Left Ventricular Activation Time in Left Ventricular Hypertrophy and in Left Bundle-Branch Block

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Simultaneous tracings of right and left ventricular leads were taken in 6 dogs with experimentally produced left bundle-branch block, and in 30 clinical cases of left ventricular hypertrophy. In the experimental study it was found that the initial phase of the QRS complex in the right epicardial lead did not change its configuration or its duration after the production of left bundle-branch block. This observation offers a new concept of the septal and left ventricular activation in left bundle-branch block. It was concluded that a considerable portion of the left ventricle is activated normally in some cases of left bundle-branch block.

A METHOD of measurement of left ventricular activation time in normal subjects was discussed in a previous paper. From a simultaneous recording of right and left precordial leads, it was found that the negativity recorded as an S wave in lead V1 was a reciprocal transmission of the positive potentials created by the activation of the left ventricular wall. Furthermore, it was concluded that the time interval from the onset of the initial deflection to the nadir of the S wave in V1 represents a better index of the left ventricular activation time than does the usually measured interval from the onset of the initial deflection (q wave) to the peak of the R wave in V7.

While there is a marked change in the configuration of the initial phase of the left precordial complexes in clinical cases of left bundle-branch block, the initial phase of the right precordial complexes remains essentially unchanged. Although the configuration of the latter is similar to that found in normal subjects, the mechanism of its production is thought to be different. In normal hearts, according to a well known concept, the septal activation from left to right causes the inscription of an r wave in both right ventricular cavity and right ventricular leads.* Upon activation of the right ventricular wall, further extension of the r wave occurs in the right ventricular leads. On the other hand, in left bundle-branch block, the initial activation of the septum from right to left causes a negativity in the right ventricular cavity. Nevertheless, despite this negativity, the right ventricular leads usually reveal r waves as seen in normal tracings. This discrepancy has been attributed to the early activation of the apical portion of the right ventricle.

Following the initial positive deflection in right ventricular leads an S wave is inscribed both in normal tracings and in left bundle-branch block. This S wave is caused by activation of the free wall of the left ventricle in normal hearts and is attributed to the abnormally slow activation of the septum from right to left in cases of left bundle-branch block. The activation of the free wall of the left ventricle in left bundle-branch block would be expected to produce further negativity and deepening of the S wave in the right ventricular leads. However, according to the generally accepted concept, activation of the left ventricular wall is manifested by the ascending limb of the S wave. But no adequate explanation is given for this.

*The term "ventricular leads" will be used throughout this paper to include both epicardial and precordial leads.
Experimental and clinical studies were undertaken to reconsider the mechanism of septal and left ventricular activation in left bundle-branch block. An attempt was also made to analyze the rS complex in right epicardial leads in dogs with experimental left bundle-branch block and in precordial leads in human patients with left ventricular hypertrophy with and without left bundle-branch block.

**Experimental Study**

**Materials and Methods**

Six dogs each weighing from 15 to 20 Kg, were used. The animals were anesthetized with sodium pentobarbital (25 to 35 mg. per Kg. of body weight). Maximum exposure of the heart was accomplished by a transverse sternal-splitting incision. Respiration was maintained with a constant flow of 100 per cent oxygen by means of a positive pressure apparatus. The pericardial fat was removed atraumatically. A small window was made in the pericardium overlying each atrium, and a small cotton-tipped intracavity electrode was inserted transatrially into each ventricular cavity. Another small cotton-tipped electrode was used to explore the entire surface of the heart through the intact pericardium in order to find complexes similar to V1 and V7 in human tracings. The rS type of complex typical of V1 could be found on the right ventricle near the septum and a qRs or Rs type of complex similar to V7 was obtained from the anterolateral wall of the left ventricle. The positions of the epicardial electrodes were similar to those of V1 and V7 in human tracings. Small saline-soaked cotton electrodes were gently sewn onto the epicardium through a small window in the pericardium. A control electrocardiogram was taken simultaneously from the 4 electrodes in the proper locations with a paper speed of 100 mm. per second, with a Sanborn 4-channel direct-writer. For more accurate meas-
measurement of the rS duration in right epicardial leads, a Sanborn Twin-Beam Cardiette was used with a paper speed of 75 mm. per second. Left bundle-branch block was produced by inserting an iridectomy knife through the posterior portion of the left ventricle near the atrioventricular groove and severing the left bundle. The diagnosis of left bundle-branch block was based on the finding of an initial positive deflection in the left intraventricular cavity lead in the presence of a sinus rhythm.

