A Clinical Appraisal of the Vectorcardiogram in Myocardial Infarction

I. The Cube System

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It has been argued that the vectorcardiogram should yield no more information than a standard 12-lead electrocardiogram. Yet many authors, including Wolff, Burch, Scherlis and Grishman and Milnor, employing different lead systems, have published data indicating that the vectorcardiogram may demonstrate abnormalities not apparent in the electrocardiogram.

Of a total of 2,545 vectorcardiograms recorded over a 2-year period in patients hospitalized at the Peter Bent Brigham Hospital 368 fulfilled the criteria, to be described below, for the diagnosis of myocardial infarction. Sixty-seven of these patients were subsequently examined post mortem. The 368 recordings fell into two groups, one in which the Grishman cube (161 cases) was employed, the other in which the corrected lead system of Frank (207 cases) was used. This communication will deal exclusively with the results obtained by the former technic. Based upon the results of a correlation between anatomic and vectorcardiographic findings, a division into distinct groups of vectorcardiographic patterns was established and these were expressed in numerical values. In addition an evaluation was made of the accuracy of the vectorcardiogram recorded by the cube technic as compared with the standard 12-lead electrocardiogram.

It has been stated that the older vectorcardiographic technics including the Grishman cube, are subject to distorting influences similar to those that affect the 12-lead electrocardiogram. These include variations in body build and dipole position resulting in differences in magnitude and direction of the effective lead axes from patient to patient. Consequently a statistical analysis similar to that applied to the results obtained with the Frank system could not be carried out. Despite these disadvantages of the cube system this material is presented because of the availability of this series of autopsied cases and the continued use of this system in many laboratories.

The modern concepts of vectorcardiography depend upon the visualization of the process of depolarization of the heart as a spatial phenomenon. Although the results of this study would most accurately have been expressed in a mathematical formulation involving spatial coordinates or spatial models they are, we think, most practically communicated to and comprehended by the non-mathematical reader when expressed as the projection of these spatial forces upon two or three mutually perpendicular planes. An abnormal spatial vector may be best appreciated in its projection in an abnormal direction upon a particular plane; however, at the same time, the direction of this abnormal spatial vector may be projected upon another plane as if it still were within normal limits. Conse-
subsequently the abnormality in its projection upon one or two planes would suffice to characterize it as abnormal in space. In these studies the magnitude of vectors was not measured. Accordingly wherever the word "vector" is used without qualification, it indicates its direction only.

Methods and Materials

All of the autopsy material in this study was analyzed in the Department of Pathology and reviewed by a senior pathologist. Routine transverse sections were made through the heart ("bread loafing") and the gross areas of fibrosis and infarction carefully mapped. In addition, a detailed drawing of the state of the coronary system was prepared. Microscopic sections were made of the heart muscle at these sites. No injection studies were done.

The majority of the postmortem examinations were performed within 2 weeks of the recording of the vectorcardiogram and the electrocardiogram. In about half of the remaining cases the interval varied from 1 to 2 months. The remainder were included in the tabulations only if there was no clinical evidence for subsequent infarction.

The material of this study fell into three categories:

Group A: Forty-three individuals, who were eventually examined post mortem. In these the diagnosis of myocardial infarction was indubitable.

Group B: Seventy-three patients (not autopsied) in whom the existence of myocardial infarction may be regarded as probable. These individuals gave a classical history of severe substernal constricting pain lasting 1 hour or more and demonstrated at least one of the following:

1. The development of significant elevation of the RS-T segment which subsequently subsided entirely.

2. The development of abnormal Q waves in patients not previously exhibiting them. Depending upon the topography of the infarct, the following electrocardiographic criteria had to be met:
   1. In the instance of anteroseptal infarction, QS complexes in leads V₁, V₂, and V₃ or QS complexes in leads V₁ and V₂ and qR or QR complexes in lead V₃, in the absence of left ventricular hypertrophy or left bundle-branch block.
   2. In anterolateral infarction, QR complexes in leads V₅ and V₆ provided that the Q waves in these leads measured at least 0.03 second in duration.
   3. In high lateral wall infarction, Q waves in leads I and aV₅, measuring at least 0.03 second in duration, confirmed by the presence of broad Q waves followed by R waves in high lateral leads.
   4. In apical or diaphragmatic infarction, Q waves in leads III and aV₃, measuring more than 0.04 second in duration and with a depth of more than 25 per cent of the amplitude of the R wave in the same lead.
   5. It is to be emphasized that there are no clearcut criteria in the contemporary literature for
Figure 2

Genesis of abnormality in initial vector in myocardial infarction: A. Arrows indicate multiple vectors at about 0.03 second. B. Resultant normal vector at 0.03 second. C. Situation when several vectors are absent as consequence of anterolateral wall infarction. D. Deviation of resultant vector ("infarct vector"). (Reproduced from Grant with permission of the author.)

the diagnosis of posterobasal (true posterior, not diaphragmatic) infarction.

