The Anatomic Substrate of Complete Left Bundle Branch Block

By Maurice Lev, M.D., Paul N. Unger, M.D., Kenneth M. Rosen, M.D., and Saroja Bharati, M.D.

SUMMARY

The conduction systems of eight cases diagnosed clinically as having complete left bundle branch block (LBBB) were serially sectioned. Left axis deviation was present in six and normal axis in two. In all cases there was evidence of ischemia of the myocardium and left bundle branch (LBB) in various parts of the coronary circulation. In all cases, the LBB showed pathologic change at its junction point with the bundle of His, with disruption, complete or incomplete, in six, and with recent and old lesions in two. There was no difference in lesions of the LBB between cases with and without left axis deviation. There is thus complete correlation between the electrocardiographic abnormality LBBB and lesions of the LBB in these cases. The pathogenesis of the LBB lesions in these cases is probably both ischemic and mechanical.

Additional Indexing Words:
Conduction system Right bundle branch
Left bundle branch block Pathology
Left bundle branch

THE ANATOMIC BASIS for the electrocardiographic abnormality complete left bundle branch block (LBBB) is today in dispute. According to one group of investigators the anatomic base lies in lesions of the LBB. According to another group, it is doubtful that lesions of the LBB are the basis of complete LBBB, and that other factors have to be sought such as lesions of the nerves or of the myocardium itself.

We therefore studied the pathology of the conduction system and the entire heart in eight cases which were diagnosed electrocardiographically as having complete LBBB. Our findings lend credence to the view that most cases of LBBB have an anatomic basis in lesions of the LBB.

Materials and Methods

The electrocardiographic criteria for the diagnosis of complete LBBB were those of the New York State Heart Association. They are as follows: 1) QRS duration of 0.12 sec or greater; 2) presence of a broad monophasic R wave in lead V₆; 3) ST depression and T-wave inversion in V₆; and 4) absence of Q waves in V₆.

Pathologically, the coronary arteries were opened by dissection in the fresh heart. The heart was then opened and examined in a manner previously described, the chambers were packed with cotton, and the heart fixed in 10% formalin (4% formaldehyde).

Histologic examination was then carried out as follows: the sinoatrial (SA) and atrioventricular (A-V) nodes and their approaches and the beginning of the penetrating portion of the bundle were serially sectioned and every twentieth section was retained. The remainder of the penetrating and all of the branching portion of the bundle, the main LBB and the first part of the right bundle branch (RBB) were serially sectioned, and all sections were retained. The remainder of the bundle branches up through the level of the moderator band were serially sectioned and every tenth section was retained. The remainder of the heart was cut into blocks and two sections were taken from each block. Alternate sections were stained with hematoxylin-eosin and Weigert-van Gieson stains. The findings in these hearts were evaluated in light of previous studies of aging changes in the conduction system of the normal heart. In these studies the SA node of seven patients, 41-50 years of age; 14 patients, 51-60; eight patients, 61-70; and three patients, 71-80 had been examined. The A-V node, bundle, and bundle branches of 14 patients, 41-50; 12 patients, 51-60; and five patients, 61-70 had been similarly examined. These patients had no history of heart disease, no evidence of LBBB, and had normal hearts at autopsy.

Results

The results are given in tables 1, 2, and 3. From the electrocardiographic standpoint (table 1) (fig. 1), the QRS duration varied from 0.12 to 0.20 sec.
There was left axis deviation ($>-30^\circ$) in six and normal QRS axis in two ($-30^\circ$ to $+10^\circ$). Five patients were in sinus rhythm and three had atrial fibrillation. One case had a prolonged P-R interval.

Seven cases were the seat of arteriosclerotic or hypertensive and arteriosclerotic heart disease (Table 2). One case showed rheumatic aortic regurgitation and acute rheumatic myocarditis.

