The Electrocardiogram in Infarction of the Lateral Wall of the Left Ventricle

A Clinicopathologic Study

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This reappraisal of the electrocardiographic manifestations of infarction of the lateral wall of the left ventricle is based on data acquired by simple and conventional technics. Categories of cases have been determined by the age of the infarct and the distribution of the lesion with respect to the circumference of the left ventricular wall and its epicardial and endocardial boundaries. Significant data have been presented in the form of illustrative diagrams and reproductions. The results do not establish a simple pattern common to all cases of infarction of the lateral wall; they do illustrate the varied yet limited electrocardiographic consequences of such lesions.

TWO notable facts justify reporting the results of another study of infarction of the lateral wall of the left ventricle. First, whereas the electrocardiographic expressions of infarction of the anterior and posterior walls of the left ventricle have been clearly defined and amply confirmed, the changes that attend infarction of the lateral wall have evaded precise and conclusive treatment. Second, the justifiable suspicion persists that the lateral wall of the left ventricle is the site of many infarcts that escape electrocardiographic detection, not only in their healed but also in their acute phase. Both these circumstances may be held to be reasonable consequences of those respects in which the lateral wall differs, so far as its contribution to the scalar electrocardiogram is concerned, from the anteroseptal and posterobasal portions of the left ventricle. The lateral portion of the free wall of the ventricle is so disposed as to preclude ready exploration by semidirect leads; in many instances, it appears to be relatively silent so far as the extremity leads are concerned.

Background

An appraisal of certain published accounts of the electrocardiographic expressions of infarction of the lateral wall will serve to identify the current state of knowledge concerning this problem.

The study of Wood and associates,1 in 1938, directed attention to the possibility of an electrocardiographic pattern of infarction of the lateral wall. Their observations, based on limited electrocardiographic exploration, included some points that have borne up under the test of time and others that have led perhaps to some confusion in past years. Significant were their suggestions that (1) some types of myocardial infarction produce a paucity of electrocardiographic evidence as compared with other types, (2) the QRS complex is usually unaffected by infarction of the lateral wall, and (3) the electrocardiographic signs of a lesion in the lateral wall may disappear rapidly and completely. However, their pathologic data included only a single case with typical findings of infarction of the lateral wall at necropsy, a second case with supportive but not definitive findings, and a third case in which they noted that the clinicopathologic correlation was rendered uncertain by the 14-month interval between the clinical suggestion of infarction and the necropsy. From this evidence they proceeded to the generalization that electrocardiographic study of infarction in other parts of the heart indicates

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Abridgment of thesis submitted by Dr. Dunn to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Medicine.

540 Circulation, Volume XIV, October, 1956
that myocardial ischemia in a certain location tends to produce a characteristic electrocardiographic pattern: "Thus it is reasonable to suppose that the 17 cases without necropsy were instances of lateral infarction."

Depression of the RS-T segment in leads I, II, and, particularly, IVr was proposed by Wood and co-workers as a characteristic finding in infarction of the lateral wall of the left ventricle. Currently accepted concepts would suggest that depression of this segment in the precordial electrocardiogram may occur in the presence of acute subendocardial injury affecting the anterior, apical, or lateral wall of the left ventricle. Existence of such a finding in a lead from the left portion of the precordium may well be an expression of subendocardial infarction of the lateral wall of the left ventricle but is not a change specific to a lesion in this location only. Thomson and Feil reported electrocardiographic changes of this type in 4 of their 9 cases of recent infarction of the lateral wall, and Myers and associates found 4 such instances among their series of 28 cases of infarction of the lateral wall. However, the electrocardiographic changes in 1 of these 4 may have been produced by a combination of left ventricular hypertrophy and the effects of digitalis.

Thomson and Feil, in 1944, recorded observations on a much larger collection of pathologic material. Interpretation of their data is rendered difficult by the fact that in most instances recent infarction of the lateral wall was attended by old infarction of the anterior or posterior wall or by infarction of the anterior or posterior wall continuous with infarction of the lateral wall.

Shaffer, in 1944, reported 5 cases of acute infarction of the lateral wall, giving concisely presented evidence that can be interpreted readily because of the homogeneity of the lesions involved. In all 5 cases, the infarcted region apparently was transmural and was reasonably well confined to the lateral wall of the left ventricle; the data, both electrocardiographic and pathologic, were secured during the acute or healing phase of the lesion. While the electrocardiographic studies were incomplete by present-day standards with respect to the number of leads recorded, the observed changes deserve summary. Two of the 5 cases were characterized by the presence of electrocardiographic changes ordinarily associated with injuries of both anterior and posterior disposition, 1 showed predominantly posterior features (Q wave and elevated S-T segment in lead III attended by depression of S-T segment in lead I), 1 showed only inverted T waves in lead I, and 1 revealed only absence of the R wave in lead IV. Shaffer concluded that not one of his cases showed the electrocardiographic features typical of infarction of the lateral wall as defined by Wood's group, and that a single consistent pattern was absent.

