Electrocardiographic Diagnosis of Myocardial Infarction in the Presence of Left Bundle-Branch Block

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It is generally accepted that most myocardial infarctions are obscured on the electrocardiogram by left bundle-branch block. The number of published cases in which this could be evaluated, however, is small, and the case reports are scattered. We have studied 30 cases of myocardial infarction with left bundle-branch block in which the location of the infarction could be determined with certainty, by autopsy, or by a previous electrocardiogram with normal intraventricular conduction. Twenty such published cases have also been collected. Electrocardiographic abnormalities have been correlated with infarctions in different locations. The possible specificity of these abnormalities is discussed.

The observation of Wilson and associates that "in the presence of left bundle branch block it is seldom possible to make a diagnosis of myocardial infarction on the basis of electrocardiographic findings alone" is still widely accepted. A number of cases in which a myocardial infarction could be recognized in the presence of left bundle-branch block (LBBB) have been reported, but these usually have been single case reports, with the exception of the groups of cases reported by Dressler et al. and by Sodi-Pallares and co-workers. Most of these have been diagnosed by Q waves in lead V6, by S-T segment or T-wave abnormalities, or by fortuitous normally conducted complexes.

Our review of 50 cases of myocardial infarction with LBBB leads us to believe that a myocardial infarction produces electrocardiographic changes almost as often in the presence of LBBB as it does with normal intraventricular conduction. In most instances the presence of the myocardial infarction is indicated by changes in the QRS complex. As would be expected, the abnormalities of the QRS complex indicating the presence of a myocardial infarction are not always the same in the presence of LBBB as they are with normal intraventricular conduction. QRS configurations similar to those seen in our cases in association with myocardial infarction are also present in published cases. Several of these QRS abnormalities have not previously been described as being associated with myocardial infarction in the presence of LBBB.

Materials and Methods

We have studied all cases with complete LBBB in our files from 1953 through June 1956. Complete LBBB was diagnosed by the usual criteria. The QRS complex should be of 0.12 second duration or longer, of sinus origin, and associated with a P-R interval of at least 0.12 second. The left precardial leads should have a broad, slurred, or notched R wave with an abnormally delayed "intrinsoid" deflection, similar complexes usually being present in lead I. There should be rS or QS complexes in lead V1 with a normal "intrinsoid" deflection. Cases in which the diagnosis of LBBB was questionable were excluded from this study, as were all cases of incomplete LBBB.

We have selected those cases in which the electrocardiogram could be evaluated by accepted criteria, such as autopsy data, a previous electrocardiogram with normal intraventricular conduction showing a myocardial infarction, or an electrocardiogram with intermittent LBBB. The electrocardiograms with intermittent LBBB had consecutive complexes with LBBB and normal intraventricular conduction. There were 30 cases of myocardial infarction with LBBB; the infarction was localized in 25 by autopsy, and in 5 by a previous electrocardiogram with normal intraventricular conduction. There were 13 cases with LBBB in which no gross infarct was found at autopsy. Five cases with intermittent LBBB were also studied.

In addition, the literature has been reviewed and all published cases of myocardial infarction with LBBB have been collected in which the location of the myocardial infarction was known—by autopsy or by normal intraventricular conduction. Inasmuch as the characteristic left preordial QRS complex is almost the "sine qua non" in the diagnosis of LBBB,

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only those cases in which left precordial leads are available were included. Twenty such cases were found. Autopsy data were available in 13 of these cases. In 4 other cases, the infarction could be localized by a previous electrocardiogram with normal intraventricular conduction, and in the remaining 3 cases the infarction was localized by an electrocardiogram with normal intraventricular conduction shortly after the infarction had occurred.

Results

QRS Complex

Comparison of the configurations of the QRS complexes within each group of cases, classified by location of infarction, reveals certain similarities.*

Anteroseptal Infarction. Of the 17 cases with anteroseptal infarction, 4 cases had a Q wave in lead V₆ (fig. 1B), similar to cases previously reported. Three of these 4 cases were autopsied and extensive anteroseptal infarction was demonstrated in each. Of the 17 cases with anteroseptal infarction, there were Q waves in lead I in 6 cases and in lead aV₆ in 10 cases. In our entire group of 43 cases, Q waves in leads I, aV₆, and V₆ were associated with anteroseptal infarction in every case except 1, in which there was a Q wave in lead aV₆ only. Patchy myocardial fibrosis was present in this case.

Seven cases, in which there was moderately extensive infarction in the anteroseptal and apical regions, had an rsR' in lead I, aV₆, or V₆ (figs. 2C and 3). The rsR' configuration in these leads usually consisted of a small r wave, a small s wave extending just below the isoelectric line, followed by a tall R'. In some leads, there was apparent respiratory variation between an rsR' and an R wave with a deep notch on the beginning of the upstroke that did not quite reach the isoelectric line. An rsR' was frequently present in the same electrocardiogram with a Q wave in lead I, aV₆ or V₆, or a notched S wave in lead V₄. The rsR' was always associated with an anteroseptal infarction in this group of cases.

