A validation of derived epicardial potential distributions by prediction of the coronary artery involved in acute myocardial infarction in humans

David Kilpatrick, M.D., and Stephen J. Walker, Ph.D.

ABSTRACT We have developed computer algorithms that enable epicardial potential distributions to be calculated from electrocardiographic body surface data. To validate this inverse transformation we obtained body surface maps during the ST segment in 55 patients with acute infarction who subsequently underwent coronary arteriography and we constructed epicardial ST segment potential distributions for each patient. From the unlabeled epicardial maps one of us predicted the coronary artery that would be found to be involved in the infarction. These predictions were compared with the results of coronary arteriography and this showed that the analysis of the epicardial map correctly predicted the coronary artery involved in 40 of 55 patients (72.7%). In another eight patients the anatomy was partially predicted. In the 15 patients in whom the prediction was incorrect or partially correct (27.3%), 11 had critical disease or occlusions of the predicted coronary artery but the infarct-related artery was incorrectly identified. This verifies that sensible epicardial potential maps can be calculated from body surface electrocardiographic data, and that these data are sufficiently accurate to predict the vessel involved in acute infarction.


THE NEED for a noninvasive measure of size and position of myocardial infarction has been well recognized.1 2 A technique that would meet some of the criteria for clinical use would be to examine the epicardial potentials during the ST segment in the patients with acute infarction. Clearly it is not possible to do this directly, but a method based on a mathematical transformation of body surface electrocardiographic data back onto the heart surface might provide equivalent data.

We have developed a system of electrocardiographic body surface mapping designed to rapidly acquire electrocardiographic data in patients with acute myocardial infarction.3 In addition, an algorithm has been developed that enables epicardial potential distributions to be calculated from the measured body surface data4–6 by use of detailed models of the torso based on geometric data obtained from computed tomographic scans. With these models, relationships between epicardial potentials and body surface potentials are derived. These relationships are inverted by mathematical regularization techniques. Various torso models can be used.

It is difficult to validate directly the epicardial potentials that are calculated. This would require simultaneous measurement of body surface and epicardial potentials in an intact human torso. A study of this type has been done in dogs,7 but there are obvious ethical and practical difficulties with undertaking this kind of study in a human subject. To test the validity of our calculated epicardial potential distributions, we have examined data from a series of patients with acute myocardial infarction who have also undergone coronary arteriography. From the calculated epicardial potential distributions during the ST segment, we have predicted the vessel involved in the infarction. These predictions have then been compared with the arteriographic data.

Methods

Patient selection. Patients with acute myocardial infarction who had subsequent cardiac catheterization before any other cardiac event and who had body surface electrocardiographic data recorded during the acute infarction were selected for study. Patients were excluded if their body surface ST segment maps

From the Department of Medicine, University of Tasmania, Hobart, Tasmania, Australia.

Funded by the Australian National Health and Medical Research Council, the Clive and Vera Ramaciotti Foundation, the National Heart Foundation of Australia, and the Royal Hobart Hospital Research Trust.

Address for correspondence: Dr. David Kilpatrick, Department of Medicine, University of Tasmania, 43 Collins St., Hobart, Tasmania 7000, Australia.

Received April 21, 1987; revision accepted Aug. 6, 1987.
DIAGNOSTIC METHODS–CORONARY ARTERY DISEASE

