

of these investigations. My assistant, Miss Ingrid Larsson, has helped me indefatigably in the work, and my gratitude is also extended to the Therese and Johan Andersson Memorial Fund for a grant which made this investigation possible.

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## CHANGES IN THE PRECORDIAL ELECTROCARDIOGRAM PRODUCED BY EXTENSION OF ANTEROSEPTAL MYOCARDIAL INFARCTION

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**T**HIS report is concerned with an electrocardiographic study of two patients, each of whom experienced two attacks of severe anginal pain within a short period of time. The observations made suggest that in each case the first attack was associated with the development of a small anteroseptal myocardial infarct, whereas the second attack was related to the lateral extension of the initial lesion. The electrocardiograms illustrate the value of multiple precordial leads in the diagnosis of this sequence.

## CASE REPORTS

CASE 1.—A 43-year-old housewife was admitted to the University Hospital on Aug. 6, 1943. On the morning of the day of admission to the hospital, about one hour after awakening, the patient noted mild precordial oppression which cleared spontaneously. It recurred about one hour later and became increasingly severe. Numbness in both shoulders, pain radiating to the left arm and hand, and a mild sense of suffocation developed. When examined by her physician shortly after the onset of these symptoms, the blood pressure was 150/120. Inhalation of amyl nitrite and two doses of morphine sulfate of 0.016 Gm. ( $\frac{1}{4}$  grain) each, subcutaneously, gave no immediate relief. She was admitted to the hospital a few hours later.

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There was no history of previous complaints referable to the heart. The patient had been examined in the outpatient clinics on several occasions. The blood pressure was recorded as 128/100 in January, 1939, 160/110 in November, 1941, and 150/90 in March, 1942. Her father died of angina pectoris. Her mother had an abnormally high blood pressure, but died of a perforated ulcer.

*Physical Examination.*—When first seen, the patient was somewhat pale and drowsy. She was not complaining of pain, possibly because the opiates which had been administered at her home had become effective. The temperature, pulse rate, and respiratory rate were normal. The heart sounds were of good quality and the cardiac rhythm was normal. The blood pressure was 118/80. The remainder of the physical examination was negative.

*Laboratory Data.*—The blood, urine, and blood serologic examinations were negative. The leucocyte count rose from 9,300 on admission to 11,850 on the fourth hospital day, and then returned to normal. The sedimentation rate on admission was 36 mm. per hour (Wintrobe method). Subsequent determinations were 0.8 mm. per minute on September 14, 0.96 mm. per minute on September 24, and 0.4 mm. per minute on February 4, 1944 (Ernstene and Rourke method).

*Clinical Course.*—The usual treatment for myocardial infarction was instituted. Except for occasional palpitation, the patient was quite comfortable during the first thirty-six hours. On the evening of August 7, precordial distress recurred, and, in the early morning hours of August 8, it became quite severe, with radiation to both arms. The pain persisted with variations in intensity for thirty-six hours. During this period, four doses of morphine sulfate of 0.016 Gm. ( $\frac{1}{4}$  grain) each, and then five doses of dilaudid of 0.002 Gm. ( $\frac{1}{50}$  grain) each, were administered. The patient grew pale, restless, and somewhat confused. She was kept in an oxygen tent for eight days. The temperature, which had been normal during the first two days, rose to 101.2° F. (R), and the pulse rate, to 116 per minute. The respiratory rate fell to 5 per minute. The blood pressure remained at about the same level as on admission. After the eighth hospital day there was progressive improvement, and convalescence was uneventful except for a mild upper respiratory infection. The patient was discharged on the fifty-second hospital day. When last seen, on Sept. 23, 1944, she was feeling well except for mild sciatica, and had been able to resume nearly full, normal activity.

