Observations on the Electrocardiogram and Ventricular Gradient in Complete Left Bundle Branch Block

By J. F. Pantridge, M.D.

Electrocardiograms from 109 patients with left bundle branch block were divided into four major groups depending on differences in standard and unipolar precordial leads. These records were then studied to find if one can draw any conclusions relative to ventricular hypertrophy or myocardial infarction in the presence of left branch block. In addition, the ventricular gradient was estimated in twenty-three instances and its value discussed.

Wilson and his associates\(^1,^2\) have shown that in the absence of precordial leads it may be impossible to differentiate the various types of intraventricular block. Studies of bundle branch block in which the diagnosis is based on the standard limb leads alone are unsatisfactory since right- and left-sided defects in conduction cannot be separated with certainty.

The object of this study is to present the electrocardiographic and some of the clinical features of a series of cases in which from examination of the standard and six or more unipolar precordial leads, it was thought that left bundle branch block was present. It was considered worthwhile to ascertain the extent to which the presence of this conduction defect obscures electrocardiographic evidence of cardiac enlargement and of myocardial infarction.

The records of 160 patients were examined. Those in which the QRS interval of the standard limb leads was not definitely greater than .12 second and those in which leads from the right side of the precordium suggested the possibility of delay in activation of the right ventricle were discarded; 109 remained for detailed study. Each of these was placed in one of the following groups.

**Group I**—45 cases. These records were considered in every respect typical of left branch block. Lead I showed a monophasic positive QRS deflection, and unipolar leads from the left precordium showed a broad flat-topped or bifid R wave with peaks of approximately equal magnitude. The amplitude of these R waves was always considerably less than that of the S wave of the leads from the right precordium (fig. 1). The symbol N/2 indicates that tracings were taken with the electrocardiograph at one half the normal sensitivity.

**Group II**—6 cases. These differed from those in group I in that an S wave was present in lead I and in one or more leads from the left side of the precordium (fig. 2).

**Group III**—13 cases. These records showed Q waves in lead I, lead V\(_L\), or the leads from the left side of the precordium (fig. 3).

**Group IV**—11 cases. The complexes of the leads from the left precordium were not of the type usually obtained from points to the left of the transitional zone in left bundle branch block. Complexes of this usual type were found in 2 of these cases when additional leads were obtained from the left side of the thorax. In one case these complexes were obtained at the posterior axillary line, in the other at the angle of the left scapula. Two cases in this group showed S waves in lead I.

The transitional zone in the majority of the cases in this study lay between the points from which leads V\(_4\) and V\(_5\) were taken. The factors responsible for its displacement to the left in cases in this group are not clear. In some it may have been due to right ventricular enlargement. However, undoubted evidence of preponderant

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From the Department of Internal Medicine, University of Michigan Medical School, Ann Arbor, Michigan.

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enlargement of the right ventricle was obtained in only 2 of these 11 cases.

Group V—34 cases. One or more precordial leads taken from points to the left of the transitional zone showed atypical R waves. In the majority of cases in this group these atypical deflections were bifid but there was a gross inequality in the voltage of the two peaks (fig. 4; see V₅ and V₆). In a few, R showed more than one notch (fig. 5).

CARDIAC ENLARGEMENT AND LEFT BUNDLE BRANCH BLOCK

The records of patients in groups I and II were studied with the object of ascertaining the relationship between the heart size and the magnitude of the deflections in the standard and precordial leads. The maximal deflections in leads I and III, the maximal depth of the S wave in leads from the right precordium, and the maximal height of the R wave in leads from the left precordium were measured, and the measurements were corrected for errors in standardization. The heart size was determined by radiologic examination which in the majority of instances included a 6 foot teleroentgenogram, an orthodiagram, and estimation of the cardiac area by the Hodges-Eyster method. Cardiac enlargement when present was classified as slight, moderate, or marked. The data obtained from this study are shown in table 1.

