Certain Clinical States and Pathologic Changes Associated with Deeply Inverted T Waves in the Precordial Electrocardiogram

By Raymond D. Pruitt, M.D., Clayton H. Klakeg, M.D. and Lemuel E. Chapin, M.D.

A correlation of the clinical and electrocardiographic findings was undertaken in 110 cases which had in common the presence of deeply inverted T waves in central terminal leads centered about position 3 on the precordium. The results of this study are reviewed. A summary is presented of changes encountered at the time of necropsy in nine cases in which electrocardiographic changes of similar type had been recorded. These observations are integrated with concepts derived from the dipole theory. The total evidence is viewed in relation to observations reported by other investigators.

The T wave of the electrocardiogram always has been a component difficult to deal with. Changes in its configuration occur at times with scant provocation, and even at its normal best, it is commonly upright when, by more simply designed relationships, it should be inverted. In this enlightened period when nearly all things basic in electrocardiography can be crammed into a tight little nutshell of theory, the conduct of the T wave provides a disturbing but intriguing expression of refusal to conform. This stubborn attachment to an individualized, and in some measure unpredictable, performance may justify application of a method which otherwise would be held archaic to a study of one of the more remarkable aberrations of this wave. The proposition basic to the study hereafter reported is that something may be learned about the origins of an electrocardiographic phenomenon by correlating it with the clinical, and in a few instances the pathologic, states in which it appears. Perhaps the argument would bear more weight if certain experiences that led to its projection were recalled.

Over a period of several years, an occasional patient has been encountered who presented few if any symptoms of cardiac disease and yet whose electrocardiogram was so manifestly abnormal that even the undiscerning in matters electrocardiographic would have been alarmed by the changes encountered. The peculiar feature of the electrocardiogram was the presence of deeply inverted T waves in records from precordial leads from all or part of the points between positions 1 and 6.

A 52 year old man came to the Mayo Clinic because of symptoms and findings indicative of polyneuritis. Careful study failed to reveal the cause of his neurologic disease but did disclose an abnormal electrocardiogram characterized by deeply inverted T waves in precordial leads V2 to V6 (fig. 1). He presented no symptoms suggestive of angina pectoris or impaired myocardial reserve. Such unusual pathologic processes as periarteritis nodosa and hemochromatosis were considered but discarded as unlikely diagnostic possibilities. Coronary sclerosis with myocardial infarction was regarded as the most likely basis for the abnormalities in the electrocardiogram, but in the absence of supporting evidence in the patient's clinical state, no definite

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INVERTED T WAVES IN THE ELECTROCARDIOGRAM

ANALYSIS OF DATA

The material to be analyzed in this study necessarily must be separated as sharply as possible into two types, namely electrocardiographic evidence and clinical data apart from the electrocardiogram.

Electrocardiographic Data. Approximately half of the electrocardiograms that had been selected during several years were found to have the essential features of the record reproduced in figure 2. The remainder had certain features of these "typical" tracings but failed in one or more ways to satisfy the

Such experiences afforded reason for collecting a group of tracings of this type and establishing a correlation between the electrocardiogram and the pertinent clinical and pathologic data. Records were considered acceptable if the T waves were inverted deeply in precordial lead V₃ and perhaps in leads from positions to the right and left of that point and if no obvious changes in the QRS complexes were present. At the outset, no limits were set for the exact degree of inversion required for inclusion in the series; when the data were analyzed, it was decided to retain all cases, arranging them in the categories to be described.

Fig. 1. A 52 year old man presented neither symptoms nor signs of cardiac disease. Note the deeply inverted T waves, particularly in precordial leads V₁ and V₄ and the absence of significant abnormalities in the QRS complexes. This is the kind of electrocardiogram classed as "typical" in this study.

