Comparison of QT Dispersion between Primary Coronary Angioplasty and Thrombolytic Therapy for Acute Myocardial Infarction

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Key words: QT dispersion, primary coronary angioplasty, thrombolytic therapy, myocardial infarction

Abstract

Background: Previous studies have documented that reduction in QT dispersion after thrombolytic treatment in acute myocardial infarction depends on reperfusion status as well as infarct site. Primary percutaneous transluminal coronary angioplasty as compared with thrombolytic therapy has been shown to result in higher patency rates of the infarct vessel.

Objectives: To evaluate whether primary PTCA has a more favorable effect on reducing QT dispersion in patients with acute MI as compared to thrombolytic treatment.

Methods: The study population included 42 consecutive patients (33 men, mean age 58±11 years) with acute MI (24 anterior wall, 18 inferior wall) who were treated with primary PTCA (group A, n = 21) or thrombolytic therapy (group B, n = 21) at 3.9±2 hours after symptom onset. QT intervals were measured before and 24 hours after treatment.

Results: On the admission electrocardiogram, patients with anterior MI had higher values of QT and QTc dispersions than patients with inferior MI (52±9 vs. 36±9 msec, $P<0.05$ and 61±4 vs. 56±4 msec, $P=0.002$, respectively). There was a significant reduction in QT and QTc dispersions from admission to 24 hours in all patients (from 50±9 to 37±9 msec, $P<0.001$ and from 59±5 to 42±5 msec, $P<0.001$, respectively), and also in group A (from 49±8 to 32±5 msec, $P<0.001$ and from 58±5 to 38±3 msec, $P<0.001$, respectively) and in group B patients (from 51±10 to 42±10 msec, $P<0.01$ and from 60±4 to 46±5 msec, $P<0.001$, respectively). QT and QTc dispersions were found to be shorter in group A at 24 hours after treatment than in group B (32±5 vs. 42±10 msec, $P<0.001$ and 38±3 vs. 46±5 msec, $P<0.001$, respectively).

Conclusions: Reperfusion therapy with primary PTCA or thrombolytic agents reduces QT and QTc dispersions in acute MI. QT and QTc dispersions after reperfusion treatment are shorter with primary PTCA than with thrombolytic therapy.

IMAJ 2001;3:333–337

Clinical and experimental studies have suggested that QT dispersion (the difference between maximal and minimal QT interval calculated on a standard 12-lead electrocardiogram) could reflect regional variations of ventricular repolarization [1] and could provide a substrate for life-threatening ventricular arrhythmias in hypertrophic cardiomyopathy [2], chronic heart failure [3], congenital long QT syndromes [4], myocardial ischemia and, particularly, acute MI [5,6]. It has been shown that increased QT dispersion is an independent predictor for ventricular tachycardia or ventricular fibrillation, and is associated with impaired cardiac function, larger post-infarction scar size and poor prognosis in patients with acute MI [6–8].

Early coronary reperfusion has been shown to be effective in reducing electrophysiological instability by decreasing QT dispersion in the recovery phase after acute MI [9]. In addition, the results of a few clinical studies suggest that reduction in QT dispersion by successful thrombolytic therapy in acute MI depends on patency status of the infarct-related artery, particularly at Thrombolysis in Myocardial Infarction grades 2 and 3 flow, compared to TIMI grades 0 and 1 flow [8,10]. Moreover, it has been reported that TIMI grade 3 flow patency rate is 54–60% with thrombolytic therapy [11,12] compared to 92–97% with primary angioplasty [13,14]. The purpose of the present study was to examine whether the higher patency rate of primary PTCA, as a reperfusion method, is reflected by a more favorable effect than thrombolytic therapy on reducing QT dispersion in patients with acute MI.