It will be seen that if allowance is made for the higher incidence of cardiac failure in those with larger hearts and for the fact that radiologic examination gives but an approximate estimate of the true heart size, the magnitude of the deflections in the precordial leads gives a reasonably good indication of the presence and degree of cardiac enlargement. A comparison...
of the records of hypertensive patients with those of patients without hypertension, shows that the average magnitude of the deflections in both the standard and precordial leads is considerably greater in the former (table 1, columns 6 and 7).

**Fig. 3.** Left bundle branch block with prominent Q waves in leads I, V₁, V₄, and V₆ (group III). Patient was a 66 year old Negro woman with arteriosclerotic and questionable syphilitic heart disease with far advanced heart failure and pericardial effusion. Patient had received digitalis.

**Myocardial Infarction Complicating Left Bundle Branch Block**

The difficulty of establishing the diagnosis of myocardial infarction in the presence of left bundle branch block is well known. It is due in large part to the fact that, with the exception of cases in which the interventricular septum is grossly involved, the potential of the cavity of the left ventricle is positive during the initial period of ventricular activation. Therefore Q waves associated with infarction and due to the transmission of the initial negative potential of the left ventricular cavity through the infarcted area do not occur. However, when the septum is grossly involved, the potential of the cavity of the right ventricle which is initially
negative in left bundle branch is transmitted to the cavity of the left ventricle. Left bundle branch block complicated by an infarct involving both the free wall of the left ventricle and the interventricular septum and extending through the greater part of the thickness of a portion of the latter, would therefore be expected to show Q waves in those leads which reflected the potential of the epicardial surface of the infarcted wall of the left ventricle. The records of cases in group III, those showing Q waves in leads I, V₃, or in leads from the left block. This is undoubtedly due to the more vertical position of the dog’s heart.

In left bundle branch block there are often no R waves in the leads from the right side of the precordium. When these deflections are absent and the transitional zone is displaced to the left, Q waves found in leads V₅ and V₆ may represent the transmission to the left side of the precordium of the potential variations responsible for the QS deflections of the leads from the right side; that is to say, the complexes of leads V₅ and V₆ in such cases probably result from the relative demarcation of the QS waves into Q and S waves, the latter being the result of the diminished thickness of the epicardial surface of the right side of the left ventricle.

The presence of Q waves in leads I and V₃ does not necessarily indicate infarction with septal involvement since these deflections might be expected if the heart were so placed that the initial negative potential of the cavity of the right ventricle was transmitted to the left arm. In this connection it may be noted that the incidence of a Q wave in lead I is much higher in experimental left bundle branch block in the dog than in clinical left bundle branch block. In addition, the presence of a Q wave in lead I is also associated with the absence of R waves in leads V₅ and V₆. The presence of an R wave in lead V₃ and its diminution or disappearance in leads V₅ and V₆ is suggestive of anteroseptal infarction. The measurements are expressed in millimeters at normal standardization.

