Inferior myocardial infarction and right coronary artery occlusive disease

A correlative study

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The present study represents an attempt to correlate the electrocardiogram and coronary arteriogram in patients with an inferior transmural infarct – or total occlusion of the right coronary artery. The influence of the collateral circulation on these findings was also evaluated.

Fifty patients with a total occlusion of the right coronary artery had characteristic electrocardiographic changes of an inferior infarct in 44 per cent, very suspicious changes in 32 per cent, and no changes suggesting an inferior infarct in 24 per cent. However, in this latter group who had no evidence of an inferior infarct, we were able to recognize a small number who showed an anterior wall infarct.

Collateral circulation was more frequently present and more extensive in those patients whose electrocardiograms did not show changes typical of inferior transmural infarction. This suggested that collateral circulation might minimize some of the electrocardiographic abnormalities which would normally result from occlusive disease of the right coronary artery.

Another 50 patients, selected because of definite electrocardiographic evidence of typical inferior transmural infarction, were evaluated by coronary arteriography. Severe obstructive disease of the right coronary artery was present in 86 per cent of the group. In the remaining 7 patients (14%) minimal or no disease was found. Infarction of the inferior wall may have resulted from occlusive disease of the anterior descending artery or have been the result of a right coronary artery occlusion with subsequent recanalization.

We conclude from our study that a careful analysis of electrocardiographic abnormalities in the inferior leads will, with certain limitations, permit us to estimate the likelihood of a severe lesion in the right coronary artery, and, in the face of definite electrocardiographic evidence of an inferior infarct, to predict the diseased artery.

The electrocardiogram remains the principal method for identifying patients with coronary artery disease. Though coronary arteriography is a more definitive approach, the electrocardiogram is more useful because it is non-invasive and easy to carry out.

This study had two purposes. First, we wanted to evaluate a group of patients with arteriographic evidence of a total occlusion of the right coronary artery in order to determine what electrocardiographic changes might result from such a lesion. One important assumption was that, in the vast majority of these patients, an inferior infarct occurred as a result of the total occlusion of the

right coronary artery. In turn, we expected that a detailed analysis of these electrocardiographic abnormalities might enhance our ability to predict the presence of a right coronary artery occlusion from an interpretation of the electrocardiogram. As a corollary to this part of the study, we examined the influence of the collateral circulation in modifying the electrocardiographic abnormalities that are commonly associated with an inferior transmural infarction.

In the second part of the study, we approached the right coronary artery electrocardiographic correlation from an opposite direction and selected a group of patients, each of whose electrocardiogram showed an inferior transmural infarction. From this

group, we determined how frequently a right coronary artery occlusion was the lesion responsible for the inferior infarction and, if the right coronary artery was not diseased, what other coronary artery lesion produced the inferior infarction.

Subjects and methods

Two groups of patients were studied.

The first group (group A) consisted of 50 patients (47 male and 3 female). Patients were included in this group solely on the basis of a total right coronary artery occlusion found during coronary arteriography. In this group, a detailed analysis of the electrocardiogram was carried out to determine what changes had occurred, particularly in the inferior leads, as a result of this occlusion.

Electrocardiographic criteria which we considered clearly diagnostic of inferior infarction (Rose and Blackburn, 1968) were first a Q in aVF of at least 0.03 s and a depth equal to or exceeding 25 per cent of the amplitude of the following R wave with additional Q waves in leads II and III, or a Q₃ of at least 0.04 s with a depth equal to the following R wave and additional Q waves in leads II and aVF.

We also examined the electrocardiogram in detail in each patient for electrical axis, aberrations (low frequency notching or slurring) in the configuration of the QRS, particularly in the inferior leads, evidence of other infarcts or fascicular block, and presence of, as well as width and depth of, Q waves in the inferior leads.

In order to assess the specificity of the several patterns found, we also examined the electrocardiograms of 100 patients with normal coronary arteriograms who were undergoing diagnostic cardiac catheterization. The diagnoses within the control group included normal subjects as well as patients with valvular disease and primary myocardial disorders.

The second group (group B) consisted of another 50 patients (45 male and 5 female). Each patient was selected for entry into this group when a clearly diagnostic residual inferior wall transmural infarction (see group A above for criteria) was found on the electrocardiogram. In these 50 patients, we directed our attention to an analysis of the coronary arteriograms. The occurrence of obstructive lesions in each branch was tabulated and the severity of each lesion noted. The electrocardiograms were also examined for the presence of other infarcts.

