QT Dispersion and Viable Myocardium in Patients with Prior Myocardial Infarction and Severe Left Ventricular Dysfunction

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Background: QT dispersion (QTd) has been found to correlate to the amount of viable myocardium in patients with Q-wave myocardial infarction and well-preserved LV function. However, this relationship is unknown in patients with severe left ventricular dysfunction.

Methods: Thirty-four patients with prior large myocardial infarction and severe left ventricular dysfunction underwent Tc-99m sestamibi single photon emission cardiac tomography (SPECT) and F-18 fluorodeoxyglucose (FDG) SPECT. Viability was defined as a defect relative count density (DCD) of at least 20% greater on FDG SPECT. QTd, corrected QT dispersion (QTcd), and QT coefficient of variation (cv) in patients with viable myocardium was compared to those without viable myocardium in the infarct area.

Results: Thirteen patients were excluded from analysis for poor FDG images or inadequate ECG tracings. Of the remaining patients, 10 (48%) were found to have viability on FDG SPECT. QTd, QTcd, and QTcv in patients with viability were: 58 ± 22 ms, 61 ± 23 ms, and $4.81 \pm 1.76\%$, respectively, which did not differ significantly from those in patients without viability (QTd = 56 ± 14 ms, QTcd = 70 ± 16 ms and QTcv = $5.06 \pm 1.20\%$ [P = NS]). Moreover, neither FDG defect size, nor LVEF correlated with QTd.

Conclusions: This study indicates no relationship between QTd and viability in patients with myocardial infarction and severe left ventricular dysfunction. **A.N.E. 2002;7(1):53-59**

QT dispersion (QTd); FDG SPECT; myocardial viability; left ventricular dysfunction

Increased QT dispersion (QTd) on the 12-lead electrocardiogram (ECG) has been shown to be a predictor of malignant ventricular arrhythmia and sudden cardiac death (SCD).¹⁻¹⁰ However, the pathophysiologic basis for the dispersion of QT intervals is incompletely understood. Several studies have shown a direct relationship between QTd and the presence of myocardium ischemia,¹¹⁻¹⁹ and an association between QTd and the amount of viable myocardium has recently been suggested.²⁰ Schneider et al.²⁰ found that QTd is significantly less in patients with chronic Q-wave myocardial infarction (MI) and viable myocardium in the in-

farct area than those without viability on fluorine-18 fluorodeoxyglucose (FDG) positron emission tomography (PET). They also found that QTd may be used to predict left ventricular (LV) function recovery after revascularization therapy. However, all patients included in this study had well-preserved LV function with a mean ejection fraction (EF) of $55 \pm 16\%$.²⁰ Given the fact that patients with severe heart failure carry an annual mortality rate of 40-50%, it is a greater clinical concern to identify those patients with coronary artery disease and severe LV dysfunction with the potential for recovery after revascularization ther-

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apy. For this reason, we studied the relationship between QTd and myocardial viability in a group of patients with prior MI and severe LV dysfunction.

METHODS

Patients

From a group of patients with prior MI and severe LV dysfunction who were referred for stress test and assessment of myocardial viability, 34 patients (26 men, age 63 ± 10 years) were sequentially included in this study after giving written informed consent. The left ventricular ejection fraction (LVEF) was measured by gated sestamibi SPECT.²¹ All patients were required to have at least one large infarct area, involving $\geq 20\%$ of myocardium, and a severe ($< 40\%$ of maximal myocardial tracer uptake) fixed defect on Tc-99m sestamibi stress/rest SPECT. This threshold was selected because previous investigators have reported that defects of lesser severity may be predicted to be viable based upon either sestamibi or thallium myocardial count density alone.²² These defects involved a total of 35 vascular territories: 18 in the left anterior descending, 14 in the right coronary artery, and 3 in the left circumflex coronary artery territories. For assessment of myocardial viability, all patients underwent FDG SPECT within 3 weeks (except one, after 6 months) of the sestamibi study.

None of the patients had a recent episode of unstable angina, and none had significant valvular disease. Furthermore, no one was on amiodarone, sotalol, or any other drug that can prolong QT interval significantly. Patients were also excluded for poor FDG SPECT images, or for inadequate ECG tracing to perform QT analysis (atrial fibrillation and electronic paced rhythm), or because the end of the T wave could not be reliably identified in ≥ 6 ECG leads.

