The Pathologic Correlates of the Electrocardiogram: Complete Left Bundle Branch Block

CHRISTOPHER J. HAVELDA, M.D., GURBACHAN S. SOHI, M.D., NANCY C. FLOWERS, M.D., AND LEO G. HORAN, M.D.

SUMMARY  To assess whether gross pathologic differences exist between hearts with left bundle branch block (LBBB) and left-axis deviation (LAXD) and those with LBBB and a normal frontal plane axis, we examined 70 hearts with LBBB in a series of 1410 sequential dissections (5%). Thirty-two hearts had LAXD and 34 had normal axes on the correlative ECG. Left ventricular enlargement occurred frequently (93%). No significant differences were found in age distribution, left ventricular weight, coronary anatomy or infarct location. Quantitative analysis revealed larger inferoposterolateral and apical infarcts in hearts with LBBB and LAXD (p < 0.01). The accuracy of various electrocardiographic signs of left ventricular enlargement and myocardial infarction in the presence of LBBB was assessed. Voltage criteria and QRS duration poorly define anatomic chamber enlargement. Anterior infarction is suggested by a q or pathologic Q wave in lead I, a q wave in leads I, V₁, and V₅, or notched S waves in V₅ or V₆. Pathologic Q waves or ST shifts in the inferior leads have high diagnostic specificity but low sensitivity for inferior infarction.

THE CLINICAL PRESENCE of complete left bundle branch block (LBBB) on the ECG is frequently associated with ischemic or hypertensive heart disease and cardiac enlargement. The prognosis of LBBB is excellent when detected at an early age in an otherwise normal subject. However, prospective long-term follow-up in an older population shows that newly acquired LBBB carries a 50% 10-year survival. Concurrent left-axis deviation (LAXD) and LBBB reportedly portend a greater incidence of myocardial dysfunction, more advanced conduction system disease and a greater cardiovascular mortality than in patients with LBBB and a normal frontal plane axis. Whether the presence of LAXD with LBBB indicates a greater degree of myocardial pathology remains unknown. LBBB can obscure the ECG findings of myocardial infarction (MI) and left ventricular enlargement (LVE); however, in the presence of LBBB, there may be subtle ECG changes that indicate the location of myocardial infarction or the presence of LVE. Some of these contentions remain controversial.

In this study we examined the gross pathologic correlates of LBBB to determine whether pathologic differences exist between hearts with LBBB and LAXD, and those with LBBB and a normal frontal plane axis. We also sought to establish the value of the ECG in detecting both LVE and the anatomic location of MI in the presence of LBBB.

Methods

During a 6-year period, information was gathered on 1500 autopsied male patients from a general hospital in whom pathologic examination of the heart was supported by correlative ECGs demonstrating supraventricular antegrade conduction. All hearts were dissected by two of the authors according to strict protocol.

In this study, the presence of myocardial deficits, either fibrosis or necrosis, of any degree is described by the term infarction. The location of such myocardial deficits was recorded on detailed protocol sheets. The myocardium of the left ventricle was subdivided into septal, anterior, and inferoposterolateral areas using clear anatomic boundaries. Each of these areas was further subdivided into two basal, two central and one apical zone, for a total of 15 zones (fig. 1). The

From the Veterans Administration Medical Center and the Division of Cardiology, Department of Medicine, University of Louisville School of Medicine, Louisville, Kentucky.

Supported in part by NHLBI grants HL-19768 and HL-16901, a grant from the Louisville and Jefferson County and Kentucky Heart Associations, and an award from the Veterans Administration.


Address for correspondence: Christopher J. Havelda, M.D., Division of Cardiology, Veterans Administration Medical Center, Building 19, 800 Zorn Avenue, Louisville, Kentucky 40202.

Received August 4, 1980; revision accepted June 10, 1981.

volume of each zone replaced by infarction was estimated using a scoring system of 0-9, where 0 indicated no damage and 9 indicated total replacement by infarction. The site and extent of the coronary artery lesions were also documented. The right (RCA), left anterior descending (LAD), and left circumflex (LCX) vessels were divided into proximal, middle and distal thirds; separate notations were made of the left main coronary artery and the first large diagonal branch of the LAD. The percent reduction of intraluminal cross-sectional area was noted and a modified Friesinger coronary artery score was assigned to each segment. According to this scoring system, a hemodynamically significant lesion was considered to be present when 75% or greater reduction in intraluminal cross-sectional area was found, and a score of 3-5 points was thus generated. Lesions of less than 75% reduction in intraluminal cross-sectional area generated scores of 1 or 2 points. Thus, a total of 15 points could be achieved for each vessel if total obstruction was present in its proximal, middle and distal thirds.

