CARDIOGRAPHIC CONTRECoup IN THE COURSE OF CARDIAC INFARCTION

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The site of stoppage in the coronary circulation is first learnt from the electrocardiogram of the resultant infarct. Yet, correlation of the information thus gained with the findings at necropsy has not invariably proved reliable, for occlusive arterial lesions with associated infarction that were not suspected from inspection of the electrocardiogram are sometimes discovered at necropsy.

To learn more about the precise distribution of such silent areas of infarction, in addition to continued comparison of cardiographic and necropsy findings, there is need to observe the successive changes in the cardiogram that may occur during the further course of cardiac pain. When the effects of impact of newer cardiographic changes on the earlier ones become known, prediction of the anatomical limits of an injury to the myocardium will then fall within the competency of the clinician.

This paper records a study which disclosed that an extension of a myocardial lesion, following further coronary occlusion, may cause certain existing cardiographic signs to regress, or even to disappear, so that an appraisal of the size and distribution of the ultimate muscle damage is thereby rendered invalid. It was to this contrecoup effect that this study was specially directed.

THE INVESTIGATION

The electrocardiogram was examined in 250 patients with cardiac infarction, in whom more than one tracing had been taken in the course of the illness. When the patients returned for re-examination they were specifically interrogated concerning any change they might have noticed in the frequency or severity of their cardiac pain, that might suggest an extension of the infarction in the intervening period. The information gained from such an inquiry, however, usually failed to tally with the conclusions reached from reading the electrocardiogram.

As my personal preference is for CR leads, these are used throughout this paper and in the figures, but the reader may regard V leads as similar to CR leads in regard to this work.

The size of the heart was always determined by cardioscopy, and hypertension with its influence on the cardiogram giving left ventricular preponderance was a special care. When either the initial or subsequent tracings showed bundle-branch block the patient was excluded from the series.

The place of the initial infarct was determined from the first cardiogram, and was regarded as belonging to one of four situations in the left ventricle, excluding the ventricular septum and possible extension into the right ventricle, in accordance with the electrocardiographic pattern that each presented (Fig. 1). These four positions, their cardiographic patterns, and the number of cases showing changes peculiar to each position are shown in the Table.

Thus, there were 68 patients with anterior infarction (TIT4 pattern), 91 with lateral infarction (TIT7 pattern), 36 with postero-lateral infarction (TIIT7 pattern), and 55 with postero-medial infarction (QTIII pattern). In the abbreviated designation applied to the separate cardiographic patterns that indicate the respective leads in which the abnormal waves are found, Roman

FIG. 1.—Diagrammatic representation of the separate sectors of the left ventricle which may show infarction, together with the cardiographic design peculiar to each. Roman numerals indicate the limb lead deformity and arabic numerals the chest lead deformity.

TABLE
DISPOSITION OF INFARCT IN 250 PATIENTS, TOLD FROM CHANGES IN INITIAL ELECTROCARDIOGRAM, ALONG WITH NATURE OF CHANGES SHOWN IN SUBSEQUENT CARDIOGRAM

Modification of initial cardiographic deformity in subsequent tracing	Site of infarction as determined by initial cardiogram			
	Anterior (TIT ₄)	Lateral (TIT ₇)	Postero-lateral (TIIIT ₇)	Postero-medial (QTIII)
Stationary	15	21	12	28
Regression	26	23	10	6
Limited spread	7	20	2	2
<i>Extension</i>				
Contiguous	8	23	9	—
Antipodal	12	4	3	19
Total	68	91	36	55

numerals denote limb leads, and arabic numerals refer to the chest leads. Thus, TIT₄, for example, refers to a deformity of the T wave in lead I and in the chest lead C₄. Similarly, QTIII signifies abnormal Q and T waves in the limb lead III.

Lead IIIR (lead III taken during deep inspiration), although not included in most of the illustrations in order to save space, was always recorded, so that whenever an abnormal Q wave or inverted T wave is reported in lead III it is to be regarded as a material or significant change.

