“Isolated” T-Wave Negativity in the “Ischemic Phase” of Myocardial Infarction in Man

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T-wave inversion, in leads localized to a small segment of the precordium, has in rare instances been described as a normal variant. The present report concerns a number of individuals in whom more or less similar T-wave changes developed under circumstances suggesting myocardial ischemia, and who went on to develop acute infarction of the anterior wall of the heart. This may be a clinical analogue of the brief T-wave inversion which has been observed early in experimental coronary artery compression.

INVERSION of the T wave has been described as the immediate consequence of experimental ligation of the descending branch of the left coronary artery.1-3 It is only on more prolonged occlusion that changes develop in the RS-T segment or in the QRS complex. In acute coronary thrombosis in man, by contrast, an elevation of the RS-T segment is generally the earliest change recorded. This may be associated with or followed by terminal inversion of the T wave or by the development of an inverted U wave. It may likewise accompany or precede changes in the QRS complex. The possibility that inversion of T waves might occur as an isolated finding in the earliest stages of acute clinical myocardial infarction has frequently been suggested or inferred but never convincingly demonstrated. Most of the publications appearing in the last decade or two on the “impending,” 4, 5 “preliminary,” 6 “premonitory” 7-9 or “formative”10 stages of myocardial infarction were written when only the three conventional leads and one, 4, 6, 7, 11 two12 or three13, 14 chest leads were recorded. These showed no changes at all4-7 or instability of the T waves4, 6, 8-13 which was difficult or impossible to localize or evaluate because of what most electrocardiographers now consider inadequate electrocardiographic exploration. Yater and his co-workers14 considered a “premonitory” phase of acute coronary artery occlusion as too vague and intangible a subject to deserve discussion; they pointed out that any anginal form of attack, except in established cases of angina pectoris, should be considered premonitory until proved otherwise. It has been noted moreover that in rare clinical instances of acute myocardial infarction associated with fever, leukocytosis or elevation of the sedimentation rate, the electrocardiogram may reveal only the development of deeply inverted T waves14-16 in all precordial leads.

It is the purpose of this paper to present six patients who may be considered as having been adequately explored, who demonstrated a possible early “ischemic” phase of acute anteroseptal myocardial infarction, and a seventh patient in whom the electrocardiogram reverted to normal. In these cases the T-wave inversion was “isolated” in two senses: namely in the temporal sense that they preceded the development of alterations in the RS-T segment and/or the QRS complex, or never progressed beyond the T-wave phase, and in the topographic sense that in this early “T-wave” phase the changes were prominent in one or two of the midprecordial leads, such as leads V3 and V4, or leads V2 and V5, while the flanking leads showed these changes to a much lesser extent (cases 2, 3 and 5) or not at all (cases 1, 4, 6, 7). Five of the patients were on the wards of the Peter Bent Brigham Hospital, one at the New England Deaconess Hospital and one was seen in the private practice of one of us (H.D.L.). None of the patients received digitalis prior to or during hospitalization.
Case 1. D. N. (P.B.B.H. 7E739), a 54 year old construction worker, had had severe kyphoscoliosis with mild dyspnea on exertion following an injury at the age of 6 months. There were no other cardiac symptoms until one month before admission when he began to suffer from angina pectoris. This became progressively more frequent and severe. Two weeks before admission one episode lasted two hours and required morphine for relief. Following this he was treated in another hospital where electrocardiograms were not remarkable, and roentgen examination showed his gall bladder and gastrointestinal tract to be normal. He was discharged from that hospital five days before admission to Peter Bent Brigham Hospital. His angina pectoris, however, rapidly became almost completely incapacitating with very little relief from nitroglycerin, and he was admitted to the Peter Bent Brigham Hospital on Nov. 24, 1952.