In this study, the upper limit of normal for the isolated left ventricular and septal weight (LVW) was 180 g; an LVW of 181-250 g represented mild-to-moderate LVE, 251-300 g represented moderate-to-severe LVE, and greater than 300 g represented marked LVE.

The ECG file for each specimen was examined and the most recent ECG record, usually taken at the time of terminal admission, and in all instances within 30 days of death, was analyzed for correlation. ECGs that demonstrated conduction from above the atrioventricular node, a QRS complex in the limb leads of 0.12 second or greater, a predominantly upright complex in leads I, aV1, and V6, and a predominantly negative complex in lead V1 were considered to be examples of LBBB. Of 1410 completed protocols, 70 ECGs (5%) met the criteria for LBBB and formed the basis of this report. In these 70 instances, frontal plane vectorial QRS-axis determinations were derived for each of the three pairs of standard limb leads at right angles. The mean frontal plane QRS axis was derived from an average of these three axes. In this study, a mean QRS axis between -31° and -90° represented LAXD.

In analyzing the accuracy of ECG signs of left ventricular enlargement and myocardial infarction to predict anatomic chamber enlargement and infarct location in the presence of LBBB, we used diagnostic, rather than nosologic, sensitivity, specificity and accuracy determinations. We considered the evaluation of diagnostic sensitivity and specificity of greater practical value than the nosologic criteria because the diagnostic specificity reflects the degree to which a positive sign specifies the disease, and the diagnostic sensitivity reflects the degree to which a negative sign excludes the disease.

Results

The mean frontal plane QRS axis was normal (+110° to -30°) in 34 hearts, while 32 hearts exhibited LAXD (axis of -31° to -90°). Three hearts exhibited a severe leftward axis shift of greater (i.e., more negative) than -90°, and one heart showed marked right-axis deviation (more than +110°). These four specimens were excluded from further comparisons between hearts with normal axes and those with LAXD. Sixty-five hearts had an increased LVW, and 32 (of 63 with adequate coronary records) had significant coronary artery obstruction. MI was found in 42 specimens; however, infarction of the septal summit was not universal, and lesions extending into the region of the left bundle (zone 3) were found in only 15 specimens.

Pathologic Features of LBBB with LAXD or a Normal Frontal Plane Axis

The mean age of patients with LBBB and LAXD was no different from that of patients with LBBB and a normal axis (67.1 ± 7.8 vs 64.6 ± 11.6 years [± sd], NS). Only five specimens (7%) had a normal LVW, and 48 specimens (69%) had moderate-to-marked LVE (table I). Of 63 hearts with adequate coronary artery records, 60 had a mean QRS axis within the normal or LAXD axis ranges. In 31 specimens there
were insignificant or no coronary artery lesions, while 15 had significant one-vessel, 12 had significant two-vessel and five had significant three-vessel coronary disease. Chi-square and student t tests, respectively, indicated no significant differences in the distribution of coronary artery lesions or coronary artery scores* between those hearts with LAXD and those with normal axes.

Of the 70 hearts with LBBB, 40 of 42 infarcted specimens and 26 of 28 specimens without infarction fell within the +110° to −90° axis range. When these 40 infarcted hearts were compared with the 26 hearts without infarction, no difference in axis distribution was found (−10.0 ± 51.6° vs −17.9 ± 50.2°, respectively [mean ± sd], NS). In the 40 infarcted specimens, the area of infarction was usually large, accounting for an average of 20% of the myocardial volume, and some degree of invasion of the infero-posterolateral zones was frequently found. The distribution of infarction in the normal-axis and LAXD groups is shown in table 2. A significant overall difference in the average infarct score between hearts with normal axis and those with LAXD was found (p < 0.05). However, no differences in infarct scores were found between hearts with normal axes and those with LAXD when comparing the septal (zones 1–5) or anterior (zones 6–10) regions. Both groups had their highest mean scores in inferoposterolateral regions, but when the average score for each zone is analyzed, a significantly larger area of infarction was found in both the infero-posterolateral (zones 11–15, p < 0.01) and apical (zones 5, 10 and 15, p < 0.005) regions in hearts with LAXD. Infarcts in these regions (zones 5, 10 and 11–15) accounted for an average of 7.75 more points in the LAXD hearts than in the normal-axis hearts. Frequent overlap into adjacent zones was seen; however, infarction of the lateral free wall, present in 34 of 42 infarcted specimens, did not correlate with the frontal plane axis. In the 28 specimens without infarction, 12 had LAXD. No differences in LVW or coronary artery score could be found between these and the 14 hearts without infarction and axes in the normal range. Hence, no explanation for LAXD could be found by gross pathologic inspection in these non-infarcted specimens.

