Right, But Not Left, Bundle Branch Block Is Associated With Large Anteroseptal Scar

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Objectives

This study sought to test the hypothesis that right bundle branch block (RBBB) patients have larger scar size than left bundle branch block (LBBB) patients do.

Background

A proximal septal perforating branch of the left anterior descending (LAD) coronary artery most commonly perfuses the right bundle branch and left anterior fascicle, but not the left posterior fascicle. Thus, proximal LAD occlusions should cause RBBB, not LBBB.

Methods

We performed electrocardiograms and magnetic resonance imaging for scar quantification in 233 patients with left ventricular (LV) ejection fraction ≤35% who were receiving primary prevention implantable cardioverter-defibrillators (ICD cohort). Scar size and location were compared among patients with RBBB, LBBB, nonspecific LV conduction delay, and QRS <120 ms. A second cohort of 20 hypertrophic cardiomyopathy patients undergoing alcohol septal ablation was studied to determine whether controlled infarction in a proximal LAD septal perforator caused RBBB or LBBB.

Results

In the ICD cohort, LV ejection fraction was similar between RBBB and LBBB patients (24.9% vs. 25.0%; p = 0.98); however, RBBB patients had significantly larger scar size (24.0% vs. 6.5%; p < 0.0001). Patients with nonspecific LV conduction delay or QRS <120 ms had intermediate scar size (12.9% and 14.4%, respectively). Those with RBBB (compared with LBBB) were more likely to have ischemic heart disease (79% vs. 29%; p < 0.0001). In the alcohol septal ablation cohort, 15 of 20 patients (75%) developed RBBB, but no patients developed LBBB.

Conclusions

In patients with LV ejection fraction ≤35%, RBBB is associated with significantly larger scar size than LBBB is, and occlusion of a proximal LAD septal perforator causes RBBB. In contrast, LBBB is most commonly caused by nonischemic pathologies. (J Am Coll Cardiol 2013;62:959–67) © 2013 by the American College of Cardiology Foundation

A conventional clinical concept is that new onset left bundle branch block (LBBB) or right bundle branch block (RBBB) that occurs with acute myocardial infarction (MI) is associated with massive MI (1–3). However, the relation between coronary artery and bundle branch anatomy suggests that very different relationships should exist between MI location and size with bundle branch block type. A prior necropsy study demonstrated that the proximal left anterior descending coronary artery (LAD) septal perforators perfuse the right bundle branch and the anterior fascicle of the left bundle branch in 90% of cases, whereas the right coronary artery (via the atrioventricular–nodal artery) perfuses the posterior fascicle of the left bundle branch in 90% of cases (4). There is dual blood supply to each of these fascicles in 40% to 50% of cases (4). This means that proximal LAD occlusions could cause RBBB and/or left anterior fascicular block. However, both proximal LAD and right coronary artery occlusions would be required typically for MI to cause LBBB. Instead, as originally described by Lenègre (5), histopathology studies demonstrated that the disruption of the
left bundle branch almost always occurs at its junction with the main bundle, often with histological findings of fibrosis or sclerosis with occasional calcification (5–7). Furthermore, at this location, the left bundle branch can be compressed between connective tissue of the central fibrous body and the base of the ventricular septum (5–7), especially when subjected to mechanical strain from a hypertrophied or dilated left ventricle (LV).

In chronic cardiomyopathy, prolonged QRS duration is associated with increased mortality (8); however, the cause of the association likely differs depending on the conduction type. In LBBB, the large delay between activation of the interventricular septum and LV free wall leads to dyssynchronous and inefficient LV contraction (9). In RBBB, activation of the LV is normal; however, increased mortality may be due to the association of RBBB and large anteroseptal MI that portend poor prognosis.

Cardiac magnetic resonance (CMR) with late gadolinium enhancement (LGE) is the gold standard for identifying the location of and quantifying the amount of myocardial scar caused by prior MI or nonischemic causes of cardiomyopathy (10). In a cohort of chronic ischemic and nonischemic cardiomyopathy patients, we used CMR to test the hypothesis that RBBB patients have a larger scar burden than LBBB patients do because RBBB patients have a higher prevalence of proximal LAD MI and less frequent nonischemic pathologies. As a proof-of-concept group, we also studied the ECG characteristics of a cohort of patients with the obstructive form of hypertrophic cardiomyopathy who underwent percutaneous alcohol septal ablation of the proximal septal perforator and could thus serve as a controlled model of proximal anteroseptal infarction.

