Assessment of QT Interval and QT Dispersion for Prediction of All-Cause and Cardiovascular Mortality in American Indians
The Strong Heart Study

Peter M. Okin, MD; Richard B. Devereux, MD; Barbara V. Howard, PhD; Richard R. Fabsitz, MA; Elisa T. Lee, PhD; Thomas K. Welty, MD

Background—Both a prolonged QT interval and increased QT interval dispersion (QTD) have been proposed as surface ECG markers of vulnerability to ventricular arrhythmias and potential predictors of mortality.

Methods and Results—The predictive values of QT prolongation and QTD were assessed in 1839 participants in the Strong Heart Study, a prospective study of cardiovascular disease in American Indians. ECGs were acquired at 250 Hz; QT intervals were measured by computer in all 12 leads and corrected for heart rate (QTc) by use of Bazett’s formula. QTD was calculated as the difference between the maximum and minimum QTc. After a mean follow-up of 3.7±0.9 years, there were 188 deaths from all causes, including 55 cardiovascular deaths. In univariate Cox analyses, prolonged QTc and increased QTD were significant predictors of all-cause mortality ($\chi^2=53.0, P<0.0001$; $\chi^2=13.7, P=0.0008$) and cardiovascular mortality ($\chi^2=14.7, P=0.0001$; $\chi^2=26.5, P<0.0001$). In multivariate Cox regression analyses controlling for risk factors, QTc remained a strong predictor of all-cause mortality ($\chi^2=16.5, P<0.0001$) and a weaker predictor of cardiovascular mortality ($\chi^2=5.8, P=0.016$); QTD remained a significant predictor of cardiovascular mortality only ($\chi^2=12.5, P=0.0004$).

Conclusions—These findings support the value of computerized measurements of QTc and QTD in noninvasive risk stratification and suggest that these surface ECG variables may reflect different underlying abnormalities of ventricular repolarization. (Circulation. 2000;101:61-66.)

Key Words: electrocardiography • risk factors • QT interval • QT dispersion

Increased QT interval dispersion (QTD) on the surface ECG has been linked to increased heterogeneity of ventricular repolarization, implicated in the genesis of ventricular arrhythmias, and has been associated with an adverse prognosis in a variety of patient populations. However, the prognostic value of QTD remains clouded by conflicting findings in patients after myocardial infarction and in patients with congestive heart failure. Prolongation of the QT interval has also been implicated in the genesis of ventricular arrhythmias, but like QTD, the prognostic value of QT prolongation remains uncertain. The variable and uncertain predictive value of QT interval prolongation and increased QTD may be due in part to difficulties in accurate and reproducible determination of T-wave offset with standard analog ECG recordings and measurement techniques. However, use of digitally acquired ECGs with computerized detection of T-wave offset significantly enhances the accuracy and reproducibility of QT interval measurements, holding promise for improving the clinical utility of QT interval and QTD. The present study was performed to examine the value of computerized QT interval and QTD measurements from digitally acquired ECGs for prediction of all-cause and cardiovascular mortality in a prospective, population-based study.

Methods

Study Population

The Strong Heart Study is a community-based study of cardiovascular disease and its risk factors in American Indians, American Indians 45 to 74 years of age during the period of July 1989 to January 1992 who were resident members of the following 13 tribes were eligible for participation in the first Strong Heart Study examination: the Pima/Maricopa/Papago tribes of central Arizona who live in the Gila River, Salt River, and Ak-Chin communities; the 7 tribes of southwestern Oklahoma (the Apache, Caddo, Comanche, Delaware, Fort Sill Apache, Kiowa, and Wichita); and the Oglala and Cheyenne River Sioux in South Dakota and the Spirit Lake Tribe.
in the Fort Totten area of North Dakota. These field centers were chosen because they represented expected low-, intermediate-, and high-risk areas for cardiovascular disease. Enrollment procedures for the study have been previously published.\textsuperscript{19} Participation rates were 71% in Arizona, 62% in Oklahoma, and 54% in South and North Dakota. Participants and nonparticipants were similar with respect to tobacco use and diabetes prevalence, but nonparticipants were more likely to be male (54% versus 41%), were slightly older (59 versus 57 years), were less likely to be hypertensive (30% versus 42%), and were slightly less obese than participants (M. Stoddart, e-mail communication).

