Coronary Arteriographic Appearances in Patients with Left Bundle-Branch Block

By C. Michael Lewis, Ph.D. (Med.), Gilles R. Dagenais, M.D., Gottlieb C. Friesinger, M.D., and Richard S. Ross, M.D.

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.

From the Department of Medicine, The Johns Hopkins University School of Medicine and Hospital, Baltimore, Maryland.
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Methods
Eight men and four women (table 1) with electrocardiographic findings of LBBB were chosen from a group of 366 patients who had been subjected to coronary arteriographic study. Patients were selected for coronary arteriography because of overt or suspected ischemic heart disease, chest pain of uncertain etiology, or an unexplained electrocardiographic abnormality such as LBBB. The finding of LBBB which was apparently clearly attributable to ischemic or hypertensive heart disease did not in itself constitute an indication for arteriography. The diagnosis of LBBB was based upon the strict criteria set forth by the New York Association. Five patients had exhibited transient LBBB over a variable period prior to the arteriographic study, and in one of these normal intraventricular conduction prevailed at the time of the study. All 12 patients with LBBB included in this study had performed a graded electrocardiographic effort test, a multiple-lead recording system being employed for this purpose.

The coronary arteriograms had been performed by the selective technic of Sones and Shirey or Judkins and were evaluated as follows: The arterial distribution pattern was determined, employing Schlesinger's criteria of "dominance" with respect to the arterial supply to the A-V node and the posterior intraventricular surface of the heart. Atherosclerotic change was graded according to a previously described scheme.
## 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>LV pressures (mm Hg)</th>
<th>Arteriogram &amp; clinical diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atherosclerotic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. WS</td>
<td>55</td>
<td>M</td>
<td>Chest pain of uncertain etiology (1955); probable angina (1958)</td>
<td>BP 136/90; ejection systolic murmur, grade II/VI, at LLSB</td>
<td>370</td>
<td>Normal</td>
<td>Fixed LBBB (1955)</td>
<td>LBBB (1966)</td>
<td>2.0</td>
</tr>
<tr>
<td>2. LT</td>
<td>37</td>
<td>M</td>
<td>Typical angina pectoris (1963)</td>
<td>BP 130/80; further unremarkable</td>
<td>230</td>
<td>Normal</td>
<td>Transient LBBB (1963)</td>
<td>Transient LBBB (1964)</td>
<td>3.0</td>
</tr>
<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</td>
<td>Normal</td>
<td>Transient LBBB (1966)</td>
<td>Transient LBBB (1967)</td>
<td>2.5</td>
</tr>
<tr>
<td>4. TC</td>
<td>41</td>
<td>M</td>
<td>Chest pain of uncertain etiology</td>
<td>BP 130/88; further unremarkable</td>
<td>240</td>
<td>Normal</td>
<td>LBBB (1966)</td>
<td>LBBB (1969)</td>
<td>3.0</td>
</tr>
<tr>
<td>Nonatherosclerotic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></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</td>
<td>Normal</td>
<td>None</td>
<td>LBBB (1965)</td>
<td>1.0</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</td>
<td>Normal</td>
<td>Transient LBBB (1955); fixed LBBB (1962)</td>
<td>LBBB (1963)</td>
<td>0.5</td>
</tr>
<tr>
<td>7. EL</td>
<td>44</td>
<td>F</td>
<td>Asthma; myocarditis (1961); vague chest pain</td>
<td>BP 115/80; S4 gallop; ejection systolic murmur, grade II/VI; at LLSB</td>
<td>285</td>
<td>Normal</td>
<td>Fixed LBBB (1961)</td>
<td>LBBB (1963)</td>
<td>2.0 (digitalis)</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</td>
<td>Normal</td>
<td>Fixed LBBB (1957)</td>
<td>LBBB (1963)</td>
<td>1.8</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</td>
<td>Normal</td>
<td>Transient LBBB (1964)</td>
<td>Transient LBBB (1967)</td>
<td>0</td>
</tr>
</tbody>
</table>
CORONARY ARTERIOGRAPHIC APPEARANCES IN LBBB

