Importance of the Great Vessels in the Genesis of the Electrocardiogram

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The electrocardiogram is the graphic representation against time of the difference in potential between points of the body caused by the current field of the heart. To examine the origin of this current field, a method of transforming body surface electrocardiographic data to the epicardial surface has been developed. The computed epicardial current density distributions in 219 patients with acute inferior myocardial infarction showed that, in 89% of patients, the current flow out of the heart during the ST segment came from two regions, not only from the infarction region but also from a region over the great vessels. This finding suggests that current flows from the ischemic region, through the low-resistance pathway provided by the intracavity blood, out the great vessels, and back to the epicardium. A similar pathway has been hypothesized when ischemia causes endocardial ST elevation, such as during a stress test or with unstable angina. To test this hypothesis, a group of patients with ST depression on the 12-lead electrocardiogram, not associated with ST elevation, was examined with body surface mapping. Ninety-four percent of patients had epicardial current density distributions that showed a region of current flow out of the heart and over the great vessels that was consistent with this hypothesis. This could explain the poor localization of coronary artery disease by electrocardiographic techniques when there is ST depression on the body surface. (Circulation Research 1990;66:1081–1087)

The electrocardiogram (ECG) is the temporal record of the potential difference between two points on the body surface recorded with time. The potential difference is set up by the current field generated by the heart. The regional information from the body surface ECG in general bears a relation to the source; however, ST segment depression has been found to be a poor localizing sign in coronary artery disease, especially with exercise.

We have studied patients with acute inferior infarction by use of body surface mapping and an inverse transformation to produce epicardial surface potential distributions. The epicardial maps during the ST segment showed a positive region over the superior aspect of the heart in addition to the expected inferior ST elevation. This suggested a selective current path from the infarcting endocardium that traveled out of the heart through the great vessels. We hypothesized that this current path might also be active in conditions such as ischemia, in which there was ST depression on the body surface 12-lead ECG. To test this hypothesis in patients with ST depression, we examined the epicardial ST maps of patients with ST depression but who showed no evidence of ST elevation on the 12-lead ECG.

Materials and Methods

Collection of Body-Surface Electrocardiographic Potentials

The system for acquisition, display, and recording of the body surface electrocardiographic data has been described previously.\(^9,10\)

In brief, a jacket with a fixed array of 50 electrodes is wrapped around the patient starting from a position 7.5 cm to the right of the sternum, so that the second column of electrodes is on the midternal line. A bedside display of the sampled electrocardiographic signals enables bad leads to be selected and replaced by an interpolation from the surrounding leads. Linear baseline drift is corrected by interpolation between baseline points picked manually in successive TP segments.

The number of electrodes in contact with the patient varies with the size of the patient, which enables virtually all patients to be studied. Electrode positions are calculated from the degree of overlap of the jacket, which is recorded at the time of mapping.
Although electrode positioning varies between patients, body surface maps comparable among patients are produced by using an interpolation scheme that takes into account the size of each patient. A spline interpolation method is used. This method produces 32 columns each of 12 rows that are evenly distributed around the thoracic surface. This format is comparable with that used by other groups. A complete recording can be made within 5 minutes of approaching the patient.

The Inverse Transformation

The epicardial potentials are calculated from the body-surface data by multiplying that data by a matrix derived from a numerical model of the electrical properties of the human torso. The technique used to model the torso has been described previously. Briefly, the model simulates, on a nonuniform rectangular grid, a three-dimensional resistor network that resembles the human torso both geometrically and electrically. The lungs, spine, sternum, and heart were included in the model. The values used for tissue resistivity in the model are those published by Rush et al.11 The torso geometry has been digitized from computed tomographic scan data obtained from a 40-year-old male subject in whom 22 computed tomographic scan slices were available, spaced at 1 cm. In the region of the heart, the slices were spaced 1 cm apart; elsewhere, slices were spaced 2 cm apart. No interpolation between slices was performed. To produce torso models of realistic size, the last (lowest) slice was repeated several times at a spacing of 2 cm to give an overall torso height of 45 cm. The extended region started just below the lungs and was homogeneous except for the inclusion of the spine.

Each computed tomographic slice was divided into a grid with a resolution of 7.5 mm when in the vicinity of the heart; as the distance from the heart increased, the resolution increased to 1 cm and then to 1.5 cm. The model contained 34,914 nodes (points at which potential calculations are performed). There were 7,237 nodes on the body surface and 807 on the epicardial surface. Intracardiac blood masses were not modeled; the inverse calculations are not affected by the internal structure of the heart because the epicardial surface nodes at which the potentials are calculated form a closed surface surrounding the heart. This surface envelops all cardiac chambers and transects the great vessels at the point at which they leave the heart. No attempt was made to model the anisotropic skeletal muscle layer. Although this is possible when using a resistor-network model, the data on muscle fiber distribution and orientation are not easily available and are not easily incorporated into the model.