Fig. 2. A 51 year old man had experienced three episodes of severe thoracic pain. The first attack, on November 10, 1952, lasted one hour; the second, on November 24, lasted 20 minutes, and the third and last, on December 4, lasted 15 minutes. The deeply inverted T waves in the tracing of 12-5-52 are unattended by changes in the QRS complexes. In the record of 6-3-53, the deflections in the T waves have reverted to an upright configuration.
FIG. 3. A 62 year old man had noted episodes of severe thoracic pain two or three times daily for a week prior to the first tracing. These attacks occurred without relation to exertion or emotional disturbances and lasted about 20 minutes. The episodes ceased after the first electrocardiogram. The record of 3-30-53 reveals deeply inverted T waves in precordial leads V₂, V₃, and V₄. A Q wave 1 mm. in depth is present in all precordial leads except V₁. The tracing of 4-6-53 is essentially unchanged; note the notched character of the R deflection in V₁ and the tiny Q waves in leads V₃ through V₆. Both T-wave and QRS changes are gone in the tracing of 1-2-54.

1. The first group included the typical cases. Of a total of 110 cases in the entire series, 62 were placed in the category of which the record in figure 2 is representative and which might be regarded as the prototype of tracings on which interest had been focused originally. This group of electrocardiograms was characterized by the presence in lead V₃ of a T deflection inverted to a depth of at least 5 mm. and as deeply inverted as the T wave in lead V₄ or more so.

2. In the second category, typical changes in the T wave were associated with some relatively minor change in the QRS complexes (fig. 3). Of the 110 cases, 20 were so classified. In 14 of these cases, this change consisted of a Q wave from 0.5 mm. to 3 mm. in depth in one, two or all of leads V₅, V₆, and V₄. In a single instance, an R wave was present in leads V₁ and V₂ but not in V₃. In another, the
4. In the fourth category, which comprised six cases, evidence of delay in the arrival of excitation at the right ventricular surface was associated with changes of a “typical” nature in the T waves (fig. 5). The characteristics of the QRS complexes in this group suggested the presence of right ventricular hypertrophy.
or of partial or complete right bundle-branch block.

5. In the final group, the degree of inversion of the T wave in lead V₃ was less than 5 mm. (fig. 6). These nine cases included five that would have been in the "typical" series (group 1) and three that would have been classed as "left-strain variants" (group 3) had the degree of inversion of the T waves been 5 mm. or more.

Clinical Data. Establishment of categories for classification of clinical diagnoses proved a problem as difficult as had that of arranging the electrocardiograms into reasonably homogeneous groups. Because certain clinical diagnoses are of established character and may be used without definition, it was deemed feasible and appropriate to present in tabular form a summary of diagnoses derived from appraisal of the clinical records (see table 1). Of the diagnostic terms used in this table, two deserve clarification with respect to their relationship to each other.

The first is "myocardial infarction," which was applied to the condition of patients who experienced an episode of pain that was severe, lasted an hour or more, and was evidently the occasion, as contrasted with any other in the patient's account, when myocardial injury occurred. The majority of these patients entered the hospital at the acute phase of their illness, and during the first few days they were found to have fever in excess of 100 F. Signs of shock were uncommon even in this group.

The second term is "severe coronary insufficiency," which included the condition of those patients whose episodes of pain varied from mild to severe, commonly lasted less than an hour, and almost always recurred several to many times over periods ranging from a few days to several months. Many of these patients never entered the hospital for treatment, and those who did come in during the acute phase of their illness never exhibited signs of shock nor displayed a significant degree of fever. Some increase in erythrocytic sedimentation rates was commonly present in patients of this

Fig. 6. A 38 year old woman had polycystic disease and moderately severe hypertension. On several occasions between August and October, 1946, she noted anginal distress while walking up a hill. The tracings of 12-30-46 reveal sharp terminal inversion of T deflections in all precordial leads. By 8-16-49 these deflections had reverted to an upright position. These tracings are of that group designated "shallow T-wave inversion" in this study.
### Table 1.—Clinical Correlation With T-wave Changes in Precordial Electrocardiograms

<table>
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<tr>
<th>Clinical Diagnosis</th>
<th>Group (Type of Electrocardiogram)</th>
<th>1 (“Typical”)</th>
<th>2 (QRS changes)</th>
<th>3 (Left-strain variant)</th>
<th>4 (Right ventricular hypertrophy or right bundle-branch block)</th>
<th>5 (Shallow T-wave inversion)</th>
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* One patient had findings of calcific aortic valvular disease. Another had noted symptoms of severe coronary insufficiency 8 months prior to these electrocardiograms.

