A Study of Unipolar Left Back Leads and Their Application to Posterior Myocardial Infarction

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The electrocardiographic diagnosis of myocardial infarction of the posterior wall is often missed because it is largely dependent on lead aVF and because this electrode position often does not sublend the diseased myocardium. To remedy this defect, a new method is described—unipolar left back leads. The validity of this method in registering potential from the posterior left ventricular wall is discussed as well as its importance in the diagnosis of myocardial infarction of the posterior wall. Left back leads can illuminate the significance of questionable Q waves.

The value of unipolar electrocardiographic leads taken from the anterior chest wall is well established. Posterior chest leads, however, are not usually employed because adequate systematic knowledge about them is not available. The greater distance separating the posterior chest from the heart might lead to the a priori assumption that back leads would not have sufficient "semidirect" relationship to the myocardial surface. This, in fact, was the conclusion of a recent study of back leads. This view has lead to the use of esophageal leads as a semidirect approach to the posterior surface of the heart. Aside from the obvious technical difficulties in using these leads, there is the further drawback that only a narrow portion of the heart is explored.

Wolferth and associates have studied the distribution of ventricular potential in leads taken from many regions of the body surface and showed that distance merely reduced the amplitude while the pattern remained faithfully reproduced. In their fundamental studies correlating electrocardiographic with postmortem findings in myocardial infarction, Myers and co-workers found that posterior myocardial infarction was most frequently missed and that the horizontal position of the heart contributed heavily to this failure. In 35 cases of necropsy-proven posterior infarction in hearts situated in the horizontal or semihorizontal position, 22 (63 per cent) showed a normal rS complex in lead aVF. In 75 cases of proven posterior infarction with the heart in intermediate to vertical position, the electrocardiogram was negative in 16 (21 per cent). The failures were found almost entirely in patients whose lesions did not involve more than one third of the posterior left ventricular surface. In a necropsy study, Levine and Phillips also noted that in 29 cases of old and recent posterior myocardial infarction 13 (45 per cent) were not diagnosed by the electrocardiographic studies.

Continental workers have attempted to increase the accuracy of diagnosis by using unconventional bipolar lead positions. Nehbi employed precordial position 7 and the second intercostal space just to the right of the sternum, while Slapak and Partilla used several points in the second intercostal space between the left sternal border and the anterior axillary line together with precordial position 7. Increased diagnostic accuracy was reported by these authors, but it is not clear what variations are found with these special leads in normal individuals. Sears and Myers analyzed a limited number of left back leads and concluded that they were helpful in determining the presence and extent of posterior infarcts but failed in a few cases in which esophageal leads were positive.

The present study was undertaken to in-
crease the accuracy of diagnosis of posterior wall infarction by the use of unipolar left back leads. The left back leads were studied in normal subjects, in patients with left and right ventricular hypertrophy, and in known and suspected cases of old and recent posterior myocardial infarction.

**Method and Subjects**

Unipolar V leads were taken along three vertical lines marked on the subject's left back. The spinous (Sp) line is drawn 2 cm. to the left of the thoracolumbar vertebral spines. The scapular (Sc) line represents the posterior projection of the anterior midclavicular line. The posterior axillary (PA) line is self explanatory. Points were marked along these lines for the placement of the electrode at the intersection of horizontal lines which passed through the spinous processes of thoracic vertebrae 2, 4, 6, 8, 10 and 12, and lumbar vertebra 2. On the posterior axillary line recordings were made only at the level of the sixth and tenth thoracic and second lumbar spines. Each lead was labeled by the letters designating the vertical line and by the number designating the spinous level at which it was recorded. The level of the second lumbar spine was labeled L-2, thus Sc8 represented the lead taken at the level of the eighth thoracic spine on the scapular line. Some idea of the anatomic relationships of the back leads may be gained from figure 1.