TC-99m Sestamibi SPECT

All patients underwent either exercise, dipyridamole or dobutamine-sestamibi perfusion SPECT, using a high-resolution low energy collimator. Resting imaging was acquired 1 hour following the intravenous administration of 9 mCi (333 MBq) of Tc-99m sestamibi for the single-day rest/stress protocol or 22 mCi (814 MBq) for the separate-day protocol. For the stress-gated study, 18 patients received a standard intravenous dipyridamole in-

fusion of 0.142 mg/kg/min infusion for 4 minutes, 2 patients underwent treadmill stress, and 1 patient had dobutamine stress testing. During peak exercise or 3 minutes following the termination of dipyridamole infusion, each patient received a 22 mCi (814 MBq) dose of Tc-99m sestamibi. Stress SPECT was then acquired 20 minutes after exercise or 1 hour after dipyridamole or dobutamine infusion. Gated SPECT was obtained by using standard acquisition and processing protocols: high resolution low energy collimator, 64 planar projections over a 180° arc, 20 seconds per projection, 64XD64 Butterworth filter, 0.52 critical frequency, power = 5.8 frames per cardiac cycle were acquired with a $\pm 50\%$ rhythm acceptance window. No patient included in the study experienced stress-induced reversible defect.

F-18 Fluorodeoxyglucose SPECT

Patients fasted overnight except for their medications and were given a solution of 100 gm of glucose the morning of the study. If the fasting blood sugar was over 140 mg/ml, insulin was also administered subcutaneously. Serum glucose levels were subsequently monitored every 10 minutes. When the serum glucose had declined, an intravenous dose of 10 mCi (370 MBq) fluorine-18 FDG was then administered intravenously. 360° SPECT was acquired 60 minutes post-infusion. A dual headed camera with detectors 180° opposed was used, equipped with an especially machined ultra-high energy collimator (General Electric Maxxus, General Electric Medical Systems, Milwaukee, WI, USA). The specifications were the following: the hole diameter 3.0 mm, the septal thickness 2.5 mm, and the septal length 60 mm. For 511 KeV photos, septal penetration was determined to be 14.08%. Image processing was optimized for the average count density of the FDG SPECT studies. A Butterworth filter with a critical frequency of 0.35 and power of 10 were used.

Image Analysis

Images of all resting Tc-99m sestamibi and FDG SPECT were reoriented along the short axis of the heart with slices of the same thickness. Side-by-side comparisons of the sestamibi images were compared to those of FDG SPECT by an experienced nuclear cardiologist, who was blinded to the QT dispersion measurements. Viability was defined as a defect relative count density (DCD),

which is a ratio of count in defect zone compared to normal, of at least 20% greater on FDG SPECT than on the rest sestamibi SPECT.

Electrocardiographic Analysis

QT interval measurement was done on hard copies of baseline 12-lead ECG obtained within 5 days of the sestamibi study and printed at 50 mm/sec paper speed.² All measurements were done manually by a single observer who was not aware of patients' clinical data and their FDG SPECT findings.

QT intervals were measured using hand-held calipers from the beginning of the inscription of the QRS complex until the end of T wave (the return of the T wave to the isoelectric line). If a U wave was present, the nadir of the curve between the T wave and U wave was identified as the termination of QT interval. Each QT interval was corrected for the patient's heart rate using the Bazett's formula: corrected QT (QTc) = $QT/\sqrt{RR}$. QTd was defined as the difference between the maximal and the minimal QT interval measured on each of the 12 leads on ECG. QTcd (corrected QTd) was calculated similarly using QTc intervals. The coefficient of variation of QT intervals (QTcv) was calculated according to the formula $QTcv = \text{standard deviation of QT intervals} \cdot 100/\text{mean QT interval on each 12 lead ECG}$.^{23, 24} "Adjusted" QTcd takes into account the numbers of leads measured and is the QTcd divided by the square root of number of leads measured.²³ In addition, the QT SD was calculated as the standard deviation (SD) of QT intervals measured in a single resting ECG.²³

Measurements were not made on leads in which termination of the T wave could not be clearly identified. But in all cases, a minimum of six measurable leads was required for inclusion in the study.²⁴ Ten or more leads could be analyzed in 13 patients (62%), 8 in 5 patients (24%), and 6 or 7 in 3 patients (14%).

Statistical Analysis

QTd parameters from patients with viable tissue were compared to those from patients without viable tissue on FDG SPECT. All data are presented as mean $\pm$ SD. Student's *t* test was used to compare group means. The null hypothesis was rejected at the 95% confidence level, indicating a probability value of $P < 0.05$ as significant. The Pearson correlation coefficient was calculated to estimate the

correlation between QTd parameters and FDG defect sizes and patients' LVEF.

