EDITORIAL COMMENT

QT Dispersions: Fact or Fiction?

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The concept of QT dispersion originated from the work of Cowan et al[1] who found that QT intervals varied between leads and the variation was greater in patients with myocardial infarction than in control subjects. They also found that anteroseptal leads (V2 or V3) provided the closest approximation to QTmax. Thereafter, QT dispersion or corrected QT (QTc) dispersion has been regarded as a marker for electrical instability in the ventricles. Independent of degree of left ventricular dysfunction, increased QT dispersion correlated with the susceptibility for reentrant ventricular tachyarrhythmias, suggesting that a simple, noninvasive measurement from a standard 12-lead electrocardiogram (ECG) makes a great contribution to identifying patients at risk for life-threatening tachyarrhythmias after a previous myocardial infarction.[2]

Some investigators disagreed with this concept, arguing that all QT dispersions in the transformed ECG result from variations in projection of the T-wave loop and cannot be due to local heterogeneity of repolarization.[3] Nevertheless, there were still a number of studies demonstrating that an increased QT dispersion was a marker for arrhythmic events and sudden death.[2,4] Patients with myocardial ischemia, myocardial infarction, ventricular hypertrophy, congestive heart failure, and drug-induced toxicity[5] were considered to be at higher risk of life-threatening arrhythmias with greater degrees of QT dispersion.[6]

There are some technical problems in most of the studies involving QT dispersion. A number of investigators have raised the issue of the difficulties in determining the ending of the T-wave.[7] Implementation of a variety of current lead selection practices resulted in widely divergent estimates of QT interval.[6] There is a need for standardization of lead selection practice for QT measurement. If measurements are confined to one or a few leads, anteroseptal leads provide the closest approximation to QTmax.[1] Since QT dispersion is a relatively small value compared with the QT interval, a relatively small error in QT measurement augments the error in QT dispersion. Manual measurement using calipers is not always accurate; interobserver and intraobserver variabilities are high. Measurement using the digitizing method is better. Secondly, there is no consensus regarding whether the U-wave should be incorporated in the QT interval measurement. Also, it is not uncommon that the U-wave cannot be distinguished from the T-wave *per se*. Perhaps the most significant one is the discovery of the circadian rhythm of the QT dispersion.[8] Patients who suffered from sudden death had a morning surge in QT dispersion. The differences in the timing of recording ECG, which was not mentioned in some of the studies of QT dispersion, might be one of the reasons for the discrepancies in the literature.

An article in this issue of the *Journal of the Chinese Medical Association* describes a new finding in the changes in QT dispersion in relation to the in-hospital cardiac events in patients undergoing percutaneous coronary intervention for acute coronary syndrome.[9] The authors retrospectively inspected the ECGs of 128 patients with acute coronary syndrome who received percutaneous coronary intervention. They measured the QTc dispersions in the ECGs before intervention and in those after intervention, and divided the patients into 2 groups: one whose QTc dispersion shortened after intervention (shortened QTc dispersion group), and the other whose QTc dispersion prolonged after intervention (prolonged QTc dispersion group). They found that the shortened QTc dispersion group had a significantly higher rate of in-hospital cardiac death and a greater pre-intervention QTc dispersion. Interestingly, they also observed that patients with cardiac death had greater pre-intervention QTc dispersion

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than those without cardiac death. These results are different from the other reports.[2] It is generally believed that QTc dispersion decreases after successful coronary intervention,[10] and that effective revascularization of total coronary artery occlusion results in decrease in QTc dispersion.[11] The findings in the current paper are difficult to interpret. On the one hand, patients who suffered from cardiac death had a higher degree of QTc dispersion, a finding compatible with previous reports. On the other hand, patients whose QTc dispersion decreased after intervention, supposed to be beneficial, had a higher mortality rate.

It is unknown if there are racial differences in the changes in QTc dispersion in relation to cardiac risks. The QTc dispersion was manually measured in this article, which might introduce some errors. The interobserver and intraobserver variabilities were not reported. Above all, the time in the day of the ECG recording was not analyzed, which might be important in view of the circadian rhythm in the changes in QTc dispersion. The baseline data of the shortened QTc dispersion group were similar with that of the prolonged QTc dispersion group, except that the percentage of the pre-intervention TIMI grade 0 or 1 flow was higher in the former group ($p = 0.06$). This background difference might be important. It could be presumed that patients in the shortened QTc dispersion group had a higher risk before intervention, resulting in worse prognosis. This finding might partly explain why patients with shorter QTc dispersion in this study carried a higher risk. The authors might need to correlate the baseline QTc dispersion before intervention with the respective risk in each of the patients and see if there is any correlation. Further, it might be useful to compare 24-hour recordings of QTc dispersion and examine the circadian rhythms.

In summary, the findings in the aforementioned article are unique. More extensive patient enrollment and more detailed and standardized measurements are required to draw a conclusion regarding the reverse relationship between cardiovascular risks and QTc dispersion in Taiwanese patients.

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