Electrophysiological Observations in Patients with Rate Dependent Bundle Branch Block

By Pablo Denes, M.D., Delon Wu, M.D., Ramesh C. Dhingra, M.D., Fernando Amat-y-Leon, M.D., Christopher Wyndham, M.D., and Kenneth M. Rosen, M.D.

SUMMARY
Electrophysiological studies were conducted in 15 patients with tachycardic rate dependent bundle branch block (RDBBB); ten with left, and five with right. No bradycardic RDBBB was observed, despite occurrence of cycle lengths (CL) longer than 1200 msec in over half the patients studied. Onset of RDBBB was abrupt in 13 patients, and gradual in two. In three patients, the CL allowing reversion to normal conduction (once RDBBB was initiated) was 50, 55, and 190 msec longer, respectively, than the CL inducing RDBBB. Bundle branch refractory periods (RP) were measured with atrial extrastimulus technique in five patients. All RP (except one) were prolonged at all tested CL when compared to patients without conduction disease. The expected decrease in RP with shortening of CL did not occur in four of the five patients.

The electrophysiological abnormality in patients with RDBBB thus appeared to be an increase in refractoriness in the affected bundle branch, along with a loss of the normal decrease in refractoriness with decrease in CL. Similar findings might be demonstrable in patients with early bundle branch disease.

Additional Indexing Words:
His bundle electrogram Cycle length Extra-stimulus technique
Refractory periods Aberrant conduction H-V interval

“UNSTABLE” (INTERMITTENT) BUNDLE BRANCH BLOCK was first described by Lewis in 1913. Several mechanisms including changing autonomic tone and changes in coronary perfusion have been implicated in this phenomenon. Most examples of intermittent bundle branch block occur at critical heart rates and are categorized as rate dependent. The underlying electrophysiological mechanisms for rate dependent bundle branch block (RDBBB) have not been totally defined.

Bundle branch refractory periods are usually shorter than the cycle lengths encountered at physiological heart rates. In addition, bundle branch refractory periods do decrease with cycle length shortens. The occurrence of RDBBB therefore implies an absence of the expected decrease in bundle refractoriness with decrease in cycle length. In this study, we examined bundle branch refractory periods at varying cycle lengths in several patients with RDBBB. In addition, observations are presented concerning: 1) conduction intervals during RDBBB; 2) mechanism of onset of RDBBB; 3) concealed retrograde conduction during RDBBB, as studied by construction of atrial pacing hysteresis curves during normal conduction, and after RDBBB has been established.

Patient Selection
The study group consisted of 15 patients with sinus rhythm and electrocardiographically documented tachycardic rate dependent bundle branch block (RDBBB), who were detected on the inpatient services of either the University of Illinois Hospital or West Side Veterans Hospital. During RDBBB, ten patients had complete left bundle branch block (LBBB), three had complete right bundle branch block (CRBBB), and two had incomplete right bundle branch block (IRBBB). All patients had diagnosed organic heart disease.

Materials and Methods

Electrophysiological Studies
Informed consent was obtained from each of the patients. His bundle (H) potentials were recorded using electrode catheter techniques. His bundle pacing was not utilized for validation of His bundle potentials. Ten consecutive complexes were measured and averaged during normal conduction and during RDBBB, for comparison of conduction intervals. Right and left bundle branch potentials were
simultaneously measured in one of the patients, utilizing previously described techniques. A second electrode catheter was placed in the high right atrium for atrial pacing. Pacing rates were increased in 5–10 beats/min increments until RDBBB was noted. In three of the patients, once RDBBB was established, pacing rates were decreased in similar increments until normal conduction was again established. This increase and subsequent decrease in pacing rates allowed the construction of hysteresis curves.

Bundle branch refractory periods were determined utilizing atrial extra-stimulus technique during sinus rhythm in five of the patients. The bundle branch refractory period was defined as the longest H1-H2 producing the appropriate electrocardiographic pattern of bundle branch block. In three of these five patients, bundle branch refractory periods were also measured at two or more shorter driven cycle lengths (CL). In all of the five patients, the cycle length inducing RDBBB was also considered the bundle branch refractory period at that cycle length. Thus, in the five patients in whom refractory periods were measured, the relationship of bundle branch refractoriness to change in cycle length could be examined.