Every one of the 9 cases in which there was infarction of the anteroseptal and apical region

* Mimeographed tables summarizing additional data on the cases are available from the authors on request.

Fig. 1. Electrocardiograms of a patient with an acute anteroseptal myocardial infarction during normal intraventricular conduction (A), transient LBBB (B), and return to normal conduction (C). Note the Q waves and abnormally elevated S-T segments in leads I, aV₆, V₆, and V₆, and the abnormal precordial R progression in B.
Fig. 2. Electrocardiograms of a patient with an old anterior infarction during normal conduction (A) and LBBB (B, C). Infarction of the septum and apex was also present at autopsy. Note the rsR' in leads I, aVL, V₃, and V₄ in C, and the notched S wave in lead V₁ in B and C.

had early notching of the S wave or QS deflection in the precordial lead just to the right of the transition zone (figs. 2C and 4B). This notching is wide and deep, and is present in complexes of relatively small amplitude. It is usually on the downstroke of the S wave or QS deflection. It is to be distinguished from the fine notching at the tip of S waves with large amplitude that is present in some right precordial complexes. This notching begins at approximately 0.03 second after the beginning of the QRS complex in most cases. In a few cases it occurred after a longer interval following the beginning of the QRS but still occurred during the early part of the S wave. In our entire group of 43 cases there was only 1 case in which a configuration like this in V₄ was not associated with an anteroseptal infarction. This case also had patchy myocardial fibrosis.

In the group of 17 cases with anteroseptal infarction, 13 had a decrease in the height of the precordial R wave, going from right to left (figs. 1B, 2C, 3, and 4B). This decrease ranged from 0.5 to 4 mm., averaging 1.6 mm. It was present in every case in which the anteroseptal infarction was extensive. It occurred in only 1 of the 13 cases without infarction at autopsy. In every case in which there was an anteroseptal infarction, 1 or more of the following was

Fig. 3. Electrocardiogram of a patient with an old infarction of the entire septum and adjacent anterior wall of the left ventricle during LBBB. Note the rsR' in lead V₃ and the initial notching of the S wave in lead aVF. Autopsied.

Fig. 4. Electrocardiograms of a patient with old anterior and posterior infarctions during normal conduction (A) and LBBB (B). Infarction of the septum and apex was also present at autopsy. Note the r' in lead aVR, the notched S wave in lead V₁, and the Q wave in leads V₂ and V₆ in B.
Fig. 5. Electrocardiogram of a patient with an old posterior infarction during LBBB. Note the notched R wave in lead aVF. Autopsied.

present: a Q wave in lead I, aV₃, or V₆; an rsR' in lead I, aV₅, or V₆; a notched S wave in lead V₄; or an abnormal precordial R progression.

Anterior Infarction. Of 3 cases with an infarction of the anterior wall of the left ventricle without septal involvement, 1 had an Rs in lead V₆. In the other 2 cases, in which the infarct was anterolateral, there was a decrease of 1 mm. in the height of the precordial R wave, going from right to left.

Posterior Infarction. Nine of the 10 cases with a posterior infarction had a notched R wave or an R' in lead aVF (figs. 4B and 5) and 8 of the 10 cases had a notched R wave or an R' in lead III. A notched R wave in lead aVF was present in 4 other cases in which a posterior infarction was not present. Patchy myocardial fibrosis was present in 2 of these cases. Patchy posterolateral fibrosis was present in a third case. In the fourth case, there was an old sub-endocardial infarct of the septum and lateral wall, and a vertical electrical axis.

Septal Infarction. Five cases had an infarction that predominantly involved the septum (figs. 3, 6B, and 7B). Each of these had initial notching of the S wave in lead aVF. In each of these cases there was a large S wave or QS deflection in lead aVF, with a small upward deflection during the early part of the S wave almost reaching the baseline. A complex similar to this was present in only 1 case without a septal infarct.

Other Cases. In our entire group of 43 cases, there were 5 cases in which QRS complexes similar to the ones described above were not associated with a transmural myocardial infarction. Gross patchy myocardial fibrosis was present in 3 of these 5 cases, and a subendocardial infarct and vertical electric axis were present in the fourth case. This case was the only one in the entire group with a vertical electric axis, all other cases having a horizontal axis. Neither infarction nor fibrosis was found in the fifth case.

