Reduction in QT Interval Dispersion by Successful Thrombolytic Therapy in Acute Myocardial Infarction

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**Background**

QT dispersion (QT_d equals maximal minus minimal QT interval) on a standard ECG has been shown to reflect regional variations in ventricular repolarization and is significantly greater in patients with than in those without arrhythmic events.

**Methods and Results**

To assess the effect of thrombolytic therapy on QT_d, we studied 244 patients (196 men; mean age, 57±10 years) with acute myocardial infarction (AMI) who were treated with streptokinase (n=115) or anistreplase (n=129) at an average of 2.6 hours after symptom onset. Angiograms at 2.4±1 hours after thrombolytic therapy showed reperfusion (TIMI grade ≥2) in 75% of patients. QT was measured in 10±2 leads at 9±5 days after AMI by using a computerized analysis program interfaced with a digitizer. QT_d, QRS_d, JT_d (QT minus QRS), and JT_d dispersion (JT_d equals maximal minus minimal JT interval) were calculated with a computer. There were significant differences in QT_d (96±31, 88±25, 60±22, and 52±19 milliseconds; P≤.0001) and in JT_d (97±32, 88±31, 63±23, and 58±21 milliseconds; P=.0001) but not in QRS_d (25±10, 22±7, 28±9, and 24±9 milliseconds; P=.24) among perfusion grades 0, 1, 2, and 3, respectively. Similar results were obtained comparing TIMI grades 0/1 with 2/3 and 0/1/2 with 3. Patients with left anterior descending (versus right and left circumflex) coronary artery occlusion showed significantly greater QT_d (70±29 versus 59±27 milliseconds, P=.003) and JT_d (74±30 versus 63±27 milliseconds, P=.004). Similarly, patients with anterior (versus inferior/lateral) AMI showed significantly greater QT_d (69±30 versus 59±27 milliseconds, P=.006) and JT_d (73±30 versus 63±27 milliseconds, P=.007). Results did not change when Bazett's QT, or JT, was substituted for QT or JT or when ANOVA included adjustments for age, sex, drug assignment, infarct site, infarct vessel, and number of measurable leads. On ANCOVA, the relation of QT_d or JT_d and perfusion grade was not influenced by heart rate.

**Conclusions**

Successful thrombolysis is associated with less QT_d and JT_d in post-AMI patients. The results are equally significant when either QT or JT is used for analysis. These data support the hypothesis that QT_d after AMI depends on reperfusion status as well as infarct site and size. Reduction in QT_d and its corresponding risk of ventricular arrhythmia may be mechanisms of benefit of thrombolytic therapy. *(Circulation. 1994;90:94-100.)*

**Key Words**

- QT dispersion
- Electrocardiography
- Thrombolysis
- Infarction
- Intervals

Thrombolytic therapy, on the other hand, has become the cornerstone of treatment for AMI. It is now indisputable that treatment of AMI with a thrombolytic agent improves survival and preserves myocardial function.21-27 Successfully reperfused patients have demonstrated a lower incidence of early and late mortality, as well as a higher left ventricular ejection fraction (with early therapy), compared with conventionally treated patients. Studies have also indicated that in-hospital and long-term benefits of thrombolysis are closely related to early reestablishment and maintenance of coronary blood flow.22,28-29

In addition to effects on mechanical function, effects of reperfusion therapy on electrical stability are of interest. The present study was undertaken to assess the effect of successful thrombolytic therapy on QT dispersion in patients after AMI and to evaluate the possible prognostic value of QT dispersion in such patients.

**Methods**

**Patient Selection**

The study population included 370 patients with AMI consecutively recruited to a multicenter, double-blind, randomized study comparing streptokinase and anistreplase (TEAM-2, second trial of Thrombolysis with Eminase in Acute Myocardial Infarction). Patient inclusion and exclusion crite-
ria, as well as other details of the study, have been previously published. All patients presented with ST-segment elevation associated with typical ischemic symptoms and received thrombolytic therapy within 4 hours of symptom onset. In addition to the original study exclusions, patients were excluded from the present study if they were not in sinus rhythm and/or had bundle branch block or any other intraventricular conduction abnormality on the discharge ECG.