Results

In all instances, the control tracings revealed the simultaneous onset of the initial deflections in all 4 leads (figs. 1 and 2).

Right Epicardial Complex. In the control tracings, the right epicardial complex was of the rS type in all instances (fig. 3). Its average duration from the onset of the r to the nadir of the S wave was 0.021 second. After the production of left bundle-branch block, the rS complexes persisted, and the interval from the onset of r to the nadir of S was unchanged. The only marked change in the right epicardial complex after the production of left bundle-branch block was slurring and widening of the ascending limb of the S wave, which accounts for the increased duration of the entire QRS complex. The average QRS duration was 0.046 second in control tracings and 0.090 second after production of left bundle-branch block.

Left Epicardial Complex. In the 6 animals, 4 had an Rs type of complex in the control tracings (fig. 1). In the other 2 animals, the complexes were of the QRS type (fig. 2). After left bundle-branch block, however, all the complexes were of the Rs type with slurring of the R wave.

Left Intraventricular Cavity Complex. All but 1 showed an rS type of complex in the control tracings, the exception being a QS complex. After the production of left bundle-branch block, all the complexes were of the QS type.

Left Intraventricular Cavity Complex. All animals showed a QS type of complex in the control tracings. These became RS in type after production of left bundle block (figs. 1 and 2). In 4 instances there was considerable delay in the inscription of the initial deflection (fig. 1).

The Relationship of the Nadir of the S Wave in Right Epicardial and the Peak of the R Wave in Left Epicardial Leads. In the control tracings, the nadir of the S wave in the right epicardial leads always occurred simultaneously with or later than the peak of the R wave in left epicardial leads. This relationship changed after the production of left bundle-branch block, when the nadir of the S wave in right epicardial leads always occurred earlier than the peak of the R wave in left epicardial leads.

Clinical Study

Materials and Methods

Thirty hypertensive patients with roentgenographic evidence of left ventricular enlargement were selected for this study. The duration of hypertension ranged from 4 to 15 years and the blood pressure varied from 160/110 to 240/140 mm. Hg, with a mean of 190/115 mm. Hg. All patients had a history of one or more episodes of congestive failure and were receiving digitalis.
preparations at the time of study. Patients with evidence of old myocardial infarction were included, but those with acute myocardial infarction were eliminated from this series. Patients with possible right ventricular enlargement were also excluded. The age of the patients ranged from 28 to 65 years with a mean of 52 years.

In all patients simultaneous recordings were made of lead V₁ and of lead V₇, with each lead from V₅ to V₇, as described in the previous paper.¹ For reasons previously discussed¹ most of the measurements pertinent to this study were those of the complexes of lead V₁ and V₇.

RESULTS

Onset and Configuration of the Initial Deflections. Of the 30 tracings studied, 25 showed the onset of the initial deflection to be simultaneous in both V₁ and V₇ (figs. 4, 5, and 6). In the remaining 5 cases, there was a delay in the onset of the initial deflection in V₇ in comparison to that of V₁ (fig. 7). In no instance did the onset of the initial deflection occur later in V₁ than in V₇. This indicates that lead V₁ is a more accurate index than V₇ for determining the onset of the initial deflection.

In V₁ the initial deflection was positive in 23 cases (table 1). In the left precordial leads, including V₅, V₆, and V₇, the initial deflection was positive in 9 instances.

Time Relationship and Duration of Complexes. In 23 of the 30 cases, the nadir of S in V₁ occurred earlier than peak of R in V₇ (figs. 6, 7, and 8). This finding was the reverse of that in normal cases. In these 23 instances, the relationship of S in V₁ and R in V₇ was exactly the same as that in experimentally produced left bundle-branch block in dogs. In 4 of the remaining 7 cases, the nadir of S in V₁ occurred later than the peak of R in V₇ (figs. 4 and 5), and in the last 3 instances the nadir of SV₁ and the peak of RV₇ were simultaneous.

