Clinically Practical Lead Systems for Improved Electrocardiography: Comparison with Precordial Grids and Conventional Lead Systems

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SUMMARY The use of limited leads for estimating total body surface potential distributions was investigated as a practical solution to the problem associated with extensive electrocardiographic sampling used in surface potential mapping. Two practical, limited lead sets of 32 leads each were derived and contrasted to a set of 30 precordial leads similar to those used in ST-segment and QRS mapping for estimating infarct size, and to a set of nine leads simulating those used in conventional 12-lead examinations. The two arrays, one of which excluded posterior sites for use in recumbent patients, showed little difference in ability to estimate 192 lead measured maps (average rms voltage error of 35 μV and average correlation coefficient of 0.97). The 30- and 9-lead arrays consistently showed twice the voltage (72 μV) and poorer pattern estimation (average correlation coefficient of 0.91) than either of the 32 lead arrays. These findings indicate the need for 20–35 properly located electrodes for accurate total body surface potential estimation. They also show that there is no difference in the abilities of a 30-lead precordial array and conventional leads to estimate maps.

EXTENSIVE MAPPING of body surface electrocardiographic potentials has been demonstrated to have significant medical merit. Improved recognition of myocardial infarction, detection of ventricular hypertrophy, estimation of extent of ischemia and sites of preexcitation have been reported.1,2 Evidence shows that cardiac states at high risk of ventricular arrhythmias can be identified by this means, and it is reasonable to expect that the size and severity of localized myocardial lesions, including infarction, can be estimated.3–11

Widespread medical use of body surface potential mapping requires practical methods of data acquisition, processing and storage, as well as the determination of criteria for differentiating various cardiac states. One significant step toward a practical system would be use of the minimal number of electrodes required to estimate accurately the total body surface potential distribution. Potential mapping studies to date have used 100 or more electrodes to acquire the electrocardiographic data, although certainly some of the data is redundant. Barr et al. reported that 24 electrodes at selected body surface sites were sufficient to obtain the useful signal content.12 Kornreich suggested that only three leads, together with the usual 12-lead electrocardiographic examination, were required to detect the additional electrocardiographic information in extensive examinations.13 In that study, however, the additional three leads were unique for each individual. In a recent study, we devised a technique for selection of a limited number of leads which permitted accurate estimation of the body surface potential distribution determined with 192 leads.14 The technique was optimal in the sense of least mean squared error for sequential lead selection.

In this study, we investigated the performance of four lead sets in their ability to estimate 192 lead potential maps. One lead set consisted of 32 electrodes selected by our technique (set I). Another lead set of 32 sites was selected by the same technique, but was constrained to exclude posteriorly located electrodes which are accessible in recumbent patients (set II). The third lead set was a 5 × 6 array of precordial electrodes similar to those used for estimation of myocardial infarct size on the basis of ST-segment elevation or the presence of pathologic Q waves (set III). The fourth lead set consisted of nine electrode sites approximating those used in the usual 12-lead examination (set IV).

Methods

Comparison of lead sets was performed on body surface potential maps from 90 normal subjects and 80 patients with 12-lead electrocardiographic evidence of cardiac abnormality. Abnormalities of the latter group included old myocardial infarctions, intraventricular conduction defects, ventricular enlargement and non-specific ST-T abnormalities (table I). Classification of infarction locations was based on conventional interpretation of 12-lead ECGs. Electrodes were placed in 16 vertical columns of 12 electrodes each. Columns were equally spaced around the thoracic circumference and horizontal electrode rows were evenly separated between a level just below the suprasternal notch and one just above the umbilicus. ECGs from the 192 electrodes were recorded simultaneously using multiplexing circuitry designed
for this purpose. ECGs from a single cardiac cycle were sampled at a rate of 1000 Hz. All ECGs were adjusted for gain and baseline, stored on digital tape and computer processed to provide isopotential contour maps on 16 mm film and hard copy paper.

The four lead sets are shown in figure 1, each superimposed on the 192-lead set from which it was selected. Set I consisted of 32 leads selected using the optimal, sequential algorithm. Set II was an array of 32 leads selected in the same way but constrained to accessible sites on recumbent patients. These lead sets are identical to the two 30-lead sets reported previously, except that two additional leads have been included in each set. From the work done in our laboratory and that reported by Barr, an exact number of required limited leads clearly cannot be specified. As the number of leads is increased, the precision with which complete maps may be estimated also increases, although the increased accuracy is small for a number of leads greater than 15. We have settled on 32 as an upper limit to the required number.

