A Correlative Study of Postmortem, Electrocardiographic, and Spatial Vectorcardiographic Data in Myocardial Infarction

By G. E. Burch, M.D., L. G. Horan, M.D., Joseph Ziskind, M.D., and J. A. Cronvich, M.S.

The spatial vectorcardiograms (sVCG) and electrocardiograms were studied with respect to the gross and microscopic data in 59 instances of myocardial infarction. Besides the early abnormalities in the QRS sF-loop frequently associated with the electrocardiographic Q of infarction, there were recognizable alterations in the sVCG associated with the anatomic location of the infarction: lateral wall infarcts shortened vectors in mid-QRS sF-loop, and posterior lateral basal infarction altered the terminal portion distinctively. The myocardial lesions included fresh infarcts, solid scars, scattered fibrosis, and scar with interwoven intact muscle fibers. Where many normal-appearing fibers were present in the lesion, the previously recorded ECG or sVCG frequently failed to show the diagnostic signs of infarction. However, by presenting the depolarization complex in greater detail, the sVCG supplemented the ECG, improving the accuracy of diagnosis of infarction, especially among the smaller, less solid lesions.

For several years it has been postulated\(^1,2\) and suggested by clinical studies\(^6\)-\(^9\) that the spatial configuration of the vectorcardiogram would be altered in a predictable fashion by myocardial infarction and that these alterations would have some diagnostic significance. As theoretic considerations of electrocardiographic studies had suggested, the early portions of the QRS sF-loop may be visualized as spatially oriented away from the centroid of the infarcted area and toward the centroid of the heart. Previous reports\(^6\),\(^9\) have been based almost entirely upon clinical data; only 3 or 4 hearts\(^5\)-\(^8\) were ever examined at necropsy, usually several weeks to years after recording of the spatial vectorcardiogram. The present investigation was undertaken 2 years ago to study the relation of the electrocardiogram (ECG) and the spatial vectorcardiogram (sVCG) to the clinical data and postmortem gross and microscopic observations in the heart regardless of the cause of death. This report is concerned mainly with the sVCG, and ECG being discussed as specifically indicated. Furthermore, spatial vectorcardiographic alterations produced by infarction that correspond to generally accepted electrocardiographic criteria of this disease are not discussed in detail; only observations of special interest are included.

Materials and Methods

The conventional ECG and the sVCG, obtained with the equilateral tetrahedral reference frame, were recorded on patients at the Veterans Administration Hospital in New Orleans who were considered liable to die within a few days or hours. The incidence of necropsies at this Hospital is high, postmortem examinations having been obtained in about 90 per cent of the fatal cases. Fifty-nine of 160 hearts obtained for careful gross and microscopic study had one or more infarcts of the ventricular myocardium. Hearts with an occasional isolated scar of less than 5 mm, in diameter but without clinical or electrocardiographic evidence of myocardial infarction were not included in this series. Such isolated, small myocardial scars in the septum have been given special consideration previously\(^13\) and will be discussed in further detail in a subsequent report.
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**PATHOLOGIC DIAGNOSIS**

- **INFARCTION**
  - Fresh
  - Stippled fibrosis
  - Coalescent fibrosis
  - Scar
- **HYPERTROPHY**
  - By weight & thickness
  - By weight alone
  - Dilatation alone
  - Dilatation & hyper trophy by weight
  - Scar, extreme thinning of wall
  - Tent but no hypertrophy by weight
  - Metallic erosions
  - Anerysmal bulge (no thinning)

**MAP DIVISIONS**

- Anterior
- Posterior

**ELECTRICAL DIAGNOSIS**

- Unequivocal by conventional criteria
- Infection suggested by terminal deformity (sVCG)
- Suggested infection by pseudo-block pattern (ECG)
- Strongly suggested: Hypertrophy

- Infection suggested by conventional criteria

Figs. 1 and 2. (See legend opposite page.)
CORRELATIVE STUDY OF DATA IN MYOCARDIAL INFARCTION

No generally accepted pathologic criteria exist for left and right ventricular hypertrophy; those based on the tables of Saphir\(^1\) were used in this study. These criteria are reliable when hypertrophy is pronounced and definite but not when it is borderline. Errors may well exist, however, in isolated instances of early hypertrophy in a small heart in which the muscle mass does not exceed the upper limits of normal for the group.

Left ventricular hypertrophy (LVH) was considered to exist if (1) thickness of the left ventricular wall, excluding any trabeculation, exceeded 15 mm. at a site 1 cm. below the valvular ring near the septum anteriorly or if (2) the weight of a heart exceeded the expected mean for the height and weight of the subject by more than two standard deviations.\(^2\) Such criteria as the appearance of a dilated chamber, hypertrophy of the trabeculae carneae, or a lower minimum of thickness of the left ventricular wall were rejected as equivocal.

