

QT-Interval Dispersion and Myocardial Viability

To the Editor:

We have read the interesting article from Gadaleta et al¹ on prolongation of the corrected QT interval as an independent predictor of risk in acute coronary syndrome without ST elevation.

We present our study's data. It included 40 patients with demonstrated ischaemic cardiopathy, who had an echocardiography stress test taken with dobutamine, with the intention of studying the presence of viable myocardium, which was considered as improved basal contractility in 2 or more segments and in at least 1 point. The protocol consisted of 3 min phases with increasing dosages of dobutamine (5.10 and 20 $\mu\text{g/kg/min}$). An ECG of 12 derivations was recorded basally and at the end of each phase. The maximum and minimum QT intervals were evaluated; the dispersion (QTD) is the difference between the 2. The QT intervals were corrected according to Bazett's formula (QTDC). Patients with viable myocardium showed a greater QTD at low dobutamine dosages (10 μg) than ischaemic patients without viable myocardium, and differences were statistically significant (71.5 [21.5] and 56.3 [17.4] ms for patients with and without viable myocardium respectively; $P=.021$), and greater QTDC (86.1 [30.8] and 60 [20.1] ms respectively; $P=.013$). The ROC curve analysis showed an area below the curve of 0.76 (95% of confidence interval, 0.60-0.93; $P=.008$) (Figure). As a result, a QTDC >59 ms predicted myocardial viability with a sensitivity of 76.9% and specificity of 55.6%. However, the increase of QTD disappeared with higher dosages of dobutamine.

Our data coincide with others who have observed that myocardial viability associates with lower QTD at rest and an increase with infusion of low dosages of dobutamine (10 μg).^{2,3} Although an even greater increase of QTD has been described with high dosages of dobutamine (20 μg), it seems to be related with inducible ischaemia. Consequently, those with persistent akinesia did not show changes in QTD.⁴

Myocardial viability after an AMI is associated with a heterogeneous myocardial repolarization, and therefore, seems to associate with a greater

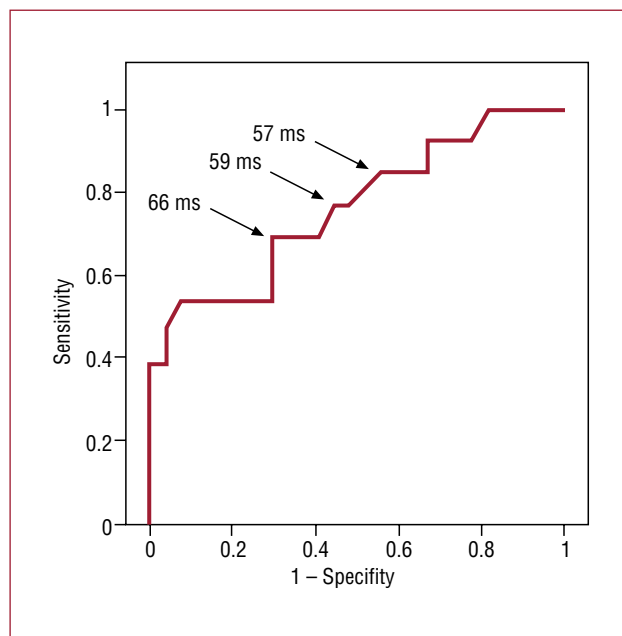


Figure 1. The ROC curve of QTd, where it shows that 59ms is the value which combines the best sensitivity and specificity for diagnosing myocardial viability.

QTd. These patients present a greater arrhythmic risk with exercise and an increased incidence of recurring ischaemic events.² It has been observed that changes in duration and QTd induced by exercise allow for identification of patients with a high risk of sudden death after an AMI.⁵

As a result, the measurement of QTd after an infusion of low dosages of dobutamine (10 µg) or during the completion of a stress test could help identify patients with viable myocardium after an AMI, with the resulting effects for the prognosis and procedure of these patients. The importance of this fact lies in the low cost and universal availability of ECG.

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Response

To the Editor:

Since 2003, when we published our first series of 102 patients, the objective was to call attention to a new application for measuring the QTc interval in patients with acute coronary syndrome without ST elevation. We showed that it was capable of predicting future ischaemic events and not arrhythmic ones.¹ The interesting work of Moreno et al is of the same indication. They were able to find out viable myocardial presence by dobutamine infusion and by using QT dispersion (QTd). By employing low dosages of dobutamine (10 µg), they found significant differences (71.5 [21.5] and 56.3 [17.4] ms) in patients with viable and non-viable myocardium, respectively ($P=.021$) and even greater QTdc (86.1 [30.8] and 60.0 [20.1] ms respectively; $P=.013$). Finally, they concluded that a QTdc >59 ms predicted myocardial viability. In 1979 Greeberg et al were the first to discuss QTc variations in the stress test.² Some years later, Sporton et al³ induced myocardial ischaemia with atrial stimulation in 24 patients and observed a clear increase of QTd in coronary patients compared to normal ones. In synchrony, by applying QTd to the stress test, Roukema et al⁴ showed for the first time that this is greater in coronary patients than in non-coronary controls (74 [7] compared to 40 [4]; $P<.003$). Recently, Carluccio et al⁵ studied the development of contraction anomalies evaluated by bidimensional echocardiograms and those induced by dipyridamole infusion. They observed 2 interesting facts; the first was the prolongation of maximum QT in patients who developed contractility alterations. The second was the increase of QTd in those with significant coronary lesions and in those where alteration of contractility was produced. However, in coronary

patients with a negative dipyridamole test, the QTd had no variation. Some received a second dosage of dipyridamole for contractile anomaly to appear, and in those, the QTd increase was at 162 % (64%). Finally, the use of aminophylline to resolve ischaemia not only reversed motility changes, but also normalized the QTd.

Finally, I believe the work of Moreno et al offers an important contribution to the application of QT and QTd intervals as predictors of ischaemic events, and as they say, given the low cost and universal availability, they should be sufficiently studied in the future.

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