Evaluation of Vectorcardiographic Criteria for the Diagnosis of Myocardial Infarction in the Presence of Left Ventricular Hypertrophy

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SUMMARY Vectorcardiograms (VCG) from a consecutive group of 77 patients with significant aortic valve disease were analyzed. All of the patients had complete left and right heart catheterization with normal coronary arteriograms and normal left ventricular contraction. Thirty-five (46%) patients met VCG criteria for anterior myocardial infarction (AMI-35%) and/or inferior myocardial infarction (IMI-14%). This was a significant increase in false positive diagnosis for both criteria compared to a group of 200 normal volunteers under age 30 and 100 patients with normal hearts by cardiac catheterization ($P < 0.01$). It was found that if the VCG diagnosis of myocardial infarction was deferred when the maximal transverse plane magnitude was $>1.8$ mV, the incidence of AMI false positive diagnosis decreased to 3% and the incidence of IMI false positive diagnosis decreased to 1%. The same rule was applied to the aortic valve disease cohort, a group of 124 patients with documented AMI and a group of 158 patients with IMI. This decreased the sensitivity of the AMI criteria from 93 to 83% and of the IMI criteria from 85 to 77%. The increase in average performance was statistically significant for the AMI criteria ($P < 0.05$) but not for the IMI criteria.

Myocardial infarction was excluded by requiring that each patient have: (1) absence of significant coronary artery disease (no greater than 25% occlusion of any vessel), (2) absence of a focal contraction abnormality on the left ventriculogram and (3) absence of a previously documented infarct (one of the following: history, positive cardiac enzymes or serial ECG changes in the acute clinical setting). In addition, patients with bundle branch block were excluded, and it was required that there be no evidence of other valvular lesions in addition to the aortic valve disease with the exception of trace mitral regurgitation.

Only patients with hemodynamically significant aortic valve disease were chosen. The group with pure aortic stenosis (AS) consisted of 28 patients; 24 had a peak to peak gradient across the aortic valve of greater than 50 mm Hg, and in four patients the gradient was between 38 and 50 mm Hg. None had greater than 1+ aortic insufficiency by the classification of Cohn et al.4 The pure aortic insufficiency (AI) group consisted of 20 patients with at least 3+ AI and less than a 25 mm Hg gradient. The combined aortic stenosis and aortic insufficiency (AS-AI) group consisted of 29 patients with at least 2+ AI; 25 with greater than 50 mm Hg gradient and four with gradients between 25 and 50 mm Hg.

Three hundred adults without evidence of heart disease were used as a control population. Two methods of selection were used so that patients with a wide range of ages would be included. The first normal group consisted of 200 healthy volunteers aged 20–29 without a history of heart disease or hypertension (NL-VOL). The second normal group was composed of 100 patients who underwent cardiac catheterization with selective coronary arteriography and left ventriculography because of chest pain suggestive of angina but in whom both a normal ventricular contraction pattern and normal coronary artery anatomy were demonstrated (NL-CATH).

VCG data were obtained from four groups of patients with strong evidence for either anterior myocardial infarction or inferior myocardial infarction for comparison with the patients with aortic valve disease. To insure that the patients with infarction were a representative population,
each subgroup was a consecutive series from September 1, 1970 to January 1, 1974 obtained from the data bank of the Duke Computer Facility where complete records of all patients undergoing cardiac catheterization for suspected coronary artery disease and of all patients admitted to the Coronary Care Unit are filed. The AMI-CCU group was composed of 46 patients admitted to the CCU of Duke University Medical Center with a history compatible with acute infarction. In addition, evolutionary ECG changes of repolarization in leads V1, V2, and V3 consisting of both ST-segment elevation and T-wave inversion (the presence or absence of significant Q waves in these leads did not influence inclusion in this group) and transient findings of both elevation of serum CPK-MB and LDH greater than LDH were required. The IMI-CCU group was composed of 75 patients chosen similarly to the AMI-CCU group except for the presence of ECG repolarization changes in leads II, III, and aVF, instead of in the precordial leads. The AMI-CATH group consisted of 78 patients who underwent coronary arteriography because of clinical symptoms and in whom both the presence of a 70 to 100% occlusion of the left anterior descending coronary artery and localized akinesia or dyskinesia of the anterior ventricular wall were proven. The IMI-CATH group consisted of 83 similarly chosen patients in whom the presence of a 75 to 100% occlusion of the right coronary artery and localized akinesia or dyskinesia of the inferior left ventricular myocardium were required. Patients were excluded from these groups if there was QRS or ventriculographic evidence of more than one area of infarction or of bundle branch block.