An early group of back tracings were taken with the subjects in the sitting position. Later it was found that somatic tremor was less troublesome when the subject was prone and the majority of tracings were taken in this position. Some acutely ill patients found it difficult to assume the prone position, and it was necessary to take the back leads while these patients were lying on their right side. In addition to the back leads, the usual 12 leads were recorded and the augmented limb leads were repeated with the subject in the position in which his back leads were taken.

The tracings were made with either a Sanborn Viso-Cardiote or a Beck-Lee Cardial. In some cases the standardization was increased to one and one-half normal for the back leads to facilitate interpretation of weak potentials.

A total of 112 patients, divided as follows, was studied: 52 normal individuals, 18 patients with left ventricular hypertrophy, 6 patients with right ventricular hypertrophy, and 36 patients with posterior wall infarction. The normal subjects were either healthy medical students or patients without clinical or electrocardiographic evidence of heart disease. In the 52 normal subjects the electrical position was classified by the criteria of Wilson9 as vertical (8 cases), semivertical (11 cases), intermediate (12 cases), semihorizontal (4 cases), and horizontal (17 cases). Because of the rarity of the horizontal electrical position in healthy young persons the group of normal subjects with horizontal hearts does not include any of the medical students but consists of an older age group, 40 to 65 years, with normal clinical, fluoroscopic and electrocardiographic findings.

Eighteen patients with clinical, fluoroscopic, and electrocardiographic evidence of left ventricular hypertrophy due to hypertensive heart disease without evidence of myocardial infarction were studied. The group with right ventricular hypertrophy con-

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**Fig. 1.** Diagram showing position of the left back leads and their relation to the ventricles. See text.
in aVL), semihorizontal to semiveritical (RS in aVL), or semihorizontal to vertical (rs or QS in aVL). When the terminal portion of the QRS in aVF is upright, the height of this R wave aids in subclassification within the area defined by aVL. None of our cases showed predominantly negative deflections in aVL. The cases were classified into two groups: horizontal to semihorizontal position, 22 cases; and intermediate position, 14 cases.

Classification of QRS Patterns

Using Wilson's concepts the initial ventricular complex may be separated into four patterns each representing the pattern obtained from a region of the heart: (1) the cavity pattern or QS wave, (2) the transition zone pattern, a QR wave, (3) the left epicardial pattern, a qR or R, and (4) the right epicardial pattern, sR or RS.

The cavity pattern is clearly distinguished from the transition pattern since the first appearance of an R wave as the electrode is placed more caudal marks the beginning of the transition pattern. Distinguishing the boundary between transition complex and epicardial complex raises a more difficult problem since both consist of a Q wave followed by an R wave. The transition zone pattern is recorded when the electrode is in such a position that it is affected by the current from both the endocardial and epicardial surfaces. It is apparent that as the electrode is shifted to a more caudal position relatively less of the endocardial and more of the epicardial potential will be recorded; hence, the Q wave becomes smaller. Therefore, the cavity Q wave must be distinguished from the small septal Q wave. This same problem is present in evaluating esophageal and aV leads. The form of the P and T waves has been used as a clue in determining when the electrode no longer faces any portion of the cavity. There are possible errors in the use of such clues, as will be discussed later.

Myers and co-workers in evaluating the diagnostic significance of the Q wave in standard lead III and in aVF, chose the following limits for a septal Q wave in aVL: Q should not exceed 25 per cent of the voltage of R and should be less than 0.03 second in duration from onset to nadir providing the total voltage of the QR wave is 0.5 mm. or greater. These criteria seem adaptable for separating the transition Q from the septal Q in back leads. As a rule in normal subjects there was either no change in the ratio of Q to R, or only a slight and gradual reduction in the relative size of the small Q below the highest level (most cephalad) at which it may be classified as septal. On the other hand, above this level a majority of the records showed a much deeper and wider Q. Myers had also noted that the duration and size of the septal Q wave is greatest near the base. This is the ground for the belief that the basal portion of the ventricular wall is activated last.

RESULTS

A. Normal Subjects

The highest (most cephalad) levels in the left back leads in which the QRS complex reflected the epicardial potential was determined for the various heart positions. All the tracings taken on the posterior axillary line presented epicardial complexes. The tracings from the Sc and Sp lines showed variation in the level of transition from cavity QRS to epicardial QRS complex (table 1).