A review of published cases of myocardial infarction with LBBB reveals most of them to be associated with QRS complexes similar to those described above. Twenty cases were found in which the location of the infarction was definitely known, by autopsy or by normal intraventricular conduction, and in which the electrocardiogram, with left precordial leads, was available for study.²⁻¹¹

There were 13 cases with anteroseptal infarction and 2 cases with anterior infarction. Thirteen of these 15 cases had Q waves in lead I, aV₃, or V₆, an rsr' in lead I or V₆, or a notched S wave in the precordial lead just to
the right of the transition zone, usually V_4. The other 2 cases not showing these QRS changes both had recent infarctions, one being 1 day old, the other being designated only as "recent."

In the group of 10 cases with posterior infarction there was a notched R wave or an R' in lead aV_F or III in 5 of the 10 cases. Of the other 5 cases, lead aV_F was not available in 3, the septum was intact in 1, and both anterior and posterior infarctions were present in 3.

A number of other cases in the literature of myocardial infarction with LBBB that lacked 1 of the criteria for inclusion in this group also had 1 or more of the following complexes—a notched S wave in the precordial lead just to the right of the transition zone, an rsR' in lead I, aV_L, or V_6, or a notched R wave or an R' in lead aV_F or III. We have not found these complexes in published examples of uncomplicated LBBB or in our nonautopsied cases in which myocardial infarction has not been suspected clinically.

The accuracy of these electrocardiographic features in indicating the presence of infarction in the combined group of 63 cases, 43 in our group and 20 from the literature, is shown in table 1. The first 4 electrocardiographic features, all associated with anteroseptal infarction, would appear to be a fairly reliable indication of anteroseptal infarction, in view of the number of cases available and the high degree of correlation. The electrocardiographic features apparently associated with posterior infarction may be causally related, or may be only coincidental. The correlation in the case of septal infarction is suggestive, but the num-
TABLE 1.—Electrocardiographic Features of Myocardial Infarction with Left Bundle-Branch Block

<table>
<thead>
<tr>
<th>Location of Infarction</th>
<th>Electrocardiogram</th>
<th>Accuracy in 63 cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extensive anteroseptal</td>
<td>Q I, aV&lt;sub&gt;L&lt;/sub&gt;, V&lt;sub&gt;6&lt;/sub&gt;</td>
<td>38/39</td>
</tr>
<tr>
<td>Moderately extensive anteroseptal</td>
<td>rsR'&lt; I, aV&lt;sub&gt;L&lt;/sub&gt;, V&lt;sub&gt;6&lt;/sub&gt;</td>
<td>10/11</td>
</tr>
<tr>
<td>Anteroseptal</td>
<td>Notched S &quot;V&lt;sub&gt;4&lt;/sub&gt;&quot;</td>
<td>19/21</td>
</tr>
<tr>
<td>Posterior</td>
<td>Abnormal precordial R progression</td>
<td>19/20</td>
</tr>
<tr>
<td>Septal</td>
<td>R' or noted R aV&lt;sub&gt;F&lt;/sub&gt;</td>
<td>11/16</td>
</tr>
<tr>
<td></td>
<td>Initial notching of S aV&lt;sub&gt;F&lt;/sub&gt;</td>
<td>5/6</td>
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![Electrocardiograms](image)

**FIG. 8.** Electrocardiograms of a patient with LBBB prior to (A) and during (B) an episode of coronary insufficiency. Note the marked elevation of the S-T junction in leads V<sub>1</sub>-V<sub>3</sub> in B. Autopsied.

The number of cases is insufficient at present to evaluate its reliability. Of the 8 cases in which a correlation similar to the above was not present, a myocardial infarction in another area was present in 3, patchy fibrosis was present in 3, and a vertical electric axis was present in 1.

**S-T Segment and T Wave**

Three cases with an acute anteroseptal infarction and LBBB were available. In the first (fig. 7B), the electrocardiogram taken on the day of the infarction demonstrates marked elevation of the S-T segments in leads V<sub>2</sub>-V<sub>4</sub>, and an isoelectric S-T segment in leads I, aV<sub>L</sub>, III, and aV<sub>F</sub>, a change in comparison with a previous electrocardiogram with LBBB. In the second case (fig. 1B), an electrocardiogram taken on the day after the infarction shows marked elevation of the S-T segment in lead V<sub>6</sub>, moderate elevation of the S-T segments in leads I, aV<sub>L</sub>, V<sub>2</sub>-V<sub>4</sub>, and V<sub>6</sub>, and depression of the S-T segments in leads III and aV<sub>F</sub>. An electrocardiogram taken the day before, when normal intraventricular conduction was present, displays similar S-T segment abnormalities. In the third case, there is marked elevation of the S-T segments in leads V<sub>2</sub>-V<sub>4</sub> and an isoelectric S-T segment in lead I, a change from previous electrocardiograms.

Abnormal S-T segments were present in 2 cases with an anteroseptal and apical aneurysm. The S-T segments are abnormally elevated in leads V<sub>4</sub>-V<sub>6</sub> in the first case, and in leads V<sub>2</sub>-V<sub>4</sub> in the second case.