In 1946, Rosenbaum and associates presented the results of a purely clinicoelectrocardiographic study on the precordial electrocardiogram in "high" lateral myocardial infarction. They contributed significantly to knowledge of the electrocardiographic consequences of infarction of the lateral wall without reporting the findings at necropsy in a single case. Pathologic correlation was confined to the statement, "We have recently heard of instances of high lateral infarction demonstrated at autopsy in which the standard extremity and precordial electrocardiograms resembled those described in this article." While they recognized the significant role that rotation of the heart or some other change in the relations of its surfaces to the usual leads must play in producing the electrocardiographic changes, they considered infarction of the lateral wall to be the type that produces characteristic findings in precordial leads V4, V6, and V7, or in leads from the upper left part of the precordium or the left axilla. A question remained, however, concerning the consistency with which such changes appeared in infarction of the lateral wall.

In 1945, Hecht presented the results of a study of 86 cases of infarction of the lateral wall. To our knowledge, his work appears in the literature only in abstract form and appraisal of his data is rendered somewhat difficult by the summarized character of the presentation.

Myers and associates, in 1949, reported their findings in 27 cases of "primary" lateral in-
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Fig. 1. Diagrams of sections of the heart on which are indicated the several components of the left ventricular wall. The sections of heart in this and all other figures in this study are viewed from above. (Originally from Achor. Reproduced with the permission of the publisher from: McQuay, N. W., Edwards, J. E., and Burchell, H. B.: Type of death in acute myocardial infarction. Arch. Int. Med. 96: 1, 1955.)

infarction. Their results are elaborately documented and deserving of careful review. Although exact appraisal of the point is difficult, the impression remains that the group of 14 cases of “high lateral wall infarction,” as that term was used in their study, corresponds in the main to the term “lateral infarction” as applied in our study. Among the 14 high lateral infarcts in their series, 5 lesions were transmural and 8 were subendocardial. In only 1 of these was there a QR pattern in lead V₅ or V₆. They stated, “On the other hand, lead aV₁ yielded a QR pattern which was considered diagnostic of lateral infarction in two cases, strongly suggestive in five cases and suspicious in four cases.”

Definition of Terms

Figure 1 presents diagrammatically the division of the heart into those component regions to which reference will be made hereafter. The anterior wall of the left ventricle is bounded medially by the ventricular septum and laterally by a line bisecting the anterior papillary muscle on its long axis. The posterior wall lies between the anterior border of the posterior papillary muscle and the junction of the free wall with the ventricular septum posteriorly. The lateral wall, therefore, includes the posterior half of the anterior papillary muscle and extends to the anterior border of the posterior papillary muscle.

The term “subendocardial infarction” refers to lesions involving the subendocardial portion of the myocardium and extending through as much as three fourths of the thickness of the ventricular wall but sparing always the epicardial layers.

Material

Cases included in this study were derived from among necropsies performed at the Mayo Clinic between 1947 and 1955. Inclusion of a case in the study was deemed appropriate if infarction of the lateral wall of the left ventricle or scarring thereof existed and if electrocardiograms were available that had been recorded at a time judged to be subsequent to occurrence of infarction. Of a series
of 47 cases fulfilling these criteria, 17 were discarded because existence of a separate infarct of the anterior wall rendered impossible the reliable ascription of electrocardiographic changes to infarction in the lateral wall. However, all cases in the following categories were retained: (1) acute transmural infarction confined to the lateral wall (6 cases); (2) acute transmural infarction of the lateroposterior wall (6 cases); (3) acute subendocardial infarction confined to the lateral wall (1 case); (4) healed subendocardial infarction confined to the lateral wall (13 cases); and (5) healed transmural infarction confined to the lateral wall (1 case). In addition to these 27 cases, 3 selected cases were retained because the sequence of electrocardiographic changes permitted reasonably exact correlation between alterations in these records and the morphologic findings. These 3 included an instance of healed transmural anteroseptal infarction complicated by acute subendocardial infarction of the lateral wall, 1 of healed transmural posterolateral infarction complicated by acute anteroseptal infarction, and 1 of old subendocardial posterolateral infarction.

The 17 cases eliminated were instances of infarction of the lateral wall of the left ventricle combined with separate anterior or anteroseptal infarcts (12 cases) and examples of subendocardial infarction of the posterolateral wall of the left ventricle (5 cases). Interpretation of data was rendered difficult in the majority of these rejected cases either because infarction of the lateral wall was associated with infarction involving other regions in such a way that a reliable correlation between the electrocardiographic changes and pathologic findings could not be formulated or because of uncertainty that the electrocardiogram was recorded after development of the infarction in the posterolateral wall of the ventricle.

