QRS-T Patterns in Multiple Precordial Leads that May Be Mistaken for Myocardial Infarction

I. Left Ventricular Hypertrophy and Dilatation

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The electrocardiograms of patients in whom myocardial infarction was excluded at autopsy are presented to bring out the following features of left ventricular hypertrophy that may be mistaken for myocardial infarction: (1) QS patterns and/or abnormal RS-T elevation in leads from the right precordium; (2) abnormal Q waves, multiphasic QRS and/or cove plane inversion of the T waves in leads from the transitional zone; (3) prominent Q waves, marked RS-T depression and/or sharply inverted T waves in leads from the left axilla.

A DETAILED analysis of the electrocardiographic findings in 161 cases of pathologically established myocardial infarction has been presented, but little attention was devoted to differential diagnosis. Full consideration of the subject requires presentation of cases with electrocardiographic findings suggestive of myocardial infarction but without evidence of such at autopsy.

Fifty cases have been selected for presentation because of abnormalities in the QRS and/or RS-T complex in Wilson precordial leads that might be mistaken for aberrations due to myocardial infarction. In all cases autopsy was performed and in none of the subjects was there pathologic evidence of myocardial infarction or of focal subendocardial myomalacia of the type attributed to coronary insufficiency by Master and associates. Postmortem examination included injection of the coronary arteries with radiopaque mass, roentgenogram, and subsequent dissection according to a previously described technic in 42 of the cases (all except Cases 3, 5, 20, 21, 33, 34, 36, 44). A large number of microscopic blocks was studied in many cases, in order to make certain that the possibility of myocardial infarction could be excluded.

The absence of pathologic evidence of myocardial infarction, however, does not rule out a coronary origin for the electrocardiographic findings. Blumgart, Gilligan, and Schlesinger produced temporary coronary occlusion in dogs and demonstrated electrocardiographic patterns typical of myocardial infarction in animals that showed no gross or microscopic evidence of a myocardial lesion at subsequent necropsy. Bohning and Katz reported one patient with serial changes in Lead CF, considered diagnostic of a rapidly healing anteroseptal infarction, whose autopsy study two years later showed narrowing of both coronary arteries, but no evidence of occlusion or infarction.

A definite electrocardiographic diagnosis of myocardial infarction had been made during life in 7 cases from this series (Cases 12, 17, 27, 28, 31, 38, 41); a diagnosis of probable infarction was made from the first tracing in Cases 11 and 49, and the possibility of infarction could not be positively excluded in several others. Upon reconsideration, after completion of the pathologic studies, it was concluded that the electrocardiographic findings had been misinterpreted in all but one of the foregoing cases and were explicable upon a basis other than myocardial infarction or the situation encountered by Blumgart and associates and by Bohning and Katz. The findings in Case 12 were not satisfactorily accounted for at autopsy and it is believed that an infarct was missed pathologi-
cally because of an inadequate number of microscopic blocks.

The electrocardiographic patterns which may be mistaken for those of myocardial infarction include (1) left ventricular hypertrophy and dilatation, (2) right ventricular hypertrophy, (3) right ventricular dilatation, (4) left bundle branch block, (5) right bundle branch block, (6) alterations in blood potassium, (7) myocardial ischemia, (8) pericarditis and subepicardial myocarditis, and (9) arrhythmias causing QRS distortion. The presentation of a sufficient number of cases for each of the foregoing nine groups was found too voluminous for a single manuscript. As a consequence, the study has been divided into four parts. The electrocardiographic features of left ventricular hypertrophy and dilatation that may be mistaken for those of myocardial infarction are considered in this article; the differentiation from right ventricular hypertrophy and dilatation, in the second; and the features of bundle branch block that may be confused with infarction, in the third; and the diagnostic errors that may be made in the presence of alterations in blood potassium, myocardial ischemia, pericarditis, subepicardial myocarditis, and arrhythmias causing QRS distortion are considered in the final manuscript.

*Electrocardiographic Features Diagnostic of Left Ventricular Hypertrophy*

Left axillary and precordial leads facing the epicardial surface of an hypertrophied left ventricle characteristically display: 511 (a) prominent R wave with slightly prolonged ascending limb and slightly delayed peak, usually preceded by a small Q wave less than 25 per cent of the amplitude of the R; (b) depression of the RS-T junction, accompanied by an inverted, diphase, or flattened T wave. Right precordial leads facing the epicardial surface of the right ventricle or right atrium characteristically display: (a) minute R wave and abnormally deep and prolonged S wave; (b) elevated RS-T junction and tall upright T wave.

*Electrocardiographic Features of Left Ventricular Hypertrophy that May Be Mistaken for Those of Myocardial Infarction*

The precordial electrocardiogram of uncomplicated left ventricular dilatation and hypertrophy may be mistaken for that of myocardial infarction because of: (A) exaggeration of the normal Q wave in left ventricular leads; (B) an RS-T pattern in left ventricular leads, characterized by either: (1) sharp inversion of the T wave accompanied by a slightly elevated or isoelectric RS-T junction instead of the customary slight depression, (2) marked RS-T depression, (3) deeper inversion of the T wave in Lead V4 than in V3 or V5, or (4) rapid changes in serial tracings; (C) QS deflections in Leads V1 and V2; (D) abnormal elevation of the RS-T junction in leads from the right side of the precordium; (E) bizarre QRS patterns in leads from the transitional zone, consisting of: (1) a multiphasic complex, (2) an abnormal initial downstroke; (F) abnormal RS-T complex at the transitional zone, including cove inversion of the T wave. These features are collectively illustrated by the electrocardiograms reproduced in figures 1, 2, and 3, obtained in Cases 1 to 12, inclusive. Autopsy revealed left ventricular hypertrophy without infarction and without coronary occlusion or narrowing in ten of these patients and a normal heart in the other two patients (Patients 2 and 11). The electrocardiograms of the two latter patients were reproduced in figures 1, B and 3, A, respectively, because of Q waves in Lead V4 that could be mistaken for those due to myocardial infarction.