All patients in the study underwent routine diagnostic coronary arteriography by the Sones technique; in addition, standard haemodynamic measurements and left ventriculography were carried out. These procedures have been outlined in detail in a previous report (Dwyer, 1970).

In each patient the site and severity of the right coronary artery obstruction was defined. The severity of the coronary artery lesion was graded from 0-5: 0=no occlusive disease, 1=luminal irregularities, 2=narrowing of 25 to 40 per cent, 3=50-70 per cent narrowing, 4=75-90 per cent narrowing, and 5=total occlusion.

The presence of collateral anastomosis from either or both the anterior descending or circumflex artery to the

distal right coronary was also determined. A $2 \times 2 \chi^2$ matrix test was used to test the significance of correlations between collateral supply and electrocardiographic changes.

Left ventriculograms in the right anterior oblique view were examined from each patient in the study. Analysis of the left ventriculograms was carried out in the patients with total occlusion of the right coronary artery. End-systolic and end-diastolic silhouettes were outlined and wall motion was analysed following establishment of the long axis (apex to mid-plane of mitral valve) and perpendiculars to the long axis at quarter-length intervals. Motion was recorded as percentage change of hemiaxes from end-diastole to end-systole.

In group A, the patients with electrocardiograms diagnostic of an inferior infarct and those with 'suspicious' or 'non-diagnostic' electrocardiograms showed 85 per cent and 83 per cent incidence, respectively, of inferior wall asynergy which we defined as akinetic, hypokinetic, or dyskinetic wall motion. To explain the occasional lack of correlation one might consider that in the patients with diagnostic electrocardiograms, wall motion abnormalities which occur on the posterolateral aspect of the left ventricle might not be appreciated by ventriculograms performed only in the right anterior oblique view: while in patients with 'non-diagnostic' electrocardiograms, the occluded right coronary artery may well have been a 'non-dominant' artery and infarction of the inferior wall never occurred. Unfortunately, once occlusion of the right coronary artery occurred we could not establish whether the vessel had been 'dominant' or 'non-dominant'. We consider that the high occurrence of inferior wall asynergy substantiates our assumption that, in the vast majority of cases, total occlusion of the right coronary artery implies an inferior wall infarction.

Clinical history and twelve-lead electrocardiograms were obtained in all patients on the day before coronary arteriography. Tracings were obtained on photographic paper by a Sanborn twin-beam recorder at a paper speed of 25 mm/s, with standardization adjusted to 1 cm per mv.

Results

Total occlusion of right coronary artery and associated electrocardiographic abnormalities (Table 1)

Diagnostic electrocardiogram Fifty patients with total right coronary artery occlusion were studied. Of the 50 patients, 22 had an electrocardiogram which we classified as diagnostic of an inferior transmural infarction. Twenty had a diagnostic Q in aVF and 2 others showed a diagnostic Q in lead III.

Suspicious electrocardiogram In the remaining 28 patients, either 'non-diagnostic' Q waves or initial R waves were seen in the inferior leads. In spite of the lack of Q waves diagnostic of an inferior infarction, we considered the patterns in 16 patients as highly suspicious. They include the following:

TABLE I *Electrocardiographic abnormalities in 50 patients with total occlusion of the right coronary artery*

No. of patients	Electrocardiogram
22	Diagnostic Q: Q aVF ≤ 0.03 s in duration, and depth = 25% of R wave amplitude; or QIII ≤ 0.04 s in duration, and depth = following R wave amplitude
8	Considerable splintering, notching, and slurring within Q of leads II, III, and/or aVF (Fig. 1)
4	Q in II, III, aVF with an electrical axis $\leq 30^\circ$ and a W configuration (Fig. 2)
4	'Embryonic' r in III, aVF with initial Q in II (Fig. 3)
5	Anterior wall infarction
7	Non-diagnostic

1) Eight patients showed conspicuous splintering, notching, or slurring within the Q wave of leads II, III, and aVF (Fig. 1). This abnormality was usually seen in all three leads. The frequency of these oscillations lies between 80 and 100 Hz. Such a distortion was seen within a Q wave in the inferior leads of only 1 of 100 patients with normal coronary arteriograms.

2) Four patients had an electrocardiogram in which the axis was less than 30° with a Q in III and aVF which resulted in a W-like configuration of the QRS complex in III and/or aVF (Fig. 2). This electrocardiographic pattern was rarely found (2 in 100) in patients with normal coronary arteriograms.