Methods

Study patients

The study population included 42 consecutive patients who were admitted to the hospital within 6 hours after the onset of acute MI (typical chest pain, ST segment elevation on admission ECG compatible with acute MI, followed by significant serum enzyme elevation). Patients were excluded from the study for any of the following reasons: atrial fibrillation or flutter, bundle branch block or any other intraventricular conduction abnormalities, pre-excitation on ECG, cardiogenic shock, cardiomyo-

PTCA = percutaneous transluminal coronary angioplasty
MI = myocardial infarction
QTc = corrected QT

TIMI = Thrombolysis in Myocardial Infarction
ECG = electrocardiogram

pathy, ventricular hypertrophy, and severe valvular heart disease. The patient group comprised 33 males and 11 females with a mean age of 58±11 years. ECG localization of MI indicated anterior MI (including anterior, anteroseptal and anterolateral location) in 24 patients and inferior MI (including inferior, inferoposterior, and inferolateral location) in 18 patients.

The study patients were treated with primary PTCA (group A, 21 patients, including primary angioplasty in 8 patients and stent-supported primary angioplasty in 13) or thrombolytic agents (group B, 21 patients, including streptokinase in 4 patients and tissue-type plasminogen activator in 17). The choice between treatment methods was independent of the clinical characteristics of patients and was dependent on the availability of the catheterization laboratory staff to perform primary PTCA. Also, coronary angiography was performed in patients treated with primary PTCA before the procedure. In these patients, anterograde perfusion of the infarct-related artery was graded according to the classification system of the TIMI trial [15]: grade 0 = no anterograde perfusion, grade 1 = minimal perfusion, grade 2 = partial perfusion, grade 3 = complete perfusion. Coronary angiography was not done in patients who received thrombolytic treatment in the acute phase of MI. Pre-discharge coronary angiography was performed in 15 patients of group B, with estimation of the mean number of stenosed arteries and TIMI perfusion grade in most probable infarct-related vessels in cases of multivessel disease. Six patients refused the procedure. Though group B patients did not undergo immediate coronary angiography reperfusion, the state after thrombolytic therapy was assessed using clinical criteria.

Complete relief of chest pain, resolution of ECG abnormalities (ST segment resolution) and development of reperfusion arrhythmias, especially accelerated idioventricular rhythm, were accepted as clinical criteria of reperfusion after thrombolytic therapy. In patients treated with primary PTCA, we performed angioplasty or stent procedure only in the infarct-related artery. Stenting was performed in patients whose infarct-related artery exhibited a non-optimal angiographic result after one or more dilations with appropriately sized balloons. Aspirin and intravenous heparin were routinely given to all of the study patients.

ECG recordings and QT interval measurements

Measurements of QT intervals were calculated at admission (before treatment) and in 24 hour ECGs (after treatment). At the time of both these ECGs, patients were not taking antiarrhythmic drugs and their electrolyte status was normal. All standard 12-lead ECGs were recorded at a paper speed of 25 mm/sec and examined retrospectively by one observer who did not know the reperfusion strategy of the study patients. Intraobserver reproducibility of QT measurements was assessed in the sample of 15 patients; correlation coefficient between two readings was 0.93 for QT maximum and 0.85 for QT minimum (for both $P < 0.0001$). Measurements of QT and RR intervals were performed manually. QT interval was measured from the beginning of the Q wave to the end of the T wave. The end of the T wave was defined as a return to the isoelectric T-P baseline. When U wave was present, the QT was measured to the nadir of the curve between the T and U waves. If the end of the T wave could not be reliably determined or if the T waves were isoelectric or of very low amplitude, measurements were not done and these leads were excluded from analysis. All patients had at least eight ECG leads that were measurable. All of the ECGs were in sinus rhythm. QTc was calculated according to Bazett's formula [16]. QT and QTc dispersions were defined as the difference between the maximum and minimum QT, and the maximum and minimum QTc interval measurements, respectively.

