ORIGINAL ARTICLES

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 extrastimuli 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) ($p < 0.00001$). 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.¹ Since then, prolongation of the QT interval has been associated with a risk of arrhythmias,² a poor prognosis after myocardial infarction,³ and drug toxicity⁴ and has been regarded as evidence that a cardiac drug has reached its target organ.⁵ 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.⁶ 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. Strong support for that hypothesis has come from analysis of patients with arrhythmogenic QT prolongation⁷ and also from survivors of acute myocardial infarction treated with placebo and sotalol.⁸ 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

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"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.

intervals from 280 ms to 400 ms was also tested in six of the patients. Ten sinus complexes separated the differently timed ventricular extrasystoles.

QT measurements were performed by one observer using a digitiser (CALCOMP 9000). All electrocardiographic analysis was performed after the studies were completed. QT intervals were measured from the onset of the QRS to the end of the T wave (defined as its return to the T-P baseline) in the last completed sinus complex preceding the ventricular extrasystole and the first sinus complex following the ventricular extrasystole. When U waves were present, the QT was measured to the nadir of the curve between the T and U wave. For the ventricular extrasystoles the interval between the pacing spike and the end of the T wave was measured. QT dispersion, defined as the difference between maximum and minimum QT, was calculated separately for the ventricular extrasystoles, the preceding sinus complexes and the following sinus complexes. QT measurements were performed only in those leads where the T wave end could be reliably identified. In a previous study we showed that QT dispersion values are directly proportional to the square root of the number of measurable leads and derived an "adjusted" QT dispersion calculated as QT dispersion divided by the square root of the number of measurable leads to correct for this influence.⁸ Accordingly, for statistical analysis, this "adjusted" QT dispersion was calculated and used to compare the dispersion occurring in sinus complexes and ventricular extrasystoles.

Student's paired *t* test was used to compared the mean "adjusted" QT dispersion occurring in sinus complexes and ventricular

extrasystoles. Confidence intervals were calculated for the differences between means.

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 $R-R/QT_{max}$,⁹ 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

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

Coupling interval	"Adjusted" QT dispersion (ms)		
	Preceding sinus complex	Ventricular extrasystole	Following sinus complex
300	22 (2)	80 (4)	23 (6)
350	18 (2)	87 (6)	18 (2)

"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.

Table 2 "Adjusted" QT dispersion in ventricular extrasystoles at varying coupling intervals

Coupling interval (ms)	"Adjusted" QT dispersion (ms)					
	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
280	—	118	—	78	—	72
300	57	95	—	75	81	101
320	—	120	72	95	101	57
350	72	113	78	66	75	78
360	78	124	107	72	95	121
400	69	78	98	61	66	107

See footnote to table 1.

which temporal dispersion of ventricular recovery was increased considerably more by a ventricular extrasystole than by a sinus complex.¹⁰ In these experiments increased dispersion of ventricular activation times rather than increased dispersion of action potential durations was responsible for the difference.

Our investigations showed that the QT dispersion of the first sinus complex after the ventricular extrasystole was almost identical to that of the sinus complex preceding the extrasystole. Thus despite the important electrical perturbations caused by the ventricular extrasystole, the electrophysiology of the ventricle returned to the pre-ventricular extrasystole conditions within a single cardiac cycle in these patients with normal hearts. This finding supports the concept of "T wave memory" whereby the characteristics of electrical recovery are retained except in the face of prolonged and consistent abnormalities of activation.^{11 12}

The methods we used may have been flawed. Surface electrocardiographic QT measurement is subject to errors but previous work has shown that variation in interlead measurements far outweigh inaccuracies introduced by any other factor including interobserver variation.¹³ It was obviously impossible to blind the observer to whether the complex being measured was sinus or extrasystolic but the large differences in QT dispersion between the two types of complex seem unlikely to be explained by observer bias alone. There are potential problems in comparing the dispersion of sinus initiated QRS complexes with that of QRS complexes induced by a pacing spike. Unlike the pacing spike the onset of the Q wave might be expected to vary between leads according to previously established variations in activation time^{14 15}; QT dispersion would then reflect dispersion of action potential durations rather than dispersion of ventricular recovery time. Previous work, however, has shown interlead variation in Q wave onset to be small compared with known regional differences in activation time,^{13 16} which is considered to reflect the greater dependence of Q wave onset on global rather than local cardiac events.¹⁶

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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