RESULTS

Of the original 34 patients, 21 remained in the study, after exclusion of 7 patients with poor FDG SPECT images and 6 with poor ECG tracing for QT analysis (3 with atrial fibrillation, 1 with electronic paced rhythm, and 2 with less than six measurable leads). Of these 21 patients, 10 patients (48%) were found to have viability on FDG SPECT with a mean DCD of 72.6 ± 30.8 . The LVEF measured by gated sestamibi SPECT in 20 of these patients was $28 \pm 10\%$.²¹ The LVEF in 1 patient could not be obtained secondary to technical difficulties. Clinical data of patients grouped accordingly to the presence or absence of viability on FDG SPECT were similar and are shown in Table 1. The global LVEF in patients with viable tissue in the infarct area was $31 \pm 9\%$ and in those without viable tissue in the infarct area was $25 \pm 10\%$. The defect size by sestamibi SPECT in the viable myocardium group was 810 ± 374 and in the non-viable group was 908 ± 426 ($P = \text{ns}$). Similarly, no difference was found in the distribution of the defect location by coronary artery territory within the two groups. The FDG defect size in the viable myocardium group also did not differ significantly from the non-viable group.

QTd, QTcd, and QTcv in patients with viability was 58 ± 22 ms, 61 ± 23 ms, and $4.81 \pm 1.76\%$, respectively, which did not differ significantly from those in patients without viability, 56 ± 14 ms, 70 ± 16 ms, and $5.06 \pm 1.20\%$ ($P = \text{ns}$, Table 2). There was no correlation between QTd and FDG defect size ($r = 0.20$, $P = \text{ns}$) as well as between QTd and LVEF ($r = -0.10$, $P = \text{ns}$), as shown in Figures 1 and 2.

DISCUSSION

The results of this study show that in patients with prior MI and severe LV dysfunction, QT dispersion parameters were independent of the amount of viable tissue as carefully assessed by FDG SPECT.

Identification of patients with viable myocardium in the infarct region is important for clinical decision making regarding future revascularization therapy. Several techniques with different sensitivity and specificity are being used to detect viable

Table 1. Clinical Data of Patients Grouped According to Presence of Viability by FDG SPECT

Clinical Features	Group with Viable Myocardium (n = 10)	Group without Viable Myocardium (n = 11)	P Value
Age (years)	66 ± 12	61 ± 6	NS
Male gender	8	9	NS
LVEF (%)	31 ± 9	25 ± 10	NS
FDG DCD	72.6 ± 30.8	45.9 ± 17.3	0.03
FDG defect size	667 ± 418	928 ± 400	NS
Sestamibi SPECT defect size	810 ± 374	908 ± 426	NS
Sestamibi SPECT defect location			NS
LAD	8	10	
LCx	2	1	
RCA	7	7	

DCD = Defect relative count density, a ratio of fluorine-18 FDG uptake in defect zone compared to normal; FDG = F-18 fluorodeoxyglucose; LAD = Left anterior descending artery; LCx = Left circumflex artery; LVEF = Left ventricular ejection fraction; RCA = Right coronary artery; SPECT = Single photon emission cardiac tomography.

myocardium. Among them, FDG PET has the highest sensitivity and specificity values for the identification of viable myocardium. The sensitivity values are between 78 and 96%, and the specificity values are between 69 and 92%.^{25, 26} However, this technique is very expensive and has very limited clinical availability. Several studies compared FDG PET and FDG SPECT for the assessment of myocardial viability, and found that FDG SPECT can provide equivalent information.²⁷⁻³¹ Other available techniques to evaluate the presence of myocardial viability include Thallium 201 scintigraphy and low dose dobutamine echocardiography,³²⁻³⁶ but both in general have lower sensitivity and specificity than FDG PET, and do not offer significant technical advantages over FDG SPECT.

Finding a simpler and more widely available technique with the capability to detect myocardial viability could have significant impact on clinical decision making. QTd on 12-lead ECG has been proposed by Schneider et al.²⁰ as a potential practical method for detection of myocardial viability.

This is a startling concept, considering the low cost and availability of the 12-lead ECG. The investigators found that QTd was significantly lower (53 ± 20 ms vs 94 ± 24 ms, $P < 0.0001$) in patients with a substantial amount of viable myocardium in the infarct region compared to those with only minimal residual viability in a group of patients with chronic Q wave MI. All patients who participated in this study underwent revascularization by either CABG or PTCA, and QTd (before revascularization) was significantly lower in those who showed improvement of LVEF after revascularization. This suggests that QTd would have the potential to predict functional recovery after revascularization therapy. All patients in this study had angiographically documented high-grade stenosis of the infarct-related coronary artery, but overall good LV function, with a mean EF of more than 50%.