As patients in whom only one electrocardiogram was recorded have been excluded from the investigation, the numbers in the separate anatomical groups, depending on a fortuitous re-attendance, are in no way an accurate census of the case incidence of each kind. Nonetheless, the figures do give

FIG. 2.—Anterior infarction. The T wave is low in lead I and inverted in CR4. In this and in subsequent figures CR leads are preferred to V leads, but the reader should know that they are interchangeable as far as this work is concerned. III R is the designation used for lead III recorded during deep inspiration.

FIG. 3.—Antero-lateral infarction. Inversion of the T wave in CR7 is added to the same change in leads I and CR4.

FIG. 4.—The inverted T wave in CR4 from anterior infarction in (A), has become upright in (B) which shows evidence of postero-medial infarction told by the appearance of Q waves in leads II and III and inversion of T wave in III.

some indication of the frequency with which each sector of the left ventricle is affected by cardiac infarction.

RESULTS

The changes taking place in the subsequent electrocardiograms and their effect on the initial changes, are to be considered for each of the four anatomical sectors. In that the sex of the patient has no importance in the analysis, it is not mentioned in the text, but the age is given as well as the period intervening between the recording of the two cardiograms.

Anterior Cardiac Infarction (TIT4 pattern)

There were 68 patients in this group. Their ages ranged from 42 to 76 years, and the mean age for the group was 57 years.

The period intervening between the recording of the initial and subsequent cardiogram varied from a few months to thirteen years, and the mean period was three years.

The cardiogram typifying this group showed either depression or actual inversion of the T wave in leads I and CR4 (Fig. 2). In 41 cases, the T wave was slightly depressed in CR7 as well (Fig. 3), indicating a small spread of the infarction into the lateral wall of the left ventricle, but in the remaining 27 cases, the lesion was strictly limited to the anterior sector. A material Q wave in leads I or CR4, namely one measuring 3 mm. or more, was present in 13 cases, a natural Q in 14, while in the remaining 41 cases the Q was absent.

Changes in Subsequent Cardiogram

The original deformity in the first cardiogram, conforming to a TIT4 pattern, when sought in the subsequent tracing, had remained stationary in 15 patients, had regressed in 26, showed a limited spread within its own sector in 7, and had extended to other sectors in 20 patients. In none had the cardiogram returned to normal (Table).

Contiguous Extension. In 8 of the 20 patients the infarct had spread into the lateral sector so that deformity of the T wave in CR7 either appeared for the first time or became exaggerated if it already existed, and the resulting cardiogram showed a TIT4T7 pattern. Although a spread into the septum was also a feature of some of these cases, with the appearance of Q waves in right-sided chest leads, this is not specially treated in this paper.

Antipodal Extension. In the remaining 12 of the 20 patients, the electrocardiographic changes told of involvement of an opposed sector of the left ventricle, representing an antipodal extension of the infarct and demonstrating a contrecoup or rebound effect.

In 10 of the 12 cases the sector that lay opposite the seat of the initial infarction in the antero-lateral sector was the postero-medial, and in 2 cases the postero-lateral.

When the *postero-medial* sector was the site of the more recent infarction, the QTIII pattern was expected to be added to the existing TIT4 pattern, but the union of these two produced a modified composite picture. Thus, the abnormality of the T wave in lead I either lessened or remained unchanged and it never increased. The deformed T in CR7 became more so. The most noticeable change was the recovery of the deformed T in CR4, so that the inverted wave became upright, and in 7 of the 10 cases it regained its normal height. This correction of T4 was regarded as following a contrecoup effect and not as a spontaneous recovery because of the constancy of the change in these 10 cases, some of which had shown no such recovery of the deformed T before the onset of the antipodal infarction.

In the final electrocardiogram the Q was always deep in lead III, and was present in lead II as well in 6 cases. The T wave in lead III, however, had remained upright in 4 of the 10 cases (Fig. 4 and 5).

When the *postero-lateral* sector with its TIIIT7 pattern became the seat of the antipodal infarction in the 2 instances of antero-lateral infarction, the T wave in lead III became inverted, the deformed T in CR7 became more so, while the T wave in lead I became less deformed, and especially so in lead CR4 (Fig. 6 and 7).