Unfortunately, the criteria for right ventricular hypertrophy (RVH) were less definitive: (1) pronounced dilatation of the right ventricular chamber, (2) hypertrophy of the trabeculae carneae, or (3) thickness of the right ventricular wall, excluding any trabeculation, of 5 mm. or more at a site near the septum just below the region of the pulmonary conus. Thicknesses of 4 to 5 mm. were regarded as equivocal and were so recorded, and such specimens were not considered to exhibit right ventricular enlargement if criteria (1) and (2) were absent.

The generally accepted electrocardiographic criteria of myocardial infarction were used.\(^3\)-\(^5\) The spatial vectorcardiographic criteria of infarc-

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**Fig. 1 Left.** Summary of the clinical, anatomic, electrocardiographic, and spatial vectorcardiographic data for 33 patients with posterior myocardial infarction. The key to symbols is in figure 2. The clinical features are listed in the following order: (C) cancer, (F) congestive failure, (AP) angina pectoris, (I) myocardial infarction, and (E) enlarged heart demonstrable roentgenographically. The presence of (C) or (E) is indicated by a solid block, their absence by a blank. The number of years of congestive or anginal failure is recorded under items (F) and (AP) except when noted only terminally (T), and the number of clinically recognized episodes of infarction under (I). In the second column, concerned with the coronary arteries (Cor. A), L\(_A\) indicates left anterior descending artery, L\(_L\) left circumflex artery, and R\(_R\) right posterior descending artery. Solid block, arterial occlusion; N, extreme narrowing; blank space, patency. In the third column, the position and depth of the predominant myocardial lesion are shown according to division of the myocardial wall into thirds: N, subendocardial; M, intramural; and P, subepicardial. In the fourth column the anatomic sites of lesions are indicated. The left ventricular cone was considered unrolled and projected on a rectangular grid divided into S, septal; A, anterior; L, lateral; and P, posterior or diaphragmatic quadrants according to conventional landmarks\(^6\) and the anatomic localizations shown at the bottom of figure 2. Each quadrant was divided into thirds, i.e., basal, central, and apical sectors. The predominant lesion for each vertical half-sector was recorded. A similar notation was employed for localization of the lesion as suggested by the conventional electrocardiogram and spatial vectorcardiogram except that all were indicated arbitrarily on the central sector of the grid. However, the vectorcardiographic finding of terminal deformity is appropriately indicated in the grid for the posterolateral basal area. In the column for right and left ventricular hypertrophy, A indicates that the diagnosis was made on the basis of evidence obtained at necropsy, E on the basis of the electrocardiogram, and V on the basis of the vectorcardiogram. Ventricular weights were determined after trimming off the vessels and fat and, when measured after fixation, corrected to fresh weight by multiplying by the ratio of the fresh total weight of the heart to the fixed total weight of the heart.

**Fig. 2 Right.** Summary of the clinical, anatomic, electrocardiographic, and spatial vectorcardiographic data for 26 patients with anterior myocardial infarctions. (See legend of figure 1 for key to abbreviations.) In both charts, in the column labeled Site, the areas of transmural scar with pronounced thinning of the wall are the same as those of aneurysmal bulging except for patient no. 11 (anterior), in whom aneurysmal bulging was not accompanied by thinning. In the column indicating depth of involvement of the wall, the upper half of the block for patient no. 6 represents the predominant anatomic change in the septal wall and the lower half the predominant anatomic change for the anterior wall. Similarly, for patient no. 17, the upper half represents the basal aspect of the lesion and the lower half the apical aspect.
Fig. 3. **Top.** The spatial vectorcardiogram for a subject with a myocardial infarct of the diaphragmatic wall of the left ventricle (patient no. 4, fig. 1).

**Bottom.** The extremely early QRS sE-loop is not displaced upward (thus, no Q in lead III), but there is an early arc-deformity as a result of direction of the trace as though away from the infarcted zone.

**RESULTS**

Results are summarized by figures 1 through 10 and tables 1 through 3. In general, the configurations of the ECG and sVCG varied according to previous expectations. Many of the hearts manifested multiple infarcts. Lesions were classified as posterior or anterior, depending on which predominated in size. Details of the anatomic location and description of the infarcts are presented in figures 1 and 2.

