Body Surface Distributions of Repolarization Potentials After Acute Myocardial Infarction

II. Relationship Between Isopotential Mapping and ST-segment Potential Summation Methods

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SUMMARY Isopotential body surface distributions derived from 14 subjects with acute anterior and 14 patients with acute inferior myocardial infarction were studied to examine three possible difficulties with ST-segment summation methods as used for evaluation of infarction severity. First, recorded sums of potentials from 150 electrodes placed on anterior and posterior thoracic surfaces and from five subsets of 42 electrodes located only on the left anterior precordium were compared. Lead subsets detected only one-half of total thoracic positivity in acute anterior infarction and the sums of potentials in these sets were significantly altered by small (approximately 1-1.5-inch) deviations in grid orientation. Second, differing isopotential distributions could yield nearly identical sums of potentials in left precordial electrode grids. Third, the effects of the wide variations in the normal ST-segment isopotential distribution were evaluated by studying 45 normal subjects and application of difference map and departure map techniques. The results illustrate the significant variation in potentials considered to be generated by the ischemic lesion caused by differences in control patterns. Thus, the recording of potential sums over limited torso areas, the attention to potential magnitudes rather than to distributions and the assumption that the normal ST segment is isoelectric represent significant problems in applying ECG methods to the quantitative evaluation of myocardial infarction.

STUDY of the electrocardiographic ST-segment is of proved value in the diagnosis and localization of acute myocardial infarction. Recent studies have attempted to extend these applications to include the assessment of the size or severity of the acute ischemic lesions. Techniques to register these ECG potentials have included standard electrocardiography and vectorcardiography, summation of potentials recorded from left precordial multielectrode grids and, most recently, body surface isopotential mapping. The last two approaches are clearly related, in that the data set recorded by the former is but a specific subset of that sensed by the latter. Thus, it may be supposed that the adequacy of left precordial electrode grid methods may be evaluated by determining the properties of the parent set of total thoracic surface potentials.

We used this approach to investigate three expected but heretofore unquantitated difficulties with the left precordial summation approach. These include (1) the assumption that the premorbid or normal ST segment is truly isoelectric, and hence, that all postinfarction potentials are due to the ischemic lesion; (2) the recording of potentials over a fixed and limited portion of the thoracic surface, even though ischemic lesions may vary in location and orientation; and (3) the attention to potential sums without consideration of the spatial distribution of the injury-current forces.

Methods

Study Populations

Twenty-eight men, ages 44-63 years, were investigated during the first 48 hours after the onset of acute myocardial infarction, as diagnosed by standard historical, electrocardiographic and enzymatic techniques. All patients were hospitalized in an intensive care area, were clinically stable with at most clinically mild congestive heart failure and were in sinus rhythm without bundle branch block. None was receiving digitalis glycosides or antiarrhythmic agents other than lidocaine, which was routinely administered. Serum electrolytes were normal in all 28 men.

Forty-five normal volunteers were also studied. Normality was assumed after a complete medical history, physical examination, standard electrocardiography and chest roentgenography. All subjects in both groups offered voluntary, informed consent before study.

Electrode Systems

ECG signals were sensed from 150 chloridized silver electrodes located on the anterior and posterior thorax, extending from the level of the clavicles to the inferior rib margins. Extremity electrodes were similarly deployed to record the standard bipolar and unipolar ECG leads and to determine the Wilson central terminal voltage.

Data Acquisition

The hardware and software in data acquisition have been described and will only be summarized here.
ECG signals were amplified by 33 capacitor-coupled, differential-input (grid electrode vs Wilson central terminal potential), variable-gain and variable-offset amplifiers. Gains were individually set at 1000-16,000 and offsets at -4.5 to 4.5 V under computer control, so that the amplifier output filled the input range of the analog-to-digital converter. Time constants were set at 2 seconds.

Five sets of ECG potentials were recorded, each including 30 grid-electrode potentials and standard leads I, II and III. These latter three leads served to document the stability of the recordings and to verify the correct merging of the five data subsets. A 20-second epoch was digitized for each subset at a sampling rate of 500 samples/channel/sec.