† Two patients had severe hypertension, 2 had moderate hypertension, 1 had mild hypertension and 1 had calcific aortic valvular stenosis. One of the 2 with severe hypertension had suggestive myocardial infarction 2 years prior to these electrocardiograms.

‡ Two patients had experienced onset of symptoms within 1 month prior to the electrocardiogram showing inverted T waves. The remaining 3 had nocturnally occurring angina pectoris.

Thus, it is evident that the implications of the terms under consideration are of a limited and peculiar character. They refer in each case to a clinical syndrome and no evidence other than that of clinical derivation was adduced in placing patients in one or the other category.

**Correlation of Clinical and Electrocardiographic Data.** The table records the correlation of clinical and electrocardiographic data. Study disclosed that the several categories of electrocardiograms bear widely differing relationships to the diagnosis of myocardial injury. The full range of variation is represented on the one hand by the tracings designated "QRS changes" and on the other by those categorized as "right ventricular hypertrophy or right bundle-branch block." Among patients whose electrocardiograms fell into the former category, 5 of 20 (25 per cent) had experienced clinical manifestations of myocardial infarction and 13 of 20 (65 per cent) had noted symptomatic expressions of severe coronary insufficiency. In the small group of patients whose
tracings evidenced right ventricular hypertrophy or right bundle-branch block in association with those changes in configuration of T waves characteristic of records included in this study, no patient presented convincing clinical evidence of myocardial injury or of coronary sclerosis.

Between the extremes represented by these two classes of electrocardiograms is that group labeled "typical." This study was designed to ascertain the clinical correlations in this group. The four other categories are included primarily as a means of defining by contrast or by similarity the probable significance of the results obtained in study of the "typical" group. Of the 62 patients in this category, nine (14 per cent) presented other clinical data strongly supporting the diagnosis of myocardial infarction and five (8 per cent) presented equivocal evidence supporting such a diagnosis. The term "equivocal" implies a high degree of qualification in the judgment that could be derived from evidence other than the electrocardiogram. In 28 of the 62 patients (45 per cent), evidence apart from the electrocardiogram supported the diagnosis of severe coronary insufficiency; in two other patients (3 per cent), equivocal evidence for such a diagnosis was elicited. Five of the 62 patients (8 per cent) gave accounts characteristic of angina pectoris and two (3 per cent) presented equivocal histories of such a condition. One patient had periarteritis nodosa attended by clinical evidence and ultimately by evidence at necropsy of involvement of the coronary arteries and focal myocardial scars. Thus, 43 of these 62 patients (69 per cent) presented substantial evidence of disease affecting the coronary arteries. Of these 43 patients, 38 (61 per cent of the total group) had clinical evidence of myocardial infarction or severe coronary insufficiency, and in five others (8 per cent) the diagnosis of angina pectoris was made on purely clinical grounds.

It is of interest that three of the 62 patients in the group whose electrocardiograms showed deeply inverted T waves unattended by changes in the QRS complexes presented absolutely no other clinical evidence of cardiac disease and that three others had no indications of cardiovascular disease apart from uncomplicated hypertension.

An attempt was made to organize such information as was available concerning the duration of a state of deep inversion in the T waves of the precordial electrocardiogram in this category of tracings called "typical." With regard to 25 of the 62 patients in this group, electrocardiograms were obtained over a period in excess of six months or over a period long enough to reveal complete reversal of deeply inverted T waves to an upright position. Of these 25 patients, only 4 failed to show such reversal when records were made over a period in excess of six months. The clinical diagnoses in these four patients and the interval over which deep inversion of T waves was recorded were chronic constrictive pericarditis (18 months), possible aortic stenosis (16 months), polyneuritis with no evidence of cardiac disease (8 months) and hypertension with equivocal evidence of severe coronary insufficiency (15 months). Of the remaining 21 of these 25 patients, one had acute toxic nephritis at the time of the electrocardiogram in which the T waves were inverted; he had made complete recovery when the essentially normal electrocardiogram was recorded seven months later. The other 20 of these 21 patients presented substantial clinical evidence of myocardial infarction or severe coronary insufficiency.