In the eight subjects with vertical position this transition was found between spinous level 6 and 10. Most of the cases showed the transition at Sc5 and Sp5. Figure 2 illustrates the vertical position. In the 16 subjects with horizontal hearts 14 presented epicardial complexes at Sc5; the other two did so at Sc4. The Sp line was slightly more variable but in 10 subjects the transition occurred at Sp3 and Sp4. In one example cavity Q waves were seen as low as Sp2. In the Sp line of four subjects in the group with horizontal hearts the Q wave became relatively deeper compared with the R wave at levels below the epicardial transition. These were the only instances among normal subjects in which the Q wave became relatively larger as the electrode was moved caudal along a vertical line.

Twenty-seven subjects were included in the groups with semivertical, intermediate, and semihorizontal positions. In 20 subjects the transition was between Sc5 and Sc6 while in 16 subjects the transition was between Sp5 and Sp16. There was some tendency for the transi-
tion point to become higher on the scapular line and lower on the spinous line as the position became more horizontal. Some of the group with semivertical hearts showed low transition levels. It might be anticipated that if more examples of vertical hearts were studied, lower transition levels would be found in this group also.

The most consistent findings in the normal subjects were: (1) the presence of epicardial complexes in all the posterior axillary leads; (2) epicardial complexes at and below Sp and Sc in subjects with hearts in the horizontal level there were many exceptions. Therefore, the P wave is not a reliable guide for determining the transition from cavity to septal Q.

Table 3* compares the level at which the Q wave meets the criteria of septal origin with the level at which the T wave becomes upright and at least one-tenth the height of R or at least 0.1 mV. in amplitude. There was only a small tendency for the T-wave change to occur at a higher level than the Q-wave change. In no instance did the T wave become upright more than two vertebral spine interspaces higher than the Q wave became septal. There

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* No septal Q wave seen—cases in which the lowest level recorded was 10
† No septal Q wave seen—cases in which the lowest level recorded was 12
‡ PA stands for posterior axillary line, Sc for scapular line and Sp for spinous line. (See text for further description.)

position; and (3) the tendency for the Q wave to become smaller at more caudal levels along either line.

To test the usefulness of the P wave as a guide to the evaluation of the Q wave, the level of transition to an infra-atrial P was compared with the level at which the Q assumed the characteristics of a septal Q defined above (table 2).* Only normal subjects showing clearly defined P-wave transition levels were tabulated. The P wave was considered to be infra-atrial when it was entirely upright without an intrinsic deflection and with approximately equal ascending and descending limbs. It is apparent from table 2* that while commonly the P and Q waves changed at the same

* Tables 2, 3, 4, 5, 6, 7 and 8 are omitted at the request of the Editor. They will be furnished on request.
except for absolute voltage. The distance between the highest back lead recording an epicardial complex and the highest reflecting the pattern of aVF represents approximately the surface projections of the area of the posterior epicardial surface that is unrecorded by aVF. Thus, in the Sc line of the patient with a horizontal heart, shown in figure 3, the area between Sc2 and Sc10 records left ventricular complexes which are not seen in aVF. This can be tabulated as eight intercostal spaces of unrecorded posterior ventricular wall. Table 4* shows the distribution of this nonregistered area in spinous intercostal spaces in the various cardiac positions. In every electrical position most of the subjects presented some posterior epicardial surface hidden from lead aVF. The unrecorded area tends to become greater in the horizontal position and less in the vertical position. Thus, among 16 subjects with hearts in the horizontal position, 14 presented hidden epicardial complexes over an area of 10 to 12 spinous intercostal spaces. There was only one subject in the group with vertical and semivertical hearts who had more than four hidden intercostal hearts. Subjects with hearts in intermediate and semihorizontal groups showed results between these extremes.

**Fig. 2.** The classic and left back leads in a typical normal subject with vertical heart position, showing relation of back leads to epicardium and cavity of heart. See text.

