Electrocardiographic Features and Prevalence of Bilateral Bundle-Branch Delay

Leonidas Tzogias, MD; Leonard A. Steinberg, MD; Andrew J. Williams, MD; Kent E. Morris, MD; William J. Mahlow, MD; Richard I. Fogel, MD; Jeff A. Olson, DO; Eric N. Prystowsky, MD; Benzy J. Padanilam, MD

Background—Definitive diagnosis of bilateral bundle-branch delay/block may be made when catheter-induced right bundle-branch block (RBBB) develops in patients with baseline left bundle-branch (LBBB) block. We hypothesized that a RBBB pattern with absent S waves in leads I and aVL will identify bilateral bundle-branch delay/block.

Methods and Results—Fifty patients developing transient RBBB pattern in lead V1 during right heart catheterization were studied. Patients were grouped according to whether the baseline ECG demonstrated a normal QRS, left fascicular blocks, or LBBB block pattern. The RBBB morphologies in each group were compared. The prevalence of bilateral bundle-branch delay/block pattern was examined in our hospital ECG database. All patients with baseline normal QRS complexes (n=30) or left fascicular blocks (4 anterior, 5 posterior) developed a typical RBBB pattern. Among the 11 patients with a baseline LBBB block pattern, 7 developed an atypical RBBB pattern with absent S waves in leads I and aVL and the remaining 4 demonstrated a typical RBBB. The absence of S waves in leads I and aVL during RBBB was 100% specific and 64% sensitive for the presence of pre-existing LBB block. Among the consecutive 2253 hospitalized patients with RBBB, 34 (1.5%) had the bilateral bundle-branch delay/block pattern.

Conclusions—An ECG pattern of RBBB in lead V1 with absent S wave in leads I and aVL indicates concomitant LBB delay. Pure RBBB and bifascicular blocks are associated with S waves in leads I and aVL.

Key Words: bundle-branch block ■ heart block

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Methods

Study Population
Patients undergoing electrophysiology study or device implantation between January 2002 and October 2012 were included in the study if they developed a new complete RBBB ECG pattern in lead V1 because of unintentional catheter trauma to the right bundle branch. Subjects were grouped by their baseline ECG morphology, which were classified as normal (normal group), left anterior or posterior fascicular block (fascicular block group), and LBBB (LBBB group). Using our hospital’s database, we surveyed 2253 consecutive RBBB pattern ECGs to determine the prevalence of BBBD pattern. The institutional review board approved the study protocols.

Definitions
Conduction system block patterns were defined in accordance with American Heart Association recommendations. LBBB criteria included QRS duration of ≥120 ms; rS or QS deflection in ECG leads V1 and V2; and slurred broad R waves in ECG leads I, aVL, and V6. RBBB criteria included QRS duration of ≥120 ms and a terminal R wave in lead V1. The following exceptions were made in definitions: for LBBB, q waves were permitted in leads I and aVL and for RBBB, S waves were not required in ECG leads I, aVL, and V6. Left anterior fascicular block (LAFB) criteria included a frontal plane axis between −45° and −90°, QR pattern in lead aVL, R-peak time in lead aVL of ≥45 ms, and QRS duration <120 ms. Left posterior fascicular block was established when the frontal plane axis was between 90° and 180° and an rS pattern in leads I and aVL along with a QR pattern in leads III and aVF and QRS duration <120 ms were documented.

Electrocardiograms
During electrophysiology study or device implantation, the surface ECG was monitored continuously and stored on a computer-based digital recording system (LabSystemPro, Bard Electrophysiology, Lowell, MA).
The ECG characteristics at baseline and after the development of catheter trauma–related RBBB were compared. ECGs were analyzed for PR interval, QRS duration, QRS axis, and the presence and depth of R/S waves in leads I, aVL, and V1. All 12-lead ECGs in the hospital’s database were recorded with a sensitivity of 10 mm/mV and a paper speed of 25 mm/s.

Statistics

Two electrophysiologists, one of who was blinded to the study groups, made all ECG measurements. For the purpose of the statistical analysis the patients with baseline normal, LAFB, and left posterior fascicular block were included in a single group, which was compared with the group of patients with baseline LBBB. Comparisons of variables between the study groups were performed using Fisher exact test and Mann–Whitney U test. The sensitivity and specificity of absent S waves in leads I and aVL for identifying BBBD were calculated. An α level of P ≤ 0.05 was the predetermined value for statistical significance. Analyses were performed using MedCalc 12.5.0.0 (MedCalc Software, Ostend, Belgium).

