QTc Prolongation Measured by Standard
12-Lead Electrocardiography Is an
Independent Risk Factor for Sudden
Death Due to Cardiac Arrest

Ale Algra, MD, PhD; Jan G.P. Tijssen, PhD; Jos R.T.C. Roelandt, MD, PhD, FACC, FESC;
Jan Pool, MD, PhD; and Jacobus Lubsen, PhD, FESC

Background. QTc prolongation has been implicated as a risk factor for sudden death; however, a
controversy exists over its significance.

Methods and Results. In the Rotterdam QT Project, 6,693 consecutive patients who underwent
24-hour ambulatory electrocardiography were followed up for 2 years; of these, 245 patients
died suddenly. A standard 12-lead electrocardiogram and clinical data at the time of 24-hour
ambulatory electrocardiography were collected for all patients who died suddenly and for a
random sample of 467 patients from the study cohort. In all patients without an intraventric-
ular conduction defect (176 patients who died suddenly and 390 patients from the sample), QT
interval duration was measured in leads I, II, and III and corrected for heart rate with Bazett’s
formula (QTc). In patients without evidence of cardiac dysfunction (history of symptoms of
pump failure or an ejection fraction <40%), QTc of more than 440 msec was associated with
a 2.3 times higher risk for sudden death compared with a QTc of 440 msec or less (95%
confidence interval: 1.4, 3.9). In contrast, in patients with evidence of cardiac dysfunction, the
relative risk of QTc prolongation was 1.0 (0.5, 1.9). Adjustment for age, gender, history of
myocardial infarction, heart rate, and the use of drugs did not alter these relative risks.

Conclusions. These data indicate that in patients without intraventricular conduction defects
and cardiac dysfunction, QTc prolongation measured from the standard electrocardiogram is
a risk factor for sudden death independent of age, history of myocardial infarction, heart rate,
and drug use. In patients with cardiac dysfunction, QTc duration is not related to the risk for
sudden death. (Circulation 1991;83:1888–1894)

Prolongation of the QT interval corrected for
heart rate (QTc) as measured in the standard
12-lead electrocardiogram is associated
with an increased incidence of sudden death due to
cardiac arrest. This relation is well known in patients
with the congenital long QT syndromes, in which
case sudden death is due to an increased susceptibil-
ity for ventricular fibrillation. In 1978, Schwartz and
Wolf first reported on this relation in patients after
myocardial infarction; they found a relative risk of
2.16 for sudden death by QTc prolongation. Observa-
tions by Schwartz and Wolf were confirmed by
some investigators but were denied by others.

In a review of the literature, Surawicz et al concluded that “QTc has little, if any, predictive
value after myocardial infarction,” whereas in another re-
view, Locati and Schwartz stated that “QTc represents
a simple and valid method of identifying patients
at high risk of sudden death.” Apparently, the
controversy still exists over the significance of QTc
prolongation as a risk factor in patients after myo-
cardial infarction. Furthermore, little is known about
the significance of QTc duration in other patient
groups.

In the Rotterdam QT Project, a cohort of 6,693
consecutive patients who underwent 24-hour ambula-
tory electrocardiography was followed up during a
2-year period for the occurrence of sudden death due to cardiac arrest. The QT Project aims to assess RR and QTc variability by 24-hour electrocardiography and their relation to sudden death due to cardiac arrest. The purpose of this study is to report on the significance of sudden death in relation to QTc duration as measured in the standard 12-lead electrocardiogram recorded at the time of 24-hour electrocardiography.

Methods

Study Cohort

The 6,693 consecutive patients who had undergone 24-hour electrocardiography in one of the four participating Rotterdam hospitals between August 1, 1980, and December 31, 1984, were included in this study. The various indications for 24-hour electrocardiography were to evaluate complaints (palpitations, dizziness, syncope, and angina) (65%), the effect of therapy (8%), the risk after myocardial infarction (10%), or a cardiac cause in transient ischemic attack or stroke (7%). Most 24-hour electrocardiograms were obtained at the outpatient clinic (75%).

Follow-up

All patients were followed up for mortality until 2 years after 24-hour ambulatory electrocardiography. Follow-up was complete in 99.5% of the patients; 716 patients had died (10.7%). Cause and circumstances of death were determined from the records of general physicians and hospitals.

