Electrocardiographic Changes in Acute Subendocardial Infarction

I. Large Subendocardial and Large Nontransmural Infarcts

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The electrocardiographic consequences of subendocardial infarction will find their most evident and characteristic expression in the acute phase of a large lesion. Five cases of this kind are presented here and the associated electrocardiographic changes are described. At what level in the myocardium between the endocardium and epicardium do the electrocardiographic expressions of subendocardial infarction give way to those of transmural infarction? Data in 6 cases of large nontransmural infarction are presented for comparison with findings in the 5 cases of subendocardial infarction.

Among the varied disorders that alter the form of the electrocardiogram, myocardial infarction is distinguished by the feature of localized loss of myocardial mass. When this destruction takes the form of a large transmural infarct, diagnostic alterations are commonly evoked in the QRS deflections of an electrocardiogram properly recorded by conventional techniques. When the region of myocardial necrosis is confined to the inner layers of the left ventricular wall, changes in the QRS complexes of the scalar electrocardiogram may not be pathognomonic of this loss of myocardial substance. This fact is illustrated in the electrocardiographic and pathologic data reproduced in figure 1a. Destruction of a thick ring of myocardium lining the entirety of the left ventricular cavity in this case failed to produce a QRS configuration distinctive of myocardial infarction.

Among several excellent articles1-3 on the electrocardiographic changes associated with subendocardial infarction, only in that of Myers and associates2 were QRS changes reported of a character held to be significant and indicative of the subendocardial injury.

In 1945, experimental studies4 of the electrocardiographic consequences of subendocardial injury led to the conclusion that such lesions commonly are attended by loss in height of the R waves and the development of a small Q deflection or a notch low on the R wave in records taken from precordial leads overlying the traumatized wall of the left ventricle. In 1954, Prinzmetal and co-workers5 reported loss of amplitude of the R deflection in 10 of 12 instances in which records were made from leads placed on the ventricular surface in a region overlying a subendocardial lesion produced by cautery.

When the clinical and the experimental data mentioned in the preceding paragraphs are combined, substantial support emerges for the view that those QRS alterations that attend subendocardial injury are of a kind subject to identification only in certain cases in which sequential records are available. Changes pathognomonic of localized loss of myocardial mass uncommonly present themselves in readily identifiable form in an isolated electrocardiogram.

If a clinical diagnosis of subendocardial infarction is to be supported electrocardiographically, experience on record would indicate that the major evidence will be derived from changes in portions of the ventricular complex other than the QRS deflection, namely the S-T segments and the T waves.

Experimental evidence supporting the concepts of the dipole theory with respect to the

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FIGURE 1. (Legend on opposite page.)
segmental shifts attending acute subendocardial injury was presented in 1948. In 1955, evidence was reported suggestive of a relationship in certain instances between deeply inverted T waves in the precordial electrocardiogram and localized subendocardial injury. The limited personal experience represented in these articles supports conclusions resting on a much larger body of evidence compiled in medical writing and summarized briefly as follows: 1. In acute subendocardial injury, as in other types of myocardial injury, segmental elevation will be recorded from a unipolar lead facing the most severely injured fibers, and segmental depression will be recorded from a lead facing the least severely injured fibers. Because of the nature of the leads commonly used in clinical electrocardiography, segmental depression as it occurs in the precordial electrocardiogram has come to be regarded as the usual expression of acute subendocardial injury or ischemia. However, segmental elevation in lead aVR, which commonly reflects variations in potential like those encountered in a lead proximate to the great orifices at the base of the heart, is an equally consistent finding in subendocardial injury. 2. Deep inversion of T waves in precordial leads overlying the left ventricular surface may well be the expression of a transmural zone of ischemia, attended by actual necrosis of myocardial fibers in certain regions, most commonly the subendocardial.

Both segmental shifts and T wave inversions identical with those encountered in subendocardial infarction may occur in transient states of myocardial ischemia and may be resolved completely within a few minutes after the ischemic state has been relieved. This observation points up a fact of critical significance, namely that, unlike the QRS changes pathognomonic of loss of myocardial mass and hence of myocardial infarction, the segmental and T-wave changes associated with subendocardial lesions afford only inferential evidence of myocardial necrosis. Furthermore, since transient ischemia can produce changes of similar character, correlations between the degree of electrocardiographic alterations and the extent of morphologic changes must be of limited accuracy.

