Dispersion of ventricular repolarization is determined by the presence of myocardial viability in patients with old myocardial infarction

A dobutamine stress echocardiography study

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Aims The study sought to investigate the relationship of myocardial viability detected by dobutamine stress echocardiography to changes of QT dispersion and to the presence of arrhythmias during dobutamine infusion in patients with old myocardial infarction. We also examined whether patency of the infarct-related artery is associated with the presence of myocardial viability and QT dispersion.

Background QT dispersion and myocardial variability have been associated with the presence of arrhythmias during late post infarction but not during dobutamine stress. Restoration of anterograde coronary flow has beneficial effects on ventricular systolic function and repolarization, suggesting that the extent of viable myocardium may determine ventricular repolarization.

Methods Seventy-five patients with previous myocardial infarction were studied in a low dose (up to 20 μg·kg⁻¹·min⁻¹) dobutamine stress echocardiography study. ECGs were obtained at rest and peak stress for measurement of QT intervals. The presence of ventricular arrhythmias (Lown grade >1b) during stress was noted. A reduction in the total wall motion score of the left ventricle at peak stress confirmed the presence of myocardial viability.

Results Dobutamine infusion increased QT dispersion in all patients (P<0.01). Patients with myocardial viability had a lower resting QT dispersion (P<0.05) and a greater increase in QT dispersion% (P<0.01) than patients without. The combination of a resting QT dispersion <65 ms or an increase in QT dispersion >30% predicted viability with a sensitivity of 67%, a specificity of 96%, and an accuracy of 78%. A patent infarct-related artery, as well as ventricular arrhythmias, were more commonly observed in patients with evidence of viable myocardium (P<0.05). Patients with arrhythmias had a higher QT dispersion than patients without (P<0.05).

Conclusion The combination of a resting QT dispersion ≤65 ms or an increase in QT dispersion >30% predicts the presence of viable myocardium and thus, may represent a simple index for the assessment of viability in everyday clinical practice. Myocardial viability is related to a patent coronary artery and to a high incidence of arrhythmias accompanied by a greater increase in QT dispersion at peak dobutamine infusion.

(Eur Heart J 2000; 21: 446–456) © 2000 The European Society of Cardiology

Key Words: Myocardial viability, QT dispersion, dobutamine stress echocardiography, arrhythmias.

See page 432 for the Editorial comment on this article

Introduction

Increased QT dispersion in recent years has been associated with an increased mortality in patients with coronary artery disease and especially during[1] or after an acute myocardial infarction[2,3]. This increase in mortality is considered to occur through an increased risk for malignant arrhythmias and sudden death[3]. Studies have shown that sudden death tends to occur during exercise in patients with coronary artery disease[4] and that its incidence is higher in patients with myocardial infarction[5]. Although increased QT dispersion has been observed after exercise treadmill testing in patients
with ischaemia and old myocardial infarction[6], the relationship of the above index to the presence of malignant arrhythmias during stress has not been adequately investigated.

Myocardial viability after myocardial infarction has been associated with an increased risk for arrhythmias during exercise treadmill testing[7], and with an increased incidence of recurrent ischaemic events[8]. Thus, an increased QT dispersion and the presence of myocardial viability have been associated with poor prognosis and the presence of arrhythmias during the late post infarction period. However, it remains unclear whether the presence of malignant arrhythmias during stress is related to changes of QT dispersion or to the presence of viable and therefore electrically irritable myocardium or to a combination of both factors.

Low dose dobutamine stress echocardiography, thalium re-injection[9,10] or 18 fluorodeoxyglucose PET[11] are well established diagnostic techniques for the detection of myocardial viability. However, these techniques are not readily available in all hospitals and require well trained operators and expensive technology. Stress electrocardiography[12] has been proposed as an alternative diagnostic tool, applicable on a large clinical scale, for diagnosis of myocardial viability. Low QT dispersion in a resting electrocardiogram has been associated with the presence of myocardial viability, detected by 18 fluorodeoxyglucose PET in 27 patients with previous myocardial infarction[13]. However, the relationship of changes in QT dispersion after dobutamine infusion to the presence of viable myocardium has not been investigated. Previous studies have shown that restoration of antegrade coronary flow may have beneficial effects on left ventricular function[14], electrophysiology[15-17] and survival[18]. However, it is not clear whether patency or persistent occlusion of a coronary artery determines the occurrence of viable myocardium within the myocardial regions this artery perfuses. Moreover, it has not been elucidated whether patency of coronary arteries influences left ventricular repolarization.