**Methods**

**ICD cohort population.** Patients referred clinically to Johns Hopkins Medical Institutions for implantable cardioverter-defibrillator (ICD) placement for primary prevention of sudden cardiac death were prospectively enrolled between November 2003 and December 2010 (11). The population has been described previously (11–14). All patients had to have LV ejection fraction ≤35%, coronary angiography, no other indications for ICD placement, and no contraindications to CMR. Patients were classified as “nonischemic” if they had no history of MI or revascularization and no evidence of coronary artery stenoses >50% of 2 or more epicardial vessels or left main or proximal LAD stenosis >50%. Other patients were classified as “ischemic.” All MI had occurred >1 month prior to enrollment. This study protocol was approved by the Johns Hopkins Institutional Review Board. All patients gave written informed consent.

**Hypertrophic cardiomyopathy alcohol septal ablation cohort.** Patients with hypertrophic cardiomyopathy referred clinically to Johns Hopkins Medical Institutions for percutaneous alcohol septal ablation were enrolled between December 2000 and November 2005 as part of a single-center prospective cohort of CMR before and after septal ablation. This study protocol was approved by the Johns Hopkins Institutional Review Board. All patients gave written informed consent.

**CMR acquisition and analysis in ICD cohort.** Patients underwent cine and CMR-LGE imaging using a 1.5-T scanner (GE Signa CV/I, GE Medical Systems, Milwau-

kee, Wisconsin; or Siemens Avanto, Siemens Medical Solutions, Malvern, Pennsylvania). Image analysis was performed using custom research software CINEtool (GE Healthcare, Milwaukee, Wisconsin). Cine images were used to measure ejection fraction and volumes, and LGE images were used to measure total scar size for the entire LV. The LGE area was outlined, and pixels with signal intensity >50% of the maximum within the LGE area were labeled as scar “core” (12,14). A region of normal myocardium without artifacts was then selected and the peak signal intensity within the normal myocardium was determined. Myocardium with signal intensity greater than peak remote signal intensity but <50% of maximal signal intensity within the LGE region was labeled the “gray” zone to represent the heterogeneous peri-scar zone (12,14). Total scar size was expressed as core +1/2 gray zone as a percentage of total LV mass (14).

Scar location was determined using the American Heart Association’s 17-segment model of the LV (15). Patients with ischemic cardiomyopathy were grouped into those having infarct with a(n) LAD, right coronary artery (RCA), and/or left circumflex infarction pattern (15). Patients with nonischemic cardiomyopathy were grouped into 6 patterns:

1. no scar present;
2. scar confined to the mid-wall myocardium;
3. scar confined to the epicardium;
4. scar confined to the endocardium;
5. transmural scar; and
6. scar at the right ventricular insertion points based on previously described patterns (16). Scar transmural involvement in each of the 17 segments was graded on a 0–4-point scale as described previously: 0 for 0% hyperenhancement; 1 for 1% to 25% hyperenhancement; 2 for 26% to 50% hyperenhancement; 3 for 51% to 75% hyperenhancement; and 4 for 76% to 100% hyperenhancement (17).

**ECG analysis for both cohorts.** ECG at the time of CMR were analyzed according to previously specified criteria (14,18,19):
Table 1  Baseline Characteristics of ICD Cohort