Electrocardiography

Standard 12-lead ECGs were performed with MAC-PC or MAC-12 digital ECG systems (GE-Marquette Medical Systems) as previously described.\textsuperscript{20} For each ECG, 10 seconds of data was digitally recorded at a 250-Hz sampling frequency to a resolution of 5 $\mu$V and stored in a Marquette MUSE system for computer measurements. Digital ECG records were available on 2140 of the 4544 eligible participants (47.1%) in whom ECGs were obtained during phase 1; the remainder of digital ECGs had been lost in a catastrophic disk crash at the Fitzsimons Army Medical Center, where the ECGs were originally stored. Participants with digital ECGs were nearly identical in age (56.1±8.2 versus 56.5±8.0 years) and were slightly more likely to be male (42.1% versus 39.1%, $P=0.04$) than those without digital ECGs. In addition, there were small but statistically significant differences in tribal representation between those with and without digital ECGs (Arizona, 33.2% versus 32.7%; Oklahoma, 30.6% versus 36.4%; and North and South Dakota, 36.2% versus 30.9%; $P<0.001$).

**QT Interval and QTD Measurement**

QT interval and QTD measurements were performed from median complexes on the digital ECGs by use of interactive software (QT-Guard, GE-Marquette Medical Systems) that detects QRS onset and T-wave offset\textsuperscript{17,18} and was validated by a single investigator (Dr Okin) who was unaware of clinical outcome. This software used a least-square fitting method to identify T-wave offset from the intersection of the maximal slope of the terminal T-wave with a threshold defined by the T-P segment.\textsuperscript{17,18} This approach has superior reproducibility compared with other automated methods of T-offset determination, with a mean difference of only 7.6 ms for QTD measurements made serially at 30 minutes, 1 day, 1 week, and 1 month after baseline study.\textsuperscript{17,18} Similarly, the mean absolute differences between computer measurements and careful manual measurement of QT interval by electronic calipers in our laboratory was only 6 ms.\textsuperscript{4} Leads with high noise levels (standard deviation of T-P segment signal divided by T-wave amplitude <0.7), flat T waves (T-wave amplitude <60 $\mu$V), and T waves with unidentifiable patterns were excluded from T-wave offset determinations.\textsuperscript{21}

Because the dispersion of QRS onset across all 12 leads is much smaller than the dispersion of T end,\textsuperscript{22} a global QRS onset of 12 leads was used for measuring QT intervals. QT intervals were measured in all 12 leads and corrected for heart rate (QTc) with Bazett’s formula.\textsuperscript{23} QTc was calculated as the difference between maximal and minimal QTc intervals. Only the 1839 participants with ≥6 total leads (mean, 9.7±0.6) and ≥3 precordial leads (mean, 4.9±0.2) with measurable QT intervals were included in the study.

**Clinical Evaluation**

All participants underwent a personal interview, including the Rose questionnaire,\textsuperscript{24} physical examination, and fasting blood and urine sampling as previously reported.\textsuperscript{19} Participants were categorized as having definite or possible coronary heart disease (CHD) on the basis of clinical and ECG evidence of coronary disease or myocardial infarction and were classified as diabetic as previously reported.\textsuperscript{25}

**Definition and Determination of Clinical End Points**

For survival analyses, observation began on the date of ECG recording. Deaths were identified in an ongoing manner from sources in each community and through annual follow-up of each participant and were verified through death certificates and medical records. Deaths were classified as cardiovascular if caused by myocardial infarction, stroke, sudden death resulting from CHD, or congestive heart failure as previously defined\textsuperscript{26-27} by an independent review panel of physicians who were unaware of QT interval or QTD findings.

