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THE PRECORDIAL ELECTROCARDIOGRAM IN HIGH LATERAL MYOCARDIAL INFARCTION

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THE observations upon which this report is based were made on a group of six patients whose routine standard and unipolar extremity electrocardiograms showed changes suggestive of myocardial infarction. The usual precordial leads presented no more and often much less evidence pointing to this diagnosis. On the other hand, extensive exploration of the left anterolateral, lateral, and posterolateral aspects of the thorax at levels higher than those usually studied yielded more significant electrocardiographic data.

Four of the six patients gave a definite history of a coronary accident a few days to one year prior to the time of our observations; one of them has recently developed a posterior lesion one year after an earlier high posterolateral lesion. In two cases the history was merely suggestive of infarction; one patient had angina pectoris, intermittent claudication, and an old posterior infarct, and the other had moderate congestive failure alone.

In four patients the changes most characteristic of myocardial infarction were recorded in the vertical line of Lead V_3, Lead V_4, or Lead V_5 but one to three intercostal spaces above the level from which these leads are taken. In these cases a diagnosis of high anterolateral infarction was made. In one patient, the most striking changes occurred in leads from the anterior and midaxillary lines at levels two or three intercostal spaces higher than the usual Lead V_5 or V_6; in this instance, the diagnosis was high lateral infarction. In one case the most definite changes were seen in records from points high in the left posterior axillary and the left scapular lines and were attributed to a high posterolateral

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infarct. The observations made were not identical in all patients since only as the study progressed were the most advantageous points for exploration revealed.*

CLINICAL OBSERVATIONS

High Anterolateral Infarction.—The first four cases to be discussed are considered examples of high anterolateral infarction.

CASE 1.—H. K., a 41-year-old bus driver entered the University Hospital on June 23, 1944, for treatment of a left hemiplegia which had appeared suddenly one year earlier. For two weeks prior to the occurrence of the paralysis he had experienced frequent attacks of pain in the chest while driving his bus. The attacks were severe enough to make him stop to rest and he was finally forced to stop working because of them. In May, 1943, while sitting in a restaurant, he suddenly developed a left hemiplegia. He was under treatment in a hospital for one month; his blood pressure was said to have been high during that period. He was able to walk with difficulty by August, 1943, but there had been little change in his condition for ten months. There was a strong familial history of cardiovascular disorders.

Examination showed a left spastic hemiplegia with a left facial paresis of central type. The retinal vessels exhibited minimal arteriosclerosis. The heart sounds were normal. The blood pressure was 120/80. There were occasional extrasystoles. The usual laboratory tests, including the blood Kahn reaction, were negative. Roentgenographic examination of the thorax showed slight cardiac enlargement and slight pulmonary congestion.

Electrocardiographic studies were made on July 28, 29, and 30. Only those made on the last day, when supplementary precordial leads were taken, are reproduced (Fig. 1). The standard and unipolar limb leads exhibit slight left axis deviation with small Q waves and slight terminal inversion of the T waves in Leads I and V_L. The usual precordial leads show tiny Q waves in Leads V_2, V_3, and V_4 and terminal inversion of the T waves in Leads V_3, V_4, and V_5. In the case of normal subjects there is a rapid increase in the size of the R waves as the exploring electrode is moved toward the left side of the precordium. In this case, therefore, the R waves are unexpectedly small in Leads V_3, V_4, and V_5. It should also be noted that R is taller in Lead V_6 than it is in Lead V_5; the opposite is normal.[1]

The tracings from points in the left midclavicular line (vertical line of Lead V_4) at higher levels (third and fourth intercostal spaces) show both QRS and T-wave changes which are strongly suggestive, if not diagnostic, of myocardial infarction. Similar but less striking alterations occur in the leads from points at the same horizontal levels but in the left anterior axillary line (line of Lead V_5). Only very slight inversion of the T waves is seen in the record from a point in the midaxillary line (line of Lead V_5) and at the level of the third intercostal space.

CASE 2.—C. K., a 40-year-old engineer entered the Heart Station of the University Hospital on March 3, 1944. Two months previously he developed burning substernal pain which

*After the first few cases had been studied, we adopted the plan of taking supplementary unipolar leads from points at the intersections of lines on the horizontal level of the fourth, third, or second intercostal space at the left sternal margin and the vertical lines of Lead V_3, Lead V_4 (left midclavicular line), Lead V_5 (left anterior axillary line), Lead V_6 (left midaxillary line), Lead V_7 (left posterior axillary line), etc., as indicated.

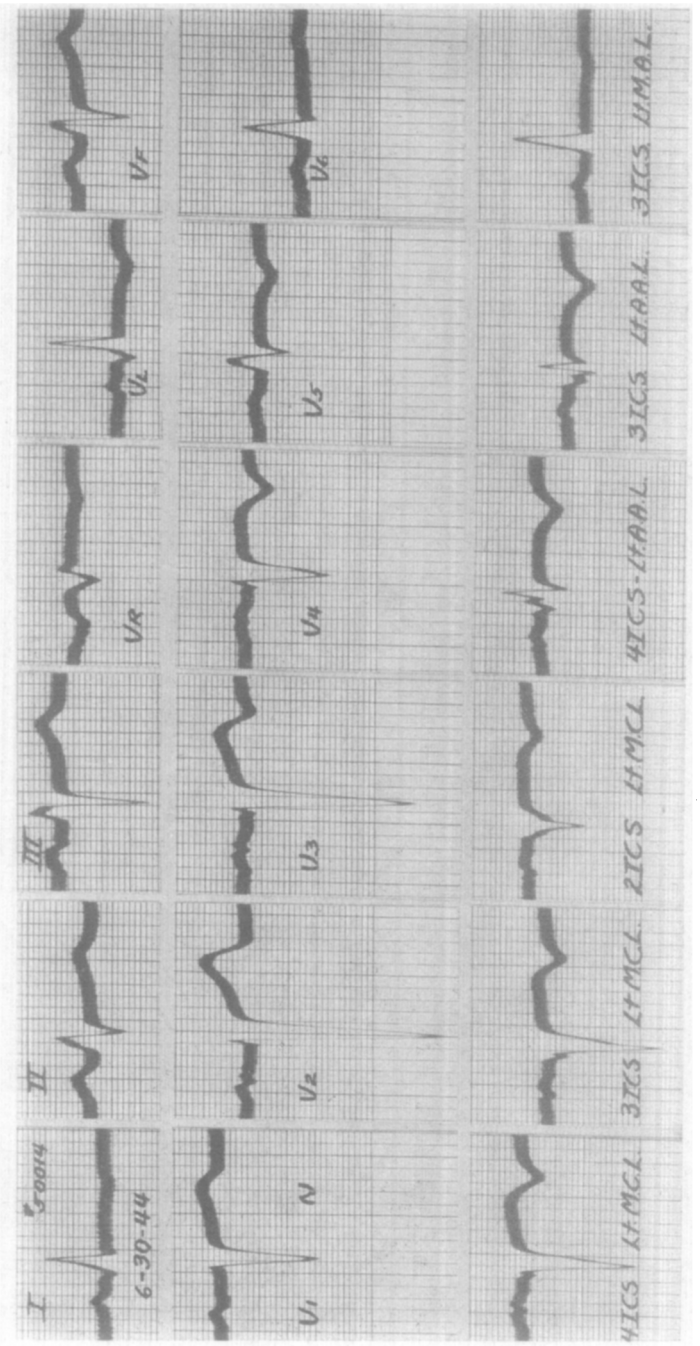

Fig. 1.—Case 1. *High anterolateral infarction.* Leads I and V_L, particularly the latter, display prominent Q waves and terminal inversion of the T deflections. The R deflection is conspicuously small in Lead V_5, and there is terminal inversion of T in Leads V_3, V_4, and V_5. The leads from the midclavicular line at the levels of the fourth, third, and second intercostal spaces exhibit large QS deflections and terminal inversion of T. Note also the prominent Q deflection and sharply inverted T wave in the lead from left anterior axillary line at the level of the fourth interspace. Symptoms strongly suggestive of infarction occurred in May, 1943.

radiated to the left arm and lasted twenty hours. He had been in a hospital for three weeks and was gradually resuming activity. There was no history of cardiac symptoms before the coronary accident.

