Left Axis Deviation
An Electrocardiographic-Pathologic Correlation Study

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Our understanding of the meaning of the electrocardiogram is greatly furthered by careful comparison of cardiac hypertrophy and cardiac damage observed post mortem with electrocardiograms taken during the last weeks of life. An electrocardiographic-pathologic correlation of 672 cases is reported in which the roles of left ventricular hypertrophy, myocardial infarction, and other factors in the production of left axis deviation are examined. A heretofore unrecognized QRS-complex syndrome of infarction is described in which the QRS forces are diagnostically abnormal but no "Q waves" are seen in the conventional leads. The roles of hypertrophy, variations in body build, chronic pulmonary disease, and various types of "papillary block" in the production of left axis deviation are studied.

Left axis deviation (LAD) is one of the commonest abnormalities encountered in clinical electrocardiography. When unassociated with other electrocardiographic abnormalities, it is usually considered of little clinical significance and is attributed to a leftward anatomic position of the heart, to incomplete left bundle-branch block, or perhaps to left ventricular hypertrophy. Recent observations, however, suggest that none of these is a common cause of LAD and that other factors, some of clinical importance, are more frequently the cause.

In the first place, detailed anatomic-electrocardiographic studies have shown that cases with marked LAD do not necessarily have a more leftward direction of the anatomic long axis of the left ventricle than do cases with more normally directed electric axes. Indeed, there proved to be very little variation in the position of the left ventricle in the chest in a wide variety of cardiac conditions. Furthermore, evidence has been presented that incomplete left bundle-branch block as ordinarily defined is an exceedingly uncommon electrocardiographic syndrome, and this evidence will be discussed further. Finally, it has been shown that myocardial infarction frequently causes an alteration in the direction of the last electric forces to be generated during the QRS interval. On comparing pre-

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infarction and postinfarction tracings in a large number of subjects it was found that nearly half show this alteration in terminal QRS vector without significant prolongation of the QRS interval, and that in a large number of these cases the terminal QRS alteration produced marked LAD. In order further to evaluate the relationship of infarction, hypertrophy, and other myocardial abnormalities to the incidence of LAD a clinico-pathologic-electrocardiographic correlative study was undertaken.

METHODS AND MATERIAL

Six hundred seventy-two consecutive cases were collected in which complete autopsies had been performed and electrocardiograms had been recorded within 5 weeks of death. From the electrocardiographic point of view, the tracings were examined for the incidence of (1) deformity of the initial QRS electric forces diagnostic of myocardial infarction by current generally accepted criteria outlined below; (2) significant left axis deviation (i.e., the mean QRS vector directed more leftward than -15 degrees on the triaxial reference figure); (3) left ventricular "strain" (i.e., mean spatial ST and T vectors relatively parallel with each other and both more than 160 degrees from the direction of the mean spatial QRS vector, whether with or without LAD); and (4) the amplitude of the QRS

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complexes in the limb leads and precordial leads, to be correlated with the heart weight and body build for a given mean QRS axis direction. Cases with QRS intervals of .12 sec. or more were not included in the study. In all cases the conventional 12-lead electrocardiogram had been recorded with V leads.

From the point of view of the autopsy correlations, the cases were obtained from hospitals connected with medical schools where, in view of the teaching activity, the postmortem examinations might be presumed to be more than usually comprehensive and detailed. However, since in nearly all cases the autopsies had been completed before this study was begun, and since no special studies in cardiac pathology were being conducted in these hospitals, it must be assumed that the postmortem examination of the hearts was no more than “routine” in its meticulousness in most cases. However, the type of information sought from the postmortem examination for this study was simple and not likely to have been overlooked or measured incorrectly. The autopsy protocols were studied for (1) the presence of myocardial infarction, seen grossly and confirmed histologically, without regard to its size, age, location, or the likelihood that more than 1 infarct might have been present; (2) the amount of fibrosis on histologic examination; and (3) the incidence of left ventricular hypertrophy. Measurements of wall thickness and descriptions of the diameter of myocardial fibers were found to be supportive but not in themselves unequivocal evidence of left ventricular hypertrophy. Therefore, the gross weight of the heart was used to identify these cases. It is recognized that this is an extremely crude index of left ventricular size, especially if the increase in weight is slight, and that cases with large mural thrombi or excessive epicardial fat might be erroneously included among cases of left ventricular hypertrophy by this method. However, for the particular purposes of this study it was more important that no case of marked left ventricular hypertrophy be overlooked than that all cases of even slight hypertrophy be included. Therefore, hearts weighing over 500 Gm. were considered to be instances of left ventricular hypertrophy if the measured thickness of the left ventricular wall was increased, and if no other cause of the increased weight had been described. This is a considerably greater heart weight than is usually used to identify hypertrophy but has the virtue of more clearly separating cases of marked hypertrophy from those that might be normal. In all cases with left ventricular hypertrophy an etiology was sought in the clinical record on the autopsy protocol to confirm the diagnosis; in all but a few instances such factors could be identified.