Table 1. Conventional Electrocardiographic Diagnostic Classification of Patients

<table>
<thead>
<tr>
<th>Condition</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>90</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td></td>
</tr>
<tr>
<td>Anterior</td>
<td>8</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>5</td>
</tr>
<tr>
<td>Anteriorinferior</td>
<td>3</td>
</tr>
<tr>
<td>Inferior</td>
<td>28</td>
</tr>
<tr>
<td>Inferolateral</td>
<td>3</td>
</tr>
<tr>
<td>Inferoposterior</td>
<td>1</td>
</tr>
<tr>
<td>Lateral</td>
<td>1</td>
</tr>
<tr>
<td>Posterior</td>
<td>4</td>
</tr>
<tr>
<td>Inferolateral-posterior</td>
<td>1</td>
</tr>
<tr>
<td>Left axis deviation</td>
<td>6</td>
</tr>
<tr>
<td>Non-specific ST-T abnormalities</td>
<td>13</td>
</tr>
<tr>
<td>Miscellaneous (LVE, RBBB, LBBB, I-V conduction defects)</td>
<td>7</td>
</tr>
<tr>
<td>Total</td>
<td>170</td>
</tr>
</tbody>
</table>

Abbreviations: LVE = left ventricular enlargement; RBBB = right bundle branch block; LBBB = left bundle branch block; I-V = interventricular.

Figure 1. Display of four lead sets compared in the study, each superimposed on the 12 × 16 array of electrodes used to obtain 192 lead maps. Correspondence of electrode location and thoracic anatomy in this figure and the following isopotential contour maps is the same. Uppermost and lowest rows of electrodes correspond to horizontal levels just below the sternal notch and just above the umbilicus, respectively. Far right and far left columns of electrodes correspond to vertical regions just left and right of the spine, respectively. A stylized sternum shows the anterior thoracic midline. Horizontal and vertical borders of the display represent electrode sites although the symbols (+, − or ×) have been left out for ease of viewing. Also, right and left borders of the display represent different electrode columns. Set I is an optimally determined array of 32 electrodes (panel A). Set II is an optimally determined array excluding posterior sites (panel B). Set III is a 6 × 5 array of precordial leads (panel C), and Set IV is a set of nine leads intended to simulate the conventional leads.
as this provides redundancy in the event of electrode or amplifier failure. Set I and II electrodes were selected using the same training data as that used for the two 30-lead sets described in reference 14. This data included all QRS distributions (over 11,000 frames) from 132 patients, of whom approximately half had documented heart disease and half had normal conventional ECGs.

Set III was a 6 × 5 array of electrodes located over the precordium and approximating those in use by other laboratories for ST-segment and Q-wave mapping in the setting of ischemic heart disease. Set IV consisted of nine sites approximating those used in conventional electrocardiography. Included are leads V1 through V6 and electrode sites close to right and left arms and left leg.

As reported in an earlier paper, the spatial redundancy in maps can be minimized using an optimal electrode selection procedure to arrive at a limited lead array. Once a limited lead set is determined, a linear transformation which minimizes the mean squared estimation error can be calculated from sample maps. This transformation permits the estimation of total surface potential distributions based on data recorded from the limited lead set. If \( \hat{\text{Z}}_2 \) is the vector of measured potentials and \( \hat{\text{P}}_2 \) is the vector of potentials to be estimated, a transformation, \( T \), is sought such that

\[
\hat{\text{P}}_2 = \hat{\text{Z}}_2 + T(\hat{\text{P}}_1 - \hat{\text{Z}}_1)
\]

where

\[
\hat{\text{Z}}_1 = E[\hat{\text{P}}_1] \\
\hat{\text{Z}}_2 = E[\hat{\text{P}}_2]
\]

and \( E \) is the mathematical expectation operator. It may be shown that

\[
T = K_{12}'K_{11}^{-1}
\]

where \( K_{11} \) is the covariance matrix of \( \hat{\text{P}}_1 \), and \( K_{12} \) is the cross covariance matrix of \( \hat{\text{P}}_1 \) and \( \hat{\text{P}}_2 \).

