The term forward problem of electrocardiography is used to denote the computation of the electric potential field generated by cardiac excitation within and on the torso surface. In this class of problems that have been studied for several decades, one starts from a model of the heart electric sources and computes the potential field within the torso volume conductor, which constitutes a solution to Laplace’s equation. Of particular interest are body surface potentials, which provide the data for clinical electrocardiography, and potentials over the epicardium, which may mirror electric events within the heart that cannot be observed in the body surface potential patterns. Indeed, simultaneous measurements with epicardial and intramural electrodes in closed-chest animals demonstrated that epicardial potentials contain a considerable amount of information about the underlying intramural electrophysiological events. A theoretical study concluded similarly that “epicardial potential maps accurately reflect the underlying source configuration and are free of the effects of body shape and size.” Several models of (equivalent) sources have been chosen to represent the electric activity of the heart, including a single (stationary or moving) dipole and multiple dipoles. Epicardial potentials have also been used as the source and starting point for computing potentials within and on the torso surface. Work on the forward problem has been extensive and conducted over many years; a comprehensive account can be found in the excellent review by Gulrajani et al.

A consistent finding of these studies is the major smoothing effect that the volume conductor exerts on the BSPM, which is of low resolution compared with the corresponding epicardial potential pattern. BSPM patterns are less sensitive to the details of activation in regions of the heart that are remote from the body surface; as a rule of thumb: the BSPM can resolve multiple epicardial potential extrema (maxima or minima) only if their separation on the epicardial surface equals or exceeds their distance to the torso surface. Importantly, the physical properties of electric fields are such that each body surface electrode (in the conventional ECG or in BSPM) records electric activity from the entire heart. Therefore, geometric relationships between regions of electric activity in the heart are not preserved in the body surface ECG and BSPM, and these methods cannot, in principle, provide information on local and regional cardiac electric activity. Another observation that is shared by many of the studies is that the inhomogeneities, when compared with a homogeneous model of a torso with the same geometry, affect the magnitudes of body surface potentials, but have only a minor effect on the BSPM potential patterns. This property is of clinical importance because most ECG diagnostic criteria rely on the morphology of electrocardiograms, rather than on their absolute voltage amplitude.

In this issue of Circulation Arrhythmia and Electrophysiology, Bear et al evaluate the accuracy of different forward models, with emphasis on effects of the inhomogeneous torso volume conductor. Similar to the earlier study by Ramsey et al, body surface and epicardial potentials were recorded simultaneously in a closed-chest animal (pig in the Bear study; dog in the study of Ramsey et al). Body surface potentials were then computed from the measured epicardial potentials in animal-specific heart–torso geometry. The torso volume conductor was assumed homogeneous in the Ramsey study, whereas Bear simulated both homogeneous and inhomogeneous torsos. Comparison with the measured body surface potentials was used to evaluate the accuracy of the forward model computation. In addition to qualitative comparison of BSPM patterns, correlation coefficients and root-mean-square errors were used to obtain quantitative measures of similarity. Correlation coefficient is a measure of similarities between potential patterns and ECG waveforms, whereas root-mean-square error also reflects amplitude differences. Ramsey concluded that “simulated body surface distributions consistently had a high correlation with the measured body distributions. However, the simulated maps usually had larger peak to peak magnitudes. A favorable result of the simulation was that the discrepancies between the simulated and measured potentials were associated primarily with the voltage reference and the peak to peak magnitudes of the simulated maps, rather than with differences in the contour patterns. Because physiological evaluation of the maps has been based primarily on the contour patterns, the simulated maps correctly represented the

Editorial

The Forward Problem of Electrocardiography Revisited

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The opinions expressed in this article are not necessarily those of the editors or of the American Heart Association.

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features usually used for map interpretation.” Moreover, the potential magnitude difference was greatly reduced by eliminating the reference offset and simple scaling of the maps. These results are consistent with the results of the Bear study, where amplitudes differed substantially (corrections for reference offset were not attempted) but “correlation coefficients between simulated and measured BSPMs were high (≈0.9) for most of the activation sequence.” This recent study used additional measures for comparing the simulated and recorded BSPMs, namely the distance between potential extrema and their relative orientation. There were differences in these measures; a careful inspection of the BSPMs in Ramsey et al shows similar differences between their simulated and measured maps as well (although they considered these differences in regions of low potential gradients to be of little significance). Inclusion of the torso inhomogeneities in Bear simulations reduced, but did not remove these differences. Given the low resolution and smoothing effect of body surface potentials and the loss of geometric relationships between cardiac electric sources in the BSPM, it is unclear whether these differences are meaningful to the interpretation of the BSPM in terms of underlying electrophysiological processes in the heart. The presence of noise in the recorded maps and inaccuracy of the closed epicardial and body surface geometries and potentials that are interpolated from a limited set of measurements could be the source of these differences.