The study groups are outlined in figure 1.

Data Collection

In all catheterization subgroups right and left heart catheterization was performed using standard techniques. The cineangiograms, taken at a filming speed of 60 frames per second, were evaluated for abnormalities of left ventricular wall motion, degree of occlusive CAD, amount of mitral and aortic insufficiency.

The VCGs were recorded using the Frank lead system with a Hewlett-Packard 1507A vectorcardiograph or an Instruments for Cardiac Research VCG-1B vectorcardiograph modified with a Hewlett-Packard oscilloscope and camera. The chest electrodes (A, C, E, and I) were placed in the fourth intercostal space as recommended for the supine position. A calibration of 1 mV per 2 cm deflection was used depending on the size of the total loop. The initial QRS forces were recorded with a calibration of 1 mV per 10 cm deflection with the P and T loops excluded. The VCG trace was composed of an electronic ECG machine.

The VCGs were recorded within one week of catheterization in all cath subgroups or within two weeks after the acute infarction in the two CCU subgroups.

Since no set of generally accepted VCG criteria for the various locations of infarction presently exists, an attempt was made to choose from the literature the most sensitive and specific criteria based on anatomical studies. The criteria are outlined as follows:

**Anterior myocardial infarction:** Initial anterior QRS forces must not exceed .10 mV in maximal anterior amplitude and also must not exceed 24 msec in duration;

**Anterolateral myocardial infarction:** Displacement of the 20 msec vector to the right and displacement of the efferent limb to the right and posterior with initial rightward QRS forces of greater than 22 msec in duration and greater than .16 msec in amplitude; 12

**High lateral myocardial infarction:** Displacement of the initial QRS deflection in the frontal plane inferiorly and to the right of the y axis with counterclockwise inscribed the vertically displaced maximal QRS vector of greater than 40°. 13 14

**Inferior myocardial infarction:** In the frontal plane, generally clockwise superior forces must be present. These are defined as forces which are initially either a) superior (rightward or leftward) or b) inferior and completely rightward for not more than 10 msec prior to becoming superior and which subsequently cross the X axis to the left

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**Figure 1** All patient groups included in this analysis are subdivided into "test" and "control" sections. AS = aortic stenosis, AI = aortic insufficiency, NL = normal, VOL = volunteers, CATH = catheterization, CCU = coronary care unit, AMI = anterior myocardial infarction, IMI = inferior myocardial infarction.
of the 0 point (or less commonly the entire efferent limb remaining superior to the X axis). At least one of the following must also be present:

1. Time from the 0 point to leftward X intercept of at least 25 msec and distance from 0 point to leftward X intercept of at least 0.3 mV.
2. A maximal frontal plane QRS vector above 15° (less than 15°).
3. A maximal superior deviation of at least 0.1 mV and a ratio of maximal superior deviation to maximal inferior deviation of at least 1.5.  

Posterior myocardial infarction: All of the following:
1. Maximal anterior QRS voltage of .5 mV or more.
2. Total anterior QRS duration of 42 msec or more.
3. Anterior access time of the maximal QRS forces of 30 msec or more.
4. Ratio of maximal anterior QRS voltage to maximal posterior QRS voltage of at least 1.0.  

The left ventricular hypertrophy criteria used were those of Romhilt et al.  

As mentioned above, only one patient met the strict anterolateral criteria. The more liberal criterion suggested by Hugenholtz et al. requires only clockwise rotation of the initial anterior forces in the transverse plane. Only four (5%) aortic valve disease patients met this criterion and each of these met anterior criteria in addition.

As shown in table 1, 11 aortic valve disease patients (14%) met inferior infarction criteria. This false positive rate was shown to be significantly increased (P < 0.01) over the NL-VOL and NL-CATH groups. The AS group and the AI group were shown to have significantly increased false positive rates of 18 and 20%, respectively (P < 0.01). The AS-AI group had a false positive rate of 7% which was not significantly increased over the control groups. As described in the Methods section, there are three parts to the inferior infarction criteria, any one of which can be used alone for the diagnosis of inferior infarction. Each of these parts was present in at least three aortic valve disease patients and no single part was responsible for a majority of the false positive diagnoses of inferior infarction.

