A Clinical Appraisal of the Vectorcardiogram in Myocardial Infarction

II. The Frank System

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In the preceding communication the results of the vectorcardiographic examination of 161 cases of myocardial infarction employing the cube system were described. In the present report the results are given for 206 cases studied by the Frank system.

The purpose of this study is to determine the practical value of this corrected lead system, which has been reported to possess many theoretical advantages over older recording systems. In addition, a comparison between the standard electrocardiogram and the vectorcardiogram is made in a fashion similar to that described previously. On the basis of distinct correlations found in the preceding study the same system of classification of types of infarct is maintained.

Methods and Materials

These studies were carried out over a period of two years on patients hospitalized at the Peter Bent Brigham Hospital. The vectorcardiographic system used was that proposed by Frank. In all other respects the technic followed that described in the previous communication. On the basis of the same criteria the material of this study was classified into three categories: Group A, namely those with indubitable infarction, 24 of the 35 patients who were examined at autopsy; group B probable infarction, 101 patients with classical sequential electrocardiographic changes and convincing evidence of tissue breakdown; and group C, presumptive infarction, 81 patients in whom, despite a convincing history, complete confirmation by laboratory tests was not forthcoming.

From the postmortem specimen the area or areas of infarction in a particular heart were classified topographically in accordance with the accompanying diagram (fig. 1).

Corresponding with the convention set forth in the previous publication, the QRS loop as inscribed in the sagittal plane was recorded as if the observer were looking from the left shoulder into the sagittal section of the body (see fig. 1 of preceding paper).

Consequently, it was necessary to reverse the values found by Pipberger in the sagittal plane in normal adults in order to conform to this manner of registration (table 1).

Criteria for Vectorcardiographic Diagnosis of Infarction

In a detailed study of 100 normal individuals, between 15 and 69 years of age, Pipberger utilizing the SVEC-III system, examined a number of vectorcardiographic parameters and determined their statistical validity. Since the direction of vectors as found by the Frank system approximates closely those registered by the SVEC-III system, his data were regarded as a standard for

*Only tables 2, 3, 7, and 10 are presented with this publication. Tables 1, 4, 5, 6, 8, 9, and 11 have been deposited as Document number 6776 with the ADI Auxiliary Publication Project, Photoduplication Service, Library of Congress. Copies may be secured by citing the Document number and by remitting $1.25 for photoprints, or $1.25 for 35 mm. microfilm. Make checks or money orders payable to: Chief, Photoduplication Service, Library of Congress.
normality in the present study. In addition, a series of normal controls in a younger age group was studied by the Frank method in this laboratory. The results agreed well with those obtained with the SVEC-III technic. In this analysis particular attention was directed to the following parameters: (1) the direction of the 0.01, 0.02, 0.025, 0.03, and 0.04-second QRS vectors in the various planes, using Helm's system of notation (fig. 2 of preceding publication); (2) the direction of the maximum QRS vector; (3) the direction of rotation (clockwise or counterclockwise) of the loop; and (4) the presence of abrupt changes in the direction of the middle or final thirds of the QRS loop (“bites” or “ares”). The magnitude of the various QRS vectors was not analyzed. Accordingly, whenever the word vector is used in this text, it refers only to the projection upon a certain plane of the spatial direction of that vector.

General Considerations

As outlined in the discussion of the preceding publication the most significant abnormalities of ventricular depolarization after myocardial infarction are to be found in the vectors generated within the first 0.03 second (fig. 1 of preceding publication). The vectorcardiograph is particularly suited for the study of the direction of these initial vectors.

The cathode ray oscilloscope allows instantaneous synthesis of rapidly changing electrical forces. In addition, a corrected lead system reduces variations and aberrations related to differences in body build, dipole position, and effective lead axes (figs. 2, 8, and 14). This is borne out by the small standard deviations found in normal controls (table 1).

Each of the observations made at a given instant is the resultant of electrical forces generated by simultaneous depolarization of many different areas of the myocardium. For example, 0.02 second after the beginning of ventricular depolarization, the resultant vector is usually oriented inferiorly and anteriorly, toward the apex of the left ventricle. Depolarization of most of the left septal mass has been accomplished and the wave of accession is now progressing along the Purkinje system in the free wall of the left ventricle. At the same time it is already proceeding from endocardium to epicardium in the neighborhood of the apex of the left ventricle. Thus, a single area of infarction in the anteroseptal region might displace the expected normal vector for this instant and would create a QRS-loop deformity “classical” for anteroseptal infarction. When multiple areas are infarcted, however, the anticipated abnormal vector may be displaced in a different direction. For example, infarction of the subendocardial and intramural laminae of the free lateral wall in addition to anteroseptal infarction would displace the infarct vector in a much more rightward direction, assuming that both areas are simultaneously depolarized. It follows that it may be difficult to predict the direction of an infarct vector when there is more than one area of infarction. This explains in part the difficulty in correlating anatomic and vectorcardiographic phenomena in multiple myocardial infarctions. The wide standard deviations found in various infarct groups must be explained in part on this basis.

Similarly, deviations of the anticipated infarct vector might be expected in the presence of right or left ventricular hypertrophy. It was found, however, that the various abnor-
Figure 2

Disparity between cube and Frank system. (The sagittal plane in the lower row [cube] is viewed from the right.) Sagittal and horizontal plane loops show an anteriorly directed force of 0.0275-second duration (11 \times 0.0025 \text{ sec.}) by the Frank technic, no anterior force by the cube technic. The 0.02-second vector in the horizontal plane is at 30° by Frank, 335° by cube. Each system shows marked posterior and leftward deflection of the peripheral QRS loop, indicating left ventricular hypertrophy. The patient was a 51-year-old man with aortic stenosis. Autopsy showed no myocardial infarction. (The right-hand frames are an amplification of initial and terminal QRS forces in the horizontal plane. \text{i} represents the isoelectric or null-point; \text{T} denotes \text{T loop}.)

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Horizontal Plane

Anteroseptal Infarct

Sagittal Plane

Anteroseptal Infarct

**Diagram of average QRSsE loop in patients with anteroseptal myocardial infarction (heavy line) contrasted with normal controls (light line) in horizontal and sagittal planes.** Note the marked difference in direction of the 0.02-second vector between patients with anteroseptal infarct and normal controls (cf. table 2). In this and succeeding figures arrows indicate direction of rotation and location of instantaneous vectors in accordance with the following scheme:

- average vector at 0.01 second;
- average vector at 0.02 second;
- average vector at 0.03 second;
- average vector at 0.04 second; and
- average maximum vector.

