QT dispersion in sinus beats and ventricular extrasystoles in normal hearts

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Abstract

Objective—Recent studies have suggested that QT interlead variability (dispersion) on the surface electrocardiogram may have potential as a measure of recovery time dispersion. To test this hypothesis further QT dispersion occurring in sinus beats was compared with that in ventricular extrasystoles.

Design—Simultaneous electrocardiograms were recorded at 50 mm/s during sinus rhythm in a drug free state while ventricular extrasystoles were introduced by programmed right ventricular stimulation at different coupling intervals. QT dispersion, defined as the difference between the maximum and minimum QT, was calculated separately for the extrasystoles and preceding and following sinus complexes. To correct for the influence of the number of measurable leads on QT dispersion, an “adjusted” QT dispersion calculated as QT dispersion/square root of the number of measurable leads, was used to compare sinus complexes and extrasystoles.

Patients—Nine patients were studied who were undergoing electrophysiological study for investigation of palpitation and were found to have electrically normal ventricles.

Results—At all coupling intervals tested “adjusted” QT dispersion was significantly greater in the ventricular extrasystoles than in either the preceding or following sinus complexes. For the coupling interval 350 ms, the 95% confidence intervals for the difference between means was 52 to 78 ms (preceding sinus complex) and 56 to 82 ms (following sinus complex) (p < 0.0001). There was no correlation between the coupling interval and the magnitude of the “adjusted” QT dispersion.

Conclusion—These results accord fully with expected differences in ventricular recovery time dispersion and offer further support for the hypothesis that QT dispersion reflects regional variation in ventricular recovery. If substantiated by invasive studies, these findings have wide implications for both the usefulness and the method of QT measurement.

As early as 1887 the QT interval of the surface electrocardiogram was recognised as a potential measure of electrical recovery in the ventricles. Since then, prolongation of the QT interval has been associated with a risk of arrhythmias, a poor prognosis after myocardial infarction, and drug toxicity and has been regarded as evidence that a cardiac drug has reached its target organ. The QT interval in these circumstances is a measure of the time from the earliest ventricular depolarisation to the latest repolarisation. But both these electrical processes are naturally temporally dispersed. A wave of excitation is followed by a wave of recovery. In an elegant review, Krikler identified the significance of the R-on-T ventricular extrasystole which not uncommonly complicates acute myocardial infarction and which shows that even early in the genesis of the surface electrocardiogram T wave some myocardial areas have regained excitability. Such dispersion of repolarisation is widely acknowledged as a likely substrate for serious ventricular arrhythmias but its existence is not exposed by a simple single measure of QT interval.

Work in our department has addressed whether interlead QT variability on the surface electrocardiogram is reflecting variation in myocardial recovery of excitability. Weak support for this hypothesis has come from analysis of patients with arrhythmogenic QT prolongation and antiarrhythmic QT prolongation and also from survivors of acute myocardial infarction treated with placebo and sotalol. We now report the effect of ventricular extrasystoles on interlead QT variability.

Patients and methods

METHODS

We studied nine patients who were undergoing electrophysiological investigation of palpitation. All were found to have electrically normal ventricles: none had evidence of preexcitation and none had inducible ventricular tachycardia. Continuous simultaneous 12 lead electrocardiograms were recorded at 50 mm/s during sinus rhythm in a drug free state while ventricular extrasystoles were introduced by programmed right ventricular stimulation either 300 ms or 350 ms after the preceding Q wave. A wider range of coupling
“Adjusted” QT dispersion (mean (SD)) in ventricular extrasystoles (coupling interval 350 ms from preceding Q wave) and preceding and following sinus complexes. “Adjusted” dispersion was calculated as the difference between maximum and minimum QT divided by the square root of the number of measurable leads.

Table 1 “Adjusted” QT dispersion (mean (SE)) in ventricular extrasystoles and preceding and following sinus complexes

<table>
<thead>
<tr>
<th>Coupling interval</th>
<th>Preceding sinus complex</th>
<th>Ventricular extrasystole</th>
<th>Following sinus complex</th>
</tr>
</thead>
<tbody>
<tr>
<td>300</td>
<td>22 (2)</td>
<td>80 (4)</td>
<td>23 (6)</td>
</tr>
<tr>
<td>350</td>
<td>18 (2)</td>
<td>87 (6)</td>
<td>18 (2)</td>
</tr>
</tbody>
</table>

“Adjusted” dispersion was calculated as the difference between maximum and minimum QT divided by the square root of the number of measurable leads. The coupling interval is the interval between the ventricular extrasystole and preceding Q wave.