Patients were considered to have died suddenly if death was observed and had occurred within 1 hour after new or more serious complaints and if its likely cause was cardiovascular. Also, patients who unexpectedly died during sleep or died while unobserved were considered to have died suddenly if circumstantial evidence pointed to sudden death from cardiovascular causes. All cases of sudden death were independently verified by two senior cardiologists. Thus, a total of 245 cases of sudden death were identified, of which 66 (27%) cases occurred in the hospital.

Collection of Baseline Data

Baseline characteristics were retrospectively collected for all patients who died suddenly and for a random sample of 467 patients from the complete study cohort (including 21 cases of sudden death). Information on the following patient characteristics at the time of 24-hour electrocardiography was collected from the medical records: known cardiovascular risk indicators (cholesterol level, blood pressure, and smoking), cardiovascular history (angina, myocardial infarction, cardiac dysfunction, and surgery), cardiovascular function tests if available, routine laboratory studies, and current drug use. Evidence of cardiac dysfunction was considered to be present if there had been a history of symptoms of pump failure (e.g., shortness of breath or treatment with digitalis) or an ejection fraction less than 40% at cineangiographic or radionuclide ventriculography.

A resting 12-lead electrocardiogram that was recorded at the time of 24-hour ambulatory electrocardiography was available in all but 24 patients. Three channel recorders at a paper speed of 25 mm/sec had been used. In each electrocardiogram, a single QT measurement was obtained. The beginning of the QT interval was taken as the earliest onset of the QRS complex in the three synchronous standard leads I, II, and III. The end of the QT interval was taken as the last point of the T wave over leads I, II, and III, where the downsloping limb joined the baseline, while we excluded U waves. QT was corrected for heart rate according to Bazett’s formula: QTc=QT/√RR. No QTc measurement was obtained in the 100 patients with an intraventricular conduction defect (Minnesota code 7) and in the 18 patients in whom no adequate RR interval was available (because of premature ventricular complexes or atrial fibrillation). Hence, QTc measurements were available in 176 patients who died suddenly and in 390 patients of the random sample. All measurements were obtained by one of the investigators (A.A.) without knowledge of the survival status of the patient. A QTc interval of more than 440 msec was considered to be prolonged.

Data Analysis

The effect of QTc prolongation on determining the risk for sudden death was expressed as the relative risk, which is equal to the risk for sudden death among patients with QTc prolongation divided by that among patients without QTc prolongation. The risk for sudden death among patients with QTc prolongation was obtained as the number of sudden deaths with QTc prolongation divided by the (estimated) total number of patients with QTc prolongation. The latter was obtained by extrapolating the QTc findings in the sample to the whole study cohort; for example, the total number of patients with QTc prolongation was obtained by multiplying the number of patients with QTc prolongation in the sample by 6,693/467, which was the inverse of the sampling fraction. The risk for sudden death among patients without QTc prolongation was obtained analogously. The 95% confidence interval of the odds ratio was used and was calculated from the data laid out for a case-control study.

We used stratified analysis and logistic regression to assess the influences of independent risk factors of sudden death unequally distributed between patients with and without QTc prolongation. Statistical difference between relative risks of subgroups was determined with use of the coefficient and its standard deviation of the interaction term of QTc duration and the subgroup factor in the logistic model.

Results

The distribution of baseline characteristics in relation to QTc duration in the 390 patients from the
random sample without intraventricular conduction defects is shown in Table 1. Female gender, advanced age, and evidence of cardiac dysfunction were preponderant characteristics among the patients with QTc prolongation.

In Table 2 the occurrence of sudden death in patients with prolonged QTc and in those with normal QTc is shown. QTc prolongation doubled the risk for sudden death. There was a marked difference between patients with and without evidence of cardiac dysfunction: If present, the relative risk was 1.0; if absent, it was 2.3. Because of the clinical significance of this difference, which was also statistically significant \(p = 0.038\), the data of both groups were analyzed separately.

The lower part of Table 2 shows the occurrence of sudden death in relation to QTc duration for patients without evidence of cardiac dysfunction subdivided according to age, gender, and history of myocardial infarction. QTc prolongation carried a higher risk for sudden death in men than in women; this difference was not statistically different \(p = 0.17\). The relative risk for sudden death of QTc prolongation was independent of age and previous myocardial infarction. Also, the relative risk was independent of the history of angina, arrhythmias, and the use of the drugs mentioned in Table 1 (not shown in Table 2).

