Comparative Quantitative Analysis of the Electrocardiogram and the Vectorcardiogram

Correlations with the Coronary Arteriogram

By David R. McConahay, M.D., Ben D. McCallister, M.D., Franz J. Hallermann, M.D., and Ralph E. Smith, M.D.

SUMMARY
Simultaneously recorded standard electrocardiograms (ECG) and Frank vectorcardiograms (VCG) were correlated with the coronary arteriograms and the left ventriculograms of 210 patients. Quantitative criteria were applied to ECG measurements made by manual technics and to VCG measurements obtained by computer technics.

As the extent of significant arteriographic disease increased, the frequency of ECG and VCG evidence of “definite” myocardial infarction (MI) increased. The VCG was diagnostic of MI in a greater number of patients than the ECG (50 versus 39, $P < 0.01$) and excelled in the detection of multiple areas of infarction in a greater number of patients (13 versus six, $P < 0.05$). This increased sensitivity was gained in anterior, inferior, and true posterior infarcts. Excluding patients with myocardial disease, all MIs diagnosed from the VCG and all but one from the ECG were associated with significant arteriographic disease in the distribution of the predicted nutrient artery.

Quantitative VCG analysis appears superior to the usual ECG analysis in the accurate detection of MI. When MI is diagnosed by either technic, significant associated coronary artery disease can be anticipated in a predictable anatomic distribution.

Additional Indexing Words:
Coronary heart disease Computer Ventriculography

SELECTIVE cinecoronary arteriography has offered the clinician a unique opportunity to demonstrate the anatomy of the coronary arteries in the living patient and to correlate this anatomy with the clinical manifestations of coronary artery disease. Any study correlating electrocardiographic data with coronary arteriographic findings must be made with a full awareness that information from arteriography is limited to a demonstration of the anatomic distribution and extent of major coronary atherosclerosis and collateral vessels and can only inferentially suggest the presence of associated myocardial ischemia or necrosis.\textsuperscript{1, 2}\ In contrast, the conventional scalar electrocardiogram (ECG) and corrected orthogonal vectorcardiogram (VCG) can detect evidence of myocardial ischemia and associated infarction but provide no direct information about the coronary arterial anatomy which underlies such ischemia. Nevertheless, such a study correlating the electrocardiographic findings and the associated in vivo coronary anatomy would permit an assessment of the screening value of the ECG and the VCG and allow a valid comparison of the ability of various ECG and VCG criteria to detect myocardial damage accurately.

Despite early reports\textsuperscript{3-5} purporting theoretical superiority of the VCG over the

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Table 1

Characteristics and Duration of Chest Pain According to Distribution of Coronary Arteriographic Disease

<table>
<thead>
<tr>
<th>Distribution of arteriographic disease</th>
<th>Sex</th>
<th>Mean age (yr)</th>
<th>Characteristics of pain</th>
<th>Duration of pain (mo)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Absent</td>
<td>Non-anginal</td>
</tr>
<tr>
<td>No disease</td>
<td>M</td>
<td>F</td>
<td>42</td>
<td>20</td>
</tr>
<tr>
<td>Single vessel (RCA 16; LAD 21)</td>
<td>30</td>
<td>7</td>
<td>49.4</td>
<td>0</td>
</tr>
<tr>
<td>Double vessel (LCA 3; LAD + LCx 11; RCA + LAD or LCx 23)</td>
<td>31</td>
<td>6</td>
<td>49.3</td>
<td>2</td>
</tr>
<tr>
<td>Diffuse (3 vessels)</td>
<td>69</td>
<td>5</td>
<td>50.9</td>
<td>0</td>
</tr>
</tbody>
</table>

*Five with aortic valve disease, four with idiopathic hypertrophic subvalvular stenosis, and two with no evidence of heart disease.

Abbreviations: LCA = main left coronary artery; LAD = left anterior descending artery and its diagonal branch; LCx = left circumflex artery and its diagonal branch; RCA = right coronary artery.

Methods

Two hundred ten patients who underwent cinecoronary arteriography and left ventriculography were studied. The patients were divided into those with and without coronary artery disease. Cinecoronary arteriography and left ventriculography were recorded in both right and left anterior oblique projection, using the 10-inch mode of the CRIM camera and a 50-cm focal spot. The cinecoronary arteriograms and cine-left ventriculograms were recorded in the right anterior oblique projection using the 10-inch mode of the CRIM camera and a 100-cm focal spot. The cinecoronary arteriograms and cine-left ventriculograms were recorded in the right anterior oblique projection using the 10-inch mode of the CRIM camera and a 100-cm focal spot. The cinecoronary arteriograms and cine-left ventriculograms were recorded in the right anterior oblique projection using the 10-inch mode of the CRIM camera and a 100-cm focal spot.

Two hundred ten patients who underwent cinecoronary arteriography and left ventriculography were studied. The patients were divided into those with and without coronary artery disease. Cinecoronary arteriography and left ventriculography were recorded in both right and left anterior oblique projection, using the 10-inch mode of the CRIM camera and a 100-cm focal spot. The cinecoronary arteriograms and cine-left ventriculograms were recorded in the right anterior oblique projection using the 10-inch mode of the CRIM camera and a 100-cm focal spot.
Table 2
Grading of Occlusion of Coronary Arteries

<table>
<thead>
<tr>
<th>Oclusion (%)</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-25</td>
<td>1</td>
</tr>
<tr>
<td>26-50</td>
<td>2</td>
</tr>
<tr>
<td>51-75</td>
<td>3</td>
</tr>
<tr>
<td>76-95</td>
<td>4</td>
</tr>
<tr>
<td>96-99</td>
<td>5</td>
</tr>
<tr>
<td>100</td>
<td>6</td>
</tr>
</tbody>
</table>

diatrizoate (Renografin 76%) within 3 to 4 sec. Selective coronary arteriograms taken at 60 frames/sec using the magnifying mode of the amplifier were recorded in multiple right and left anterior oblique positions with hand injections of 8 to 10 ml of this contrast medium utilizing the technic described by Sones and Shirey.16