**Fig. 3.** The classic and left back leads in a typical normal subject with horizontal heart position showing relation of back leads to epicardium and cavity of heart. See text.

### B. Subjects with Cardiac Pathology

1. **Left Ventricular Hypertrophy.** In most cases of left ventricular hypertrophy, the heart is in horizontal position with right ventricular QRS complexes in lead aVF. Of the 18 cases of left ventricular hypertrophy, the hearts of 11 were horizontal or semihorizontal. Figure 4 illustrates the fidelity of recording of left ventricular complexes. Thus Sc4 to Sc12 resemble V5 or V6 and Sp12 resembles aVF except for difference in voltage. Table 5* shows the distribution of the highest epicardial or septal Q wave using the criteria discussed above. In six cases in which PA

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* See footnote p. 659.
leads were taken, epicardial complexes were seen at the highest level recorded (PA4). In the Sc line, 13 of the 18 patients showed epicardial complexes in all leads from Sc2 to ScL2. Four subjects showed transition to septal Q at Sc4. One patient with vertical heart showed transition in Sc1o. The spinous line showed greater variability in the transition level.

Table 6* lists the unrecorded areas of the posterior ventricular wall measured in spinous intercostal spaces in the 11 cases of left ventricular hypertrophy in horizontal or semihorizontal heart position. This was determined in the manner described for normal hearts. In 10 of the 11 cases an area embracing eight or more intercostal spaces on the Sc line was hidden from aVF. Again the Sp line was more variable. This left ventricular hypertrophy group, therefore, corresponds to the normal group with horizontal position in the large area of unrecorded left ventricle.

2. Right Ventricular Hypertrophy. In the three subjects with cor pulmonale who were in the horizontal or semihorizontal cardiac position, tracings made in the Sc line recorded right ventricular complexes from Sc2 or Sc4 down to Sc10 (leads were not taken at lower levels). The Sp line was variable; tracings made on this line showed right ventricular, left ventricular and cavity complexes. One case of mitral stenosis and insufficiency in the electrically vertical position, which also met the criteria of Sokolow and Lyon7 for right ventricular hypertrophy, showed cavity complexes in all leads except Sc1o; the latter recorded a left ventricular complex. In the patient with transposed pulmonary veins, also in vertical position, left ventricular complexes were seen from Sc6 to ScL2 and from SpL2 to SpL2. The other leads recorded cavity complexes. The Sc line recorded right ventricular complexes at high levels. This is analogous to the recording of left ventricular complexes in the Sc line in horizontal normal hearts and horizontal left ventricular hypertrophy hearts.

3. Posterior Myocardial Infarction. In the 36 patients with posterior infarction who had definite electrocardiographic evidence of the infarct, normal left ventricular QRS complexes were seen at some level along either the Sp or Sc line on the back and a deep abnormal Q at a lower level along the same line. In all these cases a progressively deeper Q wave was noted as the electrode is moved from above downward.

* See footnote p. 639.
over the area of the infarct. This is true for both the Sp or Sc lines although in some cases it was seen in the Sp line and not in Sc or vice versa. The deepening and often widening of the Q wave on descent of the electrode position could be demonstrated along the Sc line in all but four patients in whom tracings had not been made below Sc12; when tracings were taken to the level of Sc12 or Sc12, the transition to abnormal was always demonstrable. The Sp line in four other cases failed to show a transition to a normal epicardial complex.

In two of these subjects, the presence of infarction was apparent from the progressive deepening of the Q wave at lower levels. In the other two cases, the Sp line was not diagnostic since the QR ratio remained relatively constant at all levels.

The highest levels in the back leads at which the presence of infarct could be established were charted (table 7).* At least one higher lead showed a Q wave which could be accepted as septal in origin. The area between Sc8 and Sc12 and between Sp4 and Sp10 included the vast majority of the upper limits of demonstrable infarction. Since all these subjects showed an abnormal Q in aVF, they also recorded an abnormal Q in all back leads below the highest level indicative of infarction. In a substantial number of our cases, Q waves caused by infarcts were found on a large area of the back and may be presumed to indicate a large area of infarction of the posterior ventricular wall. The size of the infarct, of course, could not be ascertained from aVF.