Electrocardiographic Diagnosis of LVE in LBBB

The sensitivity, specificity and diagnostic accuracy for various voltage criteria used in the ECG diagnosis of LVE and their relative values in predicting anatomic LVE in the presence of LBBB are given in table 3. Although most criteria had fairly high specificities and few false positives, false-negative findings were frequent, yielding poor overall sensitivity (0.30–0.62) and diagnostic accuracy (0.30–0.63). Total QRS duration did not correlate with LVW. The correlation between LVW and maximal QRS duration in either the standard limb, precordial, or all 12 ECG leads was poor (r = 0.264, r = 0.169, r = 0.186, respectively).

Electrocardiographic Diagnosis of MI in the Presence of LBBB

Fifty-seven ECG criteria proposed as predictors of the site of myocardial infarction in the presence of LBBB were gathered from the literature and were tested against the anatomic location of infarction in the correlative pathologic specimen.* 13-36 With few exceptions, these predictors showed poor correlation between the ECG criteria of infarction and the site of anatomic infarction.

Four predictors of anterior wall infarction were found: (1) An initial q wave of any size in lead I

---

**Table 1. Distribution of Left Ventricular Weight in Hearts with Left Bundle Branch Block**

<table>
<thead>
<tr>
<th>LV weight (g)</th>
<th>&lt;180</th>
<th>181-250</th>
<th>251-350</th>
<th>&gt;351</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>All specimens</td>
<td>5</td>
<td>17</td>
<td>29</td>
<td>19</td>
<td>70</td>
</tr>
<tr>
<td>Specimens with infarcts</td>
<td>1</td>
<td>12</td>
<td>19</td>
<td>10</td>
<td>42</td>
</tr>
<tr>
<td>Specimens with LAXD</td>
<td>2</td>
<td>4</td>
<td>14</td>
<td>12</td>
<td>32</td>
</tr>
<tr>
<td>Specimens with infarcts + LAXD</td>
<td>1</td>
<td>3</td>
<td>9</td>
<td>7</td>
<td>20</td>
</tr>
</tbody>
</table>

Abbreviations: LV = left ventricular; LAXD = left-axis deviation.

---

**Table 2. Comparison of Scar Score Between Hearts with Left Bundle Branch Block and Normal Frontal Plane Axis and Hearts with Left Bundle Branch Block and Left-axis Deviation**

<table>
<thead>
<tr>
<th>Zone</th>
<th>Normal axis</th>
<th>LAXD</th>
<th>Normal axis</th>
<th>LAXD</th>
<th>% total scar</th>
<th>p†</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2.00</td>
<td>1.00</td>
<td>12%</td>
<td>16%</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>1.85</td>
<td>1.85</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>0.95</td>
<td>0.90</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>0.70</td>
<td>2.05</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>1.15</td>
<td>2.95</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>0.65</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>1.25</td>
<td>1.90</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>1.35</td>
<td>1.10</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>2.00</td>
<td>1.45</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>1.30</td>
<td>2.65</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>1.70</td>
<td>2.55</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>2.20</td>
<td>2.70</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>2.05</td>
<td>2.65</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>2.25</td>
<td>3.10</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>1.75</td>
<td>3.55</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*A scar score was assigned to each zone on the basis of 0–9 point grading system, where 0 represented a zone free of scar and 9 represented a zone totally replaced by scar.

†Paired t test.

Abbreviations: LAXD = left-axis deviation.
Table 3. Value of Various Electrocardiographic Criteria in the Diagnosis of Left Ventricular Enlargement in the Presence of Left Bundle Branch