By use of the model, the epicardial potential distribution (h) and the torso surface potential distribution (b) were related by the following matrix equation:

\[ \mathbf{Th} = \mathbf{b} \]  

where \( \mathbf{T} \) is a matrix that reflects the geometry of the torso. To construct the matrix \( \mathbf{T} \), the epicardial nodes were first grouped to form 50 source regions of approximately equal size, which covered the epicardium, and the potential was assumed to be equal at all of the nodes within any one source region. Thus, the vector \( \mathbf{h} \) has 50 components: the potentials at the 50 epicardial source regions. The number of epicardial source regions chosen affects the inverse calculation, and some preliminary results of calculations with 26, 50, 74, and 98 regions have been published.12 The vector \( \mathbf{b} \) has 160 components, corresponding to 160 potentials at body surface sites where (interpolated) body surface map data were measured.

A regularization method13 was used to solve Equation 1 for \( \mathbf{h} \) in terms of \( \mathbf{T} \) and \( \mathbf{b} \). The solution obtained is given by the equation:

\[ \mathbf{h} = (\mathbf{T}^\top + \alpha \mathbf{I})^{-1} \mathbf{T} \mathbf{b} \]  

where \( \mathbf{I} \) is the appropriately sized identity matrix, \( \mathbf{T}^\top \) is the transpose of matrix \( \mathbf{T} \), and \( \alpha \) is a parameter that determines the amount of “smoothing” present in the solution. The choice of \( \alpha \) was made so that the calculated epicardial potentials were at physiologically reasonable levels while maintaining good correspondence between the measured body surface data and the body surface distribution that would be generated by the calculated epicardial potentials. There are also statistical methods for determining the optimum value of \( \alpha \) that require assumptions to be made about the nature of the body surface noise and the epicardial potentials.

Calculation of Epicardial Current Densities

Once epicardial potential distributions have been calculated, it is simple to relate body surface potential to the current density flowing out of an epicardial region. This computation essentially considers the potential at each epicardial node and adjacent non-heart nodes; Ohm’s law is used to calculate currents flowing between the nodes. The resolution for these current maps is the same as the resolution of the isopotential maps: 50 equal-sized regions around the heart, each with an approximate surface area of 5 cm².

Data Display

The epicardial potentials are plotted as isopotential contour maps on the surface of the heart. These are displayed on six perspective views generated as though one were looking at the heart in situ from viewing positions that were anterior, left lateral, posterior, right lateral, inferior, and superior to the heart. The solid lines are positive isopotential contours, the heavy solid line is the zero contour, and the dashed lines are negative isopotential contours (see Figures 1–5). The boundary of the heart visible in each view is shown by the finely spaced dotted line.
Patient Selection

Inferior Infarction. All patients who were entered into a study of the prognosis of inferior infarction were entered into this study. All patients presented with no previous history of infarction and with QRS duration 0.1 second or less, 12-lead electrocardiographic evidence of at least 1 mm of ST elevation in at least two of leads II, III, and aVF, an enzyme rise, and a history typical of acute myocardial infarction.

ST Depression. Patients who were routinely mapped in coronary care and who showed ST depression of at least 0.1 mV in at least two of the 12 standard leads and who had no detectable ST elevation were chosen for this group.

Grouping System

To group similar ST potential distributions, a hierarchical clustering technique was used. The measure of similarity between two distributions was taken to be their correlation coefficient, which is calculated as follows: Given two potential distributions A and B, considered as vectors (a) and (b), correlation coefficient = A.B/|A||B|, where A.B = Σaibi and |A| = √Σai2, |B| = √Σbibi. The correlation coefficient is independent of the magnitudes of the two vectors and gives an indication of the similarities in both pattern and position of maxima and minima. Similar maps have a correlation coefficient near 1.0. Dissimilar maps have a correlation coefficient near 0.00, and maps similar in pattern but with positive and negative values exchanged have a correlation near −1.0. Correlation coefficients have been applied to body surface maps previously in an attempt 1) to distinguish normal body surface maps from those taken from patients with myocardial infarction, 2) to assess the similarity between modeled and measured body surface maps in theoretical studies, and 3) by ourselves, to group patients with inferior infarction in a clinical correlation.

Statistical Analysis

Epicardial maps were analyzed on a regional basis, with regions over the great vessels selected for examination. Maps of both potential distribution and current density were examined. An area of positive potential over the great vessels was diagnosed when there was a confluent area of at least three of the nine superior epicardial regions positive with respect to the Wilson central terminal. With the current density maps, the current coming out of the heart is equal to that flowing into the heart so that a positive region represents current out of the heart. A positive area was defined as a confluent area with at least three of nine superior regions being positive.

Results

Inferior Infarction

A typical body surface distribution of potential of a patient with an inferior infarction is shown in Figure 1. The epicardial potentials and the epicardial current density distribution of the same patient are shown in Figure 2. The region of ST elevation on the superior aspect of the heart can be clearly seen in addition to the inferior ST elevation. This is also seen as a region of current source centered over the great vessels. The clinical and body surface electrocardiographic data from this group of inferior infarction patients are fully described in previous papers. The mean time from the onset of chest pain to mapping was 7.0 hours. Epicardial maps of both potential and current density of the 219 patients in these studies showed that 210 (96%) had a region of superior ST elevation of potential and 194 (88.6%) had a region of current flow out of the heart over the region of the great vessels.