Electrocardiograms taken during a number of episodes of coronary insufficiency were available in 2 patients. A representative electrocardiogram of one is shown in figure 8B. There is elevation of the S-T junction in leads V<sub>1</sub>-V<sub>4</sub>, and depression in leads I, aV<sub>L</sub>, V<sub>3</sub>, and V<sub>6</sub>. The T waves in leads V<sub>1</sub>-V<sub>4</sub> are tall and peaked, and the T waves in leads I and V<sub>6</sub> are diphasic. On each admission, this pattern
would soon revert to the usual pattern (fig. 8A). Similar changes in the T waves are seen in the other case (fig. 9). Figure 9A demonstrates the increased amplitude and more pointed T waves, most marked in leads V1-4.

In the 5 cases of intermittent LBBB, there was no consistent correlation between the T-wave abnormalities present during normal intraventricular conduction and those present during LBBB. When abnormal T waves were present during normal intraventricular conduction, however, the most frequent changes in the T waves during LBBB were increased positivity of T waves in the precordial leads (fig. 10), and increased amplitude of T waves in the limb leads. The abnormal precordial T waves are taller and more pointed than normal in leads V1-4, and usually diphasic, occasionally upright, in V5 and V6.

**DISCUSSION**

Wilson and his associates\(^1, 5, 6, 12-14\) have discussed the diagnosis of myocardial infarction in the presence of LBBB in a number of papers. In 1936, Wilson\(^5\) published a case of LBBB with QS complexes in leads V5 and V6, in which there was a large infarction of the anterior, posterior, and septal walls of the left ventricle. He considered that these QS complexes were due to the extensive infarction of the lower septum. In the study that summarized the experience of the Wilson group with the precordial electrocardiogram,\(^6\) it is stated that S-T segment and T-wave changes may occur if the area of the QRS is small, and that an equiphasic RS in lead V6 is suggestive of anterior infarction but is not a reliable sign. In 1944, Sodeman, Johnston, and Wilson\(^13\) reported that in 8 autopsied cases of LBBB with a Q wave in lead I, 6 had infarcts, and 5 of these 6 had septal lesions.

Dressler, Roesler, and Schwager\(^2\) in a review in 1950, presented and discussed 6 cases with definitely localized infarctions. They also summarized the electrocardiograms of appar-
ently uncomplicated LBBB in a study of 28 patients with no history of myocardial infarction or angina. In addition to the criteria previously mentioned for the diagnosis of infarction in the presence of LBBB, they observed a W-shaped QRS complex in lead II, which they felt was suggestive of a posterior infarction. They also presented definite criteria for S-T segment and T-wave configurations that are suggestive of infarction. Features considered suggestive of infarction include an isoelectric or elevated S-T junction following a prominent R deflection, a depressed or abnormally elevated S-T junction or a diphasic or inverted T wave following a deep S or QS deflection, and coronary T waves. (CR or CF leads were used in this study.)

Pantridge15 in 1951 suggested that when an R wave was present in lead V_1, the diminution or disappearance of the R waves in leads V_2-V_4, in association with Q waves in the left precordial leads, is suggestive of anteroseptal infarction. He also suggested that atypical complexes in leads from the left side of the transition zone might be associated with infarction, a low primary peak of the R wave with anteroseptal infarction, and a low second peak with anterolateral infarction. He reported that the ventricular gradient was abnormal in each of 9 cases of LBBB with either grave myocardial disease or probable myocardial infarction.

In 1952, Sodi-Pallares, Rodriguez, and Bisteni3,4 discussed the problem of the electrocardiographic diagnosis of myocardial infarction complicated by LBBB, basing their conclusions on experience with clinical cases and experimental work on dogs. They concluded that recent infarctions are suggested mainly by abnormal S-T segments and abnormal T-wave configurations, and especially, by serial changes. In old infarctions, they found a qRs complex in the left precordial leads associated with infarction of the anterolateral wall and low septum in 2 cases.

Kert,7 in 1952, presented a case of extensive anteroseptal infarction with Q waves in the left precordial leads. In 1953, Cabrera and Friedland8 presented the results of a study of a group of 45 cases with LBBB. This group included only those cases in which there was notching or slurring in the final part of the QRS complex in precordial leads with an rS or QS configuration. They found a high degree of correlation between a late notch, 0.05 second or more in duration, in these leads and anteroseptal infarction. Fruscella and Boccardelli,9 in 1954, found notching on both the downstroke and the upstroke of the S wave in similar leads in 10 cases of LBBB. Six of these cases had a clinical history of infarction. Kennamer and Prinzmetal,10 in 1956, suggested that infarction may be suspected by the loss of voltage of the R wave in the left precordial leads when compared with a previous electrocardiogram. S-T segment and T-wave abnormalities were emphasized by Moia and Acevedo11 and Somerville and Wood.12 Master and co-workers13 have found that 8 per cent of patients with acute myocardial infarction have LBBB.