<table>
<thead>
<tr>
<th>Class</th>
<th>Diagnosis</th>
<th>Arteriogram Type</th>
<th>Diameter (mm)</th>
<th>Length (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.5</td>
<td>Normal ischemic disease</td>
<td>LBBB (1968)</td>
<td>3.05</td>
<td>104/3-9</td>
</tr>
<tr>
<td>1.0</td>
<td>Normal nonischemic disease</td>
<td>LBBB (1962)</td>
<td>Normal</td>
<td>114/0-11</td>
</tr>
<tr>
<td>1.0</td>
<td>Transient ductus arteriosus</td>
<td>LBBB (1969)</td>
<td>Normal</td>
<td>100/0-11</td>
</tr>
</tbody>
</table>

Abbreviations: TNG = nitroglycerin; LLB = left bundle-branch block.

Class 0 designating the absence of detectable coronary atherosclerosis. The arteriographic findings are summarized in table 2.

The right anterior oblique projection was utilized for the measurements of the left coronary artery (LCA) length. In each patient, diastolic frames from several heart cycles were examined. This avoided the foreshortening effect of ventricular systole on the LCA length. The frames selected for measurements were those in which the catheter tip and ostium of the LCA appeared to be in the same plane. By selecting frames in this manner, the known diameter of the catheter tip (1.8 mm for a 5.5 F catheter) could be used as a reference for the LCA length measurement. All measurements of LCA length were made independently by two observers and data shown (table 2) represent the mean value of all determinations in each patient.

A control arteriographic series was provided by the random sampling of 25 coronary arteriograms from the 354 available for review. These arteriograms were analyzed in similar fashion with respect to arterial distribution pattern and prebifurcation length of the LCA. The findings were compared with those obtained in patients with LBBB and also with the results of anatomic studies reported in the literature.

**Results**

The clinical, laboratory, and hemodynamic findings in 12 patients with LBBB are summarized in table 1. Nine patients complained of chest pain. In four of these, moderate to severe coronary atherosclerosis was visualized at arteriography; rheumatic aortic regurgitation was present in case 5, myocardopathy in case 7, and a small patent ductus arteriosus in case 12, but in none was there clinical or hemodynamic evidence of a severe impairment of cardiac function. The remaining five patients had no abnormality to which the symptoms of LBBB could be attributed. In all patients, the electrocardiogram revealed a frontal axis within normal limits. The exercise electrocardiogram in 12 patients showed downward sloping S-T segment depression varying from 0 to 3.0 mm below the resting base-line level similar to the representative records shown in figure 1. In the four patients with established coronary atherosclerosis the level of S-T segment depression was 2.0 mm or more and in the eight without evidence of atherosclerosis the
Exercise electrocardiographic tests on two patients with LBBB. Negative arteriogram in case 6; class III arteriographic changes in case 3. Leads V₃-V₆ are simultaneous both at rest and during exercise. The S-T segments were abnormal at rest in both patients and further depression developed during exercise. The changes in case 3, the patient with atherosclerotic disease, were of greater magnitude than those in case 6, the patient with a normal coronary arteriogram.

S-T depression ranged from 0 to 1.8 mm except in one patient (case 7) with myocardial infarction, 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 main 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; LPC 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, LPC</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.
Figure 2
Case 11. Left coronary artery visualized in the right anterior oblique projection. The mainstem LCA measures 1 mm before its bifurcation. There is no significant irregularity of the left system.

Figure 3
Case 10. Left coronary artery visualized in the left anterior oblique projection. In this projection, the LCA length cannot be evaluated because the origin of the LPC is not well defined. Note the minimal irregularities in the midportion of the longer branch of the LAD.

Figure 4
Case 10. Same patient as in figure 3. The left coronary artery is visualized in the right anterior oblique projection. In this projection, the origin of the LPC is well defined. The length of the LCA in this frame is 4.6 mm.

Figure 5
Distribution of patients according to arteriographic measurements of the length of the left coronary artery. (A) Twelve patients with LBBB. (B) Twenty-five patients selected at random from the series. Shaded bars indicate the presence of coronary atherosclerotic disease (CAD).

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 96% 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 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 pre bifurcation 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.21 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 ¾ 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.

References

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C. MICHAEL LEWIS, GILLES R. DAGENAIMS, GOTTLIEB C. FRIESINGER and RICHARD S. ROSS

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