Of course the QRS changes ascribed to left ventricular hypertrophy reflect only an increase in ventricular size, whether due to hypertrophy or di-
triaxial reference figure, it points away from the region of the heart involved electrically by the infarct, as shown. It is the particular direction of these initial forces of the QRS interval that accounts for the abnormal "Q waves" on certain of the leads in such cases. Thus the "Q wave" patterns for different electric locations of infarction can be systematized by drawing the mean vector for the first .04 sec. of the QRS interval for each location. This is shown at the bottom of figure 1 for each of the 5 topographic locations of the left ventricle. These 5 directions of initial .04 vectors embody the QRS complex criteria used in this study.

The electric locations may or may not coincide with the actual anatomic location of the infarct. In the present study, there appeared to be at best a general similarity between the electric location of infarction and its anatomic locations as described at autopsy; however, frequently there was no agreement whatever. This should not be surprising, since the electric effects and the histologic effects are 2 quite different manifestations of infarction and there is no reason why they should necessarily coincide in location. In the discussion that follows the use of anatomic terms to identify infarction (e.g., "anterolateral infarction") is meant to indicate the type of QRS complex deformity produced by the infarct and not necessarily the actual anatomic location of the infarct. No attempt is made in this study to test present-day QRS criteria for the diagnosis of infarction against the findings at pathologic examination except as they bear on the problem of left axis deviation. The relative incidences of electrocardiographic and autopsy findings used in this study are shown in table 1.

**Left Ventricular Hypertrophy**

**As a Cause of Left Axis Deviation**

It is generally believed that left ventricular hypertrophy (LVH) is a cause of left axis deviation (LAD); indeed, LAD has often been considered an important if not essential criterion for the electrocardiographic diagnosis of LVH.\(^4\)\(^5\) Therefore, the relationship between heart weight and the incidence of LAD was studied. When cases with proved myocardial infarction were excluded, there were 77 cases with LAD. Only 35 of these (less than half) had heart weights of 500 Gm. or more; 19 had hearts weighing 400 to 500 Gm., and 23 had hearts weighing less than 400 Gm. Thus LAD is by no means diagnostic of LVH. Nor is the development of LAD dependent upon the severity of the LVH; among the 9 cases with hearts over 900 Gm.

**Table 1.—Electrocardiographic and Autopsy Findings in 672 Consecutive Cases**

<table>
<thead>
<tr>
<th>Total cases ..........</th>
<th>672</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial Infarction</td>
<td></td>
</tr>
<tr>
<td>Left Ventricular Hypertrophy (no myocard. infarc)</td>
<td>73</td>
</tr>
<tr>
<td>Controls (under 500 Gm., no myocard. infarct)</td>
<td>429</td>
</tr>
<tr>
<td>No Infarct ..........</td>
<td>113</td>
</tr>
<tr>
<td>Ht. wt. over 500 Gm.</td>
<td>33</td>
</tr>
<tr>
<td>Ht. wt. 400-500 Gm.</td>
<td>19</td>
</tr>
<tr>
<td>Ht. wt. under 400 Gm.</td>
<td>23</td>
</tr>
<tr>
<td>Myo. Infarct ..........</td>
<td>12</td>
</tr>
<tr>
<td>LVH with LAD ..........</td>
<td>6</td>
</tr>
<tr>
<td>LVH without LAD ........</td>
<td>0</td>
</tr>
<tr>
<td>Normal ht. wt. with LAD ..</td>
<td>7</td>
</tr>
<tr>
<td>Normal ht. wt. without LAD ..</td>
<td>21</td>
</tr>
</tbody>
</table>

Due to LVH, 6 had normally directed electric axes and only 3 had LAD.

This poor correlation of LAD and LVH was somewhat unexpected, so that the question was raised whether variations in body build might have influenced the results, a lean subject having a more vertical axis and a stocky subject a more leftward axis for a given heart weight. The body height and weight for each subject at the time of death had been entered in the autopsy protocol, and therefore this possibility could be studied. In figure 2 all cases of LAD and left ventricular "strain" and, in addition, all cases with hearts weighing over 500 Gm. (excluding those with infarctions at autopsy) are plotted in terms of heart weight and body build; then, for each type of body build, the cases are divided into those with normally directed electric axes, and those with LAD. For a given heart weight, whether or not it was greater than normal, the incidence of LAD was no greater among the stocky subjects (those overweight for the
left axis deviation

FIG. 2. Effect of body build and heart weight on the direction of the mean QRS axis. The cases are distributed on the abscissa according to normal or left axis deviation for 3 types of body build.

stated body height) than among lean subjects (those underweight for the stated height). In fact, there were certain lean subjects with normal or less than normal heart weights who had marked LAD while certain stocky subjects with tremendous LVH had normally directed electric axes. This does not mean that body build is without influence on the direction of the electric axis, but only that this influence is slight in most instances. In a previous study, it was shown that there is rarely more than a 20-degree variation in the direction of the anatomic long axis of the left ventricle in either the normal subject or the subject with marked RVH or LVH regardless of body build. In conclusion, while a stocky body build might cause LAD to become more leftward or a lean body build might cause a normal axis to become more vertical, it is apparent that neither variations in body build nor variations in the anatomic position of the heart can alone be responsible for LAD.