*Exaggeration of the Normal Q Wave in Left Ventricular Leads.* Q waves are present in Lead V6 in the majority of normal subjects,9 11 and represent negative potentials transmitted from the endocardial surface of the left side of the septum and anteroseptal wall of the left ventricle through the intervening structures to the axilla during the brief period that normally elapses before the impulse reaches the subendocardial layer of the lateral wall. The normal Q wave in left ventricular leads V6, V5, and V4 is almost always less than 3 mm. in depth and invariably less than 25 per cent of the amplitude of the succeeding R wave.
Fig. 1.—Left ventricular hypertrophy with electrocardiographic features suggestive of myocardial infarction. A, Case 1; B, Case 2; C, Case 3; D, Case 4; E, Case 5; F, Case 6; G, Case 7; H, Case 8.
Q waves of similar origin are present in Leads V₅ and V₆ in uncomplicated left ventricular hypertrophy and may be exaggerated in amplitude as a result of: (1) the greater voltage developed during activation of the hypertrophied septum, and (2) the improved transmission to the axilla because of the closer approach of the enlarged left ventricle to the thoracic cage. This is exemplified by the electrocardiogram reproduced in figure 1, A from 6 to 7 mm. in depth and was thus comparable in absolute voltage to the Q wave recorded in this lead as a manifestation of anterolateral infarction. However, the QRS pattern in Lead V₆ of figure 1, A could be recognized as a manifestation of left ventricular hypertrophy, rather
than infarction, by the normal duration of the Q wave and by the exceptionally tall and abnormally prolonged ascending limb of the succeeding R wave. The upstroke ranged from 35 to 38 mm. in amplitude, thereby making a normal QR ratio of 15 to 20 per cent. The ante-mortem interpretation, based on Q duration and QR ratio in V₆, was confirmed at autopsy, which revealed rheumatic mitral and aortic insufficiency and subacute bacterial endocarditis with marked left ventricular hypertrophy, but without coronary disease or myocardial infarction.

Among a series of 1375 patients studied with multiple precordial leads and subsequently studied at autopsy, no case without pathologically demonstrable infarction has been encountered thus far in which the Q wave in left ventricular lead V₆ exceeded 0.03 second from onset to nadir, and 25 per cent of the amplitude of the subsequent R wave. On the other hand, infarction confined to the subendocardial layer of the anterolateral wall may be accompanied by Q waves that are within normal limits, both as to duration and as to QR ratio. Whenever Q waves in the customary left ventricular leads approach the border line in duration or in relative amplitude, a repetition of the tracing, including high precordial leads, is advisable to investigate the possibility of a high anterolateral infarct producing marginal zonal patterns in the customary leads.

The amplitude and duration of the normal Q wave are usually greater in Lead V₆ than in V₅ and are, in turn, greater in V₅ than in V₄. Case 2, figure 1, B is included because the Q wave measured 6.0 mm. in V₄, 4.0 mm. in V₅, and 2.5 mm. in V₆. Although such relationships raise the question of anteroseptal infarction, the absolute amplitude of the Q wave loses its significance in this case when attention is directed to the time from onset to nadir of the downstroke and to the relative heights of the R waves in the three leads. The duration of the Q wave and the QR ratio are almost identical in Leads V₄, V₅, and V₆ and are within the limits

* This statement is made subject to the condition that Lead V₆ reflects the potential variations of the left ventricle, because of the occasional finding of a QS pattern in V₄ owing to the axillary transmission of the potential variations of a dilated right ventricle in a heart rotated markedly in a clockwise direction.

![Fig. 3.—Serial electrocardiograms in Case 11 (A, above) and in Case 12 (B, facing page).](http://circ.ahajournals.org/doi/abs/10.1161/01.cir.21.2.848?journalCode=circ)
Fig. 3.—Cont'd.
of normal. The interpretation, based on these two latter criteria, was confirmed by the finding of a normal heart at autopsy. Death was due to carcinoma of the colon. The parallel decrease in amplitude of the Q and R deflections as the electrode was moved from the V4 position into the axilla was a manifestation of the normal loss of potential with increasing distance from the cardiac border.

Suggestive RS-T Patterns in Left Ventricular Leads. The typical findings in leads facing the epicardial surface of the anterolateral wall of an hypertrophied left ventricle are slight to moderate depression of the RS-T junction and inverted to diphasic T waves, as illustrated by Lead V5 of figure 1, H and Lead V6 of the tracing taken on February 2 in figure 2, A. Four of the more common variants which may be mistaken for myocardial infarction are illustrated in figures 1 and 2 and will be discussed separately.

Sharp Inversion of the T Wave, Accompanied by an Isoelectric or Even Slightly Elevated RS-T Junction: This may occur in association with left ventricular hypertrophy and may mimic the cove negative T waves associated with myocardial infarction. This is illustrated by the T waves in Leads V5 and V6 of figure 1, D and those in V5 and aVL of figure 1, C. These tracings were obtained from Patients 3 and 4, respectively, neither of whom had received digitalis.

