Electrocardiographic criteria of left ventricular hypertrophy in left bundle-branch block


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SUMMARY In order to determine whether the electrocardiographic criteria of left ventricular hypertrophy apply in the presence of left bundle-branch block we studied 79 cases of intermittent left bundle-branch block and compared the QRS voltage and axis before and after its onset. Cases of incomplete left bundle-branch block were excluded. There was a statistically significant correlation between pre- and post-left bundle-branch block values of R or S wave voltage in leads I, V1, V2, V5, and V6, the Sokolow index (R V5 or V6 + S V1), and the QRS axis. There was a statistically significant reduction in R wave voltage in leads I, V5, and V6, an increase in S wave voltage in V1 and V2, and leftward shift of QRS axis, but the Sokolow index remained unchanged, after the onset of left bundle-branch block. The Sokolow criteria for left ventricular hypertrophy apply satisfactorily even in the presence of left bundle-branch block, though specificity is low, but QRS axis is unhelpful.

When left bundle-branch block is present, the diagnosis of other electrocardiographic abnormalities such as myocardial ischaemia or infarction is considered difficult or impossible (Chapman and Pearce, 1957; Cooksey et al., 1974; Susmano et al., 1976). Though some authors believe that the electrocardiographic voltage criteria for left ventricular hypertrophy apply in the presence of left bundle-branch block (Goldman, 1964), others state that the diagnosis of left ventricular hypertrophy is unreliable when there is also left bundle-branch block (Friedberg, 1966; Estes, 1970; Lipman et al., 1972).

We studied patients with intermittent left bundle-branch block and compared QRS voltage and axis before and after the appearance of the block.

Patients and methods

Three groups of patients were studied:

1. Thirty-six patients with intermittent rate dependent left bundle-branch block, observed by us.

2. Thirty-five cases of intermittent left bundle-branch block collected from published reports (Comeau et al., 1938; Vessell and Lowen, 1963; Bauer, 1964a, b; Baragan et al., 1967; Barold et al., 1968; Cohen et al., 1969; Friedberg and Schamroth, 1969; Horan et al., 1970; Krikler and Lefevre, 1970; Lewis et al., 1970; D’Cunha et al., 1971; Cohen and Voukydis, 1973; Luy et al., 1973; Rosenbaum et al., 1973; Luy, 1974; El-Sherif, 1974; Neuss et al., 1974; Rosen et al., 1975; Barnay et al., 1976).

3. Eight patients in whom a left bundle-branch block pattern was produced by pacing from the inflow tract of the right ventricle. In 2, intermittent left bundle-branch block also occurred spontaneously and in them the morphology of the pacing-induced and spontaneous left bundle-branch block could be compared.

The criteria employed for the diagnosis of left bundle-branch block were a QRS duration equal to or greater than 0.12 s and conspicuous notching or slurring of QRS (Friedberg, 1966). We did not include any cases with ‘incomplete’ left bundle-branch block since the voltage has been found to increase with its occurrence (Barold et al., 1968). The criteria for this diagnosis were absence of initial q wave in left ventricular leads, with QRS duration equal to or more than 0.10 s and slurring of QRS (Barold et al., 1968; Friedberg and Schamroth, 1969; Estes, 1970).

The voltage criteria for left ventricular hypertrophy were those employed by Sokolow and Lyon (1949). We also included the criterion proposed by Estes (1970) of maximum R or S wave voltage of 20 mm or more in a limb lead. We did not employ criteria based on intrinsicoid deflection, left axis.
Left ventricular hypertrophy in left bundle-branch block

Table  QRS changes after development of left bundle-branch block (LBBB)

<table>
<thead>
<tr>
<th></th>
<th>Pre-LBBB</th>
<th>Post-LBBB</th>
<th>Δ</th>
<th>P</th>
<th>Correlation coefficient (r)</th>
<th>Regression equation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lead I R wave</td>
<td>10.33 ± 0.52*</td>
<td>8.36 ± 0.48</td>
<td>-1.97 ± 0.54</td>
<td>&lt; 0.001</td>
<td>0.602 (P &lt; 0.001)</td>
<td>y = 2.56 + 0.561x</td>
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<tr>
<td>(n = 71) amplitude (mm)</td>
<td></td>
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<tr>
<td>Lead V1 S wave</td>
<td>13.61 ± 0.70</td>
<td>19.84 ± 0.94</td>
<td>+6.23 ± 1.24</td>
<td>&lt; 0.001</td>
<td>0.574 (P &lt; 0.001)</td>
<td>y = 14.04 + 0.426x</td>
</tr>
<tr>
<td>(n = 53) amplitude (mm)</td>
<td></td>
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<tr>
<td>Lead V2 S wave</td>
<td>14.26 ± 2.06</td>
<td>25.39 ± 1.84</td>
<td>+11.13 ± 2.02</td>
<td>&lt; 0.01</td>
<td>0.466 (P &lt; 0.05)</td>
<td>y = 18.74 + 0.421x</td>
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<td>(n = 23) amplitude (mm)</td>
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<tr>
<td>Lead V5 R wave</td>
<td>15.28 ± 1.46</td>
<td>7.88 ± 0.83</td>
<td>-7.4 ± 1.10</td>
<td>&lt; 0.001</td>
<td>0.796 (P &lt; 0.001)</td>
<td>y = 1.99 + 0.472x</td>
</tr>
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<td>(n = 24) amplitude (mm)</td>
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<tr>
<td>Lead V6 R wave</td>
<td>14.69 ± 0.90</td>
<td>9.52 ± 0.53</td>
<td>-5.17 ± 0.65</td>
<td>&lt; 0.001</td>
<td>0.833 (P &lt; 0.001)</td>
<td>y = 14.91 + 0.614x</td>
</tr>
<tr>
<td>(n = 53) amplitude (mm)</td>
<td></td>
<td></td>
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<tr>
<td>Sokolow index (mm)</td>
<td>32.95 ± 2.06</td>
<td>35.15 ± 1.52</td>
<td>+2.2 ± 1.36</td>
<td>N.S.</td>
<td>0.833 (P &lt; 0.001)</td>
<td>y = 14.91 + 0.614x</td>
</tr>
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<td>(n = 40)</td>
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<td>QRS axis (degrees)</td>
<td>13.67 ± 3.95</td>
<td>9.76 ± 4.63</td>
<td>-3.94 ± 5.21</td>
<td>&lt; 0.001</td>
<td>0.860 (P &lt; 0.001)</td>
<td>y = -23.86 + 1.032x</td>
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<td>(n = 65)</td>
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</table>