Q waves in leads III and aVF measuring between 0.03 and 0.04 second in duration or Q waves in those leads measuring from 10 to 24 per cent of the amplitude of the R wave in the same lead, were considered questionable evidence for inferior infarction. In the absence of evidence for left ventricular hypertrophy or left bundle-branch block, a decreasing amplitude of the R wave, or a failure of the R wave to exhibit a normal progressive increase in amplitude as the electrode was moved from right to left over the right precordium, was regarded as questionable evidence for anteroseptal infarction.

III. A rise of the lactic acid dehydrogenase level in the blood serum in the absence of other known causes of elevated lactic acid dehydrogenase. This was considered significant either as an isolated reading above 200 units or as a series of observations with a dynamic rise of the lactic acid dehydrogenase level to above the normal value of 140 units or as a progressive fall from above to below that value. (The presence of an elevated white blood cell count and erythrocyte sedimentation rate was considered confirmatory evidence but was not required.)

Group C. A group of 45 patients in whom the diagnosis was strongly suspected on clinical grounds and in which the vectocardiogram presented characteristic evidence of myocardial infarction but in which the criteria under group B could not be satisfied. In this group the diagnosis was regarded as presumptive.

In groups A (definite infarction) and B (probable infarction) a comparison was made between the interpretation of the standard electrocardiogram and the vectocardiogram, each recorded on the same day. Cases in group C (possible infarction) were not included in this analysis but the findings in this group were utilized to amplify or to supplement certain observations and conclusions regarding the vectocardiographic diagnosis of myocardial infarction made in groups A and B.

In all cases the routine 12 leads were recorded with a Sanborn direct-writing electrocardiograph. Leads V4R and V9R were included in all instances. The technic of vectocardiographic registration was that devised by Grishman and Scherlis. The system is so wired that the sagittal plane is oriented as if the examiner were looking at it from the patient's left side and the horizontal plane as if it were being viewed from above and from in front. Thus in the reproductions of the normal sagittal plane loop it rotates in a counterclockwise direction anteriorly, subsequently inferiorly, and finally posteriorly, then returns to the null-point.

The recording apparatus consisted of two Tektronix preamplifiers and a Dumont oscilloscope.

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VECTORCARDIOGRAM IN MYOCARDIAL INFARCTION

Figure 4

Discrepancy between electrocardiogram and vectorcardiogram. An 80-year-old woman in whom the electrocardiogram showed no evidence for myocardial infarction but in whom the horizontal-plane vectorcardiogram showed the 0.01-second vector at 360°, the 0.02-second vector at 347°, decisive evidence for anteroseptal infarct. This was confirmed at postmortem examination. PIB, peri-infarction block.

Criteria for Vectorcardiographic Diagnosis of Myocardial Infarction

No large-scale observations have been published on the vectorcardiogram with the cube technic in normal individuals. It has been observed\(^\text{10, 11}\) that there is considerable variability in the configuration of the loop as recorded by the cube system from person to person and from time to time in the
Discrepancy between electrocardiogram and vectorcardiogram. Left ventricular hypertrophy shown by each technic. The electrocardiogram indicates anteroseptal infarct and, according to Grant's criteria, anterolateral peri-infarction block. Vectorcardiogram shows 0.01-second vector at 10° and the 0.02-second vector at 14° in the horizontal plane; this is evidence against antero-septal infarct. Left ventricular hypertrophy but no infarct demonstrated post mortem.

1. Anteroseptal infarct was considered to be present if the initial forces following the 0.01-second vector were directed posteriorly to the 0° to 180° axis in the horizontal plane and posterior to the vertical axis (+90° to +270°) in the sagittal plane.

2. Anterolateral infarct was diagnosed with clockwise rotation of the horizontal plane loop, if the 0.01-second vector was directed rightward and the 0.02-second vector posteriorly.
3. **High lateral wall infarct.** Here it was required that the 0.01- and 0.02-second vectors, regardless of the direction of their rotation, be located between +90° and +180° in the horizontal plane.