Results

Normal Group

The major ECG characteristics of the groups are compared in Table 1. Among the 30 patients with normal QRS complexes, all developed a typical RBBB pattern with S waves in leads I and aVL (Figure 1). The average S wave depths in leads I and aVL were 3.15 mm (range, 1.5–7 mm) and 3.55 mm (range, 1–12 mm), respectively. In lead V1, all patients had an rSR’ pattern with a mean r wave amplitude of 0.98 mm (range, 0–2.25 mm), S wave of 1.55 (range, 0–3 mm), and R’ of 5.78 mm (range, 2.5–10 mm). At baseline 27 of 30 patients had normal QRS axis and 3 had vertical axis. With the development of RBBB pattern, the QRS axis was normal in 16 of 30 patients (mean, 33°; range, 22°–79°) and right axis deviation was present in the remaining 14 patients (mean, 104°; range, 90°–135°).

Fascicular Block Group

Among the LAFB (n=4) and left posterior fascicular block (n=5) patients, all developed a typical RBBB pattern with S waves in leads I and aVL (Figure 1). In patients with LAFB the average S wave depth in lead I was 2.81 (range, 2–3.25 mm) and in lead aVL 2.5 mm (range, 2–2.75 mm). The QRS axis showed left axis deviation before and after the development of RBBB pattern in all patients. In patients with left posterior fascicular block, the average S wave depths in leads I and aVL were 2.91 mm (range, 2.5–3.25 mm) and 3.54 mm (range 2.5–5.25 mm), respectively. The QRS axis showed right axis deviation before and after the development of RBBB pattern in all patients.

LBBB Group

All 11 patients with baseline LBBB (Table 2) had evidence of structural heart disease. Five patients had previous anterior myocardial infarction and the remaining had nonischemic cardiomyopathy. During catheter manipulation, 7 patients developed an atypical RBBB pattern with no S wave in limb leads I and aVL (Figure 2). The remaining 4 patients developed a typical RBBB pattern with S waves in leads I and aVL (Figure 3). In these 4 patients, the average S wave in lead I was 0.85 mm (range, 0.5–4.5 mm) and aVL was 2.13 mm (range, 1–3 mm). The phenomenon of absent S wave in leads I and aVL with RBBB pattern in V1 was specific to the LBBB group and never occurred in patients with baseline normal QRS or fascicular

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### Table 1. Comparison of Groups

<table>
<thead>
<tr>
<th></th>
<th>LBBB (n=11)</th>
<th>Normal and Fascicular Block (n=39)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median age (IQR), y</td>
<td>61 (57.5–64)</td>
<td>57 (32–68)</td>
<td>0.325</td>
</tr>
<tr>
<td>Male sex, n (%)</td>
<td>8 (72.7)</td>
<td>17 (43.6)</td>
<td>0.17</td>
</tr>
<tr>
<td>Device implant/EPS</td>
<td>5/6</td>
<td>4/35</td>
<td>0.017</td>
</tr>
<tr>
<td>Absent s wave in I</td>
<td>7</td>
<td>0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Absent s wave in aVL</td>
<td>7</td>
<td>0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RBBB QRS duration (IQR), ms</td>
<td>160 (157–168.5)</td>
<td>150 (136–160)</td>
<td>0.004</td>
</tr>
<tr>
<td>Left axis*</td>
<td>11</td>
<td>4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PR duration (IQR), ms</td>
<td>210 (190–230)</td>
<td>158 (144–175)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*Left axis refers to the QRS axis after the development of RBBB.

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Figure 1. Catheter trauma induced right bundle-branch block (RBBB) in patients with baseline (A) normal; (B) left anterior fascicular block (LAFB); and (C) left posterior fascicular block (LPFB). Note the presence of S waves in leads I and aVL during RBBB morphology.
blocks \((P<0.001\)). The sensitivity and specificity of absent S waves in leads I and aVL for identifying RBBB developing on top of the baseline LBBB pattern were 63.6\% (95\% confidence interval, 31.6\%–87.6\%) and 100\% (95\% confidence interval, 89\%–100\%), respectively. For the total group, the RBBB pattern was variable in lead V1 with 6 patients showing an rSR pattern, 2 patients showing an Rs pattern, and 3 patients showing a qR pattern. Mean initial r wave if present was 0.95 mm, mean S wave was 1.73 mm, and mean R’ if present was 7.4 mm. The QRS axis was left axis deviation in 7 and normal in 4 at baseline, whereas the axis was left axis deviation in all 11 patients when RBBB developed. Lead V6 was recorded in only 6 of 11 patients because of device draping interfering with the lead placement. S wave was absent in 3 (all with BBBD pattern) and present in 3 (average, 2 mm).

