Left Bundle Branch Block

Etiologic, Hemodynamic, and Ventriculographic Considerations

By JACOB I. HAFT, M.D., MICHAEL V. HERMAN, M.D., AND RICHARD GORLIN, M.D.

SUMMARY

Coronary angiography, cine left ventriculography, and the cardiovascular hemodynamics of 24 patients with left bundle branch block were studied. Seven patients were found to have significant coronary artery disease, always severe. Five had cardiomyopathy and seven had valvular disease. Only those with coronary artery disease or cardiomyopathy had abnormal left ventricular contraction patterns. The onset of left ventricular contraction was delayed in the patients with left bundle branch block, but the period of isovolumic contraction was normal in those without severe underlying disease. Left axis deviation in addition to left bundle branch block did not imply more frequent coronary atherosclerosis.

Additional Indexing Words:
Coronary angiography Left ventriculography Electromechanical delay

ALTHOUGH the ECG diagnosis of left bundle branch block (LBBB) has been made for many years, the clinical implications and the physiologic effects of the conduction abnormality have not been clarified. Previous clinical and postmortem studies have suggested that the pattern is seen most often in patients with arteriosclerotic heart disease and hypertension.1-2 Few complete studies with coronary angiography, left ventriculography, and hemodynamics3-6 in living patients with LBBB have been reported. The effects of the conduction effect on the timing of ventricular events have been investigated in man4,5,7-9 without agreement as to whether the onset of left ventricular contraction is delayed.

It is the purpose of this paper to present our findings in 24 patients with LBBB who underwent cardiac catheterization with coronary angiography and cine left ventriculography.

Materials and Methods

The electrocardiograms of 578 patients who had undergone diagnostic left heart catheterization over the past 6 years were reviewed. Twenty-four patients with complete LBBB were selected for analysis according to the criteria of the New York Heart Association10 (QRS > 0.12 sec in the standard leads, notching of the QRS with late peaking of R or R', absence of Q in left-sided leads, and ST-T waves displaced opposite to main QRS deflection).

All patients were studied in the postabsorptive state, mildly sedated with 50 mg meperidine (Demerol) and 100 mg pentobarbital sodium (Nembutal). Left heart catheterization was done retrograde via cutdown on the right brachial
Table 1

