Left Bundle Branch Block and Left Ventricular Hypertrophy: Electrocardiographic-Pathologic Correlations*

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A clinical-pathologic study was made of 50 patients with complete left bundle branch block who had postmortem examinations performed at three Salt Lake City hospitals. Forty (80 percent) were found to have left ventricular hypertrophy as determined from measured left ventricular wall thickness but only 14 had electrocardiographic evidence of this while two of the ten patients without left ventricular hypertrophy had positive electrocardiographic criteria. Twenty-six of 40 patients with anatomic left ventricular hypertrophy did not have electrocardiographic evidence of this. Forty-eight of 50 patients (96 percent) had heart weights greater than expected for body weight which probably reflected increased left ventricular mass. It is suggested that while left ventricular hypertrophy is very common in patients with left bundle branch block, its presence cannot be predictably obtained from the electrocardiogram with the criteria used.

Several authors have indicated that the electrocardiographic finding of a left bundle branch block is usually associated with significant underlying heart disease whose manifestations frequently are cardiomegaly and left ventricular hypertrophy or both.1-5 It is usually considered difficult or impossible to make the electrocardiographic (ECG) diagnosis of left ventricular hypertrophy (LVH) in the face of complete left bundle branch block (LBBB) since ventricular excitation is grossly modified when LBBB is present. Yet evidence, primarily clinical in origin, has been presented suggesting that LBBB does not obscure prominent precordial voltage as a criterion for the diagnosis of left ventricular hypertrophy.6 Scott and Norris,2 on the other hand, found all 29 patients of a necropsy series with LBBB had LVH but that only 17 of them (59 percent) had ECG features permitting this diagnosis.

This study is an attempt to determine, from pathologic studies, the incidence of LVH in a large group of patients with LBBB as well as to ascertain the accuracy of conventional ECG voltage criteria of LVH in the presence of LBBB.

METHODS

The data for this study was gathered from the clinical charts, electrocardiographic files, and necropsy reports of patients at the University of Utah affiliated hospitals (Salt Lake County General Hospital, University of Utah Medical Center, and the Salt Lake City Veterans Administration Hospital). Names to be included in the study were taken from diagnosis coded files maintained by each Heart Station which lists deceased patients who had electrocardiograms taken at that hospital. The file at the Salt Lake County General Hospital was maintained from 1956 until July, 1965 when the facilities and records therefrom were transferred to the University of Utah Medical Center. The file at the Veterans Hospital has been maintained since 1965. The clinical charts and electrocardiograms of all patients in these...
LBBB AND LEFT VENTRICULAR HYPERTROPHY

Table 1—Data from 50 Necropsied Patients with LBBB

<table>
<thead>
<tr>
<th>Diagnoses</th>
<th>No. Patients</th>
<th>Aver. Age</th>
<th>No. without Cardiomegaly</th>
<th>Aver. Heart Wt (gms)</th>
<th>No. with LVH</th>
<th>No. with RVH</th>
<th>No. with ECG Evidence of LVH</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASHD</td>
<td>23</td>
<td>76.8</td>
<td>1</td>
<td>442</td>
<td>18*</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Hypertension</td>
<td>3</td>
<td>73.3</td>
<td>0</td>
<td>452</td>
<td>3</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>ASHD + Hypertension</td>
<td>7</td>
<td>78.7</td>
<td>0</td>
<td>443</td>
<td>6</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>ASHD + AVD</td>
<td>3</td>
<td>86.3</td>
<td>1</td>
<td>363</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>AVD</td>
<td>2</td>
<td>61.5</td>
<td>0</td>
<td>633</td>
<td>2</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Hypertension + AVD</td>
<td>2</td>
<td>90.0</td>
<td>0</td>
<td>510</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>ASHD + Hypertension + AVD</td>
<td>3</td>
<td>73.7</td>
<td>0</td>
<td>610</td>
<td>3</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Misc**</td>
<td>7</td>
<td>64.3</td>
<td>0</td>
<td>499</td>
<td>4</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>50</td>
<td>75.7</td>
<td>2</td>
<td>465</td>
<td>40</td>
<td>14</td>
<td>16</td>
</tr>
</tbody>
</table>

*Two of the six patients without LVH were reported as having "dilated left ventricles."