The weight of the heart varied from 400-800 g. Seven cases had hypertrophy of both ventricles, and

## Table 1

### Age, Sex, and Electrocardiographic Findings

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yrs.)</th>
<th>Sex</th>
<th>Supraventricular Rhythm</th>
<th>P-R duration</th>
<th>QRS duration</th>
<th>Axis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>(A-120-55)</td>
<td>71</td>
<td>M</td>
<td>NSR</td>
<td>0.18</td>
<td>0.16</td>
</tr>
<tr>
<td>2</td>
<td>(A-156-55)</td>
<td>81</td>
<td>F</td>
<td>Atrial fibrillation</td>
<td>—</td>
<td>0.16</td>
</tr>
<tr>
<td>3</td>
<td>(A-126-55)</td>
<td>59</td>
<td>M</td>
<td>NSR, PACs</td>
<td>0.16</td>
<td>0.16</td>
</tr>
<tr>
<td>4</td>
<td>(A-85-52)</td>
<td>49</td>
<td>M</td>
<td>Atrial fibrillation</td>
<td>—</td>
<td>0.18</td>
</tr>
<tr>
<td>5</td>
<td>(A-95-58)</td>
<td>61</td>
<td>M</td>
<td>Sinus tachycardia</td>
<td>0.16</td>
<td>0.20</td>
</tr>
<tr>
<td>6</td>
<td>(A-85-58)</td>
<td>82</td>
<td>M</td>
<td>NSR</td>
<td>0.28</td>
<td>0.18</td>
</tr>
<tr>
<td>7</td>
<td>(A-87-55)</td>
<td>55</td>
<td>M</td>
<td>NSR</td>
<td>0.20</td>
<td>0.12</td>
</tr>
<tr>
<td>8</td>
<td>(A-24-55)</td>
<td>67</td>
<td>M</td>
<td>Atrial fibrillation</td>
<td>—</td>
<td>0.12</td>
</tr>
</tbody>
</table>

*This electrocardiogram was not available for restudy.

NSR = normal sinus rhythm; PACs = premature ventricular contractions.

## Table 2

### Gross Findings in the Heart

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Type of heart disease</th>
<th>Wt. of heart</th>
<th>Chambers</th>
<th>Coronary arteries</th>
<th>Myocardium</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Hypertensive &amp; arteriosclerotic heart disease</td>
<td>500 g</td>
<td>Hypertrophy &amp; dilatation of both ventricles &amp; left atrium</td>
<td>Three vessel narrowing. Marked narrowing of ramus septi fibrosi</td>
<td>Old postero-septal infarct. Recent antero-septal infarct.</td>
</tr>
<tr>
<td>2</td>
<td>Hypertensive &amp; arteriosclerotic heart disease</td>
<td>520 g</td>
<td>Hypertrophy &amp; dilatation of both ventricles &amp; left atrium</td>
<td>Right coronary artery narrowed. Anterior descending showed recent thrombus. Arteriolosclerosis.</td>
<td>Old antero-septal infarct. Recent septal infarct</td>
</tr>
<tr>
<td>3</td>
<td>Arteriosclerotic heart disease</td>
<td>650 g</td>
<td>Hypertrophy &amp; dilatation of both ventricles &amp; left atrium</td>
<td>Three vessel narrowing</td>
<td>Old subendocardial infarct in all walls</td>
</tr>
<tr>
<td>4</td>
<td>Rheumatic heart disease with aortic insufficiency, &amp; mitral stenosis. Acute rheumatic myocarditis</td>
<td>760 g</td>
<td>Hypertrophy &amp; dilatation of all chambers</td>
<td>No narrowing of major vessels. Narrowing of ramus septi fibrosi. Arteriolosclerosis.</td>
<td>Rheumatic myocarditis</td>
</tr>
<tr>
<td>5</td>
<td>Arteriosclerotic heart disease</td>
<td>400 g</td>
<td>Hypertrophy of left atrium &amp; left ventricle</td>
<td>Three vessel narrowing. Marked narrowing of ramus septi fibrosi</td>
<td>Old postero-septal-lateral infarct. Recent anterior wall infarct</td>
</tr>
<tr>
<td>6</td>
<td>Arteriosclerotic heart disease</td>
<td>650 g</td>
<td>Hypertrophy &amp; dilatation of all chambers</td>
<td>Three vessel narrowing. Narrowing of ramus septi fibrosi. Arteriolosclerosis</td>
<td>Old subendocardial infarct. All walls with acute degeneration of cells</td>
</tr>
<tr>
<td>7</td>
<td>Hypertensive &amp; arteriosclerotic heart disease</td>
<td>500 g</td>
<td>Marked hypertrophy of all chambers</td>
<td>Three vessel narrowing. Narrowing ramus septi fibrosi. Arteriolosclerosis</td>
<td>Recent, organizing &amp; old infarct all walls</td>
</tr>
<tr>
<td>8</td>
<td>Hypertensive &amp; arteriosclerotic heart disease</td>
<td>800 g</td>
<td>Hypertrophy of all chambers &amp; dilatation of left ventricle</td>
<td>Thrombus in anterior descending. Arteriolosclerosis</td>
<td>Recent &amp; organizing infarct of anterior, posterior &amp; septal walls</td>
</tr>
</tbody>
</table>
one case had hypertrophy of the left ventricle and not the right. In six cases the left ventricle was also dilated.