There was no cardiac enlargement. The cardiac sounds were rather loud and the heart seemed overactive. There was a faint systolic murmur at the base. The blood pressure was 170/100.

The electrocardiograms made during the patient's acute illness were available and are reproduced along with the observations made at the time of our examination in Fig. 2. The tracing taken on the day of the attack (Jan. 5, 1944) displays very slight upward RS-T displacement in Lead I and slight downward RS-T displacement in Lead III, but is not certainly abnormal. On Jan. 11, 1944, the changes in the RS-T segment had become somewhat more distinct and a tiny Q wave had appeared in Lead I. The standard electrocardiogram taken eleven days later shows, in addition, definite terminal inversion of the T waves in Lead I, and upright T waves in Lead III. A single precordial electrocardiogram (IV) was made from a point said to be in the vertical line of V_3 but two intercostal spaces higher than the usual level; it displays QRS and T-wave changes characteristic of recent myocardial infarction. The records taken at the time of our examination on March 3, 1944, display only small Q waves in Leads I and V_L; the usual precordial leads, V_1 to V_6, are well within normal limits. The records made from a point high in the anterior axillary line and from a point in the line of Lead V_3 but at the level of the second intercostal space at the left sternal margin show prominent Q waves and normal T waves. These changes are regarded as residual electrocardiographic evidence of the infarction which had occurred two months earlier; apparently, the alterations of the T wave in this case were quite transient. It is notable that only the chest leads taken at higher levels and Lead V_L display signs which can be considered significant. The record from a point high in the midaxillary line does not show changes of similar degree.

CASE 3.—T. S., a 37-year-old chiropodist entered the University Hospital on Jan. 18, 1945, complaining of attacks of dyspnea and hemoptysis. Albuminuria and fluctuating hypertension had been discovered five years earlier, and one year prior to admission he began to have paroxysmal nocturnal dyspnea. He had a typical myocardial infarction in July, 1944, after which the attacks of paroxysmal left ventricular failure grew more severe and were precipitated by excitement and emotional stress.

The patient was a small, hyperkinetic man. Marked hypertensive retinopathy was present. The heart was tremendously enlarged. A moderately loud systolic murmur and a diastolic gallop sound were heard at the apex and along the sternum. There were frequent extrasystoles. The blood pressure was 190/130 in the right arm and 130/100 in the left arm. A difference of this order was consistently present and was not altered by the position of the arms. During periods of stress the blood pressure rose as high as 290/210. Conspicuous peripheral arterial thickening was present.

Slight albuminuria and moderate reduction of urea clearance were found. A histamine test failed to give a response suggestive of a pheochromocytoma.[2] Other laboratory studies of the blood chemistry and pyelograms were negative. Roentgenographic examinations of the thorax disclosed great cardiac enlargement and moderate pulmonary congestion.

A bilateral splanchnicectomy was performed on Feb. 12, 1945. The operation and convalescence were uneventful. The patient was re-examined in the Heart Station on April 12, 1945. He was then generally improved, and the attacks of left ventricular failure were fewer

and less severe. The physical findings were not significantly different from those elicited prior to the operation. The blood pressure was 185/135. His referring physician recently informed us that he died on Aug. 8, 1945, from a cerebral hemorrhage.

Electrocardiograms made at the time of the acute infarction were available for review. On July 27, 1944, the standard leads displayed prominent Q waves,

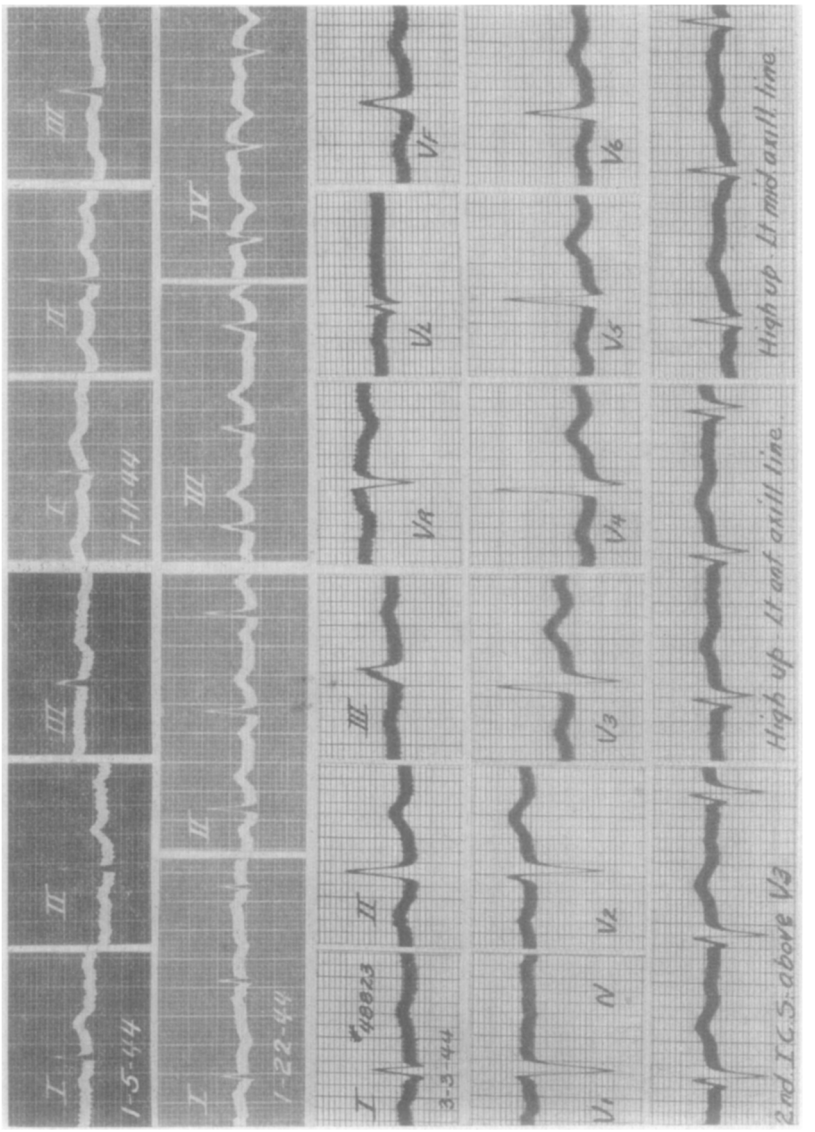

Fig. 2.—Case 2. *High anterolateral infarction.* Symptoms typical of infarction developed on Jan. 5, 1944. The record labelled "IV," which was taken on Jan. 22, 1944, was reported to have been taken by leading from a point two intercostal spaces above that specified for standard Lead IV; it displays changes diagnostic of recent myocardial infarction. The tracings of March 3, 1944, show prominent Q waves in Lead I, Lead V_6, and the leads from the upper levels of the left thorax. The ventricular complexes of the standard precordial leads (V_1 to V_6 inclusive) are well within normal limits.

slight elevation of the RS-T segment, and terminal inversion of the T waves in Leads I and II with marked depression of the RS-T segment in Lead III. The precordial leads showed unusual large QRS deflections with R waves which were definitely smaller in Lead V_3 than in Lead V_2 or V_4, tiny Q waves in Leads V_4, V_5, and V_6, and normal T deflections except for diphasic T waves in Lead V_6.