Coronary Angiography

Coronary perfusion status was determined at early angiography, performed as close to 90 minutes after the start of therapy as possible but within a maximum of 240 minutes. A single observer assessed coronary patency by reading all angiograms in a blinded fashion at a central laboratory (LDS Hospital). Anterograde perfusion of the infarct-related artery was graded according to the classification system of the Thrombolysis in Myocardial Infarction (TIMI) trial as follows: grade 0, no anterograde perfusion; grade 1, minimal perfusion—penetration of the obstructed lumen with negligible distal flow; grade 2, partial perfusion—coronary bed perfuses distal to the obstruction but at a delayed rate of filling and clearing; and grade 3, complete perfusion—coronary bed perfuses distal to the obstruction with a normal rate of filling and clearance. Interobserver variability for the determination of patency was previously shown to be small in our laboratory (TIMI grade 2/3 perfusion versus occlusion, grade 0/1 perfusion).  

Electrocardiography

All standard 12-lead ECGs were recorded at a paper speed of 25 mm/s on day 10 after myocardial infarction or immediately before hospital discharge. All ECGs were examined retrospectively by one observer who was unaware of the patients' drug and coronary patency status. Measurements of QT, QRS, JT, and RR intervals were performed using a commercially available computer program (Configurable Measurement System) interfaced with a Calcomp 9000 digitizer. With this measurement system, intraobserver, interobserver, and interstudy variabilities were shown to be small in a previous study from our laboratory.

QT interval was measured from the onset of the QRS complex to the end of the T wave, defined as its return to the T-P isoelectric baseline. The QT was measured to the nadir of the curve between the T and U waves when the latter was present. QRS interval was measured from the onset of the Q wave (or the R wave, if the Q was absent) to the end of the S wave, defined as its return to the T-P isoelectric baseline. JT interval was computer calculated by obtaining the difference between QT and QRS for each QRST complex measured. Whenever possible, the average measurement of three complexes for each lead was taken. If the end of the T wave could not be reliably determined or when the T waves were isoelectric or of very low amplitude, QT measurements were not made and these leads were excluded from analysis. A lower limit of 5 or more technically adequate leads per ECG was set for inclusion in this study.

QT dispersion was defined as the difference between the maximal and minimal QT interval measurements occurring among any of the 12 leads on a standard ECG. QTc (or heart rate–corrected QT interval) was calculated according to Bazett's formula as follows: QTc = QT/square root of the RR interval. QTc dispersion was calculated in a manner similar to QT dispersion.

JT dispersion was defined as the difference between the maximal and minimal JT interval measurements occurring among any of the 12 leads on a standard ECG. Bazett’s formula was applied to obtain JT, (heart rate–corrected JT interval), substituting JT for QT in the formula given.

Statistical Analysis

Results are expressed as mean±SD unless otherwise stated. One-factor (one-way) ANOVA was used to assess the correlation between QT or JT dispersion and perfusion grade. Simple linear regression analysis was used to determine the correlation of QT or JT dispersion with age and the number of measurable leads. ANOVA and ANCOVA were used to assess whether the QT or JT dispersion was influenced by any of the following factors: age, sex, site of infarct, infarct artery, type of thrombolytic regimen, peak cardiac enzymes, number of measurable leads per ECG, or choice of leads where minimal and maximal QT or JT intervals were measured. ANCOVA was also used to assess whether the relation of QT or JT dispersion and perfusion grade was influenced by heart rate. Spearman's rank correlation procedure was done to determine the correlation of QT or JT dispersion with peak cardiac enzymes, total lactic dehydrogenase (LDH), and LDH isoenzyme-1 (LDH-1). Last, ANOVA using factorial design was performed to determine the most important predictors of differences in QT or JT dispersion after AMI.