In all cases there was evidence of ischemia of the myocardium and of the LBB (table 2). Thus, five cases showed three vessel narrowing or occlusion. One case had narrowing of the right and occlusion of the anterior descending coronary arteries. One case had occlusion of only the anterior descending. In five cases arteriolosclerosis was present (fig. 2).* In the case with occlusion of only the anterior descending, there was also arteriolosclerosis. In five cases the ramus septi fibrosis was also narrowed. In the case with no involvement of the main coronary arteries, there was narrowing of the ramus septi fibrosis and arteriolosclerosis.

Old and recent infarcts were present in seven of the eight cases. The infarcts varied in location, but all had a septal component, and involvement of the summit of the ventricular septum was common. In the rheumatic case without infarction, there was fibrosis of the septum related to old and more recent myocarditis. It was impossible to tell what at the summit of the ventricular septum was related to infarction or myocarditis, on the one hand, and what to "sclerosis of the left side of the cardiac skeleton," as a separate entity.

The conduction system proximal to the A-V bundle was irregularly involved (table 3). In the three cases with atrial fibrillation there was marked pathology in the atria. In the one case with prolonged P-R interval, the bundle of His was markedly involved. Three cases

*An arteriole in this paper is considered to be 0.3 mm in diameter or less (entire thickness) down to the capillary level.

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**Figure 1**

Case 3. Electrocardiogram showing complete left bundle branch block. There are occasional premature ventricular contractions.

**Figure 2**

Case 7. Destruction of main left bundle branch at its origin from bundle of His. Note the arteriolosclerosis. Hematoxylin-eosin stain ×45. B = bundle of His; LBB = left bundle branch; V = ventricular septum. Arrows point to arterioles.

**Figure 3**

Case 1. Destruction of the junction of the main left bundle at its origin from the bundle of His. Note the adjacent calcification in the summit of the ventricular septum. Hematoxylin-eosin stain ×45. B = bundle of His; LBB = left bundle branch; V = ventricular septum. Arrow points to interruption.
### Table 3

