Evaluation of Precordial Orthogonal Vectorcardiographic Lead ST-segment Magnitude in the Assessment of Myocardial Ischemic Injury

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SUMMARY Relationship has been established between epicardial ST-segment elevation, considered a reliable estimate of ischemic injury in experimental myocardial damage, and ST changes by multiple-lead precordial electrocardiography. However, 35-lead precordial mapping is time-consuming and suitable only for anterior infarctions. An alternate, more rapid method for recording ST segments is an external 3-lead orthogonal vectorcardiographic (VCG) system which also can assess the entire ventricle. Accordingly, validity of VCG ST magnitude was evaluated by direct comparison with changes in epicardial ST magnitude (EST) induced by occlusion of major coronary arteries, reperfusion, and pharmacologic interventions in 15 closed-chest dogs. A total of 404 data points (average 27/dog), 20 epicardial grid and 3 Frank XYZ leads each, demonstrated close correlation (least squares linear regression) between VCG ST and EST changes \( r = 0.921 \pm 0.02 \text{ SEM} \). These data document the accuracy of precordial VCG ST in noninvasive assessment of ischemic injury in various areas of myocardium and its practicality for clinical application.

STUDIES IN EXPERIMENTAL ANIMALS have shown that changes in the sum of ST-segment elevations in each individual are proportional to myocardial damage observed 24 hours following coronary artery occlusion.\(^1\) The reliability of the 35-lead electrocardiographic precordial electrogram as an estimation of the degree of myocardial ischemic injury has been established by close experimental correlation between the sum of ST-segment elevations recorded from epicardial leads and from precordial leads.\(^2,3\) However, the procedure has certain practical shortcomings. Principally, it is not suitable in the assessment of diaphragmatic or posterior myocardial infarction. In addition, the recording of multiple precordial leads and measuring their ST sum are time-consuming processes often not ideal for clinical use in serial evaluation of the course of myocardial damage in response to rapidly acting pharmacologic interventions and related management. Even with the use of a computer, it still requires at least 15 minutes for data collection and determination of the precordial ST sum.\(^4,5\)

An alternate method for the recording of the precordial ST-segment potential is the vectorcardiographic system. Preliminary observation has demonstrated a close relationship between the precordial ST-segment sum and the vectorcardiographic (VCG) ST magnitude in a small number of patients with acute anterior myocardial infarction.\(^6\) Therefore, measurement of the vectorcardiographic orthogonal lead ST magnitude appears to be a convenient and rapid means for assessing myocardial damage which may also be applicable regardless of the anatomic location of the infarction. However, experimental evidence is lacking concerning whether the vectorcardiographic ST magnitude accurately reflects the epicardial ST sum. Accordingly, the present experimental study was undertaken to assess the relation between ST-segment changes in epicardial leads and ST alterations recorded by the vectorcardiogram following myocardial ischemia induced by transient coronary occlusion and following pharmacologic interventions.

Materials and Methods

Fifteen mongrel dogs were anesthetized with sodium pentobarbital 30 mg/kg intravenously, intubated, and ventilated on a Harvard respirator with room air. Additional sodium pentobarbital was administered for maintenance of anesthesia as required. Following a left intercostal thoracotomy the heart was suspended in a pericardial sling. The left anterior descending coronary artery or the circumflex coronary or a major diagonal was dissected free and a wire snare applied through a polyethylene tube 0.042 inch inner diameter which allowed reversible coronary occlusion from outside the chest cavity. A grid of 20 electrodes, five rows of four leads sewn to a square of elastic polyester cloth, was then placed over the anterior or inferior wall of the left ventricle to include the area of eventual myocardial ischemia. Each electrode was a 4 mm diameter nickel plated steel washer to which a number 32 insulated copper wire was soldered; the electrode centers were spaced 11 mm apart. The grid was secured to the heart by a 4-0 silk suture at each corner. The pericardium was then closed over the grid, a chest tube inserted, and the chest cavity closed. Blood pressure was monitored via a femoral artery cannula. Electrodes were then attached to the external chest wall to obtain standard orthogonal XYZ leads with the Frank system (fig. 1). The five chest electrodes were located on an anatomical horizontal plane passing through the midpoint of the left ventricle. The epicardial leads were connected to a specially constructed five channel preamplifier which allowed selection of any one of the four vertical 5-electrode columns (fig. 1). The preamplifier had DC coupling, input impedance of 10,000 megohms, a gain of 1.00, and an input bias current of less than 2 nA. The five buffered epicardial electrocardiographic rows were displayed and recorded on a multichannel os-

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Table 1. Correlation of Frank Lead System ST Magnitude with Multiple Lead Epicardial ST Sum