Clinical Data for the 24 Patients Studied

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Chest pain</th>
<th>Congestive heart failure</th>
<th>Left ventricular end-diastolic pressure (mm Hg)</th>
<th>Coronary arteries</th>
<th>Left ventriculogram</th>
<th>Diagnosis</th>
<th>ECG axis</th>
</tr>
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<tbody>
<tr>
<td>MN</td>
<td>56</td>
<td>F</td>
<td></td>
<td>X</td>
<td>23</td>
<td>3 major vessels diseased</td>
<td>Normal</td>
<td>Coronary artery disease</td>
<td>Left axis deviation</td>
</tr>
<tr>
<td>MNa</td>
<td>47</td>
<td>F</td>
<td></td>
<td>X</td>
<td>9</td>
<td>Normal; mitral regurgitation</td>
<td>Rheumatic heart disease; aortic stenosis; mitral stenosis; mitral regurgitation</td>
<td>Left axis deviation</td>
<td></td>
</tr>
<tr>
<td>WA</td>
<td>35</td>
<td>M</td>
<td>X</td>
<td>X</td>
<td>36</td>
<td>3 major vessels diseased</td>
<td>Anterior lateral; akinesia; mitral regurgitation</td>
<td>Coronary artery disease; papillary muscle dysfunction; hypertension</td>
<td>Left axis deviation</td>
</tr>
<tr>
<td>CS</td>
<td>35</td>
<td>F</td>
<td>X</td>
<td>X</td>
<td>28</td>
<td>Normal</td>
<td>Cardiomyopathy; hyperthyroidism</td>
<td>Left axis deviation</td>
<td></td>
</tr>
<tr>
<td>AM</td>
<td>56</td>
<td>F</td>
<td></td>
<td>X</td>
<td>7</td>
<td>Normal</td>
<td>Calcific aortic stenosis; hypertension</td>
<td>Left axis deviation</td>
<td></td>
</tr>
<tr>
<td>BF</td>
<td>54</td>
<td>F</td>
<td>X</td>
<td>X</td>
<td>28</td>
<td>Normal</td>
<td>Anterior inferior; akinesia</td>
<td>Alcoholic cardiomyopathy</td>
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<tr>
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<td>49</td>
<td>F</td>
<td>X</td>
<td>X</td>
<td>8</td>
<td>Normal</td>
<td>None</td>
<td>Rheumatic heart disease; aortic stenosis; aortic insufficiency; mitral regurgitation; hypertension</td>
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</tr>
<tr>
<td>AD</td>
<td>45</td>
<td>M</td>
<td>X</td>
<td>X</td>
<td>12</td>
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<tr>
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<td>F</td>
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<td>–</td>
<td>10</td>
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<td>Idiopathic dilatation of pulmonary artery</td>
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<td>X</td>
<td>–</td>
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<td>Normal</td>
<td>Normal</td>
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<td>Normal</td>
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<tr>
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<td>43</td>
<td>M</td>
<td>X</td>
<td>–</td>
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<td>X</td>
<td>–</td>
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<td>X</td>
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<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>AA</td>
<td>59</td>
<td>F</td>
<td>X</td>
<td>X</td>
<td>21</td>
<td>Normal</td>
<td>Normal</td>
<td>Generalized hypokinesis; inferior akinesis</td>
<td>Cardiomyopathy</td>
</tr>
<tr>
<td>DB</td>
<td>62</td>
<td>M</td>
<td>–</td>
<td>X</td>
<td>8</td>
<td>Normal</td>
<td>Generalized hypokinesis; inferior akinesis</td>
<td>Alcoholic cardiomyopathy</td>
<td>Normal</td>
</tr>
<tr>
<td>SF</td>
<td>56</td>
<td>M</td>
<td>X</td>
<td>–</td>
<td>9</td>
<td>3 major vessels diseased</td>
<td>None</td>
<td>Coronary artery disease</td>
<td>Normal</td>
</tr>
<tr>
<td>SK</td>
<td>59</td>
<td>M</td>
<td>X</td>
<td>X</td>
<td>24</td>
<td>3 major vessels diseased</td>
<td>Apical; inferior anterior dyskinesis</td>
<td>Coronary artery disease</td>
<td>Normal</td>
</tr>
<tr>
<td>FO</td>
<td>65</td>
<td>M</td>
<td>–</td>
<td>X</td>
<td>12</td>
<td>Normal</td>
<td>Normal; mitral regurgitation</td>
<td>Rheumatic heart disease; mitral stenosis; mitral regurgitation; aortic insufficiency</td>
<td>Left axis deviation</td>
</tr>
</tbody>
</table>
artery with a no. 8 Sones catheter. Brachial artery pressure was continuously monitored by PE 160 tubing introduced into the left brachial artery by the Seldinger technique. Measurements of the time intervals were made from simultaneous tracings of the ECG, and the left ventricular and brachial artery pressures recorded at a paper speed of 50 or 100 mm/sec. The intervals were measured in 10 consecutive beats and the results averaged. The onset of ventricular contraction was considered to be at the point in the pressure curve at which the pressure began a rapid ascent. The isovolumic contraction period was considered to begin with the onset of contraction and to end with the opening of the aortic valve. This point was determined by extrapolation back from the pressure at the onset of the rise in the brachial artery pressure. All hemodynamic measurements were made prior to coronary or left ventricular angiography. Selective coronary cine arteriography was performed by the technique of Sones and Shirey with selective injection of 75% sodium and meglumine diatrizoate into the right and left coronary arteries in at least two projections. A lesion in a coronary artery was considered significant if there was greater than 75% stenosis of the vessel lumen. In 21 patients, uniplanar cine left ventriculography was performed at rest in the right anterior oblique projection (30°) after the power injection of 45–60 ml of 75% sodium and meglumine diatrizoate into the left ventricle. The left ventriculograms were analyzed for the symmetry and synergy of the contraction pattern as previously reported, excluding all ectopic beats from study. No volume calculations were made from this uniplanar study.