<table>
<thead>
<tr>
<th></th>
<th>RBBB (n = 19)</th>
<th>LBBB (n = 45)</th>
<th>LVCD (n = 33)</th>
<th>QRSd &lt;120 ms (n = 134)</th>
<th>p Value</th>
<th>RBBB vs. LBBB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yrs</td>
<td>59.1 ± 7.8</td>
<td>59.7 ± 11.4</td>
<td>64.2 ± 12.6</td>
<td>54.6 ± 12.2</td>
<td>0.85</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>14 (74)</td>
<td>29 (64)</td>
<td>27 (77)</td>
<td>109 (81)</td>
<td>0.48</td>
<td></td>
</tr>
<tr>
<td>LVEF, %</td>
<td>24.9 ± 8.7</td>
<td>25.0 ± 9.0</td>
<td>26.1 ± 8.9</td>
<td>28.3 ± 8.9</td>
<td>0.98</td>
<td></td>
</tr>
<tr>
<td>Ischemic</td>
<td>15 (79)</td>
<td>13 (29)</td>
<td>25 (71)</td>
<td>78 (58)</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>QRS duration, ms</td>
<td>150.3 ± 16.8</td>
<td>162.2 ± 20.3</td>
<td>130.7 ± 14.1</td>
<td>98.3 ± 10.7</td>
<td>0.028*</td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
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<td></td>
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<tr>
<td>Caucasian</td>
<td>15 (79)</td>
<td>34 (76)</td>
<td>27 (77)</td>
<td>86 (64)</td>
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<tr>
<td>African American</td>
<td>3 (16)</td>
<td>10 (22)</td>
<td>8 (23)</td>
<td>42 (31)</td>
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<td></td>
</tr>
<tr>
<td>Other</td>
<td>1 (5)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>5 (4)</td>
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<tr>
<td>NYHA functional class</td>
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<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>I</td>
<td>4 (21)</td>
<td>2 (4)</td>
<td>9 (26)</td>
<td>43 (32)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>7 (37)</td>
<td>16 (36)</td>
<td>4 (11)</td>
<td>68 (51)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>8 (42)</td>
<td>27 (60)</td>
<td>22 (63)</td>
<td>23 (17)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV/BSA, ml/m²</td>
<td>114.9 ± 34.4</td>
<td>137.2 ± 50.0</td>
<td>131.4 ± 43.1</td>
<td>115.6 ± 36.3</td>
<td>0.081*</td>
<td></td>
</tr>
<tr>
<td>LVESV/BSA, ml/m²</td>
<td>89.0 ± 37.3</td>
<td>105.9 ± 51.7</td>
<td>98.8 ± 40.5</td>
<td>84.8 ± 34.6</td>
<td>0.20*</td>
<td></td>
</tr>
<tr>
<td>LV mass/BSA, ml/m²</td>
<td>73.5 ± 22.5</td>
<td>82.1 ± 32.6</td>
<td>78.9 ± 27.0</td>
<td>70.2 ± 20.8</td>
<td>0.30*</td>
<td></td>
</tr>
</tbody>
</table>

Values are mean ± SD or n (%). Bold values are statistically significant. *p < 0.05 for groupwise comparison among 4 groups.

- **LBBB**: QRS duration ≥140 ms (men) or ≥130 ms (women), QS or rS complex in leads V1 and V2 with mid-QRS notching/slurring in 2 of the leads I, aVL, V1, V2, V5, or V6.
- **RBBB**: QRS duration ≥120 ms with rR' or qR complex in lead V1 and a wide S wave in lead I (patients with left axis deviation [−180° to −45°] were classified as left anterior fascicular block + RBBB; 
- **Nonischemic LV conduction delay (LVCD)**: QRS duration ≥120 ms and QS or rS complex in lead V1, but not meeting LBBB or RBBB criteria; and
- **QRS duration <120 ms**.

**Statistical analysis.** Variables following Gaussian distributions were compared by ECG conduction type with parametric measures (2-sample Student t test, 1-way analysis of variance), whereas those not following Gaussian distributions were analyzed nonparametrically (Wilcoxon rank sum, nonparametric 1-way analysis of variance). Categorical variables were evaluated by chi-square. Analysis was performed separately comparing RBBB and LBBB patients and comparing all 4 ECG conduction types. The p values <0.05 were considered significant.

**Results**

Of the 235 primary prevention ICD patients available for analysis, 2 were excluded because only ventricular-paced ECG were available. The remaining 233 (77% were men) had a mean age of 57 years, a mean LV ejection fraction of 27%, 56% ischemic etiology, and a distribution of New York Heart Association heart failure classes of 25% class I, 41% class II, and 34% class III. There were no differences in age, sex, ethnicity, heart failure class, or LV ejection fraction between RBBB and LBBB patients (Table 1). However, there were significant differences in scar size, anatomic scar location, and prevalence of ischemic versus nonischemic cardiomyopathy. In addition, there was a trend toward larger LV end-diastolic volume in LBBB patients.