Although nothing resembling a state of statistical purity is claimed for these observations concerning duration of inversion of T waves, the reasonable surmise may be advanced that a transient and reversible state of deep inversion of the T waves of the precordial electrocardiogram bears a high correlation with the existence of clinical data supporting the diagnosis of severe coronary insufficiency or myocardial infarction.

The two remaining categories derived from the electrocardiographic findings, namely the "left-strain variants" and the "shallow T-wave inversions," were similar in that patients with angina pectoris constituted approximately half the total number in each category; no patient whose record included substantial clinical evidence of myocardial infarction or severe...
coronary insufficiency was included in either group.

Pathologic Data. Necropsy was done in only 4 of the 62 cases that constituted the “typical” electrocardiographic category. Since evidence obtained from so small a group of cases must necessarily be of most limited extent, the series was augmented by five other cases selected from the files of the Section of Pathologic Anatomy. The electrocardiographic features in these selected cases were similar to those in cases making up the “typical” category.

Healed subendocardial infarction was present in eight of these nine cases. The infarcted regions were predominantly in the lateral wall of the left ventricle in four of these eight, whereas the anterior wall was the site of predominant involvement in three and a small area of healed subendocardial scarring was present in the basal portion of the posterior wall of the left ventricle in the eighth. In the remaining case of these nine, scattered lesions of healed infarction were identified microscopically but no gross scarring was evident. The microscopic lesions were not concentrated in any one portion of the left ventricular wall. A detailed account of these pathologic data will be reported subsequently.

Comment

This study had as its objective a seemingly simple correlation involving clinical data and electrocardiographic findings. To this correlation were to be added, in those few instances in which such data were available, observations on the structural changes in the heart noted at necropsy. But like other studies of modest scope and limited objectives, this one has proved frustratingly difficult.

As already indicated, of the 110 sets of electrocardiograms in the final series, only 62 were classed as typical in the sense that they included the designated abnormalities in configuration of T waves and no other significant electrocardiographic changes. The remaining 48 sets have been distributed among four additional categories based on electrocardiographic characteristics alone. Even the introduction of five categories of electrocardio-

grams has not permitted achievement of an entirely satisfying degree of conformity among all records placed in a single class. However, to have demanded a greater degree of homogeneity would have led to a hopelessly confusing system of categories; five groups represented solution by compromise.

The other aspect of this ostensibly simple correlation entailed analysis of clinical data. Perhaps the decision should have been made to attempt to determine only whether or not there was evidence of disease affecting the coronary arteries as manifested by angina pectoris or myocardial infarction. This decision often was difficult enough, as indicated by the appreciable number of times that the evidence was rated as equivocal. The effort to distinguish a group of patients whose illness included the usual features of an attack of acute myocardial infarction from a group whose experiences might be related to severe coronary insufficiency may have represented an excessive degree of refinement in diagnostic classification. Admittedly the line of distinction was too faint in certain instances for positive delineation.

With admission of a degree of imperfection, at times distressing, in efforts directed at classification of both the electrocardiographic and the clinical data constituting this study, attention may be turned to such qualified conclusions as may be derived from such data. These conclusions will be developed in the form of answers to certain questions.

Question 1. How reliable an index is deep inversion of the T waves unattended by other significant alteration in the ventricular complex of myocardial infarction or severe coronary insufficiency? As the answer to this question, three sets of figures may be introduced.

a. Thirty-eight of 62 patients (61 per cent) who had “typical” changes in the T wave as the only significant alteration in the electrocardiogram had clinical evidence of myocardial infarction or severe coronary insufficiency.