Except for the relatively small R waves in Lead V_3, the precordial leads did not suggest fresh myocardial infarction, although the standard leads were compatible with that diagnosis. The electrocardiograms, taken on Sept. 11, 1944, displayed the usual progression of changes in the standard leads. The precordial leads on

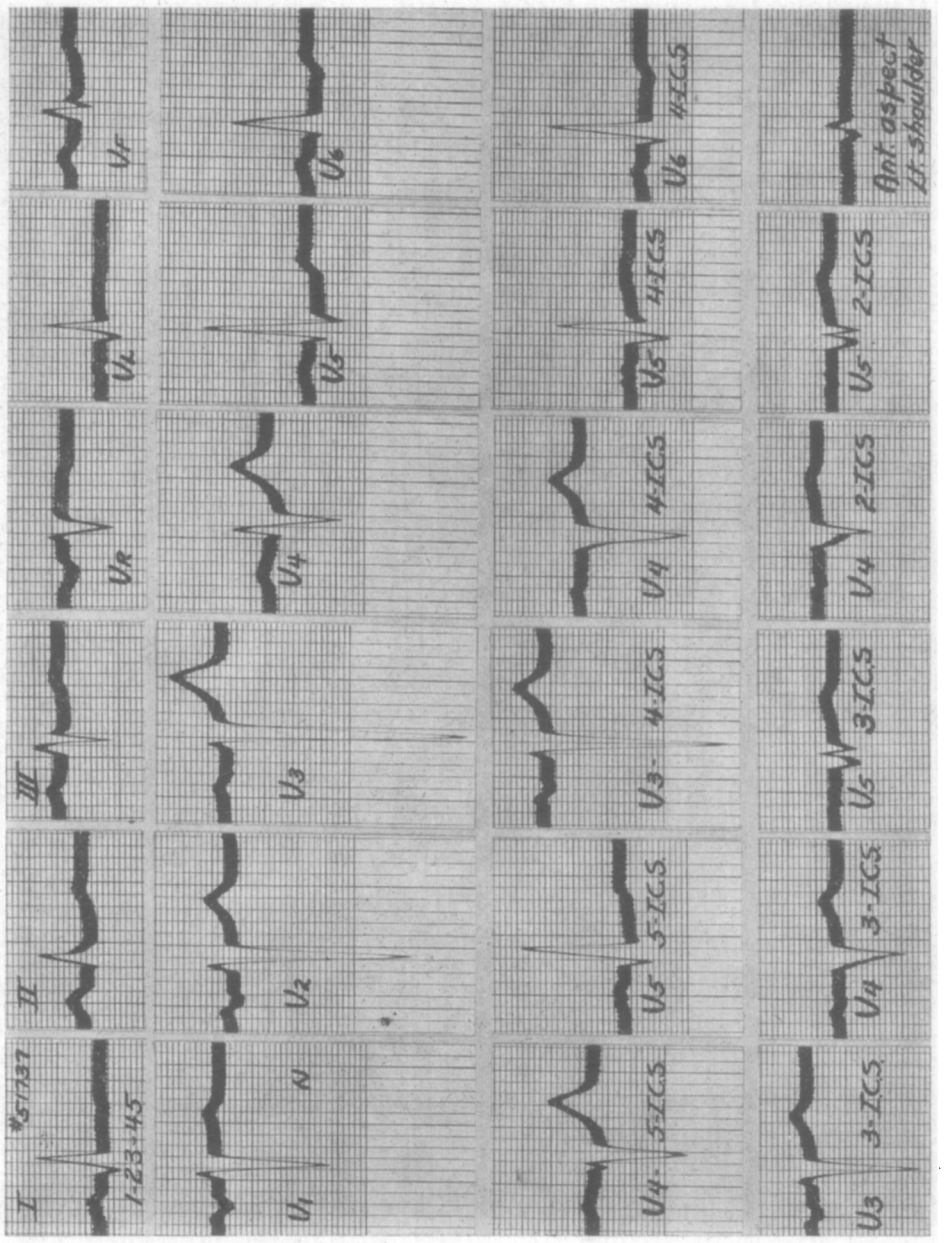

Fig. 3.—Case 3. *High anterolateral infarction.* The standard limb leads show prominent Q waves in Leads I and V_L. The standard precordial leads are distinctly abnormal only as regards the small size of R in Lead V_3. A number of the leads from the upper left chest display large Q or QS deflections which are strongly suggestive of infarction. The coronary accident occurred in July, 1944.

this occasion again showed a tiny R wave in Lead V_3 but, in addition, large QS deflections in Lead V_4 and deep, sharp, terminal inversion of the T waves in Leads V_5 and V_6. The differences between the two sets of precordial leads may

have been due to differences in the locations of the precordial points selected on the two occasions or, what seems less likely, to changes in the size or character of the myocardial lesion.

Our own electrocardiograms were taken on Jan. 20, 22, and 23, 1945. All of the records are much alike and only the last set of tracings is reproduced (Fig. 3). Leads I and V_L show small Q waves and very slight terminal inversion of the T waves. There is slight depression of the RS-T segment in Leads II, III, and V_F, but this may be the result of digitalis therapy. The usual precordial leads display smaller R waves in Lead V_3 than in Lead V_2 or V_4, tiny Q waves in Leads V_4, V_5, and V_6 and slightly inverted T waves in Leads V_5 and V_6. Curves of this type may occur in left ventricular hypertrophy, but when they do the R wave usually grows progressively larger as the exploring electrode is moved to the left. Diminution of its size such as is seen here in Lead V_3 is rare in the absence of anterior infarction.[1] The implications of this finding become apparent when one examines the records taken at higher levels in the line of Leads V_4 and V_5, for in these tracings there are large QS deflections very suggestive of infarction. As in Case 2, the inversion of the T waves previously present had cleared before the extensive electrocardiographic observations were made.

CASE 4.—W. T., a 55-year-old engineer was first seen in the Heart Station of the University Hospital on July 20, 1943. He complained of pain in the chest and calves. Five years before this, he had a quite typical myocardial infarction, and one year later he had similar but less severe symptoms. After the second attack he developed mild angina pectoris and intermittent claudication.