Each study was interpreted independently by at least two of the authors without knowledge of the patient's clinical or electrocardiographic status. In each instance the major coronary arteries and their branches including the left main coronary artery, the left anterior descending and left circumflex arteries and their diagonal branches, and the right coronary artery and its marginal branch were studied, and each was assigned a grade based on the most severe luminal narrowing noted (table 2). The location and extent of collateral vessels were also recorded. Arterial dominance (right, left, or balanced) was defined by which vessel (right coronary, left circumflex, or both arteries) supplied the posterior descending coronary artery.

Based on pathologic studies by James,17 the expected arterial supply to the various areas of left ventricular myocardium is summarized in table 3.

The right anterior oblique projection of each left ventriculogram was qualitatively evaluated for localized and generalized disorders of myocardial contraction. This visual analysis was facilitated by recording single end-systolic and end-diastolic tracings of the internal contour of the left ventricular chamber.

Electrocardiogram and Vectorcardiogram

Electrocardiograms and vectorcardiograms were obtained simultaneously by methods previously described.18 19 During a 10-sec recording period, six channels of analog data were acquired on-line in storage buffers of a digital computer.* During this acquisition, analog to digital conversion resulted in discrete voltage measurements for each lead at 4-msec intervals. Modified Frank orthogonal electrocardiograms4 were recorded continuously with the patient supine and the thoracic electrodes at the level of the fifth intercostal space. Standard electrocardiographic leads in four sets (I, II, III; aVR, aVL, aVF; V1 to V6) were switched electronically every 2.5 sec.

Automatic digital recognition and measurement programs were then applied to the Frank orthogonal electrocardiogram. Individual waves were recognized, and their onsets and terminations were determined. All of the waves of each kind (P, QRS, T) were compared, and a typical complex was automatically selected for further analysis. The voltages of the X, Y, and Z leads of this complex were printed in segments timed from the start of the R wave (fig. 1).

The location of the initial 20-msec QRS vector, the amplitude and duration of the initial rightward, anterior, and superior QRS forces, the magnitude of the maximal leftward deviation of the initial superior QRS forces, and the maximal anterior QRS accession time were derived as shown in figure 1. The VCG was then analyzed by application of quantitative criteria to these data provided by the computer readout. In addition, the VCG loops were automatically inscribed as illustrated in figure 2, using Helm's system of notation.20 and the X, Y, and Z leads were displayed in scalar form with a time base of 100 mm/sec. These reconstructed loops were used only to facilitate analysis of their contour and to derive the direction of the maximal QRS vector.

Characteristic ECG complexes from each of the four sets of leads also were selected by computer programs and displayed using digital-to-analog plotting programs with a time base of 50 mm/sec and the voltage-height relationships recommended by the American Heart Association Committee on ECG Standards21 (fig. 2). These electrocardiograms were measured subsequently by the usual manual technics.

Table 3
Expected Location of Arteriographic Occlusions Associated With Myocardial Infarctions17

<table>
<thead>
<tr>
<th>Site of infarct</th>
<th>Associated arterial disease*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anteroseptal</td>
<td>LAD</td>
</tr>
<tr>
<td>Anterior</td>
<td>LAD</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>LAD + LCx</td>
</tr>
<tr>
<td>High lateral</td>
<td>LCx</td>
</tr>
<tr>
<td>Inferior</td>
<td>RCA (if right coronary dominant)</td>
</tr>
<tr>
<td></td>
<td>LCx (if left coronary dominant)</td>
</tr>
<tr>
<td>True posterior</td>
<td>RCA (if right coronary dominant)</td>
</tr>
<tr>
<td></td>
<td>LCx (if left coronary dominant)</td>
</tr>
</tbody>
</table>

*Abbreviations as defined in tables 1 and 5.

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*IBM 1800.
Scalar Electrocardiographic Criteria

The "definite" criteria were selected from generally accepted sources in the literature, but the "possible" criteria were arbitrarily conceived by the authors to extend the sensitivity of the "definite" criteria. These criteria may be outlined as follows:

Anteroseptal myocardial infarction

Definite: One of the following (in the absence of left ventricular hypertrophy or left bundle-branch block):

1. QS deflection in leads V1, V2, and V3,22 or
2. QS deflection in V1 with qR in V2 and V3
3. QS or QR in V1 with QR in V2 and V3

(Q of 0.04-sec duration and amplitude greater than 25% of following R wave)

Possible:

1. Above criteria in presence of left ventricular hypertrophy, or
2. QS deflection in V1 with qR in V2 and V3

Anterior myocardial infarction

Definite:11 Presence of normal septal q waves in leads I, V5, and V6 with one of following:

1. Presence of an initial R in V1 followed in V2 to V4 by a Q of 0.04-sec duration and amplitude greater than 25% of R wave
2. In absence of left ventricular hypertrophy or left bundle-branch block, a decreasing amplitude of initial R wave on going from V1 to V4

Possible: Normal septal forces + Q in V3 to V4 of 0.02 to 0.04-sec duration or amplitude of 10 to 24% of following R wave

Anterolateral myocardial infarction

Definite:12 rS deflection in V1 and V2 with a decrease in amplitude of R wave in V3 and development of a Q in V4, V5, and V6 of 0.04-sec duration and depth greater than 15% of total amplitude of QRS complex