*Electrocardiograms.*—The standard leads and unipolar extremity leads taken on August 6, six and one-half hours after the onset of symptoms, show slightly inverted T waves in Lead I and sharp terminal inversion of the T waves in Lead  $V_L$  (Fig. 1). Although somewhat suggestive, these records are not diagnostic of myocardial infarction because there are no significant changes in the QRS complexes. The precordial leads taken on August 7, twenty-seven hours after the onset of symptoms, show QS deflections in Lead  $V_2$  and sharp terminal inversion of the T waves in Leads  $V_1$ ,  $V_2$ ,  $V_6$ ,  $V_4$ , and  $V_5$  (Fig. 2). These changes are characteristic of recent anteroseptal myocardial infarction. Infarcts in this location are usually not accompanied by diagnostic alterations in the standard limb leads.<sup>1</sup>

The standard and unipolar extremity leads on August 9 (not reproduced), thirty-six hours after the recurrence of pain, are quite different from the first electrocardiograms. Prominent Q waves, small or absent R waves, and upward displacement of the RS-T segment are seen in Leads I and  $V_L$ , together with downward RS-T displacement in Lead III. The same changes are present in the tracings taken on August 11, except that displacement of the RS-T segment is more striking (Fig. 1). The precordial leads taken on this same date show large QS deflections in Leads  $V_1$ ,  $V_2$ ,  $V_6$ , and  $V_4$  and pronounced RS-T displacement in Leads  $V_3$ ,  $V_5$ ,  $V_4$ , and  $V_5$  (Fig. 2). Since the cavity potential (QS deflection) is now recorded not only in Lead  $V_2$  but from several other precordial regions, chiefly to the left of this point, it is evident that the original zone of infarction had extended laterally. The RS-T displacement also suggests that further acute injury had occurred.

Standard, unipolar extremity and precordial leads taken on Sept. 9 display the expected progression of changes in the ventricular complexes (Figs. 1 and 2). The displacement of the RS-T segment has disappeared, and the T waves are now sharply inverted in Leads I,  $V_L$ ,  $V_2$ ,  $V_3$ ,  $V_4$ , and  $V_5$ . Subsequent electrocardiograms, taken on Feb. 3 and July 7, 1944, are similar except that the changes in the T waves have regressed to some extent.

CASE 2.—A 53-year-old foreman was seen in the Heart Station on March 2, 1943. He was complaining of attacks of pressing, squeezing discomfort beginning in the right arm, extending to the upper right anterior part of the thorax and sternum, and occasionally to the right cervical region. The first attack occurred on Feb. 13, 1943, while he was sawing

wood. However, subsequent attacks were not closely related to exertion, and the pain awakened him frequently at night. The discomfort usually lasted five to thirty minutes. Nitroglycerin had been found to give relief. During the evening of March 1, he had five attacks of short duration.

In 1940, he had a transient left hemiparesis during an attack of "food poisoning." The blood pressure was found to be slightly elevated at that time. He had inflammatory rheumatism at the age of 3 years, and a gastroenterostomy for peptic ulcer at the age of 39 years. His father died of a "heart attack."