b. Eighteen of 20 patients (90 per cent) who had not only these characteristic changes in the T waves but also a definite, if minor, abnormality of the QRS complex of a type associated with myocardial injury or scarring presented clinical evidence of myocardial
scarring or severe coronary insufficiency. The significance of this clinicoelectrocardiographic correlation is not its confirmation of what already is generally accepted as electrocardiographic indication of localized myocardial injury; rather, it is the measure it gives of the method used in this study for appraising the clinical data supporting the diagnosis of myocardial infarction or severe coronary insufficiency. The high incidence of supporting clinical evidence in a group of patients having established electrocardiographic evidence of myocardial infarction may be interpreted as indicating the existence of remarkably complete and accurate clinical records on these patients combined with a rather liberal attitude on the part of the investigators as to what constituted adequate support for a clinical diagnosis of myocardial infarction or severe coronary insufficiency.

c. None of the 13 patients whose electrocardiograms were of the type termed "left-strain variant" presented substantial evidence clinically of myocardial infarction or severe coronary insufficiency.

These observations delineate reasonably well the diagnostic implications of an electrocardiogram in which changes in the T wave are "typical." Such changes are not so reliable an index of myocardial injury as is found in a similar tracing in which certain minor alterations in the QRS complex are present but are far more significant in indicating such injury than an electrocardiogram in which changes are those of the kind encountered in the "left-strain variant" group.

One additional observation merits emphasis. In only one patient of the 62 who had "typical" T-wave changes was there incontrovertible evidence of a pathologic process other than coronary insufficiency or myocardial infarction as a basis for the electrocardiographic changes. This patient had chronic constrictive pericarditis and had undergone surgical exploration of the pericardium and heart. It is true that in 23 of these 62 patients the diagnosis of severe coronary insufficiency or myocardial infarction could not be established on clinical grounds, but neither could the presence of such disease be excluded as a basis for the electrocardiographic findings.

Question 2. Does the observation that the deeply inverted T waves revert in time to an upright position alter the diagnostic implications of such a record? As already indicated, in 21 of the 62 patients electrocardiograms were obtained in which such reversal in direction of T waves had occurred. It is re-emphasized that, except for the aforementioned patient who had acute nephritis at the time of the abnormal electrocardiogram, 20 of these 21 patients presented substantial clinical evidence of myocardial infarction or severe coronary insufficiency.

Question 3. Is an electrocardiogram exhibiting "typical" T-wave changes related at all consistently to any particular clinical syndrome of coronary disease? The effort expended in an attempt to answer this question probably exceeded that applied to any other portion of this study, but the answer still must be phrased in qualified terms. The impression derived was that an unusually large proportion of patients in this group presented stories of episodes of severe pain recurring over periods of days or weeks, unattended by shock or fever. As noted previously, for the purpose of this study this syndrome has been termed "severe coronary insufficiency" in contrast to "acute myocardial infarction," the latter diagnosis being applied when the episode or episodes of severe pain occurred within a more limited period and were attended by systemic manifestations of greater consequence. The ratio of patients having severe coronary insufficiency (28) to those having acute myocardial infarction (9) was approximately 3 to 1 in the series with only "typical" T-wave changes. A ratio of this same order was encountered in the series showing in addition to inversion of the T waves minor changes in the QRS complexes indicative of localized myocardial injury. Here, 13 patients were placed in the clinical category of severe coronary insufficiency, whereas to five others was attached a diagnosis of acute myocardial infarction. To conclude that "severe coronary insufficiency" as used in this study is an especially prevalent clinical counterpart among patients showing deeply
inverted T waves in the precordial electrocardiogram attended by minor QRS alterations or by none at all, appears to be reasonably justified.

**Question 4.** On the basis of such pathologic data as are available, is there a type of myocardial lesion commonly associated with inverted T waves of the kind under consideration in this study? In almost all instances in which necropsy findings are available, subendocardial myocardial infarction in the anterior or lateral wall of the left ventricle has been found.

**A Matter of Theory**

In a portion of the myocardium where the process of repolarization is prolonged with respect to its duration in other parts, a potential will exist late in electric systole that is negative relative to that existing in other portions of the myocardium. A record derived from an exploring electrode so disposed as to permit recording of changes in potential in that region where repolarization is most delayed will show inversion of the T wave.