Possible: Q wave in leads V4, V5, and V6 of 0.03 to 0.04-sec duration and depth greater than 10% of total amplitude of QRS complex

Figure 1

Portion of printout from computer program (terminal part of P-R segment, R wave, and initial part of T wave) with discrete voltage measurements in X, Y, and Z leads obtained at 4-msec sampling intervals, illustrating source of certain data from a patient with a normal VCG. The polarity is such that a vector directed leftward, anteriorly, and superiorly inscribes a positive deflection. Onset of the R wave in this example occurs at 108 msec and is directed anteriorly, superiorly, and to the right.
High lateral myocardial infarction

Definite: One or both of following:
1. Q wave in aV\textsubscript{L} of 0.04-sec duration and 50% of amplitude of following R wave (in the presence of upright P waves in the same lead and in the absence of severe left ventricular hypertrophy)
2. Q wave in lead I of 0.04-sec duration and greater than 10% of amplitude of total QRS complex (in absence of severe left ventricular hypertrophy)
Possible: Q wave in lead I or aV\textsubscript{L} of 0.03 to 0.04-sec duration

Inferior myocardial infarction

Definite: One or both of following:
1. Q wave in leads III and aV\textsubscript{F} of more than 0.04-sec duration with a depth greater than 25% of amplitude of following R wave in same lead\textsuperscript{22}
2. Initial R wave in aV\textsubscript{R} exceeding 1 mm (0.1 mv)\textsuperscript{23}
Possible: Q wave in leads III and aV\textsubscript{F} of 0.03 to 0.04-sec duration and 10 to 24% of amplitude of R wave in same lead

True posterior myocardial infarction

Definite: Initial R wave in V\textsubscript{1} or V\textsubscript{2} or both of 0.04-sec duration with RV\textsubscript{1}/SV\textsubscript{1} equal to or greater than 1 (in patients over 30 years of age and in absence of right bundle-branch block, right ventricular hypertrophy, or Wolff-Parkinson-White syndrome)

Left ventricular hypertrophy: One or more of following:
1. Amplitude of S wave in lead V\textsubscript{1} + maximal R wave in lead V\textsubscript{5} or V\textsubscript{6} greater than 35 mm\textsuperscript{24}
2. Maximal R wave in lead V\textsubscript{5} or V\textsubscript{6} greater than 26 mm\textsuperscript{24}
3. Maximal S wave in lead V\textsubscript{1} or V\textsubscript{2} + R wave in lead V\textsubscript{6} greater than 40 mm\textsuperscript{25}

Vectorcardiographic Criteria

Quantitative VCG criteria for the diagnosis of myocardial infarction based on anatomic correlative studies but in some instances on sound clinical correlative efforts were selected from the recent literature for application in this report. Certain modifications were made in some criteria in an attempt to reduce the incidence of false positive diagnoses. Again, the possible criteria were arbitrarily selected to extend the sensitivity of the definite criteria. The VCG criteria are as follows:

Anteroseptal myocardial infarction

Definite: Absent initial anterior QRS forces with either posterior and leftward displace-
element of initial 20-msec QRS vector if septal forces are absent or posterior and reduced rightward displacement of initial 20-msec vector if septal forces are only diminished. Possible: Initial posterior displacement of QRS vector for less than 20-msec duration

Anterior myocardial infarction
Definite: Preservation of initial septal forces but reduced anterior QRS forces with displacement of 20-msec QRS vector and efferent limb posteriorly and to left of E point (in absence of left ventricular hypertrophy or left bundle-branch block).

Anterolateral myocardial infarction
Definite: Displacement of 20-msec QRS vector to right and displacement of efferent limb to right and posterior with initial rightward QRS forces of greater than 22 msec in duration and greater than 0.16 mv in amplitude.
Possible: Initial rightward displacement of QRS loop for a duration greater than 22 msec

High lateral myocardial infarction
Definite: Displacement of initial QRS deflection and efferent limb inferiorly and slightly to right with counterclockwise inscription of QRS in frontal plane despite a vertically displaced maximal QRS vector greater than +40°.
Possible: Displacement of initial QRS deflection and efferent limb superiorly and to right with counterclockwise inscription of QRS in frontal plane despite a maximal QRS vector greater than +40°.

Inferior myocardial infarction
Definite: With initial superiorly directed QRS forces:
(1) One or more of the following:
(a) Early superior QRS forces must be completely clockwise in rotation, have an upwardly convex contour located more leftward than initial superior forces, and be 20 msec or more in duration and more than 0.25 mv in leftward deviation—if almost completely clockwise in rotation, then early superior forces must be 25 msec or longer
(b) Completely clockwise early superior QRS forces associated with a maximal QRS vector above +10°
(2) With initial inferiorly directed QRS forces: Initial inferior QRS forces must be rightward and completely clockwise in rotation and must be followed by early superior QRS forces of an upwardly convex contour located more leftward than initial superior forces—early superior forces must be 25 msec or more in duration and more than 0.25 mv in their most leftward deviation.

Possible: One or more of the following:
(1) Initial superior displacement of QRS loop for a duration greater than 25 msec
(2) Completely clockwise early superior QRS forces in frontal plane with maximal QRS vector between +20° and +10°

True posterior myocardial infarction
Definite: All of following:
(1) Maximal anterior QRS voltage of 0.5 mv or more
(2) Accession time of maximal anterior QRS forces of 30 msec or more
(3) Ratio of maximal anterior QRS voltage to maximal posterior QRS voltage greater than 1
(4) Total anterior QRS duration of 42 msec or more
(5) Anterior displacement of afferent limb of QRS loop in transverse plane

Possible: Four of the above criteria

Left ventricular hypertrophy: Maximal QRS magnitude in transverse plane greater than 2.2 mv if below 50 years of age, or greater than 1.8 mv if 50 years old or older (in absence of left bundle-branch block).