<table>
<thead>
<tr>
<th>LVE criteria</th>
<th>LVW &gt; 250 g</th>
<th></th>
<th>LVW &gt; 300 g</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SENS</td>
<td>SPEC</td>
<td>ACC</td>
<td>SENS</td>
</tr>
<tr>
<td>Limb leads</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a. R in lead I &gt; 20 mm</td>
<td>0.30</td>
<td>0.00</td>
<td>0.30</td>
<td>0.50</td>
</tr>
<tr>
<td>b. R in aVL &gt; 7.5 mm</td>
<td>0.34</td>
<td>0.91</td>
<td>0.43</td>
<td>0.58</td>
</tr>
<tr>
<td>c. R in aVL &gt; 11 mm</td>
<td>0.36</td>
<td>0.83</td>
<td>0.40</td>
<td>0.53</td>
</tr>
<tr>
<td>d. R₀ + S₉ &gt; 25 mm</td>
<td>0.31</td>
<td>1.00</td>
<td>0.36</td>
<td>0.53</td>
</tr>
<tr>
<td>e. R₀ + S₉ &gt; 17 mm</td>
<td>0.34</td>
<td>1.00</td>
<td>0.43</td>
<td>0.57</td>
</tr>
<tr>
<td>f. R₀ + S₉ ≥ 10 mm</td>
<td>0.36</td>
<td>0.77</td>
<td>0.54</td>
<td>0.56</td>
</tr>
<tr>
<td>g. Lewis index* ≥ 17 mm</td>
<td>0.34</td>
<td>1.00</td>
<td>0.42</td>
<td>0.57</td>
</tr>
<tr>
<td>Precordial leads</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>h. Deepest S + tallest R ≥ 35 mm</td>
<td>0.41</td>
<td>0.81</td>
<td>0.61</td>
<td>0.62</td>
</tr>
<tr>
<td>i. Deepest S + tallest R ≥ 45 mm</td>
<td>0.34</td>
<td>0.82</td>
<td>0.46</td>
<td>0.29</td>
</tr>
<tr>
<td>j. Deepest S + tallest R ≥ 50 mm</td>
<td>0.33</td>
<td>0.89</td>
<td>0.40</td>
<td>0.54</td>
</tr>
<tr>
<td>k. Deepest S + tallest R ≥ 55 mm</td>
<td>0.53</td>
<td>1.00</td>
<td>0.57</td>
<td>0.53</td>
</tr>
<tr>
<td>l. SV₁ + RV₅ or V₆ ≥ 35 mm</td>
<td>0.37</td>
<td>0.89</td>
<td>0.51</td>
<td>0.55</td>
</tr>
<tr>
<td>m. RV₅ or V₆ &gt; 25 mm</td>
<td>0.31</td>
<td>1.00</td>
<td>0.34</td>
<td>0.52</td>
</tr>
</tbody>
</table>

*Lewis index = (R₀ - S₀) + (S₉ - R₉).

Abbreviations: SENS = sensitivity; SPEC = specificity; ACC = diagnostic accuracy; LVW = left ventricular weight; LVE = left ventricular enlargement.

Definitions: True positive = ECG criteria LVE present; LVE present in specimen. True negative = ECG criteria LVE absent; LVE absent in specimen. False positive = ECG criteria LVE present; LVE absent from specimen. False negative = ECG criteria LVE absent; LVE present in specimen. Sensitivity = true negatives/(true negatives + false negatives). Specificity = true positives/(true positives + false positives). Accuracy = (true positives + true negatives)/total number tested.

Table: Value of Various Electrocardiographic Criteria in the Diagnosis of Left Ventricular Enlargement in the Presence of Left Bundle Branch

(0.76), (specificity 0.86, sensitivity 0.68, diagnostic accuracy 0.71). (2) An initial q of any size in leads I and V₁ and V₆ (specificity 0.80, sensitivity 0.45, diagnostic accuracy 0.47). This criterion when applied to the presence of a septal infarction with extension to the anterior wall demonstrated low specificity (0.40), but improved sensitivity (0.82) and diagnostic accuracy (0.78). (3) The presence of a pathologic Q wave (> 30 msec duration) in lead I (specificity 0.83, sensitivity 0.69, accuracy 0.70) (fig. 2). (4) The presence of a notch in the S wave in leads V₆ or V₅ (specificity 0.68, sensitivity 0.78, accuracy 0.75); the notch could be on the downslope, the nadir or the upslope of the S wave (fig. 1).

