Electrocardiogram and Vectorcardiogram
in Myocardial Infarction

By Harold D. Levine, M.D., Eliot Young, M.D.,
and Richard A. Williams, M.D.

By the time the patient with acute myocardial infarction is first examined, his electrocardiogram may already show changes in the RS-T segment alone, in the T wave alone, in the QRS complex alone, or in any combination of the three. Changes in any of these portions of the ventricular complex and particularly in the QRS may not appear for hours or days. Some never show distinctive electrocardiographic changes; in these the diagnosis must rest upon other than electrocardiographic evidence. The changes in the QRS complex generally long outlast the changes in RS-T and T and ordinarily constitute the telltale evidence of previous infarction for the remainder of the patient’s life.

Myocardial Infarction in Its Earliest Stages

A substantial experience with the very earliest electrocardiographic evidence of acute myocardial infarction is still lacking. There is morphologic evidence that most infarcts begin in the subendocardium and only later develop transmural “breakthrough”; an electrocardiographic counterpart at this time would be conjectural and extrapolated. One might expect in strategically located leads depression of RS-T segments outlasting simple nonsustained ischemia. In one laboratory study¹ the very earliest sequence appeared to be T-wave inversion (“ischemic”) followed by progressive elevation of RS-T segments, and the development of upright T waves of increasing height. Experience elsewhere with nonanesthesitized closed-chest dogs² indicated, to the contrary, that the earliest change was an increase in positive T-wave voltage, a change more closely approximating the very early stages of human myocardial infarction. In the clinic the earliest change to be recorded appears often to occur in the T wave, which becomes tall and peaked³ (“hyperacute”) resembling the T wave of potassium intoxication⁴ and possibly reflecting alterations in potassium distribution about a damaged area of myocardium.⁵ The T wave may then, but sometimes initially, become inverted. As the process of infarction continues, changes occur in the QRS complex. Broad (0.04 sec or longer) and prominent Q waves appear, producing QS or QR complexes where Q waves are not normally seen, and in the same leads as, or in leads contiguous to, those showing RS-T and T-wave changes. In the clinic as in the laboratory the T wave may demonstrate a gigantic waxing and waning in amplitude with sharp peaked inversion resembling the candelike T waves observed in disturbances in the central nervous system.⁶ ⁷

Subendocardial and Nontransmural Infarction

The difficulty of diagnosing acute myocardial infarction from changes in the RS-T segment and T wave in the absence of changes in the QRS complex is well recognized.⁶ ⁸ ⁹ Though changes in RS-T and T may occur in the absence of myocardial infarction, when they are associated with a suggestive history and with laboratory evidence of tissue necrosis, the case for actual myocardial infarction is strengthened. For some reason it has become common practice
to interpret tracings showing T-wave changes as suggesting an intramural site for the infarction. In many of these cases in which a QRS of normal appearance is recorded by the ECG, the VCG shows a markedly abnormal QRS (vide infra) indicating that transmural infarction has indeed occurred; this experience highlights one limitation of the ECG.

There has been little enough pathologic study of subendocardial infarction, practically none of intramural infarction as such. From considerations to be presented shortly, however, a subendocardial rather than an intramural location of the process would seem a much more likely explanation for infarction with T-wave changes only. According to the concept of electrical activation of the ventricular wall elaborated by the Ann Arbor school, significant Q waves would be expected in the epicardium overlying a subendocardial infarct. However, in a limited pathologic experience, such changes either have not been observed or, if observed, were small and unimpressive. Instead, generally there have been recorded in many or most precordial leads depression of RS-T segments with upright or inverted T waves but no Q waves; RS-T was elevated in lead aVR. At the Mayo Clinic deeply inverted T waves, in the absence of other electrocardiographic changes, were found to be evidence for subendocardial infarct. On the other hand, six large nontransmural infarcts extending from one half to three quarters of the distance from endocardium to epicardium were associated with RS-T and QRS changes commonly seen in transmural infarction. These observations are in line with the experimental and clinical observations of Prinzmetal and his co-workers suggesting a different manner of activation of the ventricular wall. This group considers that the R wave is the result of electrical activation of the outer layers of the heart, that the subendocardium is sluggish in generating injury potentials, and that functional alterations in the outer layers may be responsible for the RS-T and T-wave changes commonly observed in subendocardial injury. Finally they conclude that, in the face of clinical evidence of profound myocardial damage, the presence of a normal or almost normal electrocardiogram bespeaks a subendocardial location of the lesion, adding that RS-T depression and T inversion, when present, only lend support to that diagnosis.