Methods and Materials

A review of the necropsy records at the Mayo Clinic from 1947 through 1955 disclosed 182 cases in which acute subendocardial infarcts were present. To simplify the correlation of electrocardiographic and morphologic changes, all cases were eliminated in which a healed as well as an acute infarct was present and in which available electrocardiographic data did not permit satisfactory distinction between the electrocardiographic consequences of the old infarct and the more recent lesion. Another and larger group of cases was eliminated in which any one of a variety of disorders existed that might have rendered difficult the proper identification of the origin of the electrocardiographic changes. Excluded on this basis were cases of pulmonary embolism, acute and chronic pericarditis, valvular heart disease, congenital heart disease, myocardial aneurysm, leukemic and granulomatous myocardial infiltration, pericardial effusion, severe ventricular dilatation, acute and subacute bacterial endocarditis, abscesses of the myocardium, cardiovascular syphilis, pronounced left ventricular hypertrophy, and digitalis intoxication. Of the original 182 cases, 152 were eliminated on the basis of one or the other of the preceding considerations.

The present report concerns 11 of the remaining 30 cases. These 11 cases are distinguished from the other 19 by the larger amount of acute myocardial necrosis present at necropsy. The 19 cases not included in the present account will form the basis of a portion of a second report.

Fig. 1. a. Old circumferential nontransmural infarction in a 36-year-old man. This patient had dyspnea on exertion for 2 years, and mild angina of effort for 1 year. He did not recall any symptoms of acute myocardial infarction. This electrocardiogram was made 4 days before death. b (Case 1). Acute circumferential subendocardial infarction in a 74-year-old man. Symptoms of infarction developed 10 hours before the electrocardiogram of July 18, 1955, was recorded. The patient died the next day. c (Case 2). Acute, nearly circumferential, subendocardial infarction in a 64-year-old woman who had acute granulomatous arteritis attended by partial occlusion of coronary ostia. Anginal pain first experienced 2 months before death. Pathologically, the infarct was judged to be 3 days old.
FIGURE 2. (Legend on opposite page.)
In all of the 11 cases in this study, the acute lesion involved at least 50 per cent of the circumference of the left ventricular wall and commonly achieved or approached circumferential extent in at least 1 of the 5 slices into which the heart was sectioned at right angles to its apicobasal axis.

In 5 of the 11 cases, the infarct throughout the major portion of its extent involved no more than the inner half of the total thickness of the ventricular wall. Lesions of this character will be referred to hereafter as "subendocardial," in distinction to the lesions in the remaining 6 cases, which will be called "nontransmural." In this latter group, some significant portion of the infarct involved more than 50 per cent but less than 75 per cent of the total thickness of the ventricular wall.

**RESULTS**

**Large Acute Subendocardial Infarcts.** The 5 cases placed in this category form a group with satisfying homogeneity from both morphologic and electrocardiographic points of view.

Morphologically, a large subendocardial infarct was present involving always the anterior wall of the left ventricle together with circumferential or nearly circumferential invasion of the lateral or septal and, sometimes, the posterior portions of the left ventricular wall (fig. 1b and c, and 2). The oldest lesion in this group was in case 5 (fig. 2c) and was judged to be about 2½ weeks of age. In the other 4 cases, the infarcts ranged in age from approximately 10 hours to 3 or 4 days.

Electrocardiographically, a reasonably distinct pattern of changes could be defined as follows: 1. QRS deflections were characterized by low-amplitude R waves in V 1–4; in 3 instances in which preinfarction electrocardiograms were available (fig. 1b and c, and 2b), actual loss of amplitude occurred in R waves from these points, associated with the appearance of 8 waves in leads V 5 and V 6. 2. Q deflections were absent in all except case 1 (fig. 1b) in which such waves 1 mm. deep appeared only in leads V 2 and V 3. Complete loss of R waves was recorded in leads V 1 to V 4 in case 2 (fig. 1c). 3. Depression of the S-T segment in at least the left precordial leads was present in all instances, ranging from 2 mm. in case 5 to 7 mm. in case 1. Segmental elevation in lead aV R was a consistent finding in those 4 cases in which this lead was recorded. 4. Although some peculiarities in QRS and segmental configuration occurred in standard limb leads and extremital leads other than aV R, these changes were of lesser degree than they were in the precordial leads and varied in nature and distribution from case to case.