**Table 1. Relationship between Heart Size and Magnitude of Deflections in Standard and Preordial Leads.**

<table>
<thead>
<tr>
<th>Heart Size</th>
<th>Normal</th>
<th>Slightly Enlarged</th>
<th>Moderately Enlarged</th>
<th>Markedly Enlarged</th>
<th>Patients with Hypertension</th>
<th>Patients without Hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>I + III</td>
<td>Max. 21.5</td>
<td>33</td>
<td>22.5</td>
<td>34</td>
<td>38.5</td>
<td>21.5</td>
</tr>
<tr>
<td></td>
<td>Av. -12.4</td>
<td>17.5</td>
<td>11.4</td>
<td>14.7</td>
<td>18.8</td>
<td>12.5</td>
</tr>
<tr>
<td></td>
<td>Min. 6.5</td>
<td>5</td>
<td>6.5</td>
<td>7</td>
<td>7.0</td>
<td>5.0</td>
</tr>
<tr>
<td>S</td>
<td>Max. 46</td>
<td>68</td>
<td>76</td>
<td>80</td>
<td>80.0</td>
<td>64</td>
</tr>
<tr>
<td></td>
<td>Av. 33.3</td>
<td>43.5</td>
<td>42.5</td>
<td>47.5</td>
<td>53.2</td>
<td>35.8</td>
</tr>
<tr>
<td></td>
<td>Min. 18</td>
<td>22</td>
<td>15</td>
<td>25</td>
<td>29.0</td>
<td>15</td>
</tr>
<tr>
<td>R + S</td>
<td>Max. 60</td>
<td>82</td>
<td>90</td>
<td>117</td>
<td>117.0</td>
<td>83</td>
</tr>
<tr>
<td></td>
<td>Av. 44.5</td>
<td>57</td>
<td>57.1</td>
<td>62</td>
<td>69.3</td>
<td>46.6</td>
</tr>
<tr>
<td></td>
<td>Min. 33</td>
<td>34</td>
<td>18</td>
<td>28.5</td>
<td>30.0</td>
<td>18</td>
</tr>
<tr>
<td>QRS</td>
<td>Max. 0.16</td>
<td>0.18</td>
<td>0.16</td>
<td>0.16</td>
<td>0.18</td>
<td>0.16</td>
</tr>
<tr>
<td></td>
<td>Av. 0.144</td>
<td>0.152</td>
<td>0.146</td>
<td>0.143</td>
<td>0.150</td>
<td>0.143</td>
</tr>
<tr>
<td></td>
<td>Min. 0.12+</td>
<td>0.12+</td>
<td>0.12+</td>
<td>0.12+</td>
<td>0.12+</td>
<td>0.12+</td>
</tr>
</tbody>
</table>

I + III = The sum of the maximal deflection in lead I and the maximal deflection in lead III.

S = The maximal depth of the S wave in the leads from the right side of the precordium.

R + S = The sum of the maximal R wave from the left precordium and the maximal S wave from the right precordium. The measurements are expressed in millimeters at normal standardization.

QRS = The duration of the QRS interval in seconds.
in table 2. It will be seen that in only 4 cases (51, 64, 90, 105) did the electrocardiographic findings indicate the probability of myocardial infarction with septal involvement and that in only 2 did the clinical history support the impression obtained from the electrocardiogram.

The order of excitation of the ventricles in left bundle branch block is such that the initial peak of the usual bifid R wave in leads from the left side of the transitional zone may be expected to represent the arrival of the excitation wave at the left side of the septum. The final peak of the R wave in these leads represents in all probability the arrival of the excitation wave at the epicardial surface of the left ventricle immediately beneath the exploring electrode. Atypical complexes in leads from the left side of the transitional zone might therefore be associated with infarction of the septum or free wall of the left ventricle. Those in which the magnitude of the second peak is much smaller than that of the initial peak may result from damage of that part of the free wall of the left ventricle which lies immediately beneath the exploring electrode. Septal damage of insufficient extent to produce Q waves in the left precordial leads might cause a diminution in the magnitude of the primary peak of the R wave in these leads. For these reasons the incidence of infarction in cases of left branch block with typical complexes in the left precordial leads (groups I and II) was compared with its incidence in cases in which these complexes were atypical (group V). This comparison is largely on the basis of clinical data. However, it will be seen from table 3 that the difference in the incidence of infarction in the two groups is probably significant.

**TABLE 2. Probable Incidence of Myocardial Infarction (Based on Clinical Impressions) Related to Occurrence of Q Waves and Absence of R Waves in Certain Leads**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Position of Q Wave</th>
<th>Leads with R waves Absent</th>
<th>Clinical Impression Relating to Infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Left Precordium</td>
<td>V₁</td>
<td>Lead I</td>
</tr>
<tr>
<td>10</td>
<td>—</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>29</td>
<td>—</td>
<td>*</td>
<td>+</td>
</tr>
<tr>
<td>45</td>
<td>—</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>51</td>
<td>V₅ and V₆</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>53</td>
<td>—</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>60</td>
<td>—</td>
<td>+</td>
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<td>80</td>
<td>—</td>
<td>+</td>
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</tr>
<tr>
<td>90</td>
<td>V₅ and V₆</td>
<td>*</td>
<td>+</td>
</tr>
<tr>
<td>92</td>
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<td>*</td>
<td>+</td>
</tr>
<tr>
<td>102</td>
<td>—</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>105</td>
<td>V₅</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

* V₁ not recorded.

