Left Ventricular Dynamics in Complete Right Bundle-Branch Block with Left Axis Deviation of QRS

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SUMMARY

The time intervals defined by the simultaneous recording of an electrocardiogram, phonocardiogram, indirect carotid pulse curve, and apexcardiogram made it possible to compare the left ventricular (LV) dynamics in a group of 33 cases of complete right bundle-branch block with left axis deviation (RBBB-LAD) with two control groups, one consisting of 30 cases of complete right bundle-branch block without left axis deviation (RBBB without LAD) and the other of 30 cases of left axis deviation (LAD) alone.

The LV dynamics of RBBB-LAD differed from those of both control groups of RBBB without LAD and of LAD alone by a significantly late onset of LV ejection in the former, resulting from both late onset and slow rise of LV contraction. On the other hand, the delayed LV activity was not significantly different between the group of RBBB-LAD and a previously studied group of complete left bundle-branch block.

Normal limits for these intervals were looked for to make it possible to diagnose a late LV activity in the individual patient, and so provide a further argument in the discussion for an eventual cardiac pacing in doubtful cases.

Finally, it was not felt possible to use these intervals to try to localize the site of the conduction disturbance, whether in the bundle branch or in the ventricular wall itself.

Additional Indexing Words:
Bilateral bundle-branch block  Phonocardiography  Indirect carotid pulse curve
Apexcardiography  Electrocardiography

LEFTWARD deviation of the QRS axis in the presence of complete right bundle-branch block is not a rarity, as it was found in every third hospitalized patient with complete right bundle-branch block.1 This peculiar electrocardiographic pattern, although presented in the publications of Mahaim in 19312 and of Wilson’s group in 1934,3 was widely recognized only after the advent of multiple precordial electrocardiography. It was considered after Lenègre’s report in 19554 to be an expression of bilateral bundle-branch block, on the basis of numerous histologic studies of the intraventricular septum.5-7 Further studies, whether histologic,8 clinical,9 10 experimental,11 12 or based on His bundle recordings13 14 came in support of this statement.

Nevertheless, if one is to avoid catheterization, the direct evidence of a left intraventricular block in such cases is difficult if not impossible to provide during life. In other respects, it was possible to demonstrate both with ease and reasonable accuracy the presence of a mechanical left ventricular delay

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Received June 29, 1970; accepted for publication July 17, 1970.
in complete left bundle-branch block by phonocardiographic and mechanocardiographic (indirect carotid pulse curve, apexcardiogram) recordings. 15

The aim of this study was to try to provide, by this simple untraumatizing method, the direct proof of a delayed left ventricular activity in cases of complete right bundle-branch block with left axis deviation of QRS, and so help in reaching a decision for eventual cardiac pacing in difficult cases.

**Methods**

The various time intervals of the left ventricular (LV) dynamics in three groups of patients were compared statistically. The groups consisted of (1) 33 patients whose electrocardiograms met the classical criteria for complete right bundle-branch block combined with frontal plane deviation of the QRS axis included between $-30$ and $-90^\circ$ (RBBB-LAD), (2) 30 patients, taken at random, who had complete right bundle-branch block without left axis deviation (RBBB without LAD), and (3) 30 patients, taken at random, who had LAD of QRS included between $-30$ and $-90^\circ$, but without electrocardiographic evidence of complete bundle-branch block, right or left. Details of age, sex, and etiologic factors may be found in Table 1. The main electrocardiographic data, namely, P-R interval, QRS interval, and QRS axis, in the three groups of patients, may be found in Table 2.

Simultaneous recording of an electrocardiogram, phonocardiogram, indirect carotid pulse curve, and when possible apexcardiogram permits the analysis of the following time intervals (fig. 1): (a) a Q-OCP interval (1 + 2 in fig. 1) from the onset of QRS to the onset of the carotid pulse (OCP); (b) an electromechanical interval (EMI; 1 in fig. 1) from the onset of QRS to the onset of the systolic upstroke of the apexcardiogram; (c) pre-ejection period (PEP; 2 in fig. 1) from the onset of the systolic upstroke of the apexcardiogram to the onset of the carotid pulse, obtained by substraction of the EMI from the Q-OCP interval; (d) systolic ejection time (SET; 3 in fig. 1) from the onset of the carotid pulse to its dicrotic notch; (e) isometric relaxation phase

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**Table 1**

Etiologic Characteristics of the Three Groups of Cases: Right Bundle-Branch Block With Left Axis Deviations (RBBB-LAD), Right Bundle-Branch Block Without Left Axis Deviation (RBBB Without LAD), and Left Axis Deviation Alone (lone LAD)