**Results**

**Patient Population (Table 1)**

Thirteen of the patients were men and 11 were women. Their ages ranged from 26 to 68 years, with a mean age of 48.1 years in men and 48.3 in women. Nineteen complained of chest pain and 12 had symptoms of congestive heart failure. Seven patients had significant valvular disease with involvement of both aortic and mitral valves in three cases; five of these had histories of acute rheumatic fever. Six patients had a history of hypertension. Five patients had histories suggestive of cardiomyopathy (diagnosed on the basis of cardiomegaly, recurrent episodes of clinical failure in the absence of valvular, atherosclerotic, or hypertensive heart disease). Two had
a long history of excessive alcoholic intake, one had hyperthyroidism, and two others myocardial disease of unknown etiology. Seven had been hospitalized for presumed myocardial infarction. (Only three of these, however, were found to have abnormal coronary vessels by arteriography.) Twenty patients were in normal sinus rhythm, three in atrial fibrillation, and one in atrial flutter at the time of study. One patient had intermittent LBBB and demonstrated LBBB when studied.

**Electrocardiograms**

Two patterns of LBBB have been described, depending upon the frontal plane axis. Fourteen patients had a normal frontal plane axis, and 10 patients had marked left axis deviation (more negative than $-30^\circ$) (fig. 1). Although there was a higher incidence of patients with significant coronary atherosclerosis among the group with a normal axis (five of 14) than among those with left axis deviation (two of 10), this difference was not significant. No differences between the groups, other than axis, were apparent.

**Coronary Arteriography**

Seventeen of the 24 patients (71%) had normal coronary arteries. Seven had significantly diseased coronary arteries. Six of these had involvement of all three main vessels (the left anterior descending, the circumflex, and the right coronary arteries). One patient had disease only of the left anterior descending and circumflex arteries.

**Hemodynamics**

Five patients had valvular gradients indicating mitral stenosis of less than 0.7 cm$^2$ area in three and aortic stenosis of less than 0.5 cm$^2$ area in four. Six had normal hemodynamics, both at rest and on exercise. Twelve had elevated left ventricular end-diastolic pressures either at rest or on exercise; six with coronary disease, three with valvular disease, and three with cardiomyopathy. Two patients

![Diagram of Electrocardiograms]

**Figure 1**

Patterns of left bundle branch block: normal frontal plane mean electrical axis and left axis deviation.
with primary myocardial disease had normal left ventricular end-diastolic pressures on catheterization.

**Left Ventriculography**

Seven of the 21 patients with satisfactory ventriculograms had abnormal contraction patterns. Four of these seven had severe coronary artery disease. Among this group, all four had anterior or apical dyskinesis (large areas of paradoxical motion) in addition to areas of akinesis (no motion during contraction). The three patients with normal coronary arteries and abnormal contraction patterns on ventriculography all had severe cardiomyopathy with left ventricular failure. These patients had generalized hypokinesis with areas of akinesis anteriorly or inferiorly. The ventriculograms of the remaining 14 patients with LBBB on ECG were normal, with no differences from the patterns of contraction of patients with normal conduction. Three patients with normal contraction had mitral regurgitation.

**Timing of Left Ventricular Events (Table 2)**

In 16 patients who had suitable high speed recordings of the left ventricular pressure pulse, measurement of the time period from the onset of the QRS to the onset of left ventricular contraction (Q-OC interval) and to the onset of ejection (Q-OE interval) was performed. This was compared to the same intervals measured in 10 consecutive patients with normal QRS duration similarly studied during the evaluation of chest pain and/or congestive failure (fig. 2). (No correction for catheter delay was made in either group; catheter length was constant for all studies.) The mean duration of the Q-OC interval was longer in the group with LBBB than in those with a normal QRS duration, suggesting a delay in contraction of 0.035 sec due to the bundle branch block. This is approximately the difference in duration between a QRS of LBBB configuration and a normal QRS.