Initial observations were actually influenced very little by concomitant hypertrophy. A possible explanation for this observation may be that with left ventricular hypertrophy the abnormally large vectors directed posteriorly and to the left are usually manifested somewhat later in the period of ventricular activation.\(^\text{16-18}\) Therefore, they would not be expected to affect the forces generated between 0.01 and 0.025 second. For right ventricular hypertrophy, similar reasoning may be advanced.\(^\text{19}\) Changes caused by left bundle-branch block have been discussed previously.\(^\text{4}\)

Finally, it needs to be reiterated throughout this discussion that, although abnormalities in depolarization are present in three dimensions, our emphasis is placed, at times, on the reflection of these vectors upon a single plane when that projection appears best to indicate abnormality.

**Vectorcardiographic Observations**

The results of this study are given in tables 2 to 10. Selected data are discussed in detail in the following section.

**Anteroseptal Myocardial Infarction**

Forty-eight patients were considered to have anteroseptal myocardial infarction; seven of these were examined post mortem. The values for the 0.01- and 0.02-second and maximum QRS vectors in the horizontal plane are given in table 2. Forty-four of the 48 showed counterclockwise rotation in this plane. The average direction of the 0.02-second vector (322°) for the non-autopsied cases corresponded closely with that of the seven autopsied cases (312°). Furthermore, the measurements for this vector fall within a very narrow range (S.D. 32.4°). When compared with the direction of the normal vector for this instant, there appears to be no overlap (\(p < 0.001\)) (fig. 3). This contrasts with the wide standard deviation of 79.5° found for the 0.01-second vector. Such a wide range of values may be partially explained by the fact that in many instances the wave of depolarization may at 0.005 second not yet have reached the area of infarction. Therefore it would retain a normal anterior and rightward direction.\(^\text{20-23}\) By 0.010 second the vector has usually been displaced posteriorly as a result of the infarct. However, there were several cases in which significant displacement was not recorded at this moment of ventricular depolarization, resulting in a wide spread of observations (cf. figs. 4 and 5 with 12). Therefore the 0.02-second vector in these instances

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has greater significance than the 0.01-second vector. Similar observations were made by Howitt and Lawrie,24 using the cube system. Obviously the finding of a 0.005- and 0.010-second vector, both directed posteriorly and to the left, still indicates infarction but suggests its location in the high anteroseptal mass closer to the origin of the left bundle25 (fig. 5).

There is no difference between the direction of the maximum QRS vector (323°, table 1) in the horizontal plane in normal individuals and that in 44 patients with anteroseptal infarction (323°, table 2). Furthermore, the standard deviations for both groups of observations are very similar (40.6° and 36.3°). Therefore in anteroseptal infarction the peripheral segment of the QRSsE loop is not displaced posteriorly. This is another helpful point in the differentiation from left ventricular hypertrophy, in which condition such displacement usually occurs.14,16

In 30 of the 48 cases of anteroseptal infarction the sagittal plane QRSsE loop rotated in a counterclockwise direction (table 2, figs. 3 and 4). Again a rather wide variation of the 0.01-second vector (39°, S.D. 70.9°) was observed. There was also poor agreement between the autopsied (82°) and the non-autopsied cases (39°). The 0.02-second vector (33°, S.D. 55.2°) corresponded more closely with the mean value of the necropsied group (60°). The 0.03-second vector was tabulated here because of the excellent correspondence with the mean value in cases in which the anatomic site of the infarct was definitely known (15° vs. 17°). Moreover, the standard deviation was smaller in this group (42.9°). Furthermore, the maximum QRS vectors (349° vs. 339°) was quite similar in both groups. All these measurements differ significantly from their normal counterparts (p < 0.001).

Of the 44 with counterclockwise rotation in the horizontal plane 14 demonstrated clockwise rotation in the sagittal plane (fig. 5). In autopsied and non-autopsied cases in this group there was a close correlation for all vectors measured (table 2). In the three cases examined post mortem the anteroseptal infarct extended into the apical region (fig. 5). Moreover, other vectorcardiographic evidence for associated apical or diaphragmatic infarct was present in nine of the 11 non-autopsied cases with clockwise rotation. Conversely, the diagnosis of concomitant apical infarction could be made in only one of the 30 previously described cases with counterclockwise rotation in the sagittal plane (table 10).

In conclusion, it appears that measurement of the 0.01- and particularly of the 0.02-second vectors in the horizontal plane and the 0.03-second vectors in the sagittal plane is essential for the diagnosis of anteroseptal infarction. Absence of significant anteriorly directed forces constitutes the most reliable evidence for this diagnosis. If clockwise rotation oc-

Table 2
Mean Values (in Degrees) of Selected Instantaneous Vectors in 44 Cases with Anteroseptal Myocardial Infarction with Counterclockwise Rotation in Horizontal Plane

<table>
<thead>
<tr>
<th>Plane and rotation</th>
<th>No. cases</th>
<th>0.01 sec</th>
<th>0.02 sec</th>
<th>0.03 sec</th>
<th>Maximum vector</th>
<th>&quot;Bites&quot;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>S.D.</td>
<td>S.D.</td>
<td>S.D.</td>
<td>S.D.</td>
<td>S.D.</td>
</tr>
<tr>
<td>Horizontal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>counterclockwise</td>
<td>44 (7)</td>
<td>79.5</td>
<td>4.9</td>
<td>—</td>
<td>323 (307)</td>
<td>36.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>11.9</td>
<td></td>
<td>—</td>
<td>323 (307)</td>
<td>5.5</td>
</tr>
<tr>
<td>Sagittal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>counterclockwise</td>
<td>30 (4)</td>
<td>70.9</td>
<td>15 (17)</td>
<td>42.9</td>
<td>342 (339)</td>
<td>44.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>12.9</td>
<td>7.8</td>
<td>8.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sagittal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>clockwise</td>
<td>14 (3)</td>
<td>45.7</td>
<td>3 (357)</td>
<td>32.5</td>
<td>18 (23)</td>
<td>24.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>12.5</td>
<td>8.7</td>
<td>6.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*In this and subsequent tables the standard deviation (S.D.) is given in roman type, the standard error of the mean (S.E.) in italics. The latter has not been computed where the number of observations was less than 10.
curs in the sagittal plane, associated diaphragmatic or apical infarction must be given serious consideration (cf. fig. 4 with figs. 5 and 10).