An attempt has been made in this selection to strike a compromise between the dangerous distortion of evidence that may result from injudicious choice and the confusion of detail that may obtain in the total absence of discriminating selection.

**Results**

The results of this study can be comprehended most readily in the form of illustrations. Figures 2 through 8 present the essential electrocardiographic and morphologic data. The legends include certain clinical and pathologic facts. Whenever possible, a single illustration has been used to represent 2 or more cases constituting a type as defined by the correlation of electrocardiographic and pathologic findings.

**Acute Transmural Infarction Confined to Lateral Wall of Left Ventricle.** Cases 1 through 6 were of this type (figs. 2–4). Review of the illustrations discloses that 1 case (case 1) presented electrocardiographically as a "pure lateral," 2 (cases 2 and 5) as "apicolaterals," 1 (case 3) as a "postero-apical" and 1 (case 4) as a "posterolateral"; the electrocardiogram in 1 (case 6) was essentially normal.

QRS changes, when present at all, were of limited extent. In cases 1 and 2, tiny Q deflections were present in leads I and aV_L, and the R deflections in V_L were increased in size in relation to the S waves. In cases 3 and 4, Q waves were present in leads II and III, but these were small in case 3 and were preceded inconstantly in case 4 by a small upward deflection. In case 5, the R waves in leads I and V_L were notched on the descending limb.

Significant changes in S-T segments and T waves were present in 5 of these 6 cases. Segmental elevation in leads from regions facing the epicardial surface of the infarcted zone was attended commonly by depression of segments in leads overlying uninvolved epicardial regions. This reciprocal relationship obtained also for configuration of T waves and afforded evidence suggestive of an injury, such as myocardial infarction, confined to a local region of the ventricular wall, as opposed to a more widely distributed epicardial lesion, such as acute pericarditis. In the presence of a more generalized epicardial lesion, a similar reciprocal relationship may be encountered but the "bracketing leads" ordinarily face an epicardial surface on the one hand and an endocardial surface on the other. In the single instance (case 6) in which electrocardiograms made at the time of acute infarction disclosed no certain evidence of myocardial injury, the infarct was small and confined to a single slice of the sectioned heart.

In one instance (case 5) in which tracings were obtained 6 years after acute infarction, definitive electrocardiographic evidence of a scar was absent. T waves in lead I were of lesser amplitude than in lead III. The fact that the major evidences of injury were confined to the S-T segments and T waves allows reasonable prediction that similarly complete resolution of electrocardiographic changes encountered in the acute stage in 4 of
the other 5 cases would have occurred had the patients survived the episode.

Five of these 6 patients died during the acute phase of myocardial infarction, the immediate cause of death being rupture of the left ventricular wall. The sites of rupture are shown by the heavy black lines and arrows in figures 2, 3, and 4.

**Acute Transmural Infarction of Latero-posterior Wall of Left Ventricle.** Cases 7 through 12 were of this type (figs. 5 and 6).

The arrangement of data in figures 5 and 6 was designed to emphasize an interesting relationship between the location of the myocardial infarct and the nature of the electrocardiographic findings. The lesion in case 7 showed the most extensive invasion of the posterior wall as compared with the lateral wall of the left ventricle. The vector drawn in slice 2 of the cross-sectional diagrams indicates the essentially posterior orientation of the lesion. Correspondingly, both this case and its counterpart (case 8) display the most definitely posterior orientation of the electrocardiographic changes, which are pronounced in degree and which are manifest in aberrations of the QRS, the S-T segment, and the T-wave components of the ventricular complexes. In contrast, the lesion in case 12 was a large infarct of the lateral wall of the left ventricle that invaded to a limited degree the adjacent posterior wall. The vector hypothesized from
strictly morphologic considerations approximates a lateral direction. The electrocardiographic changes are slight in degree and have that ambivalence with respect to anterior and posterior orientation that a priori might be held appropriate to infarction of the lateral wall. Cases 9 and 11 were regarded as gradations between cases 7 and 12 on the basis of both morphologic and electrocardiographic evidence.

Perhaps this particular arrangement of data may be held to emphasize unduly the relationship of comparatively minor variations in distribution of infarcted myocardium to the electrocardiographic expressions of the lesion. However, the intent is to illustrate not an absolute association but a general and relative one.

**Acute Subendocardial Infarction Confined to Lateral Wall of Left Ventricle.** Only 1 lesion of this type was encountered. The patient survived the attack and succumbed a year later to a cerebrovascular accident. Electrocardiograms made at the time of the acute episode are reproduced in figure 7 (case 13). The changes are of minor degree and affect only the S-T segments and T waves in a manner suggestive of apicolateral injury. The scarred region, as indicated in the cross-sectional diagrams, approached transmural proportions in the second and third slices.