4. **Diaphragmatic infarct and apical infarct.** The diagnosis was made if any one of the following three criteria was fulfilled:

   a. Orientation of the 0.02- and 0.03-second vector between 270° and 0° in the frontal plane.

   b. Rotation of the frontal plane loop in a clockwise direction when the maximum QRS vector lay between 270° and 35°. Normally when the maximum QRS vector lies in this range, the loop rotates in a counterclockwise direction,\(^5\)\(^,\)\(^15\)\(^,\)\(^16\)

   c. Location of the 0.02- and 0.03-second vector between 180° and 270° in the sagittal plane.

5. **Posterobasal infarct.** Here it was required that more than 50 per cent of the QRS loop be located anterior to the 0° to 180° axis in the horizontal plane and anterior to the 270° to 90° axis in the sagittal plane and that the loop rotate in a counterclockwise direction in the horizontal plane.\(^17\)

When infarction occurred in more than one topographic area, the vectorcardiographic findings are described separately under the appropriate categories. Thus the total number of infarcts described exceeds the number of patients.

**Results**

1. **Anteroseptal infarct.** The vectorcardiograms of 62 patients fulfilled the criteria for anteroseptal infarct set forth above. Of these, 13 were subsequently examined post mortem (figs. 4 and 5). The average direction of the 0.01, 0.02, and maximum vectors are given in table 1A and B. All 13 autopsied cases showed anatomic evidence of at least anteroseptal infarct. Nine of these showed infarction elsewhere (six apical and one each posterolateral, true posterior, and anterolateral).

Fifty-four of the 62 cases demonstrated counterclockwise rotation in the horizontal plane, 18 after a very short initial clockwise and posterior segment usually of 0.01-second duration. Of the 13 autopsied cases nine rotated counterclockwise, three after such a short clockwise and posterior segment. In each of these three cases the anteroseptal infarct extended to a slight degree into the contiguous lateral or apical areas.

The remaining eight cases demonstrated clockwise rotation in the horizontal plane. Four of them came to autopsy. Two showed apical involvement of pronounced degree by the infarct; the remaining two differed from the others in showing prominent involvement of the mid-portion of the septum. Of the five non-autopsied cases that showed clockwise rotation in this plane, four exhibited associated vectorcardiographic evidence of apical or

### Table 1

<table>
<thead>
<tr>
<th>Rotation</th>
<th>No. cases</th>
<th>Total</th>
<th>Autopsied</th>
<th>Vector in degrees</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.01 sec.</td>
</tr>
<tr>
<td>Horizontal plane (A)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Entirely CCW</td>
<td>36</td>
<td>(6)</td>
<td></td>
<td>349 (327)</td>
</tr>
<tr>
<td>Short initial CW</td>
<td>18</td>
<td>(3)</td>
<td></td>
<td>291 (338)</td>
</tr>
<tr>
<td>CW, predomin.</td>
<td>8</td>
<td>(4)</td>
<td></td>
<td>312 (303)</td>
</tr>
<tr>
<td>Sagittal plane (B)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CCW</td>
<td>33</td>
<td>(7)</td>
<td></td>
<td>66 (51)</td>
</tr>
<tr>
<td>CW – CCW (figure-of-eight)</td>
<td>13</td>
<td>(2)</td>
<td>307 (310)</td>
<td>1 (326)</td>
</tr>
<tr>
<td>CW</td>
<td>13</td>
<td>(4)</td>
<td></td>
<td>269 (10)</td>
</tr>
<tr>
<td>CCW but no values</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CW, clockwise; CCW, counterclockwise.
Figure 6

Significance of rotation in the sagittal plane in evaluation of multiple infarction. Vectorcardiogram shows clockwise rotation of initial vectors in all planes. In the horizontal plane the 0.01-second vector is at 160°, the 0.02-second vector at 178°. The initial vector would have been counterclockwise in the sagittal plane if there were no apical infarct. The latter is indicated by superior displacement of initial vectors in frontal and sagittal plane. Transmural anterolateral infarction with circumferential subendocardial extension into apex demonstrated at autopsy.

anterolateral infarction. Therefore, when the initial part of the loop does not rotate in the expected counterclockwise direction, while the remainder of the loop is quite characteristic of anterosetal infarct, the possibility of infarction involving contiguous areas, especially the apical or anterolateral regions, should be considered.