Clinically, the single feature common to all 5 cases was the critical nature of the patient's condition throughout the period subsequent to the postulated time of occurrence of the large infarct.

In case 1, a state of shock existed throughout the 10 hours from the onset of pain until the patient's death. In case 2, the patient's status was characterized by fever, tachycardia, pallor, dyspnea, orthopnea, and gallop rhythm. The existence of primary arteritis attended by occlusion of the major branches of the aortic arch precluded accurate appraisal of the blood pressure in the arms; in the legs, the pressure was 140 mm. Hg systolic and 90 diastolic. In case 3, the clinical picture was confusing from the diagnostic standpoint. After 3 days of severe abdominal pains attended by nausea and vomiting the patient was semicomatose and her blood pressure was 94/72. She died on the second day in the hospital. In case 4, the patient had severe angina pectoris for several months and had status anginosus 3 weeks prior to his death, which occurred abruptly and without preceding symptoms of shock. In case 4.

![Fig. 2. a (Case 3). Acute circumferential subendocardial infarction in a 57-year-old woman who had abdominal cramps, obstipation, nausea and vomiting for 3 days and who died on April 12, 1950. The acute infarct appeared to be 2 or 3 days old. b (Case 4). Nearly circumferential, acute subendocardial infarction in a 57-year-old man who had severe angina pectoris for 3 months prior to death, which occurred suddenly on August 23, 1955. The infarct was approximately 24 hours old. c (Case 5). Nearly circumferential, acute subendocardial infarction in a 71-year-old woman. Dyspnea and symptoms ascribed to "flu" had developed 3 weeks prior to death. She did not experience thoracic pain. On admission, she had severe congestive heart failure. She died within 24 hours, on May 25, 1951. The acute infarct was judged to be about 2½ weeks old.](http://circ.ahajournals.org/)

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Figure 3. (Legend on opposite page.)
5, in which infarction occurred approximately 2½ weeks prior to death, the patient arrived at the hospital only a few hours before her death. She was in severe congestive heart failure attended by Cheyne-Stokes respiration. Her blood pressure was 118/80. The pulse rate was 120 beats per minute. She failed to respond to intensive treatment for congestive heart failure.

Large Acute Nontransmural Infarcts. The electrocardiographic behavior in this group of 6 cases can be developed appropriately by presentation of the findings in cases 6, 8, and 11.

In case 6 (fig. 3a), a 73-year-old woman had experienced episodes of anginal pain occurring at rest and lasting about 15 minutes at intervals of 1 to 2 hours for 3 days prior to her arrival at the hospital on September 9, 1955. She died abruptly and unexpectedly 15 days after admission. The acute infarct extended from one half to three quarters of the distance between the endocardial and epicardial aspects of the anterior wall of the left ventricle in the second and third slices from the apex (fig. 3a). The lesion was judged to be about 2 weeks old. Electrocardiographically, the infarct was attended by changes like those commonly encountered in an acute transmural anteroseptal myocardial infarct.

Case 7 resembled case 6 morphologically; electrocardiographically, the findings were only suggestive of acute anterior myocardial infarction.

In case 8 (fig. 3b), a 49-year-old man had experienced myocardial infarction 2 years earlier and died 14 hours after the onset of a second infarction. The acutely infarcted myocardium overlaid the subendocardial scar and extended to the junction of the inner three fourths of the left ventricular wall with the outer fourth (fig. 3b). As in case 6, the electrocardiographic changes were those commonly associated with an acute transmural lesion; the findings were those of posterior infarction. Segmental depression in leads V₃ and V₄ was of unusually great degree.

Two other patients (cases 9 and 10) among the 6 who had large acute nontransmural infarcts had similar electrocardiographic findings.