Adjustment for a host of baseline characteristics, including the unequally distributed use of diuretics and antiarrhythmic drugs (Table 1), by stratified analysis and logistic regression hardly altered the relative risk estimates, in patients with and without evidence of cardiac dysfunction.

### Discussion

**Results of the Present Study**

We observed a substantial difference in the relative risk of QTc prolongation for sudden death between patients with and without evidence of cardiac dysfunction: In the absence of cardiac dysfunction, the relative risk was 2.3; in the presence of cardiac dysfunction, it was 1.0 (Table 2). Because of this difference, which was statistically significant, we considered the two groups as two different entities. Although patients with a history of symptoms of pump failure are not the same as those with an ejection fraction of less than 40%, the relative risk in both groups was approximately 1.0. Therefore, we grouped them together. A possible explanation for the different relative risks may be that the electrophysiological properties of hearts in patients with cardiac dysfunction may be different from those in patients without cardiac dysfunction. Of note, the patients with evidence of cardiac dysfunction had a higher risk for sudden death than did the patients without dysfunction (the relative risk of 4.5 of cardiac dysfunction for sudden death can be calculated from the data in Table 2): QTc prolongation did not further increase the risk for sudden death. Neural compensating mechanisms active during cardiac dysfunction possibly preclude the expression of QTc prolongation in the form of ventricular fibrillation. In patients without evidence of cardiac dysfunction, the relative risk was independent of a history of myocardial infarction, the use of any drug, or any other important clinical variable except gender. We are not aware of any pathophysiological explanation for the intriguing difference between women and men. Also, heart rate (74 ± 18 beats/min [mean ± SD] in the 176 patients who died suddenly and 72 ± 16 beats/min in the 390 patients from the random sample) did not influence the relative risk, although Bazett’s calculation of QTc is not always considered to be adequate.\(^22\) However, no other formula has been proven to be superior to that of Bazett.\(^23\)

The data in Table 1 suggest several incomparabilities between patients with and without QTc prol-
gation, for example, the use of antiarrhythmic drugs. However, relative risks adjusted for these differences were essentially the same.

**Study Design**

Our study was primarily designed for the investigation of the relation between QTc and heart rate variability as measured in the 24-hour electrocardiogram and the risk for sudden death. The setting of this project, however, provided an opportunity to study the relation between the QTc as measured in the standard 12-lead electrocardiogram and sudden death.

The study population was heterogeneous: 31% had had a myocardial infarction, 15% had evidence of cardiac dysfunction, 16% had had a transient ischemic attack or a stroke, and 80% used one or more drugs (Table 1). The heterogeneity of the population and the size of the project offered the opportunity to study the relation between QTc prolongation and sudden death in several subgroups.

For the present study, a nested case-referent design was chosen because of efficiency considerations. Although baseline characteristics were collected only for a small subset of patients, the essential findings were produced with the same level of precision as in the conventional follow-up study. We calculated a 95% confidence interval (based on the actually observed data) that ranged from 1.4 to 3.1 for the relative risk of QTc prolongation for sudden death. If we had measured the QTc interval in all 6,693 patients and excluded patients with an intraventricular conduction defect (i.e., if we had actually observed the upper portion of Table 2), the confidence interval would have ranged from 1.5 to 2.9.

**Comparison With Other Studies**

The results of earlier studies on the relation between QTc prolongation and sudden death are summarized in Table 3. The data from the original reports were rearranged to allow a uniform presen-

### Table 2. Sudden Death in Patients Without Intraventricular Conduction Defects in Relation to QTc Prolongation and Evidence of Cardiac Dysfunction, Age, Sex, and History of Myocardial Infarction