The aim of the present study was to investigate whether changes in QT dispersion after dobutamine infusion are indicative of myocardial viability in patients with an old myocardial infarction. We also investigated whether myocardial viability, QT dispersion or both are associated with the presence of ventricular arrhythmias during low dose dobutamine stress echocardiography in this group of patients. Finally, we evaluated the impact of coronary artery patency on myocardial repolarization and viability.

### Patients and methods

#### Patients

We prospectively studied 85 patients with a previous old myocardial infarction (>6 months) and angiographically documented coronary artery disease who were referred for assessment of myocardial viability. Patients with recent myocardial infarction were excluded to ensure that dynamic changes of QT dispersion occurring in the early post infarction period would not interfere with the measurements. Patients with atrial fibrillation or left bundle branch block were also excluded. All patients had their antianginal medication (beta-blockers or calcium channel blockers) withdrawn at least 48 h before dobutamine stress echocardiography. Their clinical characteristics are shown in Table 1. As described later, 10 patients were excluded from the study, thus 75 patients are studied further.

#### Cardiac catheterization

The presence of coronary artery disease was established during routine coronary angiography using the Judkins technique. Coronary stenoses were expressed as percent diameter reduction. A >70% reduction was considered to be significant. The presence of collaterals were visually assessed.

#### Dobutamine stress echocardiography

Each patient underwent two-dimensional echocardiography using the Hewlett-Packard Sonos 2500 system, in the left recumbent position. Standard tomographic views of the left ventricle (parasternal long and short axis, apical four-and two-chamber views), were obtained at rest and continuously during the dobutamine infusion. Imaging was continuously recorded in VHS

<table>
<thead>
<tr>
<th>Table 1 Clinical characteristics of the study population</th>
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<tbody>
<tr>
<td>Age (years)</td>
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<tr>
<td>Sex (male/female)</td>
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<tr>
<td>Number of diseased coronary vessels</td>
</tr>
<tr>
<td>I-VD</td>
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<tr>
<td>II-VD</td>
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<tr>
<td>III-VD</td>
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<tr>
<td>Collateral circulation</td>
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<tr>
<td>Yes/no</td>
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<tr>
<td>EF (%)</td>
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<tr>
<td>Location of MI</td>
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<tr>
<td>Anterior</td>
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<tr>
<td>Inferior</td>
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<tr>
<td>Lateral</td>
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<tr>
<td>Multiple MI</td>
</tr>
<tr>
<td>Yes/no</td>
</tr>
<tr>
<td>LV aneurysm</td>
</tr>
<tr>
<td>CABG</td>
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<td>PTCA</td>
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<td>Diabetes mellitus</td>
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<td>Hypertension</td>
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<td>Hyperlipidaemia</td>
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</tbody>
</table>

1, II, III-VD=one, two, three vessel disease, respectively; EF=ejection fraction at coronary angiography; LV=left ventricle; CABG=previous coronary artery bypass grafting; PTCA=previous coronary angioplasty.
videotapes and was digitized online using a separate black-and-white output in a quad screen format (Tomtec system) every 5 min, at the end of each dose of dobutamine. The 12-lead electrocardiogram (Siemens Sicard) was monitored throughout the test and blood pressure (cuff sphygmomanometer) was recorded at rest and at 1-min intervals during the dobutamine infusion. Some chest electrodes were slightly displaced to optimize echocardiographic windows.

An infusion line was placed in the right or left antecubital vein. Low dose dobutamine stress echocardiography was performed to assess myocardial viability. Dobutamine was administered by incremental infusion doses of 5, 10, 15 and 20 $\mu$g $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$ (low dose protocol), using an infusion pump, at 5-min intervals to investigate the presence of myocardial viability. In 13 of the 75 patients, further incremental dobutamine doses of 30 and 40 $\mu$g $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$ were infused including 1 mg atropine at the end of dobutamine infusion to rule out the presence of ischaemia.

Dobutamine stress echocardiography was considered positive for myocardial viability when improvement in wall motion abnormalities was detected in at least two contiguous segments. The dobutamine stress test was continued regardless of the improvement in wall motion abnormalities until maximal dobutamine dose $(20 \mu g \cdot kg^{-1} \cdot min^{-1}$ in 62 and 30–40 $\mu$g $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$ in 13 patients) was infused and the protocol was completed. For the analysis of regional wall motion abnormalities an 11-segment protocol of the four tomographic views of the left ventricle was used$^{[19]}$.