The body surface mapping technique has been previously described. In brief, a jacket holding an array of 51 high-input impedance active electrodes is positioned around the body with a concentration of electrodes over the left chest. The electrodes are more densely spaced over the anterior and left chest, being positioned at the junctions of a 7.5 cm × 7.5 cm grid and more sparsely spaced over the remaining chest (a 15 cm × 7.5 cm grid). The positions of the electrodes are calculated from a measurement of the overlap of the jacket as it wraps around the chest. The second column of electrodes is always placed over the sternum. In patients with a small chest circumference the last column of electrodes may overlap the first column and thus not be in contact with the patient. In the largest patients there may be a gap between the last column and the first column of electrodes, but in practice this has never exceeded the spacing of the electrodes over the back and right chest (15 cm). The signals from the electrodes are amplified and simultaneously digitized at a sampling rate of 1 kHz. The data are checked on a graphics computer terminal and bad leads are interpolated from surrounding leads. The QRS onset is automatically picked and manually checked. Isopotential and isointegral contour maps can then be plotted. The data are stored on disk for future use. The total time from presentation of the patient to completion of data processing including the construction of an epicardial map can be as short as 5 min. Additional epicardial maps take approximately 1 min each to construct and output on the terminal and printer.

The body surface ST segment maps used in this study were each constructed from data averaged over a 20 msec interval centered on a point 140 msec after the QRS onset. The ST segment potentials are often quite low level, and averaging over a 20 msec interval reduces noise (in particular 50 Hz mains interference). The ST segment potential distributions are displayed as isopotential contour maps on a rectangle representing the thoracic surface to show both the front and back of the patient. The reference electrode used to construct the maps is a simulated Wilson central terminal calculated from the raw map data. For each electrode position an interpolated value is derived from the leads most closely representative of the two arms and the left leg and the central lead is calculated in the conventional manner.

The inverse transformation. A resistor network model of the human thorax was constructed from computerized tomographic scans of a 40-year-old male subject. Computed tomographic scan slices were digitized with a spacing of slices from the superior thorax of 2 cm and from the region including the heart of 1 cm. The lowest slice was repeated several times at intervals of 2 cm to give an overall torso height of 45 cm. This slice was inferior to the lungs and was homogeneous except for the spine. No interpolation between slices was used.

Each slice was divided into a grid of 7.5 mm in the neighborhood of the heart, increasing to 1 cm and then 1.5 cm further from the heart. In each slice the lungs, the spine, the sternum and the heart were assigned a tissue resistivity obtained from published data (lungs 2100 Ωcm, heart 380 Ωcm, bone non-conductive, other tissue regions 460 Ωcm). The ribs and great vessels were not included in this model. The resulting model had 18,924 delineated cells or nodes at which potential calculations were performed, 4602 on the body surface and 736 on the epicardial surface. A typical slice is illustrated in figure 1, which shows the distribution of the grid at the level of the heart.

No attempt was made to model the anisotropic skeletal muscle layer. Although this is possible with this type of resistor network model, the data on muscle fiber distribution and orientation were not easily available and were not easily incorporated into the model. The epicardial potentials were calculated on a closed surface defined by the external borders of the cardiac silhouette on the computed tomographic scan. This surface surrounds all chambers and passes through the great vessels at the superior border of the heart. Intracardiac blood masses were not modelled because inverse calculations of potentials on a closed surface surrounding the heart are not affected by the internal structure of the heart.

The model simulates, on a nonuniform rectangular grid, a three-dimensional resistor network. The path between two nodes is treated as a resistance element and Ohm's and Kirchhoff's laws are used to express the potential at any node in terms of the potentials at neighboring nodes. These simultaneous equations are solved by a successive overrelaxation method. The iteration is stopped when the maximum change in any variable becomes less than 10−9.

With the model we used, the epicardial potential distribution h and the torso surface potential distribution b were related by the matrix equation: Th = b, where T is a matrix that reflects the geometry of the torso. To construct the matrix T, the epicardial nodes were first grouped to form 50 source regions of approximately equal size that covered the epicardium, and the potential was assumed to be equal at all of the nodes within any one source region. The 50 regions on the heart surface were chosen for this study from previous work. To develop a series of transfer coefficients relating a potential on the epicardial surface to the body surface each epicardial region was in turn considered to have a potential of unity compared with all others with a potential of zero and a series of simultaneous equations was set up from the resistor network that when solved gave the body surface potentials. In this way a vector relating each epicardial region to the body surface was derived. These vectors can be combined together to create the matrix T relating the 50 epicardial regions to the body surface potential distribution. For the purposes of creating T, the body surface distribution was described by potentials at 160 body surface nodes, spaced evenly in 10 rows of 16 nodes.