The patient was a short, severely deformed man who had a typical attack of angina pectoris during the admission examination. He had a marked dorsal kyphoscoliosis with the convexity to the right. The blood pressure was 120/80 and the pulse rate 70. There were a few moist rales at the left base which disappeared a few hours after admission. There was no other evidence of congestive failure. The heart was apparently moderately enlarged, but the chest deformity made the evaluation of heart size difficult. The heart sounds were of good quality. There was no murmur, gallop rhythm or friction rub. The remainder of the physical examination was not remarkable. Electrocardiograms on admission (fig. 1A) showed predominantly inverted T waves in leads V₃ and V₄ with upright T waves in the flanking leads. These suggested myocardial ischemia, but were possibly a normal variant. A tracing the next day was essentially unchanged in the usual 12 leads. Precordial exploration showed the area of T-wave inversion to extend into the third left intercostal space at the vertical levels of V₅ and V₆ but not into the accessible second intercostal space. Exploration was not car-

**Fig. 1.** Isolated T-wave negativity as prelude to acute transmural antero-septal infarct. The patient was a 54 year old man with kyphoscoliosis. Angina pectoris had been present for one month, increasing in frequency and severity. Previous electrocardiograms were normal. Initial tracings recorded on day of admission show biphasic T in lead V₃, inverted T in lead V₄. The Q-T interval is normal. No laboratory evidence of tissue breakdown (A). The second set of tracings (11/26/52) shows slightly depressed RS-T segments in leads V₃, V₄ and V₅ (B) compatible with subendocardial ischemia. The third series of curves (11/28/52) shows diagnostic changes of acute anteroseptal infarction (C). The Q-T interval is normal. At this time the patient had fever and leukocytosis. The final set of tracings three months later shows residual changes of anteroseptal infarction (D).
ried out inferiorly but the T waves were upright in leads aVR, and aVF (fig. 2). These changes were interpreted as representing localized ischemia. On Nov. 26, 1952 (fig. 1B), tracings suggested subendocardial ischemia.

Leukocytosis (11,300) and fever (101 F, rectally) did not develop until Nov. 28, 1952, the day when decisive electrocardiographic evidence of acute transmural antero-septal myocardial infarction was first recorded (fig. 1C), and the sedimentation rate did not rise until five days later. The patient was treated with the chair and bed program, sedatives and anticoagulants and received nitroglycerin and narcotics for pain. During the first four days he continued to have frequent, moderately severe episodes of chest pain. Then, coincident with the appearance of definite electrocardiographic evidence of infarction, the pain disappeared. It recurred in a much less severe form 10 days later. Tracings on December 3, 9, 11 and January 14 demonstrated the usual sequence of RS-T segment elevation followed by a return to the base line with the development of terminal T-wave inversion, still present in the final tracing taken Feb. 21, 1953 (fig. 1D). Since discharge on Dec. 22, 1952, his course has been uneventful, although he still has occasional mild angina pectoris.

Case 2. L. J. (P.B.B.H.), a 61 year old widow, had rare episodes of angina pectoris for several years, hypertension for four years and a pulsating

**Fig. 2.** Topographic exploration of precordium in the same patient on 11/25/52. The area of T-wave negativity involves leads V3 and V4 in the usual six precordial leads, and extends to the vertical level of V4, V5, and V6 in the fourth intercostal space and V4 in the third intercostal space. The T wave is almost flat at the vertical level of V5 in the third intercostal space but upright in the entire accessible portion of the second intercostal space and in lead aVL. Exploration of the lower precordium is incomplete.
upper abdominal mass for one year. Eighteen days before admission while walking she developed an epigastric pain similar to her angina pectoris but lasting an hour before subsiding spontaneously. She had three similar but milder attacks in the week preceding admission; then on Dec. 15, 1952, she was awakened by severe knife-like precordial pain, radiating to her left shoulder and arm but also spreading into the left upper quadrant of her abdomen. The pain persisted in somewhat milder form until she was admitted eight hours later.