Patient Demographics

Of the 370 patients enrolled in TEAM-2, 126 patients were excluded because of the following reasons: no discharge ECG (n=65), poor-quality ECG (n=29), unavailable records (n=11), no angiographic scoring done (n=8), and the presence of bundle branch block (n=9), atrioventricular block (n=2), or atrial fibrillation (n=2). The remaining 244 patients satisfied the entry criteria for this study, including 196 men and 48 women (mean age, 57±10 years; range, 32 to 75 years). Patients were given streptokinase (n=115) or anistreplase (n=129) within 2.6±0.8 hours from symptom onset.

Angiographic Analysis

All 244 patients had coronary angiograms done at 2.4±0.9 hours (range, 0.6 to 6.5 hours) after thrombolytic therapy. The infarct lesion was identified within the right coronary artery in 111 patients (46%), the left anterior descending coronary artery in 104 patients (43%), and the left circumflex coronary artery in 27 patients (11%). Two patients had normal coronaries and were assigned a TIMI perfusion grade of 3. TIMI perfusion grades 0, 1, 2, and 3 were achieved in 41 (17%), 19 (8%), 35 (14%), and 149 (61%) patients, respectively. There was no difference in the distribution of perfusion grades between patients treated with streptokinase and anistreplase (Fig 1), justifying the pooling of results across the two drug therapies. The rate of early total patency (defined as TIMI grade 2/3) was high (75.4%) in these 244 patients and did not differ significantly between anistreplase- and streptokinase-treated patients.

ECG Analysis

The ECG was performed at 9±5 days (range, 2 to 31 days) from symptom onset. The QRS/QT interval was measurable in 10±2 leads (range, 5 to 12 leads). QT, and JT, intervals were determined for all except one patient, who had ventricular bigeminy, preventing accurate determination of RR interval.

The mean QRS, QT, and JT intervals averaged over 12 leads for all patients were as follows: 81±9 (range, 58 to 116), 385±39 (range, 304 to 535), and 305±38 (range, 225 to 444) milliseconds, respectively. The maximal QT
interval was 415±42 milliseconds (range, 319 to 555 milliseconds) and occurred in one of the precordial leads in 84 patients (34%) and in one of the limb leads in 160 patients (66%). The maximal QT was 465±35 milliseconds (range, 394 to 579 milliseconds). The minimal QT interval was 352±40 milliseconds (range, 267 to 528 milliseconds) and occurred in one of the precordial leads in 119 patients (49%) and in one of the limb leads in 125 patients (51%). The minimal QT was 393±31 milliseconds (range, 323 to 500 milliseconds).

The maximal QRS interval was 93±11 milliseconds (range, 66 to 116 milliseconds) and occurred in one of the precordial leads in 136 patients (56%) and in one of the limb leads in 108 patients (44%). The minimal QRS interval was 68±10 milliseconds (range, 45 to 99 milliseconds) and occurred in one of the precordial leads in 95 patients (39%) and in one of the limb leads in 149 patients (61%). The average QRS dispersion was 24±9 milliseconds (range, 6 to 58 milliseconds).

The maximal JT interval was 336±42 milliseconds (range, 245 to 474 milliseconds) and occurred in one of the precordial leads in 153 patients (63%) and in one of the limb leads in 91 patients (37%). The maximal JT was 377±35 milliseconds (range, 309 to 501 milliseconds). The minimal JT interval was 268±38 milliseconds (range, 174 to 431 milliseconds) and occurred in one of the precordial leads in 128 patients (52%) and in one of the limb leads in 116 patients (48%). The minimal JT was 299±31 milliseconds (range, 200 to 409 milliseconds). The average JT dispersion for the entire study group was 68±29 milliseconds (range, 15 to 173 milliseconds), and the average JT dispersion was 79±33 milliseconds (range, 17 to 194 milliseconds).

**QT, QRS, and JT Dispersion by Perfusion Status**

There were significant differences in QT and QT dispersion among the four TIMI perfusion groups. The average QT dispersion was 96±31, 88±25, 60±22, and 52±19 milliseconds and the average QT dispersion was 107±39, 98±26, 70±26, and 59±23 milliseconds in patients with TIMI perfusion grades 0, 1, 2, and 3, respectively (P=.0001 for both QT and QT dispersion, Fig 2). This was manifested as a stepwise decrease in QT and QT dispersion with increasing grade of TIMI perfusion.