In both the ECG and the VCG analyses, depolarization and repolarization abnormalities were assigned a grade based on the criteria in table 4. ECGs and VCGs were interpreted independently of each other and without knowledge of the patient’s clinical or coronary arteriographic findings.

Results

Sixty-two patients had no stenotic lesions exceeding 50% of the arterial lumen and were thus considered in this study to be functionally normal. Thirty-seven patients had significant obstructive disease (>50% luminal narrowing) limited to a single vessel and its branches; 37 had such involvement of two major arteries and their branches; 74 had such obstructive lesions in all major coronary arteries (table 1). Of 122 patients in whom coronary dominance was determined, 94 (77%) had right coronary dominance, 24 (20%) left coronary dominance, and four (3%) a balanced circulation.

As expected, the character of the presenting chest pain became more typical of angina pectoris as the extent of coronary artery disease increased (table 1). Only two of 50
patients with normal arteriograms and without evidence of obstruction of the left ventricular outflow tract complained of typical angina pectoris. In contrast, 68 of 74 patients with diffuse coronary disease presented with symptoms typical of angina pectoris.

The longer a patient had experienced angina the more likely he was to have extensive arteriographic disease. Forty-one of 74 patients with diffuse coronary artery disease had experienced angina pectoris for longer than 48 months. However, only five of 25 patients with isolated disease of the left anterior descending or right coronary artery without aortic valve disease had noted typical angina for more than 36 months prior to study. Thus the likelihood of finding an isolated lesion in a single coronary vessel at arteriography in the preoperative evaluation of a patient who has experienced typical angina for more than 3 years is small. Nevertheless one patient in this study experienced angina for more than 10 years prior to arteriographic demonstration of single-vessel disease.

Five of 74 patients with diffuse arteriographic disease and four of 37 patients with two-vessel disease had experienced typical angina for less than 12 months but only seven of 37 patients with single-vessel disease had a similarly brief duration of typical angina.

The eight patients in this series with left bundle-branch block and the 10 with either idiopathic hypertrophic subaortic stenosis or idiopathic myocardioathy are excluded from the subsequent results but will be discussed separately.

**Correlation of ECG and VCG With Coronary Arteriogram**

One hundred forty-nine patients in this series had neither ECG evidence of left ventricular hypertrophy nor clinical evidence of rheumatic heart disease (table 5). Of this group, 110 had arteriographic coronary dis-

### Table 4

**Grading of ECG and VCG Abnormalities**

<table>
<thead>
<tr>
<th>ECG-VCG abnormality</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>0</td>
</tr>
<tr>
<td>Nonspecific repolarization abnormality</td>
<td>1</td>
</tr>
<tr>
<td>Localized &quot;ischemic&quot; repolarization abnormality</td>
<td>2</td>
</tr>
<tr>
<td>Possible myocardial infarction</td>
<td>3</td>
</tr>
<tr>
<td>Definite myocardial infarction</td>
<td>4</td>
</tr>
<tr>
<td>Grades 4 and 2 in same location</td>
<td>5</td>
</tr>
</tbody>
</table>

### Table 5

**Correlation of Incidence of Definite Infarctions on ECG and VCG With Extent of Arteriographic Coronary Disease**

<table>
<thead>
<tr>
<th>Arteriogram</th>
<th>Total</th>
<th>ECG</th>
<th>VCG</th>
<th>Combined</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>39</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Single-vessel disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAD</td>
<td>15</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>RCA</td>
<td>10</td>
<td>2</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Double-vessel disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LCA or LAD-LCx</td>
<td>11</td>
<td>2</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>RCA + LAD or LCx</td>
<td>16</td>
<td>1</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Diffuse 3-vessel disease</td>
<td>58</td>
<td>23</td>
<td>24</td>
<td>26</td>
</tr>
<tr>
<td>Totals</td>
<td>149</td>
<td>31</td>
<td>40</td>
<td>42</td>
</tr>
<tr>
<td>Excluding RHD and LVH</td>
<td>192</td>
<td>39</td>
<td>50</td>
<td>53</td>
</tr>
<tr>
<td>Including RHD and LVH</td>
<td>143</td>
<td>39</td>
<td>50</td>
<td>53</td>
</tr>
<tr>
<td>Patients with abnormal arteriograms</td>
<td>143</td>
<td>(27.3)*</td>
<td>(35.0)</td>
<td>(37.1)</td>
</tr>
</tbody>
</table>

*Figures in parentheses indicate percentages.

Abbreviations: LCA = main left coronary artery; LAD = left anterior descending artery and its diagonal branch; LCx = left circumflex artery and its diagonal branch; RCA = right coronary artery; RHD = rheumatic heart disease; LVH = left ventricular hypertrophy.

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ease of at least grade 3 severity. On the basis of the described ECG criteria, definite myocardial infarcts were detected in 31 patients (28.2% of those with abnormal arteriograms). In contrast, definite infarcts were diagnosed from the VCG in 40 patients (36.4%), a difference of statistical significance ($P < 0.05$). From combined ECG and VCG findings the presence of an infarct was diagnosed in 42 different patients.

If the patients with left ventricular hypertrophy or rheumatic heart disease are included, definite infarcts were apparent on the VCG of 50 patients (36%) and on the ECG of 39 patients (27.3%) ($P < 0.01$). A total of 53 patients in the combined groups had infarcts diagnosed by one of the other technic.

With the criteria listed, the VCG also surpassed the ECG in the diagnosis of multiple areas of infarction in a greater number of patients (13 versus six, $P < 0.05$) and thus in the diagnosis of a greater total number of definite infarcts (63 versus 45, $P < 0.01$). Multiple infarcts were observed on the ECG and VCG in the presence of single or double-vessel disease although this finding was most common with diffuse arteriographic disease.

