Addendum

Following submission of this manuscript, Narula and coworkers have published a paper describing the recording of left bundle branch potentials in man.
References

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