In the ultimate cardiogram, therefore, when infarction of the anterior section of the left ventricle is joined by infarction of the antipodal postero-medial or postero-lateral sectors, evidence of the anterior disposition of the lesion, namely a depressed T4 becomes eclipsed by this contrecoup effect, but in that the T wave deformity still persists in lead I, a lesion in the lateral sector remains recognizable.

Lateral Cardiac Infarction (TIT7 pattern)

There were 91 patients in this group. Their ages ranged from 40 to 81 years, and the mean age for the group was 59 years.

The period intervening between recording the initial and subsequent electrocardiogram varied from a few months to thirteen years, and the mean period was three years.

The cardiogram typifying this group showed either depression or inversion of the T wave in leads I and CR7, with depression of the S-T and T in IIIr (Fig. 8). In 32 cases the T was slightly depressed in CR4 as well, indicating a small spread of the infarct into the anterior sector of the left

FIG. 5.—The low T waves in leads I and CR4 from anterior infarction in (A) have become upright in (B) which shows Q waves and inverted T waves in leads II, III, and CR7 from postero-medial infarction.

FIG. 6.—The inverted T waves in leads I and CR4 from anterior infarction in (A), have become upright in (B) which shows deep inversion of T waves in leads II and III, and deeper inversion of T in lead CR7, from postero-lateral infarction.

FIG. 7.—Depression of the T wave in lead I and especially of the S-T segment in CR4 from anterior infarction in (A), has disappeared in (B) which shows inversion of the T wave in leads II, III, and CR7 from postero-lateral infarction.

ventricle. In the remaining 59 cases it was confined to the lateral sector. Thus, when the former group of anterior infarction is considered jointly with the present group of lateral infarction, the lesion is found to be confined to the anterior sector in 27, to the lateral sector in 59, and is disposed antero-laterally in 73 cases. A material Q wave was present in leads I or CR7 in 6 cases, a natural Q in 42, while Q was absent in 43 instances.

FIG. 8.—Lateral infarction. Inversion of the T wave in leads I and CR7 and depression of the S-T and T wave in III R.

FIG. 9.—Inversion of the T in lead I from antero-lateral infarction in (A), has given way to a flat T in (B) which shows Q waves and S-T depression in leads II, III, and CR7 from postero-medial infarction. The deep S waves in leads II and III in (A) are absent in (B).

FIG. 10.—Postero-lateral infarction. Inversion of T wave in leads III and III R, and a low T in II and CR7. An apparent Q wave in III is absent in III R.

Changes in Subsequent Cardiogram

The original deformity in the first cardiogram, indicating lateral or antero-lateral cardiac infarction, had remained stationary in the second tracing in 21, had regressed in 23, had shown a limited spread within its own sector in 20, and had extended into other sectors in 27 cases. In no case had the cardiogram recovered completely (Table).

Contiguous Extension. Among the 27 patients in whom further infarction had taken place in sectors other than the lateral sector that contained the initial infarct, it involved adjacent sectors in 23 cases, spreading into the anterior in 16, the postero-lateral in 6 cases, and into both sectors in one case. Thus, there was added to the TIT7 pattern, inversion of the T wave in lead CR4 when it spread anteriorly, and inversion of the T in lead III when it spread posteriorly.

It was possible, therefore, in the ultimate cardiogram to tell the distribution of the infarction, which might involve either two or three of the sectors simultaneously.

Antipodal Extension. In the remaining 4 of the 27 patients, additional changes in a subsequent cardiogram told of infarction in a contraposition to the lateral sector, namely the postero-medial sector. In this circumstance the abnormal T wave in lead I either remained the same or became more natural, and the T in CR7 became more negative. The Q which had appeared in lead III was deep in every case, with depression only of the T in the same lead in two cases and inversion in the other two. In lead II a deep Q wave developed in each of the four cases: in two of these it had been absent before the newer infarction set in, and in the other two it had been natural.