In view of the apparent increased sensitivity of the VCG compared with the ECG, the question is raised by these observations whether this increased yield of infarcts as diagnosed from the VCG using quantitative criteria is accompanied by an unacceptable incidence of incorrect or "false positive" diagnoses. In this study, an ECG or VCG abnormality was considered to be falsely present if it did not correlate with a significant arteriographic lesion in, or adjacent to, a location predicted by the expected arterial distribution as described in table 3. Table 6 correlates the locations of both depolarization abnormalities and localized "ischemic" repolarization abnormalities noted by the VCG and ECG with the associated arteriographic findings to illustrate the incidence of false-positive VCG and ECG abnormalities in each of these areas.

In each instance in which a definite infarct (grade 4 or 5) was diagnosed from either the ECG or the VCG, appropriately localized arteriographic occlusions greater than 50% of the vessel lumen were present. However, four of 35 ECG diagnoses and four of 48 VCG diagnoses of a possible infarct (grade 3) were incorrect, that is, they were unaccompanied by the predicted arteriographic lesions. The frequency with which repolarization abnormalities indistinguishable from ischemic changes (grade 2) occurred in the absence of arteriographic disease is of additional interest.

No arteriographically unexplained diagno-

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Table 6

<table>
<thead>
<tr>
<th>Site of abnormality</th>
<th>2</th>
<th>3†</th>
<th>4‡</th>
<th>5‡</th>
<th>2</th>
<th>3†</th>
<th>4‡</th>
<th>5‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anteroseptal</td>
<td>0/2</td>
<td>0/4</td>
<td>0/2</td>
<td>0/3</td>
<td>0/5</td>
<td>0/3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior</td>
<td>0/7</td>
<td>0/2</td>
<td>0/2</td>
<td>0/1</td>
<td>1/4</td>
<td>0/4</td>
<td>0/2</td>
<td></td>
</tr>
<tr>
<td>Anterolateral</td>
<td>4/21</td>
<td>0/3</td>
<td>0/4</td>
<td>2/11</td>
<td>0/15</td>
<td>0/1</td>
<td>0/4</td>
<td></td>
</tr>
<tr>
<td>High lateral</td>
<td>0/4</td>
<td>1/5</td>
<td>0/1</td>
<td>0/9</td>
<td>2/3</td>
<td>0/1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inferior</td>
<td>0/6</td>
<td>3/23</td>
<td>0/14</td>
<td>0/8</td>
<td>1/5</td>
<td>2/19</td>
<td>0/20</td>
<td>0/8</td>
</tr>
<tr>
<td>True posterior</td>
<td>0/2</td>
<td>0/1</td>
<td>0/2</td>
<td>0/8</td>
<td>0/7</td>
<td>0/3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>4/38</td>
<td>4/35</td>
<td>0/22</td>
<td>0/17</td>
<td>4/31</td>
<td>4/48</td>
<td>0/38</td>
<td>0/20</td>
</tr>
</tbody>
</table>

* Numerator in fractions = ECG or VCG diagnosis unexplained by significant arteriographic disease in predicted location; denominator = total ECG or VCG diagnosis of abnormality. Rheumatic heart disease excluded.
† ECG-VCG abnormalities are referred to as possible myocardial infarction.
‡ ECG-VCG abnormalities are referred to as definite myocardial infarction.
Table 7

Frequency of Incorrect (False-Positive) ECG-VCG Diagnoses of Abnormality When Arteriogram Used as Reference Showed Significant Luminal Narrowing (>75%)*

<table>
<thead>
<tr>
<th>Site of abnormality</th>
<th>ECG (grade)</th>
<th></th>
<th></th>
<th></th>
<th>VCG (grade)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Anteroseptal</td>
<td>0/2</td>
<td>0/4</td>
<td>0/2</td>
<td>0/3</td>
<td>0/5</td>
</tr>
<tr>
<td>Anterior</td>
<td>0/7</td>
<td>0/2</td>
<td>0/2</td>
<td>0/1</td>
<td>1/4</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>4/21</td>
<td>0/3</td>
<td>0/4</td>
<td>3/11</td>
<td>0/15</td>
</tr>
<tr>
<td>High lateral</td>
<td>0/4</td>
<td>1/5</td>
<td>0/1</td>
<td>0/9</td>
<td>3/3</td>
</tr>
<tr>
<td>Inferior</td>
<td>1/6</td>
<td>4/23</td>
<td>0/14</td>
<td>0/8</td>
<td>1/5</td>
</tr>
<tr>
<td>True posterior</td>
<td>1/2</td>
<td>0/1</td>
<td>0/2</td>
<td>1/8</td>
<td>2/7</td>
</tr>
<tr>
<td>Total</td>
<td>5/38</td>
<td>5/35</td>
<td>1/22</td>
<td>0/17</td>
<td>6/31</td>
</tr>
</tbody>
</table>

*Numerator in fractions = ECG or VCG diagnosis unexplained by significant arteriographic disease in predicted location; denominator = total ECG or VCG diagnosis of abnormality. Rheumatic heart disease excluded.

Cases occurred when either the VCG or the ECG criteria for possible (grade 3) anterolateral infarct or for possible (grade 3) anteroseptal infarct were used. However, the excessive frequency of false-positive diagnoses in using the VCG criterion for possible high lateral infarct suggests that this criterion should be discarded. Three patients had definite ECG evidence of both anterolateral and inferior infarcts but only definite VCG evidence of anterolateral infarct. Each had initially directed superior QRS forces of greater than 25-msec duration on the VCG which were inscribed, however, in a counterclockwise direction, thus excluding a VCG diagnosis of definite inferior infarct with the criteria used. Perhaps in the presence of a lateral infarct, prolonged initial superior QRS forces should suffice for the VCG diagnosis of an associated inferior infarct.