Statistical analysis

The results are presented as mean ± standard deviation. The statistical analysis was performed using the Statistical Package for Social Sciences software (SPSS for Windows). Differences between groups (A vs. B, anterior vs. inferior MI) were examined by the unpaired *t*-test, and comparison of ECG data before and after treatment by the paired *t*-test. Frequencies were compared using chi-square analysis. P value < 0.05 was considered to be statistically significant.

Results

Patients' characteristics [Table 1]

Time from the onset of symptoms to the beginning of the reperfusion therapy was 3.9±2 hours for the total study population. There were no significant differences between group A and group B with regard to age, gender, cardiovascular risk factors, time from symptom onset to treatment, and MI localization. In group A patients, the infarct lesion was identified within the left anterior descending artery in 11 patients (52%), left circumflex artery in 3 patients (15%), and right coronary artery in 7 (33%). In this group, there were 14 patients with one-vessel disease, 5 patients with two-vessel disease and 2 patients with three-vessel disease. TIMI perfusion

Table 1. Clinical characteristics in patients treated with primary PTCA and thrombolytic therapy

Demographics	Primary PTCA (n = 21)	Thrombolytic therapy (n = 21)	*P*
Age (yr)	57±11	58±10	NS
Male, n (%)	17 (80%)	16 (76%)	NS
Systemic hypertension, n (%)	6 (28%)	7 (33%)	NS
Hypercholesterolemia, n (%)	6 (28%)	9 (42%)	NS
Diabetes mellitus, n (%)	1 (5%)	5 (23%)	NS
Cigarette smoking, n (%)	14 (66%)	12 (57%)	NS
Family history of CAD, n (%)	3 (14%)	1 (5%)	NS
Time to therapy (hr)*	4.1± ± 2	3.8± ± 2	NS
Anterior MI, n (%)	13 (62%)	11 (52%)	NS

* Time from symptom onset to treatment.
CAD = coronary artery disease, NS = not significant.

Table 2. Comparison of QT intervals before (admission) and after (24 hour) reperfusion therapy regardless of reperfusion strategy in study patients (n = 42)

Mean ± SD (msec)	Before reperfusion therapy	After reperfusion therapy	*P*
QT maximum	392± ± 42	389± ± 51	0.6
QT minimum	340± ± 39	354± ± 47	0.06
QT dispersion	50± ± 9	37± ± 9	< 0.001
QTc maximum	459± ± 37	445± ± 44	< 0.05
QTc minimum	399± ± 36	404± ± 40	0.3
QTc dispersion	59± ± 5	42± ± 5	< 0.001

grades 3 and 2 were achieved in 19 (90.4%) and 2 (9.6%) patients in group A, respectively. In group B, single-vessel disease was documented in 5 patients, two-vessel disease in 6 patients and three-vessel disease in 4 patients. TIMI perfusion grade 3 was registered in 12 patients, grade 1 in 2 patients, and 1 patient had grade 0 flow in the most probable infarct-related artery. Clinical reperfusion was thought to be established in 14 patients (66%) in group B.

QT dispersion and reperfusion therapy in acute MI [Table 2]

Comparison of QT and QTc dispersions before treatment and in the after treatment phase revealed a significant reduction from admission to 24 hour ECGs in all study patients treated with primary PTCA or thrombolytic agents. Also, there was a significant reduction in the QTc maximum between admission and 24 hour ECGs, but not QTc minimum.

QT dispersion and reperfusion methods in acute MI [Table 3]

There were no significant differences in QT interval measurements between group A and group B at admission. However, there were significant differences in the QT and QTc dispersions between group A and group B 24 hours after treatment (32 ± 5 vs. 42 ± 10 msec, $P < 0.001$ and 38 ± 3 vs. 46 ± 5 msec, $P < 0.001$, respectively), but not in the other QT interval measurements. Furthermore, there was a significant reduction 24 hours after treatment in QT and QTc dispersions in both groups, and QTc maximum in group A. The other QT interval measurements in both groups did not vary significantly between admission and 24 hours after treatment.