**TABLE 3.—Probable Incidence of Myocardial Infarction Related to the Existence of Typical or Atypical QRS Complexes in Precordial Leads Taken to the Left of the Transitional Zone.**

<table>
<thead>
<tr>
<th>Groups</th>
<th>No. of Cases</th>
<th>Evidences of Infarction</th>
<th>Probable Infarction</th>
<th>Possible Infarction</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>I and III</td>
<td>51</td>
<td>3</td>
<td>2</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>V</td>
<td>32</td>
<td>7</td>
<td>4</td>
<td>5</td>
<td>16</td>
</tr>
</tbody>
</table>

This view would be supported if additional leads from the left side of the precordium were obtained from patients in whom the usual precordial leads yielded complexes typical of left bundle branch block. A few of these patients might be expected to have infarction of the free wall of the left ventricle in regions not explored by the usual precordial leads. If, then, atypical complexes in which the second peak of the bifid R wave was much smaller than the
that the exploring electrode had been placed over the infarcted area.

Information of the type under consideration was obtained during a study of the effect of position of the heart on the precordial electrocardiogram in left bundle branch block. It was noted that in some patients the character of the complexes obtained in leads from the left side of the transitional zone was not affected by alteration of the position of the patient while the precordial leads were recorded, and that in others typical complexes in these leads were converted to those of an atypical form.

Figure 6 shows the limb and precordial leads in a case with typical left bundle branch block recorded with the patient in the usual recumbent position. Figure 7 shows the marked alteration that occurred in the secondary R waves in the leads V₅ and V₆ when these leads were recorded with the patient in different positions. Leads V₄, V₅, V₆, and V₇ were recorded in the supine, in the sitting, in the right lateral, and in the left lateral positions. The secondary R waves in leads V₆ in the sitting, right and left lateral positions and those of lead V₅ in the left lateral position, occur as a notch at the base of the descending limb of the primary R wave. These secondary R waves closely resemble those of leads V₅ and V₆ in figure 4.

It has been suggested that records of the type in which the initial R in lead I and in the leads from the left precordium is peaked and shows a broad notch on its descending limb represent focal left-sided block of major degree. Since, however, waves of this type may be found on additional exploration of the left precordium in patients with typical left bundle branch block, it is possible that in some cases they indicate damage of the free wall of the left ventricle.

**The Ventricular Gradient in Complete Left Bundle Branch Block**

It has been shown that the order of activation of the ventricular myocardium is not likely to affect either the magnitude or the direction of the vector which represents the lack of uniformity in the duration of its excited state. Accurate determination of this vector might therefore be expected to yield some elec-
trocardiographic information which is obscured by the presence of left bundle branch block.

This vector, the ventricular gradient, was determined in 23 cases in this series. An attempt was first made to estimate from clinical examination the extent of the myocardial damage in the cases selected. In a few the results of postmortem examination were available. On the basis of this estimation of the degree of myocardial damage the cases were divided into the following four groups: (a) Minimal myocardial disease. (b) Slight or moderate myocardial disease. (c) Grave myocardial disease without clinical or pathologic evidence of infarction. (d) Probable or proved myocardial infarction. With the exception of 3 cases in which electronic integrated electrocardiographic records were available, the following method was used in this study. A ventricular complex was selected from each standard lead at a portion of the tracing where the base line shift was minimal. Particular care was taken to ensure that the complexes selected were those recorded at approximately the same phase of respiration. Each complex was then enlarged six to eight times by means of a photographic enlarger. The recorded areas of the QRS and T components of each complex are each an average of three planimeter readings. The algebraic sum of the areas of QRS and T in each lead gave the magnitude of the QRS-T or ventricular gradient, G, in that lead. The sum of the readings in leads I and III seldom exactly equalled that in lead II. These readings were adjusted to their most probable values by adding to lead II and subtracting from leads I and III the quantity X, which is defined as the algebraic sum of leads I and III less lead II. The adjusted readings were used when calculating the mean electrical axis and the manifest area of QRS, T, and G. It was then possible to estimate roughly the error in the calculated magnitude and direction of these vectors in terms of the magnitude of X. When \( M \) represents the magnitude of the vector in millivolt seconds, \( \frac{+X}{50M} \) (degrees) is approximately twice the probable error in the calculated direction of the vector and \( \frac{+X}{M} \) per cent is roughly twice the probable error in its magnitude provided the measurement errors are random. The term "probable error" is used in its customary statistical sense. The actual error will exceed twice the probable error in approximately 20 per cent of the calculations.