Table 6 also reveals that the VCG manifested its diagnostic superiority in the detection of infarcts in three regions, namely anterior, inferior, and true posterior, and apparently did so without sacrificing accuracy. The possibility that the criteria used for the ECG diagnosis of inferior or anterior infarcts were too restricted was examined. However, extension of these criteria to include possible infarcts (grade 3) would be associated with the appearance of an increased incidence of diagnoses which were unaccompanied by the predicted arteriographic lesions.

If the arteriograms are reclassified so that an arterial occlusion must exceed 75% of the luminal diameter to be considered functionally significant, an occasional grade 4 definite infarct is then unexplained by arteriography (table 7). A single such instance on the ECG was true posterior in location, and two instances each on the VCG were inferior and true posterior in location.

A similar extrapolation of the data using nearly complete (grade 5) and complete (grade 6) arteriographic occlusions revealed an increasing incidence of arteriographically unexplained ECG and VCG diagnoses, but nevertheless when only complete arterial occlusions were considered significant, the grade 5 ECG and VCG categories maintained their accuracy. Sixteen of 17 ECGs and 18 of 20 VCGs of grade 5 severity were accounted for by complete occlusion of an appropriate nutrient artery. Many grade 4 ECG and VCG lesions, however, were unexplained by arteriographic abnormalities of this severity.

In those patients with aortic valve disease (excluded from tables 6 and 7), one of the six patients with definite evidence of an infarct on the ECG had no arteriographic basis for this finding. No similar false positive diagnoses were noted in the five patients with aortic valve disease and with VCG evidence of a definite infarct.

It was apparent from this study that the localization of infarcts by either ECG or VCG correlated well with the location of significant coronary occlusive disease. This correlation is
Table 8

Depolarization ECG-VCG Abnormalities Associated With Grade 6 Arteriographic Lesions (Complete Occlusions)‡†

<table>
<thead>
<tr>
<th>Arteriographic site</th>
<th>Collaterals</th>
<th>Normal or grade 1 ECG or VCG</th>
<th>Anteroseptal and anterior</th>
<th>Anterolateral and high lateral</th>
<th>Inferior and true posterior</th>
<th>Total infarcts</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Internal</td>
<td>Possible</td>
<td>Definite</td>
<td>Possible</td>
<td>Definite</td>
</tr>
<tr>
<td>RCA</td>
<td>No</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td>29</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>18</td>
<td>1</td>
<td>10</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>LAD</td>
<td>No</td>
<td>0</td>
<td>1</td>
<td>6</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>22</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LCx</td>
<td>No</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>13</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

ECG abnormalities

VCG abnormalities

*One ECG and one VCG entry per arteriographic lesion. Grade 2 ECG and VCG abnormalities omitted.
†Abbreviations as defined in table 5.

implied by the data analysis in table 6 as indicated by the infrequency of falsely localized infarcts. Further support is provided in table 8 in which the ECG and VCG depolarization abnormalities associated with complete arteriographic occlusion of individual arteries are correlated. Table 8 also emphasizes the frequency with which complete arterial occlusions leave no residual evidence of myocardial damage detectable by ECG or VCG. Conversely, although the incidence of ECG and VCG changes consistent with infarction increases as the extent of arteriographic disease increases (table 5), the ECG and VCG commonly reveal definite infarct patterns in the presence of significant but incomplete arterial occlusion.

Intuitively, one might expect that severe disease of the left anterior descending artery would be more easily detected by electrocardiography because the anteroseptal and anterior infarcts likely to be associated would be just beneath the exploring electrodes. However, table 8 shows that in this study severe disease of the right coronary artery was associated with a greater frequency of detectable infarcts than was severe occlusive disease of the left anterior descending artery, suggesting that either the criteria for diagnosis of inferoposterior infarcts are superior to those for anterior infarcts or myocardial infarction is a more common sequela of right coronary artery occlusion than left anterior descending artery occlusion.

Table 9

Incidence of Collateral Vessels With Increasing Extent of Arteriographic Disease

<table>
<thead>
<tr>
<th>Arteriogram</th>
<th>Total</th>
<th>With collaterals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>49</td>
<td>0</td>
</tr>
<tr>
<td>Single-vessel disease</td>
<td>19</td>
<td>10 (52.9%)</td>
</tr>
<tr>
<td>LAD</td>
<td>15</td>
<td>8</td>
</tr>
<tr>
<td>RCA</td>
<td>21</td>
<td>15 (68.6%)</td>
</tr>
<tr>
<td>RCA + LAD or LCx</td>
<td>74</td>
<td>65 (87.8%)</td>
</tr>
<tr>
<td>Diffuse disease</td>
<td>192</td>
<td>107 (74.8%)*</td>
</tr>
</tbody>
</table>

*Per cent of abnormal arteriograms.
The frequency with which collateral vessels were noted increased significantly as the extent of arteriographic disease increased ($P < 0.01$; table 9). No patient with a normal arteriogram had evidence of collateralization, whereas 53% of patients with single-vessel disease, 69% with double-vessel disease, and 88% with diffuse disease had collaterals. There was no evidence that the presence of such collateral vessels reduced the likelihood that either the ECG or the VCG would detect an infarct. In fact, as the incidence of collateral vessels increased coincident with the increase in extent and severity of arterial disease, an associated increase in the incidence of infarcts was also noted.