This contrecoup effect, whereby the QTIII pattern of the newly-formed infarct was added to the TIT7 pattern of the original lesion in the lateral sector, produced a cardiogram which made it possible to recognize the dual lesion, although some of the signs of antero-lateral infarction like a deep S wave in leads II and III had been submerged (Fig. 9).

Postero-lateral Cardiac Infarction (TIIIT7 pattern)

There were 36 patients in this group. Their ages ranged from 31 to 88 years, and the mean age for the group was 54 years. The period intervening between the recording of the initial and subsequent electrocardiogram varied from a few months to fourteen years, with a mean of three years. The cardiogram typifying this group showed depression or inversion of the T wave in leads III, IIIR, and CR7, without significant changes in other leads (Fig. 10).

A natural Q wave was present in lead II in 4 cases, in lead III in 3 cases, and in both leads in 4 cases. Q was absent from these leads in the remaining 25 cases, and in none was there a material Q wave. In CR7 a natural Q was present in 14 cases, and a material Q in one patient, while the wave was absent in the remaining 21 cases.

Changes in Subsequent Cardiogram

The deformity that characterized the initial cardiogram, giving rise to the TIIIT7 pattern, had remained stationary in the second tracing in 12 patients, had regressed in 10, showed a limited spread within its own sector in 2, and had extended into other sectors in 12 cases (Table). In no case had the cardiogram recovered completely.

Contiguous Extension. Among the 12 patients in whom further infarction had taken place in sectors other than the postero-lateral sector which contained the initial infarct, it involved the adjoining sectors in 9 cases, spreading forward into the lateral sector in 6, and to the right into the postero-medial sector in three. Thus, when it spread anteriorly a deformity of the T wave in lead I became added to that in leads III and CR7. When it spread to the right, material Q waves appeared in lead III, in addition to the inversion of the T waves already present in leads III and CR7.

Thus, when infarction spreads forward from the postero-lateral sector into the lateral its extended limits can be learnt cardiographically; but such interpretation is not possible when the lesion spreads into the postero-medial sector, unless a previous cardiogram has been recorded during a time when the infarct was contained within the postero-lateral sector.

Antipodal Extension. In the remaining 3 of the 12 patients with postero-lateral infarction, a

subsequent cardiogram indicated that infarction had taken place in the opposite or anterior sector, i.e. the antipodes. In this event the inverted T wave in lead III became upright and the T in CR7 remained or became more abnormal, while deformity of the T wave in leads I and CR4 became added (Fig. 11). Thus, this contrecoup effect obscured the cardiographic changes that told of the presence initially of infarction within the postero-lateral sector.

Postero-medial Cardiac Infarction (QTIII pattern)

There were 55 patients in this group. Their ages ranged from 37 to 89 years, and the mean age for the group was 58 years. The period intervening between the recording of the initial and subsequent electrocardiogram varied from a few months to fourteen years, with a mean of three years.

The cardiogram typifying this group showed a deep Q wave and inversion of the T wave in leads III and IIIR, along with a Q wave and depression of the T wave in lead II in most instances, while the T in CR7 was usually deformed as well (Fig. 12).

Dealing with the 55 patients in this series, a material Q wave, namely one exceeding 3 mm. in depth, was present in lead III in 52, and a natural Q in 3 patients. The T wave was always deformed in lead III, and with 5 exceptions it was inverted. Proof that such cardiographic abnormalities were significant was got by finding the same signs in lead IIIR. In lead II the Q was deep in 32, natural in 21, and absent in only 2 cases. The T wave in lead II was abnormal in 51 cases, being low in 29 and inverted in 22 cases; it was normal in only 4 cases. In lead CR7, a material Q was present in 14, a natural Q in 30, and Q was absent in 11 instances: the T wave in this lead was normal in only 5 cases, being low in 33 and inverted in 17 cases.

Changes in Subsequent Cardiogram.

The deformity characterizing postero-medial infarction in the initial cardiogram had remained stationary in a subsequent tracing in 28 patients, had regressed in 6, showed a limited spread within its own sector in 2, and had extended into another sector in 19 cases (Table). Not once had the cardiogram recovered completely.