<table>
<thead>
<tr>
<th>Case no.</th>
<th>SA node</th>
<th>Approaches to SA node</th>
<th>Approaches to A-V node</th>
<th>A-V node</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Slight thickening of SA nodal artery</td>
<td>Negative</td>
<td>Marked fatty infiltration</td>
<td>Moderate fibrosis</td>
</tr>
<tr>
<td>2</td>
<td>No changes</td>
<td>Marked arteriolosclerosis</td>
<td>Moderate fatty infiltration</td>
<td>Moderate fibroelastosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Occasional zones of focal necrosis</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Negative</td>
<td>Negative</td>
<td>Marked fatty infiltration</td>
<td>Slight to moderate fibrosis</td>
</tr>
<tr>
<td>4</td>
<td>Marked infiltration of mononuclear cells, especially around vessels. Marked proliferation of intima of vessels. Moderate acute degeneration of cells</td>
<td>Moderate to marked arteriolosclerosis. Old fibrous periarteritis. Acute myocarditis. Marked perivascular proliferation of connective tissue</td>
<td>Marked hemorrhage, arteriolosclerosis &amp; perivascular fibrosis. Aschoff bodies present</td>
<td>Slight to moderate mononuclear cell infiltration &amp; elastosis</td>
</tr>
<tr>
<td>5</td>
<td>Arterioles slightly thickened</td>
<td>Slight thickening of arterioles</td>
<td>Slight fibroelastosis</td>
<td>Moderate fibroelastosis. Slight fatty infiltration</td>
</tr>
<tr>
<td>6</td>
<td>Mild focal acute degenerative changes</td>
<td>Moderate acute degenerative changes. Moderate fatty infiltration</td>
<td>Fibrosis with small scars</td>
<td>Normal</td>
</tr>
<tr>
<td>7</td>
<td>Marked arteriolosclerosis</td>
<td>Mild arteriolosclerosis. Moderate acute degenerative changes</td>
<td>Moderate fibrosis</td>
<td>Moderate focal acute degenerative changes. Slight infiltration of mononuclear cells</td>
</tr>
<tr>
<td>8</td>
<td>Marked arteriolosclerosis. Marked fatty infiltration of periphery</td>
<td>Marked arteriolosclerosis, with acute degeneration &amp; marked early necrosis of myocardium. Moderate fibroelastosis</td>
<td>Marked early necrosis with fibroelastosis</td>
<td>Marked elastosis, and arteriolosclerosis. Moderate fatty infiltration</td>
</tr>
</tbody>
</table>

**Abbreviations:** SA = sinoatrial; A-V = atrioventricular; LBB = left bundle branch; RBB = right bundle branch; l = left; r = right.
<table>
<thead>
<tr>
<th>A-V bundle penetrating</th>
<th>A-V bundle branching</th>
<th>LBB</th>
<th>RBB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slight elastosis</td>
<td>Moderate fibrosis</td>
<td>Entire l. main bundle separated from main bundle by fibroelastic tissue. Remainder of main bundle showed small cells. Peripherally-posteriorly-moderate fibrosis. Peripherally anteriorly-acute degeneration</td>
<td>Marked fibroelastosis of second portion</td>
</tr>
<tr>
<td>Moderate to marked fibroelastosis with slight fatty infiltration</td>
<td>Moderate to marked fibroelastosis with moderate arteriolosclerosis, space formation &amp; acute degenerative changes</td>
<td>Main l. bundle subtotally separated from A-V bundle by fibroelastic tissue. Recent infarct in the periphery anteriorly &amp; posteriorly</td>
<td>First &amp; second parts infiltrated &amp; surrounded by neutrophils</td>
</tr>
<tr>
<td>Slight fibrosis</td>
<td>Negative</td>
<td>Subtotally cut off at origin from main bundle. Peripherally, vacuolated Purkinje cells</td>
<td>Negative</td>
</tr>
<tr>
<td>Moderate fibroelastosis, slight infiltration of mononuclear cells</td>
<td>Numerous vascular channels l. side replaced by spaces. Marked fibroelastosis. Old healed granuloma in one area. Infiltration of mononuclear cells &amp; neutrophils</td>
<td>Completely cut off at origin from main bundle. Occasional Aschoff bodies further down. Peripherally posterior fibers intact. Anterior fibers degenerated</td>
<td>Moderate fibroelastosis</td>
</tr>
<tr>
<td>Occasional zones of degeneration, Slight elastosis. Slight infiltration with mononuclear cells</td>
<td>Marked acute degeneration on 1. side. Moderate to marked fibroelastosis on r. side. Increase in spaces on l. side. Marked fatty infiltration at bifurcation, mostly on l. side</td>
<td>Main l. bundle posteriorly-severe acute degeneration. Anteriorly mostly small cells with severe fibroelastosis. Peripherally, cells smaller than normal posteriorly. Anteriorly acute degeneration of cells</td>
<td>Normal</td>
</tr>
<tr>
<td>Moderate to marked fibroelastosis</td>
<td>Marked fibroelastosis with basophilic degeneration of cells. Anteriorly, focal necrosis &amp; hemorrhage on l. side</td>
<td>Posterior fibers separated by fibrosis. Anteriorly, acute degeneration of cells. Marked arteriolosclerosis. Peripherally, posterior fibers destroyed by old infarct. Anteriorly, dearth of cells showing degenerative changes</td>
<td>Marked fibroelastosis</td>
</tr>
<tr>
<td>Marked focal necrosis. Marked arteriolosclerosis</td>
<td>Marked arteriolosclerosis. L. side replaced by spaces. Focal necrosis</td>
<td>Up to bifurcation, main bundle replaced by spaces. Fibers at bifurcation show acute vacuolar &amp; eosinophilic degeneration. Peripherally acute degeneration of cells</td>
<td>Marked acute degeneration</td>
</tr>
<tr>
<td>Moderate elastosis &amp; arteriolosclerosis</td>
<td>Marked arteriolosclerosis. Moderate loss of substance on l. side. Slight increase in spaces</td>
<td>Posteriorly, main bundle completely replaced by fibroelastosis. Anteriorly almost completely replaced. Posteriorly, peripherally normal. Anteriorly, markedly vacuolated</td>
<td>Moderate elastosis</td>
</tr>
</tbody>
</table>