Twenty-seven of the 192 patients without myocardopathy or left bundle-branch block had ECG evidence of left ventricular hypertrophy, and in all but two instances characteristic repolarization abnormalities were associated. Only 19 of these patients also fulfilled the VCG criteria for left ventricular hypertrophy. The incidence of left ventricular hypertrophy increased with increasing extent of arteriographic disease from 10% of the patients with normal arteriograms and 9% with single-vessel disease to 14% with double-vessel disease and 19% with diffuse arteriographic disease. However, only an occasional patient with ECG evidence of left ventricular hypertrophy was free of either underlying mitral or aortic valvular disease or systemic diastolic hypertension.

**Correlation of Ventriculogram with ECG, VGG, and Coronary Arteriogram**

As the extent of arteriographically demonstrated disease increased, the incidence of both localized (asynergic) and generalized abnormalities of myocardial contraction significantly increased ($P < 0.01$; table 10). Only one of 49 (2%) patients with normal arteriograms had an abnormality such as a localized or generalized reduction in myocardial contraction or a localized zone of akinesis or dyskinesis recognized on ventriculography. This patient had associated aortic stenosis and insufficiency. Twenty-six and a half per cent of

### Table 10

**Correlation of Localized and Generalized Disorders of Myocardial Contraction With Increasing Extent of Arteriographic Disease**

<table>
<thead>
<tr>
<th>Arteriogram</th>
<th>Total patients</th>
<th>Patients with disorders of contraction</th>
<th>Incidence (%) of collaterals in patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Localized</td>
<td>Generalized</td>
</tr>
<tr>
<td>Normal</td>
<td>49(10)†</td>
<td>0</td>
<td>1(1)</td>
</tr>
<tr>
<td>Single-vessel disease</td>
<td>34(9)</td>
<td>6(2)</td>
<td>5(3)</td>
</tr>
<tr>
<td>Double-vessel disease</td>
<td>35(5)</td>
<td>10(2)</td>
<td>9(2)</td>
</tr>
<tr>
<td>Diffuse disease</td>
<td>74(6)</td>
<td>28(2)</td>
<td>24(2)</td>
</tr>
<tr>
<td>Total</td>
<td>192(30)</td>
<td>44(6)</td>
<td>39(8)</td>
</tr>
</tbody>
</table>

*Some patients had both localized and generalized disorders.
†Parentheses indicate patients with rheumatic valvular disease.

### Table 11

**Correlation of Localized Disorders of Myocardial Contraction With Arteriographic Disease**

<table>
<thead>
<tr>
<th>Zone of asynery</th>
<th>RCA (grade)</th>
<th>LCA (grade)†</th>
<th>LAD (grade)</th>
<th>LCx (grade)†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3-4</td>
<td>5-6</td>
<td>3-4</td>
<td>5-6</td>
</tr>
<tr>
<td>Anterior</td>
<td>1</td>
<td>4</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>Apical</td>
<td>4</td>
<td>9</td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>Inferior</td>
<td>1</td>
<td>14</td>
<td></td>
<td>3</td>
</tr>
</tbody>
</table>

*Single arteriographic entry per ventriculographic abnormality.
†No patients with grade 3 or 4 disease in this category.
Table 12
Correlation of Localized Disorders of Myocardial Contraction With ECG and VCG Abnormalities*†

<table>
<thead>
<tr>
<th>Zone of asynergy</th>
<th>Normal or grade 1 ECG or VCG</th>
<th>Anteroseptal-anterior</th>
<th>Infarct</th>
<th>Anterolateral-high lateral</th>
<th>Inferior-posterior</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Possible</td>
<td>Definite</td>
<td>Possible</td>
<td>Definite</td>
</tr>
<tr>
<td>Anterior</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Apical</td>
<td>4</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Inferior</td>
<td>6</td>
<td>3</td>
<td>1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**ECG abnormalities**

**VCG abnormalities**

*Grade 2 ECG and VCG abnormalities are omitted. Single ECG and VCG entry per ventriculographic abnormality.
†Abbreviations as defined in table 5.

the patients with single-vessel disease, 34.3% with double-vessel disease, and 52.7% with diffuse disease had either localized or generalized disorders of myocardial contraction.

In table 11, each zone of localized contraction disturbance (asynergy) is correlated with the associated arteriographic abnormality. Four patients had two areas each of asynergy. Each ventriculographic abnormality was consistently explained by occlusive disease in an appropriately located nutrient artery.

It is also apparent that the likelihood of having a disorder of contraction was not reduced by the presence of associated collateral vessels (table 10). Although the numbers are small, there was no statistically significant difference at the various levels of arteriographic disease between the incidence of collaterals in those patients with contraction disorders and those without such disorders. No difference in severity of the individual arterial lesions was noted between these two groups of patients.

Table 12 correlates the relationship of localized areas of asynergy with associated ECG and VCG findings. Satisfactory localization was accomplished by electrocardiography although some overlap is noted in the apical zone. It is apparent that a myocardial infarct, at least as diagnosed by ECG or VCG, is not a consistent finding in the presence of ventricular asynergy. Many ventriculographic abnormalities associated with coronary artery disease would thus seem to occur on a basis of myocardial ischemia rather than gross scarring.

In five instances, characteristic ST-segment elevation on the ECG suggested the likelihood of a ventricular aneurysm. Two of these patients had a dyssynchronous (paradoxical) aneurysm in the predicted location, two had akinetic (noncontractile) areas in or adjacent to the predicted location, and one had a severe generalized reduction in myocardial contraction.

**Left Bundle-Branch Block**

Eight patients in this series had ECG evidence of complete left bundle-branch block. Coronary arteriographic findings were normal in six of these patients, and in each the ECG and VCG revealed only a typical complete left bundle-branch block. The single instance of atypical complete left bundle-branch block28, 29 (absence of anterior QRS forces) was associated with complete occlusion of the left anterior descending artery.