The anterior infarction criteria were applied to the AMI-CCU and AMI-CATH groups and the inferior infarction criteria for the IMI-CCU and IMI-CATH groups. The excellent sensitivity of the criteria can be appreciated from the results shown in table 1.

The VCG criteria for left ventricular hypertrophy of Romhilt et al. were applied to each subgroup and the results are shown in table 1. It is remarkable that the incidence of VCG evidence of left ventricular hypertrophy was significantly increased over normal in each of the infarction subgroups (P < 0.01).

An attempt was made to identify other VCG characteristics of the aortic valve disease patients falsely meeting infarction criteria which would differentiate them from patients with actual infarction. Figure 2 shows the distributions of maximal transverse magnitude of patients

### Table 1. Patients Meeting VCG Criteria for AMI, IMI, and LVH in the Various Study Groups

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>AMI VCG</th>
<th>IMI VCG</th>
<th>LVH VCG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic valve</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pure AS</td>
<td>28</td>
<td>7 (25%)</td>
<td>5 (18%)</td>
<td>16 (57%)</td>
</tr>
<tr>
<td>Pure AI</td>
<td>20</td>
<td>4 (20%)</td>
<td>4 (20%)</td>
<td>16 (80%)</td>
</tr>
<tr>
<td>AS–AI</td>
<td>29</td>
<td>16 (55%)</td>
<td>2 (7%)</td>
<td>28 (97%)</td>
</tr>
<tr>
<td>NL-VOL</td>
<td>100</td>
<td>3 (15%)</td>
<td>4 (2%)</td>
<td>2 (1%)</td>
</tr>
<tr>
<td>NL-CATH</td>
<td>46</td>
<td>44 (90%)</td>
<td>2 (2%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>AMI-CCU</td>
<td>71</td>
<td>71 (100%)</td>
<td>5 (11%)</td>
<td></td>
</tr>
<tr>
<td>PMI-CATH</td>
<td>78</td>
<td>71 (91%)</td>
<td>—</td>
<td>8 (10%)</td>
</tr>
<tr>
<td>DMI-CCU</td>
<td>75</td>
<td>—</td>
<td>70 (93%)</td>
<td>7 (9%)</td>
</tr>
<tr>
<td>DMI-CATH</td>
<td>83</td>
<td>—</td>
<td>64 (77%)</td>
<td>5 (6%)</td>
</tr>
</tbody>
</table>

### Table 2. Comparison of the False Positive Rates Using the VCG Criteria for AMI of Starr et al. and Those of Hugenholtz et al. As Applied to the AVD Patients

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>AMI Starr et</th>
<th>AMI Hugenholtz et</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pure AS</td>
<td>28</td>
<td>7 (25%)</td>
<td>8 (29%)</td>
</tr>
<tr>
<td>AS–AI</td>
<td>29</td>
<td>16 (55%)</td>
<td>18 (62%)</td>
</tr>
<tr>
<td>Pure AI</td>
<td>20</td>
<td>4 (20%)</td>
<td>7 (35%)</td>
</tr>
<tr>
<td>Total AVD</td>
<td>77</td>
<td>27 (35%)</td>
<td>33 (43%)</td>
</tr>
</tbody>
</table>
meeting infarction criteria. The left panel compares the patients with actual anterior infarction with the aortic valve disease patients who had false positive anterior infarction. The right panel compares the patients with actual inferior infarction and those aortic valve disease patients who had false positive inferior infarction. It is apparent that with few exceptions the aortic valve disease patients have much higher transverse magnitude than the patients with true myocardial infarction. Therefore an attempt was made to identify a specific value for transverse magnitude that could be used as an upper limit above which the definite diagnosis of infarction would not be made which would improve overall performance of the criteria in these patient groups.

Tables 3 and 4 show the percentage of correct diagnoses obtained when different values for the upper limit are used. The percent correct diagnoses is shown as percent true negatives for the aortic valve disease patients and percent true positives for the patients with actual infarction. In addition an average performance was calculated by the formula:

$$\text{average performance} = \frac{\% \text{ true negatives} + \% \text{ true positives}}{2}$$

As shown in table 3, the best average performance of the anterior infarction criteria is obtained by using an upper limit of 1.8 mV. Using this value the percent of true negatives in the aortic valve disease groups was increased to 97% compared to 65% when no upper limit was used; however, the percent true positives in the anterior infarction was reduced from 93 to 83%. The number of overall errors in