The more important clinical features, the mean electrical axis and the manifest area of the QRS, T, and G of the selected patients are shown in Table 4. The range of error, defined as twice the probable error in these determinations, and the ratio between the manifest areas of G and QRS are also shown.

It will be seen that in some cases the range of error is considerable. In case 13 the range of error in the direction of the gradient is 20 degrees. Five cases, in which the range of error in the determination of the gradient was greater than this, were excluded from this study. Since measurement of the area beneath the ventricular deflections by a planimeter is the most accurate visual method of determining the ventricular gradient, it is apparent that the error in determining this vector by the more rapid visual methods may be gross.

Vectors representing the position of the mean electrical axis and the manifest area of G and QRS have been plotted in Figure 8.

In cases 1–3 (fig. 8A), which are considered examples of minimal myocardial disease, G is in magnitude and direction within the limits at present regarded as normal,\(^\text{13-14}\).

In 2 out of 5 cases regarded as examples of slight or moderate myocardial disease, G is grossly abnormal (cases 4–9, fig. 8B). In case 8, it is abnormal, because of its direction \((-62.5^\circ)\) and in case 9, because of the magnitude of the angle between G and QRS \((92.5^\circ)\). The magnitude and the G:QRS ratio in the latter case are also markedly reduced. This may, however, be due to digitalis.

Each of the 5 cases with clinical or pathologic evidence of grave myocardial disease show an abnormal ventricular gradient (fig. 8C). In case 11, G is grossly abnormal in direction \((-60^\circ)\). The remaining cases in this group show an abnormally small G and G:QRS ratio. These patients may have received digitalis.