Similar results were obtained when JT dispersion was substituted for QT dispersion, with significant differences being observed in JT and JT dispersion among the four perfusion groups. The average JT dispersion was 97±32, 88±31, 63±23, and 58±21 milliseconds and the average JT dispersion was 113±38, 98±29, 75±26, and 67±25 milliseconds in patients with TIMI perfusion grades 0, 1, 2, and 3, respectively (P=.0001 for both JT and JT dispersion, Fig 2). Likewise, this was manifested as a stepwise decrease in JT and JT dispersion with increasing TIMI grades of perfusion.

In contrast, there were no significant differences in QRS dispersion between the four perfusion groups. The average QRS dispersion was 25±10, 22±7, 28±9, and 24±9 milliseconds in patients with TIMI perfusion grades 0, 1, 2, and 3, respectively (P=.12).

These results did not change when the anistreplase- and streptokinase-treated patients were analyzed separately. Furthermore, there was no significant difference in mean QT or QTc, and JT or JTc dispersion between patients in the two treatment regimens (63±28 versus 64±28 milliseconds, P=.61 for QT dispersion; 71±34 versus 72±33 milliseconds, P=.79 for QTc dispersion; 70±30 versus 65±27 milliseconds, P=2 for JT dispersion; and 81±34 versus 75±32 milliseconds, P=.17 for JT dispersion, between anistreplase- and streptokinase-treated patients, respectively).

The Table shows the comparison of QT or QTc, and JT or JTc dispersion among patients when observed TIMI grades were grouped as 0/1 (“occluded”) versus 2/3 (“reperfused”). Significant differences were observed in both QT or QTc, and JT or JTc dispersion between the two perfusion groups (P<.0001). These results did not change when the anistreplase- and streptokinase-treated patients were analyzed separately. As for the other comparisons, significant differences in QT or QTc, and JT or JTc dispersion were observed when TIMI grades 0/1/2 (“absent” or “incomplete” perfusion) were compared with TIMI grade 3.
Comparison of QT or QTc and JT or JTc Dispersion Between Patients With TIMI Grades 0/1 Versus 2/3 and Grades 0/1/2 Versus 3

<table>
<thead>
<tr>
<th>TIMI Grade</th>
<th>No. of Patients</th>
<th>QTd</th>
<th>QTc</th>
<th>JTd</th>
<th>JTc</th>
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</thead>
<tbody>
<tr>
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<td>60</td>
<td>94±29</td>
<td>104±35</td>
<td>94±32</td>
<td>106±36</td>
</tr>
<tr>
<td>2/3</td>
<td>184</td>
<td>54±20</td>
<td>61±24</td>
<td>59±21</td>
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<tr>
<td>P</td>
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<td>.0001</td>
<td>.0001</td>
<td>.0001</td>
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</tr>
<tr>
<td>0/1/2</td>
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<td>81±31</td>
<td>92±36</td>
<td>83±32</td>
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<tr>
<td>P</td>
<td>.0001</td>
<td>.0001</td>
<td>.0001</td>
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</tr>
</tbody>
</table>

QTd indicates dispersion of uncorrected QT interval; QTc dispersion of QT interval corrected for heart rate; and TIMI, Thrombolysis in Myocardial Infarction trial.

P value calculated by one-way ANOVA.

(“complete” perfusion) (P<.0001). These results did not change when the anistreplase- and streptokinase-treated patients were analyzed separately.

**QT Dispersion, Infarct Artery, and Infarct Site**

Increased QT or QTc and JT or JTc dispersion was also noted in patients with left anterior descending compared with those with right or left circumflex coronary artery occlusion (70±29 versus 59±27 milliseconds, P=.003 for QT dispersion; 79±34 versus 66±31 milliseconds, P=.002 for QT; dispersion; 74±30 versus 63±27 milliseconds, P=.004 for JT dispersion; and 87±34 versus 72±31 milliseconds, and P=.0008 for JT; dispersion, respectively). Similarly, increased QT or QTc and JT or JTc dispersion was noted in patients with anterior versus those with inferior, lateral, or other AMI (69±30 versus 59±27 milliseconds, P=.006 for QT dispersion; 78±34 versus 66±31, P=.004 for QT; dispersion; 73±30 versus 63±27 milliseconds, P=.007 for JT dispersion; and 85±35 versus 72±31 milliseconds, P=.002 for JTc dispersion, respectively).