The papillary muscles are made up of muscle bundles continuous with the subendocardial laminae of the ventricular wall, and they may participate with the mural endocardial layers in the process of infarction. The syndrome of necrosis of the papillary muscles described by Phillips, DePasquale, and Burch consists, in addition to the development of a diamond-shaped murmur, of certain electrocardiographic features, including changes in the U wave. The genesis of the U wave is still uncertain. During investigation of the cavitary electrogram in this laboratory enormous U waves were recorded near the apex of the right ventricle. We were puzzled about the significance of this finding but failed to follow it up. Hoffman and Cranefield have suggested that the U wave represents repolarization of the ventricular Purkinje system; in effect the U wave is the Purkinje fiber T wave. The order of depolarization of the ventricular wall is from endocardium to epicardium, of repolarization from epicardium to endocardium; as part and parcel of the subendocardium, the papillary muscles would be the last part of the ventricular myocardium to be repolarized. Prominent upright or inverted U waves are not rarely observed in acute myocardial infarction. In a more recent study, in which papillary muscle necrosis was largely associated with transmural infarction of the free wall of the left ventricle, it seemed doubtful that the electrocardiogram might selectively demonstrate ischemia or infarction of the papillary muscles. As Phillips, DePasquale, and Burch indicate, "the significance of these changes in light of some evidence relating U wave potential to repolarization of the papillary muscles remains to be clarified."

**Infarction of the Cardiac Atria**

Atrial infarction is said to occur in 1–17% of infarcts. This diagnosis is generally missed
ECG AND VCG IN MI

clinically and made only after postmortem examination by an interested pathologist. For a complete bibliography on this subject the reader is referred to the texts of Friedberg and Zimmerman. The rather poor yield of correct antemortem electrocardiographic diagnoses may be explained in part by widespread disinterest in this complication and in part by the difficulty in recognizing its presence in the ECG particularly when concomitant ventricular infarct is associated with an RS-T current of injury. The cardinal feature of atrial infarction is an alteration and displacement of the P-Ta segment, which represents atrial systole. Its vector is normally oriented superiority, rightward, and slightly posteriorly. The P-Ta segment, comprising the interval between the onset of the P wave and the end of the Ta (atrial repolarization) wave, includes the P-R segment. Its duration ranges from 0.15 to 0.45 sec. Excepting this isolated P-R segment, P-Ta is overlapped and obscured by the Q-T interval of the ventricular electrocardiogram. For all practical purposes the P-R segment is therefore the only part of the electrocardiogram in which gross changes may be detected in atrial infarction, although there have been reports of “auricular q waves” preceding the P wave in such cases. The P-R segment may normally show a slight downward displacement in lead II amounting to as much as 0.8 mm (-0.08 mv); this shift may increase in direct proportion to the heart rate and the P-wave area. Just as ventricular infarct may induce a “current of injury” with shift of the RS-T segment, so may atrial infarct induce a similar “current of injury” with displacement of P-Ta (fig. 1). This usually depresses P-R in leads II, III, and aVf, and often in leads V1 and V2. Criteria based upon clinical studies have been proposed by Liu and associates.