The area of infarction not reflected in aVF was evaluated in a manner analogous to the estimation of unrecorded ventricular surface in the subjects with normal or hypertrophied hearts. The results are shown in table 8.* In 19 subjects with hearts in a horizontal and semihorizontal position in whom infarction could be diagnosed by the Sc leads, 18 showed extension along the Sc line beyond the area reflected in aVF. In six, the unrecorded portion of the infarct projected for two spinous intercostal spaces; in seven, for four intercostal spaces; and in six, for six intercostal spaces. The Sp line showed more variability since there were four instances of 8 to 12 unrecorded intercostal spaces of infarction. However, since some of the normal subjects with horizontal hearts showed abnormal Q waves in the Sp line, the evidence points to the Sp line as being less dependable than the Sc line. In patients with hearts in the intermediate position, more of the infarcted area was reflected in aVF. In both Sp and Sc lines, 4 out of 15 cases had more than two unregistered intercostal spaces exhibiting infarction.

C. Correlation with Esophageal Leads

Five patients with definite evidence of posterior infarction were studied with both back leads and esophageal leads at 2.5 to 5.0 cm. intervals. The latter leads also demonstrated the infarct. In patient L.S. (fig. 5) the esophageal leads give the impression of a high posterior infarct beginning just inferior to the atrium, while the back leads give the impression of normal muscle just inferior to the atrium with the infarcted area at a lower level. This is best seen in Sc6. While it is possible that esophageal leads taken at closer intervals might have shown normal epicardium

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* See footnote p. 659.
between atrium and infarcted area, it is noteworthy that this was not found in the five cases studied. On the other hand, the back leads in all five cases did show a transition from normal to infarcted epicardium. Because of disagreement in interpretation of the esophageal leads in this region, we are unable to correlate the esophageal with the back lead findings with regard to the more exact localization of the infarcted area.

Two patients in whom the back leads did not demonstrate infarction were studied with esophageal leads. These patients had typical clinical histories of acute myocardial infarction two weeks prior to our study. Very small Q waves in leads II and III were the suggestive electrocardiographic findings. The esophageal leads resembled the Sp leads and showed ST-T changes at low levels but pathologic Q waves were not obtained. In these two cases, the esophageal leads gave no more information than the back leads.

One patient was studied in whom the clinical picture was suggestive of infarction, but the conventional 12-lead electrocardiogram was not diagnostic at first. In this patient, the back leads showed serial evidence of infarction as illustrated in the following summary:

Patient M. L., 65 year old male, is illustrated in figure 6. He was first seen on June 9, 1951, about 90 minutes after the onset of severe substernal pain radiating to the left shoulder precipitated by attempting to chop down a tree.

The classic leads showed S-T elevation in aVF, II, and V₆, suggesting lateral and possibly posterior ischemia. The horizontal heart position prevented the recording of the posterior epicardial surface in aVF. In this position it is anticipated that the Sc leads will reflect epicardial surface at or below Sc₂. The tracings show that the Q in Sc₁ is relatively larger than Q in Sc₂ and that from Sc₆ through Sc₁₀ the unmistakable evidence of very acute infarction with tendency to monophasic ventricular complexes is present. Sc₁₂ and L₂ illustrate the transition to right ventricular complexes normally seen in horizontal hearts.

The clinical course and laboratory studies of this patient showed the blood pressure drop, the febrile reaction, the leukocytosis, and increased sedimentation rate characteristic of acute infarction. On June 16, 1951, there were minor changes in the classic leads consisting chiefly of a small Q wave, beginning inversion of T wave in lead aVF, and lead I, and less S-T elevation. The back leads in the Sc line showed the usual serial changes seen in infarction. The small R wave in Sc₁₂ had disappeared (extension through

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FIG. 6. Serial electrocardiograms in patient M. L. showing evolution of a recent posterior wall infarct diagnosed earlier in the back leads than in the classic leads. Sp₁₂ is the same as Sp₁₁. See text.
posterior septum?). Tracings made in the PA line definitely showed the lateral extension which had been hinted at by aVL. On July 6, 1951, the first unequivocal evidence of infarction in classic leads was seen and the area of ischemia had extended laterally (V3 and V6). Back leads showed the expected serial changes with progressive inversion of T waves in the infarcted area.

