Letters to the Editor

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Is QT dispersion a reliable index of heterogeneity of ventricular repolarization and a pro-arrhythmic marker?

This letter has been prompted by a study on QT dispersion (QTd) in patients with arrhythmogenic right ventricular dysplasia, published in the European Heart Journal[4]. In this study QTd was found to be increased in individuals with right ventricular dysplasia, but the magnitude of QTd was not related to the incidence of sustained ventricular arrhythmia or sudden death. In addition, QTd was not influenced by sotalol.

Although QTd has been used for several years as an index of heterogeneity of ventricular repolarization (thought to be associated with an increased pro-arrhythmic potential), it has not been found to be a useful parameter in every-day clinical cardiology. Part of the problem is that values of QTd in various study groups overlap and, consequently, it is impossible to reliably interpret an absolute value of QTd in an individual patient, unless the magnitude of QTd is very large[5]. Even more importantly, the true pathophysiological meaning of QTd is not known. Recent basic studies have shaken our traditional understanding of the pathophysiological basis of QTd and called the methodology used in question.

In the study by Zabel et al[3], a correlation between QTd and the dispersion of epicardial action potential durations was found. These results, however, do not necessarily imply that myocardial action potentials map onto discrete areas on the body surface and are reflected as QTd. In fact, in the very same paper a correlation was also found between dispersion of action potentials and the average T peak to T end interval (TPeT). The latter has been recently suggested to represent transmural dispersion of repolarization, resulting from differences in action potentials in the epicardium, endocardium and M cells[6]. Therefore, it is fair to say that, at present, we do not know what causes QTd and how electrical dispersion should be measured. What we do know, however, is that QTd is probably not what it is commonly thought to be.

A recent study by Kors et al[5] suggests that QTd should be regarded as a manifestation of spatial T-loop morphology, and can be related to the amplitude of the T-loop and its width, with QTd being smaller for ECGs with narrow and tall T-loops. It will also depend on the angle between the axis of the terminal part of the T-loop and an ECG lead axis, i.e. the more perpendicular the terminal T axis is to the lead axis, the shorter the QT duration. For certain critical values of the width, amplitude and angle, QT will not be measurable due to too small ST-T amplitudes and the lead will have to be excluded. It seems, therefore, that some excluded lead (e.g. due to technical difficulties) may contain very important information and may be the very lead that determines the magnitude of QTd. Consequently, it is unacceptable to exclude certain leads from analysis arbitrarily, just because it is not technically feasible to obtain a good quality signal. This, unfortunately, has been done by Benn et al[1] and, in fact, many other authors.

Another problem concerns the importance of diphasic T waves or U waves that, in the study by Benn et al[1], were excluded from analysis. Since U waves have been suggested to be related to the presence and action potential duration of a subpopulation of M cells[6], the exclusion of U waves by Benn et al. might have contributed to the lack of differences in QTd between patients with and without arrhythmias as well as to the lack of an effect of sotalol on QTd. The choice of leads used for analysis is also important. Clearly, in order to assess QTd in patients with right ventricular dysplasia more thoroughly, QT variability between right rather than left ventricular leads would have to be analysed.

I believe that in order to obtain a more objective assessment of QTd, only digitized recordings should be analysed. Manual analysis, as in the present study, is not reliable. How did the authors manage to draw tangents manually?

Finally, the presented study is retrospective, which further limits its interpretation. In a recent prospective study QTd was not found to be a prognostic marker[7].

In conclusion, with all my comments in mind, I believe that the study by Benn et al[1] does not provide any pathophysiological meaningful results and does not offer any new insight into the mechanisms involved in arrhythmogenesis in patients with right ventricular dysplasia. Unfortunately, this also holds for many other studies of this kind. I think that, for the future, we should focus our clinical and basic research efforts to explain the pathophysiological basis of QTd or find other non-invasive indices of electrical heterogeneity, rather than produce indiscriminately an ever-increasing number of uninterpretable, fallacious papers on QTd.

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References


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A reply

In his letter, R. Wolk, raises some critical points with regard to QT dispersion (QTd) in reference to a paper by our group[1].
As pointed out by R. Wolk, QTd is a marker which has been found useful in group comparisons, but which may be of limited value as a prognostic factor for the individual patients. The fact that the pathophysiological meaning of QTd is largely unknown, does, however, not affect the value of QTd as a risk marker in arrhythmogenic diseases.

In order to derive reproducible and reliable results with respect to QTd, it is of major importance that the reading of the ECG signal is made meticulously. Therefore, unreadable signals have to be excluded regardless of whether they possibly could contain important information. To our knowledge, a sufficiently precise method for digitizing QT intervals remains to be validated and as the process of digitizing may distort the signal leading to errors in QT length, we presently prefer to do the reading manually.

The prospective study in which QTd was not a prognostic marker was a study of post MI patients and thus it bears no impact on our conclusions regarding patients with arrhythmogenic right ventricular dysplasia.

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