In case 11 (fig. 3c), a 58-year-old woman had been subject to angina pectoris for 3 years prior to death, which occurred 2 days after the onset of symptoms of acute myocardial infarction. In the second slice from the cardiac apex (fig. 3c), the acute infarct extended over half the thickness of the left ventricular wall from the endocardium toward the epicardial surface. The electrocardiographic changes were nonspecific. The small Q waves in leads II, III, and aVF probably were related to scarring of the posterior basal wall of the left ventricle. Segmental depression in leads I, II, aVL, and V₆, and segmental elevation in aVR and V₁ to V₄ probably were expressions of the acute infarct. The R waves in V₁ to V₄ were of low amplitude, and small Q deflections were present in V₅ and V₆.

Discussion

The electrocardiographic changes encountered in our 3 cases of large acute subendocardial infarcts conform to a pattern that might be derived from postulates based on the dipole theory and from clinical reports on the experience of others with cases in which similar types of lesion were present. The consistency with which these changes appear during the early hours of the development of such large acute lesions does not support the concept that the subendocardial region is electrocardiographically silent. However, in contrast to the consistent appearance and impressive degree of segmental changes was the limited extent of the alteration in the QRS complexes. Here again, the nature of these changes was in conformity with experimental
evidence suggesting that loss of amplitude of R waves in precordial records is a more likely consequence of subendocardial injury than is the recording of large Q waves from these same leads.

The reasonable conformity between the electrocardiographic changes encountered in these 5 cases and those changes that would have been predicted on the basis of theory and experimental evidence should not lead to complacent acceptance of the view that all acute subendocardial lesions produce similar changes, nor that a large acute subendocardial infarct exists in all instances in which such changes are present. Exceptions to either of these conclusions will be presented in the forthcoming second portion of this report, which concerns small subendocardial infarcts.

The segmental deviations and QRS changes in 4 of the 6 cases of large acute nontransmural infarcts were of a kind commonly associated with transmural infarcts of comparable distribution. This group of cases is reported in an attempt to present facts bearing on 2 interesting questions: (1) at what level of the ventricular wall in a plane parallel to the epicardial and endocardial surfaces is destruction of myocardium attended by development of Q deflections in electrocardiograms derived from direct or semidirect leads; and (2) at what level of injury in the ventricular wall does the segmental depression of acute subendocardial injury as manifest in overlying epicardial leads give way to the segmental elevation in these same leads commonly associated with acute transmural injury. Since the definable morphologic boundaries of an infarct may not and probably do not coincide with the limits of injury (using the term "injury" in distinction to "ischemic" and "normally functioning" myocardium as regards electrocardiographic consequences), deductions derived from correlation of morphologic and electrocardiographic data must be viewed with appropriate circumspection. Such evidence as is presented here suggests that the level in question for both segmental deviations and QRS changes lies not less than half nor more than three quarters of the distance from the endocardium to the epicardium. Experimental evidence and theoretic considerations bearing on this problem have been presented in an earlier report.5

**Summary**

From a series of 182 cases in which acute subendocardial infarcts were present, 30 were selected for intensive study. The availability of appropriate electrocardiographic evidence and the absence of complicating lesions that might confuse the interpretation of such evidence were principal factors determining the selection.

The findings in 11 of these 30 selected cases are presented here. The remaining 19 will constitute a portion of another report.

Five cases included in the present account made up the category of large subendocardial infarcts of circumferential or nearly circumferential extent. Electrocardiographic findings in these 5 cases consistently conformed with that pattern postulated for acute subendocardial lesions on the basis of theoretic considerations, experimental evidence, and the clinical findings included in certain earlier accounts. Low-amplitude R waves were present in precordial leads V1 to V4; these waves were completely lost in 1 case. Preinfarction electrocardiograms were available in 3 of the 5 cases. Comparison of records before and after infarction revealed actual loss of amplitude in R waves from the 4 precordial points just noted, associated with the appearance of S waves in V5 and V6. Q deflections appeared in only 1 case; these deflections were 1 mm. deep and were present in leads V2 and V3. Depression of the S-T segment in at least the left precordial leads was present in all instances, ranging from 2 to 7 mm. Elevation of this segment in lead aVR was also present in the 4 cases in which this lead was recorded. Although some peculiarities of QRS and segmental configuration occurred in standard limb leads and extremital leads other than aVR, these changes were of lesser degree than in the precordial leads and varied in nature and in distribution from case to case.
In contrast to the electrocardiographic findings in these 5 cases of large acute subendocardial infarcts were those encountered in the 6 cases of large acute nontransmural infarcts. The changes in these cases generally were similar to those that would be anticipated as a consequence of acute transmural infarcts of similar distribution.