Each segment was scored as follows: normal=1, hypokinesis=2, akinesis=3 and dyskinesis=4. For the semiquantitative analysis of regional wall motion abnormalities, a total wall motion score was used, which was the sum of scores for all 11 segments. The reduction in wall motion score between rest and 20 $\mu$g $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$ dobutamine dose (wall motion score at rest – wall motion score at low dobutamine dose) and the percent reduction of wall motion score % (wall motion score at rest – wall motion score at low dobutamine dose/wall motion score at rest) were used, as a measure of the extent of viable myocardium during the test. The 11 myocardial segments were related to each of the three coronary arteries as follows: segments 1, 2, 8–11 to left anterior descending, 3, 4 to circumflex, 5, 6, 7 to right coronary artery.

Echocardiograms were analysed by two observers (G.A., G.K.) blinded to the results of electrocardiograms, on two separate days with a time interval of 1 month. Intra-observer variability was defined as the mean difference $\pm$ SD (95% confidence intervals–CI) between the two sets of measurements and was 8 $\pm$ 0.5 ms (95% CI ranging from 7.1 to 8.7 ms). QT dispersion (QTd) was defined as the difference between maximum (QT$\text{max}$) and minimum (QT$\text{min}$) QT intervals ($\text{QT}_\text{d}=\text{QT}$max$\text{QT}$min). The difference in QTd (diffQTd=QTd at 20 $\mu$g $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$ dobutamine dose – QT at rest) and the percent difference in QTd (%diffQTd=QTd at 20 $\mu$g $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$ dobutamine dose – QTd at rest)/QTd at 20 $\mu$g $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$ dobutamine dose $\times$ 100) between rest and ‘low’ dobutamine dose were also calculated. Measurements of QT intervals were performed at 20 $\mu$g $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$ and not at higher dobutamine doses to exclude any influence of ensuing ischaemia on QT interval behaviour$^{[20]}$. All values were rate corrected using Bazett's formula ($\text{QTc}=\text{QT}/\text{SQRR}$).

(b) The presence of new ST segment elevation or an increase in baseline ST segment elevation at 20 $\mu$g $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$ was also noted. ST segment elevation was defined as new or additional ST elevation greater than 0.1 mV, 80 ms after J point in $\geq$ 2 Q wave leads. The PQ segment was considered the isoelectric line.

(c) The presence of ventricular arrhythmias (Lown grade >1b) during the test was noted$^{[21]}$.

Finally, the ejection fraction was calculated utilizing the machine’s software using Simpson’s rule.

**ECG analysis**

(a) High quality 12-lead ECGs (50 mm $\cdot$ s$^{-1}$ paper speed) were obtained at rest and at the end of each dobutamine dose for manual measurement of QT interval (ms). QT intervals were measured from the first deflection of the QRS complex to the point of T-wave offset, defined as the return of the T wave to baseline. If a U wave was present, the T wave offset was defined as the nadir of the curve between the T and U waves. Ten patients were excluded from the study, since data from $\geq$10 of the 12 leads were not available. Thus, 75 patients were finally included in this study. ECGs were analysed by a single observer (I.I.) blinded to the results of dobutamine stress echocardiography, on two separate days and with a time interval of 1 month. Intra-observer variability was defined as the mean difference $\pm$ SD (95% confidence intervals–CI) between the two sets of measurements and was 8 $\pm$ 0.5 ms (95% CI ranging from 7.1 to 8.7 ms). QT dispersion (QTd) was defined as the difference between maximum (QT$\text{max}$) and minimum (QT$\text{min}$) QT intervals ($\text{QT}_\text{d}=\text{QT}$max$\text{QT}$min). The difference in QTd (diffQTd=QTd at 20 $\mu$g $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$ dobutamine dose – QT at rest) and the percent difference in QTd (%diffQTd=QTd at 20 $\mu$g $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$ dobutamine dose – QTd at rest)/QTd at 20 $\mu$g $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$ dobutamine dose $\times$ 100) between rest and ‘low’ dobutamine dose were also calculated. Measurements of QT intervals were performed at 20 $\mu$g $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$ and not at higher dobutamine doses to exclude any influence of ensuing ischaemia on QT interval behaviour$^{[20]}$. All values were rate corrected using Bazett’s formula ($\text{QTc}=\text{QT}/\text{SQRR}$).