Atrial infarction should also be suspected when ventricular infarction is complicated by atrial arrhythmias or by atrioventricular block. Cushing found abnormal atrial mechanisms in 74% of patients showing both atrial and ventricular infarcts at postmortem, but in only 9% of cases with “pure” ventricular infarcts. On the basis of clinical experience Lown feels that atrial arrhythmias occurring during acute myocardial infarction are generally caused by “pump failure” and only rarely by atrial infarction; there is as yet no adequate postmortem substantiation for this belief. Any type of atrial arrhythmia may be seen, as well as second- or third-degree atrioventricular block. The latter facilitates recognition of an abnormally displaced P-Ta segment by bringing it out into the open where it may be seen in its entirety unobscured by the QRS complex.

Other alterations in P-wave morphology during acute myocardial infarction such as transient tall, peaked P waves in leads I, II, or III, or M-shaped, W-shaped, irregular, or notched atrial deflections, may suggest occult atrial infarction even in the absence of more substantial supporting evidence.

The right atrium, much more frequently than the left, is the site of infarction and rupture. Atrial mural thrombus formation, another complication, appears to be at least as common as thrombus formation in the ventricular chambers in ventricular infarction, occurring in up to 84% of cases. That pulmonary thromboembolism occurs in 24% of patients

![Figure 1](image)

Atrial current of injury. (A) Extremity leads on 10/14/69. (B) Same on 10/17/69, one day after open-heart surgery. Tracing shows sinus tachycardia (rate 120) and 2-mm depression of the P-Ta (P-R) segment in leads II, III, and aVf.

Circulation. Volume XLV, February 1972
with atrial infarction heightens the significance of this observation. Clearly, atrial infarction is not a development of mere academic interest. Its recognition should be regarded as a possible cue to certain forms of prophylaxis or therapy.

**Electrocardiogram and Vectorcardiogram**

Resting upon the recognition of concomitant changes in QRS, RS-T, and T of the ventricular complex, the electrocardiogram, in the experience of most observers, is quite accurate in the diagnosis of acute myocardial infarction. Thus there has been no great demand for a more accurate technique in the acute process. The QRS is judged “by the company it keeps,” i.e. RS-T and T. However, the situation may be quite different in old as well as acute myocardial infarction when RS-T and T-wave changes are not evident. Here electrocardiographic diagnosis, based solely on QRS, is subject to considerable error. To enhance the accuracy of their efforts and, in particular, to delineate changes in QRS more clearly, cardiographers have therefore seized upon the newer technique of vectorcardiography.

*Figure 2*

VCG pattern of inferior infarct in the presence of early R waves in leads III and aVF. (Top) ECG of 10/17/66. Inferior infarct might be overlooked because of small r waves in leads III and aVF. (Middle) VCG, recorded at same time as the top ECG, is diagnostic of inferior infarct. Frontal-plane loop shows an initial small inferior deflection of 0.008 sec. There then follows a superior, clockwise-directed efferent limb of 0.028-sec duration and 1.1-mv deviation to the left. Since most of the loop is clockwise, the marked left axis in the top ECG is due to the infarct and not to left anterior hemiblock. (Bottom) ECG of 5/22/65 showing definitive changes of acute inferior infarct. Second-degree A-V block is also present.

*Circulation, Volume XLV, February 1972*
Figure 3

Innocuous-appearing Q waves in vectorcardiographically demonstrated inferior infarct. (Top) ECG showing unimpressive Q waves in II and aVF (1 mm deep and 0.02 sec in duration in latter). (Bottom) Diagnostic frontal-plane VCG, recorded at same time, showing early superior clockwise forces lasting 0.026 sec, deviated 0.3 mv leftward, and displaying upward-bowed convexity. Complete obstruction of the main right coronary artery and hypokinesis of inferior wall demonstrated by angiography.

The electrocardiogram and the vectorcardiogram differ in three respects: frequency response, manner of recording, and manner of displaying the electrical activity of the heart.

It is now common experience that the VCG may show QRS changes diagnostic of myocardial infarction when the ECG is normal or shows only nonspecific changes.26-41 It is our purpose here to call attention to those situations in which the VCG may prove particularly helpful in the diagnosis, focusing attention upon those planes generally most useful for a specific diagnosis, e.g. the horizontal plane for anterior or posterobasal myocardial infarct, the frontal plane for inferior infarct. All VCGs here illustrated were obtained with the Frank leads. Except for omissions of time marks, these are exact tracings of the original loops.