The patient was a short, stocky florid man. The heart was not enlarged. The cardiac sounds were rather distant. The blood pressure was 124/80. A few râles were heard at both lung bases posteriorly. No pulsations could be felt in the left posterior tibial or in either of the dorsalis pedis arteries.

The standard and unipolar limb leads (Fig. 4) display small Q waves in Leads II and V_F and prominent Q waves and inverted T waves in Lead III. The precordial leads show only flat or slightly inverted T waves in Leads V_5 and V_6. In view of the previous clinical history, these changes may represent an old posterior myocardial infarct, but they are not of themselves diagnostic of this condition.

The patient returned to the Heart Station on Aug. 24, 1945. He had continued to have mild angina pectoris and intermittent claudication. Four months earlier, while walking in his factory, he had a sudden attack of severe dizziness and had to be assisted to his office. He noted diplopia for about one hour and a giddy sensation for several days. This latter complaint had persisted in mild degree up to the time of admission.

The findings on examination were much the same as on his initial visit. The blood pressure was 110/80. There was no evidence of postural hypotension. The hemoglobin, blood Kahn reaction, and miniature chest roentgenogram were normal.

The standard and unipolar extremity electrocardiograms (Fig. 4) are distinctly different from those taken two years earlier. There are small Q waves and flat T waves in Lead I, and the Q waves previously present in Leads II, III, and V_F have disappeared. Lead V_L exhibits prominent Q waves and sharp terminal inversion of the T waves. The usual precordial leads differ from those previously recorded in that the R waves failed to increase rapidly in size as the exploring electrode was moved to the left and the T waves are smaller. The

RS-T segment in Lead V_4 has a peculiar flattened outline and is somewhat depressed. Since these precordial records did not exhibit diagnostic evidence of infarction, leads from points at higher levels were employed. The records from

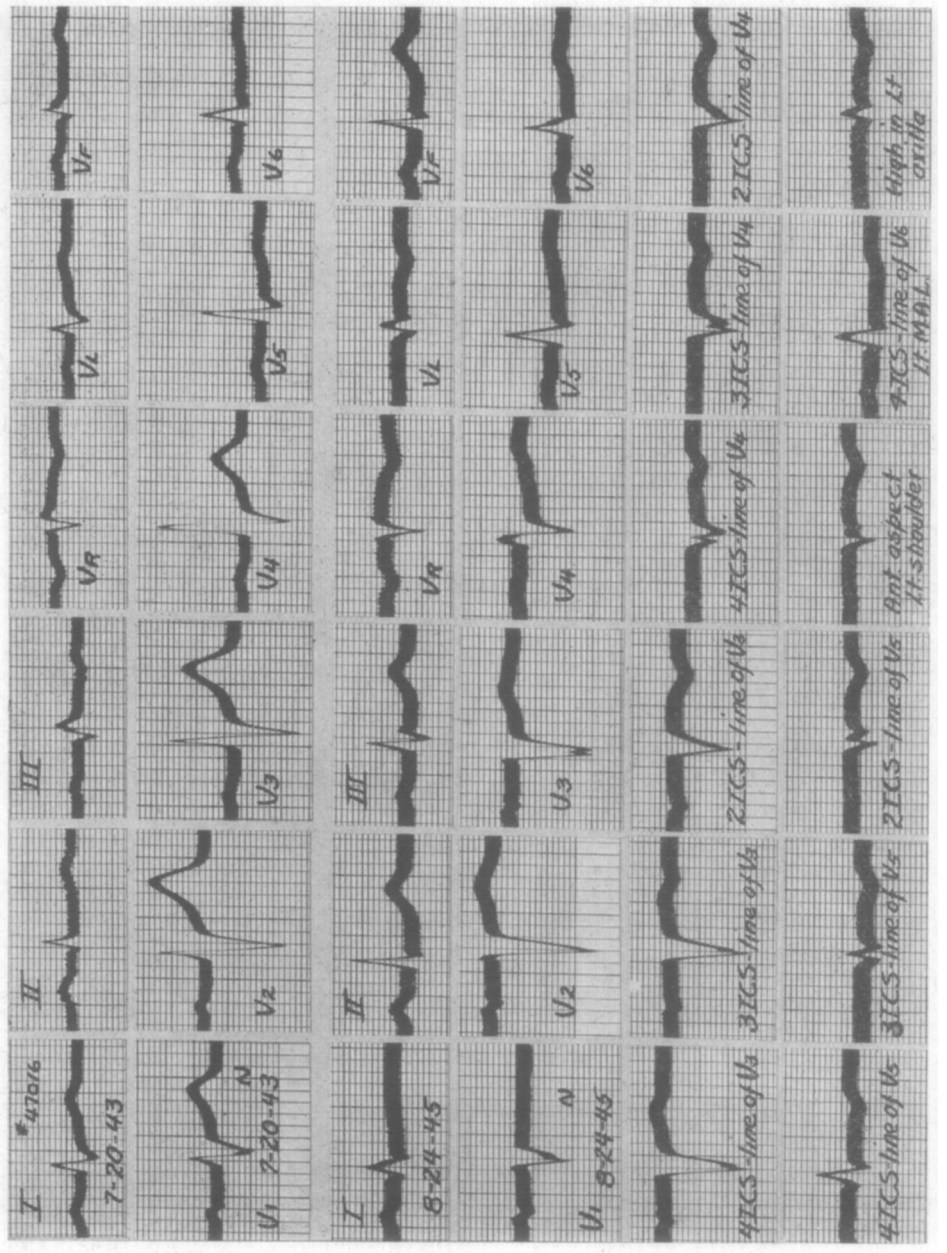

Fig. 4.—Case 4. *Old posterior infarction plus high anterolateral infarction.* The electrocardiograms taken on July 20, 1943, are strongly suggestive of old posterior infarction. There are prominent Q waves in Leads II, III, and V_F and relatively large R and T deflections in the leads from the right side of the precordium. The electrocardiograms of Aug. 24, 1945, are very different; Lead V_L shows prominent Q waves and terminal inversion of the T wave; many of the leads from the higher levels of the left thorax exhibit large Q or QS waves and terminal inversion of the T deflection. The standard precordial leads are negative apart from low-voltage T deflections and the small size of R in Leads V_2 and V_3. Symptoms suggestive of infarction occurred in 1938 and again in 1939.

points in the line of Lead V_4, but at the levels of the fourth, third, and second intercostal spaces at the sternum, and in the line of V_5 at similar levels, show changes in the QRS and T complexes which are in all respects characteristic of

myocardial infarction. Curves made from the midaxillary line at these higher levels do not display changes of like magnitude.