Experimental work on dogs with myocardial infarction and LBBB has been done by Rosenbaum and his associates.12 They concluded that when LBBB is present, infarction of the free wall of the left ventricle gives rise to no characteristic modification of the QRS complexes in the precordial leads. The location of the infarct in these dogs, however, on the anterior aspect of the apex, would make it probable that the anterior-inferior aspect of the septum was involved also. Examination of the published electrocardiograms reveals that there is present in the electrocardiograms of the dogs with an infarct a notched S wave in the precordial lead just to the right of the transition zone, corresponding to lead V_4 (point 8, dog 68; point 7, dog 70).12 There is also an rsR' in a position corresponding to lead V_6 (points 8 and 9, dog 70).12 Similar complexes are present in the corresponding epicardial leads. Such complexes are not present in the precordial and epicardial electrocardiograms of the dog in which no infarction had been produced (fig. 7).6 These complexes are the same ones we have found to be almost always associated with anteroseptal and apical infarctions in our cases. We have not found any published reports of the electrocardiographic study of experimentally produced posterior infarction in the presence of LBBB.

The reasoning leading to the belief that a
myocardial infarction can only rarely be diagnosed by the electrocardiogram in the presence of LBBB can be summarized as follows:

The presence of characteristic modifications of the QRS deflection in infarction almost always depends upon the transmission of the potential variations of the cavity of the left ventricle to the epicardial surface of the infarct and the adjacent parts of the body. ... In left bundle branch block, the cavity of the left ventricle is positive at the beginning of the QRS interval, and, consequently, Q and QS waves do not occur in leads [taken over the infarct]. ... When the septum is infarcted, as well as the free wall of the left ventricle, the cavity of the left ventricle is initially negative because the negativity of the cavity of the right is transmitted to it. Under these circumstances, the electrocardiogram may display large Q or QS deflections in leads from the left precordium.

This difficulty in the diagnosis of an isolated infarction of the free wall of the left ventricle has been extended by inference to the diagnosis of all other infarctions except one that destroys a large amount of septal myocardium and prevents the initial positivity of the left ventricular cavity. Clinically, however, an isolated infarction of the free wall, on which the generalization rests, is a relatively uncommon type of infarction. It was present in only 3 of our 30 cases of myocardial infarction with LBBB, while at least 20 of the 30 cases had infarction of the septum.

It is thought by many that the vector concept as applied in describing the origin of the changes in the QRS complex characteristic of infarction is more accurate than an interpretation in terms of cavity potential alone, called by some the "electric window" concept. In these terms, an infarction in the septum, in the presence of LBBB, may result in the loss of myocardium previously producing powerful early vectors. Various types of septal infarction—anterior or posterior, large or small—could cause various effects on the electrocardiogram.

Figure 1 demonstrates that the "cavity Q wave" associated with infarction is not always produced by cavity negativity alone. The Q waves present in leads V1,2 in figure 1A would be interpreted in these terms as reflecting the cavity negativity that is transmitted through the electrically dead anteroseptal infarction.

On the following day, however, in the presence of LBBB, r waves were present in leads V1,4 (fig. 1B). Three days later, Q waves were again present in leads V1,4 (fig. 1C). The r waves in leads V1,4 during LBBB must have been produced by right ventricular forces. The "cavity Q wave" may be present over electrically functioning myocardium, therefore, when vectors produced by the part of heart directly under the lead are overbalanced by larger vectors with an opposite direction.

Among the other QRS features associated with infarction, the notching of the S wave in lead V4 of the R wave in lead V6, and of the S wave in lead aVF have several characteristics in common. This notching is usually fairly close to the base line and it occurs during the early part of the QRS complex.

Wilson and Herrmann have described the notching of the QRS that occurs in the normal electrocardiogram and in uncomplicated LBBB. In the former, notching usually was confined to the lead of smallest amplitude or occurred relatively close to the base line. In the latter, the notching was usually near the apex of the QRS in leads of large amplitude. They believed that these 2 types of notching differed entirely in their practical significance. The reasons for this belief can be summarized as follows:

The height of a deflection in a given lead at a given instant is dependent on two factors: the manifest potential difference (E), and the cosine of the angle between the direction in which this potential is developed and the line of lead. ... The change in cosine per degree is much more rapid near 90 degrees than near zero degrees. Since, moreover, the deflection in a given lead is smallest when the electrical axis is perpendicular to the line of lead, it follows that the QRS of least amplitude will most faithfully record the changes in the direction of the vector E which occur during this interval. ... Theoretically, notches may be produced by irregularities in the growth and decline of the manifest potential difference ... Such notches will be reproduced most faithfully in the QRS of greatest amplitude ... It is probable that the notches which occur on the QRS group of [uncomplicated] bundle branch block curves are of this type.