Potential distributions from the total data set shown in table I were grouped into training and testing sets. All QRS frames of each patient in the training set were used to calculate the transformations for each of the four lead sets. All QRS and ST-T frames of patients in the test set were then used to evaluate the estimation accuracy of each lead set and its respective transformation. The rationale for testing ST-T data using transformations trained on QRS data is that ST-T potential distributions are usually simpler than QRS distributions and usually closely resemble simple QRS distributions.

Measured and estimated 192-lead distributions for each QRS and ST-T frame of each patient in the test set were compared using two criteria. The first criterion was the correlation coefficient between measured and estimated map frames. If \( \hat{\text{P}} \) is the vector of measured potentials at a given moment and \( \hat{\text{P}} \) is the vector of estimated potentials, then

\[
\rho = \frac{\hat{\text{P}} \hat{\text{P}}^T}{|\hat{\text{P}}| |\hat{\text{P}}^T|}
\]

specifies the correlation coefficient. This number is sensitive to differences in the patterns of measured and estimated potentials and is independent of amplitude.

The second criterion for evaluating each lead set was the spatial rms voltage error referred to the sites at which potentials were estimated. If \( \hat{\text{P}}_2 \) and \( \hat{\text{P}}_2 \) are the vectors of measured and estimated potentials respectively, then

\[
\tilde{E} = \frac{|\hat{\text{P}}_2 - \hat{\text{P}}_2|}{(n)^{1/2}}
\]

gives the average voltage error at the n estimated sites. This number may be directly compared to the system noise. Average values of \( \rho \) and \( \tilde{E} \) were determined for each patient and for the test group as a whole.

Results

Before comparing lead sets, we tested the sensitivity of the estimation technique to the amount of data used for training. Over 5000 QRS distributions (at 1-msec increments) from a group of 57 patient maps were used to calculate the linear transformation for the 32 leads of set I (fig. 1). Approximately half the patients had normal and half had abnormal hearts as judged by conventional electrocardiography and history. Average correlation, \( \rho \), and rms error, \( \tilde{E} \), between measured and estimated potentials were determined for each QRS and T frame of each of the 57 maps in the training set. In addition, evaluation criteria were obtained for each QRS and T map frame from a second, independent set of 57 patient maps. The results shown in table 2 indicate that there was no significant difference in pattern estimation when training or test data were used as the test set. There was a significant but small increase in the rms voltage error when test data was used in comparison to training data. These results suggest that the estimation technique was not sensitive to the training data and that a sufficient number of maps had been used in the training set. Most of the data from the 114 patients in the combined test and training sets had been used in the lead selection procedure. The fact that much of the same data were used both for lead selection and transformation calculation should have little effect on sensitivity of map estimation accuracy, since the training and testing data were completely independent.

In order to compare lead sets I–IV, over 7000 QRS frames from the 82 patient maps of the data in table I were chosen as the training set and used to determine a linear transformation for each of the lead sets. Potentials of each QRS and T frame from the maps of the remaining 88 patients which formed the test set were then estimated using each lead set and its respective transformation. Most of the 82 patient maps used in

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**Table 1**

<table>
<thead>
<tr>
<th>Lead Set</th>
<th>QRS</th>
<th>ST-T</th>
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</thead>
<tbody>
<tr>
<td>I</td>
<td>✔</td>
<td>✔</td>
</tr>
<tr>
<td>II</td>
<td>✔</td>
<td>✔</td>
</tr>
<tr>
<td>III</td>
<td>✔</td>
<td>✔</td>
</tr>
<tr>
<td>IV</td>
<td>✔</td>
<td>✔</td>
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</table>

**Table 2**

<table>
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<tr>
<th>Lead Set</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>✔</td>
<td>✔</td>
</tr>
<tr>
<td>II</td>
<td>✔</td>
<td>✔</td>
</tr>
<tr>
<td>III</td>
<td>✔</td>
<td>✔</td>
</tr>
<tr>
<td>IV</td>
<td>✔</td>
<td>✔</td>
</tr>
</tbody>
</table>

---

**Figure 1**

The spatial correlation coefficient and rms error of several lead sets for a group of 57 patient maps.
the training set had also been used for electrode selection of sets I and II. On the other hand, very few of the 88 patient maps in the test set were used for electrode selection. Figure 2 shows performance criteria and rms map voltage plotted against time for each of the four lead sets on a normal subject. Voltage errors, \( E \), tended to be largest when signals were strong and pattern errors (\( \rho \)) tended to be highest when signals were weak. Plots of this type were made for each map in the study and average performance criteria were calculated from them for QRS and ST-T intervals.