**Right versus left bundle branch block.** Figure 1 shows the CMR-LGE image of a RBBB patient with ischemic cardiomyopathy and a large anteroseptal scar involving 45% of the LV, and Figure 2 shows the CMR-LGE of a LBBB patient with nonischemic cardiomyopathy and no scar. As a group, the mean scar size in RBBB patients was significantly greater than in LBBB patients (24.0% vs. 6.5%; p < 0.0001), whereas patients with nonspecific LVCD or QRS duration <120 ms had intermediate scar size (12.9% and 14.4%, respectively) (Table 2). Of note, even though analysis in this study was performed with the pre-specified strict LBBB criteria (14,18,20), defining LBBB with conventional LVCD criteria (QRS ≥120 ms, with QS wave or rS complex in lead V1) still resulted in a large difference in scar size between LBBB (9.3 ± 10.2% LV) and RBBB (24.0 ± 15.7% LV) patients (p < 0.0001).

The difference in scar size was partially explained by a higher prevalence of ischemic cardiomyopathy in RBBB compared with LBBB patients (79% vs. 29%; p < 0.0001). However, among ischemic cardiomyopathy patients, a larger scar size persisted among those with RBBB versus those with LBBB (28.5% vs. 14.7%; p = 0.006). The scar distribution in each of the 17 segments is shown in polar plots in Figure 3. The right bundle branch runs approximately through segment 8 (mid-anteroseptal wall), which was the segment with the highest transmural scar grade among the RBBB patients, and was significantly higher than...
the mid-anteroseptal scar grade (measure of scar transmural involvement) in LBBB patients (2.47 vs. 0.89; p = 0.001).

When considering only ischemic patients, 11 of 15 (73%) RBBB patients had scar consistent with LAD occlusions (2 of whom also had scar in a second territory), and the average scar size was 34.8% of the LV (range 19% to 50% LV) for LAD-only scar. Of the 4 other ischemic RBBB patients, 2 were RCA-territory scars, 1 was a left circumflex scar, and 1 was not a typical ischemic pattern (scar near right ventricular insertion points). In contrast, among the 13 ischemic LBBB patients, there were 3 with scar patterns consistent with LAD + RCA, 6 LAD-only, 2 RCA-only, and 2 left circumflex-only. Prior necropsy studies have suggested that both LAD and RCA infarcts would usually be required for MI to be the sole cause of a LBBB, but it is unlikely for a LAD-only MI to cause a LBBB. Among the 6 LAD-only LBBB patients, only 1 of them had a large scar (35% LV), whereas the remainder had scar sizes not more than 16% of the LV (range: 5% to 16%). This suggests that in at least 5 of these 6 cases, the LAD scar was not likely the direct cause of the LBBB, but rather the LBBB developed due to stress and strain on the left bundle fibers associated with LV dilation and progressive cardiomyopathy. Indeed, there was a trend toward larger LV end-diastolic volume (corrected for body surface area) in LBBB versus RBBB patients overall (137.2 vs. 114.9 ml/m²; p = 0.081).

Among nonischemic patients, 0 of 4 RBBB patients had no scar, whereas 20 of 32 (63%) LBBB patients had no scar (p = 0.017 for comparison). There was no significant difference in the scar patterns (mid-wall, epicardial, endocardial, transmural vs. right ventricular insertion) in RBBB versus LBBB patients.

**Alcohol septal ablation.** QRS duration was normal in all hypertrophic cardiomyopathy patients prior to the septal ablation. Subsequent to the alcohol septal ablation of a proximal LAD septal perforator, 15 of 20 (75%) developed RBBB, whereas no patients developed LBBB. Figure 4 shows the ECG of a patient before and after alcohol

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**Figure 1** CMR-LGE and ECG of RBBB Patient With Ischemic Cardiomyopathy and Large Anteroseptal Scar

This patient had extensive scarring (arrows) by cardiac magnetic resonance late gadolinium enhancement (CMR-LGE) (A) from a prior proximal left anterior descending coronary artery occlusion. The patient’s electrocardiogram (ECG) (B) showed a right bundle branch block (RBBB) with left anterior fascicular block.
septal ablation that developed RBBB. The patients’ CMR-LGE image shows a focal area of LGE in the basal septum representing the area of necrosis causing RBBB.

Among those who developed RBBB, 3 of 15 also developed left anterior fascicular block. QRS duration increased by 42 ± 11 ms among those developing RBBB (p < 0.0001 compared with baseline), whereas there was no significant change in QRS duration among those that did not develop RBBB (p = 0.24). No patients developed left anterior fascicular block without RBBB.