Bundle branch refractory periods at a given CL were compared with previous data of patients with normal QRS duration and H-V intervals. To examine the effect of change in CL on bundle branch refractory periods, slopes relating cycle length and refractory period (RP) were calculated by the least squares method. These slopes (ΔRP/ΔCL) were then compared with previous data from our laboratory, the normal slope in man being (0.36 ± 0.228; mean ± 2 SD). In one patient, bundle branch recovery time was also measured at several CL at which RDBBB was present. The recovery time was defined as the shortest H1-H2 interval at which H2 resulted in normal intraventricular conduction. This recovery time was determined by delivering 19 basic stimuli (S1) at a cycle length producing RDBBB, and then one extra-stimulus (S2) which was delivered progressively later (in 10 msec increments).

Results

Mechanism of Onset and Return to Normal Conduction (Table 1)

The transition from normal conduction to complete RDBBB was sudden in 13 of the patients. In these patients, once one beat was conducted with RDBBB, all subsequent beats were conducted with RDBBB as long as the paced CL was held constant. In the remaining two patients, increasing grades of incomplete bundle branch block patterns were seen before complete RDBBB was noted. In one of these two patients, left bundle branch Wenckebach periods were noted at a heart rate of 90 beats/min; 2:1 complete left bundle branch block at heart rates of 110 to 140 beats/min, and complete left bundle branch block at rates faster than 140 beats/min.

In the other patient, fixed incomplete left bundle branch block (QRS of 0.11 sec) was noted at a heart rate of 80 beats/min. As rates were increased, increasing QRS durations were noted. At heart rates of 130 beats/min or faster, maximum QRS durations were noted (0.17 sec). In this patient, simultaneous left and right bundle branch potentials were recorded during pacing studies. At a rate of 80 beats/min, LB-V and RB-V were 40 msec (fig. 1A). As CL was decreased, LB-V shortened, while RB-V intervals remained constant. When maximum QRS duration was seen at a CL of 460 msec, the left bundle branch potential was submerged in the ventricular electrogram (fig. 1).

The paced CL producing onset and reversion of RDBBB (once RDBBB was established) was noted in three patients. These hysteresis curves are shown in figure 2. In all patients, the CL inducing RDBBB was shorter than the CL allowing reversion to normal conduction. The differences in CL were 50, 55, and 190 msec, respectively.

No patient had bradycardic RDBBB, despite observed maximum cycle lengths (H-H) ranging from 715 to 1430 msec (table 1).

Conduction Intervals

H-V interval was recorded in all patients, during both normal conduction and during RDBBB (table 1). In the ten patients with rate dependent LBBB, H-V was 51.8 ± 3.1 (mean ± sem) during normal conduction, and 52.7 ± 3.3 msec during LBBB (NS: not significant). The largest observed change in H-V interval contrasting normal conduction and LBBB was 5

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Table 1

<table>
<thead>
<tr>
<th>Pt. no.</th>
<th>H-V (msec)</th>
<th>Critical CL for RDBBB (msec)</th>
<th>Longest H-H interval observed (msec)</th>
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<tbody>
<tr>
<td>ClBBB</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>1</td>
<td>39</td>
<td>40</td>
<td>690</td>
</tr>
<tr>
<td>2</td>
<td>43</td>
<td>43</td>
<td>615</td>
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<tr>
<td>3</td>
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<td>42</td>
<td>690</td>
</tr>
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<td>10</td>
<td>67</td>
<td>72</td>
<td>460</td>
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<tr>
<td>IRRBBB</td>
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<tr>
<td>11</td>
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<td>42</td>
<td>480</td>
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<tr>
<td>12</td>
<td>70</td>
<td>72</td>
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<td>CRBBB</td>
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<tr>
<td>15</td>
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<td>460</td>
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</table>

Abbreviations: RDBBB = rate dependent bundle branch block; NC = "normal" conduction; CL = cycle length; CLBBB = complete left bundle branch block; IRRBBB = incomplete right bundle branch block, and CRBBB = complete right bundle branch block.
H-V intervals were respectively 41 and 70 msec during normal conduction in the two patients with rate dependent IRBBB, and 42 and 72 msec during RDBBB (NS). H-V was 40, 56, 67 msec during normal conduction in the three patients with rate dependent CRBBB, and 40, 56, and 64 msec during RDBBB (NS).