**Myocardiopathy**

Five of the six patients with idiopathic hypertrophic subaortic stenosis included in this study had normal coronary arteriograms. The sixth patient had complete occlusion of the proximal part of the right coronary artery and evidence of a possible inferior infarct on ECG and VCG and a definite true posterior infarct on VCG. None of the patients with
normal arteriograms had electrocardiographic evidence of definite infarct although left ventricular hypertrophy was diagnosed in three patients. Four other patients had nonobstructive cardiomyopathy of uncertain origin. A diagnosis of definite infarct was made in the absence of associated arteriographic disease in two cases from the ECG (inferior and anteroseptal) and in one case from the VCG (anteroseptal).

**Discussion**

Selective cinecoronary arteriography provides the most definitive antemortem demonstration of the coronary arterial anatomy presently available, and as such has served as our reference for establishing the presence and extent of arterial disease and as the basis for evaluation and comparison of the ECG and VCG criteria in this study. However, detailed postmortem coronary arteriographic studies and similar antemortem and postmortem correlative studies performed under more optimal technical conditions have emphasized that arteriography offers an imperfect appraisal of occlusive lesions and that when errors do occur they tend to be ones of underestimation of both the percentage of luminal reduction and the length of arterial stenoses.

Although we have considered a 50% reduction in luminal diameter (approximately 75% reduction in luminal cross-sectional area) to be functionally significant and potentially capable of causing gross myocardial necrosis, we have also analyzed our data considering as functionally significant increasing degrees of arterial occlusion (grades 4 through 6). However, it would be erroneous to conclude that a partial or complete occlusion of a coronary artery is necessarily accompanied by a myocardial infarct. The presence of severe coronary occlusive disease only provides a basis for such damage. A pathologic study would be necessary to extend the correlations in this report to the associated myocardial anatomy.

In the group of patients without left bundle-branch block or a cardiomyopathy who had abnormal arteriograms, 37.1% had evidence of a myocardial infarct on either ECG or VCG. Nine of 34 patients with single-vessel disease, 11 of 35 with double-vessel disease, and 33 of 74 with diffuse arteriographic disease had definite evidence of an infarct on ECG or VCG. There was no evidence that the presence of left ventricular hypertrophy, valvular heart disease, or collateral vessels altered the ability of either ECG or VCG to detect an infarct. On the contrary, the incidence of both collaterals and infarcts tended to increase with increasing extent of arteriographic disease.

It is of great interest that although the combined use of the ECG and VCG was superior to either technic alone, VCG criteria were apparently superior to ECG criteria in the diagnosis of myocardial infarcts. Not only did the VCG yield a diagnosis of definite infarcts in a greater number of patients (50 versus 39, P<0.01), but it also excelled in the diagnosis of multiple areas of infarcts in a greater number of patients (13 versus six, P<0.05) and thus in the diagnosis of a greater total number of definite infarcts (63 versus 45, P<0.01). Infarcts in the anterior, inferior, and true posterior areas were better detected by quantitative computer analysis of the VCG than by a conventional analysis of the ECG. As shown in table 6, this VCG advantage was attained without sacrificing specificity and, in fact, there were no patients without myocardial or aortic valve disease in whom a definite (grade 4) infarct was diagnosed in these sites on either ECG or VCG without the presence of associated appropriately localized significant arterial occlusions.

The excessive incidence of arteriographically unexplained diagnoses of infarcts by either ECG or VCG in the four patients with idiopathic cardiomyopathy, however, suggests that electrocardiographic interpretation should be approached with caution in this particular group. The possibility exists that focal electrically inactive areas of myocardial fibrosis or replacement unassociated with atherosclerosis of the major coronary vessels...
may account for this apparent inaccuracy. One of the six patients with aortic valve disease and ECG evidence of a definite infarct had no arteriographic basis for the finding, but all patients with a VCG diagnosis of such an infarct in the presence of valvular heart disease had the expected arterial occlusions.

Ventriculographic abnormalities also were observed more frequently with increasing extent of arteriographic disease, and their presence did not appear to be muted by associated collateral vessels. Each localized zone of altered myocardial contractility occurred in the distribution of a predicted compromised nutrient artery. Furthermore, when ECG or VCG abnormalities were also noted in the presence of localized ventriculographic findings, they tended to occur in the same location as the zones of asynergy. However, disturbances of contraction often occurred without revealing their presence on the electrocardiogram.

The disagreement between the results of our comparison of the ECG and VCG and those obtained in earlier studies\(^6-8\) in which no significant difference was appreciated between these two technics is most likely explained by the differences in the method of interpretation of the VCG and in the criteria applied to the VCG. The present study supports the conclusions of recent studies that a quantitative analysis of the corrected orthogonal VCG is an improvement over conventional analysis of the ECG. It also suggests that the VCG may offer a superior representation of the phasic changes in the depolarization process which can be exploited by appropriate criteria, particularly criteria applied to discrete voltage data provided by a computer.

This apparent superiority of quantitative VCG analysis over analysis of the conventional scalar ECG in the diagnosis of residua of previous infarctions emphasizes the need for evaluating these criteria with a more definitive pathologic correlative study on the one hand and an analysis of a larger group of normal patients on the other. If the results reported herein are confirmed by such approaches, an expanded application of the VCG and these criteria would seem warranted.

References

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Comparative Quantitative Analysis of the Electrocardiogram and the Vectorcardiogram: Correlations with the Coronary Arteriogram
DAVID R. MCCONAHAY, BEN D. MCCALLISTER, FRANZ J. HALLERMANN
and RALPH E. SMITH

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