When first seen she was in acute pain, clutching at her chest and upper abdomen. The blood pressure was 170/100 and the pulse rate 106. The heart was not enlarged and no murmur, gallop rhythm or friction rub was heard. There was no respiratory distress, increased venous distention or edema, but a few rales were heard at the left lung base. There was a pulsating mass in the left upper quadrant of the abdomen, measuring 5 or 6 cm. in diameter. Roentgen-ray examination showed it to be an abdominal aneurysm with calcification in its wall. Laboratory studies showed a persistent trace of albumin in her urine. Her sedimentation rate on admission was 7 mm. in 1 hour; this soon rose to 37 mm. in 1 hour.

The electrocardiographic sequence is illustrated in figure 3 and described at length in the legend. Here again localized T-wave inversion gave way rapidly to characteristic changes of acute antero-septal infarction.

Fig. 3. T-wave negativity most pronounced in leads V₂ and V₃ briefly preceding acute antero-septal infarction. The patient was a 61 year old woman with angina pectoris, hypertension and thrombosed abdominal aneurysm. There had been four prolonged attacks in the 18 days before admission. The patient suffered eight hours of precordial pain. The initial electrocardiograms (12/15/52) show inversion of T waves in leads V₁ through V₄, most pronounced in lead V₂ (A). The T waves are flat in V₃ and V₄. The Q-T interval is prolonged by 18 per cent. The patient had fever at this time but no laboratory evidence of tissue breakdown. The second set of tracings recorded later that day (B) shows elevation of RS-T segments and late inversion of T waves in leads V₁ through V₄. In conjunction with the clinical story these changes justified a clinical diagnosis of acute myocardial infarction.

The third set recorded the following day (12/16/52) shows diagnostic evidence of acute antero-septal infarction (C). The Q-T interval is prolonged 40 per cent ("post ischemic" T waves). The next two sets (12/17 and 12/31) show characteristic sequential changes (D, and E) while the final set (1/14/53) shows no decisive residual changes of infarction (F). At this time T waves were inverted everywhere but maximally in lead V₃.
Fig. 4. Isolated "ischemic" T waves followed in two and one-half weeks by decisive changes of acute anteroseptal infarct. The patient was a 54 year old man with angina pectoris for nine months; the angina had been more frequent recently but was always promptly relieved by nitroglycerin. Three-fourths hour of chest pain brought the patient to the hospital. The initial set of tracings (11/29/52) shows inversion of T in lead V4, clearly biphasic (+-) T waves in lead V5 and slightly biphasic T waves in lead V6 (A). The Q-T interval at this time is shortened to 10 per cent below expected normal ("ischemic" T waves). The second set (12/1/52) shows more deeply inverted T waves in leads V4 and V6 (B). On 12/10/52 the inversion of T waves is even more pronounced and widespread but evidence of infarction is still lacking (C). On 12/17/52 the R waves have disappeared in leads V1 and V2, are minuscule in lead V3, and the tracings are diagnostic of acute anteroseptal infarction (D). The Q-T interval is lengthened by 20 per cent ("postischemic" T waves). The tracings three months later show residual evidence of old anteroseptal infarct with inverted T waves (E).

For the first two days the patient had an elevation of her temperature, pulse and respiratory rates. Her blood pressure gradually fell to 100/80 18 hours after admission, then slowly rose to 140/80 on discharge. The chest pain was controlled with morphine. It was noted that the abdominal aneurysm remained tender and that very slight pressure over it reproduced the pain in her upper abdomen and at times in the substernal area. She suffered no recurrence of precordial pain radiating to her shoulder or arm. She had an occasional episode of upper abdominal pain, not radiating to the precordium. On Jan. 26, 1953, aortography showed that the sac of the aneurysm was already lined with a solid thrombus and no surgical treatment was recommended.

Since discharge on Jan. 30, 1953, she has continued to have occasional episodes of upper abdominal pain in addition to infrequent episodes of angina pectoris which she considers distinctively different from the abdominal pain. Her blood pressure has returned to former levels.