The difference between the mean Q-OE interval in those with normal QRS duration and those with LBBB is depicted in table 2 and figure 3. These intervals, however, are

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**Table 2**

<table>
<thead>
<tr>
<th>Timing of Left Ventricular Events</th>
<th>Q-OC (sec)</th>
<th>Q-OE (sec)</th>
<th>Q-OC vs Q-OE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal QRS (10)</td>
<td>0.116 ± 0.006</td>
<td>0.167 ± 0.0080</td>
<td>0.116 ± 0.006</td>
</tr>
<tr>
<td>LBBB (16)*</td>
<td>0.154</td>
<td>0.185</td>
<td>0.0044 ± 0.0004</td>
</tr>
<tr>
<td>With normal LVgrams (9)</td>
<td>0.154</td>
<td>0.185</td>
<td>0.0044 ± 0.0004</td>
</tr>
<tr>
<td>With abnormal LVgrams (6)</td>
<td>0.185</td>
<td>0.092</td>
<td>0.0044 ± 0.0004</td>
</tr>
</tbody>
</table>

*One patient did not undergo left ventriculography.

Values are ± standard error of the mean.
Contraction, $Q-OC = 0.06$

greatly influenced by the ability of the heart muscle to contract. If patients with abnormal contraction patterns of the hypokinetic or dyskinetic type are separated from those with normal patterns, the $Q-OE$ interval of those with LBBB and normal contraction patterns is 0.154 sec, with a mean isovolumic contraction period of 0.062 sec, which is similar to that of the patients with normal QRS duration. These findings suggest that the LBBB type of conduction abnormality in cases in which there is no other cause for abnormal contraction, causes only a delay in the onset of ventricular contraction but does not affect the synergy of left ventricular contraction.

**Discussion**

Left bundle branch block has been reported to occur most frequently among patients with arteriosclerotic heart disease and hypertension. Johnson et al.,$^1$ in a series of 555 patients with LBBB studied by only clinical criteria, found 35% to have clinical features consistent with coronary artery disease and 62% to have hypertension; 21% had both. Only 8.5% had rheumatic heart disease, and 2% had diseases that are associated with cardiomyopathy. Smith and Hayes$^2$ reported a higher incidence of arteriosclerotic heart disease, 68% among 146 patients, again using only clinical criteria for diagnosis. Thirteen percent had hypertension. A number of early reports$^3, 13, 14$ have documented that LBBB can occur in patients with otherwise normal hearts; but these were either selected case reports with no information on the frequency with which LBBB occurs or patients who were considered normal only by clinical criteria. Johnson et al.$^1$ considered 25 of their 555 patients to be normal, and Smith and Hayes$^2$ found 17 of their patients to have "no apparent cardiovascular disease." In contradistinction, only 29% of our 24 patients with symptoms sufficient to bring them to catheterization were found to have arteriographic evidence of coronary artery disease. The lower incidence of significant coronary atherosclerosis in our group than in previously reported series may be due to the fact that in the latter arteriosclerotic heart disease was diagnosed only by clinical criteria without anatomical studies in most cases; among the few autopsies reported, the

![Figure 2](image-url)

Systolic time intervals in a normal subject (QRS duration = 0.06 sec) and in a patient with LBBB (QRS duration = 0.12 sec) are determined from simultaneous left ventricular and brachial artery tracings. $Q-OC$ measures the time period from onset of the QRS to the onset of left ventricular contraction; $Q-OE$ the period from onset of QRS to the onset of ejection. The isovolumic contraction period represents the difference between $Q-OE$ and $Q-OC$.

![Figure 3](image-url)

Systolic time intervals (sec). Comparison between normal subjects and patients with LBBB. Bars represent mean and standard error of the mean.

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patients tended to be older. The pitfalls of diagnosing coronary atherosclerosis clinically in the presence of LBBB are demonstrated by the fact that only four of our 19 patients who complained of chest pain suggestive of angina were found to have coronary artery disease. In addition, four of the seven patients treated for clinical acute myocardial infarction had normal coronary arteries on angiography. The incidence of rheumatic heart disease and/or valvular disease was slightly higher than previously reported, but this can be explained by bias in selection for catheterization. Cardiomyopathy was more frequent in our series (21%), but such patients may well have been considered to have clinical arteriosclerotic heart disease if they had not been studied angiographically. Lewis et al.,6 in a recent report of 12 patients with LBBB studied with coronary arteriography, found results similar to ours: four had coronary atherosclerosis, one had rheumatic heart disease, one had cardiomyopathy, one had a small patent ductus, and five were normal, other than showing LBBB on ECG.