A regularization method was used to solve the equation Th = b for h in terms of T and b. The solution obtained is given by the equation: h = (T † + αI)−1Tb, where I is the appropriately sized identity matrix and α 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 $\alpha$, but they require assumptions to be made about the nature of the body surface noise and the epicardial potentials.

Calculation of the forward transfer matrix $T$ took approximately 36 hr on a PDP11-44 computer. Calculation of the inverse matrix took approximately 30 min. However, these calculations need only be done once for each model torso. Any set of body surface data need only be multiplied by the inverse matrix to produce an epicardial potential distribution, a process taking less than 1 min. A spline interpolation technique was used to obtain the 160 body surface node potentials from the raw data measured at 51 electrodes.

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 are anterior, left lateral, posterior, right lateral, inferior, and superior to the heart and approximately 2 m distant from it. The views are generated from the torso model, triangulating the surface formed by the 50 epicardial source regions. The views thus correspond to perspective views of the surface defined by the external borders of the cardiac silhouette on the computed tomographic scan. The triangulation is shown in figure 2. In this figure the triangles can be seen to be compressed at the borders of the views where the curvature of the heart reduces the angle of incidence of the region to the eye.

In the results, the positions of the coronary arteries have been superimposed over the six projections. The positive region has been delineated by shading all the territory above the zero contour line. The solid lines are positive isopotential contours, the dotted line is the zero contour, and the dashed lines are negative isopotential contours. The boundary of the epicardial surface is also shown in each view.

Analysis of epicardial distributions. Each epicardial distribution was coded by one of the authors (S. J. W.) so that the reader (D. K.) did not know the identity of any of the map sets. Each map set was examined and a prediction was made as to which vessel would be found to be occluded. To predict the artery involved, the standard coronary artery anatomy was plotted on a diagram with the same format as the epicardial maps. This anatomic plot was superimposed on the epicardial map of each patient and the artery supplying the territory where there was ST elevation was selected as being involved in the infarction. The size and position of the region of ST elevation influenced the decision as to which branch was involved or the choice between a dominant right or circumflex coronary artery. In those sets of data in which the region involved could be supplied by either of two branches of a coronary artery, e.g., right or left dominant systems, then two occluded arteries were identified. For each double choice an explanation was provided to justify the alternative choice. The range of possible vessels that were considered was: left anterior descending, first diagonal, second diagonal, circumflex, first obtuse marginal, second obtuse marginal, third obtuse marginal, right coronary, and posterior descending arteries (usually assumed to be right coronary artery in origin).

Coronary arteriography. Coronary arteriography was performed from the right femoral artery by conventional techniques. Each angiogram was reviewed by one of the authors (D. K.) and categorized by the distribution and the size of vessels and the percent stenosis of each vessel based on diameter measurement. The infarct-related vessel was identified in each case. The vessel dominance of each system was also noted.

Assessment of accuracy of prediction. Each prediction made from the epicardial potential data was compared with the corresponding arteriogram and was classified as either correct or incorrect. The correlation was reduced to a consideration of which of three vessels (left anterior descending, left circumflex, or right coronary artery) was involved because of the variability in the anatomy in each patient. Predictions were classified as partially correct if they were satisfactory on the basis of the normal coronary anatomy but the patient had an abnormal coronary artery distribution such that the actual predicted artery was not present or was vestigial. Additional analysis of those patients with either multiple stenoses or occlusions in the predicted territory was performed.

Results

The quality of the data obtained is illustrated in figure 3, which represents the worst data set obtained in any of the 55 patients both with respect to baseline noise and number of bad leads. This patient was a small woman in whom the last column of electrodes was overlapping and hence not in contact with the patient.

The total of 55 patients had a mean age of 58 years; the median time from the onset of pain to mapping was 22 hr, and the mean maximum level of creatine kinase was 1503 ± 1170 (normal < 200).