Several examples of individual map frames and their estimated values are shown in figures 3–5 to illustrate the accuracy with which limited lead maps estimate the 192 lead maps. Panel A of figure 3 shows a normal subject’s 192 lead isopotential contour map at a moment late in ventricular activation. Isopotential contours are spaced 100 \( \mu V \) apart and the correspondence of electrode position to surface anatomy is as illustrated in figure 1. Panels B, C, D and E of the figure show the same map estimated by each of the 4 lead sets. Correlation coefficient and rms error are shown for each of the frames. Note the particularly poor pattern reproduction and large voltage error for the precordial array (set III) and simulated conventional leads (set IV).

Panel A of figure 4 is an early QRS map frame from a patient with an old inferior wall infarct. Note particularly the low amplitude negative distribution in the lower right lateral thoracic region. Panels B, C, D and E of the figure show the estimated maps for each of the four lead sets. Note that lead sets I and II accurately estimate the distribution, while the nine lead and precordial arrays do not, particularly in the area of the negative distribution. In this case, the estimated map from nine leads better represented the measured map than did that estimated from the precordial array.

Panel A of figure 5 shows a late QRS frame from a patient with an unusual positive pole in the upper right clavicular region. Panels B, C, D and E of the figure show the estimated maps and again the relatively poor estimation of the nine lead and precordial arrays in comparison to either of the 32 lead arrays. For this case, the map estimated with the precordial array better represented the original than did the nine-lead map.

Panel A of figure 6 shows an early QRS frame from a patient with an old anterior wall infarction. Note the positive pole on the posterior thorax. Panels B and C show the estimated maps for sets I and II, which demonstrate that the posterior distribution is not “missed” by either set. Panel A of figure 7 shows a left posterior pole in a later QRS frame from the same patient as in figure 6. Panels B and C show the frames estimated from sets I and II and demonstrate the ability of set II electrodes to retain posterior distributions in spite of the lack of posterior sampling electrodes.

Performance criteria were averaged over QRS and T for each patient map in the test set and an average was calculated for the group as a whole. The results are shown in table 3. These data show large differences in the abilities of the various electrode arrays to estimate surface potentials. Both pattern

<table>
<thead>
<tr>
<th>Test set</th>
<th>Correlation coefficient</th>
<th>RMS error (( \mu V ))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>QRS</td>
<td>ST-T</td>
</tr>
<tr>
<td>Training data</td>
<td>0.967 ± 0.029</td>
<td>0.929 ± 0.053</td>
</tr>
<tr>
<td>(57 patients: 31 normal, 26 abnormal; 5167 total frames)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Testing data</td>
<td>0.961 ± 0.014</td>
<td>0.919 ± 0.044</td>
</tr>
<tr>
<td>(57 patients: 28 normal, 29 abnormal; 5389 total frames)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Table 2.** Comparison of Average Performance Criteria for Set I When Training or Test Data are Used for Testing

**Figure 2.** Plots of rms voltage \( \dot{V} \) of 192 lead map, rms voltage error \( E \), and correlation coefficients \( \rho \) for each of the four lead sets vs time for a map from a normal subject.
Figure 3. Comparison of 192-lead measured isopotential map frame late in QRS from a normal subject (panel A) and map frames estimated from lead sets I, II, III and IV (panels B, C, D and E, respectively). Isopotential maps in this and the remaining figures are plotted at increments of 100 μV. Rms voltage error and correlation coefficient are given.

Figure 4. Comparison of 192-lead measured map frame early in QRS from a patient with an inferior wall myocardial infarction (panel A) and map frames estimated from lead sets I, II, III and IV (panels B, C, D and E, respectively).
error and rms error were larger for the nine-lead (set IV) and the precordial lead (set III) arrays compared with either of the 32-lead arrays (set I and set II). The data also show there was little difference in the abilities of set I or set II arrays to estimate maps. Moreover, the average performance criteria showed that map estimation from 32 properly selected leads was accurate to a correlation of 97% and rms error of 35 μV. Also relevant to the use of precordial mapping, there were no apparent differences in the average estimation errors of 30 precordial leads compared with the nine conventional sites. These findings suggest that the precordial array and nine-site array miss electrocardiographic information.