The time intervals from the onset of the initial deflection to the nadir of the S wave in V₁ varied from 0.045 to 0.080 second, with a mean of 0.057 second. The same interval in normal individuals ranged from 0.035 to 0.055 second, with a mean of 0.048 second. In 25 tracings the time interval from the onset of the initial deflection to the peak of
TABLE 1.—Electrocardiographic Observations in
Thirty Cases of Left Ventricular Hypertrophy

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Type of QRS in V1</th>
<th>Type of QRS in V7</th>
<th>Duration from initial deflection to peak of V1</th>
<th>Duration from onset of initial deflection to peak of V7</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>rS</td>
<td>qR</td>
<td>0.065</td>
<td>0.055</td>
</tr>
<tr>
<td>2</td>
<td>rS</td>
<td>qRS</td>
<td>0.055</td>
<td>0.055</td>
</tr>
<tr>
<td>3</td>
<td>rS</td>
<td>qR</td>
<td>0.065</td>
<td>0.060</td>
</tr>
<tr>
<td>4</td>
<td>rS</td>
<td>rsR</td>
<td>0.065</td>
<td>0.070</td>
</tr>
<tr>
<td>5</td>
<td>rS</td>
<td>qR</td>
<td>0.055</td>
<td>0.080</td>
</tr>
<tr>
<td>6</td>
<td>rS</td>
<td>qR</td>
<td>0.065</td>
<td>0.080</td>
</tr>
<tr>
<td>7</td>
<td>rS</td>
<td>qR</td>
<td>0.060</td>
<td>0.080</td>
</tr>
<tr>
<td>8</td>
<td>rS</td>
<td>R</td>
<td>0.060</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>rS</td>
<td>qR</td>
<td>0.050</td>
<td>0.080</td>
</tr>
<tr>
<td>10</td>
<td>rS</td>
<td>qR</td>
<td>0.065</td>
<td>0.065</td>
</tr>
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<td>11</td>
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<td>qR</td>
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<td>qR</td>
<td>0.050</td>
<td>0.065</td>
</tr>
<tr>
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<td>qR</td>
<td>0.055</td>
<td>0.060</td>
</tr>
<tr>
<td>14</td>
<td>rS</td>
<td>qR</td>
<td>0.055</td>
<td>0.055</td>
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<tr>
<td>16</td>
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<tr>
<td>17</td>
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<td></td>
</tr>
<tr>
<td>18</td>
<td>rS</td>
<td>R</td>
<td>0.050</td>
<td></td>
</tr>
<tr>
<td>19</td>
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<td>qR</td>
<td>0.045</td>
<td>0.055</td>
</tr>
<tr>
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<td>22</td>
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<td>qR</td>
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</tr>
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<td>qrsR</td>
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</tr>
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<td>qRs</td>
<td>0.060</td>
<td></td>
</tr>
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<td>qR</td>
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</tr>
<tr>
<td>26</td>
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<td>rsrS</td>
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<td>27</td>
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<td>R</td>
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<td>0.090</td>
</tr>
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<td>rsR</td>
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<td>R</td>
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</tr>
<tr>
<td>30</td>
<td>QS</td>
<td>R</td>
<td>0.045</td>
<td>0.055</td>
</tr>
</tbody>
</table>

*In 5 instances there was a delay in the inscription of the initial deflection in lead V5, which accounts for the blank spaces in the last column.

The R wave in V7 was from 0.055 to 0.14 second, with a mean of 0.070 second. In 5 instances there was a delay in the inscription of the initial deflection in V7 and these were not included in this analysis, since no definite moment of onset could be determined. This mean duration of 0.070 second is markedly different from the normal mean of 0.042 second. It is also evident that the duration of qR or R in V7 exceeds the duration of rS, qrS, or QS in V1 in this series, which was exactly opposite from what was found in normal subjects.

The entire width of the QRS complex varied from 0.10 to 0.185 second, with a mean of 0.12 second. The same value in normal individuals ranged from 0.095 second, with a mean of 0.079 second.