**QT or JT Dispersion and Cardiac Enzymes**

There was a trend for QT dispersion to correlate positively with peak total LDH levels, a trend that did not quite achieve statistical significance (P=.07). A similar result was obtained when peak LDH-1 isoenzyme was analyzed. However, this trend was not demonstrated when JT intervals were correlated with peak total LDH (P=.46) or peak LDH-1 isoenzyme (P=.31).

**Covariance of QTd With Clinical and ECG Variables**

Results showing a difference in distribution of QT/QTc or JT or JTc dispersion among the four perfusion groups did not change when ANOVA was adjusted for infarct site, infarct vessel, and number of measurable leads. Further analysis showed that QT or QTc, or JT or JTc, dispersion was not influenced by age, sex, or choice of leads on which minimal and maximal QT intervals were measured. Last, ANCOVA showed that the relation of QT dispersion and perfusion grade was not influenced by heart rate.

**ANOVA Using Factorial Design**

To determine which are the most important predictors of differences in QT dispersion after myocardial infarction, ANOVA using a factorial design was performed. Complete data vectors were available for 236 patients (97%) in the factorial design analysis, using the nominal variables of infarct artery (P=.24), infarct site (P=.63), lead choice for measurement (P=.52), thrombolytic drug assignment (P=.94), and perfusion grade (P=.0001). The results demonstrated that perfusion grade was the single and only important factor that correlated with differences in QT dispersion. The results did not change significantly when QTd, JT, or JTc dispersion was substituted for QT or when interactions between perfusion grade and the other variables were used in the analysis.

**Discussion**

**Study Summary and Clinical Implications**

This large blinded study of 244 post–myocardial infarction patients has shown that successful thrombolysis is associated with less dispersion in QT or QTc and JT or JT, interval, measures of dispersion of ventricular repolarization. In contrast, no differences in dispersion of intraventricular conduction (QRSd) was demonstrated. Thus, by establishing patency, thrombolytic therapy may reduce the degree to which an abnormal electrophysiological milieu develops after AMI. The study also has shown that dispersion of QT or QTc and JT or JTc is greater with left anterior descending coronary artery occlusion (versus right and left circumflex occlusion) and with anterior wall site of infarction (versus inferior/lateral). Our results suggest that reduction in QT or JT dispersion after AMI depends on the reperfusion status of the infarct-related artery as well as on the infarct site and size. Reduction in dispersion of ventricular repolarization times throughout the heart, together with its corresponding risk of ventricular arrhythmia, may be a previously unreported mechanism of benefit of thrombolytic therapy.

The main goal of thrombolytic therapy in patients with AMI is the establishment and maintenance of coronary patency to improve left ventricular function and decrease mortality. Although the use of thrombolytic agents has been documented to reduce mortality rates after infarction, the mechanisms of this benefit continue to be debated, potentially including improvements in both mechanical and electrical functions. However, studies have shown that postinfarction patients with open arteries have a lower mortality rate than patients with closed arteries. Mortality rates as low as 2.5% have been reported in patients with patent arteries compared with 15% in patients with closed arteries. Mechanisms proposed to account for the beneficial effects of early and late reperfusion on mortality have been reviewed. Our study demonstrates a favorable consequence of reperfusion distinct from reduction in infarct size or improvement in myocardial function. Our results suggest that successfully reperfused patients (ie, those with TIMI grade 2/3) exhibit a more stable electrical substrate than those with persistent occlusion. Achievement of early patency might correlate with a reduced incidence of subsequent arrhythmic or cardiac death.
Considering that post-AMI patients are generally at increased risk for arrhythmic death, reduction in QT or JT dispersion, a marker of dispersion of ventricular repolarization, in these patients by reperfusion could have important clinical implications. Accordingly, the assessment of QT or JT dispersion at discharge could be prognostically useful.