3) Four patients showed an 'embryonic' (≤ 1.0 mm) R in leads III and aVF, with an initial Q deflection in lead II (Fig. 3). The initial negative deflection in lead II is the important feature in the diagnosis of a residual inferior infarct, since an initial positive deflection (rS pattern) in lead II would indicate the presence of a left anterior hemiblock.

FIG. 1 An example of Q wave distortion in the inferior leads of a patient with total right coronary artery occlusion. Low frequency oscillations (100 Hz) are present on each lead. The rapid speed (50 cm/s) (right-hand trace) showed these changes in more detail.

FIG. 2 An initial Q wave in the inferior leads with an axis $\leq 30^\circ$ with a W-like configuration of the QRS complex. This pattern was observed in 4 patients with total occlusion of the right coronary artery.

FIG. 3 In this patient, the residual electrocardiographic abnormalities after total occlusion of the right coronary artery are 'embryonic' r in III and aVF and initial Q in II.

Non-diagnostic electrocardiogram

Twelve patients showed no evidence on the electrocardiogram of an inferior infarct. However, in five of those patients, the electrocardiogram showed evidence of an anterior infarct, which enabled us to make a diagnosis of coronary artery disease though an inferior infarction could not be recognized. In each of the five patients, the left ventricular angiogram demonstrated hypokinesis (less than 25% systolic inward wall motion) of the inferior wall, indicating a strong likelihood that an inferior infarction had, in fact, also occurred. In the other seven patients, no electrocardiographic evidence of either an inferior or anterior infarction was present.

In summary, from the entire group of 50 patients with total occlusion of the right coronary artery, 22 (44%) had diagnostic Q waves, 16 (32%) had electrocardiograms suggestive of an inferior infarct. The electrocardiograms of 5 patients, though non-diagnostic of inferior infarction, showed an anterior transmural infarct, while in 7 (14%) the electrocardiogram had no evidence of inferior or other infarcts.

Influence of collateral circulation on electrocardiogram

Collateral circulation to the occluded right coronary artery may arise from the left anterior descending artery, the circumflex artery, or, as we have seen in many instances, from both of those arteries. These collaterals might well influence the extent of the inferior infarction and the resulting electrocardiographic abnormalities. The collateral circulation was evaluated in two ways. First, were collaterals present? Secondly, were collaterals arising from one or two of the major branches of the left coronary artery? This evaluation was made and the 22 patients with electrocardiograms considered diagnostic of an inferior infarct were compared with the 28 patients whose electrocardiograms were considered only suspicious or non-diagnostic of inferior infarction.

Those patients whose electrocardiograms were

not diagnostic of a residual inferior infarction (in spite of a total occlusion of the right coronary artery) showed an increased presence as well as an increased number of visible collateral vessels. In those patients, 86 per cent showed collateral anastomosis to the occluded right coronary artery. By comparison, patients with definite infarcts on the electrocardiogram showed a somewhat lower incidence (72%) of collateral channels. This difference was not statistically significant. There was, however, a significant difference ($P < 0.01$) in the presence of dual collateral channels as those patients with less characteristic electrocardiograms showed anastomoses from both the anterior descending and circumflex artery (13 of the 28 patients) while only 3 of 22 patients with diagnostic electrocardiographic evidence of an inferior infarct had a dual collateral system.

Inferior transmural infarction and associated arteriographic lesions (Table 2)

Fifty patients with an inferior transmural infarct on the electrocardiogram were studied. The arteriographic findings in these patients were subjected to a detailed analysis. The following presents an arbitrary division of those 50 patients based on the degree of disease found in the right coronary artery. 1) **Total obstruction of right coronary artery** (grade 5) was found in 23 of 50 patients (46%). Although significant ($> 50\%$) obstruction in the anterior descending or circumflex artery (or both) was also seen in nearly all these patients (19/21), we found evidence of anterior transmural infarction in only four of the group.