Prevalence of BBBD Pattern
Among 2253 consecutive RBBB ECGs in our hospital database from January 2010 to April 2012, 34 patients (1.5\%; 95\% confidence interval, 0.011–0.021) had the BBBD pattern (Figure 4). Thirty patients (88\%) had left axis deviation and the mean QRS axis was \(-57\pm21.8^\circ\) (range, 2\(^\circ\) to \(-117^\circ\)). The mean QRS duration was 133±14.8 ms (range, 120–164 ms). An S wave in lead V6 was present in 30 of 34 patients. The mean age of the group was 72.5 years (24 men, 10 women). Eight of these 34 patients had a history of second or higher degree atrioventricular block (n=4) or syncope (n=4). Eight patients had pacemaker/implantable cardioverter defibrillator implantation, 5 of whom had syncope or atrioventricular block. Left ventricular ejection fraction was <40\% in 13 patients and severe aortic stenosis was present in 4 patients.

Discussion
The chief finding of this study is that BBBD can create a characteristic QRS complex consisting of a RBBB pattern in the anterior precordial leads with absent S waves in leads I and aVL. This pattern of RBBB in V1 and LBBB in leads I/aVL, previously described as masquerading bundle-branch block or BBBD, has been postulated to be secondary to either septal myocardial infarction or bilateral bundle-branch disease.\(^3,4\) However, these theories have been based on correlations between ECG patterns and autopsy histopathology. To the best of our knowledge this is the first study to describe a BBBD pattern with direct electrophysiological evidence for its underlying mechanism. Such information is unobtainable from standard ECG analysis or autopsy studies.

In our study, the electrocardiographically defined complete LBBB was presumably not complete in the cases where RBBB developed on top of LBBB. Although there is precedent for using the phrase BBBD\(^4,5\) to describe this finding, this terminology is misleading: both bundle branches cannot be affected.

### Table 2. Clinical and Electrophysiological Features of Individual Patients With Baseline LBBB Who Developed RBBB During Catheter Manipulation

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, Sex, y</th>
<th>Procedure</th>
<th>LVEF, %</th>
<th>QRS, ms</th>
<th>Axis,(^\circ)</th>
<th>s I, mm</th>
<th>s aVL, mm</th>
<th>QRS, ms</th>
<th>Axis,(^\circ)</th>
<th>V1 Pattern</th>
<th>s I, mm</th>
<th>s aVL, mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>67, F</td>
<td>EPS</td>
<td>25</td>
<td>150</td>
<td>(-29)</td>
<td>1.5</td>
<td>1.5</td>
<td>160</td>
<td>(-75)</td>
<td>Rs</td>
<td>1.5</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>61, M</td>
<td>EPS</td>
<td>20</td>
<td>148</td>
<td>(-48)</td>
<td>1.5</td>
<td>1.5</td>
<td>136</td>
<td>(-73)</td>
<td>qr</td>
<td>0.5</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>60, M</td>
<td>EPS</td>
<td>20</td>
<td>138</td>
<td>(-54)</td>
<td>1</td>
<td>1</td>
<td>164</td>
<td>(-81)</td>
<td>rSR</td>
<td>2</td>
<td>1.5</td>
</tr>
<tr>
<td>4</td>
<td>57, M</td>
<td>BIV</td>
<td>20</td>
<td>140</td>
<td>(-72)</td>
<td>0</td>
<td>0</td>
<td>152</td>
<td>(-122)</td>
<td>qr</td>
<td>4.5</td>
<td>3</td>
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<tr>
<td>5</td>
<td>61, M</td>
<td>EPS</td>
<td>40</td>
<td>136</td>
<td>36</td>
<td>0</td>
<td>0</td>
<td>160</td>
<td>(-90)</td>
<td>Rs</td>
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<tr>
<td>6</td>
<td>65, M</td>
<td>BIV</td>
<td>30</td>
<td>160</td>
<td>(-56)</td>
<td>1</td>
<td>1</td>
<td>160</td>
<td>(-50)</td>
<td>rSR</td>
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<td>0</td>
</tr>
<tr>
<td>7</td>
<td>54, F</td>
<td>EPS</td>
<td>20</td>
<td>140</td>
<td>(-30)</td>
<td>2</td>
<td>2</td>
<td>170</td>
<td>(-76)</td>
<td>qr</td>
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<td>0</td>
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<tr>
<td>8</td>
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<td>EPS</td>
<td>25</td>
<td>176</td>
<td>19</td>
<td>0</td>
<td>0</td>
<td>162</td>
<td>(-69)</td>
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<tr>
<td>9</td>
<td>59, M</td>
<td>BIV</td>
<td>20</td>
<td>168</td>
<td>(-56)</td>
<td>0</td>
<td>0</td>
<td>212</td>
<td>(-63)</td>
<td>rSR</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>10</td>
<td>54, M</td>
<td>BIV</td>
<td>25</td>
<td>176</td>
<td>7</td>
<td>0</td>
<td>0</td>
<td>184</td>
<td>(-61)</td>
<td>rSR</td>
<td>0</td>
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<tr>
<td>11</td>
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<td>160</td>
<td>(-18)</td>
<td>1</td>
<td>1.5</td>
<td>156</td>
<td>(-59)</td>
<td>rSR</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