**Statistical analysis**

Results are expressed as mean value $\pm$ SD. Student two tail t-test and chi-square analysis were used and significance was derived by statistical tables. Comparisons between groups were performed using analysis of variance (ANOVA) with Scheffe correction and the Kruskal–Wallis test for non-parametric values. Linear regression analysis was performed to correlate changes of QT dispersion between rest and peak dobutamine dose with wall motion score. A probability value of less than 0.05 was considered significant.
improvement in wall thickening seen at ‘low dose’ until the end of the maximal dobutamine dose whereas four of the 13 showed no change in wall motion in comparison to baseline and thus, were considered negative for both ischaemia and viability. There was no difference in age, sex distribution, number of diseased vessels, presence of collaterals, ejection fraction and resting wall motion score between patients with and without evidence of myocardial viability. However, the coronary artery perfusing viable myocardial segments were patent in 27 (56%) of the 48 patients with myocardial viability. Conversely, there was a high incidence of occlusion (19/27, 71%) in the infarct-related coronary artery of patients with no evidence of viability at dobutamine stress echocardiography (chi-square=5.324, P<0.05).

Results

Patient cohort

Seventy-five out of 85 consecutive patients who underwent low dose dobutamine stress echocardiography had adequate ECGs for QT interval analysis and were included in the study. No patient was excluded due to inadequate imaging. Patient characteristics are shown in Table 1. Fourteen patients had single (I) vessel disease (19%), 27 patients had two (II) vessel (36%) and 34 patients had three (III) vessel disease (45%). The presence of collateral circulation was observed in 29 patients (39%) while 46 patients did not have any (61%) (Table 1). The coronary artery related to viable myocardial segments at dobutamine stress echocardiography, was patent (TIMI grade >1) in 35 patients (46%). The mean ejection fraction, calculated by echocardiography was 34±11%. The heart rate increased from 56±10 to 59±14 beats. min⁻¹ and systolic and diastolic blood pressure from 110±10 to 120±9 and 65±5 to 70±5 mmHg at the 20 μg . kg⁻¹ . min⁻¹ dobutamine dose.

Viability assessment

Forty-eight out of 75 patients (64%) demonstrated signs of myocardial viability as indicated by improvements in wall motion abnormalities at 20 μg . kg⁻¹ . min⁻¹. Their wall motion score improved from 21±5 to 18±5 (P<0.05). Twenty-two (46%) of the 48 patients with evidence of myocardial viability presented more than two contiguous viable segments (>2) at dobutamine stress echocardiography (15 had three segments, six, four segments and one, five segments). In the 27 patients without viability the wall motion score did not change (21±5 before and after dobutamine). Basally hypokinetic or akinetic segments, after improving their thickening at low doses, showed significant deterioration of their wall motion at peak dose (biphasic response) in only four of the 13 patients who were administered maximal dobutamine doses. Five of the 13 preserved the
Wall motion abnormalities and QT dispersion

The reduction and percent reduction (%) of wall motion score at the 20 μg·kg⁻¹·min⁻¹ dobutamine dose were not related to the difference and percent difference of QT dispersion (%diffQTd) between rest and peak stress (P=ns) even when presence or absence of myocardial viability was taken into account. However, a high resting wall motion score was associated with a greater increase (r=0.42, P<0.01) and a percent increase of QT dispersion values (r=0.33, P<0.05) at stress only in patients with myocardial viability (Fig. 2(a) and (b)).

Patients with evidence of myocardial viability in more than two (>2) contiguous myocardial segments had a higher increase (30±18 vs 18±15 ms, P<0.01) and percent increase of QT dispersion (P<0.05) at the 20 μg·kg⁻¹·min⁻¹ dobutamine infusion than patients with two or less (<2) viable segments (Fig. 3).

QT dispersion and arrhythmias during dobutamine stress echocardiography

During dobutamine infusion only two patients presented with bigeminy and two with salvos, while 22 out of 75

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**Figure 1** Patients with myocardial viability (n=48) at dobutamine stress echocardiography (DSE) present a greater percent increase in QT dispersion (%diffQTd) compared to patients without (n=27) (P<0.01). —— =mean values for each group.