Illustration of this principle can be accomplished readily under laboratory conditions where changes of limited character can be wrought in carefully delineated zones, as, for example, by cooling a small region of the epicardium. Application of this same derivative from electrocardiographic theory to an understanding of the alterations in T waves as the latter are encountered clinically is not always so evident. Perhaps no better illustration of this point could be cited than the specific problem of this study, namely the deeply inverted T waves of the precordial electrocardiogram. Exclude from consideration those changes related to ventricular hypertrophy and confine the argument to so-called "primary T-wave changes," and the discussion retains perplexing issues.

Deeply inverted T waves should appear in the precordial electrocardiogram when the process of repolarization in the epicardial myocardium is accomplished later than it is in the endocardial myocardium. A pathologic process that produces a greater degree of injury in the epicardial than in the endocardial fibers is pericarditis. It is true that in the resolving phase of acute pericarditis, T-wave inversions appear in precordial records from points overlying the involved regions. However, coronary disease and not pericarditis was the prevalent pathologic process in this study among patients whose electrocardiograms revealed characteristic inversion of T waves. Study of the changes encountered at necropsy in this series supports the accepted view that coronary disease produces its severest lesions on the endocardial aspect of the left ventricular wall and indicates furthermore that the necrotic tissue commonly is confined to the endocardial half of the left ventricular wall in patients showing only deeply inverted T waves unattended by major changes in the QRS complexes. We may postulate that superficial to these necrotic fibers lay ischemic fibers and that in those nearer the necrotic zone the process of repolarization was retarded to a degree greater than it was in the epicardial fibers. By such reasoning, the T waves in an epicardial lead or in a precordial lead from a point overlying the involved region of the ventricular wall should be upright. But our evidence indicates these deflections were, in fact, inverted.

In an attempt to account for this apparent paradox, an added perspective may be found in the review of sequential records in a case that, from the electrocardiographic standpoint, was unusually well documented. The initial set of electrocardiograms in figure 7 was obtained within a few minutes after the patient began to experience pain in his thorax. In this record, the RS-T segments in precordial leads V₃, V₄ and V₅ were depressed and the T waves were upright and exceedingly large. These alterations are of a kind encountered commonly in the electrocardiograms of patients with coronary disease who are subjected to stress, and it is this type of change that has been said to be characteristic of subendocardial myocardial infarction in previously reported studies of this lesion; in both instances, the ascription of their origin to a boundary so oriented that endocardially disposed myocardial fibers are more severely injured than the more superficially disposed fibers is justified. The transient
character of these changes is illustrated by the fact that in the patient under consideration they were no longer present in an electrocardiogram made approximately two hours after the initial tracing. This second record differed in no discernible way from that dated 9-2-51 in figure 7. In the record dated 9-3-51, approximately 36 hours after the patient first experienced pain, the T wave in precordial leads V3 and V4 had become deeply inverted, whereas in the record of 9-28-51 these deflections had returned to a nearly normal configuration.

Let us consider now the contribution that this sequence of electrocardiograms may make to the resolution of the paradox of T-wave inversion in the presence of postulated subendocardial ischemia. In this series of tracings, the initial record was of a type totally consistent with the existence of an acute subendocardial injury overlaid by a zone of ischemic myocardium. An electrocardiogram taken approximately 36 hours later revealed deeply inverted T waves in precordial leads V3 and V4, a type of change specific to the interests of this study. What redistribution of boundaries would permit the T waves to undergo so striking a reversal in direction? An answer is suggested in diagrams A and B of figure 8.

In A, representing the initial phase of the electrocardiographic sequence, the boundary of injury a is responsible for depression of the RS-T segments in complexes recorded from the electrode c. The boundary of ischemia (b-b) is the source of those forces producing the tall upright T waves at this same phase. (The lines a and, especially, b-b symbolize boundaries composed of many layers of myocardial fibers.)