It was not possible to tell whether a spread of the infarct had taken place into the adjoining postero-lateral sector in that its characteristic changes of a deformed T wave in leads III and CR7 were already present in the postero-medial variety.

Antipodal Extension. When the original infarct in the postero-medial sector had been joined by another in the antero-lateral sector in the 19 cases, its cardiographic pattern (QTIII) underwent a change. In 7 cases the cardiogram indicated that the newer infarct was situated mostly in the anterior sector with preponderant changes in leads I and CR4, and in the other 12 the lesion involved the lateral sector to a greater extent producing changes in lead I and in CR7 greater than in CR4.

The actual changes that modified the QTIII pattern of the original infarct in the postero-medial sector (Fig. 13, 14, 15, and 16) are now to be described.

In lead II a natural Q wave in 6 cases increased in its depth in 3 while a material Q in 13 cases remained so in 8, became natural in 3, and disappeared in 2 cases. The behaviour of the T wave in lead II was variable for it might be affected by both the initial and subsequent infarction.

In lead III a material Q wave persisted in 16 patients, in 9 of whom the T wave became upright; in the remaining 3 patients, in each of whom the T wave had become upright, the Q had returned to a natural depth. The inverted T wave in lead III had become upright in 12 out of the 19 but the presence of a material Q in these instances still made possible the diagnosis of postero-medial infarction. Whenever the T wave had moved upright it was usual for the S-T segment to be raised a millimetre or so. Once, both antipodal lesions were clearly indicated in the one tracing (Fig. 17). These changes did not take place in those patients in whom the infarct remained confined, even through many years, within the precincts of the postero-medial sector (Fig. 12), so that in the case of antipodal extension of the original lesion, the changes are not the outcome of a spontaneous resolution of the original tracing, but due to the direct impact on them of the cardiographic pattern peculiar to the newer lesion in the antero-lateral sector.

FIG. 11.—The inverted T wave in III with plane depression of S-T segment in CR7 in (A) indicates a limited infarction in the postero-lateral sector. The inverted T in III becomes upright in (B) which shows inversion of T in leads I, CR4, and CR7 together with a deep Q in CR4 from antero-septal and lateral infarction.

FIG. 12.—Postero-medial infarction. Q waves in leads II and III, inverted T in III and a depressed T in lead II, in both (A) and (B) recorded 10 years apart.

FIG. 13.—The inverted T waves in leads III and IIIr from postero-medial infarction in (A) have become upright in (B) which shows inversion of the T wave in leads I and CR7 from lateral infarction. The Q waves in III and IIIr have also shortened.

INTERPRETATION OF THE FINDINGS

The importance of the electrocardiogram in the recognition of myocardial disease cannot be exaggerated, and what it can accomplish in this field has not yet been plumbed, for it continues to contribute anew to our knowledge of occlusive coronary arterial disease and its effects.

Many years have elapsed since changes in the three limb leads were first ascribed to cardiac infarction, one kind of deformity being identified with infarction in the front and another with infarction at the back of the heart (Pardee, 1920). Later, Parkinson and Bedford (1928) described the changes that took place in these leads during the healing of the infarct.

FIG. 14.—The inverted T waves in leads III and III R from postero-medial infarction in (A) have become upright in (B) which shows inversion of the T wave in leads I and CR7 from lateral infarction. The Q waves in III and III R have also shortened.

FIG. 15.—Consecutive records (A), (B), and (C) show diminishing signs of postero-medial infarction as signs of antero-lateral infarction increase.

FIG. 16.—Signs of postero-medial infarction in (A) have regressed in (B) which shows changes from antero-lateral infarction.

With the introduction of chest leads (Wolferth and Wood, 1932), a more accurate localization of the lesion became possible, and thereafter several workers correlated the electrocardiographic changes in cases of cardiac infarction with anatomical findings at a subsequent necropsy (Whitten, 1930; Barnes and Ball, 1932; Barnes, 1935; Myers, Klein, and Stofer, 1948; Myers, Klein, and Hiratzka, 1949a, b, c, and d).