<table>
<thead>
<tr>
<th>QTc (msec)</th>
<th>Sudden death (n)</th>
<th>Estimated total* (n)</th>
<th>% Sudden death</th>
<th>RR†</th>
<th>95% CI‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients</td>
<td>&lt;440</td>
<td>111</td>
<td>4,357</td>
<td>2.5</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>≥440</td>
<td>65</td>
<td>1,233</td>
<td>5.3</td>
<td>2.1</td>
</tr>
<tr>
<td>Patients with evidence of cardiac dysfunction</td>
<td>&lt;440</td>
<td>47</td>
<td>516</td>
<td>9.1</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>≥440</td>
<td>30</td>
<td>315</td>
<td>9.5</td>
<td>1.0</td>
</tr>
<tr>
<td>Patients without evidence of cardiac dysfunction</td>
<td>&lt;440</td>
<td>64</td>
<td>3,841</td>
<td>1.7</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>≥440</td>
<td>35</td>
<td>917</td>
<td>3.8</td>
<td>2.3</td>
</tr>
<tr>
<td>Patients without evidence of cardiac dysfunction (age, ≤60 yr)</td>
<td>&lt;440</td>
<td>16</td>
<td>2,307</td>
<td>0.7</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>≥440</td>
<td>7</td>
<td>416</td>
<td>1.7</td>
<td>2.4</td>
</tr>
<tr>
<td>Patients without evidence of cardiac dysfunction (age, &gt;60 yr)</td>
<td>&lt;440</td>
<td>48</td>
<td>1,534</td>
<td>3.1</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>≥440</td>
<td>28</td>
<td>502</td>
<td>5.6</td>
<td>1.8</td>
</tr>
<tr>
<td>Patients without evidence of cardiac dysfunction (women)</td>
<td>&lt;440</td>
<td>17</td>
<td>1,548</td>
<td>1.1</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>≥440</td>
<td>10</td>
<td>545</td>
<td>1.8</td>
<td>1.7</td>
</tr>
<tr>
<td>Patients without evidence of cardiac dysfunction (men)</td>
<td>&lt;440</td>
<td>47</td>
<td>2,293</td>
<td>2.0</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>≥440</td>
<td>25</td>
<td>373</td>
<td>6.7</td>
<td>3.3</td>
</tr>
<tr>
<td>Patients without evidence of cardiac dysfunction (with history of MI)</td>
<td>&lt;440</td>
<td>34</td>
<td>975</td>
<td>3.5</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>≥440</td>
<td>19</td>
<td>272</td>
<td>7.0</td>
<td>2.0</td>
</tr>
<tr>
<td>Patients without evidence of cardiac dysfunction (without history of MI)</td>
<td>&lt;440</td>
<td>30</td>
<td>2,866</td>
<td>1.1</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>≥440</td>
<td>16</td>
<td>645</td>
<td>2.5</td>
<td>2.4</td>
</tr>
</tbody>
</table>

*Estimated total, estimated total number of patients; % Sudden death, 2-year sudden death rate; RR, relative risk; CI, confidence interval; MI, myocardial infarction.
†Calculated as the ratio of sudden death rates in which the rate of the category indicated by a missing RR was taken as reference; ‡Calculated as the 95% CI for the odds ratio.
TABLE 3. Comparison of Previously Published Studies and This Study