Lead aVL of figure 1, C exhibited the combination of upward bowing of the RS-T segment and V-shaped inversion of the T wave, resulting in the cove curvature originally considered diagnostic of myocardial infarction, but now identified with other lesions of the subepicardial layer of the myocardium. The tall initial R wave in these leads excluded the possibility of a central zone of transmural infarction and a marginal zone of subendocardial infarction in the area subtended by the electrode, but was compatible with any of the following three possibilities: (a) infarction limited to the subepicardial layer of the lateral wall, (b) marginal zone of subepicardial ischemia beyond the boundary of a high lateral infarct, (c) pericarditis or subepicardial myocarditis. The latter alternative was in keeping with the clinical picture and was subsequently confirmed at autopsy. The patient’s death was due to uremia secondary to malignant hypertension and the heart showed left ventricular hypertrophy complicated by subepicardial lesions typical of uremic myocardiopathy. The coronary arteries were patent and no gross or histologic evidence of infarction was found.

The contour of the T wave in Leads V5 and V6 of figure 1, D was less typical, though compatible with a specific lesion of the subepicardial layer, but was more in keeping with uncomplicated left ventricular hypertrophy. In view of the abnormally small R waves in V3 and V4, the possibility that the prominent initial R and inverted T waves of Leads V5, and V4 reflected the potential variations of the marginal zone beyond the boundary of the high anterolateral infarct should have been investigated by high precordial leads; however, this possibility was excluded by postmortem examination. The patient died of cerebral hemorrhage six months after the electrocardiogram was made and autopsy revealed uncomplicated left ventricular hypertrophy of hypertensive origin.

Marked RS-T Depression in Left Ventricular Leads: Marked RS-T depression in left ventricular leads that might be mistakenly interpreted as due to subendocardial ischemia or infarction2-7 may occur in uncomplicated left ventricular hypertrophy as a result of: (a) digitalis effect, (b) coexistence of high P waves and tachycardia.

The changes produced by digitalis in left ventricular leads are well known and consist in depression of the RS-T junction, straight downward sloping of the RS-T segment, steep ascent of the terminal limb of the T wave, and shortening of the Q-T interval. The extreme degree of RS-T depression that may occur in left ventricular leads due to the combination of left ventricular hypertrophy and full digitalization is illustrated in figure 1, E (Case 5). The RS-T junction was depressed 6 mm. below the isoelectric line in Lead V5 and 3.5 mm. in V4 and V6. Even without benefit of history, the RS-T depression can be attributed to digitalis, rather than to subendocardial ischemia or infarction, because of the characteristic shape of the T wave and the distinct shortening of the QT interval. The patient was admitted to the hos-
pital with congestive failure and was digitalized before the electrocardiogram was made, but died on the following day. Autopsy revealed a huge heart, weighing 1,000 grams, the enlargement being due principally to left ventricular hypertrophy of hypertensive origin. No evidence of infarction, coronary occlusion, or narrowing was found.

When marked RS-T depression occurs in the absence of certain of the classical manifestations of digitalis, confusion may result, as illustrated by figure 1, F (Case 6). The patient had been admitted to the hospital with marked congestive failure, due to rheumatic aortic stenosis and insufficiency, and had received digitalis before the electrocardiogram was made. The 3-mm. depression of the RS-T junction and the straightening of the RS-T segment in Lead V₆ could be explained by digitalis action in the presence of left ventricular hypertrophy, but the contour of the T wave in both V₆ and left ventricular lead aV₆, and the Q-T interval near the upper limits of normal¹² were atypical of digitalis effect. Postmortem examination revealed a 787-gram heart with a markedly hypertrophied left ventricle and a very distended, but thin-walled, right ventricle. Advanced aortic stenosis was present, but the coronary tree was widely dilated and there was no evidence of infarction. An acute epicarditis and subepicardial myocarditis was demonstrated microscopically and accounted for the modifications in RS-T pattern.

Coexistence of a deep auricular T wave and tachycardia may lead to marked pseudodepression of the RS-T segment,⁸ that could be mistaken as evidence of subendocardial ischemia or infarction. This is exemplified by Leads V₄ through V₆ of the tracing of February 19 in figure 2, A and will be discussed below.

Deeper Inversion of the T Wave in Lead V₄ Than in V₅ and V₆: This finding, as in figure 1, G and H, may arouse the suspicion of anteroseptal infarction. These tracings were obtained from Patients 7 and 8, respectively, neither of whom had received digitalis previously. The possibility of a transmural anterior infarct was excluded in both cases by the insignificant Q wave and tall R wave in left ventricular leads. Acute infarction limited to the subendocardial layer was ruled out by QRS pattern and the lack of sufficient RS-T depression. On the other hand, the possibility of infarction or inflammation limited to the subepicardial layer was excluded by the fact that the RS-T junctions were depressed rather than elevated or isoelectric. The RS-T patterns in Leads V₄ through V₆ of both patients were therefore ascribed to left ventricular hypertrophy. The progressive diminution in the depth of the inverted T wave as the electrode was moved from the C₄ to the C₆ position was attributed to loss of potential with increasing distance from the heart. This explanation was strongly supported in Case 7 (fig. 1, G) by the parallel diminution in the amplitude of the R wave. A concurrent reduction in amplitudes of the R and T waves was also noted in Case 8 when the electrode was moved from precordium to midaxilla, but not when it was shifted from the C₄ to the C₆ position. The T wave in V₄ was slightly deeper than that in V₅, but the R wave in V₄ was not quite as tall as that in V₅.