It must not be overlooked that, although less than half of the cases of LVH had LAD, nevertheless the incidence of LAD is higher among cases of LVH than it is among patients with normal heart weights, for less than a tenth of the 439 cases with neither LVH nor infarction showed LAD. Thus, although LVH does not itself cause LAD, there is some aspect of the hypertrophy mechanism that in about half of the cases brings in its train LAD. Perhaps the myocardial fibrosis that so commonly accompanies marked LVH produces an alteration in the more peripheral parts of the left ventricular conduction network analogous to what has been called "parietal block." While variations in body build do not greatly influence the direction of the mean QRS axis, they do influence the amplitude of QRS complexes in the various leads. This relation has been largely overlooked in studies concerned with establishing QRS criteria for the diagnosis of LVH. In the present series of cases, these criteria identified over 90 per cent of cases of marked LVH when the body build was normal. However, in subjects either markedly overweight or underweight for their height, the criteria were totally unreliable. For example, there were 3 subjects with heart weights over 700 Gm. due to LVH with QRS complexes not more than 20 mm. in amplitude in the precordial leads, which is within the normal range. All 3 subjects weighed over 190 pounds and none was taller than 5 feet 6 inches. On the other hand, there were 7 cases with hearts weighing less than 450 Gm. who had QRS complex amplitudes of more than 45 mm. in 1 or more precordial leads, which are amplitudes ordinarily considered diagnostic of LVH. All 7 subjects weighed less than 115 pounds and their average height was 5 feet 6 inches. Thus, obesity may obscure the QRS changes of LVH, and leanness may account for unusually large QRS complexes when the heart is normal in weight.

There is another clinical syndrome in which the amplitude of the precordial QRS complex is greatly increased and resembles that of LVH but in which the left ventricle is normal in weight. This is the syndrome of mitral insufficiency with giant left atrium. In figure 3 is shown the electrocardiogram of such a patient; the very large QRS complexes and
abnormal ST-T segments in the left precordial leads are identical with what is seen in severe LVH. However, the heart in this case was carefully dissected at autopsy and the left ventricle was found to be low normal in weight and not dilated. Atrophy of the posterior wall of the left ventricle, which is commonly seen in this syndrome, accounted for the reduced weight. The details of this case, including photographs of the left ventricle, have been published elsewhere. The increased amplitude of the QRS complexes was due to the fact that the greatly enlarged left atrium had shifted the ventricular part of the heart much closer to the left anterior chest wall. It will be noted that the mean QRS axis is vertical in this case, as it is in most cases of rheumatic mitral regurgitation with enlarged left atrium; this position reflects right ventricular dominance and is further evidence against significant left ventricular hypertrophy or dilatation. Although it is commonly thought that uncomplicated rheumatic mitral regurgitation is associated with LVH, there have been no careful autopsy studies to support this belief.

**Infarction and Left Axis Deviation**

It is well known that myocardial infarction alters the direction of the electric forces generated during the first .04 sec. of the QRS interval. This is the deformity that accounts for the "Q waves" characteristic of infarction. Recently, it has been shown that infarction may also alter the directions of the electric forces generated during the last part of the QRS interval with little or no prolongation of the QRS interval. In about one half of the cases with QRS alterations of anterolateral infarction the terminal QRS forces are caused to point leftward and superiorly, producing LAD, while in an equal percentage of cases of diaphragmatic infarction they are caused to point rightward and inferiorly, often producing right axis deviation.

The simplest explanation of these terminal QRS abnormalities attributes them to peri-infarction block. In this explanation it is presumed that the normal radial spread of excitation from endocardium to epicardium is blocked at the region of the infarct, either because of the size of the infarct or, more likely, because certain critical regions of the
cases had initial QRS changes of anterolateral infarction and 34 or two thirds of these had \textit{LAD}. On the other hand, only 20 or less than a fifth of the remaining cases of infarction had \textit{LAD}, and only one tenth of the 512 cases in whom no infarction was found at autopsy had \textit{LAD}. These data emphasize both the high incidence of terminal QRS abnormalities of the peri-infarction block type in anterolateral infarction and the importance of myocardial infarction as a cause of \textit{LAD}. In this series, over one third of all cases of \textit{LAD} occurred in cases with infarction at autopsy.

The striking electrocardiographic feature of the \textit{LAD} that occurs in anterolateral peri-infarction block is the wide angle between the initial QRS forces and the terminal QRS forces. To define this more precisely, the angle formed by the mean of the vectors generated during the first .04 sec. of the QRS interval (the "initial .04 vector") and the mean of the vectors generated during the remainder of the QRS interval (the "terminal .04 vector") exceeded 110° in all of these cases. On the other hand, among the 77 cases in this series with \textit{LAD} but no evidence of infarction at autopsy there were only 5 with as wide an angle between initial and terminal .04 vectors. Two of these had advanced pulmonary disease with thoracic deformity; the role of pulmonary disease in producing \textit{LAD} of this type will be explored later. The other 3 cases were all instances of marked left ventricular hypertrophy, 2 due to aortic stenosis and 1 to arterial hypertension with total heart weights of 650, 880, and 1050 Gm. respectively. Although an infarct may have been overlooked in these cases (they all had moderately advanced coronary artery disease), it is also possible that, as pointed out earlier, the fibrosis that accompanies marked left ventricular hypertrophy produces a conduction defect in the wall of the left ventricle with leftward deviation of the terminal QRS vectors similar to that seen in anterolateral peri-infarction block. In conclusion, anterolateral infarction with peri-infarction block is by far the commonest cause of \textit{LAD} with a wide angle between initial and terminal QRS

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**Fig. 4.** Upper figures: schema of the mechanism of initial and terminal QRS vector deformity in anterolateral peri-infarction block. \textit{A}. The typical QRS loop in anterolateral peri-infarction block with the QRS complexes; such a loop would write on the standard limb leads and lead a\textit{V}L. \textit{B}. The loop has been rotated slightly; the angle between the mean initial .04 and terminal .04 vectors is still diagnostically wide, but there is no longer a Q wave in lead I, and the Q wave in a\textit{V}L is only .02 sec. in duration.