Resort to recording the electrocardiogram after exercise (Master, 1950) and the readier recognition of the lesser abnormal signs in the resting electrocardiogram in patients with cardiac pain (Evans and McRae, 1952), have reduced if not abolished true instances of cardiac infarction in which the cardiogram was once regarded as a normal tracing.

More recently, experience with material examined at necropsy has shown that the myocardial lesion from cardiac infarction is often more widespread than that suggested by the electrocardiogram. Thus Snow, Jones, and Daber (1955), following an examination of the myocardium and the coronary arteries by arteriography in 25 cases where a clinical diagnosis of cardiac infarction had been made, discovered 41 sites at which one of the three main coronary arteries had been occluded, along with 59 separate areas of infarction.

Recovery of a deformed T wave which often follows cardiac infarction is known, but a certain permanency has been attributed to material Q waves and depression of the S-T segment. In the past the distribution of such deformities among the separate cardiographic leads has made possible the localization of the infarct to one of the five cardiac sectors, namely the anterior, lateral, postero-lateral, postero-medial, and septal. A more ambitious role is now demanded of the cardiogram, namely that subsequent to the initial infarction it should indicate whether in the course of time spread of the infarction has taken place into sectors other than the one initially affected. In order to meet this newer function, there is need to know the effect on the cardiographic pattern of injury in one sector by the superimposition of changes caused by the extension of the infarct to other sectors of the left ventricle.

In 250 patients with cardiac infarction when subsequent cardiograms were examined alongside the initial tracing, no material change had taken place in 76, while a lessening of the deformity occurred in a further 65 patients. In 31, although the graph had worsened, it indicated that the infarct was still confined to the original sector.

A study of the remaining 78 patients in whom the increased deformities had taken place in a subsequent cardiogram, presumably indicating a spread of the infarct, has been of value in that the recognition of newer cardiographic patterns can indicate the limits of the composite infarcted area. The spread in 40 of the 78 patients had been into an adjacent sector of the left ventricle, while in the remaining 38, the newer infarct occupied an antipodal sector.

Contiguous Extension. It was possible to define the ultimate sectors of injury in the 40 cases where spread had taken place from one sector to an adjoining one, except in the case of postero-medial and postero-lateral infarction. Thus, a spread from the postero-medial into the postero-lateral sector was only discernible if the initial deformity consisted of a deep Q wave in lead III and inversion of the T in III, but with a normal T wave in lead CR7.

Moreover, a spread of an infarct from the postero-lateral into the postero-medial sector could not be inferred in the absence of a preliminary cardiogram which would place it in the former sector.

Antipodal Extension. The sectors of the left ventricle involved in the antipodal disposition of the infarct were the antero-lateral and postero-medial in 33, and the anterior and postero-lateral sectors in 5 patients. The influence that the introduction of the cardiographic changes from the extended infarction exerted on these changes contingent on the initial infarct may now be considered for each of these two combinations.

The Antero-lateral—Postero-medial Contrecoup. Among the 33 patients in whom serial electrocardiograms showed the presence of infarction in these two sectors, the first to be affected was the antero-lateral in 14 and the postero-medial in 19.

The composite electrocardiographic pattern for the conjoined injury in the two sectors was

FIG. 17.—Signs of postero-medial and antero-lateral infarction evident in the one record.

expected to be TIT4T7QTIII, but the effect of the postero-medial lesion was to correct the T4 deformity and to accentuate the T7 abnormality, while the introduction of the antero-lateral lesion partially or wholly corrected the deformed T in lead III, and sometimes shortened the Q wave to less than 3 mm. in the same lead, but never caused it to disappear.

These effects minimize the size of the anterior infarct as estimated cardiographically, permitting a diagnosis of the lateral infarct only. Moreover, although the recognition of the postero-medial infarct is still possible in the presence of a material Q wave in lead III, its shortening in some cases, along with correction of the inverted T wave in this lead in one-half the cases, often casts doubt on the presence of the postero-medial lesion. Occasionally, however, both contrapositioned infarcts may be indicated in a single cardiogram.

The Anterior—Postero-lateral Contrecoup. Of the 5 patients in this group the anterior sector was the first to show infarction in 2, and the postero-lateral sector in 3.