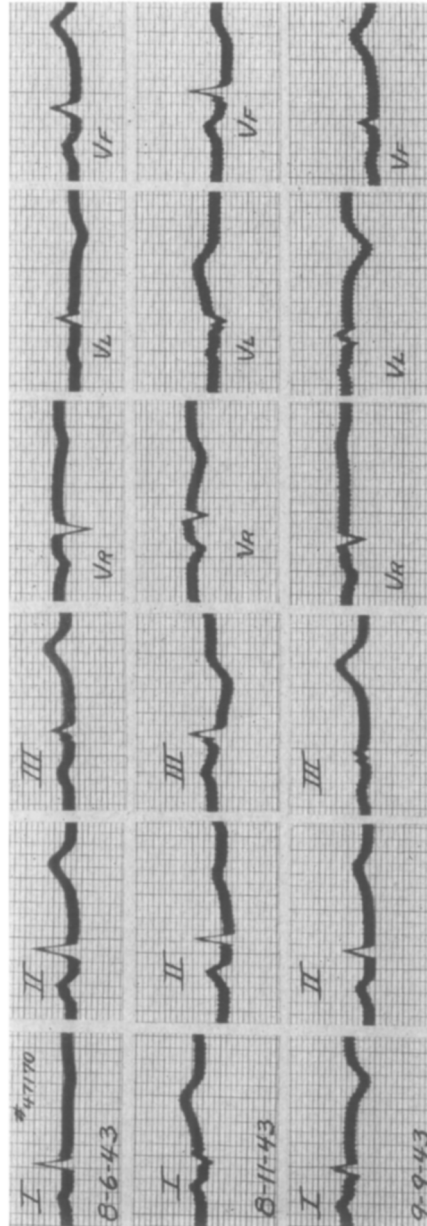


Fig. 1.—Case 1. Aug. 6, 1943: Standard leads and unipolar extremity leads taken six and one-half hours after onset of symptoms. Aug. 11, 1943: Standard and unipolar extremity leads four days after second attack. Sept. 9, 1943: Standard and unipolar extremity leads during convalescence.

*Physical Examination.*—The patient was a moderately obese, somewhat plethoric, middle-aged man. The heart was not enlarged. The aortic second sound was slightly accentuated. There were no murmurs or abnormalities of rate or rhythm. The blood pressure was 150/100. The remainder of the examination showed nothing of significance.

*Laboratory Data.*—The sedimentation rate was 9 mm. per hour (Wintrobe method). The miniature chest roentgenogram and the blood serologic reaction were negative.

*Electrocardiograms.*—The standard and unipolar extremity leads taken on March 2 show terminal inversion of the T waves in Leads I, II, and  $V_1$ , i.e., changes suggestive, but not diagnostic, of recent myocardial infarction (Fig. 3). The precordial leads taken at the same time display prominent QS deflections in Leads  $V_1$ ,  $V_2$ , and  $V_3$  and deep terminal inversion of the T waves from all the precordial points explored (Fig. 3). These changes are characteristic of infarction of the anteroseptal portion of the left ventricular wall.