The findings in this case thus are similar to

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**Fig. 3. Case 3.** Acute transmural infarction of the lateral wall in a 79-year-old woman, with onset of symptoms of infarction 1 week before death. The estimated age of the infarct on the basis of pathologic changes was 1 to 2 weeks. The electrocardiogram dated 6-20-50 was recorded on the day symptoms of infarction began and 1 week before death. Note increase in height of R wave in V1, elevation of S-T segments in II, III, and aV\(_F\), depression of these segments in I, aV\(_L\), V\(_1\), V\(_2\), and V\(_3\), inversion of T waves in V\(_4\) and V\(_6\), and upward peaking of T waves in V\(_1\), V\(_2\), and V\(_3\). These changes were thought to suggest injury of the postero-apical wall of the left ventricle.

**Case 4.** Acute transmural infarction of the lateral wall in a 61-year-old man, with onset of symptoms of infarction 4 days before death. The estimated age of the infarct on the basis of pathologic changes was approximately 1 week. The electrocardiogram was recorded on the day of death. Note Q waves in II, III, and aV\(_F\), preceded inconstantly by a small initial upward deflection. Note also slight elevation of S-T segments in I and aV\(_L\). These changes were considered indicative of posterolateral infarction.
those encountered in acute transmural infarction involving the same region.

Healed Subendocardial Infarction Confined to Lateral Wall of Left Ventricle. Cases 14 through 26 were of this type (fig. 7, cases 14, 16, and 22).

Case 14 had a counterpart in 1 other case with respect to distribution of the scar of the myocardial infarct and the nature of the electrocardiographic findings. In these 2 cases, a subendocardial scar extending from apex to base of the lateral wall of the left ventricle was

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**Fig. 4. Case 5.** Acute transmural infarction of the lateral wall in a 60-year-old man, with onset of symptoms of infarction 2 days before the electrocardiogram dated 10-18-44 and 7 years before death. At necropsy, only an old scar was present. Note elevation of S-T segments in leads I and IVR in the electrocardiogram of 10-18-44, suggestive of apicolateral injury. The tracing dated 12-12-51 provides no definitive evidence of the myocardial scar found at necropsy.

**Case 6.** Small acute transmural infarct of the lateral wall in a 78-year-old woman, with onset of symptoms of infarction 1 day before death. The estimated age of the infarct on the basis of pathologic changes was 1 to 2 days. The electrocardiogram was made within a few hours after the onset of symptoms and on the day before the patient died. Changes definitive of infarction were not identified. The S-T segments in V4 and V5 are slightly depressed.

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**Fig. 5. Case 7.** Acute transmural infarction of the posterolateral wall in a 59-year-old man, with onset of symptoms of infarction 24 days before death. The estimated age of the infarct on the basis of pathologic changes was 3 to 4 weeks. The electrocardiogram dated 9-15-52 was made 12 days after the onset of symptoms and 12 days before death. The changes are those of acute posterocostal infarction. In the record of 9-18-52, these changes have evolved toward a subacute phase. Electrocardiographic and pathologic changes of similar character were encountered in case 8, not illustrated here.

**Case 8.** Acute transmural infarction of the posterolateral wall in a 77-year-old man, with onset of symptoms of infarction 5 days before death. The estimated age of the infarct on the basis of pathologic changes was 1 week. The electrocardiogram dated 8-16-53 was made 3 days after the onset of symptoms and 2 days before death. Note tiny Q in II and QS deflections in III and aVR, increased height of R and loss of S wave in V1, slight elevation of S-T segments in II, III, and aVR, depression of S-T segments in V1, V2, V3, and V4, and upward peaking of T deflections in these same precordial leads. These changes were regarded as strongly suggestive of posterior myocardial infarction. Similar electrocardiographic and pathologic findings were present in case 10.
Case 7

Case 9

Fig. 5
attended by small Q deflections in leads II and III in electrocardiograms recorded at a time remote from occurrence of the infarct. These tracings were judged to afford evidence suggestive of old posterior myocardial infarction.

Case 16 had counterparts in 5 other cases. In each instance, a subendocardial scar in the lateral wall of the left ventricle was attended by electrocardiographic evidence that in some way was suggestive but not definitive of old apical or apicodorsal infarction. In cases 16, 17, and 18, Q deflections were present in standard leads I and II, and small Q deflections were present also in precordial lead V₂ in cases 16 and 17. Small Q deflections in V₅ followed by R waves unusually small for this point on the precordium were present in case 19. As possible evidence of an old scar, only a QR deflection in extremity leads aV₅R and aV₅L was noted in case 20. In case 21, the final member of this group, increased height of the R deflections in V₁ was the only suggestion of possible loss of myocardial mass in the wall of that ventricle contralateral to the one over which the precordial electrode was placed.