This was even more clearly demonstrated by the findings in the sagittal plane (table 1B), where clockwise rotation of the QRS loop was found in six of the 13 autopsied cases. Three of these six had associated apical myocardial infarction. In contrast, none of the seven autopsied cases with counterclockwise rotation in the sagittal plane demonstrated apical involvement. Therefore, the presence of clockwise rotation in the sagittal plane, even in the absence of superiorly directed forces sufficient of themselves to establish the diagnosis of diaphragmatic or apical infarction, would suggest that such involvement is present. Of the 49 non-autopsied cases of anterosetal infarction in whom the sagittal plane was studied, only nine demonstrated clockwise rotation; all of these had in addition evidence for diaphragmatic infarction.

Comparison between autopsied and non-autopsied cases with counterclockwise rotation showed close agreement between the values at each of the instants studied (tables 1A and B). This suggests that the observations made
in the patients studied at postmortem examination may be representative of the larger group of observations in the non-autopsied material.

Observations were also made for the frontal plane loop. Since none of these fell outside of the range of normal, they are not reported here. The presence of associated left ventricular hypertrophy changes the observed values very little. The anticipated variations will be discussed in more detail in the subsequent communication.

2. Anterolateral infarct. In the 22 patients with vectorcardiographic evidence of anterolateral infarct two types of horizontal plane loop were seen. Nineteen showed clockwise rotation with the initial 0.01- and 0.02-second vector to the right of and posterior to the nullpoint and the maximum QRS vector in the quadrant between 270° and 360° (fig. 6). In the remaining three cases the loop had a peculiar appearance in that it rotated in a counterclockwise direction to the left for a small portion of its sweep, then continued its counterclockwise course, placing the bulk of the loop to the right of and anterior to the nullpoint (table 2). Again a rather close correlation between the values observed in those cases examined post mortem and the non-autopsied cases was observed. Thus the hallmark of the QRS loop in anterolateral infarction was clockwise rotation in the horizontal plane with a marked rightward direction of the 0.01- and 0.02-second vectors.

The differentiation between anterolateral infarct on the one hand and anteroseptal infarction with clockwise rotation in the horizontal plane on the other (eight out of 62 cases) therefore is determined by the direction of the 0.01- and 0.02-second vectors (fig. 6). In the non-autopsied cases of anterolateral wall infarction these were located at 216° and 285° respectively, while the corresponding values for anteroseptal infarction with clockwise rotation were 312° and 335°. These differences were even more striking in the autopsied cases (tables 1A and 2A). There was no significant difference between the maximum QRS vectors in infarcts in either of these areas.

Study of various instantaneous vectors in the sagittal and frontal planes did not yield any consistent results. In an occasional case the frontal plane loop proved helpful because of counterclockwise rotation in the presence of right axis deviation. Since the findings in the horizontal plane are so typical and so striking, it may be concluded that study of the 0.01- and 0.02-second vectors in the horizontal plane and the direction of rotation are sufficient for the diagnosis of anterolateral wall infarction.

3. Lateral wall infarction. The findings in the seven autopsied and in the 15 non-autopsied cases were difficult to tabulate because in most instances the lateral wall infarct was only part of a larger area of infarction. The autopsied patients who had predominant lateral wall involvement demonstrated extensive rightward displacement of the initial forces in the horizontal plane (table 2B) with the 0.01-second vector at 160° and the 0.02-second vector at 154°. In four of these the horizontal plane loop continued to rotate in a counter-
Table 3

Distribution of Diaphragmatic Infarcts and Apical Infarcts Related to Duration of Superiorly Directed Forces and Direction of Rotation in Frontal Plane

<table>
<thead>
<tr>
<th>Rotation</th>
<th>Autopsy</th>
<th>No. cases</th>
<th>Number with superior forces lasting</th>
<th>Average direction of max. QRS vector*</th>
<th>Peripheral distortion of loop (&quot;bites&quot;)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.0275 to 0.0275 sec.</td>
<td>0.0275 to 0.0299 sec.</td>
<td>0.030 sec. or more</td>
</tr>
<tr>
<td>Maximum QRS: plus 270° thru 0°</td>
<td>Yes</td>
<td>(6)</td>
<td>1</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>clockwise</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maximum QRS: 20 to 60°</td>
<td>No</td>
<td>47</td>
<td>5</td>
<td>4</td>
<td>38</td>
</tr>
<tr>
<td>clockwise</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maximum QRS: 60° to 120°</td>
<td>No</td>
<td>33*</td>
<td>7†</td>
<td>11</td>
<td>12</td>
</tr>
<tr>
<td>Counter-clockwise</td>
<td>Yes</td>
<td>(6)</td>
<td>0</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>C</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>No</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
</tbody>
</table>

*Includes three cases, none autopsied, showing pronounced "bites" in frontal and sagittal planes, but not otherwise fulfilling criteria in text.