Subendocardial conduction network are involved. Therefore, the epicardium overlying the infarct is the last region of the ventricles to be depolarized, and the vectors from this region tend to dominate the last part of the QRS interval. When the initial and the terminal QRS deformities of infarction are plotted on the triaxial reference figure, they tend to be relatively opposite to one another in direction. The initial forces point away from the site of infarction and the terminal forces point toward the site of infarction. The mechanism of peri-infarction block and the way in which anterolateral peri-infarction block produces \textit{LAD} are shown in the upper part of figure 4; a case illustrating this syndrome is shown later in figure 8.

From table 1 it can be seen that there were 160 cases of proved infarction among the 672 cases of this series. Forty-seven of the 160
Fig. 5. Four cases with diagnostically wide angles between initial and terminal QRS vectors but no diagnostic "Q wave" patterns. At autopsy all 4 had gross myocardial infarction. The frontal plane QRS loops were constructed from the standard and unipolar limb leads for each case. The direction of the instantaneous vector at .04 sec. of the QRS interval is shown on each loop.

M-16, 57 y

M-46, 59 y

M-14, 81 y

M-23, 74 y

NORMAL B.P.

NORMAL B.P.

NORMAL B.P.

NORMAL B.P.

500 gm. - M.I.

500 gm. - M.I.

500 gm. - M.I.

500 gm. - M.I.

forces; if severe pulmonary disease or advanced left ventricular hypertrophy can be ruled out, it appears to be relatively diagnostic of infarction.

Might there not be cases of infarction with this wide angle but without diagnostic Q waves in any of the conventional leads because of a slight rotation of the entire QRS electric field? For example, suppose the QRS forces (fig. 4A) were rotated slightly leftward without changing their relationship to one another, as in B; now the wide angle between initial and terminal forces is still present, but no Q waves of .04 sec. are written in any of
the standard or unipolar limb leads. That this can happen is shown in figure 5. Here are 4 cases that proved to have infarctions at autopsy. They all show the diagnostically wide angle between initial and terminal .04 vectors yet have no diagnostic Q waves in any of the limb or precordial leads. (Perhaps the Q wave in Lead aV₃ and in V₄ in the fourth tracing would, for many electrocardiographers, be diagnostic of infarction).

These 4 cases illustrate a type of QRS deformity diagnostic of infarction that has not been previously recognized; in addition, they emphasize the shortcomings of "Q-wave" criteria for the QRS diagnosis of infarction. It is not generally realized that all patients, whether normal or not, have areas on the chest where Q waves of .04 sec. duration can be recorded.² The diagnostic value of Q waves lies not in the Q wave itself but in the location of the lead that recorded it, and this is a function of the direction of the initial QRS electric forces. These cases demonstrate that occasionally the initial QRS forces can be diagnostically abnormal in direction yet not produce "diagnostic" Q waves in any of the conventional leads.

But the value of vector methods in clinical

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**Fig. 6.** Diaphragmatic infarction with LAD of terminal QRS forces identical with and probably due to peri-infarction block of the anterolateral type. A and B are 2 of several tracings illustrating apparent "coming and going" of R' deflections at V₁ to V₃. The direction for the mean vector for the last .04 sec. of the QRS interval has been plotted from the conventional leads at the lower right. It can be seen that the reason for the inconstancy of the R' deflections is variation in electrode location. For certain tracings the electrodes lay in the region of electric positivity (tracing A), while for others they lay in the region of electric negativity for this electric force (tracing B).
electrocardiography is to discriminate and clarify "pattern" criteria, not to supplant them. It is likely that cases of infarction such as these were not recognized previously because the alterations in the deflections were too complex to be recognized by QRS "patterns." However, when the abnormality of electric forces for a given syndrome is known, it is quite simple to describe "pattern" criteria for its recognition. These cases are characterized by an initial R wave of over .04 sec. followed by a deep S wave in lead aVR and associated with marked LAD sufficient to produce a prominent terminal R wave on aVR or even an S wave in lead I. But plotting the initial and terminal .04 vectors may still be necessary to be certain of the abnormality in a given case.

It will be recalled that LAD occurred with other types of infarction in addition to anterolateral. There were 113 cases of proved infarction with QRS deformity other than that of anterolateral infarction, and 20 had LAD. In several, the LAD was probably due to the same mechanism that produces LAD in anterolateral infarction. For example, the anatomic location of the electric defect in strictly anterior infarction is quite near that of anterolateral infarction (fig. 1), and 5 of the 18 cases of this type had LAD. Similarly 4 of the 16 cases of apical and strictly posterior infarction had LAD. In addition, since in most cases of fatal infarction there is more than 1 infarct at autopsy, it is possible that in certain cases the deformity of the initial .04 sec. will be dominated by an infarct at one region while the deformity of the terminal .04 sec. may be dominated by an infarct at another region. Perhaps this combination accounts for the LAD in others of these cases.