**TABLE 3. Selection of the Optimal Transverse Magnitude Upper Limit for AMI Criteria**

<table>
<thead>
<tr>
<th>AMI excluded if transverse magnitude exceeds (mV)</th>
<th>1.5</th>
<th>1.6</th>
<th>1.7</th>
<th>1.8</th>
<th>1.9</th>
<th>2.0</th>
<th>2.1</th>
<th>2.2</th>
<th>No upper limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>% True negatives (AVD group, N = 77)</td>
<td>76 (99)</td>
<td>76 (99)</td>
<td>75 (97)</td>
<td>75 (97)</td>
<td>70 (91)</td>
<td>69 (90)</td>
<td>66 (86)</td>
<td>64 (83)</td>
<td>50 (65)</td>
</tr>
<tr>
<td>% True positives, (AMI group, N = 124)</td>
<td>84 (68)</td>
<td>91 (73)</td>
<td>98 (79)</td>
<td>103 (83)</td>
<td>106 (86)</td>
<td>108 (87)</td>
<td>109 (88)</td>
<td>109 (88)</td>
<td>115 (93)</td>
</tr>
<tr>
<td>Avg. performance* (%)</td>
<td>84</td>
<td>86</td>
<td>88</td>
<td>90</td>
<td>89</td>
<td>89</td>
<td>86</td>
<td>85</td>
<td>79</td>
</tr>
</tbody>
</table>

*% True Neg + % True Pos. / 2
diagnosis was shown to be significantly reduced ($P < 0.05$) when this upper limit was used.

A similar analysis was applied to the inferior infarction data. Table 4 shows that if inferior infarction is excluded when transverse magnitude exceeds 1.8 mV (the optimal upper limit for the anterior infarction criteria), the percent true negatives increases from 86% to 99%. However, the increase in average performance is not nearly as striking. Furthermore, although the total number of erroneous diagnoses is reduced, this decrease is not statistically significant at 1.8 mV or any other possible transverse magnitude upper limit.

No improvement in the separation of the aortic valve disease patients with false positive myocardial infarctions from the patients with true infarctions was achieved by adjusting the transverse magnitude upper limit according to the patient’s age in a manner analogous to that of Romhilt et al. It was found that several aortic valve disease patients under age 50 who met criteria for infarction had maximal transverse plane magnitude between 1.8 and 2.2 mV.

**Discussion**

In 1944 Wilson et al. first suggested that a QS pattern in the right precordial leads was common in the setting of left ventricular hypertrophy. It has since been well documented that such loss of anterior forces can occur in patients with left ventricular hypertrophy uncomplicated by myocardial infarction and may lead to the false positive diagnosis of anterior infarction on ECG. Hugenholtz et al. presented data to show that patients with autopsy proven left ventricular hypertrophy but without myocardial infarction retained their anterior forces with the 20 msec instantaneous vector being anterior in each of 36 patients. Subsequently, Estes, Beamer et al., Hilsenrath et al. and Brackbill and Shak presented significant numbers of cases of patients with patent coronary arteries in which the 20 msec instantaneous vector was posterior and therefore highly indicative of anterior infarction.

One of the goals of the present study was to determine the prevalence of false positive infarction in the setting of uncomplicated left ventricular hypertrophy. The definitive method of determining whether myocardial infarction has occurred and localizing the lesion is the postmortem examination. This method is impractical with aortic valve disease patients, however, since infarction often occurs just prior to death as a complication of an operative intervention or terminal severe myocardial heart failure thus making comparison of VCG and hemodynamic data with autopsy results difficult. Furthermore, autopsies were available in only a small minority of our patients. However, it is believed that high quality selective coronary arteriograms and left ventriculograms along with carefully obtained clinical histories in our patients adequately rule out previous infarction. Although there are rare cases of aortic valve disease and myocardial infarction in the absence of coronary artery disease reported, it is believed, as suggested by DeSanctis, that although the subendocardium tends to be underperfused in aortic valve disease, actual transmural infarction is rare in the presence of patent coronary arteries. Furthermore, the ventriculogram should reveal segmental contraction abnormalities in the majority of such cases.

Although postmortem examination would have provided the most definitive means of documenting the presence of left ventricular hypertrophy, the angiographic and hemodynamic data in the aortic valve disease groups provide assurance that each of these patients had left ventricular hypertrophy without coexisting coronary artery disease. The fact that 78% of these patients meet the VCG criteria of Romhilt et al. supports this conclusion, especially since only 61% of Romhilt’s autopsy series of patients with hypertrophy met these criteria.