QT dispersion and MI localization [Table 4]

Increased QT and QTc dispersions were observed in patients with anterior MI compared with inferior MI at admission ECGs (52 ± 9 vs. 36 ± 9 msec, $P < 0.05$ and 61 ± 4 vs. 56 ± 4 msec, $P = 0.002$, respectively). However, there were no significant differences in the QT and QTc dispersions between anterior and inferior MI 24 hours after the treatment. Moreover, QT and QTc dispersions were significantly shortened 24 hours after reperfusion treatment in anterior MI and also in inferior MI ($P < 0.001$ for both). There were no differences in the other QT interval measurements between anterior and inferior MI at admission, nor in 24 hour ECGs.

Table 3. Comparison of QT intervals between primary PTCA and thrombolytic therapy, and before and after treatment in each group

Mean ± SD (msec)	Primary PTCA	Thrombolytic therapy	*P*
Before treatment			
QT maximum	397 ± 42	388 ± 42	0.9
QT minimum	345 ± 39	335 ± 39	0.9
QT dispersion	49 ± 8	51 ± 10	0.2
QTc maximum	468 ± 40	449 ± 31	0.09
QTc minimum	409 ± 39	388 ± 29	0.06
QTc dispersion	58 ± 5	60 ± 4	0.02
After treatment			
QT maximum	383 ± 44	395 ± 58	0.3
QT minimum	350 ± 41	358 ± 54	0.3
QT dispersion	32 ± 5**	42 ± 10***	< 0.001
QTc maximum	445 ± 49*	445 ± 39	0.9
QTc minimum	410 ± 44	399 ± 37	0.3
QTc dispersion	38 ± 3**	46 ± 5**	< 0.001

* $P < 0.05$ vs. QTc maximum before treatment

** $P < 0.001$ vs. QT and QTc dispersion before treatment

*** $P < 0.01$ vs. QT dispersion before treatment. There were no significant differences in the other comparison of QT interval measurements before and after treatment.

Table 4. Comparison of QT intervals between anterior and inferior MI localization, and before and after treatment in each group

Mean ± SD (msec)	Anterior MI (n = 24)	Inferior MI (n = 18)	*P*
Before treatment			
QT maximum	384 ± 35	387 ± 54	0.1
QT minimum	331 ± 35	355 ± 49	0.4
QT dispersion	52 ± 9	36 ± 9	< 0.05
QTc maximum	461 ± 42	455 ± 28	0.5
QTc minimum	400 ± 41	398 ± 28	0.8
QTc dispersion	61 ± 4	56 ± 4	0.002
After treatment			
QT maximum	391 ± 50	387 ± 54	0.9
QT minimum	353 ± 48	355 ± 49	0.7
QT dispersion	38 ± 9*	36 ± 9*	0.5
QTc maximum	450 ± 45	437 ± 42	0.5
QTc minimum	411 ± 40	396 ± 41	0.2
QTc dispersion	43 ± 6*	41 ± 4*	0.2

* $P < 0.001$ vs. QT and QTc dispersion before treatment. There were no significant differences in the other comparison of QT interval measurements before and after treatment.

Discussion

The results of this study showed that reperfusion therapy leads to reduced QT and QTc dispersions in patients with acute MI, regardless of treatment methods. Our study also suggested that

primary PTCA has a more favorable effect on reducing QT and QTc dispersions at the end of the first 24 hours in patients with acute MI, compared to thrombolytic therapy. Furthermore, QT and QTc dispersions were found to be greater in anterior MI (versus inferior MI) before the reperfusion treatment, but 24 hours after treatment they significantly shortened for both anterior and inferior wall MI.

