Enhanced Detection of Ischemic but Viable Myocardium by QT Interval Dispersion on Treadmill Exercise Electrocardiograms of Patients with Healed Anterior Wall Myocardial Infarcts

YASUSHI KOIDE, M.D., MASAYUKI YOTSUKURA, M.D., KAZUHIRO TAJINO, M.D., HIDEAKI YOSHINO, M.D., KYOZO ISHIKAWA, M.D., FACC
Kyorin University, School of Medicine, Tokyo, Japan

Summary

Background: The presence of ischemic but viable myocardium in infarcted areas is an important indication for coronary revascularization, but is often difficult to detect with the use of treadmill exercise electrocardiography (ECG).

Hypothesis: QT interval dispersion (QTd) is a sensitive method for detecting myocardial ischemia and may improve the accuracy of treadmill exercise ECG testing for detecting ischemic but viable myocardium in infarcted areas.

Methods: Forty-five patients with Q-wave anterior wall myocardial infarctions who underwent treadmill exercise ECG, exercise reinjection thallium-201 (^{201}Tl) scintigraphy, radionuclide angiocardiology, and coronary angiography 1 month after infarction were enrolled in this study. The presence of viable myocardium in the infarct area was determined by exercise reinjection ^{201}Tl scintigraphy. Patients who had no redistribution in the infarct area after reinjection were included in Group 1, and those with redistribution were included in Group 2.

Results: QTd immediately after exercise, and the difference between QTd before and immediately after exercise, were significantly greater in Group 2 than in Group 1. The sensitivity, specificity, and accuracy of conventional ST-segment depres-

sion criteria for detecting viable myocardium in the infarct area were 48, 64, and 56%, respectively. The measurement of QTd immediately after exercise (abnormal: ≥ 70 ms; normal: < 70 ms) improved the sensitivity, specificity, and accuracy to 78, 82, and 80%, respectively.

Conclusions: This novel diagnostic method using QTd-based criteria significantly improves the clinical usefulness of treadmill exercise ECG testing for detecting ischemic but viable myocardium in infarct areas in patients with healed Q-wave anterior wall myocardial infarctions.

Key words: myocardial infarction, exercise electrocardiogram, QT interval dispersion, viable myocardium

Introduction

The treatment for myocardial infarction (MI) has undergone striking changes with the recent introduction of new approaches to coronary intervention, including percutaneous transluminal coronary angiography (PTCA). The presence of ischemic but viable myocardium in the infarct area is an important determinant of the potential benefit of therapeutic revascularization. However, the detection of ischemic but viable myocardium by treadmill exercise electrocardiography (ECG) using ST-segment criteria is not completely sensitive or specific. Some authors claim that ST-segment elevation in infarct-related leads is due to ischemic but viable myocardium,¹ while others propose that it is caused by abnormal wall motion.² Furthermore, reciprocal ST-segment changes might cause ST-segment depression in noninfarct-related leads.³

QT interval dispersion (QTd) measured on standard 12-lead ECGs is an indicator of heterogeneity of local ventricular repolarization.⁴ Several studies have reported that heterogeneity of the ventricular repolarization phase increases during stress test because of transient myocardial ischemia. This increase is reflected in a prolongation in QTd.^{5–7} We therefore sought to determine whether exercise-induced changes in QTd can

Address for reprints:

Kyozo Ishikawa, M.D.
Second Department of Internal Medicine
Kyorin University School of Medicine
6-20-2 Shinkawa, Mitaka-shi
Tokyo, 181-8611, Japan

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serve as an indicator of ischemic but viable myocardium in infarct areas.

Methods

Study Population

In all, 45 consecutive patients [41 men and 4 women; mean age $\pm$ standard deviation (SD) 60 ± 9 years] with healed Q-wave anterior wall MI and single-vessel coronary artery disease, who gave informed consent to participate in this study, were enrolled. All patients underwent treadmill exercise ECG, radionuclide angiocardigraphy, reinjection exercise thallium-201 (^{201}Tl) scintigraphy, and coronary angiography approximately 1 month after infarction. No patient had treatment with antiarrhythmic drugs that affect the QT interval, atrial fibrillation, intraventricular conduction disturbance, and a history of previous MI.