Case 3. R. H. (P.B.B.H., 7E761*), a 54 year old man, was well until nine months before admission when he first developed typical angina pectoris. This always abated within five minutes on resting. For three weeks prior to admission this had occurred

* We are indebted to Dr. A. W. Contratto for permission to use this case and for two of the electrocardiograms reproduced.
Fig. 5. Protracted isolated T-wave negativity with brief transient RS-T segment elevation associated with intramural myocardial infarction. Case 4, M. A., was a 47 year old man with angina pectoris. Initial tracings show inversion of T waves limited to leads V3 and V4 (A). Tracings recorded the following day with additional precordial leads are shown in figure 6. Daily tracings showed no change until 4/14/54 when the T-wave inversion gave way to RS-T segment elevation in leads V2, V3 and V4 (B). The expected subsequent progression failed to develop. Instead tracings continued essentially unchanged over those recorded on admission (C and D). The patient died two days after the last tracing. Postmortem examination showed recent intramural infarction at the apex of the left ventricle. The left photomicrograph showed normal subepicardial (E) and the right photomicrograph normal subendocardial myocardium (H). The two intervening sections (F and G) show portions of isolated and conglomerate intramural necroses. The third section shows necrotic and infiltrated muscle bordering on intact muscle layers. (X90)

more frequently. On the morning of admission, while on the way to his physician, he noted the onset of a similar pain which lasted 45 minutes and was relieved by nitroglycerin. He was then admitted on Nov. 29, 1952.

When seen in the hospital a few hours after the subsidence of pain, he was in no acute distress and physical examination was not remarkable. The blood pressure was 125/80 and showed no significant fall throughout the hospitalization. There was no fever, leukocytosis or elevation of the sedimentation rate. In the absence at that time of definite evidence for the diagnosis of myocardial infarction, but with the purpose actually of preventing myocardial infarction, he was treated with anticoagulants. During the first two weeks he required occasional nitroglycerin for chest distress; thereafter he had angina pectoris only rarely, he recovered uneventfully but still has an occasional episode of angina pectoris.

The electrocardiographic sequence is illustrated in figure 4 and described in the legend. It was shortly after the episode of severe pain that the tracings...
showed localized inversion of the T waves, but it was not until the passage of another 19 days that the tracings showed diagnostic evidence of infarction.

Case 4. M. A., a 47 year old dye-worker, had noted over a six-week period epigastric and substernal distress on walking two blocks or more, relieved by stopping, and occasionally associated with a sense of gaseous distention which at times was relieved by belching. He was admitted on April 8, 1954. Except for his sthenic habitus examination was negative. The important laboratory findings were the white blood cell counts and the corrected sedimentation rates recorded on the following dates respectively: April 8, 10,000 and 16 mm., April 9, 15,300 and 22 mm., April 10, 12,000 and 31 mm., April 12, 8800 and 32 mm., and April 20, 6200 and 33 mm. An electrocardiogram on April 9 (fig. 5A) showed T-wave inversion limited to leads V3 to V4. On the following day, April 10, 1954, extensive exploration of the precordium (fig. 6) showed that the area of dispersion of this T-wave inversion was quite widespread, thus contrasting with the rather limited distribution of this change when it is encountered as a normal variation \(^{16, 17}\) or as observed in case 1.

A transient pronounced degree of RS-T segment elevation was recorded on April 14 (fig. 5B), but this disappeared on the following day (fig. 5C) and the tracings remained substantially unchanged (fig. 5D) over those recorded on admission until his sudden demise on April 26. During the earlier part of his hospitalization he had frequent pains in both arms lasting from 10 minutes to an hour or so and relieved by nitroglycerin. He had been quite free of pain during the last week of his life, until the night before his death when it returned with unrelenting severity. Postmortem examination showed a recent thrombus in the descending branch of the left coronary artery. There was also a fairly recent intramural infarct in the region of the apex of the left ventricle (fig. 5F and G). There was no evidence

![Electrocardiogram](image)