2) **Severe disease of right coronary artery** (grade 3, 4) was present in 20 of 50 patients (40%) with a transmural inferior infarction. The right coronary artery was narrowed 75 to 90 per cent in 13 patients and 50 to 70 per cent in the remaining 7. In each of those 7 patients with a 50 to 70 per cent narrowing of the right coronary artery, severe narrowing in the anterior descending artery was also present. This raised the possibility that, in these

TABLE 2 *Arteriographic lesions in patients with transmural inferior infarction*

No. of patients	Coronary arteriogram
23	Total occlusion of right coronary artery
13	75 to 90% narrowing of right coronary artery
7	50 to 70% narrowing of right coronary artery ($> 75\%$ obstruction of LAD was present in all 7)
7	Minimal or no disease in right coronary artery ($> 75\%$ obstruction of the LAD)

LAD = left anterior descending artery.

patients, infarction of the inferior wall might have occurred as the result of critically reduced flow in the anterior descending artery rather than the right coronary artery.

3) **Minimal or no disease of right coronary artery** (grade 0, 1, and 2) was found in 7 patients (14%). In each patient the right coronary artery was a dominant vessel. Five of these 7 patients had a normal right coronary artery or showed luminal irregularities, while 2 had only a 25 to 40 per cent obstruction of the right coronary artery. Each of these was considered unlikely to be severe enough to result in an infarction of the inferior wall. The fact that infarction had occurred suggested two possibilities. One was that the right coronary artery had been occluded at the time of infarction and recanalized at the time of angiography. The second possibility is that a portion of the inferior wall was infarcted secondary to disease of the anterior descending artery. The latter explanation is made most probable by the observation that in each of the 7 patients, greater than 70 per cent obstruction was found in the anterior descending artery and an associated anterior infarction was found in 5 of those 7 patients.

Discussion

The purpose of this study was to investigate a specific subgroup of patients with significant coronary artery disease (i.e. total occlusion of the right coronary artery and inferior transmural infarction). From this subgroup, our purpose was 1) to determine, and possibly improve, the accuracy of the electrocardiogram in identifying severe disease of the right coronary artery, and 2) to determine, when an unequivocal transmural inferior infarction was present on the electrocardiogram, what were the location and severity of the coronary artery lesions.

The certain identification of patients with coronary artery disease by a simple, inexpensive, and accurate method is, of course, desirable. The electrocardiogram meets the requirements of simplicity and low cost but questions have been raised about the accuracy of the electrocardiogram in the diagnosis of coronary artery disease (Hilsenrath *et al.*, 1972). The large number of unexpected sudden deaths caused by coronary artery disease (Kuller, Lilienfeld, and Fisher, 1966) has accentuated the need for a reliable method to identify patients with coronary disease. From necropsy records of patients who have died suddenly (Kuller, Cooper, and Perper, 1972) and extensive epidemiological studies (Weinberg and Helpert, 1959), it appears that many patients with extensive and severe coronary disease die without prior knowledge of their disease.

Earlier studies (Pardee, 1930; Bayley, 1939) established the association of an abnormal QIII with an inferior wall infarction. Unfortunately, a significant number of clinically normal patients showed an abnormal QIII (Graybiel *et al.*, 1944). Addition of the augmented unipolar leg lead (aVF) (Goldberger, 1943) provided some help in the differentiation of the normal subject from the patient with a residual inferior infarction. Since Pardee's initial description of the QIII in inferior infarction, criteria have been based on only clinical and necropsy correlations. Such studies have obvious inherent shortcomings. Selective coronary arteriography and left ventriculography provide a fairly precise cardiac anatomical diagnosis which purely clinical correlations cannot provide. Studies using arteriography permit a more temporally appropriate correlation with the electrocardiogram, lack of which is frequently a shortcoming in necropsy studies.

Total occlusion of right coronary artery

Electrocardiograms of patients with total occlusion of the right coronary artery were analysed with the assumption that some degree of inferior wall infarction had occurred in all the group and should be reflected in each electrocardiogram. We do recognize that there may be a small number in this group in which an infarction has not occurred and this may be an additional factor in explaining the absence of an infarction pattern in some. Forty-four per cent (22 of 50) of those patients showed a classic inferior infarction pattern. In the 28 patients who did not have diagnostic Q waves, we found that, if certain other changes were taken into account, 16 additional electrocardiograms could be considered highly suspicious. From the remaining 12 patients the electrocardiogram gave no clue to the presence of an inferior infarction; however, in 5 patients the presence of coronary artery disease could be detected as each had an anterior wall infarction.

Some of these suspicious features have been alluded to or described by previous authors. Our intention in this report is to re-emphasize some of these characteristic alterations observed previously and to suggest their addition to older established criteria which might improve the diagnostic yield.