**Table 3** Diagnostic performance of QT dispersion and its increase by dobutamine to predict myocardial viability

<table>
<thead>
<tr>
<th></th>
<th>Resting QTd &lt;65 ms</th>
<th>Increase of QTd &gt;30%</th>
<th>Combination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensitivity</td>
<td>25/48 (53%)</td>
<td>24/48 (51%)</td>
<td>32/48 (67%)</td>
</tr>
<tr>
<td>Specificity</td>
<td>18/27 (67%)</td>
<td>23/27 (85%)</td>
<td>26/27 (96%)</td>
</tr>
<tr>
<td>(+) predictive value</td>
<td>25/34 (74%)</td>
<td>24/28 (86%)</td>
<td>32/41 (78%)</td>
</tr>
<tr>
<td>(−) predictive value</td>
<td>18/41 (44%)</td>
<td>23/46 (50%)</td>
<td>26/49 (53%)</td>
</tr>
</tbody>
</table>

QTd=dispersion; combination=rest QTd <65 ms and/or increase of QTd >30%; (+)=positive, (−)=negative.
Figure 2  High resting wall motion score (WMS) is associated with a greater increase (a) and a percent (b) increase in QT dispersion (QTd) at peak stress in patients with myocardial viability.
patients presented with multiple ventricular ectopics (Lown grade >1b). QT dispersion was increased by 47 ± 44% in patients who developed arrhythmias and by only 27 ± 22% in patients who did not develop any significant arrhythmias (P<0·01, Fig. 4). Twenty-one (43%) of the 48 patients with myocardial viability and only five (15%) of 27 patients without developed ventricular arrhythmias during dobutamine stress echocardiography (P<0·05). Wall motion score decreased by 12 ± 9% in patients who developed arrhythmias and by only 8 ± 7% in patients who did not (P<0·05) at stress. Additionally, 15 (71%) of the 21 patients with viability and arrhythmias had more than two viable myocardial segments, compared to only seven (26%) of the 27 patients without developed ventricular arrhythmias during dobutamine stress echocardiography (P<0·05). There was no relation between QT intervals and ejection fraction either at rest or at 20 μ · kg⁻¹ · min⁻¹ of dobutamine infusion.

**Figure 3** Patients (n=22) with more than two (>2) contiguous viable myocardial segments during dobutamine stress echocardiography present a greater percent increase in QT dispersion (%diffQTd) compared to patients (n=26) with two or fewer (<2) viable segments (P<0·05). ———=mean values for each group.

Discussion

To our knowledge, this is the first prospective study to demonstrate that the association of myocardial viability and incidence of ventricular arrhythmias during low dose dobutamine stress echocardiography in patients with an old myocardial infarction is correlated with a significant increase in QT dispersion. Furthermore, myocardial viability was associated with low resting QT dispersion values and a greater increase in QT dispersion during dobutamine infusion. Thus, the presence of myocardial viability, increased dispersion of ventricular repolarization and ventricular arrhythmias at stress may be the result of a similar pathological substrate.

Dobutamine and ST elevation

No new ST segment elevation was observed during the test. Only 10 (13%) out of the 75 patients had a baseline ST segment elevation greater than 0·1 mV during dobutamine infusion. All had significant (<0·1 mV) resting ST segment elevation in leads with Q waves. Seven (70%) out of 10 showed evidence of myocardial viability and only four (40%) presented with arrhythmias during dobutamine infusion. Six of the seven patients with an increase in ST elevation and evidence of viability at dobutamine infusion also had an increase in QT dispersion >30%.

Myocardial viability, QT dispersion and arrhythmias

Ventricular arrhythmias during exercise stress testing are associated with increased long-term mortality[23], particularly in patients with previous myocardial infarction[23]. Increased QT dispersion has been associated with an increased risk of fatal arrhythmias and sudden death[1–3]. Although increased QT dispersion has been
observed after treadmill testing in patients with ischaemia and old myocardial infarction\cite{6}, its relation to the presence of malignant arrhythmias during stress has not been investigated. Conversely, myocardial viability after myocardial infarction has been associated with increased risk for arrhythmias during exercise treadmill testing\cite{7}.