Case 22 had counterparts in 4 other cases. In

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![Image](https://circ.ahajournals.org/)

**Fig. 6. Case 11.** Acute transmural infarction of the lateroposterior wall in a 56-year-old man, with onset of symptoms of infarction 10 days before death. The estimated age of the infarct on the basis of pathologic changes was 2 weeks. The electrocardiogram was made 5 days after onset of symptoms and 5 days before death. Note QS deflection in III and tiny Q in aV₅L, slight elevation of S-T segments in II, III, aV₆, V₃, and V₄, depression of these segments in V₂ and V₃, and shallow inversion of T waves in II, III, aV₆, V₃, and V₄. These changes were regarded as suggestive of postero-apical infarction.

**Case 12.** Acute transmural infarction of the lateroposterior wall in a 62-year-old man, with onset of symptoms of infarction 23 days before death. The estimated age of the infarct on the basis of pathologic changes was 2 to 4 weeks. The electrocardiogram dated 11-27-51 was made on the day symptoms of infarction developed. Note slight elevation of S-T segments in I and aV₅L, depression of these segments in II, III, aV₆, V₃, V₄, and V₅, and inversion of T waves in III and aV₅L. These changes were regarded as suggestive of lateral infarction. In the tracing of 12-19-51, inversion of T waves existed in I, II, III, and V₄. Significant QRS changes did not develop, although the infarcted region was large and extended into the posterior wall of the left ventricle; the electrocardiographic changes were of minor degree and were similar to those associated with infarction confined to the lateral wall.
each instance, a subendocardial scar in the lateral wall of the left ventricle was associated with electrocardiographic findings judged to be within the range of normal.

Thus, electrocardiograms made at a time remote from the period when the lesion was acute in these 13 cases revealed evidence suggestive of posterior scarring in 2 cases, showed changes classed as possible but far from definitive evidence of apical or lateral scarring in 6 cases, and were normal in 5 cases.

**Healed Transmural Infarction Confined to Lateral Wall of Left Ventricle.** Only 1 instance of this type was encountered (fig. 8, case 27). A tiny Q deflection 1 mm. in depth in leads II and III constituted the only evidence suggestive of a myocardial scar, although pronounced scarring attended by thinning of the left ventricular wall was present.

**Selected Cases.** As already noted, 3 cases were retained from 2 categories otherwise deleted from the study.

**Old Posterolateral Infarction with Post-infarction Block.** In 2 of the 3 selected cases, the electrocardiogram revealed changes held to be characteristic of postinfarction block. Illustrated in case 28 of figure 8 are the electrocardiograms and cross-sectional diagrams in 1 of these 2. These tracings were recorded before the acute anterior infarction, and the Q waves in leads II and III, together with the prolonged QRS interval, may be ascribed to the consequences of the old transmural posterolateral myocardial infarct. The findings in case 29 were nearly identical as to both electrocardiographic changes and the pertinent morphologic lesion; the latter consisted of an old posterolateral infarct that was classed as subendocardial but that approached transmural extent.

These 2 cases provide a small but perhaps substantial basis on which to rest speculation regarding the nature of postinfarction block. In leads II and III, and predictably aVF, the final component of the widened QRS complex is an R wave. Other studies, in which esophageal leads were used, support the view that this R deflection is an expression of delayed excitation of the ventricular myocardium in the posterolateral region of the heart.a A posterolateral infarct of the type encountered in cases 28 and 29 must disturb the spread of the excitation process in such a manner that its arrival at the posterobasal fibers is delayed or its spread into them is accomplished in an anomalous fashion.

**Acute Subendocardial Infarction of Lateral Wall of Left Ventricle with Old Transmural Anteroseptal Infarction and Left Bundle-Branch Block.** This complex of morphologic and functional phenomena is presented as an excellent example of events occurring within nature in an order that precisely and definitely tests existing concepts concerning bundle-branch block and the electrocardiographic consequences of superimposed myocardial infarction.

In case 30 of figure 8, the electrocardiogram of December 10, made in all probability before the acute lateral infarct, revealed left bundle-branch block unusual in the presence of a Q deflection in leads I, aVL, V5, and V6. The presence of such a deflection suggests a loss of septal myocardium, a situation later confirmed in this case by the finding at necropsy of an old anteroseptal myocardial infarct. The final component of the QRS complexes in leads V5 and V6 in this same electrocardiogram is an R wave, the origin of which is ascribed to excitation of the apicolateral wall of the left ventricle. In the other 3 electrocardiograms illustrated in this case, the R waves diminish progressively in height at a time when clinical and pathologic findings indicated the incidence of infarction involving the myocardium of that same lateral wall.