There were 26 patients with right coronary artery occlusion, 17 patients with left anterior descending
coronary artery occlusion, and 12 patients with circumflex occlusion, as determined from the arteriographic data.

Analysis of the epicardial distributions correctly predicted the coronary artery involved in 40 of 55 patients (72.7%). Among these 40 patients, 35 had infarct-related arteries correctly predicted as a first choice and five had infarct-related arteries correctly predicted as a second choice. In another eight patients, the predictions were partially correct; they were incorrect in seven patients.

Further analysis was performed of the 15 patients in whom the predictions were either partially correct or incorrect. Eleven of these 15 patients had critical disease or occlusions in the predicted coronary artery. Although the infarct-related vessel was thought to be a different vessel, the region of ST elevation appeared to be in the territory supplied by the predicted vessel.

A typical epicardial surface map is shown in figure 4. The ST elevation is over the region of the left anterior descending coronary artery extending over the first diagonal branch. The corresponding angiogram from this patient is shown in figure 5; a tight stenosis in the proximal left anterior descending coronary artery is evident after the origin of a large diagonal vessel. The body surface map and conventionally recorded 12-lead electrocardiogram from the same patient are shown in figure 6.
FIGURE 5. The coronary arteriogram of the patient whose epicardial potentials appear in figure 4. This is a right anterior oblique view and shows a severe stenosis of the mid left anterior descending coronary artery after the origin of a large diagonal.

FIGURE 6. The 12-lead electrocardiogram and the ST segment body surface map for the patient in figures 4 and 5. The body surface map is constructed by the same convention for contour lines as on the epicardial maps (positive contours continuous, negative contours dashed, and the zero contour dotted). The electrocardiogram shows only 0.5 cm grid lines and was recorded at standard sensitivity (1 mV/cm).

FIGURE 7. ST segment epicardial potentials for a patient with right coronary artery occlusion. The format is as described in figure 1. ST segment elevation can be seen on the inferior surface of the heart.

A typical epicardial distribution from a patient with a right coronary artery occlusion is shown in figure 7. The angiographic appearance is shown in figure 8 (left anterior oblique projection) and the body surface map

FIGURE 8. The coronary arteriogram of the patient whose epicardial potentials appear in figure 7. This is a left anterior oblique projection of the right coronary artery, showing an occlusion in the midvessel.
FIGURE 9. The 12-lead electrocardiogram and the body surface map for the patient whose data are displayed in figures 7 and 8. The format is as in figure 6.

and 12-lead electrocardiogram are shown in figure 9.

Data corresponding to an incorrect prediction are illustrated in figure 10. The epicardial map shows ST elevation posteriorly, which was predicted to be in the territory of the circumflex artery. The angiogram (right anterior oblique view of left coronary artery) is shown in figure 11 and shows marked proximal circumflex disease, which was in addition to a proximal occlusion of the right coronary artery (not shown). The body surface map and conventionally recorded 12-lead electrocardiogram from this patient are shown in figure 12.

Discussion

The good correspondence between the artery involved in the infarction as predicted from the epicardial potential distributions and that determined from arteriographic data demonstrates that sensible epicardial distributions are obtained by the inverse transformation. This is an important finding, because it is difficult to validate methods of calculating epicardial potentials by direct measurement.

We did not relate the epicardial potential distribution to infarct size but rather to position of infarction. The number of regions produced by the inverse transform (50) was greater than that produced by other diagnostic techniques, such as left ventricular angiography, and encouraged an initial prediction that included the branch arteries. An analysis of these predictions proved impossible because of the wide variation in anatomy in each patient so that to score against the coronary arteriogram it was necessary to reduce the predictions down to the three main arteries. This does not indicate that the transform does not have the degree of accuracy required to predict the involvement of various branches but more that it is extremely difficult to test that accuracy in the presence of variable arterial anatomy. Further validation against radionuclide methods or nuclear magnetic resonance imaging may provide sufficient resolution to assess the accuracy of the present transform.

