Coronary Arteriographic Appearances in Patients with Left Bundle-Branch Block

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SUMMARY
Left bundle-branch block (LBBB) was found to be associated with an unusually short left main coronary artery in 11 of 12 patients studied by selective coronary arteriography. The average length of the left coronary artery in this group of 12 patients with LBBB was 4.5 ± 1.7 mm as compared to 12.8 ± 0.8 mm in a control group. Four of the 12 had evidence of ischemic heart disease, one had mild aortic regurgitation, one had myocardial infarction, and one had a patent ductus arteriosus. No etiology was discovered for the LBBB in the remaining five patients. Mechanical and hemodynamic explanations for this association of a particular pattern of coronary anatomy with LBBB are proposed.

Additional Indexing Words:
Left coronary artery  Distribution patterns  Exercise ECG

The frequent association of left bundle-branch block (LBBB) with cardiovascular disease, particularly coronary artery disease and hypertensive heart disease, often results in the implication that for all patients with this conduction disturbance the prognosis is poor. However, isolated LBBB in asymptomatic, apparently healthy persons has been shown to be associated with a relatively good prognosis, suggesting that there may be two distinct populations of subjects with this electrocardiographic abnormality. The clinical and laboratory features with special attention to the coronary arteriographic findings in 12 patients with left bundle-branch block are reported here.

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Table 1

Clinical, Laboratory, and Hemodynamic Findings in 12 Patients with Left Bundle-Branch Block

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Clinical history</th>
<th>Physical examination</th>
<th>Serum cholesterol (mg/100 ml)</th>
<th>Chest x-rays</th>
<th>Electrocardiogram</th>
<th>Exercise S-T depression (mm)</th>
<th>LV pressures (mm Hg)</th>
<th>Arteriogram &amp; clinical diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>3. OH</td>
<td>50</td>
<td>M</td>
<td>Effort-related chest pain, not relieved by TNG (1966)</td>
<td>BP 132/90; further unremarkable</td>
<td>290 Normal</td>
<td>Prev: Transient LBBB (1966)</td>
<td>Present: Transient LBBB (1967)</td>
<td>2.5</td>
<td>167/0-3</td>
<td>Class III; atypical angina</td>
</tr>
<tr>
<td>5. LH</td>
<td>40</td>
<td>M</td>
<td>Rheumatic heart disease; murmur (1946); chest pain (1960)</td>
<td>BP 150/80; early diastolic murmur, grade II/VI, at aortic area</td>
<td>220 Normal</td>
<td>None</td>
<td>Prev: LBBB (1965)</td>
<td>1.0</td>
<td>103/3-5</td>
<td>Class 0; rheumatic heart; typical angina</td>
</tr>
<tr>
<td>6. LF</td>
<td>42</td>
<td>F</td>
<td>Chest pain for 1 yr after exercise and meals; eclampsia at age 24 yr</td>
<td>BP 130/85; further unremarkable</td>
<td>225 Normal</td>
<td>Prev: Transient LBBB (1955); fixed LBBB (1962)</td>
<td>Present: LBBB (1963)</td>
<td>0.5</td>
<td>117/0-6</td>
<td>Class 0; pain of uncertain origin</td>
</tr>
<tr>
<td>7. EL</td>
<td>44</td>
<td>F</td>
<td>Asthma; myocardiitis (1961); vague chest pain</td>
<td>BP 115/80; S4 gallop; ejection systolic murmur, grade II/VI; at LLSB</td>
<td>285 Normal</td>
<td>Prev: Fixed LBBB (1961)</td>
<td>Present: LBBB (1963)</td>
<td>2.0 (digitalis)</td>
<td>103/0-16</td>
<td>Class 0; myocardiopathy; not angina</td>
</tr>
<tr>
<td>8. BG</td>
<td>51</td>
<td>M</td>
<td>Precordial pain 1 yr; relieved by TNG</td>
<td>BP 140/80; ejection systolic murmur, grade II/VI, at LLSB</td>
<td>185 Normal</td>
<td>Prev: Fixed LBBB (1957)</td>
<td>Present: LBBB (1963)</td>
<td>1.8</td>
<td>128/0-5</td>
<td>Class 0; pain of uncertain origin</td>
</tr>
<tr>
<td>9. RS</td>
<td>43</td>
<td>F</td>
<td>Precordial pain unrelated to exertion (1964)</td>
<td>BP 118/80; further unremarkable</td>
<td>260 Normal</td>
<td>Prev: Transient LBBB (1964)</td>
<td>Present: Transient LBBB (1967)</td>
<td>0</td>
<td>164/0-7</td>
<td>Class 0; pain of uncertain origin</td>
</tr>
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<td></td>
</tr>
<tr>
<td>10.</td>
<td>GT</td>
<td>51</td>
<td>M</td>
<td>Repair of hiatus hernia (1966)</td>
<td>BP 140/90; further unremarkable</td>
<td>188</td>
<td>Normal (1960) LBBB (1962)</td>
<td>1.5</td>
<td>104/3-9</td>
<td>Class I; non-ischemic disease</td>
</tr>
<tr>
<td>11.</td>
<td>CF</td>
<td>41</td>
<td>M</td>
<td>Entirely asymptomatic</td>
<td>BP 120/80</td>
<td>305</td>
<td>Normal LBBB (1968)</td>
<td>1.0</td>
<td>114/0-11</td>
<td>Class 0</td>
</tr>
<tr>
<td>12.</td>
<td>BS</td>
<td>27</td>
<td>F</td>
<td>Chest pain of uncertain etiology</td>
<td>BP 120/70; continuous murmur, grade II/V1, under left clavicle</td>
<td>Normal</td>
<td>None</td>
<td>Transient LBBB (1969)</td>
<td>1.0</td>
<td>100/0-11</td>
</tr>
</tbody>
</table>

