Electrocardiographic Changes in Acute Subendocardial Infarction

II. Small Subendocardial Infarcts

By Roderick W. Cook, M.D., Jesse E. Edwards, M.D., and Raymond D. Pruitt, M.D.

Among 24 cases of small subendocardial infarctions included in this report are 10 cases of anteroseptal distribution in which the electrocardiogram was characterized by the presence of deeply inverted T waves in some or all of the precordial leads. Two other types of change encountered among the remaining 14 cases are described. Electrocardiographic changes in this group of 24 cases are compared with changes encountered in 5 cases of large and near circumferential acute subendocardial infarction described in part I of this study.

The nature of the electrocardiographic changes attending 11 cases of large acute subendocardial infarcts from among a series of 30 cases of acute subendocardial infarction was reviewed in part I of this report. In part II, consideration will be given to the electrocardiographic changes encountered in the remaining 19 of the 30 cases that constituted this original series. Five additional cases will be considered. The latter cases failed to meet a criterion satisfied by all cases included in the initial series, namely, existence of an acute infarction at the time of necropsy. However, in each of these 5 additional cases, adequate electrocardiographic studies had been made during the presumably acute phase of an infarction, the scar of which was delineated at the time of necropsy.

The 19 cases from the initial series combined with these 5 additional cases constitute a group of 24 cases in each of which a subendocardial infarct of small or moderate extent or scarring thereof was found at the time of necropsy. These 24 cases will be placed in 3 categories, allocation being determined by the position of the subendocardial infarct in relation to the circumference of the wall of the left ventricle. Ten cases from the initial series and the 5 additional cases fall in the category of anteroseptal lesions. Eight cases from the initial series fall in the category of posterior or posteroseptal lesions, and 1 falls in the category of lateral-wall lesions.

I. Electrocardiographic Findings in Acute Anteroseptal Subendocardial Infarcts of Small or Moderate Size

Among the 15 cases in this category, 10 were characterized by the electrocardiographic finding of deeply inverted T waves in those precordial leads centered about position 3, 3 were characterized by the presence of segmental depression in the left precordial leads, and 2 by the existence of an intraventricular conduction disturbance suggestive of left bundle-branch block.

   A. Cases Without QRS Changes Related to Anteroseptal Lesion. Seven cases were placed in this subdivision. The electrocardiographic and morphologic findings representative of this division of cases are illustrated in figure 1a and b. In the case represented in figure 1a an anteroseptal lesion was attended by deeply inverted T waves in precordial leads V4 and V5 and by shallow inversion of T waves in V3, V6, and standard leads I and II. In 3 other cases, findings of similar char-
acter were encountered. In the case illustrated in figure 1b, an acute anterosepal lesion also was attended by similar T-wave changes. However, an old posterior scar was present and accounted for Q deflections in standard leads II and III. In 2 other cases, findings of similar character were encountered.

B. Cases Characterized by QRS Changes Related to Anterosepal Lesion. Three cases were placed in this subdivision. The electrocardiographic and morphologic changes are illustrated in figure 2a and b. In figure 2a, an acute anterosepal infarction was attended by deeply inverted T waves in precordial leads V_2 to V_5 and by lesser degrees of inver-

sion in V_1 and V_6. R deflections in V_1 and V_2 disappeared transiently and the R in V_3 lost amplitude. In figure 2b, comparable changes in T deflections appeared at the time of the acute infarction; the R deflections in V_1 to V_5 decreased in amplitude and in addition small Q deflections appeared in V_2 and V_3. The findings in the third case in this group resembled those in figure 2b.

2. Cases Characterized by Segmental Depression in Left Precordial Leads. The electrocardiographic and morphologic findings in the 3 cases of this division are illustrated in figure 3a, b, and c. In figure 3a, segmental depression was present in leads I, V_5, and V_6, whereas segmental elevation of comparable
degree existed in leads V₂ and V₃. R waves were absent in V₁ and V₂ and a tiny R wave in V₃ was preceded by an equally tiny Q deflection. Posterior components of the lesion found expression in small Q deflections in leads II, III, and aVF.

The findings in figure 3b included Q deflection in leads V₃ and V₅ in the record of October 24, 1949, related to old anteroseptal infarction. In the tracing of January 13, 1950, segmental depression related to the acute anterior subendocardial lesion was present in leads I, II, V₅, and V₆, whereas elevation of segments existed in aVR. Twenty-four hours later, segmental shifts were of lesser degree but loss of amplitude in the R waves of leads V₁, V₂, and V₃ suggested additional destruction of myocardial substance in the anterior wall of the left ventricle.