**Fig. 6.** Topographic exploration of precordium in same patient on 4/10/54. This is "apparent" isolated T-wave negativity in that the T wave inversion in leads V3 and V4 is clearly an extension from above into the line of the usual six leads of a large area of T-wave negativity. It is, therefore, not the very closely circumscribed, paradoxic and benign "isolated T-wave negativity" of Grant.
**FIG. 7.** T-wave inversion over midprecordium protracted over three-week period of angina decubitus preliminary to acute anteroseptal infarction. Case 5, J.F.S., was a 45 year old diabetic with an old myocardial infarct one year, and angina decubitus beginning two months, before initial electrocardiograms (A) which showed low or flat T waves. The patient admitted with exacerbation of angina two weeks before the second tracing (11/27/53) which, for the first time, showed T-wave inversion over a large part of the precordium but with upright T-waves in V_1 and flat T-waves in V_6 (B). These changes persisted throughout the remainder of hospitalization. The third set of tracings was recorded on readmission (12/31/53) for a classic episode of acute anteroseptal infarction (C).

**Fig. 8.** Isolated T-wave inversion persistent over five-year period. Case 6, F.S., was a 52 year old physician with angina pectoris for eight years. Repeated tracings from 1949 to 1954 showed isolated T-wave inversion in leads V_3, V_4 and V_5 (A). At the time of the original tracing the T wave of a postextrasystolic beat was lowered (see arrow), suggestive evidence for myocardial ischemia. In January 1954, the patient suffered a mild anteroseptal infarction (B).

whatever of extension of the infarct to the epicardial (fig. 5E) or endocardial surface (fig. 5H).

Case 5, J. F. S., a 45-year old undertaker with diabetes mellitus of 27 years' duration, suffered a myocardial infarction in November 1944, with good recovery. In October 1953, his electrocardiogram (fig. 7A) was normal except for low to flat T waves. In early August 1953, he had begun to have intermittent epigastric or low substernal pain, nonradiating, lasting from one to four minutes, never related to exertion, but at times precipitated by emotional tension. He frequently awoke from a dreamless sleep with pain which improved when he sat upright.
Nitroglycerin usually gave relief after producing an initial exacerbation of the pain. The pain became so much more frequent and severe that the patient was admitted to the New England Deaconess Hospital on November 13, 1953. Gastrointestinal studies were normal. The white blood cell count was normal on admission, rose to 12,300 on November 19 and to 12,500 on November 20; it fell to 8,200 on November
21. The sedimentation rate remained normal during his hospital stay and he was afebrile throughout. An electrocardiogram recorded during severe spontaneous pain showed pronounced RS-T segment depressions typical of "subendocardial ischemia" but these displacements disappeared when he was free of pain. In comparison with the original tracings (fig. 7A) the electrocardiograms showed no alterations until November 27, when they showed striking inversion of the T waves in leads V₂, V₃, V₄ and V₅, most pronounced in lead V₃ (fig. 7B). T was upright in lead V₁ and flat in lead V₆. During the remainder of his hospitalization the electrocardiograms showed no important changes, never developing RS-T or QRS changes in spite of persistent angina pectoris. At the time of his discharge on December 18 he was having only an occasional attack of chest pain. He was re-admitted on Dec. 30, 1953, in a fullfledged attack of acute anteroseptal infarction. The electrocardiogram now showed diagnostic changes (fig. 7C) and the white blood cell count rose to 25,300. He had a much more comfortable time of it in this second admission and was discharged improved on Feb. 18, 1954. He died suddenly in mid-June 1954, five days after an attack of chest pain unassociated with electrocardiographic or other laboratory evidence of myocardial infarction. Permission for postmortem examination was not granted.