The diagnosis of acute MI was based on the presence of typical chest pain lasting ≥ 30 min which was unresponsive to nitrates, the presence of ECG ST-segment elevations ≥ 0.1 mV in at least two limb leads or ≥ 0.2 mV in at least two precordial leads, and a plasma creatine kinase MB fraction activity at least twice the upper limit of normal. The diagnosis of Q-wave MI was based on the presence of persistent abnormal Q waves on the 12-lead ECG.

Reperfusion Therapy

Acute reperfusion therapy was performed within 6 h of the onset of chest pain with an intravenous infusion of 24 million units of recombinant tissue-plasminogen activator (alteplase) in two patients, and an only intracoronary administration of 6 million units of alteplase in six patients. Primary or rescue PTCA was performed in 13 patients using standard methods. The remaining 24 patients were treated conservatively without reperfusion therapy.

Coronary Angiography

Selective coronary cineangiography in multiple projections was performed using the Judkins technique 34 ± 8 days after infarction. Coronary angiograms were independently reviewed by two observers. Coronary stenoses were quantified visually and with the use of calipers and were classified according to criteria established by the American Heart Association. Luminal narrowing that was $\geq 75\%$ of the cross-sectional area was considered to represent a hemodynamically significant coronary artery lesion. When more than one lesion was present, the site of the culprit lesion was determined based on the presence of complete obstruction or the angiographic characteristics of the lesion (the presence of either residual thrombus or ulcerated plaque). When the findings of the two observers differed, the opinion of a third physician was used to resolve the difference. The presence of collateral circulation was defined as full contrast enhancement via collateral vessels.

Radionuclide Angiocardiography

Multigated radionuclide angiocardiography was performed 27 ± 7 days after infarction. A dose of 740 to 1110 MBq of $^{99\text{m}}\text{Tc}$ pertechnetate was injected intravenously. Gated cardiac imaging was performed with a single-crystal gamma camera (model GCA-90B, Toshiba Co., Tokyo, Japan), fitted with a low-energy, all-purpose, parallel-hole collimator, and interfaced to a dedicated minicomputer (model GMS-55U, Toshiba) operating in list mode. The left anterior oblique projection that best displayed the interventricular septum was used (approximately 45° with 10° of caudal angulation). After acquisition, the data were organized into an image sequence consisting of 20 frames per cardiac cycle using a 64×64 matrix with a $1.5 \times$ zoom. List mode data were acquired for a total of 3000 K counts. Left ventricular ejection fraction was determined from the global time-activity curve, using a signal semiautomatic left ventricular region of interest drawn on the functional phase image.

Reinjection Exercise Thallium-201 Scintigraphy

Reinjection exercise ^{201}Tl scintigraphy was performed 30 ± 7 days after infarction. The exercise test was performed on a bicycle ergometer with the patient in upright position. The protocol began at 25 W and increased by 25 W increments every 3 min. At peak exercise, 100 MBq (2.7 mCi) of ^{201}Tl was injected, and exercise was continued for 1 more min. The stress ^{201}Tl scan was obtained within 10 min after tracer injection. Three hours later, a delayed ^{201}Tl scan was obtained. Immediately after the delayed scan, 40 MBq (1.1 mCi) of ^{201}Tl was reinjected at rest, and a reinjection ^{201}Tl scan was obtained 10 min later. For each ^{201}Tl scan, single-photon emission computed tomography was performed using 32 projection images, each lasting 30 s, through a 180° arc. Left ventricular myocardial images were divided into nine segments to assess the ^{201}Tl uptake for each single-photon emission computed tomogram. The standard reinjection digital images were normalized to the peak myocardial activity for each projection (determined from the initial image set). Normalized perfusion images were presented in pairs (initial and reinjection) in a blinded fashion to two experienced observers. The images were scored qualitatively using a 4-point scale (0 = absent perfusion to 3 = normal perfusion). Each segment was classified as either having normal perfusion, rest redistribution, or a persistent defect. Rest redistribution was defined as an increase in the score by one or more points on the reinjection images compared with the post-exercise images.

