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

To the Editor:

Kors et al.1 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 al.4). 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 dispersion, which was recently demonstrated to be of possible importance in the genesis of QTD and late T-wave changes.

Although we would certainly expect that part of the T wave can be explained by the vector concept and our remarks do not include the prognostic features contained within it,5 the contribution of local and global repolarization forces to the T wave and QTD on the body surface may significantly vary between clinical situations. A minor problem of the study by Kors et al.1 in this respect is the unknown importance in the genesis of QTD and late T-wave changes.

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.6-7 This indicates the inadequacy of a simple dipole model for all practical purposes. Considering the large measurement error in determining QTD,6 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.

Jan A. Kors
Gerard van Herpen
Jan H. van Bemmel
Institute of Medical Informatics
Faculty of Medicine and Health Sciences
Erasmus University

Correspondence

Letters to the Editor must not exceed 400 words in length and must be limited to three authors and five references. They should not have tables or figures and should relate solely to an article published in Circulation within the preceding 12 weeks. Authors of letters selected for publication will receive prepublication proofs, and authors of the article cited in the letter will be invited to reply. Replies must be signed by all authors listed in the original publication. Please submit three typewritten, double-spaced copies of the letter to Herbert L. Fred, MD, % the Circulation Editorial Office. Letters will not be returned.

References


The Electrophysiological Basis of QT Dispersion: Global or Local Repolarization?
Markus Zabel and Michael R. Franz

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
Copyright © 2000 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/101/25/e235

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/