†These cases are included because their sagittal plane loop showed superiorly directed forces lasting 0.0275 to 0.0299 second.

clockwise fashion in front of the null-point and then demonstrated a marked reduction from the usual size of the loop with displacement toward the 90° to 270° axis. Similar rotation and loop configuration was observed in the non-autopsied material. Of the three autopsied patients with clockwise rotation in the horizontal plane two showed contiguous posterior and diaphragmatic infarction, and one anteroseptal subendocardial infarction.

Analysis of the data in the frontal plane yielded no consistent results.

4. Diaphragmatic infarction and apical infarction. Diaphragmatic and apical infarcts are grouped together in this study, since with the cube system of registration the difference between the direction of the initial vectors in both the frontal and the sagittal planes was not sufficient to warrant separation into two distinct groups.19

There were 98 cases of whom 15 came to autopsy (tables 3 and 4). The most important finding in these 15 was a prolongation of the superiorly directed initial QRS forces in the frontal or sagittal plane. Regardless of the initial direction of rotation of the loop, this measured 0.0300 second or more in 13 and 0.0225 second in the remaining two. If these initial forces lasted less than 0.0275 second the diagnosis of this type of infarction was not considered justified.20, 22 One of the latter two cases, however, demonstrated a second diagnostic feature, also present in six other autopsied cases, i.e., the presence of clockwise rotation of the QRS loop in the frontal plane when their maximum QRS vector was located in the segment between 270°, proceeding clockwise to 20° (table 3A) (figs. 7 and 8). The remaining 83 non-autopsied cases showed a similar distribution in the abnormality of the initial superior forces. These lasted at least 0.0275 second in all but 12 cases (tables 3A and B). The remaining patients (table 3C) demonstrated counterclockwise rotation in the frontal plane. Six of these came to autopsy; every one of these showed associated anterolateral or posterolateral infarction. All six had superiorly directed forces of 0.030 second or more. The average direction of the maximum QRS vector was 68°, which is beyond the range in which counterclockwise direction of the frontal plane is normally observed. There were three non-autopsied cases in this group; all had superior forces of 0.03 second or more and demonstrated additional evidence for lateral wall infarct in other planes.

According to Young et al.18 the extent of the initial superiorly directed forces in normal individuals studied by the Duchosal sys-
 VECTORCARDIOGRAM IN MYOCARDIAL INFARCTION

Table 4
Distribution of Diaphragmatic and Apical Infarcts Related to Duration of Superiorly Directed Forces and Direction of Rotation in Sagittal Plane

<table>
<thead>
<tr>
<th>Rotation</th>
<th>Autopsy</th>
<th>No. cases</th>
<th>Number with superior forces lasting</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.0225 to 0.0274 sec.</td>
</tr>
<tr>
<td>Clockwise</td>
<td>Yes</td>
<td>(12)</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>56</td>
<td>10*</td>
</tr>
<tr>
<td>Counterclockwise</td>
<td>Yes</td>
<td>(2)</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>21</td>
<td>5†</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>91† (14)</td>
<td>16</td>
</tr>
</tbody>
</table>

*Seven included because of decisive evidence in frontal plane.
†Included because of decisive evidence in frontal plane.

In the sagittal plane counterclockwise rotation is normal for all control series reported8,26 (table 4). Therefore the presence of clockwise rotation by itself is abnormal. This was found in 12 autopsied and 56 non-autopsied cases. In seven of the 12, associated anteroseptal or anterolateral wall infarction was demonstrated, and lateral wall involvement in the remaining five. The opposite was observed in the two autopsied cases with counterclockwise rotation in the sagittal plane; involvement of the posterior or posterolateral wall, rather than the anterolateral region, was present. Furthermore, of the 56 non-autop-
sied cases with clockwise rotation 35 revealed additional evidence of anteroseptal or anterolateral wall infarction in the horizontal plane loop. This suggests that clockwise rotation in the initial portion of the sagittal plane loop, not only indicates infarction of the inferior region of the heart, but in the majority of instances it signifies involvement of the anterior or anterolateral wall of the left ventricle as well19 (figs. 6 and 7).