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showed significant changes in the penetrating, and six, significant changes in the branching portion of the bundle of His.

In all cases, there was marked involvement of the main left bundle (table 3). The region affected was the junction point between the bundle of His and the main left bundle (figs. 2 and 3). Sometimes the left side of the bundle of His was included in the lesion. This took the form of a disruption, complete or almost complete, in continuity, with the disruptive area filled with increased spaces and fibroelastic strands in six cases. In two cases both old and recent lesions were present. The peripheral portions of the left bundle both anteriorly and posteriorly showed either fibroelastosis, vacuolization of cells, or small (atrophied) Purkinje cells. In the rheumatic case, Achoff bodies were seen further down in the left bundle. There was no difference in pathologic findings in the LBB between cases with left axis deviation and cases with a normal QRS axis.

The RBB showed partial fibroelastic replacement in two, and acute infarction (fig. 4) or severe degeneration in two.

Discussion

The literature of the anatomic base of LBBB is of significance only since the introduction of precordial leads. Lenègre, Deglaude, and Hazim,2 in a case of subaortic stenosis with patent ductus arteriosus and LBBB, found destruction of the LBB. Segers11 felt that bundle branch block (BBB) was due to lesions at the junction of the Purkinje cells and the myocardium. Glomset and Birge12 thought that BBB was not related to lesions of the conduction system, but rather to the thickness and size of chambers and other factors. Langeron, Giard and Destombes,1 in a case of calcific aortic stenosis, found significant lesions in the LBB. Sanabria19 in two cases of LBBB found no lesions in the LBB. Summarizing his studies of 25 cases of complete LBBB, Lenègre3 found total destruction of LBB in nine, subtotal destruction in nine, partial lesions in five, and mild or no lesions in two. Lev,8 in a case with a congenitally malformed aortic valve with complete LBBB, found a significant lesion in the LBB. Baragan et al.,4 further summarizing their work, found significant lesions in LBB in 44 of 48 cases of complete LBBB. One case however had only a partial lesion, and three cases no lesions in the LBB. Harper et al.7 in three cases of LBBB found significant lesions in the LBB in two. Rossi10 in a study of five cases of complete LBBB found significant lesions in three and no lesions in two. Sugiuara et al.8 found excellent correlation with lesions of the LBB in eight cases of LBBB. Ohkawa et al.,5 in one case of complete LBBB, found significant lesions in the LBB. And finally, Ekelund et al.6 found good correlation between lesions in the bundle branches and ECG findings.

Our eight cases show complete correlation between the electrocardiographic abnormality LBBB and marked lesions in the LBB. In all cases the lesion lay at the junction of the LBB and the main bundle. Here there was disruption of the continuity of the LBB by spaces with fibroelastic strands or acute degeneration of the main LBB. Pathologic changes were also found more peripherally. Despite the fact that complete serial sections were done through the LBB it was often impossible to distinguish between “complete” and “almost complete” interruption. No such changes had been found in the series of normal hearts of the same age group.