In LBBB, these notches are a result of the sudden spread of activation through the left ventricle after its passage through the septum.
In contrast to the notching in uncomplicated LBBB, due to irregularity in change of the manifest potential difference, the notching of the QRS complexes that we have found to be associated with anteroseptal and septal infarction has the characteristics of the other type of notching, due to irregularity in the movement of the electric axis. The notching in our cases has occurred relatively close to the base line, and, the complexes in the precordial lead just to the right of the transition zone have been the complexes of least amplitude in the horizontal plane. They occur when the electric axis is relatively perpendicular to the line of that lead in contrast to the notching in uncomplicated LBBB that occurs when the electric axis is relatively parallel to the line of lead.

A second difference between the notching in these cases associated with anteroseptal and septal infarction and the notching in uncomplicated LBBB is the time of the notching during the QRS interval. The abnormal notch in the S wave in lead V1 in our cases has begun approximately 0.03 second after the beginning of the QRS in most cases. The abnormal notch in the rsR’ complexes has begun approximately 0.02 second after the beginning of the QRS. In LBBB the first upstroke of the R wave in lead V6, which is due to activation of the septum, is usually 0.05 to 0.06 second in duration. In the dog, where activation of the heart does not take as long as in the human, the time required for the wave of activation to cross the septum in LBBB has been experimentally measured to be from 0.03 to 0.04 second. The abnormal configurations described above, therefore, are produced during the advance of the wave of activation through the septum and, possibly, during the early part of the activation of the adjacent free wall. The notching at the apex of the R wave in lead V6 in uncomplicated LBBB, on the other hand, usually occurred 0.05 second or more after the onset of the QRS.

Although it was anticipated that this early notching of the S wave in the precordial lead just to the right of the transition zone might be produced in a “transitional” complex, such a complex could not be found in cases of uncomplicated LBBB. An extensive precordial exploration in a group of 8 such cases, each with a broad, notched R wave in lead V6, did not reveal early notching of the S wave in any lead near the transition zone. The appearance of this notching for the first time after anteroseptal infarction in 2 cases, and its presence only in dogs with infarction also tend to identify the notching with infarction.

The decrease in height of the precordial R wave in going from right to left would appear to be due to loss of anteroseptal myocardium that had previously been producing early positive potential in the midprecordial leads. The slight decrease in height of the precordial R waves in the 2 cases with anterolateral infarction in our group may or may not be due to the infarction.

It would appear, therefore, that the Q wave and the rsR’ in leads I, aVL, and V6, the early notching of the S wave in the precordial lead just to the right of the transition zone, and the abnormal precordial R progression are all a result of the same process—infarction of myocardium in the anteroseptal area, the Q wave being produced by large infarctions, and the rsR’ and the notched S wave being produced by smaller infarctions. The downward deflection on the early part of the upstroke of the R wave, producing the rsR’ configuration, appears to have a significance similar to that of a Q wave. It could be called a “delayed Q wave,” delayed by the time necessary for passage of the wave of activation through the septum before it reaches the infarcted area.

The association of the early notching of the S wave in lead aVF with septal infarction in most cases raises the possibility that a septal infarction that is totally obscured during normal intraventricular conduction might produce recognizable changes on the electrocardiogram during LBBB. The difference in the patterns of activation in the 2 situations lends support to this possibility. During normal intraventricular conduction, the septal vector is much smaller than the left ventricular vector present at the same time. The loss of this septal vector by infarction produces little change in the resultant total vector. During LBBB, however, the septal vector is much larger dur-
ing the early part of the QRS complex than the right ventricular vector also present. In this situation, it is possible that septal infarction could produce diagnostic changes on the electrocardiogram.

The findings in posterior infarction appear to be of a more heterogeneous nature, possibly due to the wider anatomic distribution of infarcts that are included in this classification. The notch in the R wave in leads III and aVF may also have the significance of a Q wave in some cases.

The group of 13 cases without gross infarction cannot be regarded as a normal control group, as 12 of the 13 had myocardial fibrosis and 9 of the 13 had a history of congestive failure. It may be that the fibrosis in these cases was extensive enough to alter the sequence of activation, producing these patterns. Burch has observed that even a small amount of fibrosis can cause a recognizable change in the electrocardiogram. He found that septal fibrosis was present frequently in cases in which Q waves were absent in leads I, V₅, and V₆, during otherwise normal intraventricular conduction. In a series of 95 autopsied cases of diffuse, patchy myocardial fibrosis in the absence of confluent infarction, Weinberg and co-workers found a progressive increase in slurring and decrease in voltage of the QRS complexes to be correlated with such lesions. This was found in 5 of his 8 cases with bundle-branch block.

