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QT Dispersion as an Attribute of T-Loop Morphology

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Background—The suggestion that increased QT dispersion (QTD) is due to increased differences in local action potential durations within the myocardium is wanting. An alternative explanation was sought by relating QTD to vectorcardiographic T-loop morphology.

Methods and Results—The T loop is characterized by its amplitude and width (defined as the spatial angle between the mean vectors of the first and second halves of the loop). We reasoned that small, wide ("pathological") T loops produce larger QTD than large, narrow ("normal") loops. To quantify the relationship between QTD and T-loop morphology, we used a program for automated analysis of ECGs and a database of 1220 standard simultaneous 12-lead ECGs. For each ECG, QT durations, QTD, and T-loop parameters were computed. T-loop amplitude and width were dichotomized, with 250 μV (small versus large amplitudes) and 30° (narrow versus wide loops) taken as thresholds. Over all 1220 ECGs, QTDs were smallest for large, narrow T loops (54.2±27.1 ms) and largest for small, wide loops (69.5±33.5 ms; $P<0.001$).

Conclusions—QTD is an attribute of T-loop morphology, as expressed by T-loop amplitude and width. **(*Circulation*. 1999;99:1458-1463.)**

Key Words: electrocardiography ■ computers ■ potentials

QT dispersion (QTD) has been defined as the difference in duration between the longest and shortest QT intervals in any lead for a given set of ECG leads. QTD has been suggested to reflect regional variation in ventricular repolarization.[1–5] Different parts of the myocardium may have differing action potential durations, which is known to be true in a healthy heart.[6,7] In diseased myocardium, there is likely to be an increased difference in action potential durations. This increased heterogeneity within the heart muscle is then assumed to be responsible for increased differences in QT duration between ECG leads at the body surface, ie, increased QTD.[8]

It is erroneous to think, however, that the end of the T wave in a given lead is directly related to the action potential durations in some corresponding region of the heart. At the end of the cardiac cycle, when the last myocardial cells finish their repolarization, the electric field generated by these last active sources will extend throughout the trunk and be picked up by every single electrode one might choose to put in or on the body. At all electrode sites, repolarization potentials must therefore have principally the same duration. This is a consequence of simple electric field theory.[9] One cannot measure, however, the potential of individual electrodes but only the potential difference between 2 lead electrodes. If the electric signal in a lead falls to zero, the electrode potentials have become equal. (It must be remembered that the precordial leads are also essentially bipolar because the central terminal by no means constitutes a zero potential.[10]) There is only 1 end of repolarization, which is the common end for all leads together, when the electric field dissolves and all potential differences vanish.

Still, QTD cannot be dealt with simply as a measurement problem because it has been shown that QTD has a certain diagnostic capability.[3,5,11–18] In this article, we will present evidence that QTD can be regarded as a manifestation of spatial T-loop morphology; ie, we will explain the phenomenon of QTD in terms of 3 interacting factors: the amplitude of the T loop, its width, and the number of leads in which the end of the T wave could not be determined because of too low T-wave amplitudes. This will be worked out quantitatively with a large database of ECGs and a computer program for automatic measurement of QTD and T-loop parameters.

Methods

Database

All measurements were done on a database of 1220 standard 12-lead ECGs collected in the Common Standards for Quantitative Electrocardiography project.[19] All leads of each ECG were recorded simultaneously at a sampling rate of 500 Hz during 8 or 10 seconds. The clinical diagnosis of the 1220 individuals has not been released, but the database is known to contain 382 normal subjects; the rest have various abnormalities.[19]

Measurements

For data processing, the Modular ECG Analysis System (MEANS), our ECG computer program,[20] was used. The operation of the waveform recognition algorithms has been described and validated

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extensively.[20,21] MEANS determines common QRS onset and offset and T offset for all 12 leads together on 1 representative averaged beat by use of template matching techniques.[22]

For QTD measurement, the location of the overall end of T is taken as a starting point. The program then determines the end of the T wave in each separate lead by use of a threshold algorithm that is dependent on noise level. QTD is then computed as the difference between the maximum and minimum QT intervals. The T wave in a lead may be so flat that measuring its end point is impossible. In common QTD measurement practice, the lead will then be excluded from analysis. In our experiments, we excluded leads with peak-to-peak ST-T amplitudes of <50 μV (½ mm), at which level the wave is considered flat for all practical purposes. The performance of the program in determining QTD was shown to be comparable with that of human observers.[23]