**Discussion**

The principal objective of this article has been to present as lucidly as possible a correlation of electrocardiographic and pathologic data in cases of infarction of the lateral wall of the left ventricle with and without involvement of adjacent regions. Certain comments already have been made. A condensation of results will be undertaken in the section devoted to summary and conclusions. Remaining for comment are 3 points, 2 of which develop naturally from appraisal of the total body of evidence; the other appears as a rather surprising and, in some measure, a chance development.
FIG. 7. Case 13. Acute subendocardial infarction of the lateral wall in a 62-year-old man, with onset of symptoms of infarction 8 days before the electrocardiogram dated 7–8–52 was recorded. The patient survived the acute episode and died in June 1953, of thrombosis of the basilar artery. A subendocardial scar as indicated in the cross-sectional diagrams was observed at necropsy. In the tracing of 7–8–52, note elevation of S-T segments in I and V₅, and depression of this segment in III. In the record dated 7–15–52, note inversion of T waves in I, aV₆, V₇, and V₈. Additional leads from the third interspace provided no information of novel character. These changes were regarded as suggestive of apicolateral infarction.

Case 14. Old subendocardial infarction of the lateral wall in a 76-year-old man who had angina pectoris for 2 years prior to death. A subendocardial scar as indicated in the cross-sectional diagrams
1. In the identification of the presence of acute or healed infarction of the lateral wall of the left ventricle, the question is raised as to how critical a factor is the adequacy of the electrocardiographic exploration in terms of number of leads recorded. Rosenbaum and co-workers proposed that additional leads should be recorded from points on the left portion of the precordium higher than the conventional ones when abnormal findings appear in the left precordial leads or in lead aVL. Myers and associates observed that the electrocardiograms frequently were normal in a series of 27 cases of infarction of the lateral wall, except for a Q deflection regarded by these authors as significantly abnormal in aVL. Our clinical experience would lead us to support the desirability of recording the potentials from the left arm in any patient suspected of having myocardial infarction. Additional leads from the upper left part of the precordium and left axilla have not proved especially helpful inasmuch as the evidence supplied by them commonly has been similar to that obtained from the conventional precordial leads and aVL. If the latter were abnormal, the same kind of abnormality appeared in the added precordial leads, but these latter leads rarely clarified appreciably the diagnostic problem. If the conventionally derived leads were normal, so were the additional leads. Exceptions to these generalizations exist, and a reasonable question is whether the frequency of their occurrence justifies encouraging physicians to obtain additional leads that must be interpreted cautiously because of their unfamiliar character.

Review of our series of cases supports the following observations. Acute transmural infarction commonly produced segmental and T-wave changes that were minor in degree but were distributed through several conventional leads. Use of additional leads in cases of this type offers scant prospect of clarifying significantly the diagnostic problem. In contrast, evidences of an electrocardiographic relic of an ancient subendocardial infarct of the lateral wall either did not exist or were restricted to QRS changes of minor degree limited at times to a single lead among the conventional series. Among this latter category of cases, use of lead aVL may be regarded as essential to adequate electrocardiographic exploration, and the recording of additional leads from the upper left region of the precordium is desirable when minor changes in the QRS complexes are encountered in leads I, aVL, V5, and V6.

2. Included among our illustrations are 2 instances in which the R wave in lead V1 was unusually tall in relation to the depth of the S wave (fig. 2, case 1; fig. 5, case 9) and 2 others in which the R deflection in lead V1 was taller in tracings made after infarction of the lateral wall had occurred than it was prior to infarction (fig. 2, case 2; fig. 3, case 3).

Case 9 is a striking example of this electrocardiographic finding and an excellent model for illustrating its origins. As seen in the cross-sectional diagrams (fig. 5), the mass of infarcted myocardium in the posterolateral wall was located opposite to that portion of the anterior...
Fig. 8. Case 27. Healed transmural infarction of the lateral wall in a 79-year-old man who presented no history of myocardial infarction. A transmural scar distributed as indicated in the cross-sectional diagrams was found at necropsy. The electrocardiogram was recorded 1 month prior to death. Note tiny Q waves in leads II and III and inversion of T waves in leads I and V1. These changes afforded only inconclusive evidence of myocardial infarction.

Case 28. Old transmural infarction of the lateroposterior wall with electrocardiographic evidence of postinfarction block in a 58-year-old man who gave no history of myocardial infarction. The
wall of the heart over which the exploring electrode in lead V₁ commonly is placed. If the forces produced in electric systole by the posterolateral wall prior to infarction were represented by the vector shown in the second of these diagrams, then elimination of these forces consequent to infarction of the myocardium in that region would leave unopposed the forces produced in that portion of the anterior wall of the heart underlying V₁. Under such circumstances, the QRS complex in lead V₁ might become a monophasic R wave.