Anterolateral Myocardial Infarction

A total of 31 cases was available for analysis. Six of these were examined post mortem. Every case in this group showed clockwise rotation in the horizontal plane, a finding never observed in normal subjects\textsuperscript{10,11} (fig. 6, table 3). Furthermore, in the first 0.01 second the vectors had a rightward and slightly anterior direction (178°, S.D. 68.3°). The explanation for this wide standard deviation is probably the same as that already given for the similar observation in anteroseptal infarction. In view of the overlap with normal subjects, therefore, the direction of the 0.01-second vector is not decisive. However, the loop continued in a rightward direction in all cases; this was never observed in normal controls. The discrepancy in the direction of the 0.02-second vector when compared with the

\textit{Figure 4}

\textit{Anteroseptal infarction, displacing earliest (0.01 second) vector. In the vectorcardiogram in the horizontal plane the 0.01-second vector was at 352°, the 0.02-second vector at 300°; in the sagittal plane the direction of rotation of the loop was counterclockwise, the 0.01-second vector at 60°, the 0.02-second vector at 13°. Electrocardiogram suggests, vectorcardiogram is diagnostic of anteroseptal infarct. Electrocardiogram is in line with, vectorcardiogram incompatible with, diaphragmatic infarct. Extensive anteroseptal infarction; no diaphragmatic infarct found at autopsy. In this and the following illustrations the direction of rotation is indicated by the direction in which the tapering end of the teardrop points.}
normal (241° vs. 84°) (p < 0.001) indicates that this measurement is the next most important criterion for the diagnosis of anterolateral infarction (fig. 6).

Some influence on the direction of the maximum QRS vector should be expected, since the depolarization of the anterolateral region occurs later than that of the anteroseptal area20-23 and therefore would be more likely to influence peripheral loop configuration. This is illustrated by the observed values for the maximum QRS vector (non-autopsied 307°, autopsied group 296°). However, the differences between these measurements and those observed in normal controls (323°), although significant (p < 0.01), were less impressive.

Only a limited number of observations were made for the sagittal plane; they were about equally divided into two groups, one rotating counterclockwise and the other clockwise. In 15 of 19 instances of clockwise rotation in the sagittal plane additional vectorcardiographic evidence was at hand for apical or diaphragmatic involvement. In all four autopsied cases with this finding the infarct actually involved these additional areas (table 10). Conversely, in the two postmortem cases with counterclockwise rotation in this plane, associated apical or diaphragmatic infarction could not be demonstrated. In only two of the 12 non-autopsied cases with counterclockwise rotation in the sagittal plane, was there other evidence for apical or diaphragmatic infarction.

The most significant finding in the frontal plane QRS loop was a distinct preponderance of counterclockwise rotation, even in the presence of a nearly vertical maximum QRS axis. However, in this group none of the early vectors was of diagnostic importance because of the wide spread in the normal values for the 0.01- and 0.02-second vectors in this plane. In contrast to this the group with clockwise rotation showed significant superior displacement of the 0.03-second vector (315° vs. 339° for autopsied cases). This is probably best explained by the observation that in the four cases coming to autopsy associated apical infarction was present (table 3).

In summary, it is evident that the most reliable criterion for anterolateral infarction is clockwise rotation in the horizontal plane. The second most helpful evidence was rightward displacement of the 0.01-second (178°) and the 0.02-second vectors (241°). This suggests that in anterolateral infarction abnormal forces exert their maximal influence slightly later in the sequence of depolarization than in anteroseptal infarction, and thereby cause a reversal of the normal direction of rotation. Associated infarction of the apical region is frequent25 and can be demonstrated by clockwise rotation of the sagittal plane loop. The frontal plane is often helpful in that it may demonstrate counterclockwise rotation, with rightward displacement of the initial forces.

Table 3

<table>
<thead>
<tr>
<th>Plane and rotation</th>
<th>No. cases</th>
<th>Total Autopsied</th>
<th>0.01 sec S.D. S.E.</th>
<th>0.02 sec S.D. S.E.</th>
<th>0.03 sec S.D. S.E.</th>
<th>Maximum vector</th>
<th>S.D. S.E.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horizontal clockwise</td>
<td>31 (6)</td>
<td>178 (173)</td>
<td>68.3</td>
<td>241 (261)</td>
<td>43.3</td>
<td>307 (296)</td>
<td>24.2</td>
</tr>
<tr>
<td>Sagittal counterclockwise</td>
<td>12 (2)</td>
<td>87 (79)</td>
<td>75.0</td>
<td>44 (83)</td>
<td>42.1</td>
<td>17 (33)</td>
<td>28.1</td>
</tr>
<tr>
<td>Sagittal clockwise</td>
<td>14 (4)</td>
<td>296 (242)</td>
<td>85.9</td>
<td>313 (288)</td>
<td>62.8</td>
<td>2 (33)</td>
<td>62.7</td>
</tr>
</tbody>
</table>

*Total number of observations for each plane is not identical in this and subsequent tables because of exclusion of technically unsatisfactory recordings.
Discrepancy between vectorcardiogram and electrocardiogram. Vectorcardiogram diagnostic of left ventricular hypertrophy, anteroseptal (0.01-second vector 356°, 0.02-second vector 347° in horizontal plane; 0.01-second vector 347°, 0.02-second vector 356° in sagittal plane), and diaphragmatic infarcts (clockwise rotation of loop in sagittal and frontal plane, the latter in the presence of a maximum QRS at +5°). Electrocardiogram shows left ventricular hypertrophy, suggests incomplete left bundle-branch block but does not indicate anteroseptal infarct. Since it is traditionally taught that in the presence of low QRS voltage in lead aV1, a definite statement regarding diaphragmatic infarct is not warranted, this diagnosis could not be made either. Postmortem examination showed left ventricular hypertrophy and infarcts involving interventricular septum and diaphragmatic and apical surface of left ventricle.

Lateral Wall Infarction

Thirty-six cases were studied in this category (table 4). As in the previous group with anterolateral infarction, marked rightward displacement was observed for the initial vectors. A division was made into 27 patients with counterclockwise rotation (fig. 7) and nine patients with clockwise rotation (fig. 9) of the initial part of the horizontal plane loop.