In cases of infarction with terminal QRS deformity similar to that of anterolateral infarction but initial QRS deformity of some other electric location, the angle between the initial .04 vector and the terminal .04 vector may not be abnormal. For example, in diaphragmatic infarction with LAD, the initial and terminal .04 vectors are relatively parallel, (see fig. 6). Under these circumstances the recognition of infarction and its differentiation from uncomplicated LAD may be quite difficult. As will be seen later, the somewhat anterior direction of the terminal vector in this case (producing an R-prime at V1 and V2) supports the likelihood that the LAD is due to peri-infarction block.

Other Causes of Left Axis Deviation

Among the 131 cases of LAD of all causes in this series, there were 23 with no evidence of infarction at postmortem examination and with hearts weighing 400 Gm. or less. In 6 of these the important postmortem finding was severe pulmonary disease, either pulmonary emphysema or pulmonary fibrosis, with right ventricular hypertrophy and, in 2 cases, associated kyphoscoliosis. That advanced pulmonary disease may occasionally produce marked LAD has been noted in the literature previously.10-13 It is possible that the LAD is due to some myocardial factor unrelated to the pulmonary disease. However, such a factor was not apparent from the autopsies in these cases. Two of the 6 subjects were less than 50 years of age and 1 was female, which makes it unlikely that the LAD was due to the effects of chronic coronary artery disease. A more likely explanation, although entirely conjectural, suggests that LAD may be related to the reduced electric conductance of the lung in advanced pulmonary disease. It has been shown that the electric conductance of normal pulmonary tissue is not greatly different from that of other tissues.14 The electric conductance in the emphysematous lung has not been tested; however, in view of the presence of large nonconducting air sacs in the lung in this disease, it may well be greatly reduced. If this were so, the electric field surrounding the heart would be altered; the lead-field lines for leads II and III would in effect be concentrated vertically in the mediastinum, and the limb leads would now record principally the superiorly and inferiorly directed components of the electric forces of the heart. For example, QRS forces directed toward the left flank would be recorded as strictly vertically directed, while forces directed toward the left arm would be
Fig. 7. Four cases with LAD of terminal QRS forces; the terminal forces are also slightly anteriorly directed in each case producing terminal R' deflections in the certain of the precordial QRS complexes. The upper 2 cases are instances of severe pulmonary disease, the third of chronic coronary artery disease, and the fourth of hypertension with left ventricular hypertrophy.
recorded as strictly superiorly directed. With just slight differences in the anatomic position of the heart or slight variations in the magnitude of either the initial QRS forces or the more leftward terminal QRS forces in patients with emphysema, the limb leads would record mean QRS axes that are either strictly inferior or strictly superior in direction and rarely in between. Two cases with markedly leftward terminal forces due to severe pulmonary disease are shown in figure 7. To be sure, LAD was not the typical axis direction for cases of pulmonary disease in the series as a whole; there were 27 other cases with severe pulmonary disease and right ventricular hypertrophy at autopsy, and all these had the more familiar vertical QRS axis.

Excluding these cases of LAD due to pulmonary disease, there were 17 cases of LAD with normal heart weights and no evidence of infarction at autopsy. What was the mechanisms of the LAD in these cases? All 17 had abnormally directed T vectors and two-thirds had ST and T vectors characteristic of what is called left ventricular "strain" as defined earlier. On the other hand, among 395 cases with normal heart weights and no evidence of infarction at postmortem examination but with normal QRS axis directions, only 5 had ST and T vector alterations characteristic of left ventricular "strain." The high incidence of abnormalities in repolarization in the cases with LAD indicates that the T abnormality is intrinsically related to the LAD. This relationship suggests that the LAD may represent a conduction defect peripherally in the wall of the left ventricle with "secondary" T-vector alterations. It has been pointed out that uncomplicated LAD is exceedingly uncommon among subjects under 40 years of age. Although the number of cases of this type of LAD in the present series is small, their mean age was considerably higher than that of the series as a whole. For these reasons, it is concluded that LAD in older subjects without evidence of infarction or severe pulmonary disease is probably due to "parietal block" secondary to the myocardial fibrosis of chronic coronary artery disease. It is analogous, perhaps, to the mechanism of the LAD seen in peri-infarction block and in certain cases of left ventricular hypertrophy described earlier.