Results
Table 1 and the figure show the results of QT dispersion analysis of sinus complexes and ventricular extrasystoles for the coupling interval 350 ms. The mean “adjusted” QT dispersion of the ventricular extrasystole was significantly greater than that of either the preceding or following sinus complex (p < 0.00001) with 95% confidence intervals for the difference between means of 72 to 78 ms (preceding sinus complex) and 56 to 82 ms (following sinus complex). There was no difference in “adjusted” dispersion between preceding and following sinus complexes. Similar results were obtained for the coupling interval 300 ms (also shown in table 1).

Table 2 shows the results of “adjusted” QT dispersion occurring in ventricular extrasystoles at a range of different coupling intervals. There was no correlation between the “adjusted” QT dispersion and the coupling interval, although for each interval the dispersion of the ventricular extrasystole was at least three times greater than that of either the preceding or following sinus complex. In an attempt to correlate the magnitude of QT dispersion with the timing of the ventricular extrasystole in relation to the state of ventricular recovery we calculated the “prematurity index” expressed as the ratio of the coupling interval to the QT interval of the normal beat or R-R/QTmax, for each electrocardiogram (data not shown).

There was no correlation between this “prematurity index” and “adjusted” QT dispersion.

Discussion
These results are a third piece of evidence that interlead QT variability as measured on the 12-lead electrocardiogram is not merely a technical artefact but probably reflects dispersion of recovery of ventricular excitability. The adjusted QT dispersion of ventricular extrasystoles far exceeded that of the preceding or succeeding sinus complexes. Ventricular extrasystoles most certainly involve dispersion and disruption of activation, and some of the measured QT variation must be attributed to this aspect. The lack of a positive correlation between the ventricular extrasystole coupling interval and the measured dispersion suggests that activation disturbances were the predominant factor contributing to the QT dispersion. This is supported by the fact that irrespective of short coupling intervals producing what were tantamount to R-on-T ventricular extrasystoles the configuration of induced extrasystoles and stimulus to onset times were independent of the coupling interval. The increased dispersion of activation times will also lead to increased dispersion of action potential durations depending on electrical restitution, but if this were the major factor producing QT dispersion then dispersion would have been expected to vary with coupling interval. Our work in humans accords with animal studies in
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Table 2 “Adjusted” QT dispersion in ventricular extrasystoles at varying coupling intervals

<table>
<thead>
<tr>
<th>Coupling interval (ms)</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
<th>Case 4</th>
<th>Case 5</th>
<th>Case 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>280</td>
<td>77</td>
<td>77</td>
<td>77</td>
<td>77</td>
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<tr>
<td>300</td>
<td>57</td>
<td>95</td>
<td>75</td>
<td>81</td>
<td>101</td>
<td>81</td>
</tr>
<tr>
<td>320</td>
<td>120</td>
<td>72</td>
<td>95</td>
<td>101</td>
<td>57</td>
<td>101</td>
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<td>72</td>
<td>113</td>
<td>78</td>
<td>66</td>
<td>75</td>
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</tr>
<tr>
<td>360</td>
<td>78</td>
<td>124</td>
<td>107</td>
<td>72</td>
<td>95</td>
<td>121</td>
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<td>78</td>
<td>98</td>
<td>61</td>
<td>66</td>
<td>107</td>
</tr>
</tbody>
</table>

See footnote to table 1.

Thus Q wave onset, like pacing spike onset, is virtually synchronous with the earliest onset of ventricular activation.

Late in the development of one of cardiology’s classic tools we have found that the surface electrocardiogram may provide new information. All available evidence is that QT dispersion does reflect underlying regional variations in the recovery of ventricular excitability. If this view is more widely endorsed then a radical revision of QT measurements will be necessary. Classic studies from the past may not survive scrutiny.

The only scientifically acceptable QT information that would be obtained from a 12 lead electrocardiogram would be either the maximum measured QT interval (implying a search for this on all 12 leads of the surface electrocardiogram) or dispersion of QT intervals.

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1. Walker AD. A demonstration on man of electromotive changes accompanying the heart’s beat. J Physiol 1877;8:229.