QRST Nondipolar Content Revisited

Mark Potse,[a,b]   Pascal F.H.M. van Dessel,[c]   André C. Linnenbank[b,d]
[a]Institut de Génie Biomédical, Université de Montréal, Québec, Canada
[b]Academic Medical Center, University of Amsterdam, The Netherlands
[c]Krannert Institute of Cardiology, Indianapolis, Indiana, USA
[d]Heart Lung Center Utrecht, The Netherlands

Correspondence: Dr. M. Potse, Institut de Génie Biomédical, Université de Montréal, PO Box 6128 Station "Centre-Ville", Montréal (Québec) H3C 3J7, Canada. E-mail: mark@potse.nl, fax +1 514 343 6112.

Abstract. Nondipolar content of QRST integral maps (QRSTI) is a measure for vulnerability to arrhythmia. We demonstrate that the nondipolar component of the energy of the QRSTI is the same for dipolar and nondipolar maps. The higher nondipolar content for patients who are vulnarable to arrhythmia, compared to normal controls, is due to a lower dipolar component of their QRSTIs.

Keywords: Body Surface Mapping, Nondipolar Content, Ventricular Arrhythmia

1. Introduction

The QRST integral (QRSTI) of ECG body surface maps (BSM) is thought to reflect primary repolarization properties, i.e. the dispersion of refractory period (RP) in the heart [Wilson et al., 1934]. If the RP of all cells were the same, repolarization would follow the same path as depolarization, QRS and ST integral maps would cancel, and the QRSTI would be zero. However, an endo-to-epicardial and apex-to-base gradient in RP is usually present. This gradient is large enough to cause depolarization and repolarization to follow approximately opposite paths. Thus, the QRSTI is generally nonzero.

Sinus rhythm QRSTIs in several groups of patients prone to arrhythmia have deviant patterns. In the more severe cases, the pattern is characterized by multiple local extrema, whereas the normal pattern is dipolar. The conventional interpretation of this correlation is that increased dispersion is reflected at the body surface as an increased nondipolarity. This model predicts that the nondipolar component of the QRSTI is increased in patients while the amplitude of the QRSTI would be more or less constant. However, it has been reported that the amplitude of the QRSTI is also much smaller in nondipolar maps. In this paper we investigated the relation between the amplitude of the dipolar and nondipolar energy in QRSTIs and the nondipolarity, quantified using the "nondipolar content" (NDC) of the map [Abildskov et al.,1985].

2. Material and Methods

Body surface maps were recorded during sinus rhythm, from 17 patients suffering from primary electrical disease (PED), 78 patients with old infarctions, and 30 normal controls. For each patient a QRSTI was generated. The total energy (E_{QRST}) of the QRSTI was computed as the sum of the squared values of the map. The map was then expressed in terms of an orthogonal basis that has the special property that its first three basis vectors correspond to dipolar map patterns, and the others to nondipolar patterns [Abildskov 1985]. The dipolar energy (E_d) was defined as the sum of the squared values of the first three elements of the transformed map. The nondipolar energy (E_{nd}) was defined as the remaining energy and the nondipolar content (NDC) as $E_{nd}/(E_d + E_{nd})$. Thus, NDC will increase when either the nondipolar energy increases or the dipolar energy decreases.

In order to judge whether a relation between NDC and the two energies E_{QRST} and E_{nd} was present, we took the logarithm of all variables, observing that this produces more normally distributed variates and results in an approximately linear relationship. The coefficient of correlation between

energy and NDC was computed using the logarithms of the variates, and its significance level was estimated.

3. Results

The dependence of total energy and nondipolar energy of the QRSTI on NDC is shown in Fig 1. Total energy clearly decreases with increasing nondipolar content, while the nondipolar energy does not change: $r = -0.78$ and -0.16; $p = 2 \cdot 10^{-44}$ and 0.02, respectively.
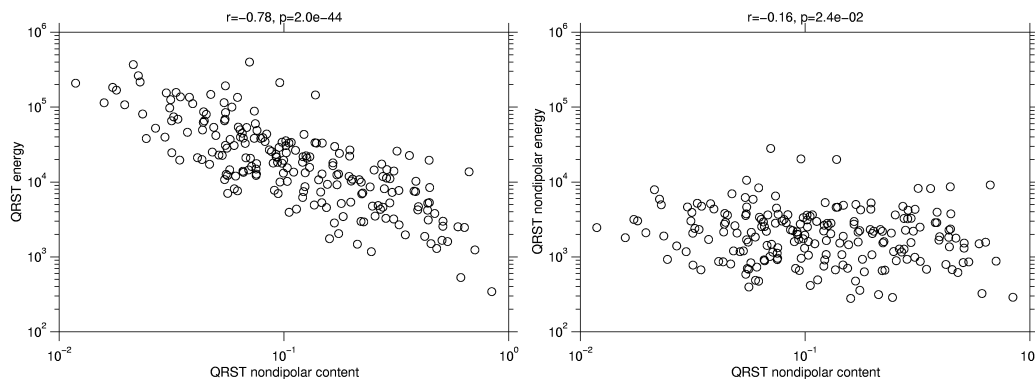

Figure 1. *Double-logarithmic scatter plots of QRSTI total energy and nondipolar energy as a function of QRSTI nondipolar content (all subjects in sinus rhythm).*

4. Discussion

From our results it is clear that large nondipolar content is not due to an increase in nondipolar energy, but rather to a decrease of the dipolar energy in the map. This implies that 1) it is not necessary to assume an increase in dispersion of primary repolarization properties to explain nondipolar QRSTI patterns; and 2) the natural global gradient in RP is diminished with increasing NDC.

Since large NDC is associated with arrhythmogenesis, either the suppression of the natural gradient in RP is arrhythmogenic, or has the same cause as the arrhythmia, or is caused by arrhythmia. The last assumption seems the most likely to us. Cardiac memory (CM) alters the RP of cardiac cells such that those that are activated later have a shorter RP. This may cause the natural RP gradient. Ectopic beats may, by the same mechanism, gradually modify the gradient and cause a smaller dipolar energy in the QRSTI, and thus a higher NDC. Thus, NDC can be correlated with arrhythmia in the past few weeks or months, and since past arrhythmia are a predictor for future arrhythmia, NDC can correlate with arrhythmogenic death, irrespective of the disease underlying the arrhythmia.

In earlier studies increased dispersion in RP proved arrhythmogenic. These results are not in conflict with ours, since we did not disprove the presence of local dispersion. We showed, however, that high NDC does not imply its presence.

The present results are in agreement with the observation [Van Dessel et al., 2001] that the standard deviation of the activation recovery interval obtained from endocardial basket catheter recordings, which is a measure of RP dispersion over the endocardial surface, correlates negatively with QRSTI nondipolar content. This is in contrast with the conventional interpretation of NDC, which predicts a positive correlation.

References

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