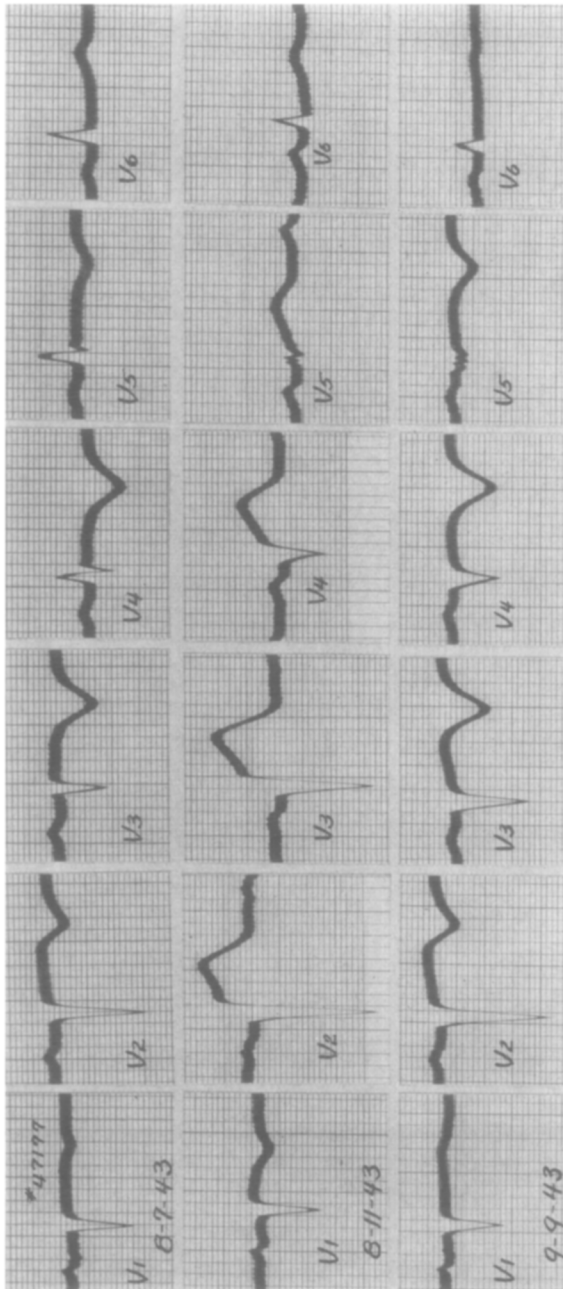


Fig. 2.—Case 1. Aug. 7, 1943: Precordial leads taken twenty-seven hours after onset of symptoms showing changes characteristic of recent anteroseptal myocardial infarction. Aug. 11, 1943: Precordial leads four days after second attack, showing evidence of lateral extension of the original lesion. Sept. 9, 1943: Precordial leads during convalescence, showing usual progression of changes.

Correlating these changes with the history, it seems probable that the infarction occurred during the preceding evening when the patient experienced the multiple, short attacks of anginal pain.

*Second Admission.*—After his first visit the patient was confined at home. On March 4, while in bed, he had an attack of pain in the right arm, thorax, and neck lasting eight hours. He took nitroglycerin tablets at intervals of five minutes (total of thirty tablets)