This evidence suggests that the change from a subendocardial to a transmural type of electrocardiographic pattern occurs when an infarct involves somewhat more than the inner half of the thickness of the ventricular wall.

**SUMMARY IN INTERLINGUA**

Ex un serie de 182 casos in que acute infarctos subendocardial esseva presente, 30 esseva seligite pro studios plus intense. Le disponibilitate del appropriate documentation electrocardiographic e le absentia de lesions complicatori apte a confunder le documentation esseva le principal factor in determinar le selection.

Le constatationes in 11 del 30 seligite cases es presentate in iste reporto. Le remanente 19 casos va fornir le material pro un altere reporto.

Cinquè del casos includite in le presente reporto constitueva le categoria de grande infarctos subendocardial dde dimensiones circumferential o quasi circumferential. Le constatationes electrocardiographic in iste 5 casos se conformava uniformemente con le postulationes pro acute lesions subendocardial, facite super le base de considerationes theoretic, de observationes experimental, e de constatationes clinice de publication anterior. Undas R a basse amplitude esseva presente in le derivationes precordial V₁ a V₄. Iste undas esseva completamente perdiite in 1 del casos. In 3 del casos, electrocardiogrammas de un periodo pre-infarccmental esseva disponibile. Le comparation del registraiones facite ante e post le infarccimento revelava un perdita de amplitude in le undas R ab le 4 punctos precordial justo notate, associate con le apparition de undas S in V₅ e V₆. Deflexiones de Q appareva in solmente 1 caso. Iste deflexiones habeva un profunde de 1 mm e occurreva in V₂ e V₃. Depression del segmento S-T in al minus le derivationes precordial sinistre esseva presente in omne casos. Illo amontava a inter 2 e 7 mm. Elevation del segmento S-T in le deriation aV₁ esseva etiam presente in le 4 casos in que iste derivation habeva esseva obtenite. Ben que certe peculiaritates de QRS e del configuration segmental occurriva in le derivationes del extremities altere que aV₁, illos esseva minus pronunciata que le alterationes in le derivationes precordial, e lor natura e lor distribution variava ab un caso al altere.

Le constatationes electrocardiographic in iste casos de grande acute infarctos subendocardial contrastava con le constatationes in le 6 casos de grande acute infarctos nontransmural. In istos, le alterationes esseva generalmente simile al alterationes expectate como consequentia de acute infarctos transmural de un simile distribution.

Iste observationes pare indicar que le transition ab le typo subendocardial al typo transmural in le configuration electrocardiographic occurre quando un infarcto comencia interes sar plus que le mediatite interior del spissitate del pariete ventricular.

**REFERENCES**


The anastomoses between coronary arteries present in the normal heart provide a basic collateral circulation, which while not sufficient to maintain myocardial contractions following acute interruption of the normal arterial supply, generally enable the survival of variable amounts of myocardium providing death does not follow the occlusion. In the survivors there is a gradual enlargement of collateral circulation quite variable in amount but sufficient to enable the ischemic myocardium to regain its contractile ability. The collateral blood comes exclusively from other arteries. The nearest artery or the anatomically shortest path of least resistance appears to be the commonest source and route.

Collateral coronary circulation was discussed from the standpoints of a historical review, physiologic studies of collateral coronary circulation, surgical approach to augmenting collateral coronary circulation, including abrasion of heart and pericardium, coronary sinus ligation, acute arterial retroperfusion of the coronary sinus, chronic arterilization of the coronary sinus, the mechanism of protection of arterial retroperfusion of the coronary sinus, implantation of internal mammary artery into myocardium, and metabolic factors operative in the augmentation of collateral coronary circulation. If and when it is possible to reduplicate the experiments of nature and completely interrupt all the normal inflow to the myocardium with survival, it should be possible to trace and measure the blood flow from the extracardiac arterial sources. Careful studies of treated and untreated groups with long-term follow-up are needed before precise answers can be reached. Much is yet to be learned, but the evidence at hand indicates that a firm foundation has been established for improving collateral circulation to the myocardium by surgical means.

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