The record of December 31, 1954, in figure 3c was made prior to development of the acute infarct of the antero-apical region. The old lateral-wall infarct probably resulted in loss of amplitude of R waves in V₅ and V₆ and in an increased R/S ratio in V₁. In the tracing of January 6, 1955, segmental deviations appeared indicative of severe subendocardial injury or ischemia. In leads I, II, aV₁, aV₂, and V₂ to V₆, this deviation was downward, whereas in aVR, and to a lesser degree in V₁, it was upward.

3. Cases Characterized by Presence of Intracavitricular Conduction Disturbance. The 2 cases in this division are represented by figure 3d. The electrocardiogram was taken a few hours prior to death in a patient found at necropsy to have an acute anteroseptal infarct approximately 1 day old. Scarring from a posterior infarct also was present. The electrocardiographic findings were a QRS interval measuring 0.12 second and a form of ventricular complex suggesting delay in excitation of the left ventricular surface. Segmental depression in leads V₃ and V₆ and elevation in aVR were of a character similar to that seen in the other cases of acute subendocardial infarction of the anteroseptal region of the left ventricle presented in section 1-2 of this discussion.

II. ELECTROCARDIOGRAPHIC FINDINGS IN ACUTE POSTERIOR OR POSTEROSEPTAL INFARCTS OF SMALL TO MODERATE SIZE

In the 8 cases in which the major portion of the acute infarct was in the posterior or posteroseptal region of the wall of the left ventricle, the electrocardiographic findings were of varied character. Depression of the RS-T segments in left or in left and midprecordial leads was present in 5 of the 8 cases and represented the only suggestion of a type of change that might be held characteristic of these subendocardial infarcts of the posteroseptal wall. In no instance did Q deflections which could be related to the acute subendocardial infarct develop in leads II, III, and aVF. The range of variation in findings among these 5 cases is represented by the findings illustrated in figure 4a, b, and c.

In the case represented in figure 4a, the patient suffered from severe and frequently recurring bouts of anginal pain throughout the last 2 months of his life. His death occurred on October 26, 1954. Necropsy disclosed a small posterior infarct estimated to be 4 or 5 days old. The electrocardiogram of September 25, 1954 was made, therefore, approximately 3 weeks before the infarct developed, yet the segmental depression in the precordial leads is of only slightly lesser degree than in the record made on October 22, 1954 at a time when the acute infarct was present.

In the case from which findings are illustrated in figure 4b a moderately large posterior subendocardial infarct estimated to be 2 days old at the time of necropsy was associated with only slight segmental elevation in leads III and aVF and with segmental depression in leads I, aV₁, and V₃ to V₆. The small Q waves in leads II and III had been present in the electrocardiogram of May 22, 1941 and probably were a product of the posterosseptal scars indicated in the diagrams of the cardiac slices.

In the case represented in figure 4c, a predominantly posterior subendocardial infarct became nearly circumferential in the 2 apically placed slices. Portions of this acute lesion appeared to be about 10 days old while
FIG. 2. a. Acute anteroseptal subendocardial infarction attended by transient decrease in voltage of R waves in precordial leads V₂ and V₃ and by deep inversion of T waves in precordial leads V₂ to V₅. The patient was a man, 47 years of age, who had an acute myo-

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a few areas were only a day or 2 old. The electrocardiogram of August 11, 1954 was made on the day of the patient’s death. Apart from the segmental depression in V₅, only loss of voltage of deflections in the standard and extremity leads suggested myocardial changes in the electrocardiogram of a patient who had left bundle-branch block prior to development of the acute infarct.

Of the 3 remaining cases of the 8 with posterior or posteroseptal subendocardial infarcts, one (fig. 5a) was characterized by the development at the time of the infarct of right bundle-branch block and segmental depression in leads I and V₂ and by segmental elevation in lead III. In the other 2, represented by the findings in the case illustrated in figure 5b, small posterior infarcts produced no identifiable abnormalities in electrocardiograms recorded soon after onset of symptoms associated with development of the lesion.

III. ELECTROCARDIOGRAPHIC FINDINGS IN ACUTE SUBENDOCARDIAL INFARCTION INVOLVING LATERAL WALL OF LEFT VENTRICLE

The records in the 1 case in this category have been published elsewhere.¹

COMMENT

To the 19 cases of acute subendocardial infarcts of small or moderate size originally included in this series were added 5 cases of anteroseptal subendocardial infarction. In all 5 of these added cases, electrocardiographic findings included deep inversion of T waves in the precordial electrocardiogram. Because of this fundamental uniformity among these added cases, observations regarding incidence of a certain type of electrocardiographic alteration must be made with appropriate allowance for the distorting influence of these added cases.