Data Processing

Specific techniques for the averaging and display of recorded thoracic potentials have also been described.8,12 The five sets of 30 electrode potentials were merged and cycles in each lead with numerically determined morphologic similarity were averaged to yield one set of 150 averaged unipolar thoracic wave forms. Data sets with significant baseline wander were discarded.

Isopotential maps were constructed from these wave forms at 2-msec intervals from the end of the QRS complex to the termination of the T wave. Potentials during the terminal 50 msec of the TP segment were averaged for service as a baseline to avoid inclusion of U-wave forces. Onsets and offsets of all intervals were manually determined from root-mean-square (RMS) potential plots. A combined linear-bilinear interpolation routine was used to draw contour lines at zero and at plus and minus 10, 20, 40, 60, 100, 150, 250, 400 and 600 μV.

Statistical differences were assessed using the paired t test for single and one-way analysis of variance routines for multiple comparisons. A 1% confidence limit was relied upon. All values were expressed as mean ± SD.

Results

Fourteen subjects had an acute anterior myocardial infarction based on standard electrocardiographic criteria (ST-segment elevation in precordial leads V1 to V6, with or without ST-segment elevation in leads I and aVF). An equal number with inferoposterior lesions (ST-segment elevation in leads II, III and aVF, with or without right precordial ST-segment depression) were studied.

Isopotential Patterns

Isopotential distributions recorded 80 msec into the ST-segment from one subject with an acute anterior and from another with an acute inferior myocardial infarction are presented in figure 1. The pattern during anterior infarction (fig. 1A) was characterized by an intense anterior maximum, or zone of peak positive potential, near the V6 electrode site. Negative potentials were diffusely spread over much of the posterior thorax. Peak positive and negative potentials were 601

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Isopotential body surface maps from a subject with an acute anterior myocardial infarction (A) and from a subject with an acute inferior myocardial infarction. Patterns are drawn from potentials sensed 80 msec into the ST segment, as indicated by × on the plots of the standard ECG lead in each panel. Plus and minus signs identify the electrode locations, with the sign corresponding to the polarity of the registered potential. The sternal notch is indicated by "V" and the six standard precordial ECG sites are indicated by solid triangles. The left and right edges of the map are along the vertebral column. Contour intervals are at a plus and minus 10, 20, 40, 60, 100, 150, 250, 400 and 600 μV; zero-level isopotential lines are overlaid. The top and bottom margins of the map are inferiorly displaced in axillary areas, reflecting the lower positions of the electrode strips in these areas. Shaded areas identify electrode sites used for lead subset studies, as detailed in the text.
\( \mu V \) and \(-66 \mu V\), respectively. As previously reported,\(^8\) this pattern remained stable during much of the ST segment.

In contrast, the distribution after acute inferior infarction (fig. 1B) was characterized by an anterior minimum, or zone of peak negative potential. Positive potentials surrounded the inferior and right lateral thoracic regions. Peak positive and negative voltages measured 157 \( \mu V \) and \(-274 \mu V\), respectively.

**ST-segment Potential Summations**

Sums of all positive potentials, registered 40 and 80 msec into the ST-segment, in six different electrode sets were computed for subjects with anterior infarction. The first electrode set consisted of all 150 thoracic electrodes. The second included only the 42 electrodes covering an area analogous to that covered by the left precordial grid described by Maroko et al.,\(^4\) i.e., electrodes located from the right parasternal to the left posterior axillary region, as indicated by the shaded zones in figure 1. This second set recorded only 45.15–69.87% (mean 57.48 \( \pm 9.40\%\)) of all positive voltages at the earlier instant, and 45.09–66.41% (mean 57.96 \( \pm 6.83\%\)) at the later instant. The differences in potential recorded by the two sets were highly significant (\( p < 0.001\)). Thus, lead sets limited to the left precordium sensed only somewhat more than half of total thoracic positivity.