Might not the LAD in these cases and in the cases of LAD associated with left ventricular hypertrophy be due to incomplete left bundle-branch block? This view also would explain the high incidence of ST and T abnormalities in these cases. However, there are 2 cogent reasons for suspecting that this is not the case. In the first place, it has been shown by several investigators that when complete left bundle-branch block develops, there is usually no change in the direction of the mean QRS axis. One would expect that this would also be the case for incomplete left bundle-branch block. Under these circumstances, one would still need an explanation for the LAD that preceded the bundle-branch block in these cases. In the second place, to prove that left bundle-branch block (either complete or incomplete) has taken place one must have a control tracing showing normal ventricular conduction recorded shortly before or after the tracing with the conduction defect. By comparing the 2 tracings one can prove that (1) the QRS alteration was sudden in onset, (2) the mode of entrance of excitation into the left ventricle was altered (as shown by a change in the directions of the initial QRS forces), and (3) infarction was not responsible for the alteration in initial QRS forces. These features must be demonstrated if a given QRS syndrome is to be attributed to a lesion in the main branch of the left bundle. (In incomplete left bundle-branch block the QRS interval must be prolonged to .09 to .11 sec. with appropriate changes in ST and T vectors, while for complete left bundle-branch block the prolongation is to .12 sec. or more). Cases with tracings showing normal ventricular conduction before and after complete left bundle-branch block fulfilling these criteria are not uncommon. On the other hand such cases are exceedingly rare among those called incomplete left bundle-branch block. In a search among 1000 cases with tracings labeled "incomplete left bundle-branch block," "left ventricular strain," or "left ventricular con-
duction defect” in several different hospitals, not a single case was found that fulfilled these criteria. Only 1 such case has been found in the literature, and here it is difficult to be sure that the initial QRS forces were altered in direction when the QRS complex change took place. It is concluded that what is called incomplete left bundle-branch block is an exceedingly uncommon conduction defect and not likely to have been the cause of the LAD in any of the cases in the present study. LAD of the type under consideration tends to develop gradually over a period of years with no certain change of initial QRS forces and, also unlike bundle-branch block, seems never to return to normal.

**R' Deflections in the Precordial Leads in the Presence of Left Axis Deviation**

In current electrocardiographic practice an R' deflection in V1 or V2 is generally attributed to right bundle-branch block if the QRS interval is prolonged, and to right ventricular hypertrophy if the QRS interval is normal, regardless of the direction of the mean QRS axis. However, from time to time the direction of the mean QRS axis is inconsistent with the precordial lead findings, giving rise to such ambiguities of nomenclature as “left bundle-branch block masquerading as right bundle-branch block,” or “left bundle-branch block with right axis deviation.” This method for interpreting the precordial leads R' deflection resulted from the assumption that a given unipolar lead was recording principally the electric events from the region of the heart immediately underlying it. From this point of view, an R' deflection in the right precordial leads meant that the last region of the heart to be depolarized was the right ventricle, since this lay immediately beneath the V1 and V2 electrodes. However, the evidence is now quite conclusive that body surface unipolar leads are more nearly recording the resultant electric activity of all regions of the heart than of the immediately underlying region. In other words, for practical clinical purposes, all electrode locations can be considered to be recording from essentially the same central resultant electric forces. This means that in interpreting the R' deflection in the right precordial leads one must take into consideration the terminal deflections of the QRS complexes in all leads of the clinical tracing. The simplest method for doing this is to base the interpretation on the direction of the terminal QRS vectors. As the cases in this study show, occasionally R' deflections are written at V1 and V2 by QRS forces that can be attributed to the right ventricle only by assuming the most extravagant and improbable torsion of the heart anatomically from its normal position.

The terminal QRS vector alterations responsible for the R' deflections at V1 and V2 in the presence of QRS prolongation to .12 sec. or more have been discussed previously. We are here concerned with those associated with a QRS interval that is normal or only slightly prolonged. Such R' deflections can be seen with 3 different directions of the terminal QRS vectors and the mechanism and clinical implications of each are different. 1. R' deflections in the right precordial leads are seen when the terminal QRS forces are directed inferiorly, slightly rightward and anteriorly, producing an S3R3S3 pattern in the standard limb leads. This is an appropriate direction for an electric force generated from the right ventricle, and since all cases in the present series with this pattern in the limb and precordial leads had pathologic findings consistent with right ventricular hypertrophy, it can be concluded that this is usually its cause. 2. R' deflections at V1 and V2 are also seen when the terminal QRS forces are directed rightward, superiorly and slightly anteriorly, producing the S1S2S3 pattern. Although most commonly seen in patients with cor pulmonale, this pattern may occur in young adults with no evidence of heart disease and is occasionally acquired as a part of the QRS deformity of infarction. It is likely, therefore, that it is due to a right ventricular conduction defect, perhaps in the region of the crista supraventricularis of the right ventricle. 3. R' deflections at V1 and V2, or both, are seen with terminal QRS forces directed superiorly and leftward, producing
an RsSsS pattern in the limb leads. There is no region of the right ventricle facing in this particular direction, and it is extremely unlikely that they are right ventricular forces.

The terminal forces in a given case of LAD with R' deflections in precordial leads may have any of these 3 directions. However, in this study we are concerned only with the cases of the third type; that is, terminal forces directed leftward and superiorly, producing the RsSsS pattern in the limb leads. It can be seen in table 1 that there were 46 cases with terminal R' deflections at V1 or V2, or both, in the entire series. In 15 of the 46 cases the terminal QRS forces were of the third type mentioned above. Eight, or half of these 15 cases, had myocardial infarction at autopsy; and all 8 showed anterolateral or strictly anterior infarction with marked LAD of the mean QRS axis. Therefore, it is probable that all 8 were examples of anterolateral peri-infarction block. Thus, the commonest cause of an R' deflection in the right precordial leads in association with LAD of terminal QRS forces is peri-infarction block. Cases illustrating this have been published by others.20, 25, 26