In B, representing the phase of T-wave inversion, the boundary of injury has become stabilized as the fibers in that zone died or recovered sufficiently to become part of the ischemic zone. The voltages arising at that boundary have disappeared from the field. In contrast, the ischemic zone, composed of fibers capable of responding to excitation but characterized still by an abnormality of the process of repolarization, has increased in size and now extends transmurally. The effective boundaries of this ischemic zone now lie at its edges and not in a plane approximating that of the epicardium and endocardium. As a conse-
Fig. 8. Diagrammatic representation of the boundaries at which arose the forces responsible for segmental and T-wave aberrations in the series of electrocardiograms reproduced in figure 7. See text for discussion.

in the electrocardiogram

sequence of this reorientation of boundaries between stages A and B, electrode c' now lies within the negative portion of the electric field and the T waves in a tracing made from this lead will be inverted.*

By this line of argument, the conclusion may be justified that deeply inverted T waves in a direct or semidirect electrocardiographic lead do not, of necessity, imply preponderant epicardial ischemia. Such inversions probably are associated with transmurally disposed regions of ischemia under which a subendocardial zone of myocardial infarction may lie. These inverted T waves are not a consequence of the existence of that infarcted tissue, but our studies, clinical and pathologic, support the conclusion that when characteristic deep inversion of the T waves develops in a patient presenting evidence of severe coronary insufficiency or myocardial infarction, it is a reasonably reliable index that one or more zones of necrotic fibers exist in the subendocardial region. Because of the more enduring character of the T-wave changes as compared with the segmental deviations associated with acute subendocardial myocardial injury, the former may serve more commonly than the latter as an index of the presence of subendocardial myocardial infarction.

In Perspective and Reflection

Review of numerous publications by Myers and associates has disclosed a scattering of electrocardiograms of the type under consideration. Case 43 of a July, 1950, publication* is that of a 15 year old girl with severe hemolytic anemia in whose electrocardiogram deeply inverted T waves were present transiently. At necropsy no myocardial lesions were found, and the changes were ascribed to acute myocardial ischemia secondary to hemolytic anemia. The electrocardiograms of April 4 in case 45 of this same paper were of the type we are concerned with. At necropsy, findings included subacute vegetative aortic valvulitis and widespread miliary abscesses of embolic origin. There was no evidence of myocardial infarction, and gross signs of pericarditis were absent. Microscopically, acute subepicardial myocarditis was present, to which were ascribed the RS-T changes. It might be questioned, however, whether or not the widespread miliary abscesses played a role in the production of these electrocardiographic changes. Case 7 of another 1950 article by Myers* is an excellent example of the type of case that poses the

* These arguments, as they depend on the significance of forces produced at the edges of a myocardial lesion, are comparable to those advanced in accounting for segmental deviations recorded in the presence of experimentally induced transmural myocardial lesions.1
difficult problem of whether or not the inverted T waves can be related solely to the consequences of left ventricular hypertrophy. Case 20 of a third 1950 publication by Myers affords illustration of T wave inversion in leads V₁ through V₄ as a consequence of pulmonary embolism and acute cor pulmonale. This record is distinguished from comparable instances in the present series by the absence of QRS peculiarities in lead V₁ or V₂ of Myers' case and their presence in the records in our series.

Among the series of articles by Myers and associates on myocardial infarction, two illustrations are included of electrocardiograms characterized by deeply inverted T waves. At necropsy, in case 150, findings included an extensive subepicardial lesion that involved the entire lateral wall and overlapped onto the anterior and posterior walls of the left ventricle. Case 125, while complicated by terminal posterolateral infarction, illustrates an instance in which inverted T waves probably were produced by an incident that left multiple small zones of fibrosis in the subendocardial half of the anteroseptal wall of the left ventricle midway between apex and base.

In relation to this latter case, Myers and his coworkers speculated that an electrocardiographic pattern characterized by deeply inverted T waves in precordial leads V₂ and V₄ “brought up the following possibilities: Acute anteroseptal ischemia, a small intramural or subepicardial infarct, acute right ventricular dilatation, and pericarditis.” With these speculations we would take no serious issue. However, we would add to this list the possibility of subendocardial infarction of a patchy or confluent type associated with transmural myocardial ischemia and would propose that this was the probable cause of the changes found in this electrocardiogram, as it probably was in many of the cases in our series.