<table>
<thead>
<tr>
<th>Investigators</th>
<th>Source</th>
<th>At risk (n)</th>
<th>Follow-up (yr)</th>
<th>Type of case*</th>
<th>QTc≥440 msec</th>
<th>QTc&lt;440 msec</th>
<th>RR†</th>
<th>95% CI</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schwartz and Wolf*</td>
<td>Post-MI</td>
<td>55</td>
<td>7</td>
<td>Sudden death</td>
<td>76</td>
<td>16/21</td>
<td>35</td>
<td>12/34</td>
<td>2.2</td>
</tr>
<tr>
<td>Boudoulas*</td>
<td>Post-MI</td>
<td>100</td>
<td>3.6</td>
<td>Cardiac death</td>
<td>31</td>
<td>4/13</td>
<td>18</td>
<td>16/87</td>
<td>1.7</td>
</tr>
<tr>
<td>Ahnve et al†</td>
<td>Post-MI</td>
<td>214</td>
<td>1</td>
<td>Cardiac death</td>
<td>23</td>
<td>10/43</td>
<td>2</td>
<td>3/17</td>
<td>13.3</td>
</tr>
<tr>
<td>Peters et al‡</td>
<td>Post-MI</td>
<td>3,692</td>
<td>2.1</td>
<td>Sudden death</td>
<td>6.6±41/619</td>
<td>3.7</td>
<td>46/1,230</td>
<td>1.8</td>
<td>1.2, 2.7</td>
</tr>
<tr>
<td>Juul-Möller§</td>
<td>Post-MI</td>
<td>96</td>
<td>1</td>
<td>Cardiac death</td>
<td>50±9/18</td>
<td>9</td>
<td>7/78</td>
<td>5.6</td>
<td>2.4, 13.0</td>
</tr>
<tr>
<td>Pohjola-Sintonen et al†</td>
<td>Post-MI</td>
<td>457</td>
<td>4</td>
<td>All death</td>
<td>30±31/104</td>
<td>22</td>
<td>79/353</td>
<td>1.3</td>
<td>0.9, 1.9</td>
</tr>
<tr>
<td>Puddu and Bourassa¹</td>
<td>Proven CAD at angiography</td>
<td>1,157</td>
<td>3.8</td>
<td>Sudden death</td>
<td>12±29/233$§</td>
<td>7</td>
<td>69/924</td>
<td>1.7</td>
<td>1.0, 2.9</td>
</tr>
<tr>
<td>Fioretti¹²</td>
<td>Post-MI</td>
<td>474</td>
<td>1</td>
<td>All death</td>
<td>13.8±20/125</td>
<td>7.6</td>
<td>25/304</td>
<td>1.8</td>
<td>1.0, 3.2</td>
</tr>
<tr>
<td>Algra et al (this study)</td>
<td>24-hr ECG</td>
<td>5,589</td>
<td>2</td>
<td>Sudden death</td>
<td>3.8±35/917</td>
<td>1.7</td>
<td>64/3,841</td>
<td>2.3</td>
<td>1.4, 3.9</td>
</tr>
<tr>
<td></td>
<td>candidates</td>
<td></td>
<td></td>
<td></td>
<td>9.5±30/315</td>
<td>9.1</td>
<td>47/516</td>
<td>1.0</td>
<td>0.5, 1.9</td>
</tr>
</tbody>
</table>

Upper part of the table shows death rates in patients with and without QTc prolongation, the corresponding relative risks, and 95% confidence intervals; lower part shows mean QTc values and standard deviations (msec) for deaths and survivors and corresponding difference and 95% confidence interval.

RR, relative risk; CI, confidence interval; MI, myocardial infarction; CAD, coronary artery disease; CD, cardiac dysfunction; ΔQTc, QTc in deaths−QTc in survivors.

*Death within a maximum of 24 hours after onset of complaints; †relative risks are calculated from the actual numbers on which the investigators based their conclusions; §QTc≥450 msec; ¶QTc>480 msec; ‡QTc<480 msec; †‡denominators estimated from sample of survivors; **estimated from standard deviations presented.

The upper part of the table shows death rates in patients with and without QTc prolongation with corresponding relative risks and 95% confidence intervals; the lower part shows mean QTc values and standard deviations for patients that died and survived with the corresponding differences and 95% confidence intervals. Because in the last four studies, data on the number of patients with a prolongation and a normal QTc were not published, relative risks as presented in the upper part of the table could not be calculated. All studies were confined to patients after myocardial infarction with the exception of that of Puddu and Bourassa,¹⁰ who used patients with angiographically proven coronary artery disease (Table 3).

Some investigators reported on death from all causes; some reported on cardiac death; and Ahnve et al¹¹ reported on major cardiac events (sudden death, circulatory standstill, and reinfarction). Some studies were not primarily designed to assess the relation between QTc prolongation and sudden death; hence, sudden death rates were not reported in all instances. The use of death from all causes dilutes the relation between QTc prolongation and outcome as is illustrated by the Beta Blocker Heart Attack Trial, in which the relative risk of 1.8 (1.3, 2.5) for sudden death was attenuated to 1.6 (1.3, 2.0) when death from all causes was used, and as illustrated by the weak associations reported in the studies by Pohjola-Sintonen et al¹³ and Ahnve et al.¹⁴