**Discussion**

In both experimental and clinical left bundle-branch block, the definitive diagnosis is believed to rest upon the findings of an initial positive deflection in the left intraventricular cavity. However, even with this method there are often differences in the configuration and onset of the positive deflection. As seen in the present experimental study, there is sometimes a considerable delay in the inscription of this initial positive deflection when it is compared to other simultaneous leads. The R wave in the left intraventricular cavity lead is preceded by an isoelectric period in these instances (figs. 1 and 2). What this isoelectric period represents is unknown. It may be that 2 equal potentials arising from opposite sides of the septum activate the septum in transverse fashion, and thus...
neutralize each other. Another possible cause of the isoelectric period might be that potentials arising in the septum are not strong enough to register. Finally, the vector of these potentials may be at such an angle to the electrode in the left ventricular cavity that no deflection can be recorded.

Using a cathode-ray oscillographic method, Kennamer and his associates found a similar discrepancy between the onset of the initial deflection in the left epicardial leads and that of left ventricular cavity potentials in experimental left bundle-branch block. These workers suggested an abnormally rapid transmission of the impulse to the epicardial surface of the left ventricle via an unknown pathway in cases of left bundle-branch block. In any event, it is suggested that the arrival of the impulse at the epicardial surface of the homolateral ventricle is not delayed when bundle-branch block is produced in dogs. The anatomic structure of the left bundle is such that a certain type of left bundle-branch block may involve only a portion of the left bundle. According to Sodi-Pallares and his associates, the delay in the activation of the septum in some cases of left bundle-branch block can be limited to a very small portion of the septum. This small diseased portion of the septum could produce a prolonged septal activation up to 0.08 second. However, the remaining portion of the septum is not affected by the block and is activated normally. This observation is supported by the findings of a qRS type of complex in the left ventricular cavity in experimental left bundle-branch block. In such an instance the greater potentials produced by the activation of the portion of the septum which is normally depolarized from left to right overwhelm the lesser potentials of this abnormally depolarized portion of the septum. This produces an initial negativity in the left ventricular cavity. Wener and others demonstrated initial negativity in the left ventricular cavity in human cases of left bundle-branch block by means of esophageal leads. Smith and his associates found both normal and delayed arrival of the impulse at the epicardial surface of the homolateral ventricle after the experimental production of "segmental bundle-branch block" in dogs.

These observations seem to cast doubt upon the classic concept that in left bundle-branch block, left ventricular depolarization does not start until completion of the septal depolarization from right to left. In addition, they suggest that clinical bundle-branch block is partial or incomplete in nature.

In a preceding paper, the nature and significance of the rS complex in right precordial leads were analyzed. It was concluded that this complex, measured from the onset of the initial deflection to the nadir of the S wave in V1, represents an accurate measurement of the left ventricular activation time in normal subjects. This conclusion also appears to be true of epicardial tracings taken from the right ventricles of normal dogs. Although there is a distinct difference between epicardial and precordial leads, the same intervals from the onset of the initial deflection to the nadir of the S wave in right epicardial leads presumably represent the left ventricular ac-
left ventricular activation time in dogs, since this interval is always identical with or greater than the interval from the onset of the initial deflection to the peak of the R wave in left epicardial leads.* That this interval showed no change after experimental production of left bundle-branch block in dogs (fig. 3) indicates that the left ventricular activation is still manifested by the same interval.

A clinical example of this situation is shown in figure 9, in which the patient developed left bundle-branch block as the result of a myocardial infarction. The block disappeared in about 1 month. In this instance, the time interval from the onset of the initial deflection to the nadir of the S wave in lead V1 remained identical in tracings obtained before and during left bundle-branch block and after its disappearance.