**Data and Statistical Analyses**

Data were stored and analyzed with SPSS, release 7.5 (SPSS Inc). Mean values were compared between groups by use of 2-way ANOVA to adjust for possible differences between study centers (Arizona, Oklahoma, and the Dakotas). Proportions were compared by use of $\chi^2$ tests. Mortality rates were calculated by the product-limit method and were plotted according to the Kaplan-Meier method,\textsuperscript{28} with comparisons of death rates between groups performed with the log-rank test. Mortality analyses were performed for both continuous and discrete variables by fitting Cox proportional-hazards models to the data after stratification by center.\textsuperscript{29} With the proportional-hazards models, the estimated relative risk of the incidence of death for positive compared with negative test outcomes was computed as the antilog of the estimated coefficient corresponding to the dichotomous variable.\textsuperscript{30} For continuous variables, the comparison in relative risk was computed for a 1-SD-of-the-mean increase as the antilog of the estimated coefficients times the SD. The 95% CI of each relative risk was calculated from estimated coefficients and their standard errors\textsuperscript{31} and Wald $\chi^2$ statistics, and probability values were calculated. To test the independence of QTc and QTD as predictors of mortality, multivariate Cox models were used, including age, sex, body mass index (BMI), diabetes, diastolic and systolic blood pressures, HDL and LDL cholesterol, albuminuria, alcohol use, history of smoking or prevalent CHD, and tribal center. For all tests, a 2-tailed $P<0.05$ was required to reject the null hypotheses that there was no difference in mortality according to QT interval or QTD findings.

**Results**

**Patient Characteristics**

After a mean follow-up of 3.7±0.9 years, there were 188 deaths from all causes and 55 cardiovascular deaths. Clinical characteristics of survivors, those who died of any cause, and participants with and without cardiovascular death are compared in Table 1. The 188 participants who died were older; had higher diastolic blood pressure, more albuminuria, a greater prevalence of diabetes and possible or definite prevalent CHD, lower BMIs, and lower LDL cholesterol levels; but did not differ in sex or smoking status compared with those who survived. The 55 participants who suffered a cardiovascular death were similarly older; had higher systolic blood pressure, higher LDL cholesterol, more albuminuria, and greater prevalence of diabetes, possible or definite CHD, and current smoking; but also had lower HDL cholesterol than participants who had not died from a cardiovascular cause.

The relation of QTc interval and QTD to clinical outcome is also shown in Table 1. Participants who died had significantly longer QTc intervals and greater QTD than those who survived, with the greatest increase in QTD in those who died of cardiovascular causes.

**QT Interval Prediction of Mortality**

In Cox analyses adjusting for possible differences between centers, QTc was a significant predictor of all-cause mortality ($\chi^2=53.0, P<0.0001$) and cardiovascular mortality ($\chi^2=14.7,$
Assessment of the Bazett-corrected QT interval as a continuous variable revealed increases of 20% to 48% in all-cause and cardiovascular mortality per 1-SD increase in QTc across the entire range from low-normal to elevated QTc (Table 2). When participants were divided into groups on the basis of a QTc partition of 460 ms, the 189 participants (10.3%) with QTc >460 ms had a significantly greater mortality from all causes by Kaplan-Meier analysis (Figure 1), with a 2.6-fold increased risk of death (hazard ratio, 2.6; 95% CI, 1.8 to 3.7). The actuarial 5-year mortality was 37% among participants with prolonged QTc and only 5% among those with lower QTc intervals.

After multivariate adjustment for age, sex, BMI, diabetes, diastolic and systolic blood pressures, HDL and LDL cholesterol, triglycerides, albuminuria, alcohol use, history of smoking or prevalent CHD, and study center, QTc considered as a continuous variable remained a strong, significant predictor of all-cause mortality ($\chi^2 = 16.5, P < 0.0001$) and a significant but statistically weaker predictor of cardiovascular mortality ($\chi^2 = 5.8, P = 0.016$). After these potential predictors of mortality were controlled for, the risk of cardiac and all-cause mortality increased 35% per SD of QTc across the range from low to elevated QTc (Table 3). After risk factors were controlled for, a QTc >460 ms was associated with a 2-fold increase in mortality.