Treadmill Exercise Electrocardiogram

Maximum symptom-limited treadmill exercise testing was performed using a standard Bruce protocol 28 ± 5 days after infarction. Patients receiving beta-adrenergic blocking drugs were instructed to taper the doses so that no such drugs were being taken 2 days before testing. Heart rate, blood pressure, and 12-lead ECG were recorded at rest and every min during

exercise and recovery until the ECG returned to baseline. In addition, three ECG leads (II, V₁, and V₅) were monitored continuously. A stress test system (ML-4500, Fukuda Denshi Inc., Tokyo, Japan) was used to record trends in heart rate, the computer-averaged ECG, and ST-segments for the three monitored leads. Blood pressure was measured by manual sphygmomanometry. Exercise testing was terminated for symptoms of angina pectoris, fatigue, or dyspnea; a decrease in systolic blood pressure ≥ 20 mm Hg; three or more consecutive premature ventricular complexes; significant ST-segment depression; or attainment of the age-related maximal heart rate (220 minus the age in years). Significant ST-segment changes during the stress test were defined as horizontal or downsloping ST-segment depressions ≥ 1 mm below the baseline 0.06 s after the J point in the noninfarct-related leads, or ST-segment elevation ≥ 1 mm above the baseline 0.06 s after the J point in the infarct-related leads.

Measurement of the QTd: The QT interval was measured manually, using calipers, in as many limb and precordial leads as possible (a minimum of 8 and a mean of 10.4 leads) from the 12-lead ECG. The QT interval was calculated from the onset of the QRS complex to the point of the return of the T wave to the isoelectric line, at baseline, and immediately after exercise, using a paper speed of 50 mm/s and a gain of 10 mm/mV. When the T wave was fused with the U or P wave, a straight line was drawn tangential to the downstroke of the T wave, and its intersection with the baseline was defined as the end of the T wave. The PR segment was used as the baseline to obviate the difficulty in identifying the end of the T wave in the presence of ST-segment depression. When the end of the T wave could not be identified with certainty, the lead was excluded from analysis. Each measurement represents the mean value of three consecutive beats. The QTd was defined as the difference between the maximum and minimum QT intervals. The Δ QTd was defined as the difference between the QTd measured before and immediately after exercise. QT values were corrected for the effects on heart rate using Bazett's formula [corrected QT interval (QTc) = QT/square root of the R-R interval].⁸ The QTcd was determined in a manner similar to that for the uncorrected QTd. The QT interval was measured while the investigators remained blinded to clinical and coronary angiographic data.

Statistics

All data are reported as the mean $\pm$ SD. The results of the exercise ECG tests were compared with the data derived from reinjection exercise ²⁰¹Tl scintigraphy to determine the sensitivity, specificity, and predictive value of an abnormal test result (positive predictive value), the predictive value of a normal result (negative predictive value), and diagnostic accuracy of the exercise ECG variables. The differences between continuous variables were assessed using the two-tailed Student's *t*-test. The differences between categorical variables were assessed using the chi-square test or Fisher's exact test, where appropriate. The test sensitivities and specificities for different criteria were compared by McNemar's

test. Simple regression analysis was used to assess the relationship between the change in the QTd immediately after exercise and the redistribution score after reinjection. A *p* value < 0.05 was considered statistically significant.

The reproducibility of the QTd measurements was tested in 30 randomly selected patients before and immediately after exercise by two independent blinded observers. Agreement between the two observers was assessed using the Bland-Altman method.⁹

Results

Patients were divided into two groups based on the results of reinjection exercise ²⁰¹Tl scintigraphy performed approximately 1 month after infarction: 22 patients were found to have fixed perfusion defects in the infarct area without evidence of redistribution after reinjection (Group 1), and 23 patients had ischemic, but viable myocardium in the infarct area, as demonstrated by redistribution after reinjection (Group 2).