Inferior Infarction

The most widely held criteria for the diagnosis of inferior infarction include early 0.025-sec vectors, which in the frontal plane are superior to the null point and which rotate in a clockwise direction.38, 39 These criteria are valid even when the very earliest forces are inferior.39, 40 The VCG can thus provide definitive evidence of inferior infarction when the ECG shows an initial R wave in lead aVF,39, 40 (fig. 2), a finding commonly taught as excluding that diagnosis.

In at least 20% of inferior infarctions the duration of early superior forces is less than 0.025 sec;40 on the other hand about 5% of normal subjects may show early superior forces lasting 0.025 sec or longer,37, 40 even up to 0.032 sec.40, 42 In view of this overlap other criteria including leftward deviation of the early superior vectors have also been emphasized.39, 40 In one report, in which a point-to-point method of analysis was utilized, a characteristic shape with an upward bowed convexity was described40 and the conclusion drawn that inferior infarction may be diagnosed if the early superior forces followed a clockwise direction, were deviated 0.25 mv or more leftward, and if they displayed this characteristic shape, even if their total duration was as short as 0.02 sec (Frank system). If on the other hand these early superior forces were preceded by initial inferior forces, the total duration of early superior forces must measure 0.025 sec or more to justify this diagnosis. The diagnosis was correct in 90 of 100 cases; there was no overlap with normal subjects. Only 42% of these 100 cases could be diagnosed on the basis of most currently used electrocardiographic criteria. In many instances in which the VCG was diagnostic a Q wave was barely discernible in lead aVF (fig. 3).

Hugenholtz et al.,38 using the Frank system, concluded that clockwise superior forces are diagnostic of inferior infarct even when they
last less than 0.025 sec, assuming that the maximal frontal-plane vector is superior to (less than) +20°. Chou and Helm, on the other hand, subsequently found that 10% of normal subjects over 40 years of age fulfilled this criterion.37 This discrepancy may be explained by differences in lead placement, the earlier observers placing the Frank chest electrodes in the fifth interspace, the more recent observers utilizing the fourth interspace. As already anticipated,40 it would seem that the more rigid restriction of the maximal vector to the area superior to (less than) +10° would validate this blanket rule regarding clockwise rotation, provided the very earliest forces are not inferior.

In an attempt to quantify morphologic VCG description, four types of mid-to-late frontal-plane QRS changes have been formulated for inferior myocardial infarct.41 Thus, 90% of the infarcts had mid-to-late inferior and leftward (in quadrant I) forces showing the following.

I. Type A: (1) The mid-to-late inferior and leftward forces are completely or almost completely clockwise (90% of inferior infarcts). (2) The distance from the most leftward point A to the lowest point B where the loop begins its final ascent to the 0 point exceeds 45% of the distance from A to the 0 point; i.e., \( \frac{A \rightarrow B}{A \rightarrow 0} > 45\% \) (about 33 1/3% of inferior infarcts). Some normal subjects who satisfied these criteria could be distinguished by one or more of the following: (a) maximal vector between +60° and +95°; (b) a convexity-to-the-right deformity of the descending quadrant I forces to the maximal leftward point A; and (c) unusual early forces with initial inferior followed by slightly superior clockwise vectors.

II. Type B (about 12% of inferior infarcts): Prominent upward bowed convexity of clockwise quadrant I vectors.

III. Type C (about 15% of inferior infarcts): An inverted V-shaped localized sharp reversal to a downward direction of the ascending afferent limb in quadrant I.

IV. Type D (6% of inferior infarcts): A figure-of-eight configuration of inferior forces in quadrant I with the afferent limb clockwise and a counterclockwise efferent limb, the latter directed partially downward early in its course. In many of these cases such mid-to-late changes were detected even when the early superior vectors, as well as the ECG, were normal.