Electrode sets three and four were constructed by shifting the 42-electrode grid one electrode column to the left or to the right, respectively. This was a translocation of approximately 1.5 inches in either direction. Sets five and six were designed by shifting the second electrode set one electrode row (1 inch) superiorly or inferiorly, respectively.

Results of these four translocations are shown in figure 2. The leftward shift of set 2 to set 3 resulted in a significant (\( p < 0.01\)) reduction in the calculated sum of positive potentials at both time points. Reductions at 40 and 80 msec after the J point ranged from 0.01–17.09% (mean 9.81 \( \pm 5.59\%\)) and from 2.00–14.04% (mean 7.97 \( \pm 4.37\%\)), respectively.

A rightward shift from set 2 to set 4 caused an increase in the sum in all cases. Magnitudes varied from 7.97–24.99% (mean 16.10 \( \pm 7.37\%\)) 40 msec into the ST segment and from 6.14–22.91% (mean 13.94 \( \pm 5.78\%\)) 80 msec into the ST segment. Superior translocations caused statistically significant differences in ST-segment potential sums at 80 msec (8.45 \( \pm 6.02\%\)), but not at 40 msec (6.77 \( \pm 6.50\%\)). Similarly, an inferior shift resulted in a significant difference only at the earlier instant (6.43 \( \pm 5.02\%\) at 40 msec and 1.61 \( \pm 5.35\%\) at 80 msec). Thus, left precordial summation methods were sensitive even to small variations in the topographic relationship between the ischemic lesions and the electrode configuration.

**Distributional vs Summation Methods**

Total thoracic isopotential patterns were compared with the results of the summation methods. Examples illustrating discrepancies between the two methods are presented in figure 3.

The patterns displayed in figure 3 were recorded from three subjects with anterior infarctions, 80 msec into the ST segment. The sum of all positive potentials (lead set 1) in figures 3A and B were 5617 and 5523 \( \mu V\), respectively. In contrast to this similarity, the sums of positive potentials sensed by lead set 2 were 3814 \( \mu V\) and 2513 \( \mu V\) in figures 3A and B, respectively. This difference resulted from the wider dispersion of positivity in the example in figure 3B, spreading from the third right column to the fourth column from the left margin, than in figure 3A. Additionally, peak positive potential in figure 3A (350 \( \mu V\)) markedly exceeded that in figure 3B (210 \( \mu V\)). Thus, similar sums of total surface positivity can result from significantly different thoracic distributions. Similarly, differing percentages of similar total thoracic potentials sums may be sensed by a limited system of electrodes.

Comparisons between figures 3A and C extend these observations. The sums of ST-segment positive potentials recorded from lead set 2 were 3814 \( \mu V\) and 3754 \( \mu V\), respectively. Total thoracic positive potential sums (lead set 1) were disparate, being 5617 \( \mu V\) in figure 3A and 6952 \( \mu V\) in figure 3C. Review of the potential distributions revealed definite differences, particularly in the locations of low-amplitude positive and negative potentials. Thus, ST-segment sums computed from limited lead systems were nearly identical, whereas the overall potential distributions may be significantly different.
Effects of Normal ST-segment Potential Patterns

That the normal ST segment is not isoelectric is well known. Two approaches were used to correct postinfarction ST-segment patterns for this normal but highly variable premorbid distribution. First, three normal subjects with differing ST-segment patterns were selected. The distributions from these normal persons, 80 msec into the ST segment, are presented in figure 4. The patterns differ in the distribution of low-level positive and negative potentials and in the magnitude of peak positive voltages (413 μV, 325 μV and 177 μV in figures 4A, B and C, respectively). Each of these three normal patterns was subtracted from the 80-msec distribution of each subject with acute infarction. The resulting difference maps were considered to represent the surface distribution generated by the ischemic lesion if the control distribution was the selected normal pattern.

In each case, the subtraction of the three normal patterns resulted in different postinfarction difference maps. One example is presented in figure 5. The postinfarction pattern from this subject with an anterior infarction was presented in figure 1A. Patterns in figures 5A, B and C were computed by subtraction of patterns in figures 4A, B and C, respectively, from that of figure 1A. Differences include variations in potential distributions and the appearance of dual maxima in figure 5B but not in the other two patterns. Peak positive potentials were 454 μV in figure 5A, 336 μV in figure 5B and 491 μV in figure 5C.