Patient Characteristics

The clinical characteristics of the patients are summarized in Table I. There were no significant differences between the two groups with respect to age, gender, coronary risk factors, therapy (medication, reperfusion therapy), Killip class, maximum plasma creatine kinase value, or left ventricular ejection fraction. However, the frequency of postinfarct angina was significantly higher in Group 2 than in Group 1 (*p* = 0.01).

Coronary angiography was performed about 1 month after infarction and showed that the incidence of clinically relevant residual stenosis was higher in Group 2 than in Group 1. In contrast, the incidence of total coronary occlusion was slightly higher in Group 1. There was no significant difference with respect to the presence of collateral circulation between the two groups.

Treadmill Exercise Electrocardiogram

The treadmill exercise ECG findings obtained approximately 1 month after infarction are summarized in Table II. There was no significant difference between the two groups with respect to heart rate or systolic blood pressure immediately before or after exercise or with regard to exercise duration. The incidence of chest pain during exercise was significantly higher in Group 2 than in Group 1 (*p* = 0.04). No significant differences were observed between the two groups with respect to the maximum ST-segment elevation in the infarct-related leads or maximum ST-segment depression in the noninfarct-related leads. There was no significant difference between the two groups with respect to pre-exercise QTd. However, the QTd immediately after exercise and the Δ QTd were significantly greater in Group 2 than in Group 1 (*p* < 0.001). The QTcd differences were similar to those for the QTd in the two groups.

TABLE I Clinical characteristics of the study patients

	Group 1	Group 2	p Value
No. of patients	22	23	
Age (years)	62 ± 9	57 ± 9	0.08
Male/female	20/2	21/2	0.99
Clinical history			
Hypertension (%)	8 (36)	10 (44)	0.63
Diabetes mellitus (%)	6 (27)	6 (26)	0.93
Hyperlipidemia (%)	7 (32)	7 (30)	0.92
Cigarette smoking (%)	18 (82)	13 (57)	0.11
Treatment			
Nitrates (%)	12 (55)	12 (52)	0.87
Beta blockers (%)	10 (45)	12 (52)	0.65
Calcium antagonists (%)	11 (50)	11 (48)	0.88
Angiotensin-converting enzyme inhibitors (%)	12 (55)	11 (48)	0.65
Reperfusion therapy			
IVT (%)	1 (5)	1 (4)	0.99
ICT (%)	2 (9)	4 (17)	0.41
Primary or rescue PTCA (%)	5 (23)	8 (35)	0.37
Kilip class (≥ II) (%)	7 (32)	9 (39)	0.68
Post MI angina (%)	1 (5)	8 (35)	0.01
Maximum CK release (IU/l)	4819 ± 2383	4290 ± 2558	0.47
LVEF (%)	47 ± 8	50 ± 11	0.45
Stenosis severity (≥ 75%)			0.01
75% (%)	1 (5)	6 (32)	
90–99% (%)	8 (38)	11 (58)	
100% (%)	12 (57)	2 (10)	
Presence of collateral circulation (stenosis severity ≥ 75% (%))	3 (14)	4 (21)	0.68

Values are mean ± standard deviation or number (%) of patients in each category.

Abbreviations: IVT = intravenous thrombolysis, ICT = intracoronary thrombolysis, PTCA = percutaneous transluminal coronary angioplasty, MI = myocardial infarction, CK = creatine kinase, LVEF = left ventricular ejection fraction, hypertension = blood pressure ≥ 160/95 mmHg, diabetes mellitus = fasting blood sugar ≥ 140 mg/dl, hypercholesterolemia = total cholesterol ≥ 220 mg/dl.