Type A resembled left posterior hemiblock as described by Rosenbaum.43 These mid-to-late forces were “wide open” and directed clockwise. Complete left posterior hemiblock is reported as uncommon in inferior infarct;43 it would seem possible however that many instances of type A deformity represent incomplete conduction delay in this fascicle.

Anterior Infarction

In classical “isolated” anteroseptal infarction only the initial 0.02 sec of the curve is altered. In contrast to normal subjects in whom all of
VCG pattern of anterior infarct in the presence of early R waves in V₁–V₆. (Top) ECG shows, as the only suggestion of an infarct, a marked notch on the upstroke of R in V₃, ordinarily discounted when present at "transitional zone." (Bottom) Diagnostic horizontal-plane VCG recorded at same time. Although the early 0.026-sec vectors are anterior and therefore well within the normal range, the latter part of the efferent limb shows a diagnostic forward-directed concavity ("bite"). Angiography showed extensive three-vessel obstructive disease and general hypokinesis.

The early 0.02-sec vectors in the horizontal plane are anterior, the initial portion of the loop is posterior and usually leftward. This part of the curve may even show a concavity directed forward. The vectors that follow immediately may be located in a normal anterior position. The remaining portion of the loop then follows an entirely normal posterior course. In some instances of anteroseptal infarction only some of the early 0.02-sec vectors are posterior. In many of these the early forces, though rightward, show minimal posterior deviation but they may be recognized as distinctly abnormal from their clockwise rotation. Furthermore, even before the 0.02-sec vector is written these forces may become normal (i.e. anterior). It is particularly in cases of this type that the VCG, displaying a very circumscribed abnormality, may be diagnostic when the ECG is normal (fig. 4). (Young E, et al.: Unpublished observations.)

Initial vectors oriented to the left do not necessarily indicate septal infarction or fibrosis, nor need the normal VCG show initial rightward forces. By the same token the ECGs of normal individuals may show no "septal" Q waves in leads I, V₃–V₆, the absence of Q waves in these leads therefore does not necessarily indicate incomplete left bundle-branch block or septal infarction or fibrosis. Nevertheless, these should be considered normal variants only if the early 0.02-sec vectors are anterior and the rest of the loop is normal.

In localized anterior infarct the instantaneous 0.02-sec vector usually lies posterior to the null point. Moreover, the 0.04-sec and maximal vectors are often more posterior than normal. Although the early 0.01-sec vector lies in a normal rightward and anterior position, the latter part of these early rightward forces may lie posterior to the null point and rotate in a clockwise direction. The succeeding leftward forces may also follow an abnormal course, perhaps figure-of-eight, perhaps clockwise. In some instances, though the leftward and posterior efferent limb may show a normal counterclockwise direction, this salient may be indented by a diagnostic concavity which is directed forward. The presence of such a "bite" when the 0.02-sec vector is hardly or not at all posterior may be the only definite evidence of infarction. (Young E, et al.: Unpublished observations.) The ECG is frequently normal in these cases (fig. 5).

The position of the 0.02-sec vector is extremely helpful in differentiating the normal from anterior infarction. Contrary to an opinion expressed recently, however, the position of the 0.02-sec vector appears to be of little if any help in differentiating anterior infarction from left ventricular hypertrophy.
Figure 6

Vectocardiographic substantiation of anterolateral infarct suspected from electrocardiogram. (Top) ECG shows no significant Q waves in V1=V6. An rs' and negative T wave in V1, and diminishing r wave from V5 to V6 are suggestive of infarct. (Bottom) Diagnostic horizontal-plane VCG, recorded at same time, is almost completely clockwise and to the right. The 0.02-sec vector is to the right of and just anterior to the null point.

(LVH). In fact, some cases of LVH may even resemble anterior infarction in showing complete clockwise rotation in the horizontal plane. In a recent attempt to solve this problem in patients showing QS complexes over the right precordium attention was focused upon the scalar voltages recorded by Frank vectorcardiographic leads. It was found that, if R in lead X equaled or exceeded 1.2 mv, LVH was correctly diagnosed in 56% of cases but that myocardial infarct was incorrectly diagnosed in 10%. If the sum total of R in leads X and Y equaled or exceeded 2.5 mv, LVH was correctly diagnosed in 66% but the yield of false-positive diagnoses of myocardial infarction was increased to 12%. Clearly there is still a great need for new ECG and VCG criteria to permit a more accurate differentiation of left ventricular hypertrophy from anterior infarct.