ST-segment potential sums were computed from each of the three difference map voltages for each subject using the lead sets 1 and 2 (total thoracic and left precordial grid deployments). For data recorded 80 msec into the ST segment, sums from both electrode patterns were significantly different (p < 0.01) when directly recorded potential patterns were compared with each of the three difference voltages and when each difference pattern was compared with any other such distribution. Thus, variations in control or
premorbid ST-segment voltage distribution cause significant variations in ST-segment distributions and potential sums computed using difference map techniques.

Similar results were recorded by subtraction of the three normal patterns from inferior infarction isopotential distributions. Isopotential difference maps computed by subtracting each normal distribution in figure 4 from the pattern presented in figure 1B are presented in figure 6. Definite differences in the distribution of, particularly, the low-level positive and negative forces are apparent. A distinct anterior minimum was always observed. Negative forces were accentuated, with peak negative voltages of −468 μV, −381 μV and −307 μV in figures 6A, B and C, respectively.

A second approach was based upon the departure map concept developed by Flowers et al. to detect abnormalities in QRS potential distributions. Confidence limits (± 2 standard deviations) for potentials at each 2-msec sampling instant and at each of the 150 electrode sites were computed using the 45 normal subjects as the data base. Deviations of potentials of each abnormal subject from these confidence limits were determined and plotted as departure maps; a result of zero was computed if the potential of the test subject at a given locus and instant was within the computed confidence limits.

An example of an isopotential distribution from one subject with an inferior lesion sensed 40 msec into the ST segment was presented in figure 1B. The departure map constructed by comparing the pattern in that map to the above described confidence limits is presented in figure 7A. The abnormal anterior negativity seen in figure 1B is accentuated after normalization, as expected from the normal positive
FIGURE 5. Three difference maps, constructed by subtracting the three normal patterns in corresponding panels of figure 4 from the anterior infarction pattern in figure 1A. Conventions are as in figure 1.

Effects of normalizing distributions from subjects suffering anterior infarction were more complex. In figure 1A, an isopotential pattern from a subject with an acute anterior infarction was presented, corresponding to potentials registered 80 msec into ST segment. A diffuse and intense anterior maximum (peak potential of 601 μV) was recorded. The departure map depicting deviations of that pattern from the normal range is presented in figure 7B. Positive potentials localized to the left anterior chest were the only ones deviating from the normal range; the maximal deviation was 262 μV.

Another example of an isopotential distribution recorded 80 msec into the ST segment of another subject with an acute anterior lesion is presented in figure 7C. The peak positive potential measured 246 μV. When compared with the normal range, no electrode potential deviated from the 95% confidence limits. The departure map was thus totally flat at this instant, as at all time points from 40–80 msec into the ST segment. The standard ECG, midprecordial ST-segment form, however, clearly indicated myocardial injury, and the clinical and laboratory data were diagnostic of an acute myocardial infarction.

Discussion

Studies detailed in this report were intended to identify and to quantitate three deficiencies of left precordial ST-segment potential summation methods for assessment of the extent of ischemic lesions. That each problem should exist was intuitive. However, the quantitative effects of each could only be assessed by comparison with recordings of total thoracic potentials from normal subjects and from those with acute myocardial infarction. Because such data only recently became available,9,12 such an evaluation has not been reported.

The current study was limited to evaluation of electrocardiographic techniques. Other noninvasive or
pathologic determinations of lesion size were not available to correlate with the electrical measurements. Thus, the actual differences in the accuracy of the methods caused by the studied effects cannot be quantitated. The purpose of the study, however, was to provide useful information relating to interpretation of frequently applied methods, not to localize the existing lesions.