In this study, left axis deviation in addition to LBBB did not imply greater disease or more frequent coronary artery disease, contrary to the report of Beach et al.8 It has been postulated that left axis deviation may be due to disease in the anterior superior radiations of the left bundle,15 suggesting more diffuse disease than in patients with LBBB with normal axis. We found no evidence for this, as fewer patients with LBBB and left axis deviation had coronary disease than did those with LBBB and a normal axis.

The pathogenesis of LBBB in the majority of our group (those with normal coronary arteries) remains open to question. Congenital abnormalities of the left bundle branch occasionally occur,16 and cannot be ruled out. Electrolyte disturbances can cause intraventricular conduction defects, but were not present in any of our patients. Fibrosis, similar to that seen in complete heart block, may be playing a role in the etiology of LBBB. Harris et al.18 reported 84% of patients with complete heart block to be free of significant coronary atherosclerosis on anatomical study, but found 40% of their patients to have localized areas of fibrosis in the bundle branches. Our group with LBBB may have a similar fibrosing process, but possibly it is at an earlier stage. It is noteworthy that 27% in Harris’ series had LBBB before the onset of complete heart block. In 31% of their cases subjected to histologic study of the conduction system,19 the lesion was near the origin or in the proximal one-third of the left bundle branch. Other workers, however, have considered the pathology found in the bundle branch system to be inadequate to explain the presence of LBBB pattern on ECG.20

The abnormal conduction pattern was found to have little effect on the contraction pattern of the left ventricle as studied by cine ventriculography. Seven of the patients, four with extensive coronary artery disease and three with cardiomyopathy, were found to have contraction abnormalities, all attributed to their underlying disease. Such disordered ventriculographic patterns are seen frequently in patients with these diseases but who nevertheless exhibit normal conduction.12 We were unable to detect evidence of asynchrony, i.e., disturbed temporal sequence of contraction, with delayed contraction of part of the left ventricular wall due to late regional depolarization. Fourteen of the 21 patients with satisfactory ventriculograms, including some with coronary disease or cardiomyopathy and all the rheumatics, and all normal subjects had normal contraction patterns. This suggests that LBBB per se does not affect left ventricular contraction.

Hemodynamic studies, with catheterization data, on the electromechanical interval in LBBB are few. Braunwald et al.4 found that the Q-onset of contraction (Q-OC) time was normal in six patients: two with myocarditis, two with rheumatic heart disease, one with coronary artery disease, and after operation in one with congenital subvalvular aortic stenosis. Bourassa et al.5 found that the Q-OC time did not change when intermittent LBBB appeared in a patient with aortic insufficiency.
In this patient LBBB reverted to left ventricular hypertrophy type QRS with the administration of oxygen, suggesting that ischemia played a part in the production of the conduction defect. These investigators have suggested that the delay in ejection seen with LBBB is due to a prolonged isovolumic contraction period possibly caused by disordered contraction. They have suggested that proximal complete block in the LBBB is not present, but that depolarization is delayed because of block in the distal branches of the left bundle branch.

The results obtained from our studies are at variance with the findings of these workers and do not support their hypotheses. We found that the delay in onset of ejection (which they also demonstrated) was due to a delay in the onset of contraction, with the isovolumic interval normal in those patients with normal contraction patterns. The magnitude of the delay in onset of contraction seen in our patients is similar to the time for conduction from the right to left ventricle through the septum.21 It also approximates the time interval by which the LBBB QRS is longer than normal QRS.

The duration of the isovolumic period is normal in those patients with normal contraction patterns. This suggests that in these patients there is not diffuse disease in the peripheral Purkinje fibers of the left ventricle but that the block is high in the left bundle. It appears that after transversing the septum the impulse enters the Purkinje network below the block and that the left ventricular wall is depolarized in a normal sequence. It is possible, as suggested by Santos et al.,9 that some patients with LBBB pattern have block in the proximal and others in the peripheral parts of the left bundle branch. Our studies, however, suggest that most patients have block in the proximal left bundle.

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