In the present study, we also found that patients with myocardial viability developed more arrhythmias during dobutamine stress than those without. At the same time, they increased their QT dispersion while patients with no evidence of viability did not. Furthermore, the majority of the patients (76%) with three or more viable segments presented significant ventricular arrhythmias, whereas only 26% of patients with two or fewer viable segments presented such arrhythmias. Thus, our data suggest that, not only the presence but also the extent of myocardial viability is related to a high incidence of arrhythmias during pharmacological stress due to a significant increase in QT dispersion by dobutamine. Fenoterol, another beta receptor antagonist has been shown to increase QT dispersion in patients with severe asthma and normal cardiac function\cite{24}.

The significant increase of QT dispersion only in patients with viable myocardium at dobutamine stress echocardiography should be attributed to the inhomogeneity in both mechanical and electrical activation produced by dobutamine. Viable myocardium has the ability to increase its contractility and modify its electrical activation–repolarization in response to dobutamine, since it possesses a sufficient number of beta receptors. Necrotic myocardium would not be expected to change its behaviour and respond to dobutamine infusion since scar tissue does not possess beta receptors; thus no significant differences from resting QT dispersion values would be expected at dobutamine infusion. In our study, patients with viable myocardium increased QT dispersion by 42% in response to dobutamine stimulation, whereas in patients with necrotic myocardium the increase was only 21%, despite the presence of a similar ejection fraction. Additionally, patients whose systolic wall thickness improved in three or more (30%) of the 11 myocardial segments increased their rest QT dispersion values to a greater extent in response to dobutamine infusion compared to patients with a smaller amount of viable tissue. Thus, presence of viable tissue corresponding to 30% or more of the left ventricular mass was related to a 55% increase in resting QT dispersion values by dobutamine. O’Sullivan et al.\cite{29} found that QTc interval and dispersion lengthen in patients with coronary artery disease after dobutamine infusion, while they shorten in healthy controls. They ascribed this finding to ischaemia. We expressly wanted to avoid the

\begin{figure}
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\includegraphics[width=\textwidth]{figure4.png}
\caption{Patients with ventricular arrhythmias (n=26) during dobutamine stress echocardiography present a greater percent increase in QT dispersion (%diffQTd) compared to patients without (n=49) (P<0.01). ——=mean values for each group.}
\end{figure}
occlusion of ischaemia in our patients. This is the reason why we limited our observation to the 'low' 20 μg · kg⁻¹ · min⁻¹ dose, at which wall motion score was improving.

A high resting wall motion score was associated with a greater increase in QT dispersion at peak dobutamine dose only in patients with viable and thus electrically active myocardium.

The lower resting QT dispersion in patients with myocardial viability may reflect a more uniform ventricular recovery due to the presence of less fibrotic and thus less electrically inactive myocardium. On the other hand, the presence of a large amount of fibrous tissue observed in patients with large infarcts and no evidence of viable myocardium may influence the duration and homogeneity of ventricular repolarization and contribute to prolonged QT dispersion values. Furthermore, infarcts with large amounts of fibrous tissue may result in enhanced mechanical stretch to myocardium or ventricular dilatation, which potentially increase both repolarization inhomogeneity and risk for arrhythmia and sudden death.

No relation between QT dispersion and ejection fraction was found, confirming findings of other investigators. The lack of correlation may be explained by the observation that within patients with similar ejection fraction those with viable myocardium present a shorter QT dispersion than those with necrotic myocardium.

**QT dispersion and prediction of myocardial viability**

Low dose dobutamine stress echocardiography, thallium or 18 fluorodeoxyglucose positron emission tomography are used for detection of myocardial viability. However, these techniques are not readily available in all hospitals and require expensive technology. Stress electrocardiography has been proposed as an alternative diagnostic tool, applicable on a large clinical scale for diagnosis of myocardial viability. Margonato et al. have suggested that elevation of the ST segment is suggestive of viability in patients with recent myocardial infarction; 94% of patients with transient ST elevation during treadmill exercise had reversible TI-201 defects. Results with dobutamine stress in patients with recent myocardial infarction have been more equivocal, since significant ST segment elevation was present only at maximal dobutamine doses and was related more to homozonal ischaemia than to the presence of myocardial viability, as described by Ricci et al. In our study we examined patients with old myocardial infarction (>6 months). Only 10 (13%) patients presented a significant increase in baseline ST segment elevation in leads with Q waves during stress and no new ST segment elevation was observed. Thus, our data suggest that new or additional ST segment elevation is an uncommon finding in patients with old myocardial infarction during low dose dobutamine stress echocardiography and therefore, this index possesses low diagnostic value for detecting myocardial viability. Additionally, the great majority of our patients with additional ST segment elevation showed an increase of QT dispersion greater than 30%. Ricci et al. found an insignificant ST elevation at low dobutamine doses, which is in accordance with our results.