The ultimate objective of electrocardiography is noninvasive determination of electrophysiological events in the heart from body surface potential measurements. In a broad sense, this is the definition of the inverse problem of electrocardiography. Similar to the forward problem, the inverse problem can be defined in terms of various equivalent cardiac sources, including epicardial potentials. The forward problem is mathematically well-posed, meaning that there is continuous dependence of the solution on the data. In contrast, the inverse problem is ill-posed in the sense that small errors in the data (measurement noise, geometry errors, and inaccurate conductivity values) can cause large unbounded errors in the solution. This necessitates the use of regularization techniques that impose physiologically based constraints or iterative schemes to stabilize the solution in the presence of these inaccuracies that are always present in the experimental and clinical environments.

Regularized and iterative inverse solutions have provided the theoretical basis for electrocardiographic imaging (ECGI; also called electrocardiographic mapping), which combines BSPM with noninvasive information about the heart–torso geometry to reconstruct noninvasively potentials, electrograms, activation sequences (isochrones), and repolarization patterns on the epicardial surfaces of the heart. The results of Bear demonstrate a limited effect of the torso inhomogeneities, relative to a homogeneous torso, on the patterns of body surface potentials in forward problem simulations. Naturally, this raises the question whether it is important to include the torso inhomogeneities in ECGI applications. This requires accurate geometric determination and segmentation of patient-specific inhomogeneities and precise determination of their conductivities, accomplished noninvasively. The conductivities can vary substantially between patients and in pathology (eg, high lung conductivity in pulmonary edema and low in cystic fibrosis; low skeletal muscle conductivity in glycogen storage disease). A noninvasive determination of these conductivities in a given patient cannot be achieved in clinical practice. Clearly, treating the torso volume conductor as electrically homogeneous (the homogeneous approximation) simplifies greatly and facilitates clinical application of ECGI. An early analytic study in the eccentric spheres model of the heart–torso system demonstrated loss of resolution of potential distributions on the posterior heart surface in inverse computations that assumed a homogeneous torso. The inverse-reconstructed potential distributions were also sensitive to changes in the conductivities of lung and skeletal muscle. However, these simulations were conducted in the absence of added measurement noise and without any form of regularization. A later study used measured epicardial potentials from a canine heart and an anatomically accurate model of the homogeneous human torso (male and female) to investigate this question. This time, measurement noise, and error in determining body surface electrode positions were incorporated, and regularization was applied. For these realistic conditions, epicardial potential patterns, electrograms, isochrones, and locations of pacing sites were reconstructed with comparable accuracy when torso inhomogeneities were not included in the calculation, supporting the use of the homogeneous approximation in clinical application of ECGI. Absolute potential amplitudes were less accurate, but as stated earlier potential patterns, electrogram morphologies, and activation isochrones usually provide the basis for physiological and clinical interpretation of epicardial (and endocardial) maps. In cases where potential magnitudes are of interest (eg, low voltages within a myocardial scar), normalized values (indexed to the maximum value in the same heart) rather than absolute values should be used. ECGI studies in patients under the homogeneous approximation confirm adequate accuracy of this approach in clinical applications; examples include determination of pacing sites in various locations around the atria with accuracy of 6.3±3.9 mm and mapping of atrial fibrillation and other atrial arrhythmias before ablation, determination of ventricular pre-excitation sites in Wolff–Parkinson–White patients undergoing ablation, mapping of focal and reentrant ventricular tachycardia mapping of substrate post myocardial infarction and in hereditary channelopathies (long QT and Brugada syndromes), and determination of the area of latest activation to guide electrode placement for cardiac resynchronization therapy in patients with heart failure. The experimental preparation developed by Bear et al provides a well-controlled framework for evaluating many more potential experimental and clinical applications of ECGI.

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