**Posterior Infarcts**

In posterior infarction the early portion of the QRS sE-loop of the sVCG was characteristically directed away from the centroid of the major infarct and toward the centroid of the heart. Thus, it was directed superiorly and to the left, frequently resulting in clockwise inscription in the frontal plane. Occasionally, with infarction of the diaphragmatic wall of the heart, there was upward arching of the inferior edge of the QRS sE-loop involving the early, but not initial, portions of the loop (fig. 3). When the lateral wall was
also infarcted, the midtemporal portion of the loop became straightened or arched, with the convexity directed to the right (fig. 4b and c and fig. 5b and c). When the infarct was "high," i.e., near the atrioventricular groove, and posterior or posterolateral, a characteristic deformity appeared in the terminal portion of the QRS sE-loop, consisting in extremely upward peaking of the trace, followed by a downward dip (or arc with upward concavity) (figs. 4 and 5). All these displacements of appropriate portions in time of the QRS sE-loop, which may be visualized as directed away from the infarcted zone, perhaps resulted from loss of electric activity that would have normally originated in the infarcted areas. This holds not only for the early portions of the QRS sE-loop but also for any other later portions, as has been discussed previously. In 6 of the 33 hearts (18 per cent), posterior or infarcts were found at necropsy when infarction had not been suspected from either the ECG or the sVCG. On careful re-examination of spatial vectorcardiographic traces of these hearts, after completion of the anatomic and pathologic studies, we still failed to find electrocardiographic or spatial vectorcardiographic evidence of infarction. Figure 1 shows the location of these "infarcts" in each heart. Evaluation of the pathologic diagnosis of these 6 hearts provided two interesting characteristics: 1. In 5 of these hearts, the "infarct" was composed not of a large single fibrotic scar but rather of many small, isolated, irregular scars of a few millimeters in diameter, which tended to coalesce. In all instances, however, myocardial tissue around and among the small scars appeared normal (fig. 6). 2. In 3 of the hearts, complete occlusion was not detectable in any coronary artery.

Thus, it appears that such a lesion is not an infarct in the usually accepted sense but is rather a multiplicity of small scars grouped together to give the impression, on casual inspection, of a large, single infarcted area or scar.

As shown in table 1, there was a preponderance of lesions of stippled or coalescing fibrosis among the hearts with posterior infarction. However, when all the examples of discontinuous fibrosis of both posterior and anterior classifications were grouped with all small in-
tramural scars and contrasted to the larger, solid, subendocardial or transmural infarcts and scars, certain relations appeared. Of 31 examples of the first group with "minor" infarction, 22 patients had died as a result of cancer and its complications or from unrelated diseases (ruptured peptic ulcer or fracture of the skull with bronchopneumonia). Thus, only 9 had symptomatic heart disease, and only 4 of these had had clinically recognized angina pectoris or episodes of myocardial infarction. In the second group with "major" infarctions, the clinical state and death of the patient were dominated by cancer in only 5 of 28 instances but by symptomatic heart disease in 23 instances, in 22 of which angina pectoris or clinically recognized episodes of myocardial infarction had been evident. The diagnosis of infarction, on the basis of the ECG and sVCG, was missed in the group with minor infarction on 9 occasions but in that with major infarction only once.

Accuracy of the diagnosis of posterior infarction from both ECG and sVCG is summarized in tables 1 through 3. An unequivocal diagnosis of posterior infarction was made from the ECG alone in 12 of the 33 cases (36 per cent) and from the sVCG alone in 8 cases (24 per cent). In 9 cases (27 per cent), strongly suggestive evidence was found in the sVCG alone and in 4 cases (12 per cent), in the ECG alone (table 3). Thus, the clinical and other data were of considerable importance in arriving at the correct diagnosis before death. From the sVCG of 6 of 21 patients, an accurate diagnosis of a high or basal posterolateral infarct was made on the basis of the terminal deformity in the QRS sF-loop. In 5 of these instances, this infarct was not suspected from the ECG, although late changes in the QRS complex were subsequently interpreted in other tracings, before death of the patients, as suggestive evidence of high or basal infarction.

The general location of the posterior infarcts for 33 hearts and the accuracy of electrocardiographic and spatial vectorcardiographic diagnoses are indicated in table 2. The sVCG and the conventionally recorded ECG were equally accurate in the diagnosis and localization of the posterior infarcts if the mid and late deformities of the QRS sF-loop were considered. The "are-shaped" deformities in the midportions and the terminal deformities of the QRS sE-loops are newly recognized criteria of infarction manifested by the spatial vectorcardiogram. The ECG undoubtedly displays associated deformities
CORRELATIVE STUDY OF DATA IN MYOCARDIAL INFARCTION

TABLE 1.—Infarctions Classified by Site and by Character

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<td>Spotty fibrosis and intramural scars</td>
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<td>9</td>
<td>18</td>
<td>17*</td>
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<tr>
<td>Solid scars or infarcts</td>
<td>28†</td>
<td>23</td>
<td>23</td>
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<td>Total</td>
<td>59</td>
<td>32</td>
<td>41</td>
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*In the ECGs of 3 patients with scattered fibrosis of the posterior wall, the only sign of abnormality was absence of Q in I, V5, and V6. Thus, 3 fewer diagnoses than indicated were missed when this criterion was used.