As regards acute anteroseptal subendocardial lesions, the conclusion appears justified that 2 types of change occurred most commonly but not in the same cases. These were deeply inverted T waves in the precordial electrocardiogram and depression of the S-T segments in all precordial or in left precordial leads, usually associated with elevation of segment in lead aV₉. QRS changes related to the acute infarction either were absent or were of limited extent. When present, these changes consisted of loss of amplitude in R waves in precordial leads V₁, V₂ and V₃ sometimes associated with appearance of small Q deflections in these same leads. Such changes occurred in 5 among 15 cases (3 cases under heading I-1-B and the first 2 cases under I-2). In 2 other cases (heading I-3) an intraventricular conduction disturbance suggestive of left bundle-branch block was present.

In no instance among the 8 cases of acute posterior or posteroseptal infarction were there Q waves related to the acute lesion. Only the finding of segmental depression in the left or left and midprecordial leads in 5 of the 8 cases could be held suggestive of a recurring pattern. Appearance of segmental depression in precordial leads in such cases is a finding discordant with simple predictions based on the dipole theory. Lesions of this distribution, if they influenced at all the segmental position in precordial leads, should produce elevation. The most likely basis for this discrepancy between theoretic prediction and recorded fact is lack of conformity between the boundaries of the infarct as defined at necropsy and the boundaries of the ischemic zone responsible for the segmental deviation. If the ischemic zone involved not only the myocardium immediately adjacent to the infarct but also extended into the apicobasal subendocardial regions, then segmental devi-
Fig. 3. a. Acute patchy anteroseptal subendocardial infarction attended by depression of RS-T segments in precordial leads V₅ and V₆ and by Q deflection in leads V₂ and V₃. The Q waves in leads II, III, and aVF may be ascribed to the lesion in the posterior wall of the left ventricle. This lesion was older by approximately 1 week than the remainder. The patient, a man aged 82 years, died on April 7, 1954. Neither the clinical nor the pathological evidence rendered certain the presence of all portions of the patchy anteroseptal lesions when the

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ations of the kind recorded in the cases under
discussion would be in keeping with predictions based on generally accepted theory.

A lack of conformity between the boundaries of the infarct and the boundaries of the
ischemic zone reasonably should be anticipated. That such a lack of conformity occurs is substantiated by 2 other features of the data bearing on the correlation between
infarcted myocardium and segmental deviations in the electrocardiogram. First, the
correlation between size of infarct and extent of segmental deviations among the cases constituting this entire series is poor, even when allowance is made for possible failure to obtain an electrocardiogram at that time when segmental deviations were at their maximal extent. For example, an instance of striking segmental shifts in relation to a comparatively small infarct is found in the case represented in figure 3c. Second, in at least 1 case in this series (case represented in figure 4a), segmental deviations of similar distribution and degree were present in electrocardiograms made approximately 3 weeks before and on the day of occurrence of the infarct of the posterior wall of the left ventricle as found at the time of necropsy. While the infarct presumably developed in that portion of the myocardium where ischemia was most intense, and while existence of the zone of ischemic myocardium
was responsible for the segmental changes, presence of infarcted tissue was only incidental to existence of segmental deviations.

In summary of these observations bearing on T-wave inversions and segmental shifts, it may be said that subendocardial infarction is the morphologic consequence of intense and sustained ischemia. In some instances the total region of ischemia may have been little larger than the infarct that developed. In other instances the infarcted myocardium may have represented only a small fraction of the total region of ischemia. Although segmental deviations and inversion of T waves were the principal electrocardiographic attendants of the acute subendocardial infarcts in this series, these electrocardiographic changes bear a specific relation to the ischemic and not to the infarcted tissue. The extent of infarcted tissue cannot be predicted from the degree of electrocardiographic changes encountered, and the very presence of infarcted myocardium is a deduction justified by, but not inherent in, the nature of the electrocardiographic changes.

When the electrocardiographic findings in the 5 cases of large and near-circumferential subendocardial infarcts presented in part 1 of this report are compared with the changes encountered in the present group of cases, certain differences are noted.