Anterolateral infarct may show early rightward forces exceeding 0.022 sec in duration. According to the size of the infarct and the precise timing of its vectors within the QRS cycle, the loop may be markedly abnormal because it is inscribed in a completely clockwise direction and located in the midline close to the +90° to +270° axis in the horizontal plane. In some of these cases, because of minimal rightward deviation of these early forces, Q waves may be insignificant or absent in the ECG. The only clue that the QRS has been significantly altered may be “poor R-wave progression” or diminished height of the R wave in the left precordial leads. It is especially in this type of infarct in which the only obvious ECG abnormality is in the T waves that the term “intramural” infarct is often erroneously applied (fig. 6).

It is commonly taught that an infarct located high on the left lateral wall of the heart should show, as a clue, a broad, prominent Q wave in lead aV1, and, in corroboration, similar changes in special leads higher on the left precordium than the usual site of application. We have seldom been able to make the diagnosis in this way. We would agree with Massie and Walsh that a “significant” Q wave and a negative T wave may be observed in lead aV1, in normal individuals presumably because of an unusual heart position. Although these findings in normal subjects are usually associated with an inverted P wave, we have observed them even when the P wave is upright.

Chou and Helm, employing the Frank lead system, suggested that a counterclockwise frontal-plane loop with a maximal QRS
vector exceeding +40° indicates high lateral infarct. Our own experience indicates that at this angle there is significant overlap with normal subjects. Clearly, more accurate criteria are needed for the diagnosis of high lateral infarct. It is our belief that other aspects of the frontal-plane loop may in time prove helpful. Attention might also be directed to the initial deflection (R wave) in lead aVF.

**Posterobasal Infarction**

Posterobasal, dorsal, or “true” posterior infarct is one of the more difficult ECG or VCG diagnoses. The posterior wall of the left ventricle is said to be depolarized late in the QRS cycle. To the extent that this is so, it is difficult to account for prominent anterior forces (upward deflections) early as well as late over the right precordium in these cases of myocardial infarction. In some cases of posterobasal infarct the late part of the leftward forces may be located posterior to the null point. It may thus be difficult to differentiate some posterobasal infarcts from normal variants which may likewise show predominantly anteriorly located vectors. In fact, with the Duchosal (double cube) VCG reference system all of the leftward vectors may normally be anterior. In the Frank system, now the most widely used technique, posterior forces are accentuated; in this VCG reference system entirely anteriorly directed leftward forces are abnormal and may, among other causes, be due to posterobasal infarct. In many of these patients the direction of inscription may also be definitely abnormal with marked figure-of-eight or even completely clockwise patterns. In some of these cases the ECG may show an R/S pattern in lead V1 which is difficult to distinguish from the normal, but the VCG is often quite decisive (fig. 7.8).

Difficulty in distinguishing right ventricular hypertrophy (RVH) from posterobasal infarct is experienced with the VCG as well as with the ECG. Vectorcardiographic criteria, derived from Frank lead recordings, which are very helpful in making a proper differentiation, were proposed recently. If the horizontal-plane mean QRS falls in the left anterior quadrant and the terminal rightward appendage measures less than 1 mv, the diagnosis of posterobasal infarct is favored (90% accuracy) over RVH, but differentiation from normal subjects cannot necessarily be made. When right bundle-branch block is associated with posterobasal infarct the entire curve may lie anterior to the null point; the same is true of
some subjects with right bundle-branch block who do not have heart disease.51, 52

Generally, multiple infarcts can each be diagnosed by the VCG because the pattern of one does not alter or simulate the pattern of the other.36, 37 Nevertheless, one report has shown that when there is an inferior infarct the additional presence of an anterior infarct may alter the shape of the upward-bowed convexity of early superior frontal-plane vectors as well as the direction of their inscription.10 However, there is need for further VCG criteria of combined infarcts which lie directly opposite each other. In these cases, i.e. combined anterior and posterobasal or high and inferior locations, the ECG may be normal because the abnormal Q changes of one infarct cancel out those due to the other. It is particularly in these difficult examples that the VCG has been relatively unexplored but may yet yield useful information.