†Includes arbitrarily one fresh intramural infarct (no. 4 of fig. 1) surrounded by transmural coalescent fibrosis.

at the same time in the cycle, but their recognition has simply not yet been developed. Studies of Langner indicate the value of higher fidelity and more rapid paper and film speed in electrocardiography for demonstrating such changes, but recent experience suggests that some of them can be detected with conventional recording technics. However, whether or not this can be made clinically reliable without improvement in recording technic remains to be determined.

The presence of RVH and LVH did not seem to impair the accuracy of electrocardiographic more than vectorcardiographic inter-

Fig. 6 Left. Cross section of the posteroseptal wall of the left ventricle of a heart with small areas of coalescing fibrosis. The fine stippling throughout the myocardium is visible in the photograph, with clusters of small scars indicated by the arrows; others can be seen on close inspection of the illustration. Right. Microscopic appearance of a section of the left ventricular wall of a heart with coalescent myocardial scarring. Note persistent muscle fibers coursing through the regions of scarring.
pretation of the infarction. Interestingly, either RVH or I.VII was considered to be present, on the basis of the ECG and sVCG, on 4 occasions when it did not actually exist. This type of error is known to occur in myocardial infarction, but the mechanisms and reasons therefore are not clear.

The relation of the size of ventricles to each other and to infarction is shown in figure 7. The 2 ventricles were directly related to each other in size regardless of total weight. Posterior and anterior infarctions were about equally frequent in the normal sized hearts and in those with hypertrophied ventricles (fig. 7). The ventricles of patients with malignant disease were usually small, even in the presence of infarction.

Anterior Infarcts

The spatial vectorcardiographic changes diagnostic to anterior myocardial infarction were essentially as previously described1-12 an abnormal early part of the QRS sE-loop directed as though away from the infarcted zone toward the centroid of the heart (fig. 8). The postmortem studies suggested some points of interest that deserve special comment. In 7 of the 26 hearts (27 per cent) in which anterior myocardial infaracts were found at necropsy, the infarction was not detected from the sVCG (table 1). Four of these, however, were instances of multiple small scars with intervening areas of non-scarred tissue; they presented precisely the same problem described for the multiple coalescing scars in posterior infarction. One heart had a small subendocardial scar beneath apparently viable muscle. Classical left bundle-branch block in one of the patients

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**Fig. 7.** Relation of the right and left ventricular masses of 50 of the hearts with myocardial infarction with symbols to indicate the site of infarction and the presence or absence of cancer. Maximal arbitrary normal limit was set at 50 Gm. for right and 170 Gm. for left ventricular weight. The estimate of hypertrophy by thickness of the wall is not shown but corresponded well to the weight.

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**Fig. 8.** Spatial vectorcardiogram of a subject with anteroseptal myocardial infarction (patient no. 6, fig. 2). Note that in the superior planar view the direction of inscription of the early portion of the QRS sE-loop is to the right and then posteriorly, thus in a clockwise direction away from the anteroseptal junction rather than in the normal counterclockwise direction.
prevented detection of infarction by either sVCG or ECG. For reasons to be discussed, not all such lesions need be expected to produce vectorcardiographic or electrocardiographic abnormalities of sufficient degree to lead to an interpretation of infarction (fig. 9). However, subtle changes of diagnostic importance may exist in these traces, awaiting recognition, since the ECG taken several weeks later was considered to reflect infarction or fibrosis in 2 of the 7 hearts (patient nos. 2 and 26, fig. 2). This may represent a change in conduction about the area of fibrosis, for the pathologic appearance was that of scarring and fibrosis of long standing in each instance. The general location of all the anterior infarcts is shown by table 2.

The accuracy of electrical diagnosis of anterior infarction is summarized in table 3. Accuracy of recognition of the anterior infarcts by sVCG or ECG did not generally differ, except in an occasional patient.

The terminal deformity in the sVCG, although of supplemental use in this group, did not enhance detection of basal scarring and infarction overlooked on ECG, in contrast to the experience with posterior infarctions (figs. 1 and 2). In only one patient with anterior infarction was the terminal deformity in the sVCG the sole diagnostic sign. This may be explained on the basis of the more extensive nature of the infarctions in this group of patients. Also noteworthy is the fact that the terminal deformity appeared in

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**Table 2.** Infarctions not Diagnosed Electrically by Site of Lesion

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<td>slightly anterior</td>
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<td>3 1 1</td>
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<td>Total</td>
<td>59</td>
<td>17* 19</td>
</tr>
</tbody>
</table>

*This sum excludes 3 instances in which the only electrocardiographic evidence of abnormality was absence of Q in I, V₅, and V₆; lesions of the posterior wall but not septal wall were found at necropsy. Thus, if included, these tracings would increase total numbers of those missed to 15 and 20, respectively.