Case 6. F. S., a 52 year old physician, had noted substernal pain on walking against a cold wind during the past eight years. In 1949, an electrocardiogram, made in connection with a survey conducted by the United States Public Health Service showed T-wave inversion localized to leads V₂ and V₄ (fig. 8A). For two weeks he suffered from what we have chosen to call "paroxysmal nocturnal angina," a type of angina decubitus which would wake the patient from his sleep and which would be relieved by sitting upright in bed. Later the night pain was not thus relieved and he had prolonged pain during the day. On admission (Jan. 29, 1954), his white blood cell count was 12,400. His corrected sedimentation rate rose to a high of 27 mm. in 1 hour on Feb. 1, 1954, and during the first 36 hours of hospitalization he had a slight fever (maximal temperature 100.8 F.). For a few days he continued to have episodes of pain promptly relieved by nitroglycerin and on one occasion by right carotid sinus stimulation which at the same time produced cardiac arrest lasting seven seconds. Electrocardiograms (fig. 8B) showed initial and sequential changes characteristic of acute anteroseptal infarction from which he made a satisfactory recovery.

Case 7. M. K., a 44 year old man, consulted an otolaryngologist because of a choking feeling in the throat occurring after meals, at rest or during work. He was referred for an electrocardiogram which showed notching with minimal inversion of the T waves in leads V₁ and V₃ and flat T waves in lead V₆ on March 4, 1954 (fig. 9A). Repeat tracings recorded five days later were within normal limits (fig. 9B), but a Master two-step exercise test at that time (fig. 9C) induced changes characteristic of acute coronary insufficiency.

Although we know of no long-term observations regarding the localized T-wave inversions described by Grant and his coworkers (16) as a normal variant, it would, we believe, be expected to remain a fixed feature of the electrocardiogram. In this case, by contrast, the disappearance of the localized T-wave changes would appear to make them doubly significant. The latency of "ischemic" changes was then demonstrated by the "positive" two-step test. In this connection it is of interest that, whereas the spontaneous change consisted of notching of the T wave, the induced change consisted of RS-T segment depression. This experience is similar to that in case 5 (J.F.S.) in which RS-T segment depression was induced by the two-step test and was recorded in much greater degree during a spontaneous episode of pain, though the subsequent spontaneous development was the evolution of profound T-wave inversion.

In the clinic, then, it appears that these early T-wave changes, whether or not associated with evidence of tissue breakdown, may progress through the stages of RS-T and QRS changes after a short (cases 1, 2 and 3) or longer (cases 5 and 6) interval; they may progress to the stage of RS-T segment shift and, rather than proceed to develop more definitive changes of myocardial infarction, actually recede to the stage of pure T-wave inversion (case 4); or they may regress from a phase of T-wave inversion to a normal electrocardiogram (case 7).

**DISCUSSION**

The early ischemic changes recorded by Bayley in dogs were present for only a matter of seconds or minutes. It has been suggested that, if similar electrical changes actually occur clinically in man, it is extremely unlikely that they would be recorded, since the patient simply does not present himself to the physician during the first few moments of an acute coronary thrombosis. The observations of Graham and Laforet on accidental coronary artery ligation in man probably come as near to the experimental set-up of Bayley as we are likely to come; one of these showed T-wave negativity, the other increased T-wave posi-
tivity. The first patient described by Roesler and Dressler\textsuperscript{26} showed transient inversion of T in lead V\textsubscript{3} during the subsidence of a spontaneous attack of angina pectoris at the height of which RS-T segment elevations had been recorded. Is it possible that the changes, which are induced in the dog over a minute or so by deliberate ligation, may develop spontaneously in man over the course of hours or days? If the process of closure, whatever its precise mechanism, were slow and progressive, it seems conceivable that such changes might indeed be recorded clinically. In the present group of cases the interval between the known presence of T-wave inversion and the development of electrocardiographic alterations diagnostic of acute myocardial infarction was 4 days in the first case, 10 hours in the second, 19 days in the third, 34 days in the fifth, and 5 years in the sixth case. In the fourth case there was a five-day interval between the T wave inversion and RS-T segment elevation which then disappeared. And in the seventh case the T-wave inversion disappeared and more profound changes never developed. In some of those cases the interval might have been found to be shorter if more frequent tracings had been taken.

