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The Electrophysiological Basis of QT Dispersion: Global or Local Repolarization?

To the Editor:

Kors et al1 present a very interesting article on the genesis of QT dispersion (QTD) in the 12-lead surface ECG. The authors provide support for the hypothesis that differences in the QT interval on the surface ECG are due to different projections of a common T-wave vector. Although the article is an important methodological contribution and it fuels a necessary rethinking of the clinical and diagnostic value of QTD, we would like to caution against a mechanistic view of its main conclusion.

Many arguments may still be made for the widely appreciated “local” hypothesis of QTD genesis, which we supported after reporting a significant correlation between local myocardial measurements and the surface ECG.2,3 Although it is notoriously difficult, if not impossible, to explain local myocardial repolarization by means of surface ECG recordings (the unsolved “inverse” problem of electrocardiography), a common T-wave vector may reflect some simplification of the electric forces during ventricular repolarization. The vectorcardiographic calculation itself presumes that the T wave can be explained by an electric dipole and that it will average existing local differences in repolarization forces.

Importantly, by using the technique of body surface potential mapping, a large number of elaborate studies spanning decades have proven that the nature of repolarization has nondipolar contents as well (recently reviewed by Taccardi et al4). Under certain arrhythmogenic clinical situations, it may be exactly this local deviation from dipolarity that may reveal the arrhythmogenic substrate. In addition, viewing repolarization as a global electric dipole also neglects the contribution of any transmural repolarization. The vectorcardiographic calculation itself presumes that the T wave can be explained by an electric dipole and that it will average existing local differences in repolarization forces.

Because the truth may lie somewhere in the middle between the vector concept of QTD and an explanation by underlying local repolarization, more specific experimental and human studies are needed to ascertain the exact contribution of local and global repolarization forces to recordings on the body surface.

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Circulation 2002;105:2257

Response

We thank Drs Zabel and Franz for their kind letter, which gives us the opportunity to further clarify our thinking about the controversial and difficult problem of QT dispersion (QTD). We completely agree that not all information in the T wave or in the ECG in general can be represented by a dipolar model. However, there will always be one last electrical source when repolarization comes to an end. The field generated by this source will be captured by every single electrode on or in the body. Repolarization must, therefore, come to an end at all electrode positions simultaneously. Only when the lead electrodes have equal potential will the signal in this lead become zero before the common end of repolarization.

The closer to the source, the higher the electrode potential. This is essentially a function of the inverse of the square of the distance. Thus, an epicardial electrode will pick up huge signals from the local myocardium and much attenuated signals from remote parts. The measurement of differences in action potential duration is possible close to the heart because of the rejection of low-level signal contributions from distant sources. On the body surface, however, amplitude differences between local and distant sources are sharply diminished. Because of this blurring of local cardiac information, we cannot accurately deduce action potential duration distributions in the heart.

For our explanation of QTD in terms of low amplitude and equal electrode potentials, we made use of a single dipole model. Our experiments do not exclude multipolar sources but, in our opinion, circumstantial evidence makes it unlikely that they play an important role. Previous studies have shown that ECGs and vectorcardiograms can very well be synthesized from each other when the reconstruction is performed using a transformation per individual.1-2 This indicates the adequacy of a simple dipole model for all practical purposes. Considering the large measurement error in determining QTD,3 we think any subtleties due to nondipolar components are unlikely to be discernible at the body surface. However, further research in this respect, as suggested by Drs Zabel and Franz, is to be welcomed. The greatest challenge will be to distinguish between the measurement problem and the equality of electrode potentials, without which QTD cannot exist.

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Circulation. 2000;101:e235-e236
doi: 10.1161/01.CIR.101.25.e235
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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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:
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