†This sum includes 2 instances in which the lesion was missed in sVCG but suggested in ECG several weeks later, after clinical evidence of a new, fresh infarct had appeared.
Evidence of infarction | Electrical diagnosis |
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Posterior Infarct</td>
</tr>
<tr>
<td></td>
<td>ECG</td>
</tr>
<tr>
<td>Unequivocal by conventional criteria</td>
<td>12</td>
</tr>
<tr>
<td>Strongly suggestive</td>
<td>4</td>
</tr>
<tr>
<td>Suggested by terminal deformity only (sVCG)</td>
<td>5</td>
</tr>
<tr>
<td>Suggested by absence of Q in I, Vs, and Ve only (ECG)</td>
<td>21 (63%)</td>
</tr>
<tr>
<td>Total accurate</td>
<td>12</td>
</tr>
<tr>
<td>Missed</td>
<td>33</td>
</tr>
<tr>
<td>Grand total</td>
<td></td>
</tr>
</tbody>
</table>

*Six examples of pseudoblock pattern included.
†Septal lesions found at autopsy.

3 cases of anterior infarction with posterolateral involvement without, as well as in 5 cases with basal involvement.

The general anatomic location of the infarcts in the 26 hearts in which anterior infarctions predominated is shown in table 1 and figure 2. Nineteen of the 26 hearts had lesions of the posterior wall as well, however. It has long been known that the presence of a posterior lesion may render vectorcardiographic or electrocardiographic detection of anterior infarction more difficult.

As with the varieties of posterior infarction, the loss of electric activity in the region of infarction reduced the local contribution to the successive mean instantaneous vectors. Thus, appropriate portions of the QRS sE-loop were, in general, displaced or oriented away from the region of infarction toward the centroid of the heart.

**Posterior and Anterior Infarction**

The over-all differences in clinical, pathologic, and electric manifestations of the "posterior" and the "anterior" infarcts are presented in figures 1 and 2. As previously noted, anterior infarction was found primarily in patients with symptomatic heart disease and posterior infarction in patients with clinically concealed or asymptomatic heart disease. The anterior descending branch of the left coronary artery was grossly narrow or occluded in all except 2 of 25 instances of anterior infarction in which the coronary arteries were specifically examined; in both of these, however, the apical myocardium was involved, with subsequent electric detection. In only 16 of 31 instances of posterior infarction was the anterior descending branch of the left coronary artery grossly narrow or occluded. In 9 of the remaining 15 the apical myocardium was not involved, and in 6 of these the infarct was not detected from the ECG.

In 3 of these 6, the infarction was detected solely from the sVCG, by its terminal deformity, whereas in the other 3 it was completely missed. The apical portion of the myocardium was involved in all except 2 of the 26 hearts with anterior infarcts but in only 17 of the 33 with posterior infarcts. With allowance for differences in sampling, there is, thus, a rought clinical and anatomic similarity between the group with posterior infarcts and group I of Sayen, Sheldon, and Wolfert and between our group with anterior infarcts and their groups II through IV.

**Left and Right Ventricular Hypertrophy**

The left ventricle was hypertrophied in 20 of the 26 hearts with anterior infarction. Thirteen of these hearts had right ventricular hypertrophy also, and another had right ventricular dilatation (fig. 2). Right ventricular hypertrophy was detected from the ECG in 3 instances and from the sVCG in none, whereas left ventricular hypertrophy was detected from the ECG in 10 instances (3 only suggestive) and from the sVCG in 15. The
accuracy of diagnosis was increased slightly when both types of recordings were used together.

The anterior infarct was missed electrocardiographically in 1 of the hearts with left or right ventricular hypertrophy or both. One heart also had a left bundle-branch block, which is expected to reduce the diagnostic reliability of the ECG, although it has not yet been proved to be a complete handicap in interpreting the sVCG.

In general, ventricular hypertrophy presented essentially similar problems for detection of anterior infarction from the ECG and sVCG as discussed previously for posterior infarction. In only one patient with anterior myocardial infarction did both the ECG and sVCG suggest the presence of hypertrophy when it did not exist. This may be attributable to the localization and extent of the infarcts in this heart.

**Ventricular Aneurysms**

There were 11 ventricular aneurysms, 3 of which were in a miscellaneous group composed of a posterolateral, a posteroapical, and an apical and posteroapical infarct. Infarction in the other 8 instances involved either 3 or 4 of the "quadrants" of the left ventricular cone. Six of these 8 patients with aneurysms associated with extensive myocardial infarction exhibited a common electrocardiogram and electrocardiogram of a subject with anteroseptal and postero-apical myocardial infarction with aneurysmal bulging of the thinned scar in the central portion of the septum (patient no. 22, fig. 2). The superior planar view shows the abnormal rightward and clockwise inscription, which is associated with the early, abnormal, downward deflection in lead I. This configuration of the QRS sE-loop is probably partly due to the left ventricular hypertrophy.