Abbreviations: TNG = nitroglycerin; LL SB = left lower sternal border; LBBB = left bundle-branch block.

**Results**

The clinical, laboratory, and hemodynamic findings in 12 patients with LBBB are summarized in Table 1. Nine patients experienced chest pain. In four of these, the symptoms of LBBB could not be explained by coronary atherosclerosis. A control arteriographic series was provided by the random sampling of 25 coronary arteriograms from the 324 arteriograms analyzed for review. These arteriograms were compared with those obtained in patients with LBBB and also with the results of angiographic studies reported in the literature.

**Coronary Arteriographic Appearance in LBBB**

class 0 designating the absence of detectable coronary arteriosclerosis. The arteriographic findings are summarized in Table 2. The right anterior oblique projection was utilized for the measurements of the LCA length. Each patient, except for the patient with LCA 18 mm (Table 2), had a small patent ductus arteriosus. The right anteroposterior view of the left anterior descending coronary artery (LCA) length was employed in each patient. The LCA length in the four patients with LBBB was measured independently by two observers. The mean value of all determinations for the LCA length, measurement.

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S-T depression ranged from 0 to 1.8 mm except in one patient (case 7) with myocardialopathy, who was receiving digitalis at the time of the exercise study and had 2.0 mm S-T depression.

The cinecoronary arteriographic findings in these patients are outlined in table 2 and are illustrated by the examples in figures 2 to 4. The incidence of a left dominant arterial distribution pattern (four of 12 cases of LBBB) is higher than in the total arteriographic experience in the remaining 354 cases (9.3%). In two patients with LBBB the two major divisions of the left coronary artery, the anterior descending and posterior circumflex trunks, appeared to take origin from separate ostia in the left aortic sinus. These two cases represent the sole occurrence of this anomaly in our arteriographic experience.

The prebifurcation length of the main LCA in patients with LBBB was found to vary between 0 mm (double ostia) and 22.3 mm, with a mean and standard error of the mean (SEM) of 4.5 ± 1.7 mm. This was comparable to a mean value of 12.8 ± SEM 0.8 mm in 25 patients selected at random from the series of 354 arteriograms. The difference between the two mean values was at the 0.001 level of significance, and the distribution of the two sets of values is strikingly dissimilar (fig. 5). If a single patient (case 3) with an exceptionally long left coronary artery (22.3 mm)
Table 2