Patient 7 had no symptoms referable to the heart and died of carcinoma of the colon. Patient 8 was admitted to the hospital with congestive failure and expired suddenly of pulmonary embolism. Autopsy revealed left ventricular hypertrophy of hypertensive origin in both patients, without infarction or coronary narrowing. Thus deeply inverted T waves that progressively diminish as the electrode is moved from the C₄ to the C₆ position may occur as a manifestation of uncomplicated left ventricular hypertrophy and are usually, though not necessarily, accompanied by parallel decrease in the amplitude of the R waves.

Rapid Changes in RS-T Segment and T Waves of Left Ventricular Leads in Serial Tracings: These often arouse the suspicion of myocardial infarction, but may occur in association with left ventricular dilatation and hypertrophy without demonstrable myocardial lesion, particularly when the heart is subjected to some extrinsic stress. This is exemplified by figure 2, which reproduces the serial electrocardiograms of two patients with thyrotoxicosis and associated left ventricular dilatation and hypertrophy.

Patient 9 (fig. 2, A) was a 34 year old woman
with severe Graves' disease. The tracing of January 27, 1943, obtained when the basal metabolism rate was +80, showed very tall R waves in Leads V1 and V6, which were of normal duration and thus not diagnostic of left ventricular hypertrophy. In Leads V6 and I, the RS-T segment was slightly depressed and the T wave, inverted, whereas in Lead V1, the T wave was coarsely notched. Subsequent electrocardiograms showed significant changes in the RS-T segments and T waves, but not in the QRS complexes. The tracing of February 2, obtained when the basal metabolism rate had fallen to +51 under iodides, showed a diphasic wave in Lead V6, a doubled T wave in V1, and flattened T wave in Lead I. Although the basal metabolism rate did not decrease further, the record of February 11 showed normal upright, pointed T waves in all leads. Cardiac glycosides could be positively excluded as a cause of the RS-T changes, since none was given to this patient at any time. Because of the absence of significant changes in the QRS complexes, the RS-T evolution in the tracings made between January 27 and February 11 was much more likely due to subsidence of external stress upon the left ventricle than to healing of a small myocardial infarct.

Thyroidectomy was performed on February 17 and was complicated by a postoperative thyroid crisis. Electrocardiogram on February 19, obtained during the crisis, showed a sinus tachycardia of 154 and striking changes in the RS-T segment of Leads III, V4, and V6, without significant alterations in QRS. Although the QRS-T pattern in Lead III was strongly suggestive of recent posterior infarction, the diagnosis is not justified unless parallel changes can be demonstrated in Lead II and particularly in Lead aVr.\(^5\) Lead aVr was not obtained on this patient, but the findings in Lead II failed to confirm the presence of posterior infarction. The apparent RS-T depression in Leads V4 and V6 might have represented a reciprocal effect of a recent posterior infarction or a direct effect of acute ischemia or infarction limited to the subendocardial layer of the anterolateral wall; however, the contour of the RS-T segment and T wave was not typical of either of these alternatives. More careful study showed that the depression was apparent, rather than real, and was due to two factors: (1) the presence of a tachycardia sufficient to cause superimposition of the P wave on the antecedent T and thereby prevent the usual diastolic return of the string to the isoelectric line, (2) a prominent auricular T wave following an exceptionally tall P wave.\(^9\)

The patient died on February 23 and autopsy disclosed a 420-gram heart, showing left ventricular hypertrophy, ascribed to thyrotoxicosis, after failure to demonstrate evidence of hypertension or other recognized causes. The coronary vessels were normal and no evidence of infarction was found in either the posterior or the anterolateral walls. The postmortem findings were thus in keeping with the electrocardiographic interpretation. The QRS-T pattern in Lead III was probably derived principally from its left arm component. AlthoughLead aV L was not obtained, the findings in Lead I suggested that the potential variations of the left arm paralleled those of the V4 and V6 positions. Thus the Q wave, elevated RS-T junction, and inverted T wave of Lead III represented, respectively, the reciprocals of the prominent R wave, depressed RS-T junction, and upright T wave, which should have been recorded in Lead aV L.

The patient whose electrocardiograms are reproduced in figure 2, B (Case 10) was admitted to the hospital with thyrotoxicosis complicated by auricular fibrillation, congestive failure, and right lower lobar pneumonia and received 1.6 Gm. Cedilanid prior to the electrocardiogram of September 11, the second hospital day. The QR pattern in Leads V1 and V6 was indicative of left ventricular hypertrophy. The inverted T waves of Leads V6 and I and the diphasic T wave of V1 were probably not due exclusively to digitalis, because of the subsequent appearance of upright T waves in these leads despite continuation of digitalis in doses sufficient to maintain the apical rate between 50 and 60. The improvement in the T waves was in keeping with decrease in load upon the left ventricle, consequent upon recovery from the pneumonia, and decrease in the thyrotoxicosis.

Thiourca was employed and the course was complicated by a toxic psychosis, which led to
death on October 14. The heart weighed 478 grams and showed left ventricular hypertrophy, believed to have been due to hyperthyroidism because of the failure to find evidence of hypertension or other known causes. The coronary vessels were normal and the myocardium showed no evidence of infarction. Thus, the serial changes in the T waves were not due to coronary disease.