TABLE II Treadmill exercise electrocardiogram findings

	Group 1	Group 2	p Value
No. of patients	22	23	
Exercise duration (s)	348 ± 127	354 ± 109	0.86
Chest pain during treadmill exercise test	1 (5%)	7 (30%)	0.04
Characteristics before exercise			
Heart rate (beats/min)	68 ± 9	72 ± 14	0.27
Systolic blood pressure (mmHg)	112 ± 12	113 ± 10	0.77
QTd (ms)	57 ± 11	60 ± 8	0.28
QTcd (ms)	60 ± 12	65 ± 13	0.16
Characteristics immediately after exercise			
Heart rate (beats/min)	123 ± 11	122 ± 8	0.85
Systolic blood pressure (mmHg)	142 ± 18	146 ± 21	0.51
Maximum ST-segment elevation in infarct-related leads (mm)	1.0 ± 0.9	1.3 ± 1.0	0.40
Maximum ST-segment depression in noninfarct-related leads (mm)	0.4 ± 0.5	0.7 ± 0.7	0.12
QTd (ms)	55 ± 15	77 ± 17	<0.0001
QTcd (ms)	79 ± 24	110 ± 25	0.0001
ΔQTd (ms)	−2 ± 13	17 ± 16	0.0002
ΔQTcd (ms)	19 ± 20	45 ± 23	0.0004

Values are mean ± standard deviation or number (%) of patients in each category.

Abbreviations: ECG = electrocardiogram, QTd = QT interval dispersion, QTcd = corrected QT interval dispersion, ΔQTd = difference in QT interval dispersion before and immediately after exercise, ΔQTcd = difference in corrected QT interval dispersion before and immediately after exercise.

FIG. 1 Reproducibility of QTd measurements before (A) and immediately after (B) exercise in 30 randomly selected study patients, as assessed by the Bland-Altman method. There are < 30 points because of data overlap (B). QTd = QT interval dispersion, SD = standard deviation.

Reproducibility of QTd Measurements

The mean difference between two observers for the measurement of QTd before exercise ($n = 30$) was 9.1 ± 17.3 ms with a correlation coefficient for the independent measurements of 0.06 ($p = 0.74$). Immediately after exercise, the mean difference between observers was 4.5 ± 9.1 ms with a correlation coefficient of 0.82 ($p < 0.0001$). The mean value for the absolute differences in QTd measurements for the two observers was 16.9 ms before and 8.9 ms immediately after exercise (Fig. 1).

Relationship between QTd Immediately after Exercise and Perfusion Defect Score after ReInjection

The relationship between QTd immediately after exercise and the redistribution score after reinjection was determined. A significant correlation ($r = 0.740$) was found between the two variables ($p < 0.0001$; Fig. 2).

Accuracy of Detection of Ischemic but Viable Myocardium in the Infarct Area Based on QTd Immediately after Exercise

Based on the sensitivity and specificity data presented in Figure 3, we identified patients with ischemic but viable myocardium in the infarct area based on a QTd ≥ 70 ms immediately after exercise, a QTcd ≥ 100 ms immediately after exercise, a Δ QTd ≥ 10 ms, and a Δ QTcd ≥ 30 ms. The diagnostic values for exercise QTd, QTcd, conventional ST-segment depression, and ST-segment elevation are summarized in Table III. The use of a QTd ≥ 70 ms immediately after exercise as a predictor of ischemic but viable myocardium had a sensitivity of 78%, a specificity of 82%, a positive predictive value of 82%, a negative predictive value of 78%, and an accuracy of 80%. The sensitivity and specificity of this new criterion were significantly higher than those for methods based on conventional ST-segment depression in noninfarct-related leads and ST-segment elevation in infarct-related leads ($p < 0.05$).

FIG. 2 Relationship between the QTd immediately after exercise and the redistribution score after reinjection. There are < 45 points because of data overlap. QTd = QT interval dispersion.

Discussion

Our study shows that changes in the QTd during treadmill exercise testing can be used to detect the presence or absence of ischemic but viable myocardium in the infarct area in patients with MI.