The six attacks of acute myocardial infarction described in the present series of cases, as well as in the series of Miller and Drori,\textsuperscript{24} were preceded by periods of varying duration in which the patient suffered from angina pectoris. Delineation of the period of actual myocardial infarction from that of pure angina pectoris would be ill-defined and arbitrary. It would be difficult then to decide whether the T-wave changes described belong properly with the period of angina pectoris or with that of myocardial infarction. In four of these six episodes of acute infarction the phase of T-wave inversion was associated with such evidences of tissue breakdown as fever, leukocytosis or elevation of the sedimentation rate.

There is a large, well-founded body of opinion which separates from angina pectoris and acute myocardial infarction an intermediary type of response on the part of the heart to oxygen deprivation, namely, the status or phase of "coronary failure"\textsuperscript{21-23} or "coronary insufficiency."\textsuperscript{24-27} It might properly be maintained that the present T-wave changes are electrocardiographic manifestations, not necessarily the only type of electrocardiographic response, of that status. One could consider the subsequent electrocardiographic changes as the result of infarction of previously ischemic areas, whether or not these areas were then the site of small-scale necroses. In our opinion this is possible but unproved. The only fatal and autopsied case in this series, one of the two which never developed QRS changes, showed grossly evident infarction of a peculiar topographical distribution, namely an intramural infarct, which failed to involve the epicardium or endocardium. It would appear to be desirable to report, as much more convincing than any of the above-described cases, any instance of sequential changes, first in the T wave, then in the RS-T segment and QRS complex, occurring in a single, brisk, clear-cut episode of acute myocardial infarction not preceded by angina pectoris. To the best of our knowledge, such a case has not been described. The early T-wave changes as recorded in the experimental animal also deserve confirmation. Inversion of the T waves is a nonspecific affair.\textsuperscript{29} Among its many possible causes is myocardial ischemia. Although coronary insufficiency induced by various "stress tests" is generally manifested by a depression of the RS-T segments, a reversal in the direction of the T wave at the apical lead may similarly constitute a "positive" or "abnormal" test. Furthermore, areas of T-wave negativity, limited, as in the present cases, to or about lead V\textsubscript{4}, have also been reported in apparently healthy males.\textsuperscript{18} It is obvious from the present study that such changes, recorded in clinically suspect circumstances, may represent a dynamic development rather than a static condition and therefore demand careful continued study for the subsequent evolution of diagnostic electrocardiographic evidence of myocardial infarction.

The "transitional zone" of the QRS complex is currently regarded as that region at one side of which one type of QRS complex is recorded and at the other side of which another type of QRS complex is recorded. Depending upon
whether this zone lies parallel to the line of the usual six precordial leads, or intercepts the line of these leads more or less perpendicularly, the "transitional zone" may appear to be broad and distributed over two or more precordial positions, or it may be abrupt and closely demarcated. Depending upon the spatial orientation of the heart, this zone may be projected to one or more of the unipolar limb leads. Although there are some objections to the validity of this entire concept, in general the "transitional zone" is regarded as the projection upon the body surface of the plane of the interventricular septum. Since this plane must continue through the entire body, it must intercept the body surface twice. There must, therefore, be two such "transitional zones," one in a general way about 180 degrees around the torso from the other. Grant has suggested that, just as there may be a "transitional zone" for the QRS complex, so may there be such a "transitional zone" for the T wave. If this is accepted, can one explain the areas of localized T-wave negativity described in the present series of cases as the result merely of the projection of two such transitional zones to an area within the confines of the usual six precordial leads, leads showing T-wave negativity being flanked by leads showing T-wave positivity? It seems difficult to imagine that the two extremities of this zone should lie so close to one another as the distance, say, from lead V5 to lead V4. This might assume that 180 degrees, more or less, of the circumference of the heart can be encompassed by moving the electrode 20 degrees or so on the precordium. This would seem to require a very small heart lying very close to the chest wall, or a very large chest wall closely opposed to a relatively normal sized heart. These postulates seem unlikely.