In those instances where counterclockwise rotation was observed, the 0.01-second vector was directed at 129°. This is significantly different from the normal (94°) (p < 0.001). This cannot be said for the direction of the 0.02-second vector as projected on this plane (81° vs. 84° in the normal). However, in 17 instances the loop made a sudden and abrupt rightward and posterior turn after the 0.02-second vector and continued with clockwise rotation into the left posterior quadrant (between 270° and 360°), as if the ventricular activation curve suddenly met an obstruction (figs. 7 and 8). Such "bites" have been described by Burch, using the tetrahedron, by Portheine, using the Schellong reference system, and by Howitt and Lawrie, using the cube system. An explanation for this phenomenon may be offered along lines previously...
discussed. At 0.025 second, when this abrupt reversal of direction is usually encountered, the endocardium and Purkinje system are usually fully depolarized.\textsuperscript{20, 21, 22, 27} The activation wave then advances more slowly toward the epicardium.\textsuperscript{28} If this slower phase of activation, progressing as it were from cell to cell, meets an electrically inert area in the outer half of the free lateral wall, the remaining regions of the myocardium divert the resultant vector during that time.\textsuperscript{29} This could result in the sudden reversal from counterclockwise to clockwise rotation; such rotation was observed in the terminal limb of the loop in all of our cases (figs. 7 and 8, see also fig. 14), and has never been encountered in any of the normal subjects studied by corrected lead systems.\textsuperscript{10, 11, 31, 44}   

Of the 9 cases with clockwise rotation, four were examined post mortem. Each of these showed very extensive infarction involving the entire free left ventricular wall, extending both anteriorly and posteriorly. Depending upon the degree of involvement, then, the loop may rotate in a clockwise direction from its very onset or at some time after initial normal activation. In the first instance the loop will be entirely clockwise (fig. 9), in the latter a "bite" will be inscribed (figs. 7 and 8).   

In the patients showing the most extensive lateral wall infarction significant rightward displacement of the 0.03-second vector and of the maximum QRS vector was observed in the frontal plane. This occurred regardless of whether the loop rotated in a clockwise (five cases) or in a counterclockwise (four cases) direction (table 4B).   

Thus either an abnormal rightward direction of the initial portion of the QRS loop in the horizontal plane or a "bite" in its middle portion is required for the vectocardiographic diagnosis of lateral wall infarct.   

It is significant that of the 36 cases, 19 showed associated infarction in the apical and diaphragmatic regions. As described by Port-\textsuperscript{33} heine,\textsuperscript{12} it was largely in this group that the high incidence of "bites" was noted. These may be transient (fig. 11). In none of the four autopsied cases with counterclockwise rotation in the sagittal plane was apical or diaphragmatic infarction noted. On the other hand, in each of the three non-autopsied cases (table 4C) with clockwise rotation, vectocardiographic evidence for apical infarction was present. This re-emphasizes the importance of examining the direction of rotation in the sagittal plane to establish the presence or absence of associated diaphragmatic or apical infarction.   

### Apical and Diaphragmatic Infarction   

There was considerable difference in the configuration of loops in both the frontal and the sagittal planes between cases with apical

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**Table 7**

*Mean Values (in Degrees) of Selected Instantaneous Vectors in 51 Cases of Apical and 71 Cases of Diaphragmatic Infarction with Clockwise Rotation in Frontal Plane*

<table>
<thead>
<tr>
<th>Plane and rotation</th>
<th>No. cases</th>
<th>0.02 sec.</th>
<th>0.025 sec.</th>
<th>0.03 sec.</th>
<th>Maximum vector</th>
<th>No. of &quot;bites&quot;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apical infarction</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal clockwise</td>
<td>51 (7)</td>
<td>289 (271)</td>
<td>307 (300)</td>
<td>327 (323)</td>
<td>42 (38)</td>
<td>10</td>
</tr>
<tr>
<td>Sagittal clockwise</td>
<td>14 (4)</td>
<td>263 (269)</td>
<td>294 (303)</td>
<td>324 (358)</td>
<td>6 (17)</td>
<td>2</td>
</tr>
<tr>
<td>Sagittal counterclockwise</td>
<td>35 (3)</td>
<td>225 (262)</td>
<td>207 (257)</td>
<td>202 (251)</td>
<td>57 (84)</td>
<td>8</td>
</tr>
<tr>
<td>Diaphragmatic infarction</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal clockwise</td>
<td>71 (5)</td>
<td>317 (314)</td>
<td>338 (328)</td>
<td>358 (342)</td>
<td>48 (25)</td>
<td>11</td>
</tr>
<tr>
<td>Sagittal clockwise</td>
<td>37 (5)</td>
<td>290 (298)</td>
<td>309 (313)</td>
<td>339 (330)</td>
<td>28 (355)</td>
<td>4</td>
</tr>
<tr>
<td>Sagittal counterclockwise</td>
<td>34 (0)</td>
<td>226 (—)</td>
<td>192 (—)</td>
<td>135 (—)</td>
<td>37 (—)</td>
<td>7</td>
</tr>
</tbody>
</table>

*Since these data are a composite of the subgroups described in tables 5 A, B, and C, tables 6 A, B, and C, and in the text, no S.D. or S.E. has been calculated.

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Anterolateral infarction. Diagram (upper) of horizontal plane QRS loop. Note clockwise rotation of loop and wide separation between 0.02-second vector with this type of infarction and that recorded in normal subjects. There is also displacement of the maximum QRS vector (cf. table 3). Demonstration of infarct (lower) in vectorcardiogram, not in electrocardiogram. The 0.01-second vector in the horizontal plane is at 145°, the 0.02-second vector at 270°. Clockwise rotation is present in the horizontal plane. Patient presented a classical clinical attack with elevated lactic acid dehydrogenase.
infarction and those with pure diaphragmatic infarction (tables 7A and B). The entire category of 124 cases with infarction in inferior areas of the heart was therefore subdivided according to the direction of rotation of the frontal plane loop and according to the position of the maximum QRS vector in the frontal plane. Thus, in contrast to the method used in the previous publication, apical and diaphragmatic infarctions are here considered separately.

Apical Infarction

Of 53 patients, 51 displayed clockwise rotation in the frontal plane loop. These were subdivided into three groups: (1) a category in which the maximum QRS vector was in the range between 270° and 32° (proceeding clockwise) (fig. 10, table 5A); (2) a group with the maximum QRS vector between 52° and 141° (table 5B), and (3) a group with the maximum frontal plane QRS axis located within the range of normal (32° to 52°) (table 5C). In this first category (39 cases) superior displacement of the entire frontal plane QRSsE loop was the rule. Paralleling this was a significant superior displacement of the maximum QRS vector (mean value 5°, S.D. 18.5°) from the normal (p < 0.001). The corresponding measurement in the autopsied group was 13° (table 5A). The maximum QRS vector therefore serves as a reliable guide to loop displacement in this group.