Two ECG criteria were specific predictors of inferior wall infarction. First, the presence of pathologic Q waves (> 30 msec duration) in the inferior leads accurately predicted anatomic inferior infarction (specificity 1.00). However, the sensitivity (0.50) and diagnostic accuracy (0.57) of this finding were low. The value of this criterion was not enhanced by applying it only to ECGs that demonstrated LAXD (specificity 1.00, sensitivity 0.46, accuracy 0.55). Further, a pathologic Q wave in lead aVF was again highly specific (1.00), but sensitivity (0.47) and diagnostic accuracy (0.54) were poor. The presence of an initial negative vector (first 30 msec) in the inferior leads was a specific (0.85), but not a sensitive (0.46) or accurate (0.54), indicator of inferior infarction. This criterion was not improved by applying it only to specimens that demonstrated LAXD (specificity 1.00, sensitivity 0.46, accuracy 0.56). Second, ST-segment depression or elevation in lead III was a specific indicator (0.78) of inferior wall infarction, but the sensitivity (0.52) and accuracy (0.60) of this finding were low (fig. 2).

ECG criteria predicting septal infarction had low specificities, while criteria predicting extension of a septal infarction to the anterior wall were quite sensitive. Criteria predicting the presence of an isolated lateral free wall infarction together with criteria comparing QRS and ST magnitudes in leads V₁ and V₅ showed poor sensitivity, specificity and diagnostic accuracy.

Discussion

Pathologic Features of LBBB with and Without LAXD

The combination of LBBB and LAXD has undefined clinical significance. Various investigators have found no clinical or electrophysiologic differences between groups with LBBB and a normal axis and those with LBBB and LAXD. Some have disputed the hypothesis that LAXD coexistent with LBBB is evidence for additional block in the left anterior fascicle. Dhir et al. suggested that at
least a more serious pathologic state is predicted with LBBB and LAXD than with LBBB and a normal axis. These workers reported a large number of patients who had chronic LBBB with and without LAXD in whom they compared their clinical, electrophysiologic and long-term survival characteristics. They concluded that patients with LBBB and LAXD had a greater incidence of clinical myocardial dysfunction, longer mean PR, AH and HV intervals, longer atrial and atioventricular nodal effective refractory periods and a greater cardiovascular mortality. Spurrell et al. noted prolonged His-Q intervals in patients with LBBB and LAXD and suggested that the concurrence of LAXD with LBBB indicated more widespread disease of the conduction system.

Pathologically, the anatomic basis for LBBB has been attributed to both ischemic, degenerative and mechanical factors. Little information is available, however, comparing the pathologic substrate of LBBB and normal axis with that of LBBB and LAXD. We found no significant differences in age distribution, LVW, extent of coronary artery obstruction, or site of MI between patients with LBBB and a normal axis and those with LAXD. LVE was present in approximately 70% of the hearts studied, an observation in keeping with previous reports. However, no significant differences were noted between the LVW of hearts with a normal axis and those with LAXD, which suggests that an increase in LVW did not play a role in the leftward axis shift.

Although it has been suggested that LBBB connotes coronary artery disease (CAD), it may frequently be seen in its absence. When LBBB occurs in the clinical setting of CAD, the degree of coronary obstruction is said to be severe. Beach et al. reported 10 patients with LBBB and no detectable CAD or underlying heart disease. Compared with patients with LBBB and obvious heart disease, patients with LBBB and no other detectable heart disease had lesser degrees of LAXD. Such a clinical distinction has been challenged by Haft et al. who found that LAXD in addition to LBBB did not imply more frequent CAD. The incidence of significant CAD in our series was approximately 50%. Only five of 44 specimens with CAD had significant three-vessel disease, and the presence of LBBB was not associated with a specific coronary lesion or combination of lesions. Further, the distribution of severity of CAD was similar in hearts with a normal axis and those with LAXD.

Our study was designed to permit quantitative assessment of the degree and location of myocardial deficits, and MI was found in over 50% of the hearts dissected. The infarcts were usually large, frequently involved the inferoposterolateral and apical regions, and were more extensive in these regions when associated with LAXD. This latter finding offers an explanation not previously available for left-axis shifts in qualitative pathologic studies. Proposed mechanisms for the genesis of LAXD include delay in left anterior fascicular conduction or loss of inferiorly directed vectorial forces resulting from tissue loss. Although it might be argued that arborizations of the left anterior fascicle would more likely be involved by infarction of the septal summit, this was not demonstrated in our series. Thus, our findings could be interpreted as supporting the latter contention. They do not, however, completely explain the presence of LAXD in hearts with minimal or no inferior infarction.

Morphologically, LBBB is frequently associated with LVE, CAD and MI. The concurrence of LAXD does not imply greater degrees of LVE or coronary artery occlusion, but in the presence of known atherosclerotic heart disease does suggest the likelihood of more extensive inferoposterolateral and apical infarction.