<table>
<thead>
<tr>
<th></th>
<th>RBBB-LAD (33 cases)</th>
<th>RBBB without LAD (30 cases)</th>
<th>Lone LAD (30 cases)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td>49.8</td>
<td>35.4</td>
<td>44.3</td>
</tr>
<tr>
<td>Range</td>
<td>(11-75)</td>
<td>(8-75)</td>
<td>(11-76)</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>28</td>
<td>20</td>
<td>24</td>
</tr>
<tr>
<td>Female</td>
<td>5</td>
<td>10</td>
<td>6</td>
</tr>
<tr>
<td>Etiologic factors:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Idiopathic conduction disturbance</td>
<td>12</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>7</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Valvular heart disease</td>
<td>5</td>
<td>9</td>
<td>15</td>
</tr>
<tr>
<td>Congenital heart disease</td>
<td>6</td>
<td>14</td>
<td>3</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>3</td>
<td>4</td>
<td>7</td>
</tr>
</tbody>
</table>

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**Table 2**

Electrocardiograms in the Three Groups of Cases*

<table>
<thead>
<tr>
<th>Groups</th>
<th>P-R interval</th>
<th>QRS interval</th>
<th>QRS axis</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBBB-LAD</td>
<td>20 $\pm$ 3.3 (29)</td>
<td>14 $\pm$ 1.8 (33)</td>
<td>$-66.7 \pm 17$ (33)</td>
</tr>
<tr>
<td>RBBB without LAD</td>
<td>18 $\pm$ 3.6 (26)</td>
<td>13 $\pm$ 1.6 (30)</td>
<td>$+126 \pm 30$ (25)</td>
</tr>
<tr>
<td>LAD</td>
<td>18 $\pm$ 4 (29)</td>
<td>9 $\pm$ 1 (30)</td>
<td>$-39.7 \pm 12$ (29)</td>
</tr>
</tbody>
</table>

*For each item the figures refer from left to right to the mean value, standard deviation and number of cases. P-R and QRS intervals are expressed in hundredths of a second and QRS axes in degrees.
Diagram of sequence of left ventricular contraction and ejection (and end of right ventricular ejection) in the normal heart. L1 = electrocardiographic lead 1; CP = indirect carotid pulse; ACG = apexcardiogram; PCG = phonocardiogram; "0" is the lowest diastolic pressure and indicates mitral valve opening. Intervals are numbered:

1. Electromechanical interval; 2. pre-ejection period; 3. systolic ejection time; 4. isometric relaxation phase. The interval Q to the onset of the carotid pulse is the sum of 1 + 2.

(IRP; 4 in fig. 1) from the aortic component A2 of the second heart sound to the lowest diastolic point 0 of the apexcardiogram; and (f) A2-P2 interval between the aortic and pulmonary components of the second heart sound. Except for this last interval which results from asynchronism in the end of left and right ventricular ejections, all the other intervals are related to the various events of LV dynamics. For the present study the most important ones were the Q-OCP interval which marks the onset of LV ejection, the EMI which marks the onset of LV contraction, and the difference between these two intervals, the PEP which is a measure of the time necessary for the LV contraction to overcome the aortic diastolic pressure, that is, the speed of the LV contraction rise. All the values studied were an average of measurements made from four cardiac cycles, after they had been corrected for cardiac rate by dividing the measured value by the square root of the preceding R-R interval of the electrocardiogram. The curves were recorded with photographic four-channel or eight-channel Hellige Multicardiotest, at a speed of 50 mm/sec.

Results

Comparison of the Two Groups of Complete Right Bundle-Branch Block, One With and One Without Left Axis Deviation (RBBB-LAD vs. RBBB Without LAD; fig. 2, table 3)

By comparison with the cases of RBBB without LAD, the cases of RBBB-LAD showed significantly longer overall Q-OCP interval (P < 0.001), EMI (P < 0.001), and PEP (P < 0.01), while both the systolic ejection time and the isometric relaxation phase were not significantly different. This means that LV ejection started later in RBBB-LAD than in RBBB without LAD, and this delay resulted from both a late onset and a slow rise of LV contraction. The same delay was found in the end of LV ejection (decrease of the A2-P2 interval).