The mean value of the 0.02-second vector for the autopsied cases (265°) appears to be representative for this entire subgroup (277°). These findings correspond to those of Pearce and Chapman for the standard electrocardiogram. Also the 0.025-second vector (294°) and the 0.03-second vector (323°) showed pronounced superior deviation. All of these showed significant differences from the normal (p < 0.001).

Location of the maximum QRS vector in this range would ordinarily be associated with counterclockwise rotation of the entire loop. This was noted, for example, in all of our cases with left axis deviation due to left ventricular hypertrophy. Furthermore, in our normal controls the one case with the most extreme leftward displacement of the maximal QRS vector in the frontal plane, assuming clockwise rotation, showed a maximal QRS of 20°. Hence, the observation of initial clockwise rotation in association with the maximum QRS vector in the range between 270° and 20° in the frontal plane, in itself constitutes one of the most reliable signs of apical infarction (figs. 10 and 12). This also applies when the superiorly directed forces are of shorter duration than 0.0275 second. This phenomenon is readily apparent in the vectorcardiogram of figure 5, where the electrocardiogram does not justify this diagnosis.

Of this first group the sagittal plane loop (table 5A) displayed initial counterclockwise rotation in 28 of 38 cases (figs. 10, 11, and 13). The observed values demonstrated some overlap with those found in the normal.
Lateral wall infarction. Diagram (upper) of horizontal plane QRSsE loop. The 0.01-second vector is displaced significantly to the right. The peripheral part of the loop is twisted into a figure-of-eight pattern ("bite"). Horizontal plane vectorcardiogram (lower) shows the latter, one of the characteristic changes of lateral infarct. This was confirmed at autopsy. The electrocardiogram failed to indicate the correct diagnosis. P refers to P loop.
ever, in this group there was considerable individual variation and the two autopsied cases showed marked superior displacement well beyond the range of normal.

In the remaining 10 clockwise rotation was observed in the sagittal plane (figs. 5 and 10). In eight of these 10, additional evidence of either anterolateral or lateral wall involvement was present in the horizontal plane QRSSF loop (table 10). This corresponds with earlier observations made with the cube system.1 Conversely, of the 28 cases with counterclockwise rotation in the sagittal plane only two demonstrated evidence of additional anterolateral infarction.

It is clear then that in this first category with superiorly displaced maximum QRSSF axis in the frontal plane the diagnosis of apical infarction can be made on the basis of the initial clockwise rotation alone. Furthermore, superiorly displaced initial vectors are the rule up to 0.03 second. In these cases ad-

Figure 8

Difference between vectorcardiogram and electrocardiogram in localization of infarct. The Frank vectorcardiogram (third frame) locates the infarct in the anterolateral region (0.01-second vector at 73°, 0.02-second vector at 316°); the electrocardiogram suggests an anteroseptal location. The cube technic also indicated that the infarct was anteroseptal (right-hand frames). At autopsy the infarct was in the anterolateral wall.

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Figure 9

Vectorcardiogram and electrocardiogram in lateral wall infarct. The horizontal plane loop here shows clockwise rotation throughout its entire sweep. The 0.01-second vector is at 102°, 0.02-second vector at 103°, and 0.03-second vector at 111°; this indicates that the anteroseptal region is spared by the infarct. T loop absent in horizontal and frontal planes. The sagittal plane loop rotates counterclockwise in its initial segment. This is obscured by T loop in nonamplified lower frame but, with amplification and "dissection" of loop (upper frame), omitting the T loop, this salient is clearly demonstrated (arrows). The 0.02-second vector is at 160°, the 0.03-second vector at 149°. The frontal plane loop shows counterclockwise rotation. At autopsy the entire free left ventricular wall was infarcted.

Additional anterior myocardial infarction should be suspected if the sagittal plane loop rotates in an abnormal direction. This principle is well illustrated by figures 5 and 12 (table 10).

The remaining 14 patients fell into three small subgroups, none of which satisfied the complete criteria outlined above. The details of these categories follow:

In the second group with apical infarction (eight cases) (table 5B) the maximum frontal plane QRS vector was found in a range between 52° and 141°. In all, the loop rotated in clockwise direction, which is normal in this plane, when the maximum axis is in this range. The diagnosis, therefore, depended entirely on the duration of superiorly directed forces and their direction (fig. 11). Because of wide scatter, Pipberger10 could give no quantitative data for the 0.01- and the 0.02-second vector in the frontal plane. However, comparison of the mean values for this second subgroup of cases with apical infarction with our own control observations and those of Mori31 in a group of older men showed a significant superior displacement of the 0.02-second vector (292°) and of the 0.025-second vector (300°), (table 5B). The 0.03-second vector (338°) was also considerably displaced from the normal value of 28° (p <0.01 in all).

Three of the eight cases showed counterclockwise...
rotation in the sagittal plane. Although the early vectors demonstrated a considerable increase in the duration of superiorly directed forces (cf. tables 5B and 5A), only the 0.03-second vector differed significantly from the normal (219° vs. 138° for the normal).

In the remaining four cases in which sagittal plane observations were made, clockwise rotation was present. Three of these were examined post mortem. Two showed associated anterolateral infarction, while the remaining case demonstrated severe pericarditis and myocarditis, involving the anterior wall of the heart as well as the apical lesion. Thus, clockwise rotation in the sagittal plane appears to occur when the anterior wall as well as the apex is involved (table 10).

In the third subgroup with clockwise rotation in the frontal plane the maximum QRS was found...
in a normal range (32° to 52°, table 5C). Here again, there was marked superior displacement of the early vectors. As in the previous subgroup the 0.03-second vector in these four cases differed significantly from the normal (321° vs. 28°).

All four demonstrated counterclockwise rotation of the sagittal plane QRS loops. All of these had, by vectocardiogram, associated posterobasal infarction. The values for the single autopsied case in this subgroup were in close agreement with the remaining cases. However, in all, these measurements differed very little from the normal.

A last category consists of two patients demonstrating counterclockwise rotation in the frontal plane; both showed abnormal upwardly directed forces. Additional lateral wall infarction was found in each at autopsy (table 5D, fig. 9). In the one patient who demonstrated clockwise rotation in the sagittal plane, the infarct extended into the anterior wall; in the other the rotation was in the expected normal counterclockwise direction and the additional infarct was confined to the lateral wall.