In all 4 cases with probable myocardial infarction, G is markedly abnormal (fig. 8D). In
<table>
<thead>
<tr>
<th>No.</th>
<th>Age</th>
<th>Diagnosis</th>
<th>BP</th>
<th>Heart Size</th>
<th>Evidence of Failure</th>
<th>History of Infarction</th>
<th>Digitals</th>
<th>Heart Rate</th>
<th>α</th>
<th>M</th>
<th>E%</th>
<th>M-QRS/QRS</th>
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<tr>
<td>1</td>
<td>24</td>
<td>Functional systolic murmur</td>
<td>114/68</td>
<td>Normal</td>
<td>—</td>
<td>—</td>
<td>76</td>
<td>QRS −10</td>
<td>69</td>
<td>3</td>
<td>5</td>
<td>1.2</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>T 75</td>
<td>42</td>
<td>5</td>
<td>10</td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>QRST 19</td>
<td>84</td>
<td>5</td>
<td>9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>56</td>
<td>Cholecystitis. No apparent cardiac abnormality.</td>
<td>—</td>
<td>Normal</td>
<td>—</td>
<td>—</td>
<td>97</td>
<td>QRS −9</td>
<td>69</td>
<td>6</td>
<td>11</td>
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<td></td>
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<td></td>
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<td></td>
<td>T 122</td>
<td>36</td>
<td>11</td>
<td>23</td>
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<td>Thyrotoxicosis</td>
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<td>Normal</td>
<td>—</td>
<td>—</td>
<td>65</td>
<td>QRS 38</td>
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<td>4</td>
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<td>22</td>
<td></td>
<td></td>
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<td>4</td>
<td>72</td>
<td>Myxedema</td>
<td>120/80</td>
<td>Normal</td>
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<td>—</td>
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<td>108</td>
<td>1</td>
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<td>9</td>
<td>18</td>
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<tr>
<td>5</td>
<td>37</td>
<td>Essential hypertension Cholecystitis</td>
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<td>—</td>
<td>88</td>
<td>QRS 2</td>
<td>71</td>
<td>5</td>
<td>9</td>
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<td>10</td>
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<td>39</td>
<td>Rheumatic heart disease. Mitral stenosis</td>
<td>120/90</td>
<td>? slight enlargement</td>
<td>—</td>
<td>—</td>
<td>75</td>
<td>QRS 6</td>
<td>89</td>
<td>1</td>
<td>2</td>
<td>0.53</td>
</tr>
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<td>7</td>
<td>71</td>
<td>Arteriosclerotic heart disease. Hypertension.</td>
<td>105/100</td>
<td>Slight enlargement</td>
<td>—</td>
<td>—</td>
<td>78</td>
<td>QRS 22</td>
<td>36</td>
<td>11</td>
<td>19</td>
<td>1.2</td>
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<tr>
<td></td>
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<td>Death from bronchopneumonia. P.M. heart weight 400 Gm. No evidence of infarction.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>T 99</td>
<td>20</td>
<td>30</td>
<td>60</td>
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<td></td>
<td></td>
<td>QRST 47</td>
<td>45</td>
<td>12</td>
<td>24</td>
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<td>8</td>
<td>49</td>
<td>Chronic Nephritis</td>
<td>232/126</td>
<td>Moderate enlargement</td>
<td>—</td>
<td>—</td>
<td>73</td>
<td>QRS −62</td>
<td>181</td>
<td>4</td>
<td>8</td>
<td>0.45</td>
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<td></td>
<td></td>
<td>T 123</td>
<td>101</td>
<td>1</td>
<td>2</td>
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<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>QRST −69</td>
<td>81</td>
<td>7</td>
<td>14</td>
<td></td>
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</tr>
<tr>
<td>9</td>
<td>82</td>
<td>Carcinoma of bladder, postoperative death. P.M. patchy myocardial fibrosis</td>
<td>128/69</td>
<td>Moderate enlargement, 406 Gm.</td>
<td>—</td>
<td>—</td>
<td>79</td>
<td>QRS −55</td>
<td>61</td>
<td>1</td>
<td>1</td>
<td>0.19</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>T 111</td>
<td>72</td>
<td>1</td>
<td>1</td>
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<td></td>
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<td></td>
<td></td>
<td>QRST 37</td>
<td>11</td>
<td>4</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>74</td>
<td>Arteriosclerotic heart disease</td>
<td>118/50</td>
<td>Moderate enlargement</td>
<td>Moderate congestive failure</td>
<td>—</td>
<td>+ 107</td>
<td>QRS 22</td>
<td>33</td>
<td>6</td>
<td>12</td>
<td>0.36</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>T −151</td>
<td>21</td>
<td>15</td>
<td>30</td>
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<td>QRST 10</td>
<td>12</td>
<td>4</td>
<td>8</td>
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<td></td>
<td></td>
<td></td>
<td>QRS −65</td>
<td>220</td>
<td>2</td>
<td>4</td>
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</tr>
<tr>
<td>11</td>
<td>72</td>
<td>Arteriosclerotic heart disease</td>
<td>140/90</td>
<td>Moderate enlargement</td>
<td>Mild congestive failure</td>
<td>—</td>
<td>+ 65</td>
<td>QRS −60</td>
<td>67</td>
<td>1</td>
<td>1</td>
<td>0.30</td>
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<td></td>
<td></td>
<td></td>
<td>T 112</td>
<td>153</td>
<td>3</td>
<td>6</td>
<td></td>
<td></td>
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<tr>
<td>12</td>
<td>60</td>
<td>Arteriosclerotic heart disease. Angina pectoris. P.M. gross myocardial scarring</td>
<td>200/100</td>
<td>Moderate enlargement, 480 Gm.</td>
<td>—</td>
<td>—</td>
<td>+</td>
<td>54</td>
<td>QRS</td>
<td>—76</td>
<td>41</td>
<td>1</td>
</tr>
<tr>
<td>13</td>
<td>74</td>
<td>Arteriosclerotic heart disease</td>
<td>130/75</td>
<td>Normal</td>
<td>—</td>
<td>—</td>
<td>+</td>
<td>77</td>
<td>QRS</td>
<td>29</td>
<td>35</td>
<td>4</td>
</tr>
<tr>
<td>14</td>
<td>57</td>
<td>Rheumatic heart disease. Hypertension. Pulmonary tuberculosis.</td>
<td>170/90</td>
<td>Moderate enlargement</td>
<td>Moderate congestive failure</td>
<td>P.M. Gross fibrosis left ventricle. Coronary vessels patent Suggestive</td>
<td>+</td>
<td>70</td>
<td>QRS</td>
<td>42</td>
<td>57</td>
<td>1</td>
</tr>
<tr>
<td>15</td>
<td>65</td>
<td>Arteriosclerotic heart disease</td>
<td>122/86</td>
<td>Slight enlargement</td>
<td>Moderate congestive failure</td>
<td>Highly suggestive history</td>
<td>+</td>
<td>72</td>
<td>QRS</td>
<td>—62</td>
<td>87</td>
<td>1</td>
</tr>
<tr>
<td>16</td>
<td>59</td>
<td>Arteriosclerotic heart disease</td>
<td>126/90</td>
<td>Marked enlargement, 750 Gm.</td>
<td>Gross congestive failure</td>
<td>P.M. Old infarction involving the apex with mural thrombosis</td>
<td>+</td>
<td>102</td>
<td>QRS</td>
<td>—64</td>
<td>68</td>
<td>4</td>
</tr>
<tr>
<td>17</td>
<td>53</td>
<td>Arteriosclerotic heart disease</td>
<td>140/103</td>
<td>Marked cardiac enlargement</td>
<td>Mild congestive failure</td>
<td>Two typical clinical attacks</td>
<td>—</td>
<td>100</td>
<td>QRS</td>
<td>—36</td>
<td>36</td>
<td>3</td>
</tr>
<tr>
<td>18</td>
<td>49</td>
<td>Essential hypertension</td>
<td>226/122</td>
<td>Slight enlargement</td>
<td>—</td>
<td>Two fairly typical attacks</td>
<td>—</td>
<td>94</td>
<td>QRS</td>
<td>—52</td>
<td>103</td>
<td>9</td>
</tr>
</tbody>
</table>