In 30 clinical cases of left ventricular hypertrophy 9 showed typical left bundle-branch block (figs. 7 and 8). The duration of the interval from the onset of the initial deflection to the nadir of the S wave in lead V1 was measured in all 30 tracings. It ranged from 0.045 to 0.080 second with a mean of 0.057 second (table 1). This duration is significantly longer than in normal subjects, in which the range was from 0.035 to 0.055 second, with a mean of 0.048 second. In these clinical cases without right ventricular hypertrophy, the prolonged duration of rS in lead V1 may be due to a hypertrophied left ventricle, or a diseased septum or both. The possibility that a diseased septum causes the prolonged rS interval was ruled out in the present experimental studies. The only possible cause for the increased time interval of rS is therefore hypertrophy of the left ventricle. Since the activation of the left ventricle appears to be manifested by the rS interval in lead V1, not only in normal subjects but also in cases of left bundle-branch block, it follows that there is no delay in the onset of the left ventricular activation in the latter instances. This new concept differs markedly from the classic one.12 The following alternative suggestions are offered to explain the mechanism of septal and ventricular depolarization in left bundle-branch block.

The septum may be activated from either direction or it may be activated simultaneously from right and left, depending upon the location and severity of the block in the left bundle. Whatever the direction of the initial septal activation may be, there is no delay in the arrival of the impulse at the subendocardial aspect of either ventricle. Therefore, depolarization of the free wall of the ventricles begins normally. At the same time, an abnormal potential occurs in the septum at the site of the block. Because of, the location of the electrode in right ventricular leads, both epicardial and precordial leads register only the overwhelming potentials created by the nor-

*The epicardial leads over the left ventricle of dogs appear to show a constant s wave. Which region of the heart causes this s wave is not clear. It may be the right ventricle near the pulmonary conus. The duration of rS in right epicardial leads of dogs seems to represent left ventricular activation time, but does not necessarily represent the activation time of the last portion of the heart to be depolarized.
nally activated ventricles, and the septal potentials are masked. The potentials derived from forces created by the activation of the free wall of the left ventricle produce a normal appearance of the Rs wave in right ventricular leads. In contrast, left ventricular leads register the combined forces created by the normally activated ventricles and the abnormally activated diseased portion of the septum. Thus there occurs a slurring in the upstroke of the R wave. There is also a notch of the ascending limb of the R wave indicating the completion of the activation in the undiseased portion of the left ventricle. Simultaneous with the notch of the ascending limb of the R wave in left ventricular leads, the S wave in right ventricular leads inscribes its nadir as seen in normal tracings. The abnormal part of the septum then completes its activation and is followed by the activation of the portion of the left ventricle which is supplied by the diseased left bundle. These last forces appear to be directed toward the anterior portion of the heart\textsuperscript{10, 13} rather than posteriorly, as in the normal heart. For this reason, the right ventricular leads register relatively positive potentials in the ascending limb of the S wave and a plateau in the R wave in left ventricular leads (fig. 8). This last force directed toward the anterior portion of the heart is often seen as a delayed positivity at the transitional area of clinical precordial leads (figs. 7 and 8).

This new concept, that the left ventricle begins its activation in a normal manner in cases of left bundle-branch block, is supported by the work of Braunwald and Morrow.\textsuperscript{14} These investigators simultaneously catheterized both left and right ventricles in patients with left bundle-branch block. They found that the onset of both ventricular pressure curves occurred at the same instant. This observation is not compatible with the usual concept of left bundle-branch block, which postulates complete interruption of conduction in the left bundle, and tends to substantiate the new concept discussed above.

With this new concept it is possible to explain certain findings that have not been well understood in the past, for example, the occurrence of a pure R wave in the left ventricular cavity lead in some cases of left bundle-branch block.\textsuperscript{5} According to the classic theory,\textsuperscript{12} the activation of the left ventricle always follows that of the septum. The inscription of an S wave in the left ventricular cavity lead is then expected, since the potentials are directed from within outward during the depolarization of the left ventricle and therefore register negative potentials. The absence of the S wave may be explained by the new concept as follows. Left ventricular activation begins in a normal manner. The activation of the blocked portion of the septum also starts about the same instant. Probably because of the location of the electrode in the left ventricular cavity, the septal potentials predominate in the cavity. In other words, the potentials created by the activation of the left ventricle do not produce any recordable negativity in the cavity, since its potentials are hidden in the positive deflection caused by the abnormally depolarized diseased septum. When the activation of the diseased septum takes more time than the normally activated left ventricle, there will be a continuous inscription of an R wave in the left ventricular cavity until the completion of activation of the diseased septum. The activation process then arrives at the portion of the left ventricular subendocardium that is supplied by the diseased portion of the left bundle. Normally, this potential associated with the depolarization of the blocked portion of the left ventricle would be recorded as an s wave in the cavity. However, if the blocked portion of the left ventricle is small, its potentials may be too weak to be recorded. Thus there may be no negativity inscribed as an s or S wave in some cases of left bundle-branch block. The size, location, and degree of severity of the block of the left bundle seem to be important in the inscription of a pure R wave in left ventricular cavity leads in some cases of left bundle-branch block.