Clinical significance of QT interval in acute MI

Several studies have confirmed that interlead variations in QT interval measurements reflect regional variation in ventricular repolarization, and that increased dispersion results in prolongation of the vulnerable period and, consequently, enhanced susceptibility to ventricular arrhythmias [1–5,17]. Increased QT dispersion is a well-known finding during acute MI and was found to be significantly greater in patients with MI who had malignant ventricular arrhythmias than in those without arrhythmias [5–7]. Previous research has shown that in patients with acute MI who developed ventricular fibrillation within the first 24 hours after admission, QT dispersion was significantly longer (88 ± 30 msec) than in those without ventricular fibrillation (56 ± 24 msec) [18]. It has been reported that QT dispersion > 80 msec was associated with ventricular tachycardia, with a 68% sensitivity and 88% specificity in the chronic stage of MI [7]. On the other hand, several investigators have reported that QT dispersion does not predict early ventricular fibrillation during acute MI [19,20]. This discrepancy may be related to variations in QT measurements over time and the different mechanisms of arrhythmogenesis at each stage of MI.

Influence of coronary reperfusion on QT dispersion in acute MI

Studies have documented that treatment of acute MI with thrombolytic agents or primary PTCA leads to reestablishment and maintenance of coronary patency, preserves myocardial function, and improves survival [11–14]. It has also been pointed out that establishing sustained patency may reduce electrophysiological instability by reducing QT dispersion, thus reduction in QT dispersion may be an additional mechanism of the benefit of reperfusion [9,10]. There are a few clinical reports in the literature concerning QT dispersion in acute MI, including analysis of perfusion status of the infarct-related artery [8–10]. Karagounis et al. [10] showed that patients with TIMI grades 2/3 had less QT and QTc dispersions on their post-myocardial infarction ECG (on the 10th day) than those with TIMI grades 0/1 flow (51 ± 22 msec vs. 74 ± 23 msec for QT, 59 ± 25 msec vs. 84 ± 24 msec for QTc, respectively). The same was found for TIMI grade 3 compared to TIMI grades 0/1/2 flow (50 ± 20 msec vs. 65 ± 28 msec for QT, 57 ± 23 msec vs. 74 ± 31 msec for QTc, respectively). Moreover, a previous study indicated a significant reduction in the QTc dispersion and QTc maximum from the acute (2nd day) to the recovery phase (14th day) of MI in patients with early coronary recanalization (TIMI grade > 2 within 12 hours after the onset of MI) compared to patients without recanalization, but not in the QTc minimum [9]. These results are compatible with our findings. Other investigators observed that in patients with reperfusion the QT interval was transiently prolonged within 12 hours and later shortened, but in patients without reperfusion it became progressively prolonged on serial ECGs recorded within 72 hours after the onset of MI [21]. However, in these studies only thrombolytic agents had been used. Accordingly, we found a significant reduction in the QT and QTc dispersions with thrombolytic treatment. Moreover, we could demonstrate a significant reduction in the QT, QTc dispersion and QTc maximum with primary PTCA, as with the other reperfusion strategy.

Few studies have investigated the reduction of QT dispersion in the late phase of MI treated with angioplasty of the infarct-related artery [22]. We could not find any report that compared QT dispersion in primary PTCA and thrombolytic therapy in the acute phase of MI. However, it is known that primary PTCA leads to a higher patency rate than do thrombolytic agents [11–14]. On the other hand, reduction of the QTc dispersion is dependent on reperfusion status after MI [8,9,10,21]. In our patients treated with primary PTCA, the total patency rate (defined as TIMI grades 2 and 3) was high (100%). We could not demonstrate immediate patency status in patients treated with thrombolytic therapy because angiography was not performed in these patients in the acute phase of MI. Although there was a significant reduction in QT and QTc dispersions in both groups, we found that QT and QTc dispersions were shorter in the primary PTCA group 24 hours after treatment than in the thrombolytic group. This result can be explained by the higher TIMI grade 3 patency rate achieved with primary PTCA.