**Miscellaneous causes were: three cases, idiopathic cardiomyopathy; one case, old dissecting aneurysm which had occluded the right coronary ostium; one case, diffuse myocardial calcium oxalate deposition; two cases, no detectable heart disease.

ASHD—Arteriosclerotic heart disease.

AVD—Aortic valvular disease (stenosis and/or calcification).

files with a diagnosis of LBBB were reviewed to determine whether criteria for this diagnosis as defined by the New York Heart Association were present and if an adequate necropsy had been performed. Pathologic data included recorded heart weight, left and right ventricular wall thickness and adequate description of the coronary arteries and heart valves. Of 146 patients whose names were listed in the files as having LBBB, 98 could not be included because of a lack of pathologic information, failure to meet strict criteria for LBBB, or lack of adequate clinical information. Of the 50 patients who were included in the study, there were 27 men and 23 women. The average age was 75.7 years (range 34-102 years). Vectorcardiograms were not available for any of the patients.

A patient was considered hypertensive if review of the clinical chart revealed diastolic blood pressures above 95 mm Hg for longer than 3 days in the hospital or on three clinic visits. Pathologic diagnosis of arteriosclerotic heart disease, valvular disease or other cardiac disease were made from the pathologist's written report on the gross and microscopic findings at necropsy.

A left ventricular thickness of 13 mm or greater was defined as LVH and a right ventricular thickness of 5 mm or greater was defined as right ventricular hypertrophy (RVH). In our study we have used left ventricular thickness as the most definitive criterion for LVH. It was recognized that there might be some instances of left ventricular enlargement due primarily to left ventricular dilatation without having a thickened left ventricular wall as defined above. Even under the most favorable circumstances, the postmortem diagnosis of relatively mild left ventricular dilatation is difficult and subjective. As such, we used cardiomegaly in the absence of isolated RVH, as an additional, albeit more indirect, index of left ventricular enlargement which would include instances of dilatation as well as hypertrophy. Cardiomegaly was defined by the method of Smith as a heart weight which was greater than would be expected on the basis of body weight (heart weighing greater than 0.43 percent of body weight in men or greater than 0.40 percent in women).

An electrocardiographic diagnosis of LVH was made when any one of the following criteria were met: 1) Positive deflection greater than 26 mm in lead V₆ or V₇; 2) SV₁ + RV₅ or RV₆ having a total deflection of at least 36 mm; or 3) R₁ + S₉ having a total deflection of 26 mm or greater.

It is recognized that voltage criteria for LVH, even in the absence of LBBB, are not entirely satisfactory since they are associated with errors of both the false positive and false negative kinds. Their evaluation in this study was prompted by two considerations. First was that other ancillary diagnostic clues to the presence or absence of LVH are obscured by LBBB (eg ST segment and T wave changes, QRS complex duration, delayed intrinsiocid deflection and left axis deviation). Second was a previous report which suggested that precordial voltage criteria could still be used in the face of LBBB.

RESULTS

Table 1 summarizes the findings of this study. Thirty-six of the 50 patients had significant arteriosclerotic heart disease. Twenty-three of these 36 had arteriosclerotic heart disease as the only pathologic diagnosis while the additional 13 patients had concomitant hypertension and aortic valvular disease or both (stenosis and calcification or both). Three of these patients had a combination of arteriosclerotic heart disease, hypertension and aortic valvular disease or both. Three patients had hypertension without arteriosclerotic heart disease while two patients had hypertension with concomitant aortic valvular disease. Only two patients had aortic valvular disease as the only cardiac pathologic finding. The incidence of arteriosclerotic heart disease and hypertension or both was 82 percent.

Of the seven patients listed as miscellaneous in Table 1, three were diagnosed as having an "idiopathic cardiomyopathy" and one patient had an old dissecting aneurysm of the ascending aorta which had caused occlusion of the right coronary ostium. Another 34-year-old patient had the finding of...
calcium oxalate deposition throughout the myocardium in the absence of coronary artery disease. This patient had undergone two renal transplants for chronic renal failure. There were no other pathologic findings to explain the LBBB, although she did have a history of hypertension prior to the kidney transplants. The other two patients had no pathologic findings to explain the LBBB although one of them had received radiation therapy to the mediastinum for bronchogenic carcinoma. The last patient had minimal coronary artery disease with a normal heart weight and a left ventricular thickness of 11 mm.