The date of the infarction responsible for the electrocardiographic changes recorded in 1945 is not clear. It may have occurred at the time of the severe

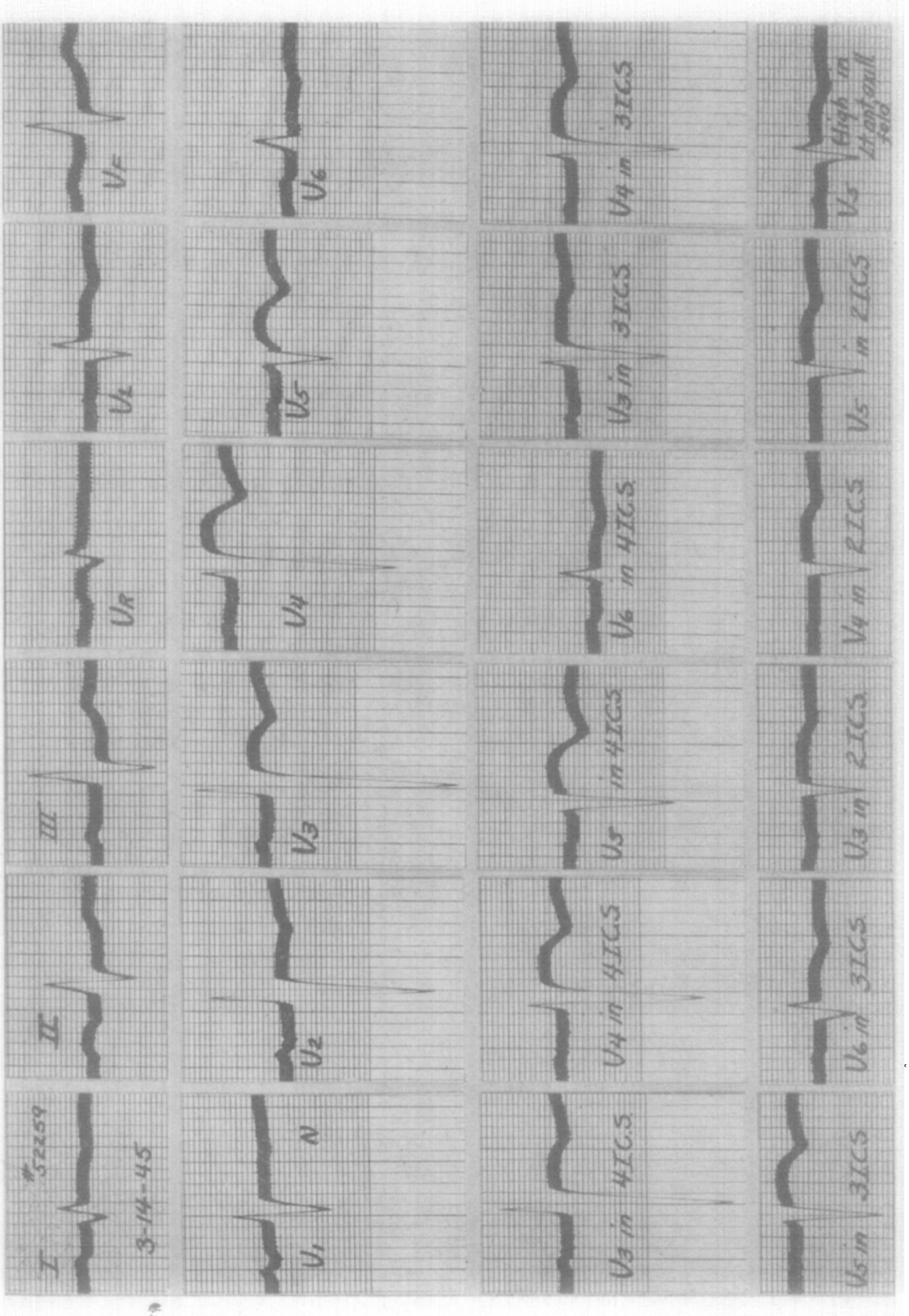

Fig. 5.—Case 5. *High lateral infarction.* The limb leads show large Q waves and terminal inversion of the T wave in Lead V_L. The standard precordial tracings show abnormally small R deflections in Leads V_1 and V_2; there are changes in the RS-T segment and the T wave strongly suggestive of infarction in Leads V_3, V_4, and V_5. In a number of the leads from the higher levels of the left lateral thorax, similar changes in the T complex are combined with large Q or QS deflections.

attack of dizziness, or the patient may have regarded the symptoms associated with it as merely one of his many attacks of angina pectoris.

High Lateral Infarction.—One case is considered an example of high lateral infarction because the most striking electrocardiographic changes occurred in unipolar leads from the upper lateral aspects of the left thorax and left axilla.

CASE 5.—J. L., a 67-year-old tailor entered the University Hospital on March 4, 1945, complaining of dyspnea and visual difficulty. He had noted exertional dyspnea and intermittent ankle edema for many years. For one year there had been paroxysmal nocturnal dyspnea. Ten months before he was first seen, he had been in a hospital for ten days because of these complaints. There was no history suggesting an acute myocardial infarction. Cataracts had caused progressive reduction in vision.

The patient was moderately dyspneic and appeared chronically ill. Minimal pulmonary congestion and emphysema were noted. The heart sounds were normal; no murmurs were heard. The blood pressure was 140/84. There was peripheral arteriosclerosis and minimal pitting edema of the ankles. The urine, blood, stool, and blood Kahn examinations were negative. Roentgenographic examination of the thorax showed marked cardiac enlargement and slight pulmonary congestion.

The patient responded well to treatment for congestive cardiac failure. He was discharged on March 17, 1945. He returned for a cataract extraction on May 14, 1945. When last seen on Aug. 20, 1945, his condition was unchanged.

The standard limb and precordial leads taken on March 6 and March 13, 1945, are similar to those taken on March 14, 1945, which are reproduced in Fig. 5. In Lead I there are a tiny R wave preceding a deep S deflection, slight upward RS-T displacement, and slight terminal inversion of the T waves. Lead V_L is similar except for the absence of the small initial R. Leads II, III, and V_F show slight RS-T depression, possibly due to digitalis which the patient was receiving. The standard precordial curves are distinctly abnormal since the R wave grows progressively smaller in successive leads, becoming smallest in Lead V_5. There is pronounced upward displacement of the RS-T segment in Leads V_3, V_4, and V_5 and terminal inversion of the T waves in Leads V_2 and V_6 in addition. Inasmuch as these changes were strongly suggestive of myocardial infarction, but did not include the presence of prominent Q or QS deflections in the leads from the usual precordial sites, additional tracings from points at higher levels were taken. The characteristic changes sought were recorded from regions high up in the line of Lead V_5 (see V_5—2 I.C.S.) and in the vertical line of Lead V_6 (see V_6—3 I.C.S.).

High Posterolateral Infarction.—One case has been classified as an example of high posterolateral infarction because the most characteristic electrocardiographic phenomena appeared in records taken from points high in the left posterior axillary line and over the left scapula. This case was unusually interesting because of the length of the interval which elapsed between the onset of symptoms and the appearance of the electrocardiographic changes. The patient recently developed a typical posterior myocardial infarct.

CASE 6.—J. A., a 46-year-old moulder, was admitted to the University Hospital on Sept. 16, 1944, complaining of severe retrosternal pain. Three years previously he began to have incapacitating intermittent claudication. On Sept. 10, 1944, he had attacks of severe, crush-

ing, retrosternal pain which radiated to the left arm and hand. These attacks were severe and prolonged and were only partially relieved by opiates. Five days before admission, the blood pressure was said to be 210/110.