If, on the other hand, the plane of the "transitional zone" lies almost parallel with the line of the precordial leads, and since these six leads do not lie in a single straight line (leads V2 and V4 or V3 and V5 being staggered off that line), one can, as suggested by Grant, postulate that the middle two or three precordial leads lie on the negative side of the "transitional zone" for T waves, while the flanking leads lie on its positive side. This explanation is entirely reasonable. However, as observed in the present study, the attendant and subsequent clinical course and their association when recorded with other electrocardiographic evidence of myocardial ischemia (e.g., positive Master test or postextrasystolic T-wave changes) make it much more likely, it seems to us, that these changes are to be regarded, somehow, as manifestations of myocardial ischemia.

Some of the earlier studies in which only a single apical lead showed T-wave inversion cannot be considered adequate for the present study. On more thorough exploration of the right side of the precordium this change might have been found to be associated, at that time, with definitive evidence of acute anteroseptal infarction. In such circumstances the inverted T waves would have represented "ischemia" at the margins of the infarct. Lepeschkin has differentiated two types of pointed T-wave inversion in myocardial infarction, an early "ischemic" pattern associated with normal or shortened Q-T interval, and a later "post-ischemic" pattern associated with a prolonged Q-T interval. In the present study the Q-T interval was normal in cases 1, 4, 5, 6 and 7, prolonged in case 2 and shortened in case 3 at the phase of early T-wave inversion. When definitive electrocardiographic evidence of infarction was present, the Q-T interval was normal in cases 1, 4 and 5 and prolonged in cases 2, 3 and 6.

This discussion has been restricted to changes in certain of the precordial leads and their relation to subsequent anterior wall infarction. This is a reflection, we feel, of the anatomic fact that in the precordial leads alone is it possible in a general way to delineate the changes generated in a restricted area. This assumes the validity of the proposition that, although each of the leads in current use records the totality of the electrical changes produced in the heart as a whole, the individual leads, depending upon their proximity to the heart, are influenced to a much greater extent by changes in neighboring than in distant parts of the heart. Thus because of the relative remoteness of the left leg from the posteroinferior aspect of the heart, leads aVr, II and III record the
total and composite effect of changes in the back of the heart. Likewise, depending upon the position of the heart, lead aV_4 may reflect predominantly the changes produced in the entire lateral aspect of the heart. Were we able to record changes in selective portions of the posterior or lateral aspects of the heart, as we can on the anterior surface of the heart, it should be possible to demonstrate localized T-wave inversion in incipient myocardial infarction in the corresponding regions.

**Conclusions**

Inversion of the T waves, restricted to, or predominant in, circumscribed areas of the precordium, may precede, herald, or, at times, constitute an integral part of the electrocardiographic sequence of acute infarction of the anterior wall of the heart. These changes may remain fixed for long periods of time or show an instability in the direction toward or away from normality. In either case they may represent the clinical counterpart of an “ischemic” phase of myocardial infarction described in the experimental animal. The Q-T interval is generally normal when these changes are recorded but it may be shortened or lengthened. This experience emphasizes the need for protracted observation of patients showing these changes and justifies scepticism toward regarding them, especially under clinically suspect circumstances, as normal variations.

**Sumario Español**

La inversión de las ondas T, restringida a, o predominantemente en áreas circunscritas del precordio, puede preceder, anunciar, o, a veces, constituir una parte integral de la secuencia electrocardiográfica de infarto agudo de la pared anterior del corazón. Estos cambios pueden permanecer fijos por largos períodos de tiempo o mostrar una instabilidad en la dirección hacia lo normal o anormal. En uno u el otro caso puede representar la contraparte de la fase isquémica del infarto del miocardio descrito en el animal experimental. El intervalo Q-T es generalmente normal cuando estos cambios son registrados pero puede estar prolongado o acortado. Esta experiencia enfatiza la necesidad para una observación prolongada de pacientes que muestren estos cambios y justifica escéptico en considerar estos cambios, especialmente bajo circunstancias de sospecha clínica como variaciones normales.

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