An increase in the QTd reflects heterogeneity of local ventricular repolarization,⁴ and such increases are associated with an increased risk of ventricular arrhythmias.¹⁰⁻¹⁴ The association between QTd and the presence of viable myocardium following MI has been studied extensively. The QTd is lower in patients who have responded to thrombolytic therapy than in those who have not achieved thrombolysis.¹⁵ Patients with viable myocardium in the infarct region have lower QTd values than patients without viable myocardium in the infarct region, and the QTd is considered a sensitive indicator of the presence of viable myocardium.¹⁶ Several studies have evaluated the relationship between QTd and the presence of transient myocardial ischemia in patients with no prior history of myocardial infarction.⁵⁻⁷ The QTd has been shown to increase in response to transient myocardial ischemia,^{5,6} and a significant positive correlation has been demonstrated between the QTd and the

TABLE III Diagnostic value of treadmill exercise electrocardiogram for the detection of residual ischemia after myocardial infarction

	Conventional ST-segment depression criteria	ST-segment elevation Criteria (≥ 1 mm: positive)	QTd immediately after exercise Criteria (≥ 70 ms: positive)	QTd immediately after exercise Criteria (≥ 100 ms: positive)	Δ QTd criteria (≥ 10 ms: positive)	Δ QTcd criteria (≥ 30 ms: positive)
Sensitivity, %	48 (11/23)	57 (13/23)	78 (18/23) ^b	74 (17/23) ^a	70 (16/23)	70 (16/23)
Specificity, %	64 (14/22)	59 (13/22)	82 (18/22) ^b	77 (17/22)	73 (16/22)	77 (17/22)
Positive predictive value, %	58 (11/19)	59 (13/22)	82 (18/22)	77 (17/22)	73 (16/22)	76 (16/21)
Negative predictive value, %	54 (14/26)	57 (13/23)	78 (18/23)	74 (17/23)	70 (16/23)	71 (17/24)
Diagnostic accuracy, %	56 (25/45)	58 (26/45)	80 (36/45)	76 (34/45)	71 (32/45)	73 (33/45)

^a $p < 0.05$ vs. conventional ST-segment depression criteria.^b $p < 0.05$ vs. conventional ST-segment depression criteria and ST-segment elevation criteria. Abbreviations as in Table II.

FIG. 3 Sensitivity and specificity curves for QTd during treadmill exercise testing. (A) Based on the QTd immediately after exercise (abnormal: ≥ 70 ms), the sensitivity and specificity of exercise electrocardiographic (ECG) testing were 78 and 82%, respectively. (B) Based on the QTcd immediately after exercise (abnormal: ≥ 100 ms), the sensitivity and specificity of exercise ECG testing were 74 and 77%, respectively. (C) Based on the Δ QTd (abnormal: ≥ 10 ms), the sensitivity and specificity of exercise ECG testing were 70 and 73%, respectively. (D) Based on the Δ QTcd (abnormal: ≥ 30 ms), the sensitivity and specificity of exercise ECG testing were 70 and 77%, respectively. Abbreviations as in Table II. —■— = Sensitivity, —●— = specificity.

degree of ischemia.⁷ These previous reports suggest that transient myocardial ischemia occurring in viable myocardium within the infarct area may contribute to changes in the QTd.

Recently, Naka *et al.*¹⁷ studied the relationship between changes in the QTd during exercise and the presence of ischemic but viable myocardium in the infarct area in patients with a prior history of anterior wall MI. Patients without viable myocardium in the ischemic region had no significant change in the QTd after exercise. In contrast, patients with ischemic but viable myocardium had a significant increase in the QTd after exercise. These previous results support the hypothesis that the QTd increases in response to exercise-induced transient ischemia in viable myocardium in the infarct area. However, the diagnostic sensitivity of the QTd in assessing the presence of ischemic but viable myocardium, and the relationship between the QTd and the extent of infarction have yet to be determined. A methodologic limitation of the study by Naka *et al.* was the use of conventional exercise ²⁰¹Tl myocardial scintigraphy, which often underestimates

the presence of ischemic but viable myocardium.¹⁸ We therefore performed exercise ²⁰¹Tl myocardial scintigraphy using a reinjection technique, which is more sensitive for detecting ischemic but viable myocardium,¹⁹ to determine the relationship between changes in the QTd after exercise and the presence of ischemic but viable myocardium. We also assessed the sensitivity of this technique in detecting ischemic but viable myocardium.