**Discussion**

Our study supports the premise that RBBB has a strong association with large anteroseptal scar in cardiomyopathy patients, and occlusion of a proximal LAD septal perforator causes RBBB. In contrast, the majority of LBBB patients have nonischemic cardiomyopathies and, even among those with ischemic cardiomyopathy, LBBB patients have significantly lower overall scar burden than RBBB patients do, whereas LVCD and QRS duration <120 ms patients have intermediate scar sizes. These results oppose the conventional clinical concept that LBBB is caused by massive septal infarction. Instead, LBBB is more likely caused by a combination of sclerosis and fibrosis combined with mechanical strain on the left bundle fibers near the left bundle junction with the main bundle, where the conduction fibers are sandwiched between the connective tissue of the central fibrous body and the base of the ventricular septum (5–7). However, it is possible that these observations are an artifact of survival bias. The relationship between proximal LAD occlusions and RBBB (but not LBBB) was further confirmed by our cohort with hypertrophic cardiomyopathy in whom alcohol ablation of a proximal LAD septal perforator led to RBBB in 75% of patients, but
not to LBBB. This evidence that LAD occlusions cause RBBB, but not LBBB, has important potential clinical implications for primary prevention ICD and cardiac resynchronization therapy (CRT) patients, such as those in this study, but also potentially for acute MI patients. CRT studies performed in the past decade were limited by the lack of distinction between ventricular block types. CRT trials enrolled patients with QRS duration ≥120 ms (without distinction between conduction types) and showed that CRT reduced heart failure symptoms, heart failure

### Table 2  CMR-LGE Scar Size of ICD Cohort

|                     | RBBB | LBBB | LVCD | QRSd <120 ms | p Value  
|---------------------|------|------|------|--------------|-----------
| All                 | 19   | 45   | 35   | 134          | <0.0001*  
| Scar size, %LV            | 24.0±15.1 | 6.5±9.4 | 12.9±10.0 | 14.4±13.7 | <0.0001*  
| Scar core, %LV            | 17.8±11.1 | 4.3±6.1 | 9.4±7.1 | 10.6±10.4 | <0.0001*  
| Scar gray, %LV           | 12.3±9.2 | 4.2±7.0 | 7.0±6.4 | 7.6±7.9 | <0.0001*  
| Ischemic             | 15   | 13   | 25   | 77           |          
| Scar size, %LV            | 28.5±13.7 | 14.7±9.7 | 17.4±8.2 | 22.5±11.0 | 0.006*  
| Scar core, %LV            | 21.0±10.2 | 10.0±6.2 | 12.6±5.7 | 16.7±8.8 | 0.002*  
| Scar gray, %LV           | 14.7±8.6 | 9.0±7.3 | 9.5±6.0 | 11.6±7.1 | 0.072  
| Nonischemic           | 4    | 32   | 10   | 57           |          
| Scar size, %LV            | 7.3±4.7 | 3.2±7.1 | 1.8±1.9 | 3.4±8.1 | 0.27   
| Scar core, %LV            | 5.7±3.4 | 2.0±4.3 | 1.4±1.6 | 2.3±5.4 | 0.110  
| Scar gray, %LV           | 3.1±4.5 | 2.3±6.0 | 0.8±0.8 | 2.2±5.4 | 0.80   

Values are n or mean ± SD. Bold values are statistically significant. *p < 0.05 for groupwise comparison among 4 groups. CMR-LGE = cardiac magnetic resonance late gadolinium enhancement; %LV = percentage of left ventricle; other abbreviations as in Table 1.