QS Deflections in Leads V1 and V2 (Fig. 1, C, D, E, F). Such deflections may raise the question of infarction of the interventricular septum, but are not diagnostic of a septal lesion because of their occasional presence in normal subjects, and in persons with left ventricular hypertrophy. The registration of a QS complex in Leads V1 and V2 as a normal variant is favored by a cardiac rotation that brings the right atrium beneath the sternum, carries the left apex backward, and tilts the mitral orifice to the right and forward, thereby facilitating transmission of left ventricular cavity potentials through the interatrial septum to the precordium. Conditions suitable for the registration of a QS deflection as a normal variant were present in Patients 4 and 6 (fig. 1, D, F), as shown by (1) distinct auricular intrinsicsoid deflections in Leads V1 and V2, indicating proximity of the electrode to the right atrium; (2) prominent late R waves in Lead aVR, indicating backward displacement of the left apex. While the apparent relation of the electrode to the heart was strongly in favor of interpreting the QS complex of Patients 4 and 6 as a normal variant, it did not positively exclude the possibility of septal infarction. On the other hand, the absence of demonstrable backward rotation in Lead aVR of Patient 3 did not rule out positional factors as the cause of the QS in Leads V1 and V2 of figure 1, C.

When there is uncertainty as to the significance of QS complexes in V1 and V2 the following additional steps are advisable: (1) search for evidence of infarction in precordial leads to the left and right, (2) repetition of the tracing in a different posture to determine the stability of the QS pattern in Leads V1 and V2. The findings in leads to the left of V2 were not diagnostic of infarction in any of the three cases, but the abnormal elevation of the RS-T junction in Leads V3 and V4 of Patient 6 and the abnormally small initial R wave in the same leads of Patient 4 warranted further investigation. The abnormal RS-T displacement in Leads V3 and V4 of Patient 6, figure 1, F, was referable to an epicarditis, as discussed above. Lead V3R was obtained from Patient 4 and revealed a QS complex similar to that in Lead V1, figure 1, D. This represented the expected finding if the QS pattern in V1 and V2 were a normal variant. The tracings reproduced in figure 1, D, F were obtained with the patients in the sitting position. Reposition with the patients in the recumbent position revealed a distinct initial R wave in the first two precordial leads in Case 6, a small initial R wave in V1 and V2 in Case 4, and a somewhat larger initial R wave in Leads V2 and V1. This confirmed the interpretation of the QS complexes in V1 and V2 of Cases 4 and 6 as a normal variant referable to cardiac position. On autopsy, no evidence of infarction of the septum was found in these two cases or in Case 3.

Elevation of the RS-T Segment in Right Precordial Leads. This is an expected finding in left ventricular hypertrophy and should not be misinterpreted as evidence of myocardial infarction when the segment maintains its normal upward concavity and the T wave is upright and normal in shape, as in figure 1, C, D, G, H. Excessive elevation of the RS-T segment in right precordial leads sufficient to arouse the suspicion of infarction may occur in association with left ventricular hypertrophy as a result of (1) digitalization, (2) superimposed pericarditis.

The combined effects of digitalis and left ventricular hypertrophy on the RS-T segment and T wave in right precordial leads may mimic those associated with acute anteroseptal infarction, as exemplified by figure 1, E (Case 5). Inspection of the first three precordial leads revealed the following changes suggestive of acute anteroseptal infarction: (a) marked RS-T elevation, amounting to 4 mm. in V1, 5 mm. in V2, and 8 mm. in V3; (b) straightening of the RS-T segment; (c) precipitous descent of the T wave. However, the possibility of anteroseptal infarction was rendered very unlikely by the prominent initial R waves, measuring 4 mm.
in V1, 10 mm. in V2, and 13 mm. in V3, and was excluded as a result of other evidence indicating that the RS-T patterns in V1 through V3 were referable to digitalis action, namely: (a) the shortening of the Q-T interval, (b) the fact that the RS-T complex in V1 through V3 was reciprocal to the digitalis RS-T pattern in V4 and V. As indicated above, autopsy revealed left ventricular hypertrophy without infarction and thus confirmed this interpretation.

Although Patient 6 had left ventricular hypertrophy and had received digitalis prior to the electrocardiographic recording (fig. 1, F), the findings in the first four precordial leads could not be satisfactorily explained by this combination because the extreme upward RS-T displacement in right ventricular leads V2 and V4 was disproportionate to the slight elevation in right atrial lead V1 and to the depression in V6. It was therefore necessary to consider the possibility that an acute anteroseptal infarct may have caused the RS-T elevation in V2 and V4 and may have produced reciprocal depression in V6. High precordial leads were taken, but revealed a small R, deep S, and elevated RS-T segment comparable to that in Leads V3 and V4. Anteroseptal infarction could not be ruled out by the absence of a diagnostic QRS pattern in a single tracing, but its possibility was excluded in this case by a study of previous electrocardiograms. The first tracing, made eight days prior to that in figure 1, F, showed RS-T depression in V6, typical of digitalis action, but no abnormal elevation in right ventricular leads. The latter was first noted two days later and steadily increased in two intervening records, to reach a maximum in the tracing reproduced in figure 1, F. The serial changes in the RS-T segment in V3 and V4, together with the presence of a constant QRS pattern, were indicative of pericarditis. The pathologic findings, as pointed out above, consisted of marked left ventricular hypertrophy, right ventricular dilatation and acute epicarditis, and subepicardial myocarditis. There was no evidence of infarction of the septum or anterior wall.