The relative value of the VCG versus the ECG diagnosis of myocardial infarction is still argued. Indeed, McConahay et al.53 on the basis of findings at coronary cineangiography, found that the combined use of ECG and VCG was superior to either technique alone. Earlier reports54, 55 showed at least a theoretical advantage of the VCG over the ECG, but no significant difference was noted between the two techniques in the earlier studies of Burch et al.56 or Johnston57 or in the more recent cooperative study headed by Simonson.58 The latter, however, emphasized that

"the most important reason for the relatively low diagnostic accuracy of the VCG is the lack of quantitative evaluation" and stated that "there is little doubt that truly quantitative analysis of VCG information will be the most important step for improvement of its diagnostic accuracy." Our own recent experience and that of McConahay et al.53 seem to substantiate Simonson's conclusions. It seems to us that the accuracy of ECG interpretation might well be enhanced if ECG criteria were critically reevaluated in the light of information contained in the VCG.

Left Hemiblock or Trifascicular Block and Myocardial Infarction

Left anterior hemiblock (LAH) by substituting an initial R wave in inferior leads in some cases can eliminate the expected Q wave of diaphragmatic infarct; this initial R wave is the result of the early rightward inferiorly directed forces caused by this conduction defect. Q waves may not then be observed in leads III and aVF, and the existence of diaphragmatic infarct might be missed.43 Myocardial infarction may be masked in another way. In anteroseptal myocardial infarction the Q waves expected in the right precordial leads may, with the onset of LAH, be replaced by R waves. The same vectorial explanation given above applies here.43

Figure 8

(Top) ECG of a 50-year-old male, proven normal by angiography, showing an intrinsic deflection of 0.03 sec and R/S ratio greater than 1 in V1. (Bottom) Horizontal-plane VCG is normal because it is counterclockwise and much of the late part of the loop is posterior.
are examples of "false-negative" diagnoses of myocardial infarct.

On the other hand a "false-positive" diagnosis of anteroseptal infarct may also be made in "pure" LAH.43 This can produce small Q waves in the right chest leads if the latter are recorded above the electrical center of depolarization (as in emphysema) or because of high application of the chest electrodes. Such an erroneous diagnosis may be avoided, it is said, by showing that these Q waves disappear when the electrodes are applied an interspace or so lower. Simulation of anteroseptal infarct can also occur in "pure" left posterior hemiblock (LPH) for exactly the opposite reason; misleading Q waves may appear in the right precordial leads if the electrodes are applied below the conventional level.

Yet another instance of "infarct simulation": LAH may lead to the development of impressive Q waves in lead aV_{1} and thus simulate high lateral-wall infarct. Contrariwise, with a large lateral-wall infarct all QRS changes could erroneously be attributed to LPH, and the infarct could be missed. LPH should not necessarily be diagnosed in the scalar electrocardiogram in the face of a large lateral infarct.43

It is conceivable that some of Rosenbaum's concepts on hemiblock may need to be modified in light of the following considerations: (1) Whether or not hemiblock or infarct is present certain changes in the spatial relationship of heart to recording electrode (e.g., those induced by respiration, by change in posture, or by changing level of electrode placement) may modify the appearance of the ventricular complex. (2) Further criteria are needed to differentiate simulated or concealed hemiblock from infarct as well as from other cardiac abnormalities. As illustrated in figure 2 and discussed briefly in the following paragraph, the VCG may well be helpful in this differentiation. (Young E, et al.: Unpublished observations.)