To derive T-loop parameters, vectorcardiographic leads X, Y, and Z were reconstructed from the standard ECG leads.[24,25] The following parameters were taken to characterize T-loop morphology: initial and terminal axes, width, and maximal amplitude. Each parameter can be determined for the spatial T loop and its projection on the frontal (XY), horizontal (XZ), and sagittal (YZ) planes. The initial axis, T_1, is obtained by vectorially adding the instantaneous heart vectors during the first half of the T loop (half is defined as half the geometrical circumference of the loop). The terminal axis, T_2, is obtained similarly for the second half of the T loop. The greater the width of the loop, the more divergent the initial and terminal parts. We thus defined T width as the angle between T_1 and T_2. To quantify the effect of T-loop width and amplitude on QTD, we dichotomized these parameters, taking 250 μV (small versus large amplitudes) and 30° (narrow versus wide loops) as threshold values. The amplitude threshold was based on previous reports on normal values of T-loop amplitude; the width threshold was, after some geometrical manipulations, derived from the rule of thumb that a normal T loop should have a length $\geq$2.5 times greater than its width.[26]

Statistical Analysis

Statistical analysis was performed by use of Student's *t* test for unpaired samples. Data are presented as mean±SD.

Results

QT Duration and Terminal T Axis

As argued above, the fact that the T wave in some lead becomes zero before the T waves in other leads signifies that the potentials of the lead electrodes have become equal within the limits of the measurement accuracy. This is equivalently expressed by saying that the orientation of the electric heart vector has become perpendicular to the lead axis, as already stated by Einthoven et al.[27] Consequently, the shortest QT durations are expected to occur in leads perpendicular to the axis of the terminal part of the T loop (T_2). To test this hypothesis, we assume a simplified but common model for the directions of the lead axes. In the frontal plane, they are taken to range from −30° for lead aVL, 0° for lead I, to 120° for lead III, in 30° steps. The precordial leads are supposed to lie in a transversal plane, with V_6 at −30°, V_5 at 0°, and again in 30° steps to V_1 at 120°. In these same 2 planes, for each of the 1220 ECGs, the direction of T_2 was calculated, as were the 6 QT durations, 1 of them being the maximum QT (QT_{max}). Thus, there is a difference between QT measured in the lead and QT_{max}.

Figure 1 shows the mean of the 1220 differences for each lead as a function of the angle between lead axis and T_2 for the frontal and horizontal planes separately. For leads parallel to T_2 (angle between lead axis and T_2 of 0° or 180°), the mean difference between QT in the lead and QT_{max} is smallest. (In fact, QT in that lead will often be QT_{max}.) The larger the angle is between T_2 and the lead, the shorter QT tends to be and the larger the difference is until, when a lead is perpendicular to T_2 (at −90° or 90°), the mean difference is largest, ie, QT is shortest. For each lead, we tested whether the mean difference at 0° or 180° was equal to the difference at −90° or 90°. All differences between these means proved highly significant ($P<0.001$) for all leads.

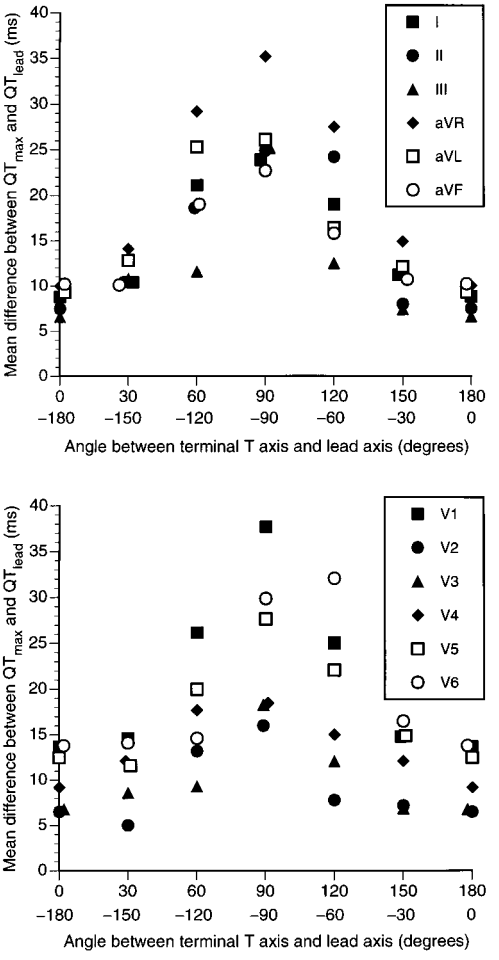

Figure 1. Difference between QT_{max} and QT duration measured in individual leads (QT_{lead}) as function of angle between axis of terminal part of T loop and lead axis for extremity leads (A) and precordial leads (B). Angles are grouped in classes of 30°; eg, class (90, −90) contains all angles between 75° and 105° and between −105° and −75°.