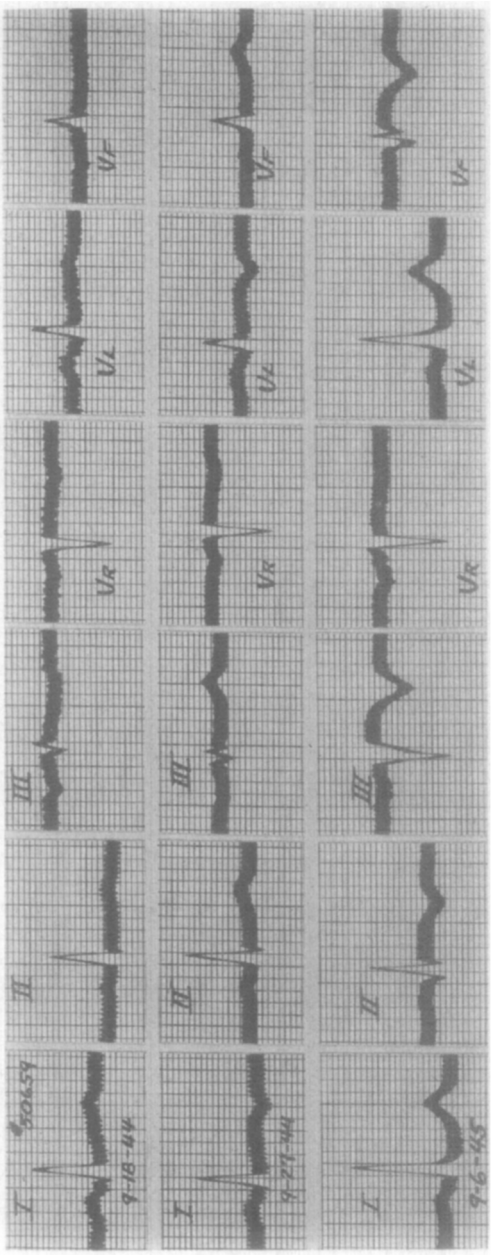

Fig. 6.—Case 6. *High posterolateral infarction followed by posterior infarction.* Standard unipolar limb leads. The tracings of Sept. 18 and Sept. 27, 1944, show delayed terminal inversion of the T deflection in Leads I and V_L. The tracing of Sept. 6, 1945, is characteristic of recent posterior infarction. The first myocardial infarction occurred on Sept. 16, 1944; the second occurred on Sept. 4, 1945.

The patient was a heavy, muscular man and was in acute painful distress. The heart was not enlarged and the cardiac sounds were normal. The blood pressure was 150/90. The left leg was somewhat cool, and the pulse in the left dorsalis pedis artery was absent. The routine urine, stool, blood Kahn, chest roentgenographic, and blood examinations were negative except for slight leucocytosis during the first week in the hospital.

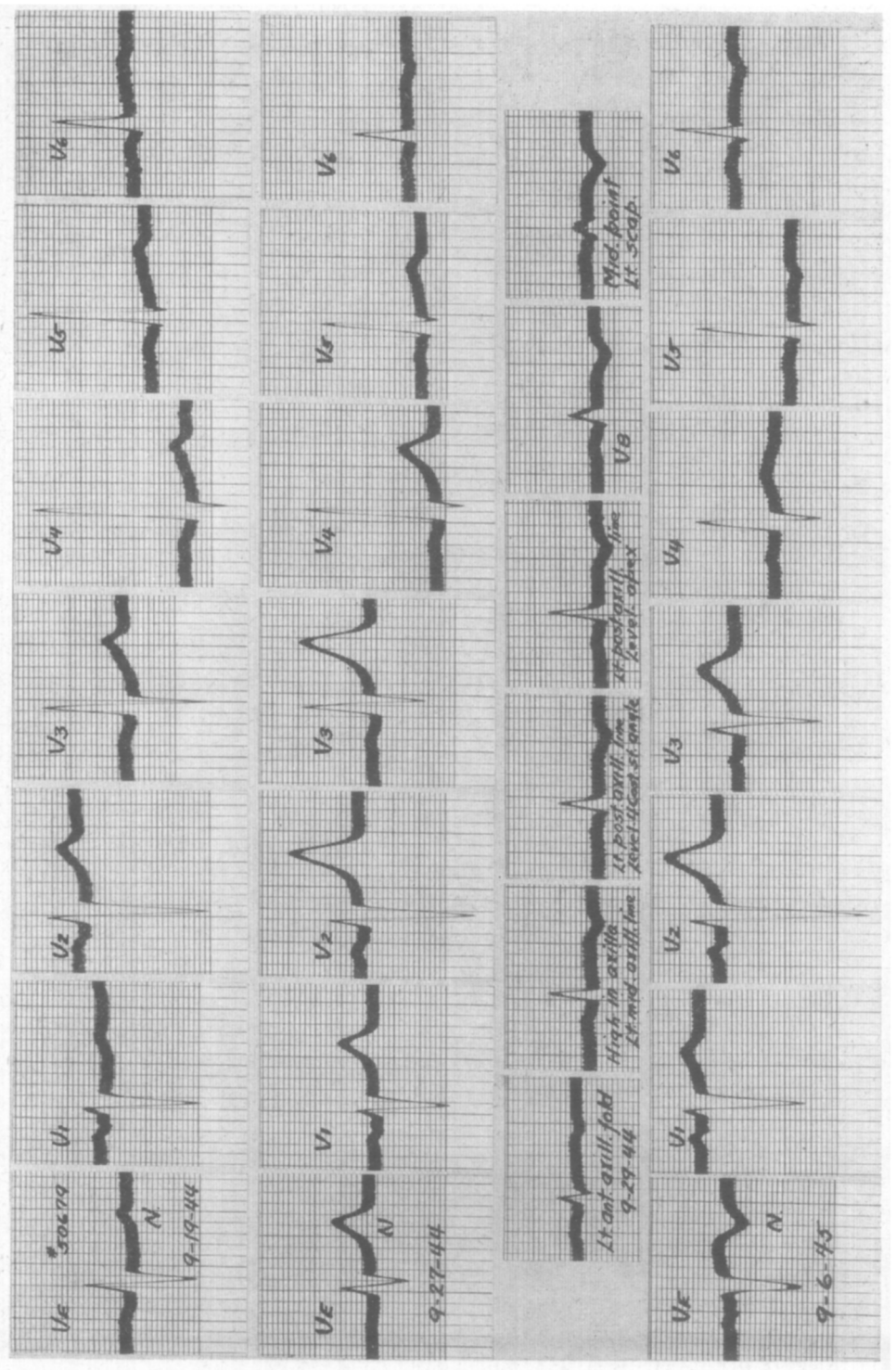

Fig. 7.—Case 6. *High posterolateral infarction followed by posterior infarction.* Precordial and other thoracic leads. The precordial leads taken on Sept. 27, 1944, illustrate the development of large pointed T waves in the leads from the right side of the precordium and of terminal inversion of the T wave in Lead V_6. More striking terminal inversion of the T deflection is exhibited by the leads from the higher levels of the left thorax. The tracings of Sept. 6, 1945, show sharp terminal inversion of T in Lead V_8 and less pronounced changes in the T complex in Leads V_5 and V_6.

The usual treatment for myocardial infarction was instituted. Two lesser attacks of pain occurred during the patient's stay in the hospital. There was some fever, maximum rectal temperature 103.2° F., during the first five days. The patient was discharged on the twenty-third hospital day.