In summary, the detection of clockwise rotation in the frontal or in the sagittal plane and the measurement of the maximum QRS vector are both so simple and accurate and their reliability as demonstrated in the autopsied cases is so striking, that it should be considered the most important criterion on which the presence of diaphragmatic or apical infarction can be determined. This was present in 68 of the 98 patients with this type of infarction. In 22 of the remaining 30 cases the duration of superiorly directed initial forces, whether rightward or leftward, was 0.0275 second or more, irrespective of rotation of the remainder of the loop. This corresponds in significance to the measurement of a wide and deep Q wave in leads III and aVF of the standard electrocardiogram.27, 28

Figure 7
Value of sagittal plane in multiple infarction. Here the diaphragmatic component of infarct was demonstrated only in vectorcardiogram. The anterolateral component was indicated in both electrocardiogram and vectorcardiogram. Initial vectors rotate clockwise in all planes and show large superior displacement in frontal and sagittal planes. This loop differs from that of left bundle-branch block (fig. 9) in the clockwise rotation of its initial component in the horizontal plane (see insert in horizontal plane) and in the absence of delay in its total duration.
VECTORCARDIOGRAM IN MYOCARDIAL INFARCTION

With an occasional exception the precision and ease with which the short time interval of 0.0275 second to 0.030 second can be measured in an oscilloscopic recording is preferable to similar measurements made in the ordinary electrocardiogram. Furthermore, the data recorded in the sagittal plane are not easily visualized in the standard electrocardiographic tracing. These considerations explain many of the discrepancies found between the standard electrocardiogram and vectorcardiogram (table 5, figs. 7 and 8).

5. Posterobasal infarction. There were 13 clinical and nine postmortem cases showing posterobasal ("true posterior") infarction. In only two of the nine autopsied cases and one of the 13 clinical cases was this infarct limited to the posterobasal region. In nearly all of the remaining cases infarction was also demonstrated post mortem or by vectorcardiographic criteria in the diaphragmatic or lateral aspects of the heart. In the horizontal plane most showed the greater bulk (more than 60 per cent) of the QRS loop to be located anterior to the 0°-to-180° line. Counterclockwise rotation occurred in this plane in seven of the nine autopsied cases; the direction of their 0.03-second vector was at 30°, and of the 0.04-second vector at 14°. Similar rotation and similar direction of the 0.03-(32°) and 0.04-second vectors (9°) was found in 11 of the 13 non-autopsied cases.

Discussion
Findings at Postmortem Examination
One of the most significant findings in the autopsied cases was the infrequency of infarcts involving a single topographic area. Of
the 43 cases examined post mortem only 16 showed such a discrete infarct; 19 showed either two infarcts or a large infarct extending into more than one region. The remaining eight demonstrated more than two infarcts. Indeed, the 16 showing single infarcts presented a vectorcardiogram that was considered "classical" for infarction of that particular area. This contrasts with the complexity of the vectorcardiographic loop in the remaining cases. It is theoretically possible for a second or third area of infarction to displace or even cancel the abnormal forces expected from a single infarct (fig. 2). This is probably one of the reasons for the wide variations in some of the observed values. Nevertheless, in most instances, the vectorcardiographic loop facilitated a more vivid and graphic visualization even of multiple infarcts than did the electrocardiogram.

Excepting four instances in which there was a discrepancy between the number of infarcts predicted from the vectorcardiogram and the findings at autopsy, the QRS-loop abnormalities corresponded in all of the 45 to the findings at autopsy. In one of these four an extreme degree of diffuse necrosis of the left ventricular wall had induced a pattern of posterobasal infarct while the area of myocardial necrosis actually extended from the posterior into the lateral wall.29 In the second case a posterobasal infarct was correctly diagnosed but an extension into the apex of the left ventricle was not detected. In the third patient a lateral wall component of an apical infarction was thought to be present but not confirmed at autopsy. In the last individual there were two discrete myocardial infarctions, one anteroseptal and the other involving the posterior portion of the septum and the contiguous posterior wall of the left ventricle. The vectorcardiogram here showed only the anteroseptal infarct. It is likely that cancellation effects account for the failure of the vectorcardiogram to detect the second infarct.