Unfortunately, alteration in the height of a normally occurring R wave may not be particularly helpful in establishing the presence of myocardial infarction. Perhaps vectorcardiographic study of this disturbance in the normal balance of electric forces may afford a means of using the phenomenon to greater diagnostic advantage.

3. A third point warrants comment, even though it bears on a matter apart from the correlation of pathologic and electrocardiographic data that is the theme of this article. As mentioned previously, death occurred in 5 of the 6 cases in which electrocardiograms were made during acute transmural infarction of the lateral wall, and the cause of death in every case was rupture of the left ventricular wall. Review of the cross-sectional diagrams of the heart in these cases discloses that the site of rupture in cases 1, 3, and 4 was approximately in the center of the infarcted myocardium at a point between the anterior and posterior papillary muscles where the ventricular wall is less thick. The rupture in case 6 was in the same general location but disposed 1 slice toward the apex; in case 2, it was at the junction between infarcted and uninfarcted myocardium along the anterior margin of the infarct.

Summary and Conclusions

Pertinent electrocardiographic and pathologic findings have been correlated in 30 cases of myocardial infarction involving the lateral wall of the left ventricle studied at the Mayo Clinic.

In acute transmural infarction confined to the lateral wall of the left ventricle, electrocardiograms made up of 6 leads (standard limb leads and precordial leads V₁, V₃, and V₅) or 12 leads (the 6 just mentioned plus unipolar extremity leads and precordial leads V₂, V₄, and V₆) recorded in the conventional manner commonly disclosed evidence of acute myocardial injury. QRS changes of magnitude and character sufficient to be definitive of myocardial infarction rarely developed. Segmental elevation and subsequent inversion of T waves commonly occurred in leads presumably so oriented as to face the epicardial aspect of the infarcted myocardium. However, the disposition of these changes varied among the several cases, suggesting in one or more instances apical injury (changes in leads V₄ and V₆), posterior injury (changes in leads II, III, and aV₅) or lateral injury (changes in leads I and aV₆) or some combination of 2 or more of these variants. No consistently recurring “pattern” could be derived.

When acute transmural infarction involved both the lateral and posterior portions of the wall of the left ventricle, the electrocardiogram was recorded 8 days prior to death and before development of the acute myocardial infarct outlined in the cross-sectional diagrams. The significant correlation, therefore, involved the electrocardiogram and the old infarct of the lateroposterior wall. Note a QRS interval of 0.12 sec, Q deflections in leads II and III, S deflections in leads I and V₁, and absence of evidence of right bundle-branch block in V₁. Electrocardiographic and pathologic changes of similar character were encountered in case 20.

Case 30. Healed transmural anteroseptal infarction and acute subendocardial lateral infarction in the presence of left bundle-branch block in a 61-year-old woman who had a history of angina pectoris for 6 months. The onset of symptoms of acute myocardial infarction was on December 13, 1952, and death occurred on December 20. The age of the acute infarct as estimated at necropsy was 5 to 7 days. The electrocardiogram dated 12–10–52 reveals a QRS interval in excess of 0.12 sec. The Q waves in leads I, aV₆, and V₄ are related to the old anteroseptal infarct in the presence of left bundle-branch block. The progressive diminution in height of the final R deflection in V₁ and V₄ in subsequent tracings resulted from and was pathognomonic of infarction of the lateral wall.
graphic changes assumed a posterior character to a degree roughly proportional to the extent of involvement of the posterior ventricular wall.

As might be predicted from the character of the electrocardiographic changes encountered in the acute stage of transmural infarction of the lateral wall, the electrocardiographic relics attending healed lesions of that wall were of minor extent or were absent. Among electrocardiograms made in 13 cases of healed subendocardial infarction of the lateral wall of the left ventricle, 2 revealed evidence suggestive of posterior scarring, 6 presented changes classed as affording possible evidence of apical or lateral scarring, and 5 were regarded as normal. According to the limited data in this study, only among cases in which routine electrocardiograms are suggestive of apical or lateral scarring would there appear to be an indication for securing additional leads from the upper left portion of the precordium or the left axilla.

Two cases of healed lateroposterior infarction attended by electrocardiographic evidence of postinfarction block were encountered.

A single case of acute subendocardial infarction of the lateral wall of the left ventricle combined with old transmural anterosetal infarction in the presence of left bundle-branch block presented a basis for the evaluation of currently accepted concepts concerning bundle-branch block and the electrocardiographic consequences of superimposed myocardial infarction.

A change in QRS configuration peculiar to lesions of the lateral wall as contrasted to myocardial infarcts of other localizations is an increase in height of the R deflection in precordial leads centered around position 1. The diagnostic value of this finding is reduced by the fact that it is a quantitative rather than a qualitative change.

Infarcts of the lateral wall of the left ventricle, particularly in their acute phases, are not electrocardiographically "silent"; their voice is, however, one of weak and mingled tones.