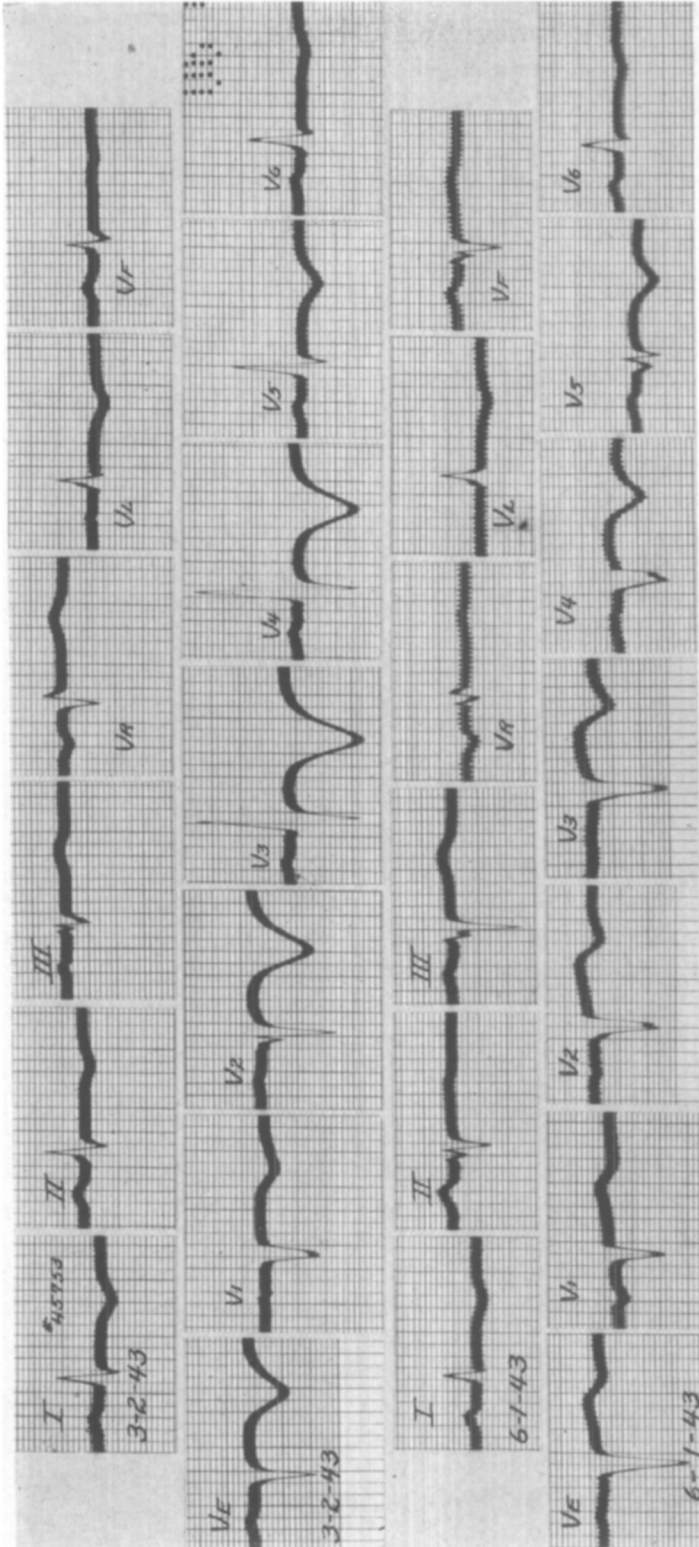


Fig. 3.—Case 2. March 2, 1943: Standard, unipolar extremity and precordial leads showing characteristic of recent anteroseptal myocardial infarction. Patient had five short, sharp attacks of anginal pain eighteen hours before, June 1, 1943; Standard unipolar extremity and precordial leads showing signs of the lateral infarct which probably occurred on March 4, 1943.

without obtaining relief. He remained in bed for five weeks and thereafter gradually resumed activity. He had no more prolonged attacks of pain, but mild anginal distress occurred on moderate exertion or excitement and was relieved by rest or nitroglycerin. He returned to the Heart Station for re-examination on June 1, 1943.

*Physical Examination.*—The findings were unchanged from those at the time of the first examination except that the heart sounds were rather faint. The blood pressure was 150/100.

*Electrocardiograms.*—Comparison of the standard and unipolar extremity leads taken June 1 with those recorded three months earlier discloses only minor changes. The R waves are smaller in Leads I, II, and  $V_1$ , the S waves are larger in Leads II, III, and  $V_2$ , and the T waves have become upright in Lead II (Fig. 3). The true significance of these changes becomes evident only when the precordial leads taken at the same time are examined (Fig. 3). QS deflections are now present in Leads  $V_1$ ,  $V_2$ ,  $V_3$ ,  $V_4$ , and  $V_E$ . The T waves have become upright in Leads  $V_1$  and  $V_E$ , but are again inverted in all the other precordial leads. A W-shaped QRS complex is now seen in Lead  $V_3$  in which the initial ventricular complex was previously of normal form. It is evident that extension of the original antero-septal infarct laterally to involve the anterior and anterolateral regions of the left ventricle was responsible for these changes. The history suggests that this extension occurred on March 4, when the patient had the prolonged attack of anginal pain.

#### DISCUSSION

These two patients are quite similar with respect to their history and the character and sequence of development of the electrocardiographic changes. In each case, the initial infarct was associated with relatively minor clinical manifestations and only T-wave changes in the standard and unipolar extremity leads (Figs. 1 and 3). The precordial leads, however, show characteristic QRS and T-wave changes due to antero-septal myocardial infarction in the leads from the extreme right side of the precordium (Case 1, Fig. 2, Lead  $V_2$ , and Case 2, Fig. 3, Leads  $V_1$ ,  $V_2$ , and  $V_E$ ). The full import of the initial clinical manifestations might have been overlooked if multiple precordial leads, and particularly leads from the right side of the precordium, had not been taken. This becomes still more apparent if Leads  $V_4$  and  $V_5$  of the records taken after the initial infarct are examined; these display only T-wave inversion, which, albeit pronounced, in the absence of QRS changes, does not permit an electrocardiographic diagnosis of myocardial infarction. Therefore, it can be seen that standard limb leads and single precordial leads from the region of the cardiac apex would not have been as helpful in these cases as tracings of the type reproduced here.