### TABLE 1. Clinical Characteristics, QT Interval, and QTD Measurements in Participants According to Survival Status*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Survivors (n=1651)</th>
<th>All-Cause Death (n=188)</th>
<th>$P$</th>
<th>Survivors and Non-CVD Death (n=1784)</th>
<th>CVD Death (n=55)</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>55±8</td>
<td>59±8</td>
<td>&lt;0.001</td>
<td>55±8</td>
<td>60±9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Sex, % male</td>
<td>43</td>
<td>49</td>
<td>0.104</td>
<td>43</td>
<td>55</td>
<td>0.097</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>30.7±6.2</td>
<td>29.0±6.6</td>
<td>0.006</td>
<td>30.6±6.3</td>
<td>29.3±5.2</td>
<td>0.199</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>78±11</td>
<td>76±12</td>
<td>0.038</td>
<td>77±11</td>
<td>79±12</td>
<td>0.304</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>127±20</td>
<td>132±25</td>
<td>0.082</td>
<td>127±20</td>
<td>139±26</td>
<td>0.002</td>
</tr>
<tr>
<td>HDL cholesterol, mg/dL</td>
<td>45±13</td>
<td>46±17</td>
<td>0.151</td>
<td>45±13</td>
<td>41±13</td>
<td>0.019</td>
</tr>
<tr>
<td>LDL cholesterol, mg/dL</td>
<td>117±32</td>
<td>107±41</td>
<td>0.001</td>
<td>115±33</td>
<td>128±51</td>
<td>0.002</td>
</tr>
<tr>
<td>Triglycerides, mg/dL</td>
<td>147±131</td>
<td>147±155</td>
<td>0.980</td>
<td>147±129</td>
<td>178±243</td>
<td>0.081</td>
</tr>
<tr>
<td>Albuminuria, mg/g</td>
<td>2.7±2.0</td>
<td>4.0±2.6</td>
<td>&lt;0.001</td>
<td>2.7±2.1</td>
<td>4.8±2.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diabetes or IGT, %</td>
<td>60</td>
<td>75</td>
<td>&lt;0.001</td>
<td>61</td>
<td>80</td>
<td>0.004</td>
</tr>
<tr>
<td>Prevalent CHD, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>None</td>
<td>83</td>
<td>69</td>
<td></td>
<td>82</td>
<td>62</td>
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<tr>
<td>Possible</td>
<td>15</td>
<td>24</td>
<td></td>
<td>16</td>
<td>27</td>
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<td>Definite</td>
<td>2</td>
<td>7</td>
<td></td>
<td>2</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>Smoking, %</td>
<td></td>
<td></td>
<td>0.655</td>
<td>0.026</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>31</td>
<td>27</td>
<td></td>
<td>31</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Previous</td>
<td>34</td>
<td>35</td>
<td></td>
<td>34</td>
<td>35</td>
<td></td>
</tr>
<tr>
<td>Current</td>
<td>35</td>
<td>38</td>
<td></td>
<td>35</td>
<td>45</td>
<td></td>
</tr>
<tr>
<td>QTc, ms/s(^{1/2})</td>
<td>429±23</td>
<td>441±28</td>
<td>&lt;0.001</td>
<td>430±24</td>
<td>440±28</td>
<td>0.002</td>
</tr>
<tr>
<td>QTD, ms</td>
<td>21±16</td>
<td>26±22</td>
<td>0.020</td>
<td>21±16</td>
<td>33±31</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

CVD indicates cardiovascular disease; BP, blood pressure; and IGT, impaired glucose tolerance.

*Adjusted for differences between tribal centers with 2-way ANOVA.

### TABLE 2. Center-Adjusted Cox Proportional-Hazards Models for Prediction of All-Cause and Cardiovascular Disease Mortality Examining QTc and QTD as Continuous Variables*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Comparison, ms†</th>
<th>Hazard Ratio</th>
<th>95% CI</th>
<th>$\chi^2$</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prediction of all-cause mortality</td>
<td>QTc 24</td>
<td>1.48</td>
<td>1.33–1.64</td>
<td>53.0</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td>QTD 17</td>
<td>1.20</td>
<td>1.08–1.35</td>
<td>11.3</td>
<td>0.0008</td>
</tr>
<tr>
<td>Prediction of cardiovascular disease mortality</td>
<td>QTc 24</td>
<td>1.47</td>
<td>1.20–1.79</td>
<td>14.7</td>
<td>0.0001</td>
</tr>
<tr>
<td></td>
<td>QTD 17</td>
<td>1.45</td>
<td>1.26–1.67</td>
<td>26.5</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

*Adjusted for possible center effects.