<table>
<thead>
<tr>
<th>Dog</th>
<th>Location of coronary occlusion</th>
<th>Duration of occlusion (min)</th>
<th>Additional interventions</th>
<th>Number of observations</th>
<th>Correlation coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Prox LAD</td>
<td>18</td>
<td>None</td>
<td>17</td>
<td>.96</td>
</tr>
<tr>
<td>2</td>
<td>First and second CM branches</td>
<td>210</td>
<td>I</td>
<td>46</td>
<td>.96</td>
</tr>
<tr>
<td>3</td>
<td>Prox LAD</td>
<td>20, 20</td>
<td>40 min reperfusion</td>
<td>30</td>
<td>.95</td>
</tr>
<tr>
<td>4</td>
<td>Prox + dist LAD</td>
<td>31</td>
<td>None</td>
<td>27</td>
<td>.78</td>
</tr>
<tr>
<td>5</td>
<td>Prox LAD</td>
<td>60</td>
<td>None</td>
<td>22</td>
<td>.84</td>
</tr>
<tr>
<td>6</td>
<td>Mid LAD</td>
<td>95</td>
<td>I</td>
<td>50</td>
<td>.93</td>
</tr>
<tr>
<td>7</td>
<td>Prox + dist LAD Diagonal</td>
<td>120</td>
<td>M, I, M</td>
<td>24</td>
<td>.97</td>
</tr>
<tr>
<td>8</td>
<td>Mid LAD</td>
<td>190</td>
<td>None</td>
<td>34</td>
<td>.97</td>
</tr>
<tr>
<td>9</td>
<td>First CM branch</td>
<td>17, 17</td>
<td>N</td>
<td>19</td>
<td>.92</td>
</tr>
<tr>
<td>10</td>
<td>Second CM branch</td>
<td>17, 17</td>
<td>M, P</td>
<td>30</td>
<td>.75</td>
</tr>
<tr>
<td>11</td>
<td>Second CM branch</td>
<td>156</td>
<td>I, I, I</td>
<td>26</td>
<td>.81</td>
</tr>
<tr>
<td>12</td>
<td>Prox LCF</td>
<td>18</td>
<td>None</td>
<td>8</td>
<td>.98</td>
</tr>
<tr>
<td>13</td>
<td>Mid LCF</td>
<td>135</td>
<td>P, M, N</td>
<td>34</td>
<td>.99</td>
</tr>
<tr>
<td>14</td>
<td>Prox LAD</td>
<td>4</td>
<td>None</td>
<td>3</td>
<td>.99</td>
</tr>
<tr>
<td>15</td>
<td>Prox LCF</td>
<td>78</td>
<td>M</td>
<td>34</td>
<td>.97</td>
</tr>
</tbody>
</table>

Abbreviations: Prox = proximal; Dist = distal; LAD = left anterior descending artery; CM = circumflex marginal artery; LCF = left circumflex artery; M = methoxamine; I = isoproterenol; N = nitroprusside; P = propranolol.
for a vector in three dimensional space: \( \text{VCG-ST} = \sqrt{X^2 + Y^2 + Z^2} \). For each dog a least squares linear regression of VCG ST magnitude versus EST was calculated and the correlation coefficient was obtained.

**Results**

A total of 404 data points (average 27 per dog) were recorded during the interventions (table 1). Each data point representing the 20 epicardial ST segments and the three Frank lead ST segments were recorded at 2 to 5 minute intervals following coronary artery occlusion, reperfusion, and during pharmacologic administration. For each dog table 1 indicates the site and duration of occlusion, the pharmacologic agents employed, the number of recorded data points, and the correlation between the VCG ST magnitude and EST. The mean correlation coefficient \( r \) for all dogs was 0.921 ± 0.02 SEM. Since the number of data points varied among the animals, a number weighted average for \( r \) was calculated and was found to be similar at 0.918. Eight dogs were given drugs which alter myocardial oxygen requirements to demonstrate that the VCG ST and EST magnitudes were similarly affected (figs. 2–5). Isoproterenol uniformly increased the ischemic ST segments (fig. 5); methoxamine decreased ischemic ST segments (figs. 3 and 4); and nitroprusside increased the epicardial and precordial ST segments. The concordant movements of the VCG ST segments and the epicardial ST segments are demonstrated in figure 1 following coronary occlusion and in figures 2 through 5 following pharmacologic intervention.

**Discussion**

The present study establishes the close correlation between epicardial and precordial ST-segment changes following experimental coronary occlusion and drug-induced alterations in myocardial oxygen supply and requirements. Furthermore, the work herein extends this close internal-external electrical relationship to precordial electrocardiographic recordings employing the Frank vectorcardiographic lead system. Thereby an accurate and practical modality has been described for the noninvasive clinical evaluation of myocardial damage occurring in any portion of the left ventricular wall. This vectorcardiographic technique offers a valuable method for rapid, sequential examination of myocardial ischemic injury in acute coronary disease for the assessment of the course of native disease, the effects of pharmacologic and related interventions, and for the guidance of optimal application of therapeutic agents in the limitation of infarct size (figs. 3–5).