The relationship between the nadir of the S wave in right ventricular leads and the peak of the R wave in left ventricular leads pre-
LEFT VENTRICULAR ACTIVATION TIME

Table 2.—Duration of QRS Intervals in Normal Group and in Left Ventricular Hypertrophy

<table>
<thead>
<tr>
<th>Duration of r in V1 (from the onset of the r to its peak)</th>
<th>Mean (sec.)</th>
<th>Standard deviation (sec.)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I*</td>
<td>0.050</td>
<td>0.005</td>
<td></td>
</tr>
<tr>
<td>Group II*</td>
<td>0.018</td>
<td>0.004</td>
<td></td>
</tr>
<tr>
<td>Duration of rS, qRS, and QS in V1 (from the onset of the initial deflection to the nadir of the S wave)</td>
<td>Group I</td>
<td>0.048</td>
<td>0.006</td>
</tr>
<tr>
<td>Group II</td>
<td>0.057</td>
<td>0.008</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Duration of qR and R in V7 (from the onset of the initial deflection to the peak of the R wave)</td>
<td>Group I</td>
<td>0.041</td>
<td>0.007</td>
</tr>
<tr>
<td>Group II</td>
<td>0.070</td>
<td>0.018</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Duration of the widest QRS complex</td>
<td>Group I</td>
<td>0.079</td>
<td>0.010</td>
</tr>
<tr>
<td>Group II</td>
<td>0.122</td>
<td>0.021</td>
<td></td>
</tr>
</tbody>
</table>

*Group I represents cases of normal individuals, and group II represents the present studies of left ventricular hypertrophy with and without left bundle-branch block.

Sents an interesting problem. In normal human subjects and in normal dogs the nadir of S in V1 always occurred simultaneously with or later than the peak of R in V7 (figs. 1 and 2). In 23 of 30 cases of left ventricular hypertrophy the relationship was reversed: the nadir of S in V1 occurred earlier than the peak of the R in V7. Rapaport and his associates also found this relationship between the nadir of the S wave in right precordial leads and the peak of the R wave in left precordial leads in cases of left ventricular hypertrophy. They attributed these changes to rotation of the heart in association with left ventricular hypertrophy. Experimentally, however, this reversed relationship between the nadir of the S wave in right epicardial lead and the peak of the R wave in left epicardial lead was seen only when left bundle-branch block was produced.

It is postulated that an interpretation of findings of experimental studies may be applied to the precordial tracings in cases of left bundle-branch block. As long as the nadir of the S wave in lead V1 coincides with or occurs later than the peak of the R wave in lead V7, the possibility of left bundle-branch block is most unlikely, regardless of the pattern or the duration of the entire QRS complex (fig. 4). Such instances may be classified as normal or left ventricular hypertrophy, depending upon the time interval from the onset of the initial deflection to the nadir of the S wave in V1. On the other hand, when the nadir of the S wave in lead V1 occurs earlier than the peak of the R wave in lead V7, left bundle-branch block should be strongly suspected. In such cases, if the interval between the onset of the initial deflection in V1 to the nadir of the S wave is increased, it is probable that left ventricular hypertrophy is also present.

To determine the statistical significance of the clinical studies, a t test was performed (table 2), comparing the mean of the time intervals from the onset of initial deflection to the nadir of S wave in V1 and the time interval from the onset of initial deflection to the peak of R wave in V7. The p value was less than 0.001. This indicates that the former time interval is always shorter than the latter. The result is the reverse of that found in normal hearts.