In the present study of the 77 patients with uncomplicated aortic valve disease, 35% met the VCG anterior infarction criteria of Starr et al. and 43% met the VCG anterior infarction criteria of Hugenholtz et al., both values being significantly increased over comparable normals. Ten patients (13%) had total absence of anterior forces, a pattern not seen in any of the normals. Also, four of those patients retaining anterior forces moved clockwise initially in the horizontal plane, another pattern not seen in any of the normal patients. Only one of these four was found to meet strict anterolateral infarction criteria.

To our knowledge there has been no objective evidence of an increased incidence of false positive inferior infarction with left ventricular hypertrophy in the literature. Therefore it was somewhat surprising to find that 14% of the aortic valve disease patients met inferior infarction criteria, a significant increase over the control population.

These figures are thought to estimate the prevalence of false positive anterior and inferior myocardial infarction in a group of patients with uncomplicated aortic valve disease since every attempt was made to obtain a consecutive series of patients. Whether these figures can be validly applied to patients with ventricular hypertrophy secondary to other causes is not known.

Kini et al. studied 830 patients who were felt to have left ventricular hypertrophy upon clinical grounds but with no myocardial infarction by history. They found absence of

**Table 4. Selection of the Optimal Transverse Magnitude Upper Limit for IMI Criteria**

<table>
<thead>
<tr>
<th>AMI excluded if transverse magnitude exceeds (mV)</th>
<th>Modified AMI criteria</th>
<th>No upper limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.5</td>
<td>1.6</td>
<td>1.7</td>
</tr>
<tr>
<td>76 (99)</td>
<td>76 (99)</td>
<td>76 (99)</td>
</tr>
<tr>
<td>% True negative (AVD group, N = 77)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>101 (64)</td>
<td>113 (72)</td>
<td>119 (75)</td>
</tr>
<tr>
<td>% True positive (IMI group, N = 138)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avg. performance (%)</td>
<td>81</td>
<td>85</td>
</tr>
</tbody>
</table>

*% True Neg + % True Pos.
anterosterior forces in 114 or 14%, a figure which is similar to the 13% incidence in our aortic valve disease patients. These authors compared their 114 patients to 365 patients with documented infarction and loss of anterior forces. They also found that the only significant difference between the vectors of patients with left ventricular hypertrophy and those with true myocardial infarction was in amplitude rather than direction of initial instantaneous vectors. By using two scalar measurements, R amplitude in lead X ≥ 1.2 mV and the sum of amplitudes of R in leads X and Z ≥ 2.5 mV, 66% of left ventricular hypertrophy cases and 88% of myocardial infarction records were correctly classified.

Because of the high prevalence of false positive anterior and inferior infarction which was documented in the present study, we also sought guidelines for more accurate use of the infarction criteria in the setting of left ventricular hypertrophy. The only distinguishing characteristic of the aortic valve disease patients with falsely positive myocardial infarctions was presence of transverse magnitudes which were clearly greater than those of a large group of patients with true positive infarctions. This observation led to an attempt to identify a specific value for transverse magnitude above which the VCG diagnosis of myocardial infarction could not be made. Since it was not clear whether sensitivity or specificity was more important, an average performance was calculated which equally weighted true positives in the infarction group and true negatives in the hypertrophy group. Using an upper limit for the diagnosis of infarction of 1.8 mV and applying it to aortic valve disease patients and patients with true anterior infarction, the average performance was shown to be significantly increased with the true negatives increasing from 65 to 97% and the true positives decreasing only from 93 to 83%. Although the number of erroneous diagnoses could be decreased by applying such a modification to the inferior infarction criteria, the improvement was not statistically significant.

Clockwise rotation of initial forces in the horizontal plane was never seen in either the NL-CATH or NL-VOL groups, and was rarely present (5%) in the patients with aortic valve disease. Therefore, a diagnosis of probable anterior infarction could be made when this characteristic accompanies both transverse magnitude ≥1.8 mV and anterior forces ≤.1 mV and ≤24 msec.

It is the opinion of the authors that in patients meeting anterior or inferior infarction criteria and having a maximal transverse plane magnitude of greater than 1.8 mV, the diagnosis of definite infarction should not be made on the basis of VCG alone. If this rule is followed, the incorrect diagnosis of anterior or inferior infarction in the presence of left ventricular overload due to aortic valve disease will occur in less than 5% of patients. However, the diagnosis of true anterior and inferior infarction will be further reduced by 10% to 83% and 77%, respectively.

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