There has been substantial controversy regarding the reliability of directional ST-segment changes during acute myocardial ischemia. Supporting the accuracy of this technique have been the investigations of Maroko and coworkers who have shown that acute ST-segment elevation in an individual epicardial electrode of 2 mV or greater was associated with histologic evidence of infarction under the electrode when coronary occlusion was maintained for 24 hours. Greater levels of ST elevation were accompanied by more marked evidence of tissue infarction. In addition, these investigators showed that the degree of ST-segment elevation correlated closely with the extent of creatine phosphokinase depletion in the myocardium beneath the electrode. Further, the number of electrodes with ST-segment elevation indicated the size of the infarction, and the height of the ST segment indicated the severity of the ischemic process as manifested by the concurrent muscle enzyme diminution and ultimate fibrosis. It was further demonstrated that changes in the infarcted area induced by drugs could be measured by concordant alterations of epicardial ST segments and histologic damage. Thus when an intervention reduced in-

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** Representative demonstration of the simultaneously obtained ST-segment sum alterations recorded by the epicardial grid electrodes (open circles) compared to the ST-segment magnitude obtained from the external Frank XYZ leads (closed circles). The mean systemic arterial blood pressure (BP) is indicated by the open triangles with solid lines. Time and intervention (arrow) are expressed on the horizontal axis, and the ST-segment sums on the vertical axis as well as the mean blood pressure. Coronary occlusion on this and subsequent figures was carried out at time zero. Marked concordant EST and VCG-ST changes were recorded following occlusion which gradually decreased over the 3-hour period of observation.

![Figure 3](http://circ.ahajournals.org/)

**Figure 3.** Representative demonstration of concordant VCG-ST and EST following coronary occlusion, with methoxamine and reperfusion. The format is the same as figure 2.
Infarct size as outlined by epicardial electrodes, there was a concomitant decrease in the size of the ultimate histologically defined infarct. Conversely, interventions leading to an enlarged infarct area were associated with an increased number of epicardial electrodes showing ST elevation as well as increased tissue creatine phosphokinase depletion and pathologic documentation of expanded necrosis. From these observations, total myocardial damage was shown to be proportional to the sum of epicardial ST-segment elevations.

Simultaneous epicardial and precordial electrocardiographic recordings from dogs which underwent coronary ligation revealed close correlation of the precordial ST-segment sum and the epicardial ST-segment sum (r = 0.92). Subsequently, clinical work has shown directionally similar changes of the precordial ST sum with other indirect noninvasive measures of the area of acute infarction. Since the precordial mapping technique is limited to anterior located infarctions and is time-consuming in application, the feasibility of vectorcardiographic lead recordings in the assessment of ischemic injury has been suggested by a previous study showing that such recordings correlate well with the precordial ST-segment sum obtained by the 35-lead precordial blanket.

Our experimental findings validate the accuracy of the precordial vectorcardiographic lead technique by demonstrating that the VCG ST magnitude correlates closely with the EST sum. In addition, the observations herein document the close relation between VCG-ST and EST for both anterior and circumflex coronary occlusions, thereby extending the clinical application of the VCG method to infarction other than anterior in location.

Finally, it is important to emphasize that the principal purpose of the present study was to show the close correlation between external vectorcardiographic and epicardial lead ST-segment changes during acute alterations of myocardial energetics in different anatomical sites of the left ventricle. Although it is recognized that considerable controversy exists concerning the theoretical aspects of epicardial and precordial ST-segment mapping, this investigation was not specifically addressed to the problem of the electrophysiologic basis of ST-segment changes occurring with acute myocardial damage. Clearly ST-segment mapping possesses certain values and limitations which must be appreciated in its application and interpretation. Indices of multiple lead precordial ST-segment potentials do not provide direct quantification of myocardial infarction size. Furthermore, ST-segment changes associated with acute myocardial infarction are not stable but have a natural course of their own, as typified by the representative example in the present report (fig. 2) of the temporal alterations of ST-segment sums in a dog following coronary occlusion. Therefore, use of ST-segment deviations to estimate the extent and intensity of ischemic injury necessarily must take into account the duration of acute myocardial damage, as well as a number of factors unrelated to ischemia.
present time, the most important clinical utility of ST sum variations appears to be the assessment of directional changes in ischemic injury associated with acute myocardial infarction, concerning evaluation of the efficacy of interventions designed to limit acute myocardial damage. In this regard, the external 3-lead Frank vectorcardiographic system possesses important advantages over the relatively cumbersome 35-lead systems designed to limit acute myocardial damage. In patients with acute myocardial infarction, such vectorcardiographic monitoring has recently been shown to be of prognostic value relative to complications in patients with acute myocardial infarction.

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References
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