Lenègre and his associates,3,18 Lev,19 Lev et al.,20 Unger et al.,21 and Sugiuara et al.8 had previously pointed out that the place of predilection for lesions of the LBB was at its origin from the bundle of His. Lenègre and his associates3, 18 pointed out that this is a spot where the fine fascicles of the LBB are normally sandwiched in between the connective tissue of the pars membranacea and that of the summit of the ventricular septum and are thus constantly subject to trauma related to the pull of the left ventricle and aorta. Our present work emphasizes that involvement of the summit of the ventricular septum by infarction, and fibrosis of the summit hastened by hypertension or due to spread from a scarred mitral valve, may be

Figure 4

Case 2. Acute infarction of RBB. Hematoxylin-eosin stain ×120. Arrows point to RBB.
ANATOMIC BASE OF LBBB

added factors in the development of this mechanically induced lesion in the LBB. Aortic regurgitation may constitute a hemodynamic factor predisposing the summit of the ventricular septum to lesions.

In all our cases there is a second factor in the lesions of the LBB — that of ischemia of the LBB. In five of our cases there was three vessel narrowing and in one the anterior descending was occluded while the right coronary artery was narrowed. Both of these vessels are concerned in the blood supply to the left main bundle branch. In addition, the ramus septi fibroso was often narrowed. This vessel is likewise concerned with the blood supply to the left main bundle. Furthermore, arteriosclerosis of the conduction system was frequent. In the case with occlusion of the anterior descending only, the arterioles were also involved, and in the case with rheumatic aortic regurgitation in which the main vessels were not involved, the arterioles were narrowed, as was the ramus septi fibroso. The involvement of the arterioles in some cases of LBBB has previously been pointed out.

Thus, we believe there are two factors in the production of lesions in the LBBB in our cases, one mechanical and one ischemic. We are left with the question as to whether the mechanical factor alone, in the form of sclerosis of the left side of the cardiac skeleton, by itself or exacerbated by hypertension, can produce LBBB without the influence of arteriosclerosis of the conduction system. It is well known that there is an "idiopathic" type of right bundle branch block with left axis deviation which we consider to be due to sclerosis of the left side of the cardiac skeleton. But few reports of the existence of LBBB of this type alone have been published. Lenègre and Sugihara et al. include a number of cases of LBBB with aortic regurgitation or hypertension in their series. But they do not indicate whether these particular cases were accompanied by arteriosclerosis.

The pathologic changes in the bundle of His deserve attention. Three cases showed significant changes in the penetrating portion, and six cases, significant changes in the branching portion. This raises the question of longitudinal dissociation in the His bundle, making possible in some cases the production of bundle branch block by lesions in the His bundle. However correlation with the consistent total or subtotal lesion at the beginning of the left bundle branch, found in all of our cases, is more probable.

The lesions of the RBB, although significant histologically in four cases, could not be detected electrocardiographically by our present criteria. The prolonged P-R in case 6 might be correlated with the significant lesions in the bundle of His and the RBB. His bundle recordings would be necessary to further delineate the electrophysiologic site of delay. The three cases with atrial fibrillation had extensive disease of the atria.

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References

5. OIKAWA S, SUGIURA M, IZUKA T, SHIMADA H, OKADA R: Three cases of idiopathic cardiomegaly in the aged, with special references to the morphological specificity and to the conduction system. Jap Heart J 12: 305, 1971
11. SEGERS M: Nouvelles bases d'interprétation de l'électrocardiogramme normal et pathologique. Acta Cardiol (suppl IV): 1, 1948
12. GLOMSET DJ, BIRGE RF: A morphologic study of the cardiac conduction system. V. The pathogenesis of heart block and bundle branch block. Arch Pathol 45: 135, 1948
19. LEV M: The normal anatomy of the conduction system in man...
22. FINK RJ, JAMES TN: Normal blood supply to the human His bundle and proximal bundle branches. Circulation 47: 8, 1973
23. LASCANO EF: Irrigación normal de nódulo de Tawara, haz de His y sus ramas. Rev argent Cardiol 10: 23, 1943
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