Diaphragmatic Infarction

As in the preceding section, a division into four subgroups was established. The first category consisted of 36 cases showing clockwise rotation in the frontal plane with a maximum QRS vector ranging between 262° and 32°. Three of these came to autopsy (table 6A, figs. 10 and 12). A close agreement was observed for both the autopsied and non-autopsied cases with regard to the 0.02-second vector (319° vs. 329°), the 0.025-second vector (334° vs. 345°), and the 0.03-second vector (346° vs. 360°). Since the very early vectors in this subgroup showed some overlap with normal measurements, clockwise rotation remained the most significant evidence for diaphragmatic infarction in this group, assuming a maximum QRS axis of 7° (± 20.2°). However, the 0.025- and the 0.03-second vectors

Figure 11

Apical infarction (VCG 257) manifested by (1) superior displacement of initial forces; (in the frontal plane the 0.02-second vector is at 257°, 0.025-second vector at 318°, and 0.03-second vector at 328°; clockwise rotation is present. In the sagittal plane the 0.02-second vector is at 202°, the 0.025-second vector at 193°, and the 0.03-second vector at 185°; counterclockwise rotation is present); and (2) by abrupt deviation of loop at 0.04 second ("bite"). The latter was a transient phenomenon, the vectorcardiogram 2 months later (VCG 373) showing similar abnormality of the initial forces with smoothing out of the remainder of the QRS loops. There is evidence of additional involvement of the posterobasal and posterolateral regions, best seen in amplified horizontal plane loop (right frame of each row).
also proved to be significantly different from their normal counterparts ($p < 0.001$).

Thirty of these 56 demonstrated clockwise rotation in the sagittal plane as well (fig. 12). Again, considerable variation was found between individual values in this group, resulting in a large standard deviation (table 6A). All three autopsied cases showed clockwise rotation in this plane in addition to significant superior forces; therefore, a further diagnosis of anterior wall infarction was made. In each case this was confirmed at autopsy (figs. 5 and 12).

In the remaining 26, counterclockwise rotation was present in the sagittal plane (fig. 13). As in previous groups, the variation in individual measurements was wide, resulting in large standard deviations. Distinction from the normal was impossible in many instances.

In the second subgroup there were six cases (table 6B) (fig. 13). Here the maximum frontal QRS vector ranged between 52° and 142°. Counterclockwise rotation existed in all. The vectors at 0.02 second and 0.025 second showed significant superior displacement, when compared with the cases reported by Mori et al.31 and with our normal controls.11 However, this did not apply to the 0.03-second vector.

The observations in the sagittal plane of this group were divided into three cases with clockwise rotation (each of which showed associated anteroseptal or anterior wall infarct, confirmed in the autopsied patient) and three with counterclockwise rotation. The latter were as difficult to separate from normal controls as those presented in table 6A.

There were nine cases in the third subgroup of diaphragmatic infarction. All had their maximum QRSsE vector in the frontal plane within the normal range (32° to 52°, table 6C). Although the observed measurements for the 0.02-second vector (306°), the 0.025-second vector (334°), and the 0.03-second vector (355°) showed more superior displacement than those in group 6B, separation from normal controls remained difficult.

Finally, there remained a fourth subgroup of 10 cases with counterclockwise rotation of the frontal plane QRSsE loop (tables 6D and 6E). In eight the QRSsE loops were located in the right superior quadrant, with their maximum axis between 180° and 270°. All of them presented vectocardiographic evidence of infarction in both the anterior and posterior walls. But since none of these was eventually examined post mortem, further discussion of these cases is omitted.

In summary, it appears that, if the maximal QRS vector lies above 20° in the frontal plane, initial clockwise rotation in this plane is one of the most reliable signs of diaphragmatic infarction (fig. 10). This was present in 56 of the 71 cases and in 94 of the total of 122 cases of both apical and diaphragmatic infarction (table 8). In no other condition, excepting the Wolff-Parkinson-White syndrome, has clockwise rotation been noted in association with such a pronounced degree of superior displacement of the maximum axis.

Finally, superior orientation of initial forces directly upward or leftward is the rule with diaphragmatic infarction. However, since there was some overlap with the normal in the direction of the 0.03-second vector, the instantaneous frontal plane 0.025-second vector was analyzed in more detail. In the data given for normal older men by Mori31 and for younger individuals studied in this laboratory (0.025 second at 36°),11 as well as for the more heterogeneous age group measured by Pipberger,10 the normal extrapolated value for this instant lies below the 0° to 180° axis. By contrast, the 0.025-second vector invariably lies well above this axis (338° for the entire group of 71 patients, 326° for the five who came to autopsy) in patients with diaphragmatic infarction (table 7A). For the total group of 122 cases the direction of this vector was 317°, contrasting with a normal value of 36° ($p < 0.001$). Similar findings have been reported by Milnor and co-workers32 and others.24,30 It is of interest that displacement of the same 0.025-second vector above the 0° to the 180° axis was also found to be the most reliable evidence for diaphragmatic or apical infarction with the cube technic.1

The sagittal plane showed counterclockwise rotation in 69 of 122 cases (fig. 10). For all initial vectors (table 8) studied there was some overlap with the normal, however, if the 0.025-second vector points above the 0° to 180° axis in this plane, apical or diaphragmatic infarction is probably present. The mean value for this vector in all 69 cases was
Two cases with diaphragmatic and anteroseptal infarction. In each case the former was demonstrated by prominent superiorly directed forces in the frontal and sagittal planes, the latter by clockwise rotation in the sagittal plane. The first case (VCG 269) differed from the second (VCG 709) in the presence of an initial early anterior vector (0.03-second). At first sight this suggested that the anteroseptal area was intact but the alternative explanation, that the persistent anterior force was the effect of an (continued at bottom of next page)
Posterobasal Myocardial Infarction

Of the total 33 cases available for analysis, 29 demonstrated counterclockwise rotation in the horizontal plane loop (table 9). Each showed considerable anterior displacement of the initial part of the QRS loop (fig. 11, upper). As in the previous study there appears to be little or no influence on the direction of the vectors at 0.01, 0.02, and 0.03 second in most cases with posterobasal infarcts. Similar statements have been made in the literature. If the infarct extends to the posterolateral region, however, the initial vectors are much more strikingly and distinctly displaced anteriorly (figs. 11 and 13). Since the influence of an area of electrically inert myocardial tissue, ordinarily depolarized late in the QRS interval, should be reflected in the late vectors, it appeared worth while to study the 0.04-second as well as the maximum QRS vectors. The mean 0.04-second vector was located at 16° (normal 356°), a significant difference (p < 0.05), and the maximum QRS at 357° (normal 323°). Since subepicardial infarction of the posterior wall would tend to reduce the magnitude rather than change the direction of late QRS forces, an analysis of the area of the QRS loop anterior to the 0° to 180° axis may also prove worthwhile. This is one of the few circumstances encountered during this entire study in which measurement of the magnitude of the various instantaneous vectors might be more productive than the analysis of the direction alone.