Finally, map estimation errors were grouped by diagnostic classification for each lead set in order to determine whether performance errors were sensitive to data types. Only maps from normal subjects (n = 38) and patients with old anterior (n = 7) or inferior infarctions (n = 13) were statistically compared, as these were the groups with sufficient numbers. No significant differences in performance criteria of each lead set over all three classes were found (p < 0.01, paired t test).

Discussion

Improved detection of cardiac disease through the use of body surface potential mapping has been reported in recent years. Most of these early reports of success in such areas as detection of infarction, ventricular hypertrophy, and preexcitation pathways have been based on mapping systems using many (100–200) electrodes. The refinement of electrocardiographic diagnosis, both in the accuracy and resolution with which disease may be classified and measured, is clearly indicated by these studies.

Our investigation was aimed at verifying the hypothesis that surface potential maps estimated from a limited number of properly located thoracic leads are accurate enough to be used in place of the cumbersome and expensive complete mapping systems. Confirmation of this hypothesis would permit more widespread use of mapping, because of the greater practicality of acquiring and processing data provided by the limited lead systems.

Our results indicate that the use of 32 appropriately located leads provides an accurate estimate of the total body surface distribution of electrocardiographic potentials. However, several points must be considered. First, the problem of finding the number and placement of electrodes required for adequate sampling of surface potentials has a nonunique solution. Our experience and that reported by Barr,12 and more recently by Warren (PhD dissertation, Duke University, 1977), suggests that for a given number of leads, there are many lead sets which will perform comparably with insignificant differences between them. In this sense there is no absolute optimum array, but a great many "near-optimal" sets. As to the
number of leads required, results suggest the need for 20-35 electrodes. The goal of limited lead mapping is to provide an accurate representation of the total body surface ECG. Any limited lead set has a probability of missing significant map features such as small, localized poles, or poorly estimated potential distributions, in individual cases. These errors should be minimized, while keeping the number of electrodes within practical limits. The 32 leads of set II, currently used by our laboratory for clinical mapping studies, appear to be a reasonable compromise between accuracy and practicality. Moreover, this set provides some redundancy to compensate for data loss resulting from electrode failure or excessive noise on a given lead.

A second consideration is the type and amount of data needed for training the transformation and the accuracy of the limited lead system when applied to all varieties of cardiac abnormalities. All maps reported in this study were obtained from adult patients. Training data used in the calculations of the transformation included maps from normal subjects and patients.
with diagnosed heart disease. The result, showing little or no difference in performance criteria when test and training sets were mutually exclusive, is a positive statement of adequate training. The fact that performance criteria for a given lead set were not significantly different for any of the diagnostic classes further supports this conclusion. In addition, maps from patients with various types of heart disease were reproduced accurately from limited leads, suggesting that inclusion of many more maps with widely differing diagnostic classifications would not reduce estimation errors. The implication of these results is that the relationship between measured and estimated potentials is primarily determined by the physical relationships of the sites, e.g., body shape and size and site-to-site distances. The generator characteristics of the heart appear to be secondary in their effect on interrelationships of surface potentials. Therefore, a separate transformation might be desirable for each body type, i.e., adult, adolescent and infant, male and female. This could become impractical if carried to extremes, and further studies should be performed to determine whether significant improvement in map estimation results from such “tailored” transformations.

Finally, the increased ease with which set II electrodes can be applied and the insignificant reduction of performance compared with the unconstrained 32-lead set (set I) appears to justify its use as a clinically practical system. The estimated distributions in figures 6 and 7 demonstrate that the lack of posterior sampling sites does not significantly affect estimation pattern or voltage errors. Typically, 10–20 minutes are required for marking the patient’s chest, applying the electrodes and recording the data. This is well within practical times for data acquisition on a large scale.

In summary, the results demonstrate a practical and accurate method for estimating total body surface potential distributions using 32 properly located electrocardiographic leads. The results suggest that information recorded from precordial electrode arrays frequently miss or distort information of definite diagnostic importance.

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