H-V was prolonged (greater than 55 msec) in two of the ten rate dependent LBBB patients (and in one additional LBBB patient during RDBBB), and in three of the five patients with rate dependent incomplete or complete RBBB. H-V prolongation in the above patients suggested additional His bundle or bilateral bundle branch disease.

Bundle Branch Refractory Periods (Table 2)

Bundle branch refractory periods were measured in five of the patients (fig. 3). These were compared to refractory periods measured in 14 previously reported normal patients. Figure 4 shows a plot of refractory period versus cycle length in the present five cases and the previously reported 14 cases. Bundle branch refractory periods were prolonged at all measured CL in the RDBBB patients, except for one refractory period at one CL in patient 15.

The effects of change in CL on bundle branch refractory periods were also studied in the five patients with RDBBB (figs. 3 and 4). The normal response is a decrease in bundle branch refractory
Table 2

<table>
<thead>
<tr>
<th>Pl. no.</th>
<th>CL (msec)</th>
<th>AV-FRP* (msec)</th>
<th>BB-RP (msec)</th>
<th>ΔRP/ΔCL</th>
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</thead>
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<tr>
<td>(9)</td>
<td>980</td>
<td>430</td>
<td>680</td>
<td>- .05</td>
</tr>
<tr>
<td>(11)</td>
<td>640</td>
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<td>(13)</td>
<td>1080</td>
<td>405</td>
<td>970</td>
<td>- .83</td>
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<tr>
<td>(14)</td>
<td>600</td>
<td>400</td>
<td>490</td>
<td>+ .37</td>
</tr>
<tr>
<td>(15)</td>
<td>830</td>
<td>435</td>
<td>450</td>
<td>- .10</td>
</tr>
</tbody>
</table>

Abbreviations: CL = cycle length; AV-FRP = A-V nodal functional refractory period, and BB-RP = bundle branch refractory period.

*R = Shortest propagated H+-H₂ interval.

Discussion

Rate dependent bundle branch block (RDBBB) is diagnosed when bundle branch block appears at a critical heart rate.⁴⁻⁹ This should be differentiated from functional bundle branch block, in which the block is dependent on a preceding long cycle length. Functional bundle branch block (aberrancy) may be a physiological response, while RDBBB is seen almost exclusively in patients with heart disease.¹²,¹⁶,¹⁸

The transition from normal QRS to bundle branch block pattern can be gradual or abrupt. Thirteen of our 15 patients had an abrupt change in QRS morphology, while in two, the change was gradual. Previous reports have described examples of both abrupt and gradual onset, the former being most common in tachycardic RDBBB block.⁴,⁷,⁹,¹⁶⁻¹⁹

The sudden failure of conduction is typical of the diseased His-Purkinje system.²⁰ Abrupt onset RDBBB resembles Mobitz type II A-V block, which generally occurs in patients with established bundle branch block, and presumably reflects sudden failure of conduction in the functioning contralateral bundle branch. Although type II block may be repetitive (paroxysmal heart block), it frequently occurs with only single dropped beats. This is in contrast to the bundle branch block seen in the patients with sudden onset RDBBB, which was almost always maintained, once initiated. This probably reflects the effects of concealed retrograde conduction to the blocked bundle branch, maintaining the bundle branch block.²¹

Gradual onset of RDBBB was noted in only two of our patients.