BIV indicates biventricular defibrillator implantation; EPS, electrophysiology study; F, female; LBBB, left bundle-branch block; LVEF, left ventricular ejection fraction; M, male; and RBBB, right bundle-branch block.
be completely blocked. The mechanism could be conduction delay in the left bundle branch with conduction block in the right bundle branch or variable conduction delay in both bundles. It is also possible that myocardial infarction or other pathophysiologic entities might generate a similar ECG pattern, but in our patients developing a new RBBB pattern during catheter manipulation, the described QRS complex was unique to patients with pre-existing LBBB. The BBBD pattern never occurred in patients with ECG evidence of normal left bundle conduction or only fascicular block.

In typical RBBB, the terminal negative deflection (S wave) in the lateral limb leads (I and aVL) is because of delayed right ventricular (RV) depolarization compared with left ventricular depolarization. When concomitant LBBB is present, left ventricular depolarization is also delayed. If the left ventricular depolarization time equals or exceeds the RV depolarization time, lateral lead S waves might be expected to be absent. A majority of the BBBD patients in this study lacked the terminal negative forces (S waves) in the lateral ECG leads, consistent with this hypothesis. Presumably, left ventricular depolarization time exceeds RV depolarization in these patients although we cannot readily explain why the terminal R waves in lead V1 (which also represents delayed RV depolarization) persist when lateral S waves disappear. Lead V1, a right parasternal unipolar lead, records all posterior–anterior vectors of activation. The outflow tract region is among the latest to be depolarized during activation of the normal heart RV. Thus, there may be delayed depolarization of anterior or outflow tract RV myocardium giving rise to a delayed posterior–anterior vector without a significant delay in the left to right vector. The pattern could be dependent on multiple factors including conduction times in the bundle branches and Purkinje fibers, local myocardial conduction velocity, ventricular cavity size, cardiac position, and prior infarct. Further studies with vector electrocardiography and 3-dimensional activation mapping could help clarify the issue.

An understanding of the electrophysiological mechanism underlying this unique BBBD pattern may facilitate clinical practice. We identified this pattern of BBBD in 1.5% of consecutive ECGs reported as RBBB in our hospital database. A high percentage of such patients (8 of 34; 24%) had heart block or syncope. The prevalence of heart block seems to be much higher than the 9% reported with the typical RBBB+LAFB—traditionally considered the commonest ECG pattern preceding the development of complete heart block. Most BBBD ECGs (all 11 catheter induced and 30 of 34 from the hospital database) exhibited RBBB+LAFB pattern with the added finding of absent S wave in leads I and aVL. Thus, although most of these BBBD patients would be identified by traditional criteria for having a risk of heart block, the distinct pattern of BBBD described in this study could conceivably predict a significantly higher risk. A second clinical application might arise in the selection of RBBB patients more likely
to respond to cardiac resynchronization therapy. Patients with RBBB have been shown to be poor responders to biventricular pacing. It is conceivable that in patients with BBBD, the benefit of biventricular pacing may equal or exceed that seen in LBBB patients.

Our study has several limitations. The number of patients identified with the BBBD pattern is relatively small. Direct recordings from the bundle branches were not obtained and may have differentiated block versus conduction delay at each site. It is possible that some patients with LBBD pattern on ECG may also have BBBD. By design this study was incapable of identifying such patients. The evaluation of the clinical features of the BBBD pattern from hospital database is retrospective. A larger, prospective study could clarify the rate of progression to heart block and response to biventricular pacing in these patients.

In summary, this study provides electrophysiological proof that an ECG pattern of RBBB in lead V1 with absent S wave in leads I and aVL is specific for BBBD. Pure RBBB and bifascicular blocks are associated with S waves in leads I and aVL.

Disclosures

None.

References


CLINICAL PERSPECTIVE

Right bundle-branch block (RBBB) patterns showing left anterior or left posterior fascicular block are well known and herald an increased risk for development of complete heart block; however, a RBBB pattern indicative of concomitant underlying left bundle-branch block or delay remains ill defined. Such an ECG pattern is termed bilateral bundle-branch delay. We describe the electrophysiological basis and the ECG features of bilateral bundle-branch delay. A diagnosis of bilateral bundle-branch delay was made when a catheter-induced RBBB pattern developed in patients with pre-existing left bundle-branch block or delay during electrophysiological studies. A pattern of RBBB in lead V1 with absent S waves in leads I and aVL identified bilateral bundle-branch delay with high sensitivity and specificity. Isolated RBBB and bifascicular blocks are associated with S waves in leads I and aVL. Further studies are needed to determine whether this uncommon ECG pattern predicts adverse clinical outcomes.
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