**Figure 3 Segmental Scar Distribution in the ICD Cohort**

Polar plots show the average transmural scar extent within each of the 17 American Heart Association–defined myocardial segments graded on a 0-to-4-point scale (0 for 0% hyperenhancement; 1 for 1% to 25% hyperenhancement; 2 for 26% to 50% hyperenhancement; 3 for 51% to 75% hyperenhancement; and 4 for 76% to 100% hyperenhancement) (17). The figure is scaled from no scar (light yellow) to a transmural scar grade of 2.5 (dark red). Note that the right bundle branch runs through segment 8 (mid-anteroseptal), which had the highest scar grade of any segment in RBBB patients. ICD = implantable cardioverter-defibrillator; LVCD = left ventricular conduction delay; QRSd = QRS duration; other abbreviations as in Figures 1 and 2.
This hypertrophic cardiomyopathy patient had a baseline ECG (A) showing left ventricular hypertrophy, but not bundle branch block. Alcohol septal ablation was performed in a proximal left anterior descending coronary artery septal perforator leading to necrosis highlighted by arrows in the CMR-LGE image (B). Post-septal ablation ECG (C) showed that RBBB developed. Abbreviations as in Figure 1.
hospitalization, and mortality (21–26). However, more recent analysis has suggested that the benefit from CRT is driven by LBBB patients (26–28), especially in the MADIT-CRT (Multicenter Automated Defibrillator Implantation Trial) that enrolled New York Heart Association class I and II patients (27). In that trial, neither RBBB patients nor nonspecific LVCD patients benefited from CRT. Furthermore, large outcome studies of Medicare patients demonstrated that, among CRT patients, mortality is highest with RBBB, intermediate with LVCD, and lowest with LBBB (29,30). CRT likely benefits LBBB patients most because RBBB and LVCD patients have normal Purkinje activation of the LV, but poor prognosis with RBBB may also be due to RBBB association with large scar burden, as demonstrated in the present study.

Our results have potential implications for acute MI patients, as well. Prior studies have described the association between new onset LBBB and occlusion of the proximal LAD artery and large MI size (2). However, this was based on remote studies using Q waves in leads V1 to V3 to determine anteroseptal MI location and creatine kinase–measured infarct size (1). Recent studies using CMR-LGE have demonstrated that in LBBB, large R waves (not Q waves) in leads V1 to V3 represent anteroseptal MI (14,18). As discussed previously, post-mortem studies support the observation that proximal LAD septal perforators most commonly perfuse the right bundle branch and the anterior half of left bundle branch, whereas the RCA (via the atrioventricular-nodal artery) most commonly perfuses the posterior half of the left bundle branch (4). Thus, proximal LAD occlusions can cause RBBB and/or left anterior fascicular block; however, both proximal LAD and RCA occlusions would typically be required for infarcts to be the cause of LBBB. In addition, some patients diagnosed with LBBB by conventional ECG criteria do not have activation consistent with LBBB and the recently proposed strict LBBB criteria (14,18,20) used in the present study should be assessed in future studies in patients presenting with symptoms of acute coronary syndromes.

Our data from the hypertrophic cardiomyopathy patients undergoing septal ablation of a proximal LAD septal perforator support this concept as 15 of 20 patients developed RBBB (with 3 developing left anterior fascicular block in addition to RBBB), whereas no patients developed LBBB. This is consistent with previous studies (31,32). Qin et al. (31) reported that 62% of patients developed RBBB after septal ablation, whereas 6% developed LBBB. It is possible that the limited number of patients supposedly developing LBBB did not actually develop LBBB, but rather left anterior fascicular block that caused the patients to meet conventional ECG criteria for LBBB, but they would not have met strict criteria for LBBB used in this study (20).

**Study limitations.** All patients enrolled in the ICD cohort portion of this study had an ejection fraction ≤35% and met criteria for primary prevention ICD. Thus, the results cannot necessarily be extrapolated outside of this population. Although we comment on how our findings might translate to patients presenting with bundle branch block and acute MI, this should be interpreted with caution and requires further study. Although the subgroup comparison of ischemic patients still demonstrates smaller scar in the LBBB group, this may be an artifact of survival bias; specifically, that patients with large anterior MI who develop or have LBBB are less likely to survive long enough to qualify for an ICD. Studying hypertrophic cardiomyopathy patients undergoing alcohol septal ablation of a proximal LAD septal perforator did allow us to investigate the direct effect of proximal LAD occlusion in an acute setting; however, directed alcohol infusion down a septal perforator may not be representative of naturally occurring LAD coronary occlusions.

**Conclusions**

In a chronic cardiomyopathy cohort, RBBB is associated with ischemic cardiomyopathy and large anteroseptal scar, whereas LBBB is associated with nonischemic etiologies. The large myocardial scar in RBBB patients may explain why they have even worse mortality than do nonspecific LVCD patients receiving CRT. This study highlights that the LAD branches perfuse the right bundle branch and left anterior fascicle, not the entire left bundle branch. Future clinical prognostic studies should specifically distinguish between RBBB and LBBB patients rather than consider them homogeneously. Assessment of scar burden as another risk factor may also be of value.

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