Q waves in leads V₅ and V₆ with a late “intrinsicoid” deflection could be produced either by an anterolateral infarction with a so-called peri-infarction block or a block involving one of the major subdivisions of the left bundle, or by an extensive anteroseptal infarction with LBBB. We believe that the cases presented here with Q waves in leads V₅ and V₆ are cases with LBBB, because each one was associated with an anteroseptal, rather than anterolateral, infarction. It should be mentioned that a Q wave in leads I, aV₃, or V₆ during LBBB may not be necessarily indicative of infarction, as suggested by the case presented by Lapin and Sprague, in which a Q wave was present in these leads only during inspiration.

There was no clinical evidence of a myocardial infarction in their patient.

Although the correlation of the QRS features described above with myocardial infarction has appeared to be high in this group of cases, 63 cases is not a large number for statistical purposes when they are broken down into several categories. Furthermore, the QRS changes in lead aVF that appear to occur with the predominantly septal and posterior infarcts are of a minor character, and occur in only a few leads. When the R’ and notched R wave in lead aVF are used as an indication of posterior infarction, there are a large number of “false positives;” this feature, especially, needs to be evaluated further. More extensive correlation of electrocardiographic changes with clinical and autopsy findings is necessary before these electrocardiographic features can be applied with confidence in the diagnosis of myocardial infarction. Further experimental studies are in order also, to place these findings on a sound basis. It should also be remembered that with LBBB, as without it, a myocardial infarction may occur without apparent change of the electrocardiogram.

Another electrocardiographic pattern, the “LBBB with precordial leads that erroneously suggest a RBBB,” has been presented by Sodi-Pallares and Rodriguez and by Richman and Wolff as being due to a particular type of myocardial infarction with LBBB. Sodi-Pallares and Rodriguez suggested that this pattern was produced by infarction of the anterolateral wall and moderately extensive infarction of the septum, while Richman and Wolff suggested that it was produced by infarction of the septum and of the lateral and diaphragmatic walls of the left ventricle. The question of whether either type of infarction can change the precordial leads in LBBB to resemble those in RBBB, would appear to require experimental demonstration.

Although it has been stated that characteristic changes in the S-T segment and T wave are usually obscured by the alterations of the T complex produced by the conduction defect except when the area of the QRS is small, each of the 3 cases in which electrocardiograms were taken during the acute stage of a myocardial
infarction has marked S-T segment elevation (figs. 1B and 7B). The area of the QRS complex in each of these cases was fairly large. These few cases would suggest, therefore, that characteristic S-T segment elevations may be present during LBBB in cases with acute myocardial infarction, even when the area of the QRS is fairly large. The S-T segment changes that we have observed have been similar to those described by Dressler and co-workers and Sodi-Pallares and associates. It is of interest to note the close similarity between the complexes with marked S-T segment elevation in leads V4 and V5 in figure 7 during acute anteroseptal infarction in the presence of LBBB (fig. 7B), and the electrocardiograms from epicardial leads taken by Kennamer and Prinzmetal. Two minutes after ligation of a branch of the anterior descending coronary artery in a dog with LBBB.

Although the apparent abnormalities noted in the T wave may be the result of an abnormal ventricular gradient, little reliance can be placed on these changes alone, in view of the many causes of T-wave abnormalities. Sodeman has observed that diphasic or upright T waves in lead I are not necessarily abnormal in LBBB. We also have observed many instances of slight terminal positivity of the T waves in leads I, aVL, and V5 in cases not suspected of myocardial damage.

Although not related directly to LBBB, it should also be mentioned that a myocardial infarction can be diagnosed occasionally when, in the presence of LBBB, there is a postextrasystolic beat with normal intraventricular conduction. Infarction has also been suspected by the occurrence of premature beats “of the ‘intermediate type,’ that is, due to excitations which activate the 2 ventricles in normal sequence because of suitable site of ventricular focus.”

**Summary**

A study of the electrocardiograms of 50 patients with myocardial infarction and left bundle-branch block (LBBB) reveals that QRS changes that appear to be characteristic of myocardial infarction are present in many such cases.

Extensive anteroseptal infarction in the presence of LBBB is associated with Q waves in leads I, aVL, and V6, and an abnormal precordial R progression. When the infarction is less extensive, rsR complexes are present in the same leads, and the S wave or QS deflection in the precordial lead just to the right of the transition zone, usually V4, is deeply notched. One or more of these findings were present in each of the 17 cases of anteroseptal infarction in our group. The notching of the early part of the upstroke of the R wave in left precordial leads producing an rsR complex, would appear to be the equivalent of a “delayed Q wave,” in its significance in indicating anteroseptal infarction.