From the evidence in the back leads we could postulate a large posterior infarct at the first examination while the classic leads were only suspicious. Extension to the septum and laterally was demonstrable in subsequent tracings. The Sp leads were not informative of myocardial infarction and evidently represent cavity or transitional complexes. Therefore, the diagnosis was dependent on changes in the Sc line. The important diagnostic lesson is that the back leads showed the posterior myocardial infarction earlier than the classic leads because the back leads subtend the infarcted region.

**Discussion**

In this investigation of the left back leads, a major problem was the differentiation of the cavity Q waves from Q waves caused by infarction. This necessitated a study of the back leads in normal and abnormal hearts in various electrical positions. Figures 2 and 3 illustrate rather typical findings in left back leads in vertical and horizontal hearts. In the vertical heart (fig. 2), both Sp and Sc leads show a recognizable progression from cavity to transitional to left epicardial complexes. Leads Sp12 and Sc12 subtend the cavity in the same manner as lead aVR, and accordingly, these complexes closely resemble each other. Leads Sc6 to Sc12 and Sp12 to Sp12 subtend the epicardial surface of the left ventricle in the same manner as leads V5, V6 and aVF, and again these complexes closely resemble each other (except for differences in voltage). Transitional complexes obtained partly from the cavity and partly from the epicardium are recorded in Sc6 to Sc12 and Sp6 to Sp12. The PA leads reflect only epicardium since they do not face the cavity.

In the normal heart in the horizontal position (fig. 3) only Sp6 “looks” partially into the cavity, whereas PA, Sc and the remaining Sp leads reflect either right or left epicardial complexes, or transitional complexes along the septum as noted in Sp12 and Sc12. This transition from left to right ventricular complexes resembles that seen in the precordial leads. Figure 3 also demonstrates for the first time that the rS complex seen in aVF in horizontal hearts is also transmitted to the left back leads so that the right ventricle not only rests on the diaphragm but also points posteriorly.

With the exception of the horizontal heart position, the transition from cavity to epicardial complexes occurs at varying levels in the other electrical positions. Hence, the significance of the Q wave must be evaluated by comparing the various back leads in a given individual. The registration of a deep, wide Q wave at any given back position may not be indicative of infarction. However, once the transition to a septal Q wave is recorded the
finding of a cavity-like Q wave at a lower level in the same line is abnormal. This type of evaluation is similar to the interpretation of a small or absent R wave in lead V3. With sufficient clockwise rotation, V3 normally records a cavity complex and a small or absent R wave is expected. However, if the same patient had a definite R wave in lead V2, the absent R in V3 is abnormal.

There were four exceptions to this empiric finding, all in the Sp line in normal hearts in the horizontal position. In these patients the Q wave became progressively deeper from Sp2 to Sp10, although normal left ventricular complexes were present in the Sc leads. Also, the transition from right to left ventricle in the precordial leads occurred at V3 and the initial R in aVR was absent. These four exceptions may be explained by considering the relationship between the Sp line and the base of the heart. When the heart is horizontal, the Sp line tends to be in the same sagittal plane as the base. Accordingly, relatively minor mediastinal or apical shifts may determine whether cavity or epicardial complexes are recorded in the Sp line. Our suggested hypothesis is depicted in figure 7. In figure 7a the plane of the base of the heart is almost parallel to the Sp line so that cavity or transitional complexes are noted in both high and low Sp leads. As the apex rotates more posteriorly (fig. 7b) the plane of the base of the heart faces more anteriorly so that the Sp line does not “look into” the cavity and epicardial complexes are registered. This is also illustrated in figure 3. When the superior portion of the base lies more medially than the inferior portion (fig. 7c), upper Sp leads will show transitional complexes.