The even greater value of multiple precordial leads in these two cases is seen upon examination of the precordial electrocardiograms taken after the second attack of severe pain (Figs. 2 and 3). Since the changes in the QRS complexes are now recorded from a much larger area, it is evident that the initial zone of infarction has grown larger by lateral extension. Although one could have suspected from the clinical picture that an extension of the original infarct or a second infarct had occurred, the exact situation was not revealed until the multiple precordial leads were repeated. In Case 1, these records were made soon enough so that acute injury effects were also recorded (Fig. 2), thereby further substantiating the impression that additional muscle had been infarcted. This was not possible in Case 2 because the second set of records was not taken early enough to show such changes.

In Case 1, the standard and unipolar extremity leads also showed significant changes when the infarct extended, particularly in Leads I and  $V_L$ . This is usually the case in anterior and anterolateral infarction, for these leads ordinarily reflect the form of the electrocardiogram in Leads  $V_5$  and  $V_6$ . How-

ever, in Case 2, Leads I and  $V_L$  do not display changes of similar degree, although they do resemble Lead  $V_6$ . This difference is probably due to a slight difference in the position of the heart in the two cases. That this is true is supported further by the fact that, after the extension of the infarct in Case 2, the QRS complex in the unipolar lead from the left leg ( $V_F$ ) showed greater change than that from the left arm ( $V_L$ ).

Several recent reports have pointed out that "premonitory" or "prodromal" symptoms may precede myocardial infarction.<sup>2-10</sup> In both of the cases presented here, the symptoms accompanying the initial infarct were of the type which have been reported as frequently indicating "impending" infarction. Electrocardiograms taken on such patients during the interval between the onset of the prodromal symptoms and the occurrence of the myocardial infarct have usually been of normal form. However, in some of the records which have been published, there is inversion of the T waves in Lead I, in a single precordial lead, or in both, similar to that observed in the two cases reported here. On the other hand, in some cases of anteroseptal infarction (as shown by leads from the right side of the precordium,  $V_1$ ,  $V_2$ , and  $V_3$ ), the standard leads and precordial leads from the region of the cardiac apex ( $V_4$ ,  $V_5$ , and  $V_6$ ) are well within normal limits.<sup>11</sup> Therefore, if multiple precordial leads had not been taken, thereby revealing that anteroseptal infarction had already occurred, the two patients discussed here might also have been considered to have experienced only premonitory symptoms. The extension of the original lesion which occurred a few days later would then have been erroneously considered the initial infarction. The opinion that prodromal pain may actually be an expression of myocardial infarction in some cases has also been set forth by Dressler,<sup>12</sup> as well as by earlier observers.<sup>3, 8, 10</sup>

Bayley<sup>13-15</sup> has shown that the T-wave changes in patients with symptoms of impending infarction are the result of myocardial ischemia. He has presented important new evidence regarding the nature and manner of development of such changes. The alterations in the T waves which he has observed both clinically and in experimental animals are very similar to those recorded in the two cases presented here. However, the presence of significant QRS changes in the leads from the right side of the precordium in the records following the initial infarct indicate that, in addition to ischemia, actual infarction was present.

#### SUMMARY

Electrocardiographic studies are reported on two patients, each of whom had anteroseptal infarction, followed in a few days by lateral extension of the initial lesion. The worth of multiple precordial leads in the diagnosis of extension of such infarcts is illustrated.

Evidence is again presented that infarcts which are anteroseptal in location, as shown by diagnostic changes in leads from the right precordial area, often fail to produce equally significant changes in the limb leads.

It is suggested that, in cases of coronary arterial disease, some of the attacks of pain which have usually been considered prodromal symptoms of myocardial infarction, actually represent the development of a small, anteroseptal infarct, and that the more characteristic symptoms of acute coronary thrombosis which often occur later are due to an extension of this initial lesion. The true situation must be recognized, if such patients are to be properly treated.

We do not wish to convey the impression that we are convinced that all attacks of so-called prodromal pain represent actual myocardial infarction. The data, at present available, bearing on this problem are inadequate to justify this conclusion. Some attacks of this character appear to be due to acute processes developing in the coronary arteries or to transient myocardial ischemia associated with such processes.<sup>7</sup>

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