There are many reports in the literature relating the risk of subsequently developing serious ventricular arrhythmias (eg, ventricular tachycardia) with the perfusion status of the infarct artery in patients after AMI.\(^{39-41}\) An increased frequency of late potentials on signal-averaged ECG has also been demonstrated after AMI in patients with occluded compared with perfused infarct-related arteries in some but not other studies.\(^{42-44}\) However, the unique contribution of this study is the demonstration that a strong correlation exists between the early perfusion status of the infarct-related artery after thrombolysis and the degree of dispersion of ventricular repolarization times.

Current measures of recovery time dispersion, ie, epicardial monophasic action potentials and body surface mapping, are either invasive or impractical for routine use.\(^{45-47}\) The 12-lead ECG, as used in the present study, is a simple and accessible clinical tool that is routinely available. As Day et al\(^{18}\) suggested, such an easily accessible, reasonably accurate, noninvasive method for assessment of dispersion of repolarization could significantly alter our approach to arrhythmia risk assessment, redefining current concepts, predicting arrhythmogenesis, improving prognostication, and perhaps guiding therapy.

**Dispersion of Conduction Versus Repolarization**

The QT or QT\(_c\) interval is dependent on ventricular conduction time as well as ventricular repolarization. In an effort to determine whether one or both of these variables contribute to interlead differences in QT interval, QRS intervals were measured, and JT and JT\(_c\) intervals were calculated. No significant variation in QRS interval was noted by perfusion grade. In contrast, the relation of perfusion grade and dispersion was equally significant whether QT or JT was used in the analysis. These data support the hypothesis that changes in repolarization are responsible for the QT dispersion noted in our patients. Thus, measurement of the QT interval appears sufficient for assessing interlead variation that might reflect regional variations in ventricular repolarization.

**Previous Studies of QT Prolongation and Dispersion**

The clinical significance of QT interval prolongation has been the subject of much debate.\(^{48-49}\) Evidence to date in published literature favors an association between a prolonged QT or an increased QT dispersion and an increased risk of sudden arrhythmic death. The possibility that persistent prolongation of the QT interval might be associated with an increased risk of complex and often fatal cardiac arrhythmias derives from clinical and experimental data. The long QT syndrome, for example, is associated with a very high incidence of ventricular fibrillation.\(^{50-51}\) Drugs that prolong the QT interval, such as quinidine, may also cause fatal arrhythmias and sudden death.\(^{20,52,53}\) Other investigations have shown that even in patients without intraventricular conduction defects and cardiac dysfunction, QT prolongation is a risk factor for sudden death.\(^{6}\) Although the explanation for QT prolongation in these patients is not certain, endocardial monophasic action potential studies have demonstrated that there are regional differences in the duration of myocardial repolarization that may be reflected in the surface ECG. Investigators have demonstrated that interlead variations in QT interval measurements (reflecting regional variations in ventricular repolarization) are significantly greater in patients with myocardial infarction and arrhythmia than in those without and that excessive dispersion of recovery times increases the susceptibility to malignant arrhythmias.\(^{15,17,18,20,54,55}\) Such increased dispersion results in prolongation of the vulnerable period and thereby enhanced susceptibility to ventricular tachyarrhythmias. Recently, emphasis has been placed on QT\(_d\) rather than QT prolongation per se as the cause of increased risk, because some conditions associated with a prolonged QT (eg, therapy with amiodarone or sotalol) may reduce the risk of sudden death in association with a reduction in QT\(_d\) but an increase in QT.\(^{15,18}\)

Other investigators have proposed the hypothesis that afterdepolarizations are the basis for the long QT syndrome.\(^{51}\) This hypothesis links clinical observations with cellular electrophysiological phenomena that suggest that these afterdepolarizations are the basis for the U wave on the ECG and that triggered activity is responsible for initiation of the associated arrhythmias. Although there is growing interest in the afterpotential hypothesis, studies to evaluate this theory in the post-infarction setting have not been reported.