QT dispersion and infarct site

As has been previously shown, QT and QTc dispersions are greater with anterior MI than inferior MI [6,8]. Furthermore, it has been reported that QT and QTc dispersions are dependent on the infarct size, and the greater values of QT and QTc dispersions associated with anterior MI can be explained by larger infarction [7,8]. Our findings are compatible with these results. In addition, we observed a significant reduction of QT and QTc dispersions with reperfusion therapy in both sites of MI.

The SD of the QTc dispersion was too low, and the SD of the QT dispersion was relatively low in our study compared to previous reported results [7–10]. The lower values of SD in our patients can be attributed to the relatively small study population, vigorous selection of leads for QT analysis (reduced up to 8), and the homogenous group of patients: all had chest pain on presentation and arrived within 6 hours of the chest pain onset.

Study limitations

A limitation of this study is that angiography was not performed and patency status or TIMI grades flow could not be examined in patients who received thrombolytic treatment in

the acute phase of MI. Thus, we could not assess the relationship between QT dispersion and TIMI grade flow in both groups. Moreover, there is no consensus for QT interval assessment with a standard method of analysis or of lead selection. In this study, QT interval was measured manually. Finally, this study was a retrospective investigation, though there were no significant differences in clinical characteristics between the groups.

Conclusions

The results of the present study demonstrate that reperfusion therapy with primary PTCA or thrombolytic agents reduces QT and QTc dispersions in patients with acute MI. QT and QTc dispersions are shorter with primary PTCA compared to thrombolytic therapy after 24 hours treatment. The result of shorster QT and QTc dispersions with primary PTCA after 24 hours treatment could be attributed to the higher TIMI grade 3 patency rate achieved by primary PTCA. Finally, our findings support the association between a higher value of QT or QTc dispersions and anterior MI.