A few of the records which display the progression of changes in this case are shown in Fig. 6. The standard and unipolar limb leads taken on Sept. 16, 1944, are not abnormal. The same leads taken on September 18 exhibit deflections of similar outline except for slight changes in the RS-T segment in Lead I and some flattening of the T wave in Lead II. The precordial leads of September 19 (Fig. 7) show a small depression of the RS-T segment in Leads V_1 and V_2 and very slight elevation of this segment in Lead V_6, but these curves are not certainly abnormal. The standard and unipolar extremity leads of September 25 and September 27 are much alike and both display terminal inversion of the T waves in Leads I and V_L. The T waves in Leads II, III, and V_F have become very large, tall, and upright. Furthermore, the usual precordial tracings of September 27 are very different from those taken eight days earlier, for the T waves are much taller in the leads from the right side of the precordium and there is slight terminal inversion of the T wave in Lead V_6. These changes represent the first unequivocal electrocardiographic evidence of the myocardial infarction which, judging from the clinical data, occurred at least ten days before they appeared. Additional records from points in the axilla, high in the mid-axillary line (Lt. mid. axill. line), in the posterior axillary line (Lt. post. axill. line Level—4 Cost. st. angle), and over the left scapula (Mid. point Lt. scap.) exhibit more characteristic changes in the T complex but no significant alterations of the QRS deflections (Fig. 7).

The patient was readmitted to the University Hospital on Sept. 4, 1945, complaining of severe pain in the chest. He had been comfortable except for mild angina pectoris until three days before when he had anginal pain lasting ten minutes. On the day of admission he had a very severe attack which persisted several hours despite repeated hypodermic injections.

The patient was slightly dyspneic. Fine râles were heard at the bases of both lungs. The heart was slightly enlarged and some precordial tenderness was noted. The blood pressure was 132/70. The blood, urine, stool examinations, and the circulation time were normal.

The patient was again treated for myocardial infarction in the usual manner. There was some recurrence of pain during the third week, but the hospital course was not otherwise remarkable. He was discharged on the twenty-sixth day.

The standard and unipolar limb leads of Sept. 5, 1945, are characteristic of recent posterior myocardial infarction and show prominent Q waves and deep terminal inversion of the T waves in Leads II, III, and V_F (Fig. 6). Records taken on September 6 and September 21 are similar except for greater inversion of the T deflections. The precordial leads (Fig. 7) are similar to those taken on Sept. 27, 1944, except that terminal inversion of the T waves is present in Leads V_5, V_6, and V_E. Leads from points low in the left posterior axillary line also show typical T-wave changes but those from points at higher levels, which exhibited significant changes after the first infarction, fail to show such alterations. The contrast of changes in Leads V_L and V_F produced by the two infarcts and the appearance with the second infarction of deep inversion of the T waves in the lead from the ensiform cartilage (V_E) are additional features of interest.

DISCUSSION

Wood, Wolferth, and Bellet[3] have proposed criteria for the electrocardiographic diagnosis of acute lateral or midventricular infarction. The features which they considered important were depression of the RS-T segment in Lead IV, and usually in Leads I and II, and the absence of signs of posterior infarction in Lead III. Using these criteria Thomson and Feil[4] reviewed the electrocardiograms of nineteen patients who were found at post-mortem examination to have lateral myocardial infarction. Their studies did not disclose a consistent correlation between the post-mortem and the electrocardiographic findings; only four of nine patients with recent lateral infarction showed the pattern described by Wood, Wolferth, and Bellet. Bosco[5] has employed the same criteria in an effort to establish lateral infarction as a distinct anatomicoclinical entity different from infarction involving the anterior or posterior walls of the left ventricle. It has been recognized[3, 4] that changes of this same type may result from digitalis, may occur in records made during attacks of angina pectoris, and may appear under other circumstances. Wilson[6] has pointed out that the most reliable signs of myocardial infarction consist of a sequence of characteristic alterations in both the QRS and T complexes. It has been our experience in this laboratory that lateral infarction of the more usual variety produces these characteristic changes in unipolar precordial leads from the left lateral thorax (Leads V_5, V_6, and V_7, which is from the posterior axillary line) at the level of the cardiac apex.[1] The records of the group of cases presented in this report are quite different from those of the more usual examples of lateral infarction, and we believe that they represent an important, although probably somewhat uncommon type of lateral myocardial infarction.

If the electrocardiographic observations made in these six patients are reviewed, certain features appear to be significant. The standard electrocardiograms of all but one of the patients show tiny or small Q waves and flat or slightly inverted T waves in Lead I. The magnitude of the changes in the T complex varies from case to case, but this would be expected since the electrocardiographic studies were made at very different stages of the infarction in the different instances. Although there are no Q waves in Lead I in the records of Case 5 which are reproduced, they were present in tracings taken earlier. This difference may be accounted for by a shift in the position of the heart. The ventricular complexes of the unipolar lead from the left arm (V_L) are similar in general outline to those of Lead I in all cases and exhibit small Q waves and flattening, sight inversion, or sharp terminal inversion of the T deflections. On the whole, the changes in Lead V_L are more striking than those in Lead I, and, in the instance of high lateral infarction (Case 5), the difference is pronounced, probably because the zone of infarction was in such a position as to face toward the left shoulder as well as toward the upper left axilla.

The character of the complexes of the standard precordial leads distinguishes these cases from the usual type of lateral infarction. There are no changes diagnostic of infarction in the QRS complexes of the leads from the left side of the precordium, and, in all but two cases (1 and 5), distinctive altera-

tions in the T complex are absent in the leads from this region. On the whole, the deviations from the normal in the standard precordial leads were least impressive in the cases of high anterolateral infarction. In one instance (Case 2) these leads are normal, in two (Cases 3 and 4) they show only tiny Q waves and minor changes in the T waves of the leads from the left side of the precordium, and in the remaining one (Case 1) the R waves are unusually small in all of these leads. Terminal inversion of the T waves is also seen in Leads V_4 and V_5 in this case. The usual precordial leads in the case of high lateral infarction (Case 5) display a pronounced decrease of the height of the R wave in successive leads. This case exhibits slight to moderate inversion of the T waves and some upward displacement of the RS-T segment in the leads from the left side of the precordium. This displacement is apparently of the persistent type since the clinical data do not suggest that the infarction was recent. There were, however, no roentgenographic signs of ventricular aneurysm, such as have been observed in some instances of persistent displacement of the RS-T segment.[7] In the single instance (Case 6) of high posterolateral infarction, the usual precordial leads showed at first only slight depression of the RS-T segment in Leads V_1 and V_2. Subsequently when the changes in other leads had become apparent, the T waves of the leads from the right side of the precordium became taller and slight terminal inversion of the T deflection appeared in Lead V_6.