We observed no exercise-induced increases in the QTd in patients without evidence of viable myocardium. In contrast, in patients with ischemic but viable myocardium, exercise produced an increase in the QTd. Moreover, when a QTd value ≥ 70 ms immediately after exercise was defined as a criterion for ischemic but viable myocardium in the infarct area, the resulting diagnostic sensitivity was significantly better than that based on the presence of ST-segment depressions in the noninfarct-related leads or ST-segment elevations in the infarct-related leads. We also found a significant positive correlation between the redistribution score after reinjection and the QTd immediately after exercise. Although the mechanisms responsible for our findings are unknown, increases in the QTd most likely result from localized abnormal wall motion caused by transient myocardial ischemia and increased heterogeneity of ventricular repolarization.

The reproducibility of measurements of resting QTd has been questioned.^{20, 21} The reproducibility of the measurements of resting QTd was relatively poor in our study; however, the QTd measurements obtained immediately after exercise were highly reproducible. The improved reproducibility of QTd measurements immediately after exercise is probably due to exercise-induced T-wave normalization, increased T-wave amplitude, or better definition of the terminal portion of the T wave immediately after exercise. When the T wave is inverted or its amplitude is low, the QT interval may be difficult to measure. However, T waves frequently normalize, the T-wave amplitude frequently increases, and the terminal portion of the T wave sharpens immediately after exercise in patients with MI. It is possible that in the presence of normalized T waves, increased T-wave amplitude, and better T-wave definition immediately after exercise, the reproducibility of QTd measurements improves. The greater reproducibility of the QTd immediately after exercise makes this variable a useful diagnostic indicator for detecting viable myocardium in the infarct area.

The sensitivity of detection of ischemic but viable myocardium in the infarct area based on the presence of ST-segment depression in the noninfarct-related leads was relatively poor in our study, which is consistent with previous studies using positron emission tomography.^{1, 22}

Clinical Limitations

We had no opportunity to validate the usefulness of QTd for detecting ischemic but viable myocardium using positron emission tomography. However, recently, Marin-Neto *et al.*²³ have reported that reinjection exercise ²⁰¹Tl scintigraphy and

positron emission tomography were concordant in 98 and 93% of myocardial regions, respectively, in patients with moderate and severe left ventricular dysfunction. Then, we believe that there is no problem in our results obtained by reinjection ²⁰¹Tl scintigraphy instead of using positron emission tomography. Only patients with single-vessel coronary artery disease were studied to eliminate the effects of stenoses at other sites on the QTd. Therefore, further studies are necessary to determine whether the proposed criteria for ischemic but viable myocardium in the setting of single-vessel disease can also be used in patients with multivessel disease. The analysis of QTd is sometimes difficult because the end of the T wave cannot always be reliably determined, leading to spurious measurements of QTd. In cases where we could not accurately determine the end of the T wave, these leads were excluded from analysis. Our study group consisted primarily of men, and therefore the results may not apply to women. Although our conclusions are supported by statistical analyses, only a limited number of patients were studied. Therefore, the validity of our findings should be confirmed in a larger number of patients.

Conclusions

We have developed an improved method for detecting ischemic but viable myocardium in infarct areas in patients with healed Q-wave anterior wall myocardial infarctions using treadmill exercise ECG. The diagnostic accuracy for detecting ischemic but viable myocardium was increased markedly by measuring the QTd immediately after exercise. The sensitivity and specificity of a QTd ≥ 70 ms immediately after exercise for detecting ischemic but viable myocardium were 78 and 82%, respectively. This result suggests that QTd measurements immediately after exercise may be a replacement for the nuclear reinjection technique for detecting ischemic but viable myocardium.

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