Coronary Arteriography in 12 Patients with Left Bundle-Branch Block

<table>
<thead>
<tr>
<th>Patient</th>
<th>Mainstem LCA length (mm)</th>
<th>Distribution pattern</th>
<th>Sinus node artery</th>
<th>A-V node artery</th>
<th>Anterior perforating</th>
<th>Posterior perforating</th>
<th>Atherosclerotic change</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. WS</td>
<td>1.8</td>
<td>Left</td>
<td>LPC</td>
<td>LPC</td>
<td>–</td>
<td>?</td>
<td>Class IV; LAD obstructed</td>
<td>Collaterals: RCA to LAD; LCA to LAD</td>
</tr>
<tr>
<td>2. LT</td>
<td>4.0</td>
<td>Left</td>
<td>RCA</td>
<td>–</td>
<td>+</td>
<td>?</td>
<td>Class III; triple artery narrowings</td>
<td>RCA small: early bifurcation</td>
</tr>
<tr>
<td>3. OH</td>
<td>22.3</td>
<td>Right</td>
<td>RCA</td>
<td>LPC</td>
<td>?</td>
<td>?</td>
<td>Class III; triple artery narrowings</td>
<td>Trifurcation, tortuosity of LAD; collateral: sinus node artery to septum</td>
</tr>
<tr>
<td>4. TC</td>
<td>6.0</td>
<td>Right</td>
<td>LPC</td>
<td>RCA</td>
<td>+</td>
<td>+</td>
<td>Class II; RCA</td>
<td></td>
</tr>
<tr>
<td>5. LH</td>
<td>2.4</td>
<td>Left</td>
<td>RCA &amp; LPC</td>
<td>LPC</td>
<td>+</td>
<td>+</td>
<td>Class 0</td>
<td>Moderate aortic regurgitation</td>
</tr>
<tr>
<td>6. LF</td>
<td>2.4</td>
<td>Left</td>
<td>?</td>
<td>RCA</td>
<td>?</td>
<td>?</td>
<td>Class 0</td>
<td>Relatively small arteries; tortuosity of LAD</td>
</tr>
<tr>
<td>7. EL</td>
<td>0</td>
<td>Right</td>
<td>RCA</td>
<td>RCA</td>
<td>+</td>
<td>+</td>
<td>Class 0</td>
<td>Normal coronary arteriogram</td>
</tr>
<tr>
<td>8. BG</td>
<td>0</td>
<td>Right</td>
<td>LPC</td>
<td>RCA</td>
<td>+</td>
<td>?</td>
<td>Class 0</td>
<td>LAD trifurcation; tortuosity of LAD, LCA</td>
</tr>
<tr>
<td>9. RS</td>
<td>4.7</td>
<td>Balanced</td>
<td>RCA</td>
<td>RCA</td>
<td>+</td>
<td>+</td>
<td>Class 0</td>
<td>Marked tortuosity of all systems</td>
</tr>
<tr>
<td>10. GT</td>
<td>4.6</td>
<td>Right</td>
<td>RCA</td>
<td>RCA</td>
<td>+</td>
<td>+</td>
<td>Class I</td>
<td>LAD slightly narrowed</td>
</tr>
<tr>
<td>11. CF</td>
<td>1.0</td>
<td>Balanced</td>
<td>RCA</td>
<td>LPC</td>
<td>+</td>
<td>+</td>
<td>Class 0</td>
<td>Normal coronary arteriogram</td>
</tr>
<tr>
<td>12. BS</td>
<td>4.5</td>
<td>Right</td>
<td>RCA</td>
<td>RCA</td>
<td>+</td>
<td>–</td>
<td>Class 0</td>
<td>Normal coronary arteriogram; patent ductus arteriosus</td>
</tr>
</tbody>
</table>

Abbreviations: LCA = left coronary artery; RCA = right coronary artery; LAD = left anterior descending artery; LPC = Left posterior circumflex artery.
is excluded from the series of patients with LBBB, the distance from left coronary ostium to bifurcation in the remaining 11 patients does not exceed 6.0 mm, and the mean value is $2.9 \pm 0.6$ mm. Even if the two instances of double ostia are considered to represent
special cases and are excluded from these mean calculations, a value of 5.4 mm ± SEM 1.9 is obtained for these 10 patients, which is still far lower than that in the randomly selected control series.

Reference to figure 5 and table 2 will show that within the group of patients with LBBB, arterial distribution pattern and LCA length do not provide a basis for differentiation of patients with coronary atherosclerosis from those without evidence of coronary disease. It is of interest, however, that the only patient in the series with a main LCA longer than 6.0 mm (case 3) was included in the group with significant coronary atherosclerosis. This patient, it should be noted, was also the one in whom the A-V nodal artery had a left-sided origin despite a dominant right coronary distribution pattern. Anterior septal branches of the left anterior descending system were not identified in this patient, and the interventricular septum appeared to be perfused via collateral flow from the artery to the sinus node.

Discussion

In 76 to 86% of documented cases LBBB has been shown to be clinically associated with coronary artery disease and hypertensive heart disease. The conduction disturbance is also encountered in patients with rheumatic heart disease or myocardial disease, particularly when signs of left ventricular hypertrophy or dilatation are present and these factors may have been important in two of our patients (cases 5 and 7). According to Rossi, the histologic changes in the conduction system are not sufficient to explain the bundle-branch block, so that the associated disease condition, whether coronary atherosclerosis, hypertensive cardiovascular disease, or myocardial disease, must be considered important in the pathogenesis of the conduction defect. Where advanced atherosclerosis is held responsible, the lesion would presumably have to be an extensive one, causing interruption of the nutrient supply to all ramifications of the left bundle branch. It is also possible to speculate on the probable etiologic role of focal fibrosis and congenital abnormality of the bundle itself or metabolic alterations in the conduction tissue. According to Bauer, the etiologic factors are the same in both transient and fixed forms of LBBB. In our series, patients with the transient form of block were equally divided between those with significant coronary atherosclerosis and those without. The present report and others emphasized that LBBB may be present in the absence of coronary atherosclerotic changes or hypertensive heart disease.

