Letter to the Editor

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In their recent paper, Kilpatrick et al have used measured body surface potentials to calculate epicardial potentials by using an inverse solution. Particularly interesting, and apparently novel, is their use of their model to determine normal current density at the epicardial surface. A somewhat unanticipated finding is the presence of current leaving the heart superiorly in the case of inferior injury.

Figure 1 of their paper shows a body surface isopotential map (BSPM) of a patient with an inferior infarction. This map is rather “dipolar” in appearance. Potentials are predominantly negative over the top half of the thorax (superiorly) and positive over the bottom half (inferiorly). Calculated isopotential epicardial maps for the same patient shown in Figure 2 depict two regions of highly positive potential: one on the superior aspect and one on the inferior aspect of the heart. A current density map exhibits similarly located regions of positive (outward) current. The authors postulate that the conducting pathway provided by the great vessels is responsible for the positive potential and current maxima on the upper surface of the heart, but they provide no further justification for their hypothesis.

Note that the inverse solution for potential or current density on the epicardial surface involves only the body surface potentials and the nature of the volume conductor outside the heart. On the other hand, an explanation of epicardial currents involves the nature of cardiac sources and the volume conductor properties of the heart and torso.

Two questions arise in our minds. Is the inverse solution plausible? Is the explanation for the epicardial currents plausible?

From a consideration of currents in a volume conductor it is hard to see how current leaving the heart superiorly as shown in the figure will fail to produce a relative potential maximum somewhere on the superior body surface. Other investigators have shown a general similarity between body surface and epicardial potential distributions, a finding at variance with the one presented by Kilpatrick et al. Extensive examples of measured canine BSPMs and simultaneous epicardial recordings are given by Barr and Spach.1 These maps all indicate that a region of positive heart potentials can be correlated in space to a region of thorax with positive potential. A similar result is given by Rudy and Messinger-Rapport2 in their studies using a heart suspended in an electrolytic tank for both measured and inversely computed epicardial potentials. These authors go on to say that “the general features of the measured epicardial potentials are preserved” on the calculated body surface maps. In earlier work, Walker and Kilpatrick3 showed a measured BSPM, a series of computed epicardial maps, and a series of BSPMs computed from the computed epicardial potential maps. The quite diverse epicardial maps all gave very similar body surface maps, with the correlation coefficients greater than 0.99 for four of the five examples presented. This result demonstrates the great caution necessary in interpreting epicardial maps calculated from inverse procedures.

Let us now examine possible explanations for positive potentials and current density on the superior surface of the heart. Potential maxima on the superior and inferior surfaces of the heart could result from the simultaneous presence of forces directed superiorly and inferiorly. Unless the superiorly directed forces are quite weak, however, one would expect corresponding maxima on the body surface. If only inferiorly directed forces are present, as would seem to be consistent with an inferior injury, and as argued in the paper, then superior outward current would have to result from the nature of the volume conductor. The authors postulate that a highly conducting blood mass in the great vessels is responsible but offer little justification. It is not clear how the great vessels can possibly distort the field as proposed. We would have preferred to see some biophysical argument for how a source for inferior injury can result in upward currents with the proposed conducting pathway.

It is our feeling that the conjecture of an erroneous inverse solution is at least as likely as the conjecture of the role of the great vessels. The burden of proof is on the authors of the paper.

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References


Reply to the Preceding Letter

We can appreciate the concerns mentioned by the authors of this letter. We had the same concerns as the authors of the letter when we made the initial observations regarding the inverse solution. However, there is a relative potential maximum on the superior body surface. The maximum negative potentials in patients with inferior infarction usually occur on the upper chest. Toward the neck and posteriorly in the superior regions there is a definite relative positivity. We can find no reason to suspect that the general features of the inverse transformation are incorrect although direct measurement is clearly required for complete validation.

Our paper presented the hypothesis that this superior potential represented current flow out of the cavity of the ventricle. This hypothesis is not new. Since writing the original article we have become aware of two papers from the 1950s in which this was clearly the mechanism postulated for the occurrence of ST depression.1,2

This should undoubtedly remain as a hypothesis, but we believe that it is a sensible hypothesis that at present fits the available data and aids in the understanding of the electrocardiogram.

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Comments on "Importance of the great vessels in the genesis of the electrocardiogram"
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