**Electrocardiographic Criteria for LVE in the Presence of LBBB**

The electrocardiographic criteria for LVE in the presence of normal conduction have included either the use of a point-score system or voltage criteria alone. In the presence of LBBB, QRS duration, delayed intrinsictoid deflection and ST-T abnormalities may be obscured by the altered activation sequence. QRS voltage criteria could, therefore, conceivably attain a greater importance in the detection of LVE in the presence of LBBB. Scott et al. evaluated a number of the ECG criteria of LVE in the presence of LBBB and found that they were present in 60% of his autopsy series. However, the majority of these patients had anatomic LVE; thus, Scott et al. considered these criteria unreliable because of the high incidence of false-negative diagnoses. Cokkinos et al. in a study of 79 patients with intermittent LBBB, demonstrated significant correlation of precordial lead voltage criteria for LVE between tracings taken before and after the development of LBBB. Our findings support those of Scott et al.
In our series, voltage criteria of LVE in both the precordial and standard leads had a fairly high specificity, but low sensitivity and diagnostic accuracy. However, the incidence of LVE in association with LBBB is high and the diagnosis of LVE can be made by inference alone with a 69% accuracy. Thus, any test that detects LVE in the presence of LBBB must have not only a specificity higher than the underlying incidence of LVE, but also a high degree of sensitivity to result in a predicted accuracy that is clinically useful. Although a number of the criteria listed in table 3 carry specificities greater than the 69% incidence of definite LVE in our series, and hence improve the diagnostic capabilities of the ECG somewhat, they infrequently predict absence of LVE.

Thus, clinically, LVE is frequently present with LBBB, but is poorly defined by the ECG.

Electrocardiographic Criteria Predicting Infarct Location in the Presence of LBBB

In assessing the various ECG criteria proposed to predict the site of anatomic infarction in the presence of LBBB, our findings generally concurred with the suggestion that the presence of LBBB tends to obscure the ECG representation of myocardial infarction. However, certain ECG findings remain significantly related to the location of infarction in the presence of LBBB. The presence of pathologic Q waves (>30 msec duration) in lead I or q waves of any size in leads I, V₁ and V₅ represents a contradiction of expected activation during LBBB and has been proposed to indicate both anterior and septal infarction. Our findings indicate that with anterior or septal infarction, these signs carry a reasonable diagnostic specificity and sensitivity. Although others have disputed the value of notching of the S wave in leads V₁ or V₅ in detecting anterior wall infarction, we found this to be a fairly reliable sign.

Most ECG findings suggesting the presence of inferior wall infarction were unreliable. The presence of ST-segment changes and pathologic Q waves in the inferior leads were reasonably specific criteria, although their overall sensitivities and diagnostic accuracies were poor. The value of pathologic Q waves in the inferior leads in the presence of LBBB is disputed. Timmis et al. noted the disappearance of inferior Q waves on spontaneous conversion of LBBB to normal conduction, while Abben et al. in a study of 256 patients with intermittent LBBB, found that atypical ECG findings during LBBB, including Q waves in the inferior leads, were of little value in predicting the presence of MI as determined by electrocardiographic criteria. We reported a high degree of specificity for this criterion with no false-positive findings. However, the problem lies in obtaining enough documented instances of both pathologic Q waves in the inferior leads and the presence of LBBB to resolve the discrepancy between these reports. For example, we found only eight such instances, all with inferior scars. However, for these eight instances to be detected, more than 1500 consecutive postmortem examinations had to be performed to detect 70 patients with LBBB, and eight with concomitant inferior infarction criteria.

Such findings may underscore the differences between pathologic and clinical correlative studies. An autopsy series may preselect older patients with more advanced disease, while clinical criteria may underestimate the extent of myocardial scarring. Nevertheless, from an electrocardiographer's point of view, the recognition of subtle changes in the ECG pattern of LBBB will improve diagnostic capabilities to some extent, but should not lessen the value of serial observation or clinical correlation.

References

17. Master AM, Daut S, Jaffe HL: Bundle branch and intraventricular block in acute coronary artery occlusion. Am Heart
PATHOLOGIC CORRELATES OF LBBB/Havelda et al. 451

46. Romhilt DW, Estes EH Jr: A point-score system for the ECG diagnosis of left ventricular hypertrophy. Am Heart J 75:752, 1968
The pathologic correlates of the electrocardiogram: complete left bundle branch block.
C J Havelda, G S Sohi, N C Flowers and L G Horan

_Circulation._ 1982;65:445-451
doi: 10.1161/01.CIR.65.3.445
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1982 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/65/3/445

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org/subscriptions/