Fig. 10. Spatial vectorcardiogram and electrocardiogram of a subject with anteroseptal and postero-apical myocardial infarction with aneurysmal bulging of the thinned scar in the central portion of the septum (patient no. 22, fig. 2). The superior planar view shows the abnormal rightward and clockwise inscription, which is associated with the early, abnormal, downward deflection in lead I. This configuration of the QRS sE-loop is probably partly due to the left ventricular hypertrophy.
cardiographic pattern: an abnormally prolonged and predominantly upright QRS with a wide, early downward deflection, due to infarction, in leads I, V_L, V_5, or V_6 or any combination of these. The corresponding QRS sE-loop was relatively long, narrow, pointed, and directed upward, slightly to the left, and posteriorly (fig. 10). The trace was inscribed slowly and slightly irregularly, the early portion being to the right of, and posterior to, the isoelectric point. All the loops considered typical had small concave areas directed downward in some portion of the QRS sE-loop, suggesting infarction of the diaphragmatic myocardial wall, in addition to the early “anterolateral” are deformity seen best in the superior plane.

**DISCUSSION**

These studies reveal certain interesting points. For example, instead of a single large scar, 31 hearts had many small, separate, coalescing scars with microscopic evidence of apparently normal muscle distributed among the fibrotic areas. The muscle could have been active enough electrically to produce complexes that did not exhibit evidence of infarction in the ECG and sVCG. Perhaps the plan of choosing subjects who were expected to die soon had the effect of emphasizing 2 bands in the spectrum of myocardial infarction-coronary artery disease. Two disease groups provided most of the material for study: (1) patients who were dying from cancer but whose health had never been seriously jeopardized by their myocardial damage and (2) patients who were dying from serious, symptomatic heart disease produced by repeated major infarction and scarring.

These observations are of interest, first, because a better understanding is needed of the term “infarction” as interpreted by the clinician and pathologist and, second, because absence of electrocardiographic and spatial vectorcardiographic findings suggestive of infarction in a heart with apparent postmortem evidence of an infarct requires better understanding.

Possibly the subjects with lesser degrees of infarction may have had repeated episodes of angiospasm, with or without pain, and, perhaps, occlusion of small vessels with resultant death of small segments of myocardium with formation of a scar. Conceivably, small segments of muscle may also have been injured during the periods of coronary insufficiency, with only gradual death of the segment and formation of scar; several episodes of insufficiency of coronary blood flow and associated disturbances may have been required before death of the segment occurred. In any event, the mechanism by which the scars formed is unknown, but the resultant lesions or “tombstones” were evident at necropsy. Fibers of electrically active myocardium probably coursed around and among the myriad scars. With depolarization and repolarization of these fibers, as much as 90 mv. of potential difference could have been produced across the cellular wall, so that sufficient electric sources among these coalescing scars remained to maintain an essentially adequate order of electric activity throughout the cardiac cycle. Some alterations must have been produced, but they were minor and too subtle to be detected in the records. Septal scars and fibrosis have been shown to produce alterations in the Q wave in certain leads of the ECG of some subjects. This type of anatomic or structural alteration is not merely of academic interest but is of considerable practical importance, especially if the ECG and sVCG are to be evaluated properly from both experimental and clinical points of view.

It would appear preferable, therefore, to regard the lesions in the first group as multiple, coalescing, small scars rather than as a single large infarct. The clinician and pathologist may interpret these lesions as wishes, but any confusion resulting from arbitrary inclusion of both kinds of lesions in a single diagnostic category of infarction is to be avoided if proper electrocardiographic and vectorcardiographic criteria of diagnosis are to be established. Not only are these lesions anatomically and electrocardiographically different but they may also differ in other respects.
More careful postmortem inspection of the heart for disease is seriously needed. All too frequently, the postmortem report supported by a cursory pathologic examination is erroneously considered the final verdict. Only if the postmortem study is adequate should it be considered indisputable. It is believed that slicing of the unrolled left ventricular cone parallel to the endocardial and epicardial surfaces will display the extent of gross myocardial lesions to best advantage. Multiple serial layers may thus be inspected by means of serial slicing. Conventional ring slices fail to reveal adequately, if at all, some of the small lesions, but preference of technic is an individual matter. The significant requirement is a careful, thorough study to insure reliable information. The type, size, location of lesions and associated observations should be carefully described in detail to permit critical clinical, electric, and anatomic correlations.

The temporal phases of electrocardiographic and vectorcardiographic phenomena should also be considered more carefully. Changes in the ECG and sVCG can reasonably be expected to occur during the intervals appropriate for activation of the diseased segment of the myocardium. The present studies confirm the previously introduced impressions that detectable changes in the later portions of the QRS sVCG loop occur when infarction exists in those portions of the myocardium that are depolarized later in the acceision process. Although these sVCG changes are readily recognizable, it was not always possible to see such alterations in the ECG. Thus, attention must be directed beyond the early portions of the ECG and sVCG for detection of infarcts in all portions of the myocardium. During interpretation, the complexes of the ECG and sVCG must be considered temporally, not only from one cardiac cycle to the next but also within each cycle in relation to the simultaneously occurring electric and other physiologic phenomena of all portions of the myocardium.