These findings appear to provide a significant aid in the clinical diagnosis of left bundle-branch block. A larger group of cases, especially with pathologic correlation, is desirable further to substantiate the findings of the present studies.

Summary and Conclusions

Simultaneous tracings of right and left ventricular leads were made in 6 dogs with ex-
experimentally produced left bundle-branch block and in 30 clinical cases of left ventricular hypertrophy.

In the dogs the duration from the onset of the initial deflection to the nadir of the S wave in the right epicardial lead did not change after the experimental production of left bundle-branch block. On the other hand, there was a marked change in the configuration and duration of the left epicardial complexes.

After experimental production of left bundle-branch block, there sometimes occurred a delay in the onset of the initial deflection in leads obtained from the left ventricular cavity. The possible causes for this delay were discussed.

In patients with clinical left ventricular hypertrophy, the interval from the onset of the initial deflection to the nadir of the S wave in V₁ was prolonged. This finding strongly suggests that this interval represents left ventricular activation time in cases of left ventricular hypertrophy, with or without associated left bundle-branch block.

The nadir of SV₁ occurred earlier than the peak of RV₇ only in cases of left bundle-branch block. This phenomenon appears to offer a useful criterion for the clinical diagnosis of left bundle-branch block.

It was concluded that in left bundle-branch block a considerable portion of the left ventricle begins its activation at the normal time and in a normal manner. Only that portion of the left ventricle supplied by the blocked part of the left bundle is depolarized later. This concept implies that complete functional block of the entire left bundle is doubtful, but that in practically all instances of left bundle-branch block, the block is incomplete.

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Summary in Interlingua

Electrocardiogrammas simultaneae a derivatione dextero- e sinistro-ventricular esseva obtente ab 6 canes con bloco de branca sinistre a production experimental e 30 casos clinic de hypertrophia sinistro-ventricular.

In le canes, le duration ab le declaration del deflexion initial usque al nadir del unda S in le derivation dextero-epicardial non se alte rava post le production experimental de bloco de branca sinistre. Del altere latere, il occurreva marcate alterationes in le configuration e le duration del complexos sinistro-epicardial.

Post le production experimental de bloco de branca sinistre, il occurreva in certe casos un retardo in le declaration del deflexion initial in derivationes ab le cavitate sinistro-ventricular. Le causas possibile de iste retardo es discutite.

In patienes con clinic hypertrophia sinistro-ventricular, le intervallo ab le declaration del deflexion initial usque al nadir del unda S in V₁ esseva prolongate. Iste facto es un forte indication que le intervallo in question representa tempore de activation sinistro-ventricular in casos de hypertrophia sinistro-ventricular, con o sin associate bloco de branca sinistre.

Le nadir de SV₁ occurreva plus tosto que le zenit de RV₇ solmente in casos de bloco de branca sinistre. Iste phenomeno pare representar un criterio de utilitate in le diagnose clinic de bloco de branca sinistre.

Esseva conclusite que in bloco de branca sinistre, un portion considerabile del ventriculo sinistre comencia su activation al tempore normal e in un manera normal. Dispolarisation retardate occurre solmente in le portion del ventriculo sinistre que es alimentate per le parte blocaite del branca sinistre. Isto significa implicitemente que bloco functional complete del branca sinistre total es dubitose e que in practically omne casos de bloco de branca sinistre il se tracta de un bloco incomplete.
LEFT VENTRICULAR ACTIVATION TIME

REFERENCES


The Eskimo is often cited as a race which has little atherosclerosis, despite a high-fat diet. However, the incidence of atherosclerosis among Eskimos is actually unknown and many of them do not consume a high-fat diet. Accordingly, the serum cholesterol and blood pressure of 842 Eskimo men, age 17 to 53, were studied. The mean cholesterol level of 214.4 mg. per 100 ml. was not unusual. There was considerable variation of the mean depending on geographic location, northern Eskimos having higher levels than those of the south. The average systolic pressure was 126.9; the diastolic was 74.3 mm. Hg. Measurements of blood pressure showed much less variation.

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Left Ventricular Activation Time in Left Ventricular Hypertrophy and in Left Bundle-Branch Block
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