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d. Acute antero-apical subendocardial infarction associated with a healed anteroseptal lesion in a man, aged 56 years at the time of death. The electrocardiogram of October 24, 1949 was recorded approximately 12 hours after the patient experienced an attack of pain in the chest. The Q deflection in precordial lead V4 was ascribed to effects of an anteroseptal infarct that presented as a scar at the time of necropsy. The tracing of January 13, 1950 was taken 5 hours after onset of a second episode. Significant features in this record included depression of RS-T segments in leads I, II, aV3, V6, and V5, elevation of segment in aV6, and loss of amplitude of R waves in V2 and V4. In the record of January 14, 1950 the R waves in V2 and V4 were even smaller. c. Acute anteroseptal subendocardial infarction in a man, aged 53 years, who had experienced an initial infarction 8 years previously. Episodes of severe chest pain occurred on December 29, 1954 and January 6, 1955. In the record of December 29, 1954 the tall R in V1 and the small R in V6 probably are expressions of the old infarct in the lateral wall of the left ventricle. In the record of January 6, 1955, major segmental deviations have appeared in relation to an episode of severe chest pain which began 6 hours before this tracing was made. Death occurred 12 hours later. The acute infarction was judged to be less than 24 hours old. d. Acute anteroseptal and old posterolateral infarction in a man, aged 48 years. Pain related to the acute lesion began on the day of the patient's death. At necropsy the acute infarct was judged to be about 12 hours old. The electrocardiogram shows changes indicating delay in excitation of the surface of the left ventricle. There was a Q deflection in aV4. Segmental depression and upright T waves were present in leads V4 and V5.
FIG. 4. a. Acute subendocardial myocardial infarction in a man, aged 73 years, with severe angina pectoris and development of status anginosus in September 1954. On October 21, 1954, 5 days before death a particularly severe episode of pain occurred, probably with occurrence of a small subendocardial infarct of the posterior wall of the left ventricle. Note that segmental depression in leads I, II, and V5 to V6 and elevation of segment in aVR were only slightly greater in the record made after development of the infarct than in the tracing made 3 weeks earlier. b. Acute posterolateral subendocardial infarction

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associated with healed lesions of posterior and septal location. The patient was a man, 58 years of age. The tracing of November 10, 1952 was recorded 1 day after the onset of symptoms of myocardial infarction and 1 day prior to the patient's death. Q waves were present in the tracing dated May 22, 1941 and these persisted unchanged in the record of November 10, 1952. Segmental depression and inversion of T waves were present in leads I, V₅, V₆, and V₇ of the latter record. The RST segment in aVR and V₁ was elevated. c. Acute posterior-apical subendocardial infarction associated with anterior subendocardial scarring in the presence of left bundle-branch block. The patient was a woman, 60 years of age. Symptoms of myocardial infarction developed on August 10, 1954 and the patient died the next day. The changes in the electrocardiogram between January 30, 1954 and August 11, 1954 probably are a consequence of the acute lesion, since the old infarct was believed to have occurred in July 1953. Note loss of amplitude of QRS complexes in standard limb and extremal leads and greater segmental depression in lead V₆.
1. Whereas the pattern of deeply inverted T waves in the midprecordial lead was not encountered in the group of large, near-circumferential infarcts, it appeared in 5 of the cases originally included among the small acute subendocardial infarcts and in 5 additional cases appended to the series.

2. Segmental depression in precordial leads and segmental elevation in lead aVR were common attendants both of acute anteroseptal and of acute posterior or posteroseptal infarcts of small or moderate size. However, the incidence of this change approximated 50 per cent in the 19 cases in the original series of this type of infarct, whereas segmental depression occurred in all cases of large acute subendocardial infarction. Furthermore, the degree of depression commonly was greater in the presence of the larger infarcts.

3. When QRS changes did attend development of small anteroseptal subendocardial infarcts, the nature of these changes was similar to the nature of those occurring with large subendocardial lesions. However, the incidence of such changes was less in association with the small lesions (3 among 8 cases; in 2 additional cases there were intraventricular conduction disturbances) than with the large lesions (all 5 cases). Q waves related to the acute infarction did not appear in lead II, III, or aVF in the group of 8 posterior or posteroseptal lesions.

SUMMARY

1. The electrocardiographic findings have been presented in 19 cases of acute subendocardial infarcts of small or moderate size, and in 5 additional cases of anteroseptal infarcts in which electrocardiograms were recorded at a time when other clinical data indicated the lesion was acute.

2. Three types of electrocardiographic change related to the acute phase of the infarction were encountered most commonly. These were: (a) depression of the RS-T segments in all precordial or in the left precordial leads, (b) deeply inverted T waves in some or all of the precordial leads, and (c) diminished height of R waves sometimes associated with appearance of small Q waves in certain precordial leads.

3. Because QRS changes are of so limited an extent and RS-T-segment and T-wave changes afford no specific indication of myocardial infarction as contrasted with subnecrotic myocardial injury or myocardial ischemia, the electrocardiographic delineation of subendocardial infarction is commonly inferential as to the fact of occurrence and nebulous as regards extent and configuration of the lesion.

REFERENCE

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RODERICK W. COOK, JESSE E. EDWARDS and RAYMOND D. PRUITT

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