The errors in prediction frequently involved patients with severe multivessel disease in whom chronic occlusion of one artery could have disrupted collateral flow via that artery to a previously critically diseased vessel. In two patients, there was no way to explain the discrepancy between the epicardial maps and the angiograms on this basis. In these patients it is possible that the infarction was occurring in a territory without a critically stenosed vessel, following coronary artery spasm for example.
Coronary arteriography is not perfect in predicting the occluded vessel in all patients with acute infarction and in some patients in this series considerable doubt existed, especially in those who had non-Q wave infarction. Left ventricular angiograms showing the regions of akinesis were considered in order to improve the coronary arteriographic predictions in these patients. Allowing for this lack of precision in coronary arteriographic data, the results are surprisingly good. Although the correlation is based on a rather subjective assessment of the epicardial potential distributions, the technique is working well enough to be used for anterograde prediction of the vessel involved in an infarct.

The point of the validation was not to compare the accuracy of an epicardial map against either the 12-lead electrocardiogram or the body surface map but to show that the transform produces reasonably accurate results. The inverse transform and epicardial surface perspective display allow a direct comparison of the position of ST elevation with the anatomy of the coronary arteries, unlike the 12-lead electrocardiogram or body surface maps. To perform a similar study using either of the latter two methods would require a learning set of patients in which criteria for prediction would be derived, and a testing set to validate the empirically derived criteria. For this reason we have only examined the epicardial maps since they offer the only electrocardiographic method of direct prediction. It is likely at least as high a level of accuracy could be obtained with the use of body surface maps and multivariate analysis, since the original patient data are the same.

The inverse transformation used in this study is based on the computed tomographic scan of a single patient. All patient data were analyzed with the use of the same transformation, with no attempt to match patients for size or sex. This is not an ideal situation, because our studies with multiple torso geometries have shown that torso geometry affects the epicardial potentials to a significant extent. The use of a library of inverse matrices that could be matched with the torso geometry of each patient should further improve the accuracy of this method.

In this work we have used a degree of regularization that is suitable for the potentials during the QRS of the electrocardiogram but is not optimized for the ST segment, but the qualitative accuracy achieved appears satisfactory to predict the region of ST elevation. Further discussion of the choice of regularization parameter has been included in other publications.

The advantages of this system are that it is noninvasive, rapid, and repeatable. A complete validation of such a technique may require patients with implanted epicardial electrodes undergoing simultaneous epicardial and body surface mapping. Such a study has been

FIGURE 11. The coronary arteriogram of the patient whose epicardial potentials appear in figure 10. This is a left anterior oblique projection of the left coronary artery, showing a tight narrowing of the circumflex coronary artery. The right coronary artery was occluded (not shown in this figure).

FIGURE 12. The 12-lead electrocardiogram and the body surface map for the patient whose data are displayed in figures 10 and 11. The format is as in figure 6.
performed previously in dogs, and showed that there was agreement with respect to major features but not in detail between the measured and calculated epicardial potentials. However, the transform used by these authors assumed a homogeneous torso, which is less accurate than the torso model used in our work. The present transformations can be further improved by matching inverse matrixes and patients for body habitus, sex, and data from a chest x-ray.

Although further validation is needed, these results are sufficiently encouraging to suggest that epicardial transformation of body surface potentials in humans is possible and already produces reasonably accurate results during the ST segment in patients with acute infarction.

We thank Anne Duffield as well as the staff of the ICU, Royal Hobart Hospital, for assistance in performing mapping, and David Lees for his excellent preparation of the figures.

References
A validation of derived epicardial potential distributions by prediction of the coronary artery involved in acute myocardial infarction in humans.

D Kilpatrick and S J Walker

Circulation. 1987;76:1282-1289
doi: 10.1161/01.CIR.76.6.1282

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1987 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/76/6/1282