References

1. Zabel M, Portnoy S, Franz MR. Electrocardiographic indexes of dispersion of ventricular repolarization: an isolated heart validation study. *J Am Coll Cardiol* 1995;25:746–52.
2. Buja G, Miorelli M, Turrini P, Melacini P, Nava A. Comparison of QT dispersion in hypertrophic cardiomyopathy between patients with and without ventricular arrhythmias and sudden death. *Am J Cardiol* 1993;72:973–6.
3. Barr CS, Nass A, Freeman M, Lang CC, Struthers AD. QT dispersion and sudden unexpected death in chronic heart failure. *Lancet* 1994;343:327–9.
4. Linker NJ, Colonna P, Kekwick CA, Till J, Camm AJ, Ward DE. Assessment of QT dispersion in symptomatic patients with congenital long QT syndromes. *Am J Cardiol* 1992;69:634–8.
5. Higham PD, Furniss SS, Campbell RW. QT dispersion and components of the QT interval in ischaemia and infarction. *Br Heart J* 1995;73:32–6.
6. Paventi S, Bevilacqua U, Parafati MA, Di Luzio E, Rossi F, Pelliccioni PR. QT dispersion and early arrhythmic risk during acute myocardial infarction. *Angiology* 1999;50:209–15.
7. Puljevic D, Smalcelj A, Durakovic Z, Goldner V. Effects of postmyocardial infarction scar size, cardiac function, and severity of coronary artery disease on QT interval dispersion as a risk factor for complex ventricular arrhythmia. *Pacing Clin Electrophysiol* 1998;21:1508–16.
8. Moreno FL, Villanueva T, Karagounis LA, Anderson JL. Reduction in QT interval dispersion by successful thrombolytic therapy in acute myocardial infarction. TEAM-2 Study Investigators. *Circulation* 1994;90:94–100.
9. Endoh Y, Kasanuki H, Ohnishi S, Shibata N, Hosoda S. Influence of early coronary reperfusion on QT interval dispersion after acute myocardial infarction. *Pacing Clin Electrophysiol* 1997;20:1646–53.
10. Karagounis LA, Anderson JL, Moreno FL, Sorensen SG. Multivariate associates of QT dispersion in patients with acute myocardial infarction: primacy of patency status of the infarct-related artery. *Am Heart J* 1998;135:1027–35.
11. The GUSTO Angiographic Investigators: The comparative effects of tissue plasminogen activator, streptokinase, or both on coronary artery patency, ventricular function and survival after acute myocardial infarction. *N Engl J Med* 1993;329:1615–22.
12. Cannon CP, McCabe CH, Diver DJ, Herson S, Greene RM, Shah PK, Sequeira RF, Leya F, Kirshenbaum JM, Magorien RD. Comparison of front-loaded recombinant tissue-type plasminogen activator, antistreptase and combination thrombolytic therapy for acute myocardial infarction: Results of the Thrombolysis in Myocardial Infarction (TIMI) 4 trial. *J Am Coll Cardiol* 1994;24:1602–10.
13. Grines CL, Browne KF, Marco J, Rothbaum D, Stone GW, O'Keefe J, Overlie P, Donohue B, Chelliah N, Timmis GC, Vlietstra RE, Strzelecki M, Puchrowicz-Ochocki S, O'Neill WW. A comparison of immediate angioplasty with thrombolytic therapy for acute myocardial infarction. The Primary Angioplasty in Myocardial Infarction Study Group. *N Engl J Med* 1993;328:673–9.
14. De Boer MJ, Suryapranata H, Hoorntje JC, Reiffers S, Liem AL, Miedema K, Hermens WT, van de Brand MJ, Ziljstra F. Limitation of infarct size and preservation of the left ventricular function after primary coronary angioplasty compared with intravenous streptokinase in acute myocardial infarction. *Circulation* 1994;90:753–61.
15. Chesebro JH, Knatterud G, Roberts R, Borer J, Cohen LS, Balen J, Dodge HT, Francis CK, Hillis D, Ludbrook P. Thrombolysis in myocardial infarcion (TIMI) trial, Phase I: a comparison between intravenous plasminogen activator and intravenous streptokinase. *Circulation* 1987;76:142–54.
16. Ahnve S. Correction of the QT interval for heart rate: review of different formulas and the use of Bazett's formula in myocardial infarction. *Am Heart J* 1985;109:568–74.
17. Kuo CS, Munakata K, Reddy CP, Surawicz B. Characteristics and possible mechanism of ventricular arrhythmia dependent on the dispersion of action potential durations. *Circulation* 1983;67:1356–67.
18. Van de Loo A, Arendts W, Hohnloser SH. Variability of QT dispersion measurements in the surface electrocardiogram in patients with acute myocardial infarction and in normal subjects. *Am J Cardiol* 1994;74:1113–18.
19. Leitch J, Basta M, Dobson A. QT dispersion does not predict early ventricular fibrillation after acute myocardial infarction. *Pacing Clin Electrophysiol* 1995;18:45–8.
20. Tomassoni G, Pisano E, Gardner L, Krucoff MW, Natale A. QT prolongation and dispersion in myocardial ischemia and infarction. *J Electrocardiol* 1998;30:187–90.
21. Niwa H, Maeda T, Shimoyama N, Tenda K, Yonemochi H, Saikawa T, Takaki R. Serial changes in QT interval during acute myocardial infarction: comparison of QT interval changes of reperfused cases and non-reperfused cases. *J Cardiol* 1993;23:41–50.
22. Szydlo K, Trusz-Gluza M, Drzewiecki J, Wozniak-Skowerska I, Szczogiel J. Correlation of heart rate variability parameters and interval in patients after PTCA of infarct related coronary artery as an indicator of improved autonomic regulation. *Pacing Clin Electrophysiol* 1998;21:2407–10.

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We must try to find ways to starve the terrorists of the oxygen of publicity on which they depend

Margaret Thatcher, British Conservative Prime Minister (1925–). Coinage of the phrase "oxygen of publicity" has been ascribed to Britain's then chief Rabbi, Lord Jakobovits