![Diagram](http://circ.ahajournals.org/doi/fig/10.1161/01.CIR.80.4.665)

Fig. 8. Classic and back leads showing digitalis effect in a patient with posterior myocardial infarction. See text.

The four exceptions in the normal horizontal hearts may fit into the concept diagrammed in figure 7c.

In the 112 patients studied, the left back leads obtained in normal hearts and in those with the various cardiac lesions record with surprising fidelity (except for voltage) not only the QRS complexes but also the ST-T changes of left ventricular hypertrophy and/or strain (fig. 4), and in one patient digitalis effects (fig. 8). The back leads also register a considerable area of the posterior left ventricular surface not recorded in aVP, especially in the semihorizontal and horizontal heart position.
It should also be noted that only minimal changes occur in the aV leads in the supine or prone posture so that this posture hardly influences the cardiac electrical position.

Margo and Foscarini were unable to correlate posterior chest leads with anatomic or clinical conditions and felt that the exploring electrode is so far distant from the heart that cavity and epicardial potentials cannot be distinguished. These authors used leads on a plane with $V_6$ equivalent to our PA$_{10}$, Sc$_{10}$, and Sp$_{10}$ plus Sc$_{10}$ on the right back. Their leads, limited to a horizontal plane in the back, do not permit analysis of transition from cavity to epicardial complexes which is the basis of our study. Our study shows that multiple leads along a vertical line in the back are necessary for proper interpretation. Also, our evidence indicates that left back leads are semidirect leads and yield localized information about the posterior heart surface.

The criteria of Myers for infarction Q waves in lead aV$_F$ is supported by his necropsy data. The finding in the back leads that the Q wave becomes progressively deeper (and occasionally wider) as the electrode is moved caudally on a vertical line, even though the largest Q wave may fall short of satisfying Myers' criteria, is highly suggestive of myocardial infarction, and particularly so in the Sc line. This has been seen in patients with clinical and electrocardiographic evidence of posterior myocardial infarction. However, postmortem correlation is necessary for confirmation.

From the results in our five cases of myocardial infarction studied by both esophageal leads and left back leads it would appear that the former tend to record deep Q waves over a relatively wider area than the latter. This may lead to the false diagnosis of high infarcts by esophageal leads and perhaps prevent the diagnosis of such infarcts by the back leads. The esophageal leads are limited to one vertical line and may leave much of the posterior surface unexplored. The back leads do not have this limitation. When a heart is in the horizontal position, the esophagus may be in a particularly disadvantageous relationship to the ventricle. The esophageal electrocardiogram will represent chiefly the uppermost base of the ventricles and the proximity of the A-V groove could lead to errors in interpretation. This is suggested by our finding that the Sp line is unreliable in horizontal heart position and this line roughly parallels the esophagus.

The criteria for the diagnosis of posterior infarction in the back leads may well be applied to studies with esophageal leads. The significance of a deep Q wave cannot be based solely on the form of the accompanying P wave. That this is true for the esophageal leads has been recently demonstrated by Sandberg and associates and our results with back leads tend to be confirmatory. That rigid application of the P-wave criterion can lead to apparent over-diagnosis of high posterior infarction is illustrated in a recent report by Benchimol and co-workers. Their cases 9 and 10 were presented as diagnostic of infarction although there was only a rather vague past history and no other objective confirmation. Their cases showed moderately deep, narrow T waves for 2.5 to 5 cm. below the level at which the P lost its intrinsic deflection. However, in each case the T waves became progressively smaller as the recording electrode moved from the cavity to the transitional to the epicardial zones. From our study of left back leads, we would consider these cases as showing a normal tendency for transition of the Q wave at a slightly lower level than the P wave rather than indicating infarction. Neither the P wave nor the T wave may be reliable guides for evaluating the meaning of a deep Q wave. Instead, the demonstration of deepening Q waves as lower left back leads are recorded would be more dependable guides.