Comparison of the Two Groups of Left Axis Deviation, One With and One Without Complete Right Bundle-Branch Block (RBBB-LAD vs. LAD; fig. 3, table 3)

By comparison with the cases of lone LAD, the cases of RBBB-LAD showed significantly longer overall Q-OCP interval (P < 0.001), EMI (P < 0.001), and PEP (P < 0.001). In the latter group the systolic ejection time was significantly shorter, while the isometric relaxation phase and the A2-P2 interval were not significantly different. Here again, LV ejection started later in RBBB-LAD than in lone LAD, and this resulted from both a late onset and a slow rise of LV contraction. However, these two groups were not strictly comparable, especially in what concerned the average QRS duration and left axis deviation which were both more pronounced in RBBB-LAD than in lone LAD, respectively 0.14 ± 0.018 sec and 0.09 ± 0.01 sec (P < 0.001) and −66.7° ± 17 and −39.7° ± 12 (P < 0.005) (table 2).

Comments

Since Mahaim's report in 1931, many studies have shown that the electrocardio-
Table 3

Measurements* of the Various Intervals in the Three Groups of Cases Studied Herein and in Two Previously Published Groups of Complete Left Bundle-Branch Block (LBBB) and Normal Conduction (NC)

<table>
<thead>
<tr>
<th>Intervals</th>
<th>RBBB-LAD (33 cases)</th>
<th>RBBB without LAD (30 cases)</th>
<th>Lone LAD (30 cases)</th>
<th>LBBB (30 cases)</th>
<th>NC (42 cases)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Q-OCP</td>
<td>177 ± 34</td>
<td>144 ± 25</td>
<td>135 ± 18</td>
<td>192 ± 35</td>
<td>139 ± 21</td>
</tr>
<tr>
<td>EMI</td>
<td>53 ± 18</td>
<td>33 ± 24</td>
<td>38 ± 8</td>
<td>57 ± 19</td>
<td>32 ± 13</td>
</tr>
<tr>
<td>PEP</td>
<td>124 ± 38</td>
<td>111 ± 35</td>
<td>97 ± 20</td>
<td>134 ± 35</td>
<td>107 ± 21</td>
</tr>
<tr>
<td>SET</td>
<td>284 ± 30</td>
<td>298 ± 25</td>
<td>307 ± 28</td>
<td>309 ± 45</td>
<td>306 ± 39</td>
</tr>
<tr>
<td>IRP</td>
<td>123 ± 38</td>
<td>111 ± 29</td>
<td>123 ± 30</td>
<td>135 ± 31</td>
<td>105 ± 31</td>
</tr>
<tr>
<td>A2-P2 I</td>
<td>35 ± 26</td>
<td>49 ± 22</td>
<td>28 ± 13</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

*Measurements of the various intervals (means and standard deviations) in milliseconds. All values are average measurements made from four cardiac cycles after they had been corrected for heart rate by dividing the measured value by the square root of the preceding R-R interval of the electrocardiogram.

Abbreviations: Same as text and table for RBBB-LAD; RBBB without LAD, lone LAD. LBBB = left bundle-branch block; NC = normal conduction; Q-OCP = Q onset to onset of the carotid pulse; EMI = electromechanical interval; PEP = pre-ejection period; SET = systolic ejection time; IRP = isometric relaxation phase; A2-P2 I = interval between aortic and pulmonary components of the second heart sound.

Figure 2

Comparison between the various intervals in right bundle-branch block with left axis deviation (RBBB-LAD) and in right bundle-branch block without left axis deviation (RBBB without LAD). The numbers are average values expressed in milliseconds; for abbreviations see table 3. NS = not significant.

graphic pattern of RBBB-LAD coincided with interruptive histologic lesions of both bundle branches.4-8 In their recent monograph on the subject, Blondeau and Lenègre4 found total or subtotal destruction of the right bundle branch in 38 of 43 cases together with major
lesions on all (15 cases) or part (17 cases) of the left bundle-branch fibers. The common occurrence in these patients of complete heart block, of Stokes-Adams attacks, or sudden death\(^1,9,10\) brings further support to these histologic findings.

A new approach was afforded by His bundle recordings which demonstrated the LV delay in these cases\(^18,14\) but this required catheterization and the use of very sophisticated material. A similar result was attained with ease, reasonable accuracy, and a minimum of nuisance to the patients by analysis of phonocardiographic and mechanocardiographic tracings. It was thus pointed out that the LV dynamics of RBBB-LAD differed from those of both RBBB without LAD and of lone LAD by a late onset of LV ejection in RBBB-LAD, resulting from both a late onset and a slow rise of LV contraction. Furthermore, when these results were compared with those of a previous study on chronic complete left bundle-branch block (LBBB),\(^15\) no significant difference in the LV dynamics was found between the cases of RBBB-LAD and those of LBBB on the one hand (fig. 4, table 3) and between the cases of RBBB without LAD and those of "normal" conduction on the other (fig. 5, table 3). In other words, the delayed LV activity in the cases of RBBB-LAD probably resulted from the superaddition of a left intraventricular block to the complete RBBB. The late LV activity could not be attributed to the RBBB itself. The delayed LV dynamics of RBBB-LAD in comparison with those of lone LAD were probably due to the presence in the former group of more cases with a more severe degree of left intraventricular block, parallel with the degree of left axis deviation, \(-66^\circ\) and \(-39^\circ\), respectively.