The wariness with which sound clinicians have viewed the general problem of interpretation of changes in the configuration of T waves as an isolated electrocardiographic finding is well represented by Rosenbaum's statements, as he expanded in some measure the earlier comments of Wilson and associates: “The diagnosis of myocardial infarction can be made on the basis of T wave or RS-T segment changes only if a characteristic sequence of alterations, such as shown here, is observed. Even if such a sequence is recorded, it is usually best to be certain that the clinical picture is compatible with the diagnosis. The diagnosis is almost never justified from a single record which shows changes confined to the T waves and RS-T segments. It is probable that infarcts that give rise to changes of this type are relatively small in their extent, or they may be in an unusual location in respect to the type of leading now employed. Most patients with infarcts of this type do well and have no serious impairment of function upon recovery.”

Findings in the present study certainly demand no radical revision of this position. Rather our results lend support to every phase of the summary. Perhaps the position of one interpreting such an electrocardiogram is strengthened by the knowledge afforded by the results of this study of what apparently is the largest collection of such records with which medical literature thus far has been encumbered. The present-day “electrocardiographer” is reduced at times to the necessity of laboring over fields that remain unexplored solely by virtue of their total unloveliness.

**SUMMARY AND CONCLUSIONS**

1. The presence of deeply inverted T waves in electrocardiograms from central terminal leads centered about position 3 on the precordium unattended by significant changes in the QRS complexes in tracings derived from these or other leads was related in 38 of 62 patients (61 per cent) to clinical evidence of myocardial infarction or severe coronary insufficiency.

2. A transient and reversible state of deep inversion in the T waves obtained from these same leads bears an exceedingly high correlation with the presence of data supporting the diagnosis of severe coronary insufficiency or myocardial infarction.

3. “Severe coronary insufficiency,” as used in this study to describe a clinical state differing in some respects from the usual symptomatic manifestations of acute myo-
cardial infarction, was an especially prevalent clinical counterpart of electrocardiograms of the type noted in the first paragraph of this summary.

4. In almost all cases in which necropsy findings were available and in which electrocardiograms had shown deformities of the kind under consideration, subendocardial myocardial infarction was found in the anterior or lateral wall of the left ventricle.

5. Expressed in terms of the dipole theory, deeply inverted T waves of the kind described in this study are commonly an expression of the presence of a transmurally disposed region of myocardial ischemia under which there may be a subendocardial zone of myocardial infarction.

SUMMARIO E CONCLUSIONES IN INTERLINGUA

1. Esseva interprende un correlation del constataiones clinic e electrocardiographic in 62 casos (seligite ex un serie de 110 casos), que habeva in commun le tracto de undas T a inversion profunde in derivationes terminal central concentrate circa le position 3 del pre-cordio sed nulle cambiamentos significative del complexos QRS in iste o altere derivationes. In 38 inter le 62 casos (61 pro cento), le presentia de undas T a inversion profunde esseva associate con evidentia clinic de infarcimento myocardiac o sever insufficientia coronari.

2. Transientia o reversibilitate del profunde inversion del undas T in iste derivationes occurreva in altissime correlation con datos supportante le diagnose de sever insufficientia coronari o infarcimento myocardiac.

3. "Sever insufficientia coronari"—que se interpreta in iste studio como un stato clinic differente in alicun respectos ab le usual manifestationes symptomatic de acute infarcimentos myocardiac—esseva un facto clinic prevalen-

tissimemente associate con electrocardiogrammas del typo descritibe in le prime paragraphe de iste sumario.

4. In quasi omne casos in que observationes necropitic esseva obtenite e in que le electrocardiogrammas habeva exhibite deformitates del typo sub consideration, infarcimentos myocardiac subendocardiac esseva trovate in le pariole anterior o lateral del ventriculo sinusire.

5. Expressite in le terminos del theoria dipol in undas T a inversion profunde del typo descritibe in iste studio es generalmente indicative del presentia de un region de ischemia myocardiac in disposition transmural sub le qual se trova possibilemente un zona subendocardiac de infarcimento myocardiac.

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