Lead Exclusion

In terms of T-loop parameters, we will examine the situations in which flat ST-T waves may occur in a lead. If a T loop has a narrow, elongated ("spindlelike") shape, its projection on a given lead axis will result in a well-discernible T wave as long as the angle with the lead axis is narrow enough (Figure 2A). The more perpendicular to a lead the loop becomes, the smaller its projection is and the lower the ST-T amplitude is in the lead until it may decrease to <50 μV, which was used as an exclusion criterion (Figure 2B). This will be the case in 1 lead only unless the T loop is perpendicular to 1 of the planes, in which situation it will be perpendicular to all leads

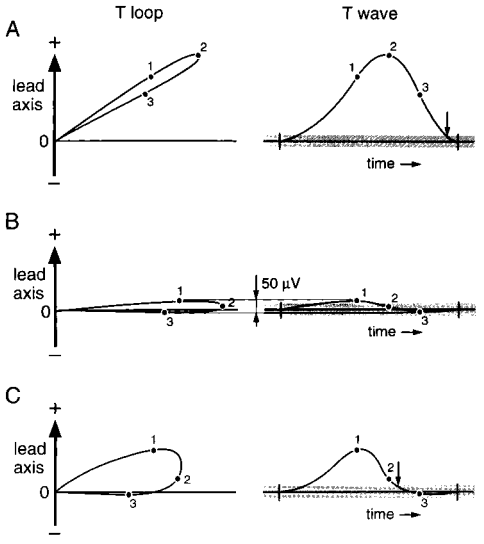

Figure 2. Lead exclusion and shortening of QT duration. Projection of T loops on hypothetical lead axis, plotted against time, yields T waves depicted. For time instants 1, 2, and 3, corresponding points on T loops and T waves are indicated. Shaded zone represents noise band. Ends of T waves are marked by vertical arrows and do not coincide with end of repolarization because of limited measurement accuracy. A, Narrow, elongated T loop at oblique angle with lead axis results in well-discernible T wave in that lead with well-defined end, almost coinciding with real end of repolarization. B, If same T loop is rotated to be perpendicular to lead axis, low ST-T amplitudes result. When peak-to-peak amplitude sinks to <50 μV, lead is excluded from further analysis. C, Wide T loop with its terminal part perpendicular to lead axis gives rise to T wave that ends before overall end of repolarization but is not excluded from analysis.

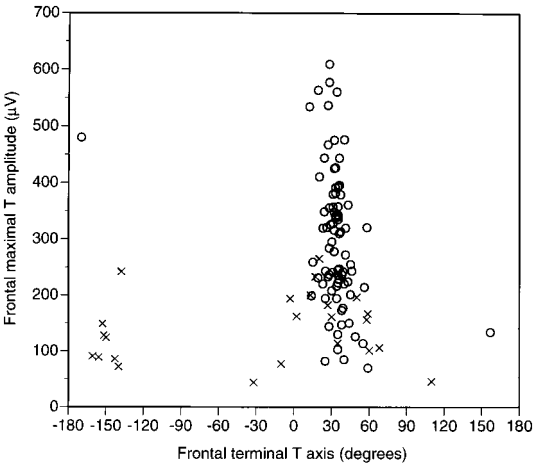

Figure 3. Maximum amplitude of frontal T loop versus terminal axis of frontal T loop for all ECGs in which lead III was excluded from measurement because of too low ST-T amplitude. Narrow T loops are marked as "o"; wide T loops, as "x." Good-sized T loops are never excluded when they are wide but are excluded if they are narrow and have terminal T axis perpendicular to lead III at 30° and −150°. If maximum T amplitude is small, both narrow and wide T loops may be excluded.

of the plane. The smaller the T loop, the less perpendicular to a lead it has to be before the ST-T amplitude is so low that it results in exclusion of the lead. Moreover, a small-enough T loop will produce low ST-T waves in more leads than the 1 more or less perpendicular to it.

A wide, round T loop with its terminal axis perpendicular to a lead will tend to have an initial axis at an oblique angle of incidence to the lead, so its projection will not become zero and the ST-T wave will stay large enough not to be excluded (Figure 2C). Exclusion will occur only if the loop is overall very small.

Of the 1220 ECGs, leads were excluded in 429 ECGs (35.2%), of which 326 had 1, 79 had 2, and 24 had >2 excluded leads. To illustrate the relationship between lead exclusion and T-loop parameters, Figure 3 shows a scatterplot of frontal T_2 versus frontal amplitude for those ECGs in which lead III was excluded, with narrow loops marked as "o" and wide loops as "x." As expected, lead III is excluded only if the ECG has a tall, narrow T loop with a T_2 that clusters at $\approx$30° and −150° (which is perpendicular to the lead axis of III, assumed to lie at 120°) or if the amplitude of its T loop is small.