From Table 1 it can be seen that a total of ten of 50 patients did not have LVH as determined from left ventricular thickness. It would appear that isolated arteriosclerotic heart disease is not uncommonly associated with LBBB in the absence of left ventricular hypertrophy since five of the 23 cases of LBBB with arteriosclerotic heart disease did not have LVH. The presence of LBBB in a hypertensive patient was usually associated with LVH with LVH being found in 14 of the 15 hypertensive patients. Aortic valvular disease also correlated well with LVH, being found in nine of the ten patients in this category. Three other patients who did not have LVH included one with myocardial oxalate deposition, one with idiopathic cardiomyopathy and one patient without discernable significant cardiac abnormalities.

Fourteen patients had RVH in addition to LVH and increased heart weight. No patient in this series had RVH without associated LVH. Forty-eight of 50 patients (96 percent) are noted to have cardiomegaly as determined by heart weight alone (supra vide) and one of the other two had a thickened left ventricular wall with a normal heart weight.

Sixteen of 50 patients (32 percent) had ECG voltage criteria for LVH as shown in Table 2. The pathologic correlation, however, was not good as only 14 of the 40 patients with a thickened left ventricular wall met the ECG criteria for LVH. Two out of ten patients without LVH at necropsy also met the ECG criteria for LVH. In addition, 26 of the 34 patients without electrocardiographic evidence of LVH had LVH at necropsy. Since no patient in this series had RVH without LVH, it is possible to consider increased total heart weight as a possible alternative criterion for anatomic LVH. If this is done, the false positive rate remains the same but the false negative rate increases with all 34 of the patients without ECG evidence of LVH having cardiomegaly at necropsy.

**Discussion**

Our study found very poor correlation between the presence of anatomic LVH and the ECG criteria for LVH in these patients with LBBB regardless of whether left ventricular thickness or cardiomegaly was used as the criterion for LVH. Both false positive and false negative errors occurred but the latter were much more common. This contrasts with the study of Pantridge et al. who suggested that there was a good correlation between high voltage and cardiac enlargement in the presence of LBBB. His study, however, was not based on left ventricular size determined anatomically but rather on cardiac size determined by clinical methods. Scott and Norris have reported that 17 of 29 cases of LBBB and LVH did have electrocardiographic evidence of LVH. However, they included in their study nine cases with Q waves in leads I, aVL, V5 and V6, which would not be considered as LBBB in this study.

The occurrence of ten cases (20 percent) of LBBB without anatomic LVH in this report is higher than would be expected from the study of Scott and Norris who found all of their 29 patients with LBBB to have LVH at necropsy. However, it is possible that, in some of their cases, the diagnosis of LVH was made on the basis of total heart weight alone. If cardiomegaly is used as the criterion of anatomic LVH in our cases the incidence of LVH rises from 80 percent to 96 percent.

Our data, therefore, suggest that the ECG diag-
nosis of LVH in the face of LBBB, with the criteria used, should not be attempted. It was also noted that the mere presence of left bundle branch block in our series carried with it a high correlation with LVH (80 percent to 96 percent depending on whether left ventricular wall thickness or cardiomegaly is used as the criterion).

It should be cautioned that our data probably provide a maximal estimate of the incidence of LVH in LBBB since Johnson and associates\(^\text{11}\) have presented evidence suggesting that cardiomegaly in LBBB adversely affects prognosis. An autopsy series such as ours might be therefore weighted by including an unusually high percentage of patients with cardiomegaly. Thus, the association of anatomic LVH and LBBB may be somewhat less pronounced in living patients than our necropsy series would suggest.

**References**

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Medical students wishing to enter the 1971 Alfred A. Richman Essay Contest of the American College of Chest Physicians must be enrolled in an accredited medical school at the time of submitting their applications.

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