Several investigative groups¹³⁻¹⁷ have concluded that their studies did not confirm the observations in the original report of Schwartz and Wolf.⁶ However, in all studies, the observed relative risk or QTc difference points to an increased risk for (sudden) death with QTc prolongation. The confidence intervals in these studies all include the null value (1 for the relative risk, 0 for the QTc difference) or in the terminology of the original reports: The relation of QTc with the risk for (sudden) death was not statistically significant at the 5% level. Several of the smaller studies were inconclusive when considered separately; however, when taken together, they corroborate the observations of Schwartz and Wolf, a phenomenon often observed in meta-analyses. The relative risk found in our patients without cardiac dysfunction compares well with those summarized in Table 36⁻¹⁰,¹²,¹³,¹⁷ and especially with those from the large Beta Blocker Heart Attack Trial.⁸
Variability of the QTc Interval

Repeated measurements of QTc allow investigators to identify temporary prolongation of QTc intervals, which may be an indication for a (temporarily) increased risk for sudden death. Schwartz and Wolf measured QTc every 2 months during follow-up and found that patients with a high variability of QTc were more likely to die suddenly than were those with low QTc variability. This hypothesis will be further investigated by analysis of the variability of QTc and heart rate in the 24-hour electrocardiograms that led to entry of our patients into the Rotterdam QT Project.

Limitations of the Study

The criteria used for classification of the sudden deaths were pragmatic because information on cause and circumstances of death had to be obtained between 1 and 4 years after death. Actually, more strict criteria can be used in a prospective study because details are not lost with time.

At a paper speed of 25 mm/sec and a measurement accuracy of 0.5 mm, the resulting QT interval measurement error is 20 msec. The measurements were obtained by one reader only who used the original recordings in the patient records. The reader was not aware of the patient survival status; therefore, measurements were not biased. It is well known that measurement errors may attenuate relative risk estimates. Nevertheless, we observed substantial relative risk estimates. We can only speculate how much larger these estimates would have been with perfect measurements.

A cutoff value for QTc prolongation of 440 msec was chosen because of its frequent use in the international literature. Furthermore, the 440-msec cutoff yielded a balanced distribution of sudden deaths over the QTc categories, resulting in a narrow 95% confidence interval of the relative risk estimate. We thus decided to use the 440-msec cutoff to facilitate the comparison with other studies presented in Table 3.

The occurrence of sudden death in the cohort was conditional on the interventions that took place since 24-hour electrocardiography. Therefore, the findings during routine analysis of the 24-hour electrocardiograms may have influenced intervention strategies. This would jeopardize the validity of the estimation of the effect of the QTc interval derived from the standard 12-lead electrocardiogram on sudden death. However, in clinical practice, only extreme QTc prolongation would lead to specific intervention; the other electrocardiographic parameters (heart rate variability and QT interval variability) are not generally used in clinical practice. Thus, the validity of the relative risk estimate is probably not influenced by the findings on the 24-hour electrocardiograms. Nevertheless, when interpreting the results of this study, one should always realize that the findings are conditional on the patient’s therapy because

24-hour electrocardiographic findings, per se, possibly induced certain therapeutic regimens.

Conclusions

The data from our study indicate that in the absence of evidence of cardiac dysfunction, QTc prolongation may double the risk for sudden death. Age, history of myocardial infarction, heart rate, and drug use do not affect the strength of the association. The findings from our study are in accord with those in the literature provided that these are taken together. Furthermore, our study indicates that in patients with cardiac dysfunction the high risk for sudden death is independent of QTc prolongation. Because of the stability of the relative risk over many subgroups, our results apply to a broad population of patients.

Acknowledgments

We thank M.J. Janse, MD, PhD, University of Amsterdam, The Netherlands, for his critical comments; Mr. J.L.H. Le Brun and Mrs. S. van der Does-van der Linden for their great help with the retrieval and coding of the patient records; and the cardiology staff of the University Hospital Rotterdam-Dijkzigt, the Bergwegziekenhuis, the Sint Franciscus Gasthuis, and the Zuiderziekenhuis, Rotterdam for their cooperation.

References


**KEY WORDS** • electrocardiography • sudden death • risk factor • cardiac dysfunction • meta-analysis
QTc prolongation measured by standard 12-lead electrocardiography is an independent risk factor for sudden death due to cardiac arrest.
A Algra, J G Tijssen, J R Roelandt, J Pool and J Lubsen

Circulation. 1991;83:1888-1894
doi: 10.1161/01.CIR.83.6.1888

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/83/6/1888