In the remaining four cases with clockwise rotation in the horizontal plane (table 7) differentiation from right ventricular hypertrophy proved very difficult. This is discussed in greater detail later.

Discussion

Electrocardiographic Observations vs. Vectorcardiographic Data

It has long been recognized that the electrocardiographic diagnosis of myocardial infarction, particularly old infarction, is subject to considerable error. Katz, Johnson, Gray, Paton, and others have estimated the percentage of error to be between 20 and 75 per cent. It is known also that a diagnostic electrocardiographic abnormality, present at one time, may disappear partially or completely for a variable period of time, frequently to return at a later date. Patients with well-established inferior myocardial infarction, for example, may lose the diagnostic evidence of their infarction because of a decrease in depth and duration of the Q waves in leads III and aVF (fig. 14). Similarly, patients with old anteroseptal infarction may show a waxing and waning in the voltage of the precordial R waves so that at one time the tracings are and, at other times, are not
Posterobasal infarction. Upper diagram shows horizontal plane QRS loop. The large anterior salience is important in the diagnosis, since the vectors at 0.01, 0.02, and even 0.03 second do not differ significantly in location from corresponding instants in the normal vector loop. Vectorcardiogram shows large superior forces in frontal (0.025-
(continued at bottom of next page)
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characteristic of infarction. If in such instances of known previous infarction some method were available that permanently retained the diagnostic evidence for infarction, one could not help concluding that such a method possessed greater accuracy than the currently available electrocardiogram.

Of the 125 cases falling in groups A and B, all with convincing vectorecardiographic and clinical evidence for myocardial infarction, only 76 in electrocardiograms taken immediately after the vectorecardiogram demonstrated definite evidence of infarction according to the criteria enumerated above. In 18 of the remaining 49 cases (table 11) the electrocardiogram demonstrated infarct in only one area with certainty, whereas the vectorecardiogram indicated infarction of multiple and frequently contiguous areas. In an additional 20, the vectorecardiographic evidence for infarction could be regarded as only questionable. The remaining 11 satisfied none of the diagnostic vectorecardiographic criteria for infarction (9.6 per cent) (figs. 4, 5, 6, 7, and 12). It is to be emphasized that this series is weighted in favor of the electrocardiogram, since in all cases it had been diagnostic for infarction some time in the past; it was this very fact that led to their inclusion in the present study.

When this material was studied from the opposite point of view, that is, from the standpoint of the vectorecardiographic findings in patients who at one time or another had shown diagnostic electrocardiograms, it was found that all such patients exhibited vectorecardiographic evidence of these infarcts at the present time. Furthermore, there were several instances, not included in this analysis of 125 cases, in which electrocardiograms had been interpreted as diagnostic of infarction; each of these patients showed no infarction in the vectorecardiogram and had no infarction at postmortem examination.

A discrepancy between the conventional 12-lead electrocardiogram and the vectorecardiogram as derived by the Frank lead system, is not unexpected in the light of the observations of Frank, Langner, and Pipberger. Among other factors such differences have been attributed to the inconstancy of the direction and magnitude of conventional effective lead axes, and to variations in the dipole position and in body build. A number of systems, including that of Frank, have been designed to correct these errors and have resulted in a narrower range of normal values, less overlap between normal and pathologic measurements, and improved reproducibility of the loops from time to time (fig. 14). Although in isolated instances comparisons were made between the loops recorded with the cube and with the Frank system (figs. 2 and 8), a wholesale confrontation of the two techniques was not attempted.

Of the 24 cases examined at autopsy (group A) it was possible accurately to predict the area of infarction from the vectorecardiogram in 21 cases. In the remaining three, vectorecardiographic localization was inaccurate. In two of these, widespread infarction was found; presumably as a consequence of this the resultant vector incriminated an area in which no infarct was found. In the third case the reason for the inaccuracy is uncertain; it may be explained by a peculiar rotation of second vector at 298°, 0.03-second vector at 308°), and sagittal (0.025-second vector at 221°, 0.03-second vector at 204°) planes establishing presence of diaphragmatic infarct. The horizontal plane shows counterclockwise rotation and preponderance of anterior forces, lasting 0.065 second, with initial rightward component (0.02-second vector at 120°, 0.04-second vector at 54°), indicating posterobasal and posterolateral involvement. This is borne out in the sagittal plane where the loop rotates counterclockwise with 80 per cent of the area of the loop anterior to the null-point. Such rotation in the sagittal plane with marked anterior displacement has been the rule in uncomplicated posterobasal infarction (cf. fig. 11). The electrocardiogram is clearly diagnostic of diaphragmatic infarction; the tall R waves in leads V3R, V3R, and particularly V1 could be confused with right ventricular hypertrophy, but posterobasal infarction can be inferred from this change in the presence of diaphragmatic infarction.

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Effect of inspiration on vectorcardiographic and electrocardiographic ($V_F$, middle continuous line; $V_6$, lower continuous line) evidence of infarction involving apical, posterobasal, and posterolateral region of the heart. In the vectorcardiogram the large superior and anterior forces persist in the frontal and horizontal planes on inspiration, although the projection of these abnormal forces on the sagittal plane is so altered, that they approach variation of normal. This could be demonstrated repeatedly. By contrast the broad prominent Q wave in leads III (not shown in picture), $V_F$, and $V_6$ is replaced by an R wave on inspiration. This observation of a persistent superior vector in the frontal plane vectorcardiogram contrasting with the disappearance of the Q wave in a$V_F$ on inspiration is in accordance with the predicted performance of the Frank lead system. Posterobasal infarction is indicated by the large anterior counterclockwise force in the horizontal and sagittal planes. The “bite,” beginning at 0.0275 second in the frontal and horizontal planes, signals posterolateral involvement. In the electrocardiogram the tall R wave over the right precordium might be attributed to counterclockwise rotation of the heart on its longitudinal axis, but such an interpretation is not justified in the vectorcardiogram.