Schneider et al. have shown that a QT dispersion ≤ 70 ms on a resting electrocardiogram has a sensitivity of 85% and a specificity of 82% to predict myocardial viability, detected by 18-fluorodeoxyglucose positron emission tomography in 44 patients with old myocardial infarction. We also found that a resting QT dispersion < 65 ms has a sensitivity of 53% and a specificity of 67% to predict myocardial viability detected by dobutamine stress echocardiography in 75 patients with old myocardial infarction. The differences in sensitivity and specificity of resting QT dispersion between the two studies could be attributed to the differences in the number and characteristics of the patients examined. In our study, the mean ejection fraction in patients with and without viability was 34% and the majority of patients presented akinetic segments within the infarct related area. Conversely, in the study by Schneider et al. the mean ejection fraction was 50%, wall motion abnormalities were assessed by left ventriculography and thus a higher number of hypokinetic segments with increased F-18 fluorodeoxyglucose uptake may have been included in the infarct related area.

At the 20 μg · kg⁻¹ · min⁻¹ dobutamine dose, an increase of QT dispersion values >30% was observed in 24 out of the 48 (51%) patients with viability but only in four out of the 27 without (P<0.01). This finding had a similar sensitivity, but a higher specificity and a positive predictive value for viability compared to resting QT dispersion values. When the presence of a resting QT dispersion < 65 ms was combined to a QT dispersion increase > 30% after dobutamine, a sensitivity of 67%, a specificity 96% and a positive predictive value 78% for predicting viability was found. Thallium-201 scintigraphy yields sensitivity values of 74% and specificity values of 75% in predicting viability. Thus, the ability of resting QT dispersion and its increase after dobutamine infusion to detect viable myocardium in patients with old myocardial infarction is comparable to that of other expensive and not readily available diagnostic techniques. Consequently, the calculation of QT dispersion both at rest and after dobutamine infusion is a reasonable initial approach to estimate the presence of a substantial amount of viable myocardium in patients with old myocardial infarction considering the simplicity, availability and low cost of a stress electrocardiogram.

**Patency of coronary vessels, myocardial viability and ventricular repolarization**

Previous studies have shown that restoration of antegrade coronary flow may have beneficial effects on
left ventricular function\textsuperscript{14}, electrophysiology\textsuperscript{15-17} and survival\textsuperscript{18}. Successful thrombolysis after myocardial infarction has been shown to decrease repolarization inhomogeneity, as reflected by lower QT dispersion values\textsuperscript{13,19} and improved electrical stability\textsuperscript{13,20}. Furthermore, patients with an occluded infarct related artery have a higher prevalence of ischaemia during pharmacological and exercise stress testing, regardless of presence of collateral flow\textsuperscript{21}. In the present study, patency of coronary vessels was associated with higher occurrence of viable myocardium within the myocardial region this artery perfused. However, patency of coronary arteries was not associated with QT dispersion values, suggesting a lack of influence by the left ventricle on repolarization, although other factors may confound this relationship.

In conclusion, dobutamine infusion increases QT dispersion in patients with previous myocardial infarction, but has a greater effect on those with viable myocardium. This may reflect greater changes in ventricular recovery in this group of patients. The higher incidence of ventricular arrhythmias in patients with myocardial viability during dobutamine stress echocardiography and the greater increase in QT dispersion at peak stress may be associated by a similar pathological process. This finding may augment the ability of the surface electrocardiogram to predict myocardial viability.

**Study limitations**

We have used dobutamine stress echocardiography to assess myocardial viability in patients with myocardial infarction. The technique is extensively used in daily clinical practice to identify viable myocardium as it yields a sensitivity and specificity ranging between 74% and 92% and 77% and 95%, respectively, compared to other techniques\textsuperscript{10,31}. There is no gold standard for identifying myocardial viability other than by examining recovery of ventricular function after revascularization. Several investigators have shown that dobutamine stress echocardiography predicts recovery of ventricular function after revascularization with greater accuracy in patients with myocardial infarction\textsuperscript{13-15}. This method is as useful as positron emission tomography in predicting ventricular recovery in this group of patients\textsuperscript{18}.

**References**


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Eur Heart J, Vol. 21, issue 6, March 2000