But these are overall results, and if they are to have a practical value, for example in helping to decide on the advisability of cardiac pacing in doubtful cases, their diagnostic value has to be assessed in the individual patient.

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**Figure 3**

Comparison between the various intervals in right bundle-branch block with left axis deviation (RBBB-LAD) and in left axis deviation alone (lone LAD). Same presentation and abbreviations as for figure 2.
Prolongation of the Q-OCP interval may be considered as the easiest criterion to demonstrate disturbed left intraventricular conduction. In our previous study on chronic
LBBB, the upper "normal" limit for the corrected Q-OCP interval was tentatively set at 175 msec, as this limit included 95% of the 42 cases without electrocardiographic evidence of bundle-branch block. When considered alone this interval would detect late LV ejection in only 33% of the 33 cases of RBBB-LAD. But because a prolonged Q-OCP interval may result from a lone increase of the PEP (slow LV contraction rise or high diastolic hypertension), it may occur from other causes than left intraventricular block, namely, severe myocardial disease, and severe diastolic hypertension. For this reason, a prolonged EMI (delayed onset of LV contraction) was suggested as a more specific index of left intraventricular block.17 But an apexcardiogram is not always recordable, and indeed it was obtained in only 24 of our 33 cases of RBBB-LAD. However, in the same study on LBBB, the upper "normal" limit for the corrected EMI was tentatively set at 52 msec, as this limit included 95% of the 42 patients with "normal" conduction. When considered alone, this interval would detect a late LV contraction in 37% of 24 cases of RBBB-LAD. Combination of both criteria made it possible to detect late LV activity in 45% of 24 cases of RBBB-LAD.

These results appear not to be in keeping with the results of the overall study of RBBB-LAD and with those of the histologic study reported in the monograph of Blondeau and Lenègre.1 These investigators found destruction of part or all of the left bundle-branch fibers in 74% of 43 cases of RBBB-LAD. This discrepancy may be due to the fact that our upper "normal" limits are too high. This was particularly so for the Q-OCP interval which, when set at 175 msec, would have left undiagnosed an otherwise obvious case of intermittent complete LBBB in which this interval had increased from 95 msec in normal conduction to 166 msec in the presence of the complete LBBB pattern. This is why more sensitive upper "normal" limits were looked for and were tentatively set at levels slightly above the respective average values (fig. 5, table 3), that is, 149 msec for the corrected Q-OCP interval and 39 msec for the corrected EMI. These limits appeared to discriminate best between normal and abnormal conduction, though at the expense of many false positives. If they are accepted, one may expect three types of answer: (1) delayed LV activity (corrected Q-OCP interval > 175 msec or corrected EMI > 52 msec or both), (2) undelayed LV activity (corrected Q-OCP interval < 149 msec and corrected EMI < 39 msec) and (3) possibly or probably delayed LV activity (corrected Q-OCP interval included between 149 and 175 msec, or corrected EMI included between 39 and 52 msec or both). These criteria would thus make it possible to subdivide the 24 cases of RBBB-LAD into cases exhibiting an obvious LV delay (45%), cases with a possible or probable LV delay (46%), and cases without LV delay (9%).

In our previous paper on LBBB, it was assumed that normal EMI (with long PEP) meant arborization block, whereas prolonged EMI (with normal PEP) meant bundle-branch block. This assumption presupposes a normal conduction in the right bundle branch. In the latter case, a prolonged EMI probably results from the delay of the activation wave in crossing through the interventricular septum, from the normal right bundle branch to the left ventricle. When the right bundle branch is interrupted as well, as is known to occur in almost all cases of RBBB-LAD, the site of onset of ventricular depolarization remains conjectural, and a prolonged EMI may mean only a difficult spread of the activation wave from the incompletely blocked left bundle branch to the LV myocardium (arborization block). Further, a normal EMI does not necessarily exclude a bundle-branch lesion, if the reference point—onset of ventricular depolarization—is delayed. For these reasons, it was not felt possible to use these time intervals in this study, for localizing the site of the left intraventricular block.

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Circulation. 1970;42:797-804
doi: 10.1161/01.CIR.42.5.797

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