*Mean ± SEM

deviation, or ST or T wave changes, since they may not give a true reflection of normal baseline values in patients prone to develop left bundle-branch block. Similarly, the P wave terminal forces were not measured, as they are often abnormal in patients with coronary heart disease, even in the absence of left ventricular overload (Porfong and Stake, 1976). The correlation coefficient and the paired t test were employed for comparison of pre- and post-left bundle-branch block values.

**Results**

1. There was a significant correlation between pre- and post-left bundle-branch block R or S wave voltage in the following leads only (Table): lead I (Fig. 1), V1 (Fig. 2), V2, and V5 and V6 taken together (Fig. 3). There was also an excellent correlation between pre- and post-left bundle-branch block Sokolow index (Fig. 4).

2. The S wave voltage increased significantly in leads V1 and V2, and R wave voltage decreased in leads I, V5, and V6. The Sokolow index did not change significantly (Table). Fourteen patients had a Sokolow index equal to or more than 35 mm before the onset of left bundle-branch block; 13 of these had values equal to or more than 35 mm post-left bundle-branch block. Twenty-six had an index equal to or less than 35 mm before the onset of left bundle-branch block, and in 7 of these the index was equal to or more than 35 mm post-left bundle-branch block.

3. There was a significant correlation between the pre- and post-left bundle-branch block QRS axis. A significant leftward shift occurred (Table). In the 13 patients in whom there was evidence of left ventricular hypertrophy before the onset of left

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Fig. 1  Correlation of pre- and post-left bundle-branch block R wave voltage in lead I.

Fig. 2  Correlation of pre- and post-left bundle-branch block S wave voltage in lead V1.
bundle-branch block, the degree of axis change was similar to that in the 40 patients without left ventricular hypertrophy. Of the 13 former patients, 6 had an axis to the left of −30° after the development of left bundle-branch block, while 17 of the 40 in the latter group had a similar degree of left axis deviation.

(4) The T wave became mainly negative in the left ventricular leads (I, aVL, V5, V6) in most patients, even in 8 in whom it had been negative pre-left bundle-branch block. In a few it became biphasic.

(5) In 2 patients transient left bundle-branch block occurred both spontaneously and by pacing from the right ventricle. The QRS morphology was similar in these two circumstances (Fig. 5).

Discussion

Cabrera and Gaxiola (1966) found that most patients with left bundle-branch block have anatomical left ventricular hypertrophy at necropsy. Similarly, Sodi-Pallares et al. (1964) found left ventricular hypertrophy in the great majority of patients with incomplete left bundle-branch block. However, our study was purely electrocardiographic. In a parallel way, through necropsy studies have been carried out in order to establish the diagnosis of myocardial infarction in cases of left bundle-branch block, Luy et al. (1973) studied the electrocardiographic changes in the infarction pattern when intermittent left bundle-branch block occurred, and others have studied the same changes when left bundle-branch block was induced by right ventricular pacing in acute myocardial infarction (Castellanos et al., 1973). We think that our observation that QRS
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morphology is similar in spontaneously occurring and pacing-induced left bundle-branch block supports the validity of the latter studies, though we had only two such cases.

From our results we consider that the voltage criteria for the diagnosis of left ventricular hypertrophy can be applied to a certain extent when there is left bundle-branch block. There was an excellent correlation in some leads: in leads I, V5, and V6 the QRS voltage decreased, while in leads V1 and V2 it increased post-left bundle-branch block. The Sokolow index did not change, and the correlation between pre- and post-left bundle-branch block values was excellent. However, 7 of 26 patients had a Sokolow index which increased from less than to more than 35 mm with the onset of left bundle-branch block. On the other hand, only 1 of the 14 patients with a value exceeding 35 mm pre-left bundle-branch block did not have a similar high value post-left bundle-branch block. Thus, in left bundle-branch block a Sokolow index of 35 mm or more is very sensitive but not very specific for the electrocardiographic diagnosis of left ventricular hypertrophy. Neither QRS axis changes nor left axis deviation exceeding –30° post-left bundle-branch block were helpful in differentiating between patients with and without left ventricular hypertrophy before the development of the conduction disturbance.

References


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D V Cokkinos, J N Demopoulos, E T Heimonas, C Mallios, N Papazoglou and E M Vorides

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