The composite electrocardiographic pattern for the conjoined sectors was expected to be TIT4TIII T7, but the impact of the postero-lateral lesion was to diminish the T wave deformity in leads I and CR4, and deform the T in CR7, while the introduction of the anterior lesion corrected the T wave inversion in lead III.

The result of this anterior—postero-lateral contrecoup effect, therefore, is to submerge all evidence of the postero-lateral lesion, and to minimize the anterior distribution of the infarct, connoting only its lateral position.

SUMMARY AND CONCLUSIONS

It is usual for the electrocardiographic prediction of the position of gross infarction to be confirmed at a subsequent examination of the heart at necropsy. Such localization is made in regard to some five sectors of the left ventricle, namely anterior, lateral, postero-lateral, postero-medial, and septal; the recognition of an extension of either the anterior or postero-medial infarction into the right ventricle, as may happen, is not yet feasible.

Not infrequently, however, the extent of infarction at necropsy has been greater than predicted by the cardiogram, so that although the tracing invariably tells of the presence of infarction, it sometimes fails to define its extent accurately. Thus, an infarct has been discovered in a sector of the heart other than the one correctly predicted by the cardiogram. Meanwhile therefore the tracing, although invaluable in diagnosis, does not give material assistance in prognosis, for it may fail to define an infarct in sectors other than the one containing the initial main infarct. Occasional experience had already shown me that such failure applied chiefly to infarcts that were situated in opposed or antipodal sectors, and not when the infarct extended into adjacent sectors.

In the belief that serial electrocardiography would throw light on the problem and show how further infarction, causing newer deformities in the tracing, might modify cardiographic changes identified with the initial infarction, I collected the notes of 250 patients whom I had seen with cardiac infarction and in whom successive electrocardiograms had been recorded during the course of the illness. Inspection of the initial electrocardiogram sited the infarct in the anterior sector of the left ventricle in 68, the lateral sector in 91, the postero-lateral sector in 36, and the postero-medial sector in 55 cases.

In subsequent cardiograms the changes were found to have remained stationary in 76, and to have regressed in 65 cases. In 31, although the deformities had increased, they indicated that the infarct was still confined to its original sector. In 78 cases, however, a subsequent cardiogram told of the spread of the infarct into another sector, involving an adjacent sector in 40, and an antipodal sector in 38 cases.

When the infarct had spread into an adjacent sector it was possible to judge its newer boundaries except in two circumstances. First, it was not possible to recognize a spread from the postero-medial to the postero-lateral sector, even when two cardiograms were available, unless the T wave in CR7 in the first tracing that showed a QTIII pattern was normal. Secondly, a spread from the

postero-lateral to the lateral sector could not be detected in the absence of two cardiograms and where the first lesion was seen to lie within the precincts of the former sector.

The infarct had extended into an *antipodal* sector in 38 cases when the ultimate cardiogram confirmed involvement of the antero-lateral and postero-medial sectors in 33 and of the anterior and postero-lateral sectors in 5 cases.

In the first variety the effect of the postero-medial infarction was to correct the inverted T4, and so destroy the evidence of the anterior lesion in half the cases; while the antero-lateral infarct in turn corrected the inverted T wave in lead III in one-half, and the deep Q wave in one-fifth of the cases, signs that signified the postero-medial infarction.

In the second variety the cardiographic signs of anterior infarction cancelled those of postero-lateral infarction, while the latter modified the signs of the former, leaving behind only evidence of infarction of the lateral sector.

This contrecoup effect, whereby the cardiographic pattern identified with infarction in one sector of the left ventricle influences materially the pattern belonging to a sector situated antipodally, calls for caution in pronouncing on the distribution of occlusive coronary disease and resulting cardiac infarction, unless a tracing recorded earlier in the illness has shown a lesion that at the time was confined to a single sector.

In the absence of this preliminary cardiogram, a knowledge of the design of the composite tracing that emerges from interaction of the electrical changes produced by two or more infarcts occupying either adjoining or antipodal sectors will help to define the limits of the extended myocardial injury.

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