ST Depression

There were 93 patients with ST depression on the 12-lead ECG. Of these, 53 patients had non-Q wave infarction, and 40 had no evidence of infarction. The body surface map patterns of these patients were grouped into seven categories; the mean maps are shown in Figure 3. In 88 (93.7%) of the epicardial maps of these patients, a region of current flow out of the great vessels was seen. The 12-lead ECG and body surface map of a typical patient are illustrated in Figure 4. In Figure 5, the epicardial maps of the patient illustrated in Figure 4 show a similar pattern superiorly to that seen in inferior infarcts (Figure 2).

Discussion

The Origin of ST Potentials

During the ST segment in patients with infarction, current flows from the region of ischemia or infarction to the normal muscle and effectively causes the potential of the infarcting region to be elevated compared with the normal region. In our patients
with inferior infarction, the presence of a region of ST elevation over the great vessels was unexpected. A possible explanation in these patients is that the low resistance pathway of the blood appears to form a selective conduction path from the abnormal region of endocardium, to both the normal endocardium and the epicardium, by a circuit including the great vessels. According to this hypothesis, all patients who have endocardial ST elevation should show a similar current path and have superior ST elevation.

The genesis of ST depression on the epicardium was studied by Holland and Brooks, who concluded that the presence of endocardial ST elevation, when observed from the epicardium, appeared as epicardial ST depression. This was explained by using the solid-angle theory but without consideration of the current flow through the great vessels.

Most experimental situations remove the heart or at least remove the external conduction pathway by open-chest procedures. This importance of the low-impedance great vessels would explain why ST depression is poorly localized. It would also suggest that quantitative measures of ischemia should include leads where ST elevation is seen. On the body surface, ST depression is shown as a region whose potential is below that of the reference terminal, and unless the reference terminal is chosen at an extreme, then there must be regions on the body surface that are positive compared with the reference terminal (regions of ST elevation).

Epicardial Potential Calculations

The epicardial potentials are calculated from body surface map data by using a standard transformation

**FIGURE 2.** Epicardial maps of a patient with an acute inferior infarction. Left panel: Isopotential maps. Right panel: Current density maps. In the two sets of data, the format is identical. The six projections for each epicardium are orthogonal and drawn in perspective. As a consequence, the contour spacing is nonlinear especially at the edges of each projection. To help orient the plots, a set of coronary arteries has been superimposed on each epicardium. The left anterior descending coronary artery is seen on the right of the anterior projection and on the left of the lateral projection. The circumflex coronary artery and its branches run down the right border of the lateral projection and in the center right of the posterior projection. The superior plane is oriented with anterior toward the top of the page, as though one were looking down at the heart from above. The inferior surface is viewed from below the heart looking up; the posterior portion is uppermost, and the left side points to the left. The positions of the coronary arteries in the inferior view are arbitrary and represent a balanced coronary distribution. P represents the pulmonary artery and A the aorta. The heavy line represents a zero potential line; continuous lines, positive contours; and dashed lines, negative contours. The contour interval is given for each set of data.
derived from a resistive network model of the thorax of a 40-year-old male. This inverse transformation requires further validation; however, it produces sensible results even when used with a single body model. Other workers also have inverse solutions that appear stable, but so far none have looked at acute infarction. The ST elevation seen over the great vessels was present in most cases and suggests that future models of current flow in the thorax should include the great vessels.

In the thorax, the heart is the only current generator. This means that current flowing out of a region of the heart must also flow back into the heart. As a consequence, the total current density, over every heart surface, is zero, and current density distributions on the heart are independent of the reference terminal. We have used current density transforms in this paper to overcome any uncertainties in the relativity of the potentials arising from the use of the Wilson central terminal.

Clinical Significance

The obvious clinical significance of the selective current path is in the interpretation of ST depression. Many workers have shown that, although body surface ST elevation was highly related to the region of ischemia, body surface ST depression was poorly related if at all. Using precordial body surface map patterns on exercise, Fox et al. showed some general relation of ST change to position especially in single-vessel disease. Montague and colleagues were able to differentiate single-vessel disease through subtle changes to the map pattern that were statistically but not positionally related to the ischemic region. If the current from endocardial ST elevation was flowing out of the great vessels back to the epicardium, then most of the epicardium could look negative relative to the superior aspect of the heart.

We conclude that the great vessels form an important low-resistance current path in the human intact thorax that must be considered when examining the genesis of ECGs. This is particularly important with endocardial events and suggests that ischemia may be better monitored by sampling from leads representing the great vessels.

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FIGURE 3. Average map patterns of the groups of patients with ST depression on the 12-lead electrocardiogram. Each map was grouped into one of seven groups by using the algorithm described in the text, and the average map for each group was calculated. The format is identical with that in Figure 1.

FIGURE 4. Top panel: Twelve-lead electrocardiogram of a patient with ST depression. Bottom panel: Surface map of the same patient. The map contour is the same as in Figure 1.
Anne Duffield, Wendy Herbert, and Peter Lavercombe and the staff of the ICU, Royal Hobart Hospital.

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