†Relative risks calculated for a 1-SD increase in the mean.
(95% CI, 1.4 to 3.0) increased risk of death and a 2.1-fold (95% CI, 1.0 to 4.4) higher risk of cardiovascular death.

QTD and Mortality
In Cox analyses that adjusted for center, QTD was a significant predictor of all-cause mortality ($\chi^2=11.3, P=0.0008$) and cardiovascular mortality ($\chi^2=26.5, P<0.0001$). QTD as a continuous variable was associated with 20% and 45% higher all-cause and cardiovascular mortality per SD of QTD (Table 2). When participants were divided into groups by use of a QTD partition of 58 ms (the upper 98th percentile in a separate group of normal subjects with 97% specificity in a normal subset of the current population), QTD $>58$ ms was present in 76 participants (4.1%) and was associated with significantly greater all-cause mortality by Kaplan-Meier analysis (Figure 2), with an $\approx$2-fold (hazard ratio, 1.9; 95% CI, 1.1 to 3.3) increased risk of death. Actuarial 5-year mortality was 32% among participants with abnormal QTD and 18% among those with QTD $\leq 58$ ms. When cardiovascular mortality only was examined (Figure 2), QTD $>58$ ms was associated with a 3.4-fold (95% CI, 1.5 to 7.5) increased risk of cardiovascular death. Actuarial 5-year cardiovascular mortality was 18% among participants with QTD $>58$ ms and only 5% among those with more normal QTD.

Multivariate Cox analyses (Table 3) demonstrated that after adjustment for other potential predictors of mortality,

**Table 3. Multivariate Cox Proportional-Hazards Model for Prediction of All-Cause and Cardiovascular Disease Mortality Examining QTc and QTD as Continuous Variables**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Comparison, ms†</th>
<th>Hazard Ratio</th>
<th>95% CI</th>
<th>$\chi^2$</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prediction of all-cause mortality</td>
<td>QTc 24</td>
<td>1.35</td>
<td>1.17–1.56</td>
<td>16.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>QTD 17</td>
<td>1.10</td>
<td>0.96–1.26</td>
<td>1.3</td>
<td>0.25</td>
<td></td>
</tr>
<tr>
<td>Prediction of cardiovascular disease mortality</td>
<td>QTc 24</td>
<td>1.35</td>
<td>1.02–1.78</td>
<td>5.8</td>
<td>0.016</td>
</tr>
<tr>
<td>QTD 17</td>
<td>1.34</td>
<td>1.13–1.59</td>
<td>12.5</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
</tbody>
</table>

*Adjusted for age, sex, BMI, diabetes, diastolic and systolic blood pressures, HDL and LDL cholesterol levels, triglyceride level, albuminuria, alcohol use, history of smoking or prevalent CHD, and study center.
†Relative risks calculated for a 1-SD increase in the mean.
QTD considered as a continuous variable remained a strong, significant predictor of cardiovascular mortality ($\chi^2=12.5$, $P<0.0001$) but was no longer an independent predictor of all-cause mortality ($\chi^2=1.3$, $P=0.25$). After these risk factors were controlled for, QTD $>58$ ms was associated with a 2.8-fold (95% CI, 1.1 to 6.6) increased risk of cardiovascular death. When both QTc and QTD were entered with other risk factors into the Cox multivariate regression analysis for prediction of cardiovascular death, both QTc ($\chi^2=10$, $P=0.0012$) and QTD ($\chi^2=4$, $P=0.05$) remained significant independent predictors of cardiovascular mortality.

Discussion
The present findings demonstrate the predictive value of the QT interval and QTD measured by computerized ECG for noninvasive risk stratification in a population-based sample. After adjustment for multiple known predictors of adverse outcome, QT interval corrected for heart rate remained associated with both all-cause and cardiovascular mortality and increased QTD remained a significant predictor of cardiovascular mortality. This additive risk prediction suggests that increased QTc and QTD on the surface ECG reflect different aspects of abnormal ventricular repolarization.