Anterior infarction without septal involvement was present in 3 cases in our group, 1 case having an Rs configuration in lead V4, and the other 2 having an abnormal precordial R progression.

Nine of the 10 cases with posterior infarction in our group were associated with a notched R wave or an R’ in lead aVF. A similar configuration was present in lead III in 8 of the 10 cases.

Each of 5 cases with predominantly septal infarction in our group was associated with initial notching of the S wave in lead aVF.

Similar complexes were present in most cases collected from the literature, and in published examples of the electrocardiograms of dogs with myocardial infarction and LBBB.

The electrocardiograms of several cases demonstrate that the “cavity Q wave” does not always reflect pure cavity negativity. Theoretic reasons, clinical experience, and experimental data are discussed that suggest that the early notching of the R wave in leads I, aVL, and V6, and of the S wave in leads aVF and V4, are different from the usual notching in uncomplicated LBBB, and are related to myocardial infarction.

Abnormal elevation of the S-T segment was present in leads I, aVL, and in the precordial leads in each of the 3 cases of acute anteroseptal infarction in the group. S-T segments were also abnormally elevated in the precordial leads in 2 cases with an anteroseptal and apical
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aneurysm. These S-T segment elevations appear to have the same significance during LBBB as they have during normal intraventricular conduction. No consistent correlation of T-wave changes with ischemia could be demonstrated.

Further clinical, autopsy, and experimental evaluation is necessary before these findings can be applied with confidence in the diagnosis of myocardial infarction.

ADDENDUM

Since completion of this paper, 8 additional autopsied cases of myocardial infarction with LBBB have been studied, with findings similar to those reported above. Since completion also, the English edition of Demetrio Sodi-Palleres’ book New Bases of Electrocardiography, has become available. He discusses the problem of the electrocardiographic diagnosis of myocardial infarction in the presence of LBBB and emphasizes that in the case of septal infarcts, LBBB actually aids in the detection of the infarct. His experimental work is summarized in the book and several new clinical cases are presented.

SUMMARIO IN INTERLINGUA

Le studio del electrocardiogrammas de 50 patientes con infarimento myocardic e bloco de branca sinistre ha revelate que alterationes de QRS, apparentemente caracteristic de infarimento myocardio es presente in multe tal casos.

Extense infarimento anteroseptal in le presentia de bloco de branca sinistre es asociate con undas Q in le derivationes I, aV_L, e V6 e un anormal progression de R precordial. Quando le infarimento es minus extense, complexos rsR’ es presente in le mesme derivationes, e le unda S o le deflexion de QS in le derivation precordial justo al dextera del zona de transition (usualmente V4) es profundemente indentate. Un o plures de iste aspectos eseva presente in cata un del 17 casos de infarimento anteroseptal in nostre gruppo. Le indentation del prime parte del ascendentia del unda R in derivationes sinistro-precordial producente un complexo rsR’ pare esser le equivalente de un “retardate unda Q” in su qualitye de indicazione de un infarcimento anteroseptal.

Infarimento anterior sin implication septal eseva presente in 3 casos de nostre gruppo. Un de iste casos haeva un configuration Rs in derivation V6, e le altere 2 habeva un anormal progression de R precordial.

Nove del 10 casos con infarcimento posterior in nostre gruppo eseva associate con un indentate unda R o un R’ in le derivation aVF. Un simile configuration eseva presente in derivation III in 8 del 10 casos.

Omne le 5 casos con infarcimento predominantemente septal in nostre gruppo eseva associate con indentation initial del unda S in le derivation aVF.

Simile complexos eseva presente in le majoritate del casos colligite in le litteratura e in publicate specimens de electrocardiogrammas de canes con infarcimento myocardic e bloco de branca sinistre.

Le electrocardiogrammas de plure casos demonstra que le “unda Q a cavitate” non reflecte semper un pur negativitate de cavitate. Rationes theoretic, experiencias clinic, e datos experimental es discute que indica que le indentation al inicio del unda R in derivationes I, aV_L, e V6 e del unda S in derivationes aVF e V4 non es identic con le indentation usual in non-complicate bloco de branca sinistre sed es relationate con infarcimento myocardic.

Anormal elevationes del segmento S-T eseva presente in derivationes I, aV_L, e le derivationes precordial in omne le 3 casos de acute infarcimento anteroseptal in nostre gruppo. Le segmentos S-T eseva etiam anormalmente elevate in le derivationes precordial in 2 casos con aneurysma anteroseptal e apical. Iste elevationes del segmento S-T pare haber le mesme signification in bloco de branca sinistre como in normal conduction intraventricular. Nulle correlation uniforme inter alterationes de unda T e ischemia poteva esser demonstrate.

Additional evaluationes clinic, necroptic, e experimental es necessari ante que iste constatazioni pote esser applicate con confidentia al diagnose de infarcimento myocardic.

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