It must be remembered that myocardial infarct is a significant cause of hemiblock. Coronary heart disease is responsible for no less than 41% of cases of LAH.43 It should be kept in mind nonetheless that some normal subjects as well as patients with pulmonary disease may show an ECG with marked left-axis deviation. In some of these cases the VCG is very helpful because the frontal-plane loop is characteristic and not at all suggestive of LAH.

The presence of LAH and right bundle-branch block (RBBB) in a patient with documented coronary artery disease suggests stenosis at least of the anterior descending branch of the left coronary artery. Frank anteroseptal infarct43 is the condition common to its development. An angiographic study carried out at this hospital showed in fact (Williams RA, et al.: Unpublished observations) that the descending branch of the left coronary artery was critically obstructed in all cases of anterior infarct complicated by LAH. Myocardial infarct accounts for a considerably smaller percentage of cases of LPH.43

It is common practice to infer an active myocardial process if the ECG shows definite changes in relation to previous tracings, but an abrupt metamorphosis in the ECG is not bona fide evidence of fresh myocardial infarction. Generally the latter does not blossom full grown but rather passes through a gradual sequential transformation. Conceivably such intermediate developments could be missed if events proceeded very rapidly or if observations were made too infrequently. However, this hardly accounts for all such abrupt mutations for they may be seen when the ECG is under very close surveillance, and they commonly disappear as suddenly as they appear. The development or reversion of conduction disturbances, on the other hand, may change the ECG abruptly. The sudden appearance or disappearance of the full-fledged QRS changes of infarct could thus be due to the emergence or submersion of evidence of old infarct, revealed at one time, concealed at another.

Acknowledgment

We are grateful to Dr. Richard Gorlin under whose direction and in whose Cardiovascular Division Laboratories all angiographic studies referred to in this publication were carried out.
References

1. Bayley RH, LaDue JS, York DJ: Electrocardiographic changes (local ventricular ischemia and injury) produced in the dog by temporary occlusion of a coronary artery, showing a new stage in the evolution of a myocardial infarction. Amer Heart J 27: 164, 1944


10. Wilson FN, MacLeod AG, Barker PS: The interpretation of the initial deflections of the ventricular complex of the electrocardiogram. Amer Heart J 6: 637, 1931


29. Levine HD, Hellem HS, Wittenborg MH, Dexter L: Studies in intracardiac electrogra-
ECG AND VCG IN MI

phy in man: I. The potential variations in the right atrium. Amer Heart J 37: 46, 1949
31. MASTER AM: P wave changes in acute coronary occlusion. Amer Heart J 8: 462, 1933
32. BLOOM NB: The auricular complex in coronary thrombosis. Amer Heart J 24: 602, 1942
42. DRAPER HW, PEPPER CJ, STALLMAN FW, LITTMAN D, PIPBERGER HV: Corrected orthogonal electrocardiogram and vectorcardiogram in 510 normal men (Frank lead system). Circulation 30: 853, 1964
45. YOUNG E, WOLFF L, CHATFIELD J: The normal vectorcardiogram. Amer Heart J 51: 713, 1956
47. KINI PM, EDDLEMAN EE Jr, PIPBERGER HV: Electrocardiographic differentiation between left ventricular hypertrophy and anterior myocardial infarction. Circulation 42: 875, 1970
48. GOLDBERGER E: Differentiation of normal from abnormal Q waves. Amer Heart J 30: 341, 1945
51. PENALOZA D, GAMBOA R, SIME F: Experimental right bundle-branch block in the normal heart: Electrocardiographic, vectorcardiographic and hemodynamic observations. Amer J Cardiol 8: 767, 1961
56. BURCH GE, HORAN LG, ZISKIND J, CRONVICH JA: A correlative study of postmortem, electrocardiographic, and spatial vectorcardiographic data

Circulation, Volume XLV, February 1972


Electrocardiogram and Vectorcardiogram in Myocardial Infarction
HAROLD D. LEVINE, ELIOT YOUNG and RICHARD A. WILLIAMS

Circulation. 1972;45:457-470
doi: 10.1161/01.CIR.45.2.457
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1972 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/45/2/457.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/