Figure 14

Pitfalls in Vectorcardiographic Diagnosis

On occasion the vectorcardiographic appearance of myocardial infarction may be simulated by other conditions. These are left bundle-branch block, which may be confused with anterolateral infarction, right ventricu-
lar hypertrophy, which may mimic posterobasal infarction, septal hypertrophy (as part of left ventricular hypertrophy), which may be mistaken for lateral wall infarction, and "giant right atrium," which may produce changes resembling anteroseptal infarction.

The differentiation between left bundle-branch block and anterolateral infarct has been discussed in the previous communication dealing with the cube system. Since left bundle-branch block is regarded as only an electrocardiographic diagnosis and since anatomic evidence for this lesion is frequently not forthcoming, it would appear to be futile to belabor the method of differentiating the two conditions. Furthermore, none of the patients in this series with left bundle-branch block by vectorcardiogram was subjected to postmortem examination. Suffice it to say, all but two of the 14 individuals whose electrocardiogram showed left bundle-branch block demonstrated an initial anterior force by vectorcardiogram as discussed by Frimpter et al. It is in these patients, lacking the characteristic anterior part of the loop, in whom the diagnosis of anterolateral infarct may be considered. This differentiation was possible in a case recently studied by the injection-dissection technic, where the electrocardiographic finding of left bundle-branch block obscured the existence of myocardial infarction but where the vectorcardiogram led to the correct diagnosis.

There is no real problem in differentiating between right ventricular hypertrophy on the one hand and posterobasal infarction on the other, when there is already convincing evidence of diaphragmatic or apical myocardial infarction in the frontal and sagittal planes. The problem may be more difficult when there is no associated diaphragmatic infarction. A distinguishing point may be the direction of rotation of the horizontal plane QRS loop. In 29 of 33 cases of posterobasal infarction counterclockwise rotation was found. We have not observed this direction of rotation in adults with right ventricular hypertrophy, if there is a similar degree of anterior displacement of the loop. Furthermore, in none of the patients with this so-called "pseudo right ventricular hypertrophy" (figs. 13 and 14) was the maximum QRS vector in the frontal plane deviated to the right of 50°, as it is in true right ventricular hypertrophy. It seems possible that there may be a rare case of mild right ventricular hypertrophy in which the horizontal plane QRS loop rotates entirely in a counterclockwise direction, but here the problem of differentiation from posterobasal infarction hardly arises, for there is no comparable degree of anterior salience of the loop.

Twenty-seven patients were observed who displayed a large initial rightward force in the frontal and horizontal plane suggesting lateral wall infarction (table 4). All fell in group B excepting a 32-year-old woman in whom there was considerable doubt that she had actually sustained a myocardial infarction. She had a massive heart, a blood pressure of 180/40, and severe free aortic regurgitation. From the discussion in the previous communication it was concluded that in isolated instances pronounced septal hypertrophy (as seen in one autopsied case) may simulate lateral wall infarction. It appears that the patient under discussion belongs in this category.

In three other patients, each a young woman in the thirties, the vectorcardiogram and the electrocardiogram were interpreted as showing anterior wall infarction (fig. 15). In two of these patients postmortem examination showed a giant right atrium associated with an atrial septal defect of the secundum variety; roentgen-ray examination showed rotation of the heart, so that the bulk of the right ventricle was situated anteriorly. At autopsy there was no evidence of myocardial infarction. The electrocardiogram in all three patients resembled the pattern described by Sodi-Pallares. Rheumatic heart disease with severe right-sided hypertrophy and dilatation, or congenital heart disease with similar changes, may induce QS or QR complexes over the right precordium. These are explained as ventricular cavity potentials transmitted through a giant right atrium or as the effect of a shift of the ventricular mass to the left, displacing the entire QRS field left-
Figure 15

Electrocardiogram and vectorcardiogram in “giant right atrium.” The electrocardiogram of this 45-year-old woman with severe rheumatic heart disease suggests extensive anterior wall infarction. Anterolateral infarct seems to be indicated by clockwise rotation of the horizontal plane loop with the 0.08-second vector at 180° and clockwise rotation in the sagittal plane. However, the presence of a maximum QRS vector in the frontal plane at 120° with clockwise rotation in the plane nullifies the significance of these findings, since with the suspected degree of anterolateral wall infarction, counterclockwise rotation would be expected in the frontal plane (cf. fig. 9). At autopsy no infarction was found.

ward and bringing normal initial negativity toward the center of the chest wall. The abnormal vectorcardiogram in our cases can be explained similarly.

This study was undertaken primarily to establish quantitative criteria for the vectorcardiographic diagnosis of myocardial infarction. Such quantitative data based upon objective measurements, expressed in degrees rather than upon qualitative terminology alone, have not yet been available. It demonstrates that, for the most part, measurements made in patients with infarction fall into discrete and statistically significant groups. It is recognized that the approach in this study had to be peripheral and exploratory. Consequently, it does not forge the final link establishing these criteria as unequivocally valid. In certain instances this method has given decisive information not present in the

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electrocardiogram but the study does not prove beyond doubt the invariable superiority of the "modern" vectorcardiogram over the "traditional" electrocardiogram. The number of autopsied cases studied was too limited to justify such a conclusion. Judgment awaits the accumulation and publication of larger scale postmortem material. Such studies are now underway here and elsewhere.

Summary

Two-hundred six patients with myocardial infarction were studied with the Frank vectorcardiographic lead system and the standard 12-lead electrocardiogram in common use. Distinctive and statistically significant measurements separated these individuals from normal controls similarly studied.

Of 125 patients with convincing evidence of past myocardial infarction all showed abnormal vectorcardiograms. Only 76 of these presented electrocardiograms diagnostic of infarction at the time of the diagnostic vectorcardiogram. Eleven (9.6 per cent) showed no evidence of infarction in their electrocardiographic tracings.

The theory is held by some that modern vectorcardiography should be superior to conventional electrocardiography. The present observations in suspected myocardial infarction suggest that this method may yield confirmatory evidence in most cases, additional and complementary data in some, and decisive information not otherwise available, in a few.

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A Clinical Appraisal of the Vectorcardiogram in Myocardial Infarction: II. The Frank System
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