QTD and T Loop

Several factors come into play to explain the relation between QTD and T-loop morphology. We hypothesize that spatial T amplitude, T width, and number of excluded leads (which in turn depends on axis, amplitude, and width, as explained above) affect QTD. If the angle between the axis of the terminal part of the T loop (T_2) and the lead axis deviates sufficiently from 90°, the end of the T wave in that lead will coincide approximately with the end of the spatial T loop, ie, with the cessation of all repolarization activity (Figure 2A). If T_2 is perpendicular to the lead, however, the projection on the lead axis will be zero, and the T wave in that lead will end before the overall end of repolarization (Figure 2C). QT duration will then be shorter in that lead than in a less perpendicular lead, and QTD results.

There is a difference between narrow and wide loops, as discussed above. In a narrow loop, when T_2 is perpendicular to a lead, T_1 also is approximately perpendicular, and the projection of the whole loop on the lead axis is small, so the ST-T wave may become subthreshold and be excluded (Figure 2B). If in a round loop the terminal part is perpendicular to a lead, QT duration is likewise shortened. The initial part, however, because of the roundness of the loop, will generally be less perpendicular to the lead, and the ST-T wave retains sufficient amplitude not to be excluded. QTD might therefore be measured in round loops, while being absent in narrow loops by exclusion of the very leads by which it would become manifest.

Overall decreased spatial amplitude will result in decreased ST-T amplitudes. This also leads to increased QTD because of the increased uncertainty in determining the end of low T waves,[23,28,29] as long as the measurement is not excluded because of too low ST-T amplitude.

The Table shows the mean QTD of ECGs in subgroups by spatial T amplitude and width, distinguishing between ECGs in which all 12 leads or in which <12 leads could be measured. Mean QTDs are smallest for narrow, high-amplitude T loops (54.2±27.2 ms) and largest for small, wide loops (69.5±33.5 ms, $P<0.001$). The percentage of ECGs with $\geq$1 excluded leads is lowest for large, wide T loops

Mean QTD of 1220 ECGs for Different Categories of T-Loop Width and Amplitude

		Lead Exclusion‡		QTD, ms		
Width*	Amplitude†	n	%	All	12 Leads	<12 Leads
Narrow	Large	196/600	32.7	54.2±27.1	57.6±26.2	47.2±27.8
Narrow	Small	63/80	78.8	57.9±27.7	60.8±23.4	57.1±28.9
Wide	Large	70/314	22.3	62.9±26.3	66.2±26.6	51.4±22.2
Wide	Small	100/226	44.2	69.5±33.5	73.3±32.5	64.6±34.3

*Narrow, width <30°; wide, width ≥30°.
†Small, amplitude <250 μV; large, amplitude ≥250 μV.
‡ECGs with <12 leads measured.

(22%) and highest for small, narrow loops (79%). The mean QTD of ECGs with≥1 leads excluded is between 3.7 and 14.8 ms shorter than the QTD of ECGs with no excluded leads.

For the presentation of these results, correlation coefficients that presuppose linear relations between variables are not the appropriate means. The relationships between our parameters and QTD are highly nonlinear: The exclusion of leads is an all-or-none decision; the increases in amplitude and width do not lead to a proportional increase in QTD; and the interplay between the 3 factors also creates unforeseeable nonlinearities.

Discussion

We argued on physical grounds that a commonly suggested explanation for the mechanism underlying QTD, ie, local differences in action potential durations, does not hold. Instead, we propose an alternative explanation that relates QTD to T-wave morphology. We showed that ECGs with narrow, tall T loops have relatively small QTD values, whereas wide, small T loops have the largest QTDs. Width and amplitude also determine whether a QT duration is measurable in all 12 leads or in <12 leads, which in turn affects QTD. Thus, QTD should be considered an attribute of T-loop morphology.

We also demonstrated that the T axis is associated with QT duration: The more perpendicular the terminal T axis is to the lead axis, the shorter the QT duration is. Conversely, the more parallel the T axis is with the lead axis, the longer the QT duration is. One may wonder whether this fact alone would not be sufficient to explain the differences in QTD between patient and control groups that have been reported in many studies. However, this is not the case because the lead axes of the extremity leads and the precordial leads are periodically distributed over the frontal and transversal planes, respectively. The direction of the terminal T axis determines which lead will have the shortest QT duration (the lead perpendicular to the T axis) and which will have the longest (the lead parallel with it). If the direction of the terminal T axis changes, the leads with the shortest and longest QT will change, but the difference between the longest and shortest QT duration, ie, QTD, will remain the same, independent of the terminal T axis. To explain differences in QTD, T-loop morphology has to be taken into account.