Bizarre QRS Patterns in Leads From the Transitional Zone. In taking the six precordial leads, the electrode is customarily moved from points over the right ventricle to points over the left ventricle. If the electrode happens to cross the septum when shifted from one chest position to the next, an abrupt change from an rS pattern, representing the potential variations of the right ventricle (Lead V4 of figure 1, A), to an Rs deflection, representing the potential variations of the left ventricle (Lead V6 of fig. 1, A), is recorded.* On the other hand, if the electrode happens to straddle the anterior terminus of the septum in one or more leads, transitional complexes, representing varying mixtures of right and left ventricular effects, are recorded. The usual finding at the transitional zone is a QRS complex of relatively low voltage, consisting of R and S deflections that are intermediate in amplitude between those in adjacent leads to the right and left, as exemplified by V3 of figure 1, B. These intermediate complexes often exhibit coarse slurring or notching, as in Lead V4 of figure 1, C and V5 of figure 1, H. Through determination of temporal relationships of such notches with the peaks of the R waves in leads to the right and left, these notches may be recognized as manifestations of the transitional zone and not an indication of an interventricular conduction defect. For example, the notch at the nadir of the S wave in Lead V3 of figure 1, H is synchronous with the peak of the R wave in Lead V4 and thus marks the arrival of the impulse of the epicardial surface of the anterior apical wall of the left ventricle. In a few cases of left ventricular hypertrophy, bizarre QRS patterns, characterized either by a multiphasic complex or by an initial downstroke, may be found at the transitional zone and may be mistaken for patterns due to myocardial infarction unless analyzed in reference to the findings in adjacent leads.

Multiphasic QRS Complexes at the Transitional Zone: From a hasty glance at figure 1, F, one might suspect a localized conduction defect in the anterolateral wall of the left ventricle from the quadrifasic rsIt's complex in V6, as compared with the smooth, unnotched Rs and Rs deflections in V4 and V6, respectively.

* The lower case letter is used to indicate a relatively small deflection, the upper case letter to indicate a relatively large deflection.
The fact that the QRS interval, as measured in V5, was similar to that in V4 is against such an interpretation. Furthermore, the peak of the initial R wave in V5 was synchronous with the R wave of right ventricular origin in Lead V1, whereas the peak of the R' deflection in V6 was synchronous with the R wave of left ventricular origin in V6. Hence, Lead V5 was a transitional lead registering separately the intrinscoid deflections from the anterior walls of both ventricles. Multiphasic QRS complexes of low voltage in the transitional zone are more difficult to analyze and may require additional leads.

Prominent Q Wave at the Transitional Zone: Q waves in the precordial leads that measure 0.03 second or more from onset to nadir and exceed 25 per cent of the amplitude of the subsequent R wave are ordinarily the result of myocardial infarction, but occasionally may occur in leads at the transitional zone in the absence of infarction. The diagnostic difficulties under these circumstances are exemplified by Cases 11 and 12.

Patient 11, a man 60 years of age, came to the Outpatient Department in June, 1947, with symptoms referable to carcinoma of the stomach. The first electrocardiogram, made on June 27 and reproduced in figure 3, A, showed sinus rhythm interrupted by frequent premature auricular beats. The QRS-T pattern in the limb leads and in V5 and V6 was considered normal. In Leads V1 and V2, a minute initial R wave could be made out, followed by a deep S wave. This small initial R wave disappeared from V1 and V4, and a QS deflection was recorded in the former, a triphasic QRS complex in the latter. The triphasic QRS deflection in Lead V4 began with a downstroke, which ranged from 2 mm. to 5 mm. in depth and from 33 per cent to 200 per cent of the succeeding R wave. The change from an rS deflection in V2 to a QS in V3 and the abnormal QR ratio in every cycle of V4 pointed strongly towards anteroseptal infarction, whereas the contour of the RS-T complexes indicated that infarction, if present, was old and healed. However, an unequivocal diagnosis of anteroseptal infarction was not justifiable from the precordial electrocardiogram for the following reasons: (1) the time from onset to nadir of the Q wave in V4 was only 0.02 second; (2) Q waves were not detected in V5 and V6 as would have been expected if an infarct had been responsible for the relatively deep Q waves in V3 and V4; (3) the Q waves were confined to leads that were transitional in type, as indicated by: (a) the low voltage of the QRS in V5 and V4, as compared to V2 and V5; (b) marked respiratory fluctuations in the depth of the Q wave and the QR ratio in Lead V4.

During the course of preparation for operation, the tracing was repeated to further investigate the significance of the findings in V5 and V4. No essential change was found in the QRS-T pattern of Leads V1, V2, V3, V5, and V6. In Lead V3 there was a much larger S wave, comparable in voltage to the S wave of V1 and V2. A small initial R was consistently present in Lead V5, but showed respiratory fluctuations in amplitude. The findings in Lead V5 on July 22 indicated that this lead reflected chiefly the potential variations of the right ventricle, rather than the transitional zone. Lead V5 displayed a prominent initial R wave and a tall upright T wave, similar to those in V3 and V6, indicating that the electrode had crossed the septum and reflected the potential variations of the anteropapical wall of the left ventricle. The negative findings in leads immediately to the right and left of the septum in this tracing constituted further evidence signifying that the pattern in V3 and V4 of the first tracing was representative of transitional zonal effects, rather than anteroseptal infarction. The patient died postoperatively and autopsy revealed a heart of normal weight with a widely dilated coronary tree and no evidence of myocardial infarction. There was moderate right ventricular dilatation, which may have represented a postoperative complication.