**QT Dispersion in Healthy Subjects**

Previous studies have shown that healthy subjects exhibit a small degree of QT dispersion.\(^{11,12,46}\) These studies have also shown that interobserver differences in QT measurement are small compared with the typical values for QT dispersion observed in patients with myocardial infarction. In our study, the QT dispersion noted in patients with patent arteries (54±20 milliseconds) was in the same range established for healthy subjects in the studies of Sylven et al (54±27 milliseconds),\(^{46}\) Mirvis et al (59±12.9 milliseconds),\(^{11}\) and Cowan et al (48±18 milliseconds).\(^{12}\) In contrast, patients in our study who had nonpatent coronary arteries showed a substantially greater and statistically significantly increased QT dispersion (94±29 milliseconds). These indicate that true increases in QT dispersion (presumed to be due to regional differences in repolarization) can be differentiated from normal, physiologically occurring interlead variations and those due to technical artifacts (ie, caused by differences in unipolar versus bipolar leads, differential tissue attenuation or cancellation of vectors, etc). Because homogeneity of recovery time is believed to protect against arrhythmias and dispersion of recovery time is believed to be arrhythmogenic, the reduction in QT and JT dispersion seen in our reperfused patients may be interpreted as evidence for this mechanism of benefit of thrombolytic therapy.

**Study Limitations**

A limitation of QT interval assessment is that it is not always measurable in every lead or may be difficult to
measure with precision in certain leads, a problem recognized by previous investigators. In this study, QT interval could be measured adequately in all 12 leads in only a minority of patients (14%), although in most cases, all except 1 or 2 leads (average, 10) were evaluable. The difference in QT interval variation in measured leads between patients with patent and non-patent arteries was not explained by a difference in the number of or distribution of leads contributing to QT dispersion determination. Thus, with the use of a reliable computerized measurement system, the QT interval was able to be accurately assessed in the majority of leads, and a reliable measure of QT dispersion could be determined.

There is no universally recognized standard method of analysis of or lead selection for QT interval assessment. Cowan et al. found divergent estimates of QT interval among methods and underscored the need for standardization of lead selection. Some investigators have used only the precordial V1 through V6 leads in the selection of maximal and minimal QT intervals. In the present study, all leads that were technically adequate were measured, neglecting the concern about whether the "best" leads or lead groups were chosen for measurement.

Bazett's formula for calculation of QT, has been found to give suboptimal correction, as it includes a slight overcorrection of the QT interval for fast heart rates. However, none of the other proposed methods of rate correction for QT have been generally accepted. In view of potential errors that may be generated by suboptimal correction of the QT interval for heart rate, it has been suggested that including the uncorrected QT interval and heart rate as well as QT, data in published reports would be helpful to reader interpretation. In our study, results were reported for both uncorrected and heart rate-corrected intervals and were consistent whether QT or QTc, measurements were used in the analysis, strengthening the validity of the relation between QT dispersion and perfusion status noted.

Finally, the study was limited by the absence of long-term outcome information, which was not collected in TEAM-2. Other studies are needed to verify the predicted better clinical outcome of patients with reduced QTc after thrombolysis.

Conclusions

The results of the present study show that successful thrombolysis is associated with less QT and JT dispersion on the convalescent (predischarge) ECG in patients after AMI. The results were equally significant whether QT or JT was used in the analysis. Our data support the hypothesis that QT dispersion after myocardial infarction depends on reperfusion status, infarct site, and infarct size. Reduction in QT dispersion may be a mechanism of benefit of thrombolytic therapy.

Acknowledgments

This work was unfunded. The original TEAM-2 study was supported by a grant from SmithKline Beecham Pharmaceuticals, Philadelphia, Pa.

References


15. Merx W, Yoon MS, Han J. The role of local disparity in conduc- tion and recovery time on ventricular vulnerability to fibrillation. Am Heart J. 1977;94:603-610.


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Circulation. 1994;90:94-100
doi: 10.1161/01.CIR.90.1.94

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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