The diagnosis of ventricular enlargement and hypertrophy by either ECG or sVCG was poor. The high incidence of combined hypertrophy, with the electric effects of one enlarged chamber tending to neutralize that of the other, may account for this. The criticism of den Boer should be kept in mind when attempts are being made to distinguish left ventricular hypertrophy from anterior myocardial infarction; similar orientation of the QRS sVCG loop may result from either. However, in some instances of posterior myocardial infarction, the classical pattern of LVH was deformed only by an otherwise paradoxic upward displacement of the early portions of the QRS sVCG loop. Left ventricular hypertrophy was not suspected from the electrocardiogram, but both lesions were readily suspected from the sVCG.

Experience is necessary to detect readily deformities that indicate specific lesions in the heart. Spatial vectorcardiograms recorded by other methods and reference systems should display changes essentially analogous to those described in this report, provided the methods and reference systems conform to certain general requirements for satisfactory recording of the sVCG. Of course, direct observations with postmortem correlations are necessary to evaluate the accuracy of this statement. Similarly, further inquiry should be made into the repolarization process, which may be as revealing as closer scrutiny of the depolarization complex has been. Although the TsV loops and T waves were not studied extensively in these observations, they did not seem to depart from the patterns predicted by present-day concepts in vectorcardiography and electrocardiography.

The combination of clinical data and electrocardiographic and spatial vectorcardiographic findings resulted in a correct diagnosis of infarction before death in all but 5 instances. Failure to recognize in the ECG and sVCG evidence of infarction in about 15 per cent of the hearts that exhibited postmortem evidence of lesions is not surprising for general hospital medical and surgical services. The percentage would probably be smaller in a cardiologic service, with more careful and complete electrocardiographic exploration. Patients without definite electrocardiographic evidence of myocardial in-
farction are frequently, and appropriately, treated by the clinician for infarction, but with a certain element of doubt. This mental reservation on the part of the clinician sometimes arises from injudicious or excessive faith in the infallibility of interpretation of the tracings. The importance of reliable, meticulously obtained clinical information cannot be overemphasized in the diagnosis and proper management of infarction.

This study was undertaken not to determine whether the ECG or the sVCG was superior but rather to learn what may be obtained from the sVCG, to search for new vectorcardiographic indications of abnormality, and to investigate opinions previously expressed concerning the sVCG and ECG. Our observations indicate the necessity for evaluating the interpretation of the ECG and the sVCG at necropsy. A few fibers of viable and electrically active muscle dispersed among the scars (fig. 6) would probably be sufficient to minimize the traditionally recognized diagnostic electrocardiographic alterations of myocardial infarction. The discrepancy between electric and pathologic diagnoses emphasized the need for studying in greater detail the configurations of the ECG and sVCG in search of reliable, but presently unrecognized, alterations diagnostic of infarction, however subtle they may be.

SUMMARY AND CONCLUSIONS

Of 160 unselected consecutive necropsies of patients in whom the electrocardiogram and spatial vectorcardiogram were recorded shortly before death, 59 hearts had myocardial infarcts. The ECG and sVCG were carefully analyzed with respect to postmortem findings in the heart. Thirty-three myocardial infarcts were classified as predominantly posterior and 26 as predominantly anterior. Right ventricular enlargement was recorded in 26 instances, left ventricular enlargement in 37, combined ventricular enlargement in 24, and ventricular aneurysms in 11.

In addition to previously described total and segmental displacements of the QRS sE-loop with resultant arcs, abrupt shorten-

BURCH, HORAN, ZISKIND, CRONVICH

ing, and other deformities of the QRS sE-loop characteristically directed as though away from the infarction, a deformity of the terminal limb was observed for high or basal posterolateral infarction. This deformity consisted of pronounced upward peaking, followed by a downward dip or arc with upward concavity. The interpretations of the sVCG and ECG were equally efficient in the detection of anterior and posterior infarction, there being individual differences among patients.

Of the 11 patients with ventricular aneurysms, 6 had a similar electrocardiographic pattern: predominantly upright, abnormally prolonged QRS with a definite, early, downward deflection in leads I, V6, V5, or V6, or any combination of these. This was different from that of either LBBB with septal infarction or lateral infarction with a prolonged QRS complex. The QRS sE-loops of 7 patients were alike; they were directed primarily upward and slightly to the left and posteriorly, with early displacement to the right of, and posterior to, the isopotential point and with arcing deformities suggestive of both anterolateral and posterior myocardial infarction.