The presence of 'embryonic' r waves in leads III and aVF is not an uncommon finding after an inferior infarction. Hoffman, Taymor, and Gootnick (1964), in vectorcardiographic studies, confirmed this observation and emphasized the unreliability of the 0.01 s vector in diagnosing inferior infarction. However, since the rotational characteristics of the 0.02 s and later vectors are superior and clockwise after an inferior infarction, an initial Q deflection in lead II of the electrocardiogram can usually be

found which provides the clue to the diagnosis of residual inferior infarction.

Pronounced notching and slurring *within the* 'non-diagnostic' Q waves of leads II, III, and aVF was a common alteration after right coronary artery occlusion. The frequencies of these oscillations seen in our records are of relatively low frequency and lie between 80 and 100 Hz. Since the finding is rare in normal subjects, an electrocardiogram with such distortions should be viewed as highly suspect for a residual inferior infarction. This observation does not imply that when such notching or slurring is seen in later parts of the QRS complex or in other leads, that an infarction is present.

The number of patients in which Q wave notching and slurring can be appreciated is partly related to the quality of the recording equipment. We realize that such small amplitude deflections may be significantly attenuated by direct writers with a poor frequency response. Though our electrocardiograms were performed with optical equipment with an extended fidelity response, similar observations should be possible with current electrocardiographic equipment which adheres to recently formulated standards (Report of Committee on Electrocardiography, American Heart Association, 1967).

High frequency oscillations in the electrocardiogram have been studied by several investigators and correlated with clinical diagnoses (Langner, 1953; Flowers and Horan, 1971). However, to our knowledge, there has been no previous attempt to use low frequency oscillations as a diagnostic aid.

Finally, a small number of the group showed associated findings which when seen in combination should alert one to the possibility of a residual inferior infarct. These included 'non-diagnostic' Q waves in II, III, and aVF, an axis less than 30°, and a W-shaped configuration of the QRS in lead III and/or aVF.

Collateral circulation

Although it is well established that collateral vessels have little effect on the patient's ultimate prognosis, our results suggest that both the presence and the number of collateral anastomoses influence the configuration of the electrocardiogram after total occlusion of the right coronary artery. In the patients with diagnostic inferior infarction patterns, collateral anastomoses were seen less frequently and rarely had a dual collateral supply (from both anterior descending and circumflex artery), whereas those patients with more extensive collateral anastomoses had electrocardiograms far less diagnostic of an inferior infarct. Martinez-Rios *et al.* (1970) re-

ported a group of patients with normal electrocardiograms in spite of the presence of severe coronary artery disease, and reached a similar conclusion that the presence of extensive collateral circulation might be an important factor in minimizing the electrocardiographic changes.

Correlation of transmural inferior infarction and arteriographic lesions

When the electrocardiogram shows a transmural infarction, diagnosed by conventional criteria, certain conclusions can be drawn regarding the site and severity of the obstruction. The right coronary artery is significantly diseased in 86 per cent of the patients while in the remaining 14 per cent the inferior infarction occurs in the presence of a right coronary artery with little or no disease. It is possible that an anterior descending artery lesion results in an inferior infarct. This can probably be best explained by the fact that the anterior descending artery frequently 'wraps around the apex' and supplies a significant portion of the inferior wall. These patients frequently have an anterior wall infarct in addition. Unfortunately in these patients, the electrocardiographic findings suggest significantly more coronary disease than is indeed present. Williams *et al.* (1973), in a recent correlative study of arteriographic and ventriculographic abnormalities in all types of transmural infarction, observed similar arteriographic findings in patients with inferior transmural infarction.

Electrocardiographic evidence of an inferior infarct in patients with a normal or minimally diseased right coronary artery may also be explained by occlusion of the right coronary artery with subsequent recanalization of that vessel. Bruschke *et al.* (1971) reported a series of patients in whom angiographic studies at the time of infarction showed severe narrowings or occlusions of the coronary arteries which on subsequent arteriographic examination were found to be normal.

Our study delineates the extent of confidence one may have in predicting right coronary artery lesions from electrocardiographic changes in the inferior leads. In evaluation of the patients with coronary artery disease, electrocardiographic-angiographic correlations of this type have greater value and validity than earlier studies which have attempted to correlate electrocardiographic abnormalities with findings at necropsy. In addition, we have emphasized several electrocardiographic changes which are not characteristic but which should be considered as highly suggestive of a residual inferior infarct and severe right coronary artery disease.

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