Whenever the terminal QRS forces are leftward in direction, as is the case in anterolateral peri-infarction block, there is always an area on the upper left chest where unipolar leads will record positivity for the last part of the QRS complex. Whether or not this area of terminal positivity will extend down far enough on the anterior chest to include the region where the conventional precordial electrodes are placed depends upon how far anteriorly the leftward terminal forces are directed. The distribution of terminal positivity on the chest in such a case is shown in figure 8. Since only 1 in 5 cases of anterolateral peri-infarction block shows terminal R' deflections in the precordial leads, it appears that the area of positivity for these terminal QRS vectors is near, but does not often include the region of the chest where the precordial leads are placed in these cases. However, the region of positivity for these leftward terminal forces is usually so near the location of the precordial leads that slight variations in the placement of precordial electrodes in a given case may cause the R' deflection to come and go from tracing to tracing. Figure 6 illustrates such a case; the terminal R' deflection was present in the precordial leads in some tracings and absent in others. It can be seen that for certain of the tracings, the electrodes for V1 and V2 lay in the area of relative positivity for terminal QRS vectors, writing terminal R waves, and for others they lay in the area of relative negativity for the vector, writing terminal S waves. When the terminal QRS vector is not sufficiently anteriorly directed to produce R' deflections in the precordial leads, a shallow notch on the ascending limb of the S wave instead of an R' wave may be seen in the precordial leads. How this can take place may be seen by studying figure 8. Cabrera and Friedlander24 have also called attention to this shallow notching of the S wave in the precordial leads as a sign of infarction.

Among the 15 cases of LAD with terminal R' deflections at V1 and V2 due to leftward and superiorly directed terminal QRS forces (RsSsSs) there were 2 cases with severe pulmonary disease, no evidence of myocardial infarction, and normal heart weights. A possible explanation for the LAD in these cases has already been suggested. It is well to remember that verticalization of the QRS forces by pulmonary emphysema will apply to rightward forces as well as to leftward forces. Possibly these 2 cases represent verticalization of anteriorly directed SsSsSs: forces which, as mentioned earlier, are commonly seen in the presence of chronic cor pulmonale.

Of the remaining 5 cases with LAD and R' deflections in the right precordial leads, 2 had normal heart weights and no evidence at autopsy of either pulmonary disease or myocardial infarction, and 3 were instances of marked left ventricular hypertrophy. One of each of these is shown in figure 7. It is generally accepted that uncomplicated left ventricular hypertrophy causes the terminal QRS forces to be more posteriorly directed than normally (as evidenced by the shift of QRS transition to the left).8 The fact that the terminal forces were more anteriorly directed
Fig. 8. Unipolar lead QRS complexes in a case of LAD due to anterolateral peri-infarction block. The unipolar deflections were recorded from a number of points on the anterior surface of the chest indicated by the solid dots on the torso at lower right. The location of the electrodes for V₁ and V₂ are shown by open circles on this figure. The null contour and direction for the mean vector for the last .04 sec. of the QRS interval, shown on the torso, were plotted from the chest unipolar deflections. They indicate the distribution of positivity and negativity on the chest for the terminal part of the QRS complex. The electrode locations for the conventional precordial leads lie in the area of electric negativity for the terminal .04 sec. vector. However, they are near the null contour for this force, explaining why the terminal S waves would be shallow in the conventional precordial leads. Had the conventional precordial leads been recorded slightly higher on the chest, terminal R waves would have been recorded in the QRS complexes for these leads.
than normally in these 3 cases is further evidence that the LAD of left ventricular hypertrophy is due to a conduction defect.

In the summary, the analysis of the incidence of R' deflections in the precordial leads in subjects with LAD has disclosed another electric similarity between the LAD of anterolateral peri-infarction block, the LAD of marked left ventricular hypertrophy, and the LAD of chronic coronary artery disease: they all may be associated with terminal QRS forces that are directed sufficiently anteriorly to cause R' deflections to appear at V1 or V2. This finding adds further evidence to the likelihood that the LAD for each is due to a conduction defect distal in the conduction network of the left ventricle, a sort of "parietal block." The fact that the terminal forces are more commonly directed anteriorly in cases of peri-infarction block than in either of the other 2 syndromes is useful from a clinical point of view and also suggests that the similarity of the LAD in these 3 syndromes is a superficial and strictly electrocardiographic one. The underlying electrophysiologic abnormality may well be quite different in the 3 syndromes; but too little is known of the left ventricular conduction mechanism in man to permit further speculation.

SUMMARY AND CONCLUSIONS

Six hundred seventy-two consecutive cases in which an electrocardiogram had been recorded within 5 weeks of death and in which detailed postmortem examination had been performed are the basis of this report; those under the age of 30 years and those with QRS interval duration of .12 sec. or more have been excluded.

One third of all cases with left axis deviation had myocardial infarction at autopsy. Of 160 cases of proved infarction, 67 had left axis deviation and two thirds of these had the QRS deformity of anterolateral infarction. Thus, left axis deviation is considerably more common in anterolateral infarction than in any other type of infarction or in any other single category of heart disease. The mechanism of the left axis deviation in this type of infarction is shown to be peri-infarction block, characterized by a diagnostically wide angle between the initial and the terminal QRS forces. Four cases are shown with proved infarction in which this angle was diagnostically wide but in which there were no diagnostic "Q waves" in any of the conventional leads. This is an electrocardiographic pattern of infarction that has not been previously recognized.