There were two additional cases, not included in this series, where a divergence was observed between the vectorcardiogram and the postmortem findings. One demonstrated a normal electrocardiogram and vectorcardio-

gram. At necropsy this patient showed an intramural infarct on the posterior wall of the left ventricle, measuring only ½ cm. in its greatest diameter. Classical electrocardiographic and vectorcardiographic findings for anteroseptal infarct were recorded in the second patient. She was a 37-year-old woman with rheumatic heart disease and very severe mitral regurgitation. Striking left atrial enlargement was found at postmortem examination but there was no coronary artery disease. A similar vectorcardiographic phenomenon was observed in three patients during the study with the Frank lead system. A possible explanation for these observations will be presented in the succeeding communication.19

Also not included in this series of myocardial infarctions were four patients in whom the electrocardiogram raised the question of a myocardial infarct. In each instance the vectorcardiogram failed to confirm this diagnosis but showed left ventricular hypertrophy in three. The patient with a normal vectorcardiogram had no heart disease. In the three whose vectorcardiograms showed left ventricular hypertrophy considerable anteriorly directed forces were noted in the horizontal plane, so that both the 0.01- and 0.02-second vectors were anterior to the 0° to 180° axis. In each of these three cases, the electrocardiogram had raised a question of an old anteroseptal infarct, either on the basis of deep QS complexes from V₁ to V₄, or of minimal R waves from V₁ to V₃, which became even smaller as the electrode was moved onto the left precordium. In each of these, autopsy revealed a severe degree of left ventricular hypertrophy; in two the result of severe aortic stenosis and, in the third, of free aortic regurgitation (fig. 5). Two of these patients had been treated for prolonged periods of time as having acute myocardial infarction because of associated R-ST-segment and T-wave changes. It is clear that in these instances the vectorcardiogram was of great value.

Conditions Simulating Myocardial Infarction
The manner in which left ventricular hypertrophy is differentiated from anteroseptal infarction,20 anteroseptal wall infarction from

HUGENHOLTZ, WHIPPLE, LEVINE
VECTORCARDIOGRAM IN MYOCARDIAL INFARCTION

Table 5

Comparison of Vectorcardiographic and Electrocardiographic Findings

<table>
<thead>
<tr>
<th></th>
<th>Group A (Postmortem)</th>
<th>Group B (Clinical)</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Infarct</td>
<td>No Infarct</td>
<td></td>
</tr>
<tr>
<td>VCG</td>
<td>24†</td>
<td>2</td>
<td>55‡</td>
</tr>
<tr>
<td>Diagnostic of Infarction</td>
<td>9</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>VCG</td>
<td>0</td>
<td>1</td>
<td>3§</td>
</tr>
<tr>
<td>Negative for Infarction</td>
<td>0</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>43</td>
<td>6§</td>
<td>73</td>
</tr>
</tbody>
</table>

*Not included in this group were four cases referred to on page 820 in which precise localization of infarcts was inaccurate.
†Included in this group were three cases in which electrocardiogram showed only a single infarct while vectorcardiogram correctly localized more than one area of necrosis as found at postmortem examination.
‡Included in this group were 10 non-autopsied cases in which vectorcardiogram showed more than one infarct while the electrocardiogram indicated only one.
§The total number of patients indicated in this chart differs from that in groups A and B as presented in text because cases in which VCG or autopsy showed no infarct are included, when there was a discrepancy between VCG and ECG.

Giant right atrium,20 and right ventricular hypertrophy of minor degree from true posterobasal infarction is discussed in the subsequent publication.19

Left bundle-branch block can be differentiated from anterolateral infarction on the basis of the following considerations. In left bundle-branch block clockwise rotation in the horizontal plane is preceded by a short but definite initial anterior counterclockwise segment lasting approximately 0.010 to 0.012 second. Such an initial component was observed in all of our cases and in 21 of 25 cases studied by Frimpter et al.31 by the Frank system. In addition, in left bundle-branch block the loop characteristically moves slowly throughout its entire sweep and the time markings are spaced closely together. Such changes are not observed in anterolateral infarction. The contrast between the vectorcardiogram of left bundle-branch block and that of anterolateral infarct is well illustrated in figures 6, 7, and 9.

There were two instances in this series where lateral wall infarction was suspected on the basis of counterclockwise rotation and excessive initial rightward forces in the horizontal and frontal planes. Each was 37 years old and each had severe aortic regurgitation. In the one autopsied case no infarct was found. The explanation for the unusual rightward and anterior forces of the spatial QRS loop in these cases may be the excessive degree of septal hypertrophy. This increased the normal vector, caused by septal depolarization, to such a degree that lateral wall infarction was simulated. However, the obvious physical signs of cor bovinum in patients with such a vectorcardiogram should lead one to suspect the correct diagnosis.