Angiographic study of the coronary distribution in this series of patients with LBBB is not helpful in differentiating etiologically explained from isolated cases of block. It was, nevertheless, of value in distinguishing cases of LBBB from the random group of 25 cases selected from 354 patients studied arteriographically and control data recorded in the literature. Analysis of arterial distribution patterns in the 354 arteriograms revealed right preponderance in 69.5%, left dominance in 9.3%, and a balanced coronary circulation in 14.4%, with an indeterminate distribution in 6.8%. These percentages reflect close agreement with the findings of Baroldi and Scomazzoni in pathologic studies on 522 human hearts and those of Fulton, but differ sharply from the findings in our 12 patients with LBBB, in whom the incidence of left coronary preponderance is much greater. Likely explanations of this finding are lacking, but it might be speculated that predominant right coronary perfusion of the posterior third of the septum and the A-V node confers relative immunity from ischemia, the right coronary system being a less frequent site of obliterative disease or hemodynamic overloading. A dual vascular supply of the crux of the heart from both right coronary and left circumflex systems in a balanced circulation might similarly be surmised to preserve viability of this region. It is recognized, however, that other than nutritive factors may be active in the pathogenesis of delayed intraventricular conduction and that an arborization block associated with diffuse myocardial involvement rather than localized
myocardial ischemia may account for the electrocardiographic appearance in LBBB.

The prebifurcation length of the mainstem LCA in 25 patients selected at random from the series of 354 arteriograms ranged from 7.5 to 20.5 mm (mean, 12.8 mm) and is similar to those reported from a pathologic study by Baroldi and Scomazzoni23 of a range of 3 to 23 mm (mean, 13.5 mm). The highly significant difference observed in the length of the main LCA in our patients with LBBB (mean, 4.5 mm) represents a finding not previously documented. A double ostial origin of the left coronary artery has been infrequently noted to date16, 23 and was observed in two of our patients with LBBB. It might be postulated that an unusually short (less than 6 mm) or absent main LCA in some way predisposes to the development of LBBB. It is conceivable that early septal branches of the left anterior descending coronary artery, normally protected from shearing forces by the relative “binding down” of the parent vessel, are now subjected through systolic kinking of the anterior descending artery to unusual stress. Presumably the slack normally provided by the main LCA trunk minimizes disruption of the coronary flow to the endocardial surface of the left ventricle near the summit of the muscular interventricular septum, where the left bundle branch originates. The finding of a short main LCA in two of our patients with LBBB who also had severe coronary atherosclerosis gives rise to the suggestion that this morphologic anomaly may play a predisposing role in the pathogenesis of LBBB. Detailed microscopic analysis of the main left bundle branch and its sources of perfusion would, however, be necessary to substantiate this hypothesis. On the other hand, it is possible that this morphologic pattern merely reflects an associated congenital anomaly of the conduction tissue or an accessory pathway analogous to the bundle of Kent and as such could be regarded as a genetic marker.

Beach et al.21 suggested that electrocardiographic findings are helpful in differentiating cases of atherosclerotic LBBB from those without evidence of ischemic heart disease. These authors reported 10 patients with LBBB who had normal coronary arteriograms and suggested that a normal frontal plane mean QRS axis in LBBB may indicate that the bundle-branch block is not due to coronary artery disease.24 The findings from the present study and those of Haft et al.22 failed to confirm this etiologic differentiation based on the frontal axis.

The electrocardiographic exercise test is generally considered to be uninterpretable in patients with LBBB. However, in the current study the degree of S-T segment depression encountered in the patients with benign LBBB was less than in those with ischemic LBBB. These results and those reported previously21, 25, 26 suggest that useful information may be obtained from the ECG exercise tests in the presence of LBBB, but a definitive conclusion cannot be supported by the small number of patients evaluated.

Follow-up clinical evaluation of these 12 patients with isolated LBBB ranging from ½ to 14 years (mean, 6.9 years) revealed no deaths in the series at the time of writing. The patients with ischemic heart disease continued to experience symptoms of coronary insufficiency, while those with unexplained or isolated LBBB have developed no evidence of ischemic heart disease. The prognosis for patients who have LBBB without any demonstrable associated cardiovascular disease is almost certainly better than that for patients with heart disease. Epidemiologic studies2, 8, 7, 8 support this contention, and the continued asymptomatic status of all our patients in the former category lends further corroboration.

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