\[ \alpha = \text{The angle in the triaxial reference system} \]
\[ M = \text{The magnitude in millivolt seconds (4mv. = 1 unit)} \]
\[ E_\alpha = \text{The range of error in estimating the angle (twice the probable error)} \]
\[ EM\% = \text{Range of error in estimating the magnitude} \]
\[ M_{QRST}/QRS = \text{magnitude QRS} \]

597
cases 16 and 18 it is abnormal in direction (−81° and −45°) and in cases 15 and 17 it is markedly reduced in magnitude and the G:QRS ratio is small. Digitalis can be excluded as the
cause of the reduction of the magnitude of G and G:QRS ratio in case 17.

**Summary**

A study of the electrocardiogram in cases of complete left bundle branch block shows that the presence of this conduction defect does not obscure the evidence of left ventricular enlargement in the precordial electrocardiogram.

The usual electrocardiographic evidence of myocardial infarction is with few exceptions completely obscured by left bundle branch block. Variations in the form of the QRS complex in leads from the left side of the transitional zone in left bundle branch block are described. The significance of these variations in relation to the diagnosis of myocardial infarction is discussed.

The ventricular gradient has been determined in 18 cases of complete left bundle branch block. The results support the belief based on theoretic and experimental evidence that changes in the magnitude and direction of this vector will reveal electrocardiographic information usually obscured by this conduction defect. Despite the use of greatly enlarged records and an accurate planimeter the sum of the areas in leads I and III did not always closely approximate that of lead II. This implies large uncertainties (probable errors) in the calculated magnitude and direction of the ventricular gradient and indicates the difficulty in determining these quantities precisely.

**Acknowledgments**

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J. F. PANTRIDGE

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