Left axis deviation was seen in less than half of the cases with proved left ventricular hypertrophy. Neither the severity of the hypertrophy, nor the anatomic position of the heart in the chest, nor the body build of the patient plays a significant role in the development of left axis deviation in these subjects. Incomplete left bundle branch block is an extremely rare cause of left axis deviation in these or other cases. Left axis deviation of left ventricular hypertrophy represents a type of "parietal block" in the more distal parts of the left ventricular conduction network and is perhaps the result of the myocardial fibrosis that accompanies marked left ventricular hypertrophy.

Seventeen subjects with no evidence of myocardial infarction or left ventricular hypertrophy at autopsy had marked left axis deviation. The relatively advanced average age of these patients and the presence of myocardial fibrosis in the majority of them suggest that this is due to a conduction defect similar to that seen in left ventricular hypertrophy and related perhaps to the chronic coronary artery disease that was present. Six cases with severe pulmonary disease had marked left axis deviation, possibly due to "verticalization" of the electric field of the heart by the reduced electric conductance of the emphysematous lung.

Among 15 cases with LAD and R' deflections in precordial leads V1 and V2, over half were instances of anterolateral peri-infarction block, and this appears to be the commonest cause of R' deflections in the precordial leads in LAD. The remaining cases were equally divided among cases of severe pulmonary disease, cases of left ventricular hypertrophy, and elderly patients with chronic coronary artery disease.
That the terminal QRS forces can occasionally be somewhat anteriorly directed in cases of left axis deviation due to left ventricular hypertrophy or chronic coronary artery disease, is considered further evidence that the left axis deviation in these cases is due to a parietal block in the left ventricle, analogous to that seen in anterolateral peri-infarction block.

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SUMMARIO IN INTERLINGUA

Le base del presente reporto es 672 casos consecutive in que electrocardiogrammas habeva essite obtenite intra 5 septimanas ante le morte e in que protocollos autoptic esseva disponibile. Casos de patientes de infra 30 annos e de patientes con intervallos QRS de un duration de 0,12 sec o plus habeva essite excludite.

Un tertio de omne casos con deviation axial sinistrostrorse involveva constatationes autoptic de infarimento myocardial. De 160 casos demonstrat de infarimento, 67 habeva deviation axial sinistrostrorse, e duo tertios de istos habeva le deformitate de QRS que es caracteristic de infarimento anterolateral. Ergo, deviation axial sinistrostrorse es considerabilemente plus frequente in infarimento anterolateral que in ulle altere typo de infarimento o in ulle altere categoria individual de morbo cardiac. Es monstrate que le mechanismo del deviation axial sinistrostrorse in iste typo de infarimento es un bloco peri-infarimental, caraterisate per un diagnostic angulo obtuse inter le fortias de QRS initial e terminal. Es signalate 4 casos de infarimento demonstrat in que iste angulo esseva diagnosticamente obtuse sed in que nulle del derivationes conventional monstrava diagnostic “undas Q.” Isto es un configuration electrocardiographic de infarimento que ha non previamente essite recognoscite.

Deviation axial sinistrostrorse esseva notate in minus que un medietate del casos con demonstrate hypertrophia ventricular. Ni le severeitate del hypertrophia ni le position anatomic del corde intra le thorace ni le conformation corporee del patiente ha un rolo significative in le disveloppamento de deviation axial sinistrostrorse in iste subjectos. Incomplete bloco de branca sinistre es un causa rarissime de deviation axial sinistrostrorse in tal o altere casos. Deviation axial sinistrostrorse de hypertrophia sinistroventricular representat un typo de “bloco parietal” in le portion plus distal del rete de conduction sinistro-ventricular e es forsan le resultato del fibrosis myocardial que accompania marcate hypertrophia sinistro-ventricular.

Dece-septe individuos sin evidentia autoptic de infarimento myocardial o de hypertrophia sinistro-ventricular habeva marcate grades de deviation axial sinistrostrorse. Le relativemente alte etate median de iste patientes e le presentia de fibrosis myocardial in le majoritate de illes pare indicar que isto es debite a un defecto de conduction simile al defecto observate in hypertrophia sinistro-ventricular e relationate, forsan, al presentia de chronic morbo de arteria coronari. Sex casos con sever morbo pulmonar habeva marcate deviation axial sinistrostrorse, possiblemente in consequentia de “verticalisation” del campo electric del corde per le reduceite conductantia del pulmone emphysematose.

Inter 15 casos con LAD con deflexiones de R’ in le derivation precordial V₁ e V₃, plus que un medietate eseva casos de bloco peri-infarimental anterolateral. Iste bloco appareva le plus communemente causa de deflexioner de R’ in le presentia de deviation axial sinistrostrorse. Le remanente casos representava in numeros equal sever morbo pulmonar, hypertrophia sinistro-ventricular, e patientes de etate avantiate con chronic morbo de arteria coronari. Le facto que le terminal fortias de QRS in casos de deviation axial sinistrostrorse es a vices orientate levemente in un direction anterior, debite a hypertrophia sinistro-ventricular o chronic morbo de arteria coronari, es considerate como un proba additional que le deviation axial sinistrostrorse in iste casos es causate per un bloco parietal in le ventriculo sinistre, de manera analoge a lo que es incontrate in bloco peri-infarimental anterolateral.
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