Vectorcardiographic versus Electrocardiographic Findings

In the evaluation of this technic as compared with standard scalar electrocardiography certain considerations should not be forgotten. Either of the technics is subject to two types of error, namely, the error of the method itself and the error of the individual. The former, which is integral to the technic, is the truth that this investigation seeks. But it is subject to certain human factors implicit in the art as opposed to the science of electrocardiography. Furthermore, there is the stubborn resistance of some to the acquisition of a new discipline, on the one hand, and the bias presented by the vested interest, on the other. No one happily invests 4 or 5 years of his career in a technical and intellectual problem then readily concedes its futility.

Bearing these limitations in mind an at-
Figure 9

Left bundle-branch block. (This is the only figure presented in this communication in which the sagittal plane is viewed from the patient's right side.) The loop moves slowly throughout its entire sweep in all planes. The initial counterclockwise rotation in the horizontal plane is best shown in the amplified insert (arrow). The total duration of the QRS in the vectorcardiogram is 0.16 second. At autopsy septal fibrosis, involving the left bundle, was found.

A temptation was made to assess the relative value of the vectorcardiogram, as recorded by the Grishman cube technic, against the standard electrocardiogram in the detection of myocardial infarction. The electrocardiograms were reviewed and interpreted without knowledge of the clinical, pathologic, or vectocardiographic findings. The 122 cases considered were those falling in groups A and B. The results are given in table 5. The electrocardiograms of 10 of the patients in group A and of nine in group B failed to indicate myocardial infarction. In addition there were 17 patients in groups A and B whose electrocardiograms yielded only questionable evidence. In all 36 the vectorcardiogram was interpreted as indicating myocardial infarction. This was confirmed in each case that was studied post mortem (figs. 4 and 7). Such discrepancy need not be too surprising, if one considers the high incidence of undiagnosed myocardial infarction found at autopsy as reported in various series. On the other hand, such a large degree of variance does come unexpected, since it has been stated that the information recorded by an uncorrected lead system cannot be other than that registered by the standard electrocardiographic lead.

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VECTORCARDIOGRAM IN MYOCARDIAL INFARCTION

system. Although this discrepancy may be due to the application of scalar electrocardiographic criteria that are too rigid, the alternative explanation seems more likely, namely, that even the "uncorrected" vectorcardiogram, either by virtue of the method of lead application or of the manner of registration of planar forces on the cathode ray screen, or of both, allows for more strict diagnostic criteria, and in selected cases may lead to the correct diagnosis.

Summary

A total of 161 vectorcardiograms was recorded with the Grishman cube system in patients with suspected myocardial infarction. In the group of 49 cases subsequently examined post mortem and of 73 non-autopsied cases, in whom the diagnosis of infarction appeared certain, a comparison was made between the standard electrocardiogram and the vectorcardiogram taken in succession. The vectorcardiogram demonstrated more infarcts than the electrocardiogram. Of a total of 122 cases in which the vectorcardiogram showed infarction, 19 had normal and 17 equivocal electrocardiograms. A number of simple and easily applicable criteria for the vectorcardiographic diagnosis of infarction are given. It appears that even the older, uncorrected cube-lead system permits a more accurate diagnosis than currently available standard electrocardiographic procedure. It is suggested that this method of exploration may be useful in cases of suspected myocardial infarction in which the routine electrocardiographic examination leaves doubt.

References


The Early History of Instrumental Precision in Medicine

Avenbrugger lived on to see his famous colleague, De Haen, write his fifteen volumes without a word on percussion. Van Swieten did it no greater justice. In his huge history of medicine, Sprengel mentions it as rather subtle. Yet were the contents of this booklet of twenty-two pages, more practically valuable to man than all these men wrote or all the results of the vast and bloody campaigns during which it slept, until in 1808, one year before the grave, when this contented music-loving German died at 87, Corvisart translated it into French, and proclaimed its undying value to a waiting world.—S. Weir Mitchell, M.D., Transactions of the Congress of American Physicians and Surgeons held at Washington, D.C., 1891. New Haven, The Congress, 1892, p. 180.
A Clinical Appraisal of the Vectorcardiogram in Myocardial Infarction: I. The Cube System
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