Restudy, after the autopsy, of the tracing of June 27 uncovered the following additional data that might account for the unusual normal variations found in Leads V3 and V4. The QRS interval measured 0.08 second in V1 and V2, 0.07 second in V3 and V4 and 0.06 second in V5 and V6. This difference in time interval indicated that the forces responsible for the initial R in V1 and V2 were not represented in the
remaining leads. The R wave in leads facing the right ventricle and atrium is generally derived in part from the septum and in part from the free wall of the right ventricle. Activation of the septum usually results in transmission of positive potentials to the right precordium and negative potentials to the left precordium, either because of earlier arrival of the impulse in the left side of the septum or greater magnitude of forces developed in the left than in the right half of the septum. A comparison of the initial R waves in right precordial leads showed that it was largest in V1, the lead furthest removed from the septum, and decreased as the electrode approached the septum. This relationship, together with the absence of a Q counterpart in Leads V4 and V6, was strongly against a septal origin for the R wave in V1 and V2. On the other hand, the diminishing R wave in the first three leads was compatible with a right ventricular origin, since activation of the relatively thick base of the right ventricle near the tricuspid orifice should produce greater electromotive force than activation of the relatively thin apex of the right ventricle. If depolarization of the two sides of the septum began simultaneously and produced forces of approximately equal magnitude, one might expect precordial transmission of negative potentials from the endocardial surface as a consequence of extinction of positive potentials in the center of the septum. The findings in Leads V3 and V4 can be accounted for by this hypothesis, assuming that the electrode was situated just to the right of the septum at V2 and straddled the septum at V4. The downstroke in Lead V3 was probably initiated by negative potentials reaching the right ventricular cavity from activation of the right side of the septum and continued by left ventricular cavity potentials transmitted to the right after depolarization of the septum. The initial downstroke in Lead V4 was probably derived from the right side of the septum in the same manner as the first part of the QS in V5, whereas the succeeding R wave was undoubtedly due to activation of the anteroseptal wall of the left ventricle, as shown by the synchrony of the intrinsicoid deflection with that in V5. The origin of the triphasic QRS complex in Lead V4 of the tracing of June 27 was thus analogous to the origin of the quadriphasic QRS in Lead V4 in Case 6, figure 1, F.

The tracings reproduced in figure 3, B were obtained from Patient 12, a 46 year old man, who gave a history of shortness of breath on exertion since April, 1944, and sudden paroxysmal nocturnal dyspnea on July 15, followed by progressive dependent edema. He denied the presence of chest pain. Physical examination revealed signs of rheumatic aortic stenosis and insufficiency complicated by marked congestive failure. The patient was partially digitalized during the last three days in July and received an additional 4½ grains during the three-day interim between the two tracings.

Sinus rhythm was present on August 1, except for two late ventricular premature beats of the fusion type in Lead III. Leads V4, V6, and I showed a minute Q wave, tall slurred upstroke, slightly delayed intrinsicoid deflection, slightly depressed RS-T junction, and diphasic T wave, typical of left ventricular hypertrophy. The deep S wave in V1, which extended below the lower edge of the record, was consistent with left ventricular hypertrophy, but the initial R was unusually large, measuring 0.8 to 1.0 millivolts, and raised the question of coexistent right ventricular hypertrophy. Some cycles of Lead V2 were similar to those of V1; others displayed a minute Q, followed by a slightly smaller R, but a comparable S wave. A minute Q wave was consistently present in Lead V3 and was followed by a much smaller upstroke, ranging from 1 to 3 mm. in amplitude. Lead V4 showed marked fluctuations in the QRS contour, despite a uniform P-R interval and a regular ventricular rhythm. These fluctuations were present during quiet breathing, but were markedly accentuated by deep breathing, which was in progress while the strip dated August 1 was made. The variability of the QRS pattern in V4 indicated that the electrode was at the transitional zone and was attributed to respiratory shifting in cardiac position. Nevertheless, the prominent notched Q wave present in every cycle of V4 was interpreted as evidence of anteroseptal infarction, because of consistent prolongation in time interval from its onset to nadir and because of an habitually abnormal
Q-R ratio. The diagnosis of anteroseptal infarction was strongly supported by elevation of the RS-T junction in V₃ and V₄. The RS-T displacement in these leads could not have been a transitional phenomenon, since the RS-T junction was depressed in leads further to the right and left, whereas the T wave, which was upright and doubled in V₃ and V₄, was inverted to diphasic both in V₁ and in V₅ and V₆. A diagnosis of recent anteroseptal infarction was confirmed by the cove inversion of the T waves in V₃ and V₄, which took place during the next three days. These RS-T changes were believed independent of digitalis, not only because of the small dose administered during interim, but also because of the lack of Q-T shortening. The QRS pattern was essentially the same as in the previous tracing, but the respiratory fluctuations were not as marked in V₄ because of the fact that this tracing was made during quiet breathing.

The patient returned to the hospital on September 17, 1944, with congestive failure. An electrocardiogram made the day following admission showed no significant change in QRS pattern, except a T-wave evolution consistent with the healing of an infarction. Death occurred on September 20, 1944, and autopsy revealed a 637-gm heart, showing marked left and moderate right ventricular hypertrophy associated with rheumatic aortic stenosis. The coronary tree injected well and showed no narrowing or occlusion. The heart was opened by the Schlesinger technique and no evidence of infarction was found on gross inspection. Seven microscopic blocks were taken from the anteroseptal wall of the left ventricle, extending from apex to base, and showed no myocardial lesion apart from hypertrophy and slight perivascular fibrosis consistent with his old rheumatic infection. Similar negative findings were observed in the posterior wall, but unfortunately no blocks were taken from the lateral wall of the left ventricle. Discard of the gross specimen has made further pathologic study impossible.