Patients in whom posterior infarcts are most likely to be missed by routine electrocardiography are those whose hearts are in semihorizontal to horizontal positions. These include most cases of left ventricular hypertrophy. This explains the high incidence of posterior infarction not diagnosed in the usual electrocardiogram in Myers' series. Fortunately in these cases most, if not all, of the PA and Sc leads represent the epicardial
surface and a wide area is available for exploration. It is in such patients that left back leads are most likely to find application. The present results also suggest that in some cases with more vertical heart position a significant area of posterior infarction may be diagnosable only by back leads. In subjects in whom the presence of infarction is established by other leads, the back leads could be helpful in demonstrating the extent of infarction.

The introduction of new electrocardiographic leads raises the question whether the additional information gained warrants the added time involved. The left back leads are not indicated for routine use. They should serve to supplement the routine leads when an equivalent Q wave is found in aVF or when a clinically suspected myocardial infarct is not demonstrated by routine methods. In these problems the back leads may substitute for esophageal leads and are easier to take. The entire series of 17 posterior leads is often not necessary in clinical problems. The Sc line is usually most productive of useful and reliable information. If ScL2 is recorded first and successively higher leads are taken along this line one may gather this information with a minimal number of leads. A large Q wave at a low position and an epicardial complex at a higher position are the significant findings. Occasionally this may be seen after only three or four leads. When the entire Sc line is normal, then exploration of the PA and Sp lines in a similar manner is indicated.

**Summary**

1. Unipolar left back leads are semidirect leads; they recorded with fidelity the electrical events of the posterior surfaces of the left or right ventricles, and the cavity in the 112 patients studied.

2. In normal subjects with hearts in the horizontal position consistent recordings of epicardial complexes are found in tracings made in the PA and Sc lines. With the heart in other positions, transitions from endocardial to epicardial complexes are found along the Sc and Sp lines at variable but recognizable levels, while the tracings made in the PA line record only epicardial complexes.

3. In right and left ventricular hypertrophy changes similar to those seen in the conventional leads are recorded in the back leads.

4. In all 36 cases of posterior myocardial infarction established clinically and by conventional electrocardiographic leads, the back leads also showed the infarct. In addition, the back leads showed the transition from unaffected posterior ventricle to the area of infarction (that is from septal Q to infarct Q), and thereby allowed some estimation of the size of the infarct. In some patients clinically suspected of myocardial infarction, in whom the conventional leads did not clearly demonstrate the infarct, the left back leads offered specific evidence of infarction.

5. In every electrical heart position, most of the normal and abnormal patients presented some posterior epicardial surface hidden from lead aVF. This electrocardiographically unrecorded area is least in the vertical position and most in the horizontal position.

6. An interesting finding in the back leads, noted for the first time, was a transition from left to right ventricular complexes in horizontal hearts in either normal subjects or patients with left ventricular hypertrophy. This may be comparable to a similar transition seen in the precordial leads.

7. A concept is advanced that progressive deepening of the Q wave as the lower left back leads are recorded may indicate myocardial infarction just as well as the accepted criteria for an abnormal Q wave in lead aVF. Thus, the significance of questionable Q waves in lead aVF, upon which the diagnosis of posterior myocardial infarction usually depends, can be better evaluated by means of the left back leads.

**Sumario Español**

El diagnóstico electrocardiográfico de infarto de la pared posterior del miocardio es a menudo pasado por alto debido a que depende grandemente en la derivación aVF, y debido a que la posición del electrodo muy a menudo no subtiende el miocardio averiado. Para remediar este defecto, un método nuevo se describe—derivaciones unipolares del lado izquierdo de la espalda. El valor de este método registrando
potenciales de la pared posterior del miocardio se discute así como su importancia en el diagnóstico de infartos posteriores del miocardio. Derivaciones del lado izquierdo de la espalda pueden iluminar el significado de ondas Q de origen problemático.

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A Study of Unipolar Left Back Leads and Their Application to Posterior Myocardial Infarction

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