So far as the supplementary leads taken from the upper left thorax are concerned, it may be said that, in all but one instance (Case 3), Q or QS and T-wave changes characteristic of myocardial infarction were recorded. In the single exception, only QRS changes were present, presumably because the infarction had occurred six months before. Earlier records of the usual type displayed characteristic alterations in the T wave. It was necessary to place the exploring electrode one to three intercostal spaces above the usual levels to obtain diagnostic electrocardiographic changes. In some instances (Case 6) the zone which yielded the most significant changes was relatively small whereas in others (Cases 1 and 4) it was much larger. This difference may have depended upon the relative size of the infarcted region, but we have no evidence bearing on this question. The distinction between anterolateral, lateral, and posterolateral infarcts is here based upon the position of the vertical lines through the points which yielded the most pronounced electrocardiographic changes. If the most pronounced evidence of infarction occurred in leads from points lying directly above those used in taking Leads V_3, V_4, and V_5, the case was put in the first group; if it occurred in leads from points above those used in taking Leads V_5 and V_6, it was placed in the second; and if it occurred in leads from points in the left posterior axillary line (V_7) and the left scapular line, it was placed in the third group.

Our experience with electrocardiograms obtained by means of unipolar leads from the higher levels of the chest has been rather limited. In order to control the observations made in our cases of high lateral infarction, we have examined tracings* obtained in a similar way from a small group of normal subjects and

*Some of the tracings utilized were taken in this laboratory[8]; others were taken by Miss Annie Mary Lyle of the Prudential Insurance Company.[9]

from a group of patients with various kinds of electrocardiographic abnormalities. Prominent Q or QS waves were not encountered in leads from the higher levels of the left anterolateral and lateral thoracic areas when the heart was normal. In general, the ventricular complexes of leads from the higher levels of the left anterolateral and lateral thoracic areas are transitional in form between those of the standard unipolar lead from the left side of the precordium and those of the unipolar lead from the left arm (Lead V_L). Whenever, for any one of a variety of reasons, there are large Q waves or QS deflections in Lead V_L, prominent Q waves will usually be present in some leads from the upper levels of the left thorax. It is, therefore, our present opinion that prominent Q waves, QS deflections, and sharply inverted T waves in Leads of the kind in question have an important bearing upon the diagnosis of myocardial infarction only when they give rise to ventricular complexes which are more typical of this lesion than the ventricular complexes of either the standard precordial leads or the unipolar lead from the left arm. The chief indication for additional leads from the upper levels of the left thorax is the presence of changes suggestive of infarction in Lead V_L without corresponding changes in the standard leads from the left side of the precordium.

There are several possible explanations for the occurrence of the most striking signs of infarction in leads from thoracic levels above those usually explored. We believe that this phenomenon is usually due to infarction of parts of the wall of the left ventricle which are closer to the base of this chamber than those more commonly involved. Some of the infarcts studied by Thomson and Feil[4] seem to have been of this sort, and we have recently heard of instances of high lateral infarction demonstrated at autopsy in which the standard extremity and precordial electrocardiograms resembled some of those described in this article.[10] It is, of course, possible that the peculiarities of the electrocardiographic patterns we have described are sometimes the result of rotation of the heart or some other change in the spatial relations of its surfaces to the standard electrocardiographic leads. It is also difficult to predict what modifications of the more common electrocardiographic patterns produced by infarction might arise as a consequence of ventricular enlargement following a coronary accident.

The delayed appearance of electrocardiographic evidence of myocardial infarction in Case 6 (Figs. 6 and 7) is, perhaps, deserving of comment. The clinical findings were sufficiently characteristic at the time of the patient's first admission to the hospital to justify the diagnosis made, but supportive electrocardiographic data were not obtained, despite frequent examinations, until ten days later. Some considerations which may account for the delayed appearance of electrocardiographic signs of infarction have been discussed elsewhere.[11] It has been pointed out that in leads from the precordium and extremities, in contrast to leads from the surface of the heart itself, the effects produced by the parts of the infarct responsible for RS-T displacement may obscure those produced by the muscle responsible for inversion of the T deflection. This is likely to happen when the muscle zone ischemic enough to give rise to changes of the first kind in direct leads is approximately equal in size to the zone ischemic only to the degree necessary to give rise to changes of the second kind. A loca-

tion of the infarcted region which is unfavorable with respect to the leads employed may also account for the late appearance of characteristic electrocardiographic evidence of infarction. Finally, the extension of an initially small infarct may explain the apparently late development of electrocardiographic changes in some instances. In the case under consideration the location of the infarct was unusual and certainly unfavorable as far as its detection by means of the usual extremity and precordial leads was concerned. It seems probable, however, that the factor first mentioned was an important cause of the late appearance of inversion of the T wave in Leads I, V_L, and V_6.

SUMMARY

Six cases of suspected infarction of the basal parts of the lateral wall of the left ventricle are reported. The usual unipolar limb leads and the six standard precordial leads failed to furnish unequivocal evidence of myocardial infarction in these cases. Unipolar leads from points on the anterolateral, lateral, and posterolateral aspects of the upper left thorax supplied electrocardiographic data of greater diagnostic value.

The types of lesions differentiated have been classified as high anterolateral, high lateral, and high posterolateral infarcts on the basis of the vertical lines in which the most significant electrocardiographic changes were recorded.

The opinion is expressed that in these instances the electrocardiographic changes typical of infarction were most pronounced in leads from the upper left thorax because the infarcted region was more basal and more lateral than is usually the case. It is, however, admitted that rotation of the heart or some other change in the relations of its surfaces to the usual leads may have been responsible for some of the electrocardiographic peculiarities encountered.

It is recommended that unipolar leads from the higher levels of the left thorax be taken when the clinical history and Lead I, or Lead V_L, both suggest that myocardial infarction has occurred and the standard leads from the left side of the precordium fail to display changes of the kind and magnitude expected.

REFERENCES

1. Wilson, F. N., Johnston, F. D., Rosenbaum, F. F., Erlanger, H., Kossman, C. E., Hecht, H., Cotrim, N., Menezes de Oliveira, R., Scarsi, R., and Barker, P. S.: The Precordial Electrocardiogram, AM. HEART J. **27**: 19, 1944.
2. Roth, G. M., and Kvale, W. F.: A Tentative Test for the Diagnosis of Pheochromocytoma, J. Lab. & Clin. Med. **30**: 366, 1945.
3. Wood, F. C., Wolferth, C. C., and Bellet, S.: Infarction of the Lateral Wall of the Left Ventricle: Electrocardiographic Characteristics, AM. HEART J. **16**: 387, 1938.
4. Thomson, H. W., and Feil, H.: Infarction of the Lateral Wall of the Left Ventricle. Pathologic and Electrocardiographic Study, Am. J. M. Sc. **207**: 588, 1944.
5. Bosco, G. A.: Sindrome Coronario Lateral, Buenos Aires, 1943, Imprenta Ferrari Hnos.
6. Wilson, F. N.: Diseases of the Coronary Arteries and Cardiac Pain, Edited by Robert Levy, New York, 1936, The Macmillan Company, p. 281.
7. Rosenbaum, F. F., Johnston, F. D., and Alzamora, V. V.: Persistent Displacement of the RS-T Segment in a Case of Metastatic Tumor of the Heart, AM. HEART J. **27**: 667, 1944.
8. Rosenbaum, F. F., and Wilson, F. N.: Unpublished observations.
9. Lyle, A. M.: Personal communication to F. N. Wilson.
10. Myers, G. B.: Personal communication to F. F. Rosenbaum.
11. Wilson, F. N., Rosenbaum, F. F., and Johnston, F. D.: The Interpretation of the Ventricular Complex of the Electrocardiogram, Advances in Internal Medicine, II, New York, Interscience Publishing Co. In press.