We chose a simple model for the lead axes, and more sophisticated lead models would have been possible. However, more realistic sets of lead axes also have limitations because each was derived from only 1, often not even heterogeneous, mathematical or physical torso model. Moreover, we use the lead model only to illustrate the relationship between QT duration and terminal T axis (Figure 1), not to demonstrate that QTD is an attribute of T-loop morphology (the Table).

Mean differences between maximum QT and QT durations in individual leads were studied before by Cowan et al,[11] but they did not relate their findings to the T axis. Several investigators[11,28,30] have suggested that QTD might be due to the different projections of the heart vector on the different lead axes. To the best of our knowledge, an explanation in terms of T-loop morphology has not been given before.

Priori et al[31] have related QT duration to T-wave morphology as expressed in the principal components of the 8 independent ECG signals. They defined an index of complexity of repolarization (CR) as the ratio between the first and second eigenvalues and showed that CR discriminates between long-QT syndrome patients and control subjects. Their index, however, was not significantly correlated with QTD, and they did not try to explain the phenomenon of QTD in terms of the index. The approach of these authors is mathematical and is not concerned with T-loop shape. We expect the first eigenvalue to be related to the maximum amplitude of the T loop and CR to its width.

In a study by Badilini et al,[32] myocardial infarction patients and individuals with long-QT syndrome were shown to have increased QTD compared with normal subjects. Badilini et al also assessed T-loop roundness, similar to CR, and planarity. T-loop amplitude and number of excluded leads were not taken into account. Only moderate correlations between QTD and T-loop parameters were found, which should not come as a surprise in view of the nonlinearity of the relationships, as indicated above. Again, an explanation of the phenomenon of QTD, the major objective of our study, is not offered.

The relation between epicardial action potential durations and QTD has been investigated by Zabel et al.[8] Action potential durations were measured on a rabbit heart suspended in a tank and were varied by administration of D-sotalol. The dispersion of action potential durations corre-

lated with the QTDs measured at the surface of the tank. The authors do not explain how the heterogeneity of repolarization in the heart is connected to QTD. Changes in the course and duration of action potential durations in the heart will certainly cause changes in the surface ECG, but this does not mean that areas with longer or shorter action potential durations in the heart are mapped onto discrete areas of increased or decreased repolarization potential duration on the body surface, resulting in a QTD increase. In our thinking, heterogeneity of repolarization leads to greater variability in T-loop morphology. A characteristic of T-loop morphology is width, and we demonstrated that larger T-loop width increases QTD; low T-loop amplitude is a second factor. This does not contradict our principle that local variations in repolarization potential durations on the body surface cannot exist, considering that all repolarization potentials must end at the same moment. The determination of the end of T in the ECG is the measurement of a potential difference, and this measurement yields zero simply when the lead electrodes have equal potential, which can occur in any lead at which the repolarization vector becomes perpendicular to the lead axis.

Our results would also explain why increased QTD is associated with a variety of pathologies, as has been reported in many previous studies.[3,5,11–18] In clinical vectorcardiography, it is a well-known fact that normal T loops usually have elongated, narrow shapes with spatial T amplitudes of $\approx$500 μV.[33,34] Wide T loops, on the other hand, are considered a sign of various forms of pathology, as are small amplitudes of the T loop.[35] In the present study, the difference in mean QTD between these normal (long and narrow) and abnormal (small and wide) T loops was 15.3 ms. In previous studies that compared QTDs of myocardial infarction patients and control subjects (2 groups that constitute about two thirds of our study population), differences between mean QTDs ranged from 15 to 26 ms.[4,11,13,29] Our results are in accordance with these findings. The thresholds of 250 μV for amplitude and 30° for width are not very critical. Changes of 15% to 20% up or down did not give essentially different results.

As has been said, the shape of the T loop somehow reflects the distribution and course of action potentials in the ventricular myocardium. Unfortunately, although certain T-loop characteristics are helpful in recognizing pathological conditions, our understanding of the relationships between T-loop morphology and the pathophysiology of specific repolarization abnormalities is limited. In that respect, one might object that we are not better off with T-loop morphology than with QTD. But as we have argued, QTD is not a physically sound concept. Its existence is due to a measuring problem that can be understood in terms of T-loop morphology. This suggests that T-loop parameters may have a discriminative and prognostic value that is at least as good as that of QTD. Moreover, they can be measured more easily and less ambiguously than QTD.

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