QT Dispersion
Ventricular repolarization is a complex process occurring nonuniformly in space and time, with the ST segment and T wave on the surface ECG reflecting an integrated signal from multiple repolarization wave fronts. Although simple QTD on the surface ECG provides only an imperfect estimate of the degree of heterogeneity of repolarization, several studies have suggested that QTD helps identify patients at increased risk of ventricular arrhythmias or clinical events in a variety of clinical settings. However, the value of QTD in risk stratification has not been uniform.

Two recent studies highlight these conflicting findings on the predictive value of QTD. de Bruyne et al demonstrated that increased computer-measured QTD on digitally acquired ECGs predicted all-cause and cardiovascular mortality after adjustment for age, prior myocardial infarction, and overall QT interval duration in 5812 men and women $\geq5$5 years of age in the Rotterdam Study of the elderly. In contrast, Zabel et al found no relation between QTd (or other ECG measures of dispersion of repolarization such as the T-peak to T-end interval and T-wave area) and adverse outcome in a prospective study of 280 patients after myocardial infarction that used a custom computer program to measure QT intervals from optically scanned analog ECGs. The present study extends the application of QTD by demonstrating in a population-based sample of middle-aged to elderly adults with a high rate of mortality (10% over 3.7 years starting from a mean of 55 years of age) that increased QTD at baseline conveys significant additional risk for cardiovascular death after accounting for age, sex, and multiple cardiac risk factors, independent of the impact of QT prolongation.

QT Interval
The QT interval on the surface ECG reflects a summation of the net potential differences during ventricular depolarization and repolarization, with the end of the T wave closely approximating the longest duration of ventricular repolarization. QT interval prolongation has been implicated in the origin of ventricular arrhythmias, possibly because of less uniform recovery of ventricular excitability in the setting of regional differences in cardiac sympathetic nervous system activity. However, similar to QTD, the value of increased QT interval for risk stratification has been variable both in patients after myocardial infarction and in population-based studies. The present study demonstrated that an increased QTc, considered as either a continuous variable or by identifying durations exceeding a discrete partition value, predicted increased risk of all-cause and cardiovascular mortality even when numerous other risk factors and increased QTD are taken into account.

Although the increased risk of cardiovascular death in participants with prolonged QTc can be interpreted as reflecting the known associations of increased QTc with both underlying cardiovascular disease and an increased risk of ventricular arrhythmias, the ability of a prolonged QT interval to predict all-cause mortality is less readily explained. It is possible that an increased QTc is a marker of underlying disorders such as diabetes and renal dysfunction with an increased risk of early death, in which electrolyte abnormalities and/or medications could contribute to QT prolongation. On the other hand, QT prolongation might also reflect profound alterations in neurohumoral balance that could predispose to an increased risk of mortality.

Study Limitations and Implications
This study and previous investigations are affected by fundamental limitations in the use of QT interval and QTD measurements. However, the present study more completely addresses these limitations than have previous studies. First, the accuracy and reproducibility of QT interval and dispersion measurements have been limited by difficulties with reliable identification of T-wave offset. However, the computerized method used to determine T-wave offset in the present study has greater reproducibility than manual measurements or other computer-based methods. Second, although the partition values used for QTc (460 ms) and QTD (58 ms) are somewhat arbitrary, they correspond to the 97th percentile values in the apparently normal subset of the present population. Additionally, risk stratification by both QTc and QTD was statistically significant when these criteria were examined as continuous variables (Tables 2 and 3), suggesting that risk increases with increasing values of these measures and is not dependent on the choice of specific partition values. Moreover, risk stratification remained statistically significant even if alternative thresholds of 50 ms for QTD and 440 ms for QTc were used, further suggesting that the overall predictive value of these measures in not strongly dependent on the precise partition values selected. Indeed, the relative mortality risk was >2-fold higher for our partition values, even after adjustment for other potential risk factors, highlighting the potential clinical significance of these findings for identifying patients in the general population who are at increased risk of death. Further study is necessary to
address the predictive value of changes in QT interval and QTD over time.

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References
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