Loss of RDBBB

Normalization of conduction in patients with RDBBB should depend upon the following factors: 1) refractory period of the bundle branch and its response to change in cycle length; 2) antegrade conduction time in the unblocked bundle branch, transseptal conduction time, and retrograde conduction to the site of RDBBB.²¹ Theoretically, the
Case 9. Determination of left bundle branch refractory period at two different cycle lengths. In each panel, electrocardiographic leads II, III, V₁ and His bundle electrograms (HBE) are shown. CL = cycle length, A₁, H₁, A₂, and H₂ are the atrial and His bundle electrograms of the basic drive and premature atrial stimuli respectively. CL, A₁-A₂, H₁-H₂ intervals are listed in msec. Panels A and B) Shows sinus rhythm at CL of 980 msec. At an A₁-A₂ interval of 690 msec, the H₁-H₂ is 695 msec, and H₂ is conducted with normal QRS configuration. When A₁-A₂ is decreased by 20 msec, the resulting H₁-H₂ is 680 msec. Note that H₂ is conducted with left bundle branch configuration in panel B. Panels C and D) Atrioventricular (AV) node driven at a CL of 750 msec. At an A₁-A₂ interval of 700 msec, the resulting H₁-H₂ is 710 msec and H₂ is conducted with normal QRS configuration. When A₁-A₂ is decreased by 680 msec, the resulting H₁-H₂ is 700 msec. Note that H₂ is conducted with left bundle branch configuration. Also note that RP increases as CL has decreased.

The cycle length at which RDBBB occurs should be shorter than the cycle length at which the rate dependent bundle branch block reverts to normal conduction. This was previously observed by Fisch et al. In the present three cases, the differences between the CL inducing RDBBB, and the CL allowing recovery were 50, 55, and 190 msec. The latter value is longer than expected from theoretical considerations, and may reflect additional depression of conduction from repetitive antegrade or retrograde concealed conduction to the blocked bundle branch, a "fatigue" phenomenon.

Conduction Intervals

The literature concerning the effect of onset of rate dependent LBBB on conduction intervals is controversial. Berkowitz et al.22 based on limited observations, suggested that H-V prolonged once rate dependent LBBB was established. He implied that this reflected the unmasking of a longer conduction time in the right bundle branch system. In contrast, Narula et al. and Rosen et al., also with only limited observations, suggested that H-V remained constant, despite the onset of rate dependent LBBB. They implied that right and left bundle branch conduction times (to the onset of ventricular activation) were similar, so that development of LBBB was not accompanied by prolongation of H-V interval. The present study confirms their previous observations, by demonstrating insignificant change in H-V interval in ten patients with rate dependent LBBB. Since rate dependent LBBB does not prolong H-V interval, prolongation of H-V interval in patients with chronic LBBB presumably reflects conduction delays in either the His bundle or right bundle branch, and not...
physiologic differences in left and right bundle branch conduction times.

**Bundle Branch Refractory Periods in RDBBB**

Bundle branch refractory periods are usually shorter than the cycle lengths encountered at physiological ranges of heart rates. In addition, bundle branch refractory periods normally decrease with a decrease in cycle length. RDBBB will occur when the spontaneous (or driven) CL becomes equal to or exceeds the refractory period of a bundle branch. There are three mechanisms by which this could occur, these being: 1) an absolute increase in bundle branch refractoriness with preservation of the normal cycle length-refractory period relationships; 2) a change of the normal bundle branch refractory period-cycle length relationship with either an increase in refractory period with decreasing CL (negative slope), no change in refractory period with decreasing CL (zero slope), or less than normal decrease in refractoriness with decreasing CL (positive slope, but less positive than normal); 3) a combination of 1) and 2).

Four of our patients had prolonged bundle branch refractory periods at all CL tested. In one patient, all bundle branch refractory periods were prolonged, except for one refractory period measured at a CL of 690 msec. The slopes of ΔRP/ΔCL in four of our patients were flat or negative, an abnormal response. In only one patient were the cycle length-refractory period relationships within the previously reported range, despite prolonged refractoriness of the bundle branches at all CL in this patient. Thus, the findings in our patients suggest prolonged bundle branch refractory periods, as well as abnormal cycle length-refractory period relationships in the genesis of RDBBB.

**Clinical Implications**

Since many patients with RDBBB ultimately develop permanent bundle branch block, we would speculate that the electrophysiological abnormalities demonstrated in the present group of patients apply to patients with early disease in the bundle branch system. It is possible that demonstration of either prolonged bundle branch refractory periods and/or a
loss of the normal decrease in refractory period with decrease in cycle length could predict the subsequent development of bundle branch block in some patients.

References

Eectrophysiological observations in pateints with rate dependent bundle branch block.
P Denes, D Wu, R C Dhingra, F Amat-y-leon, C Wyndham and K M Rosen

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