Perhaps related to the high incidence of combined ventricular hypertrophy was a low incidence of electrocardiographic or vectorcardiographic diagnosis of ventricular hypertrophy. Additional interference with the diagnosis of infarction may have resulted, but there was a fairly consistent direct relation between the sizes of the right and left ventricles regardless of total weight of the heart. The T sE-loops seemed to obey previous predictions for alterations produced by infarction.

Further physiologic, as well as more detailed anatomic, study will be necessary to evaluate more thoroughly the relation between the electric events of the heart and the clinical and pathologic state of the myocardium.

SUMARIO IN INTERLINGUA

In un serie de 160 non-seligite necropsias consecutive de patientes pro qui electrocardiogrammas e spatio-vectorcardiogrammas
CORRELATIVE STUDY OF DATA IN MYOCARDIAL INFARCTION

habeva essebite registrate brevemente ante morte, infarctos myocardial esseva incontrate 59 vices. Le electrocardiograms e le spatio-vectoeardiogrammas esseva analysate detaliatemente con respecto al constatationes facite in le cordes post morte. Trenta-tres infarctos myocardial esseva classificate como predominantemente posterior e 26 como predominantemente anterior. Allargamento dextero-ventricular esseva registrate in 26 casos, allargamente sinistro-ventricular in 37 casos, combine allargamento ventricular in 24 casos, e aneurysmas ventricular in 11 casos.

A parte le previebite descritbe displaciaments total e segmental del ansa sE de QRS con resultante arcos, accurtation abrupte, e altere deformitates del ansa sE le quales es characteristicamente orientate de manera a sugerger le tendentia a distantiar se ab le infarimento, un deformite del segmento terminal esseva observate pro infarimentos posterolateral alte o basal. Iste deformitate consisteva de un pronunciate dentification in alto, sequite per un valle o un arco a concavitate in alto. Le interpretationes del electrocardiograms e del spatio-vectoeardiogrammas esseva equalemente efficace in le detection de infarimento anterior e de infarimento posterior, con differentias inter le patientes individual.

Ex le 11 patientes con aneurysma ventricular, 6 habeva simile configurationes electrocardiographie: Un predominantemente erete e anomalmente prolongate QRS con un definite precoce deflexion in basso in le derivationes I, V_L, V_5, o V_6 o un combination de istos. Iste configuration differeva ab illo de bloco de branca sinistre con infarimento septal o infarimento lateral con prolongation del complexo QRS. Le ansas sE de QRS de 7 patientes esseva le mesmes. Illos habeva un direction primarimente in alto e levemente postero-sinistrorse, con displaciament prococe al latere dextere, posterior al puncto isopotential e con deformitates arcante que sugervene infarimento myocardial tanto antero-lateral e etiam posterior.

Possiblemente relationate con le alte incidencia de combine hypertrophia ventricular eseva un basse incidence de diagnoses electrocardiographie o vectoeardiographie de hypertrophia ventricular. Il es possibile que un interferentia additional con le diagnose de infarimento esseva le resultato, sed il existeva un satys constante relation inter le dimensions del ventriculos dextere e sinistre, sin reguardo al peso total del corde. Il pareva que le ansa sE obediva previe prediictiones in re le alterationes produceite per infarimento.

Studios physiologic additional e etiam plus detaliate studios anatomic va esser necessari pro evalutar plus precisemente le relation inter le evenimentos electric del corde e le stato clinic e pathologic del myocardio.

REFERENCES


The authors analyzed 1950 electrocardiograms to determine the significance of depression of the T-U segment and of the presence of natural Q waves in certain leads. A trough-like deformity of the T-U segment extending below the level of the U-P segment was not found in 500 electrocardiograms of healthy adults. It was observed once (Fallot's tetralogy) in 250 patients with noncoronary heart disease and among 15 with cardiac pain it was the only abnormality in 7. A natural Q wave in adults is defined as one not over 0.04 second wide and between 1 to 3 mm. deep except in lead 3 where it may be deeper. Among 500 electrocardiograms in healthy adults natural Q waves were not isolated to leads 1, 2, 3 or CR, or confined to the following combinations of leads: 1 and 2; 1 and CR; 2 and CR; CR, and CR; 1, 3 and 3R; 1, 2 and CR; 2, CR, and CR; 1, 3, 3R and CR; 2, 3, 3R and CR; 3, 3R, CR, and CR; 1, 2, 3, 3R and CR; 1, 3, 3R, CR, and CR. For this reason, natural Q waves so occurring were regarded as evidence of myocardial injury. Although natural Q waves in leads 1, CR, and CR, are found in healthy adults, the combination occurred so much more frequently in older persons and those who later developed myocardial infarction that this combination may likewise prove to be an early electrocardiographic sign of a myocardial injury.
A Correlative Study of Postmortem, Electrocardiographic, and Spatial Vectorcardiographic Data in Myocardial Infarction
G. E. BURCH, L. G. HÖRAN, JOSEPH ZISKIND and J. A. CRONVICH

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