It has been estimated that more than 2 million patients in the United States suffer from heart failure, with coronary artery disease being the most important etiology. There is an average annual

Table 2. QT Parameters of Patients Grouped According to Presence of Viability by FDG SPECT

QTd Parameters	Group with Viable Myocardium (n = 10)	Group without Viable Myocardium (n = 11)	P value
QTd	58 ± 22 ms	56 ± 14 ms	NS
QTcd	61 ± 23 ms	70 ± 16 ms	NS
QTcv	4.81 ± 1.76%	5.06 ± 1.20%	NS
QT SD	20 ± 9 ms	19 ± 4 ms	NS
Adjusted QTcd	19 ± 8 ms	23 ± 5 ms	NS

QTd = QT dispersion; QTcd = Corrected QT dispersion; QTcv = Coefficient of variation of QT intervals; QT SD = Standard deviation of QT intervals.

Figure 1. Correlation of QT dispersion and left ventricular ejection fraction by gated sestamibi SPECT. No correlation was found.

mortality rate of 40-50% in patients with severe heart failure, and of these, nearly one-half of the patients die from progressive pump failure and one-half from sudden cardiac death. The potential application of QTd to identify those patients with severe ischemic cardiomyopathy and significant viable myocardium, a reversible condition by revascularization therapy, has great potential to impact on public health, by predicting, and perhaps preventing, death from both pump failure and sudden cardiac death. Unfortunately, no difference in QTd was found in our study between those with substantial amount of viable myocardium in the infarct area and those without. Furthermore, no correlation was found in our study between LVEF or FDG defect size and QTd parameters. In contrast to the study by Schneider et al, all patients in our study had much more severe global LV dysfunction, with an EF of $28 \pm 10\%$.

The reasons for these inconsistent results are not clear, but it is probably not entirely due to the differences in LV dysfunction. Conflicting data exist in the relationship between QTd and LVEF. Pye et al.¹⁰ have previously shown a significant correlation of LV dysfunction and QTd in a mixed population of patients with CAD and dilated cardiomyopathy (DCM). However, most other data have shown no correlation between LVEF and QTd, including the present study and several previously published studies.^{3,7,18,37} Based on these observations, it is unlikely that the different results found in our study are due to the lower LVEF in our patients.

It is interesting to note that Schneider et al. in-

cluded patients from a group of patients with angina, angina equivalent, or abnormal exercise test.²⁰ In contrast, patients included in our study were believed to be free of significant ischemia and had only fixed defect on stress test. Thus, it may be that an increased QTd is more related to the presence of myocardial ischemia than the degree of LV dysfunction. Numerous studies have previously shown this relationship.¹²⁻¹⁹ An abnormal increase in QTd has been described during exercise,¹⁶⁻¹⁸ by incremental atrial pacing,^{12,13} as well as in dipyridamole- and dobutamine-induced reversible ischemia.¹⁶ Furthermore, even daily activities not causing significant ST segment changes may increase QTd in the presence of myocardial ischemia,¹⁸ which could be clinically silent.

Based on these observations, it is likely that the correlation between QTd and myocardial viability previously observed is due to the different degrees of chronic resting ischemia, and in the absence of significant resting ischemia, no correlation between QTd and myocardial viability is found as in our study.

There are, however, several limitations to our study that need to be considered. First, we used FDG SPECT, instead of FDG PET, for assessment of myocardial viability. However, it is unlikely this would have caused the negative result, given that many studies have shown that FDG SPECT and FDG PET have comparable results for assessment of myocardial viability, especially in patients with LVEF = to 35%.³⁰ Second, the number of patients studied is relatively small. Third, QTd measurements were done by a single observer. However, QTd parameters were measured blindly and were remarkably similar in the viable and nonviable

Figure 2. Correlation of QT dispersion and FDG SPECT defect size. No correlation was found.

groups. Finally, a common limitation of all QTd studies is the difficulty involved in the measurement of QT intervals. To avoid this, only ECGs with at least six reliably measurable leads were included. No difference was found however in the "adjusted" QTcd, which corrects for number of leads analyzed.

In conclusion, this study indicates no correlation exists between QTd parameters and viability in patients with a large MI and severe left ventricular dysfunction. QT dispersion does not appear useful for assessment of myocardial viability.

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