**SUMMARIO IN INTERLINGUA**

Pertinente constataziones electrocardiographic e pathologic essea correlationate in 30 casos de infarimento myocardial involvente le pariete lateral del ventriculo sinistre, studiate al Clinica Mayo.

In casos de acute infarimento transmural restringite al pariete lateral del ventriculo sinistre, electrocardiagrammas consistente de 6 derivationes (le derivationes standard de extremitate e le derivationes precordial V1, V5, e V6) o de 12 derivationes (le 6 derivationes justo mentionate, le derivationes unipolar de extremitate, e le derivationes precordial V2, V4, e V6), omnes registrate in le maniera conventional, revelava generalmente manifestaciones de acute lesion myocardial. Se desenvolpava rarmente alterationes de QRS de magnitude e de character sufficiente pro esser interpretate como signos definitive de infarimento myocardial. Elevation segmental e inversion subsequente del undas T occurreva communemente in derivationes que essea supponitemente orientate de maniera a esser confrontate con le aspecto epicardial del infarite myocardio. Tamen, le disposition de iste alterationes variava ab un caso al alteres, suggesterent—pro un o plure patientes—lesion apical (alterationes in derivation V5 e V6), lesion posterior (alterationes in derivation II, III, e aVr), lesion lateral (alterationes in derivation I e aVL), o le un o le altere combination de 2 o plures de iste variantes. Il non essea possibile derivar un specific "configuration" de recurrentia regular.

In casos in que le infarimento transmural involveva le portiones tanto lateral como etiam posterior del pariete del ventriculo sinistre, le alterationes electrocardiographic exhibiva un character posterior de grados grossiermente proportional al mesura de involvimento del pariete postero-ventricular.

Viste le character del alterationes electrocardiographic incontrate in le stadio acute del infarimento transmural del pariete lateral, il non essea un surprisa notar que le reliquias electrocardiographic associate con curate lesiones de ille pariete essea paucu importante si illos essea presente del toto. Inter le electrocardiogrammas obtenite in 13 casos de curate infarimento subendocardial del pariete lateral del ventriculo sinistre, 2 exhibiva aspectos suggestive de cicatrisation posterior, 6 mons-
trava alterationes que poteva esser interpre-
tate como manifestationes de cicatrisation
apical o lateral, e 5 esser considerate como
normal. Super le base del restringite datos del
presente studio, il pare que le casos in que le
electrocardiogrammas de routine suggere le
presentia de cicatrisation apical o lateral es le
sol casos in que il pot esser indicate obtener
derivationes additional ab le portion supero-
sinistre del precordio o ab le axilla sinistre.
Esseva incontrate 2 casos de curate in-
farcimento latero-posterior con signos electro-
cardiographic de bloco postinfarcimental.
Un sol caso de acute infarcimento sub-
endocardical del pariete lateral del ventriculo
sinistre in combination con ancian infarcimento
transmural anteroseptal e bloco de branca
sinistre offereva un base pro le evulatiun
de currentemente acceptate notiones in re bloco
de branca e le consequentias electrocardio-
graphic de superimponite infarcimento myo-
cardial.
Un alteration del configuration de QRS que
es restringite a lesiones del pariete lateral (in
contrasto con infarcimentos myocardial de
altere locationes) es un augmento de elevation
del deflexion R in derivationes precordial
centrate circa le position 1. Le valor diagnostic
de iste constatation es reducere per le facto que
il se tracta de un alteration quantitative plus
tosto que qualitative.

Infarcimentos del pariete lateral del ventricu-
culo sinistre, specialmente in lor phases acute,
non es electrocardiographicamente “silente,”
seu lor voz ha solmente sonos que es debile e
miscite.

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The Foxglove is a plant sufficiently common in this island, and as we have but one species, and
that so generally known, I should have thought it superfluous either to figure or describe it; had I
not more than once seen the leaves of Mullein gathered for those of Foxglove. On the continent of
Europe too, other species are found, and I have been informed that our species is very rare in some
parts of Germany, existing only by means of cultivation, in gardens.

Our plant is the Digitalis purpurea of Linnaeus. It belongs to the 2d order of the 14th class,
or the Didynamia Angiospermia. The essential characters of the genus are, Cup with 5 divisions.
Blossom bell-shaped, bulging. Capsule egg-shaped, 2 celled.—WILLIAM WITHERING. An Account
of the Foxglove, and Some of Its Medical Uses. Bringham, 1785.
The Electrocardiogram in Infarction of the Lateral Wall of the Left Ventricle: A Clinicopathologic Study
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Circulation. 1956;14:540-555
doi: 10.1161/01.CIR.14.4.540
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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The online version of this article, along with updated information and services, is located on the World Wide Web at:
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