JACC Vol. 14, No. 6
November 15, 1989:1537–8

Editorial Comment

QRST Area Maps and Cardiac Arrhythmias*

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Cardiac conditions favoring susceptibility to arrhythmias are fundamentally electrical and most likely to be recognized by examination of the heart's electrophysiologic properties. The only practical noninvasive examination of these properties is the body surface electrocardiogram (ECG), but the 12 lead examination has not proved useful for the prediction of arrhythmias. More extensive sampling of the ECG adds importantly to the evaluation of cardiac electrophysiologic properties in a manner relevant to arrhythmia prediction. Sufficient sampling has been convincingly shown to furnish a regionally selective examination of electrophysiologic conditions. Nonuniformity of these conditions is an important component of arrhythmia susceptibility, and its identification is likely to require regionally selective examination.

QRST area maps to detect predisposition to arrhythmias. The cardiac conditions pertinent to reentrant arrhythmias include primary nonuniform propagation of excitation and nonuniform recovery sequence due to disparate local recovery properties leading to secondary nonuniform propagation. Abnormalities of propagation will be evidenced in the distributions of QRS parameters in serial isopotential maps and maps of the QRS area. Abnormalities of recovery sequence will be evidenced in serial isopotential maps of the ST-T deflection and of the area of that deflection. Nonuniformity of local recovery properties has special relevance to arrhythmia prediction because it has been experimentally documented to affect vulnerability (1) and is the determinant of the QRST deflection area (2). The combination of QRST area and selective regional sensitivity provided by ECG mapping is a theoretically sound approach to the recognition of local inequalities of repolarization that predispose the ventricles to arrhythmias.

The present study by Tsunakawa et al. (3) in this issue of the Journal adds to previous evidence that QRST area maps are useful for the recognition of arrhythmia vulnerability. It also contributes to the definition of appropriate methods of analysis and of clinical conditions in which maps are useful for evaluating vulnerability.

Analysis of QRST area maps. In this study (3) nondipolarity was estimated with use of a dipole model. The dipole that best accounted for the measured body surface distribution of QRST areas was calculated and the residue was taken as the nondipolar component. This approach is related, but not identical, to that of prior studies in which the nondipolar content of QRST area maps was determined on the basis of eigenvector analysis (4). That analysis was not based on a physiologic model, and relation to disease or physiologic states such as that of nondipolar content to arrhythmia vulnerability is purely empiric. The analysis used in the present study is based on a physiologic model that assumes a dipole characterization of the heart. At present, the optimal method for analyzing QRST and other ECG distributions for arrhythmia prediction is uncertain and both methods of determining nondipolarity deserve further study.

Clinical conditions. The condition for which the usefulness of the QRST map was tested was ventricular tachycardia in association with myocardial infarction. Nondipolarity was higher in patients with infarction than in normal subjects and higher in patients who had ventricular tachycardia ≥10 days after infarction than in other patients. These findings are similar to those of studies in which the nondipolar content evaluated by eigenvectors was higher in patients with ventricular tachycardia than in normal subjects (5) and in patients with sudden death after infarction than in other patients with infarction (6).

One especially interesting finding in the present study (3) was that nondipolarity of QRST area maps in patients with ventricular tachycardia during the first 10 days after infarction was not higher than that in patients without tachycardia. This finding may have been related to the time difference between map measurements and the occurrence of tachycardia, as the authors suggest. Alternatively, it may reflect different mechanisms of tachycardia in acute and chronic phases of myocardial infarction.

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0735-1097/89/$3.50

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