The first problem studied was the effect of the variable topographic relationship between ischemic lesions and fixed, limited-size electrode grids. If variable portions of the electrical fields generated by two lesions in different subjects are sampled, it would be unlikely that the sensed information could provide quantitative comparisons of the two abnormalities. Indeed, it is widely accepted that ST-segment summation methods cannot quantitate tissue loss but can only determine directional changes of given insult. The quantitative basis for this is expressed in figures 1–3. First, percentages of total thoracic positivity sensed by left precordial grids varied from 45–70%, related to the distribution of positive potential on the chest surface. Second, small differences in lesion and electrode grid locations produced significant changes in calculated potential sums (fig. 2). This would clearly be expected from the nonuniform and complex thoracic isopotential distributions (figs. 1 and 3). Similarly, small differences in electrode deployment would result in analogous errors.

A second problem was investigated with the use of summation methods without consideration of the distribution of the abnormal potentials. Body surface isopotential mapping has been repeatedly shown to be advantageous by relating surface and epicardial electrical events. This is dependent upon not only the wide area explored, but also upon the method of display of potential patterns. In particular, close attention to locations and movements of extrema and low-level potential forces are needed to detect variations in source activity. The possible resulting errors are illustrated in figure 3; similar potential sums may be computed for distributions that differ in many other

Figure 6. Difference maps computed by subtracting each of the three normal distributions of figure 4 from the pattern after inferior infarction in figure 1B. Conventions are as in figure 1.
ways, falsely suggesting a similarity in lesion properties. As noted by Spach et al.,\textsuperscript{12} such differences may reflect very different source characteristics.

The last problem explored was the effect of the normal ST-segment potential distribution on postinfarction map patterns. That this difficulty should exist is predicated upon the common operational consideration that the normal ST segment is truly isoelectric and that all potentials recorded after an event are due to an experimental or natural occurrence. Obviously, this would affect all ECG measurements, not just those derived from limited-size grid systems. Two studies have detailed the normal ST-segment potential pattern.\textsuperscript{12,14} Distributions during the midportions are typically characterized by an anterior maximum near the V\textsubscript{3} electrode site, in figure 4. The magnitude of the potential varies considerably, as is well recognized in routine electrocardiographic study. In the 45 normal subjects reported here, peak potentials 80 msec into the ST segment were 177-413 $\mu$V. Thus, the range of normal upon which an infarct pattern is superimposed may be significant.

This remains a difficult problem to approach because, in contrast to routine ECGs, control isopotential maps are rarely available for comparison. Two approaches were used in this study. First, three normal patterns with widely varying maximal potentials were subtracted from each postinfarction distribution. The resulting difference map depicts the potential that would have been generated by the in-
In the context of myocardial infarction itself if the normal pattern used as the sub-tractor had been the pattern before infarction. As anticipated, the difference maps and potential sums varied significantly, depending upon the baseline distribution (fig. 5).

A more readily applicable approach to the consideration of normal distributions was proposed and applied to evaluation of QRS potentials by Flowers and associates. This method of departure mapping, as applied to ST-segment forces in this report, is based upon detection at any instant and thoracic locus of potentials that deviate from the 95% confidence limits of a normally distributed control population.

When applied to patterns detected after inferior infarction, marked deviations from the normal pattern were determined (fig. 7). In contrast, potentials beyond the normal range after anterior infarctions were typically restricted to intense positive forces localized to portions of the left precordium (fig. 7B). This would be expected from the general similarity of anterior infarction injury-current distributions (fig. 1) and the normal ST-segment isopotential form (fig. 4). An extreme example of this similarity was presented in figure 7, in which the individual patterns during the ST segment were not statistically abnormal. In contrast, the sequence of potential variations during the ST segment at individual electrode sites was deformed in a way characteristic of myocardial injury. This observation is directly analogous to the patterns described by Pardee, in which the ST segment is altered in shape without major elevation.

These data suggest that departure mapping can detect limited distributions of abnormal potentials after acute infarction, and may thereby be of value in quantitating infarction-generated electrical forces. However, electrical distributions at individual instants must be considered within the context of the temporal evolution of ventricular repolarization. Thus, potential sums or distributions recorded at a specific time may lead to false quantitative or diagnostic conclusions.

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