Proceeding upon the assumption that an infarct had not been overlooked pathologically, one might be tempted to explain the QRS pattern in V₃ and V₄ as a transitional zonal phenomenon analogous to that already discussed in connection with Case 11. However, the serial changes in RS-T complex could not be accounted for in this manner. Although the RS-T evolution was compatible with a localized area of acute pericarditis or subepicardial myocarditis, no traces were found at autopsy. Thus, we were dissatisfied with the hypothesis that the abnormal Q waves in V₃ and V₄, fulfilling the criteria for infarction, represented a rare variant of the transitional zone, whereas the RS-T evolution in the same leads was due to a localized, pathologically undetectable pericarditis. This made it necessary to reconsider the alternative possibility that an infarct was present in August, 1944, but was missed at autopsy seven weeks later. Complete healing during the interim cannot account for the discrepancy between the electrocardiographic and pathologic findings because the Q-wave abnormalities were still present in a tracing obtained two days before death. Experience during the past four years with other cases, particularly Case 144 of a previous report,⁷ has suggested a better explanation. The electrocardiogram in Case 144 revealed abnormal Q patterns in V₁ and V₄, but not in V₅ and V₆, whereas autopsy disclosed an infarct localized to the lateral wall of the left ventricle. The potential variations of the epicardial surface of this lateral infarct were transmitted to the precordium, rather than to the axilla, because of marked counterclockwise rotation of the heart. Since a comparable degree of counterclockwise rotation was present in Patient 12 of this series, it seems most likely that a lateral infarct was responsible for the RS-T abnormalities in V₃ and V₄, but was missed at autopsy because of failure to take sections from the lateral wall.

Midprecordial Leads with Bizarre RS-T Patterns Suggestive of Infarction. These may be encountered as a result of: (1) displacement of the transitional zone for the T wave to the right of that for the QRS; (2) registration at the transitional zone of an intermediate RS-T complex, consisting of an RS-T segment resembling that in leads to the right and a T wave like that in leads to the left.

The deeply inverted T wave in Lead V₃ of Patient 7, figure 1, G, in association with an
almost equiphase RS deflection of transitional type, might raise the question of a marginal zone of subepicardial infarction. Its close resemblance to the T wave in left ventricular lead V₄ suggested that the T wave in V₃ was chiefly left ventricular in origin, and like that in V₄, could have been due to uncomplicated left ventricular hypertrophy. Autopsy confirmed the latter and excluded infarction. The fact that the transitional zone for the T wave was located to the right of that for the QRS may have been due to counterclockwise rotation of the heart during mechanical systole, which begins during the registration of the intrinsicoid deflection.

The inscription of an elevated RS-T segment like that in leads further to the right, followed by sharp inversion of the T wave like that in leads to the left, may produce an effect simulating the cove negative T wave of recent myocardial infarction. This was observed in Patient 47, as a manifestation of the transitional zone in uncomplicated left ventricular hypertrophy and in Patient 31 with uncomplicated left bundle branch block, and will be reported in future communications.

**Summary**

QRS-T patterns in Wilson precordial leads that may be mistaken for patterns due to myocardial infarction include those of left ventricular hypertrophy and dilatation; right ventricular hypertrophy and dilatation; left bundle branch block; right bundle branch block; alterations in blood potassium; myocardial ischemia; pericarditis and subepicardial myocarditis; and certain arrhythmias. To bring out the differential diagnosis, cases have been selected for presentation in which myocardial infarction was diagnosed, or at least considered in the electrocardiographic interpretation during life, but was subsequently excluded by meticulous postmortem examination. The electrocardiographic differentiation of myocardial infarction from left ventricular hypertrophy is considered in this article and the differentiation from the remaining lesions will form the subject of future reports.

The electrocardiograms of 12 patients, in whom autopsy revealed either left ventricular hypertrophy or a normal myocardium and excluded the possibility of myocardial infarction, have been presented because of one or more of the following signs suggestive of myocardial infarction:

A. Unusual depth of the Q waves customarily recorded in leads from the left axilla in the presence of left ventricular hypertrophy or greater voltage of the Q wave in Lead V₄ than in V₂ or V₆.

B. RS-T patterns in leads from the left precardium or axilla, characterized by either: (1) sharp inversion of the T wave, accompanied by a slightly elevated or isoelectric RS-T junction, instead of the customary slight depression; (2) marked RS-T depression; (3) deeper inversion of the T wave in Lead V₁ than in V₂ or V₆; (4) rapid changes in serial tracings.

C. QS deflections in Leads V₁ and V₆.

D. Abnormal elevation of the RS-T junction in leads from the right side of the precordium.

E. Bizarre QRS patterns in leads from the transitional zone, characterized by either: (1) a multiphasic complex, (2) an abnormal initial downstroke.

F. Abnormal displacement of the RS-T segment and/or inversion of the T wave in leads from the transitional zone.

The correlation of electrocardiographic and pathologic findings and the differentiation from the pattern of myocardial infarction are brought out through a detailed analysis of each electrocardiogram.

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QRS-T Patterns in Multiple Precordial Leads that May Be Mistaken for Myocardial Infarction: I. Left Ventricular Hypertrophy and Dilatation
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