Role of Collateral Flow in a Pharmacological Stress Test (a Combination of Low-Dose Dobutamine and a Vasodilator) as a Predictor of Wall Motion Reversibility

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The role of collateral flow was evaluated in a pharmacological stress test [a combination of low-dose dobutamine (DOB) and a vasodilator] as a predictor of wall motion reversibility at rest after percutaneous transluminal coronary angioplasty (PTCA) using ultrafast computed tomography (UFCT). Segments with wall motion abnormalities before PTCA were divided into two groups; ie, either with or without collateral flow. Patients were scanned at rest for baseline and again after 5 min of intravenous administration of 4 µg/kg per min of DOB after nitroglycerin (0.3 mg sublingually) or isosorbide dinitrate (2.5 mg bolus intravenous injection). Three months after PTCA, patients were scanned again and wall motion was compared with the previous findings. In collateral-dependent segments, the sensitivity of the pharmacological stress test as a predictor of wall motion reversibility was 87.5% and the specificity was 83.3%. In collateral-independent segments, the sensitivity was only 41.7%, while the specificity was 95.2%.

Our findings demonstrate that the pharmacological stress test we used satisfactorily predicted wall motion reversibility in collateral-dependent segments, but tended to underestimate wall motion reversibility in collateral-independent segments. Therefore, collateral flow may be an important factor in accurately predicting wall motion reversibility by this pharmacological stress test.

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For the past two decades, cardiac nitroglycerin1–6 and epinephrine stress tests7,8 have been used to evaluate cardiac reserve function and the reversibility of wall motion in left ventriculography. Recently, a clinical stress test which uses a new catecholamine, dobutamine (DOB), has been developed to evaluate hibernating myocardium using echocardiography9. However, the details of the administration (including, for example, dose and duration) and the mechanism of the effect of DOB on hibernating myocardium have not yet been clarified, and several questions remain to be answered. Dilsizian and Bonow have suggested that the administration of a positive inotropic agent (even at low doses) may merely increase myocardial demand under conditions of an exhausted coronary flow reserve, thereby producing myocardial ischemia and persistent regional dysfunction.10 In addition, Rahimtoola11 has observed that because the contraction force of normal myocardium increases with inotropic stimulation, its tethering effect on akinetic segments may appear to improve wall motion in these segments.

Vanoverschelde et al12 recently reported that there are cases in which myocardial blood flow does not decrease with collateral
flow, but instead shows segmental dysfunction and reverses at rest after revascularization. Therefore, chronic reversible myocardium can originate not only from contraction-perfusion matching for hypoperfusion\(^1\) but also from some disturbance in the contraction mechanism, even though blood flow may have been almost normal at rest due to collateral flow.

Therefore, the primary purpose of this study was to evaluate a pharmacological stress test [a combination of low-dose DOB and a vasodilator (nitroglycerin or isosorbide dinitrate)] as a predictor of wall motion reversibility, and to examine the role of collateral flow in this test. Organic nitrates\(^14\)\(^-\)\(^16\) which dilate not only epicardial coronary artery, but also collateral artery, increase blood flow and oxygen supply downstream from a coronary stenosis to improve the contraction force of the co-administered catecholamine. Accordingly, this combination was expected to improve the sensitivity of the pharmacological stress test beyond that of a test which uses only low-dose DOB (Fig. 1). The secondary purpose of this study was to attempt to perform a quantitative analysis using ultrafast computed tomography (UFCT), which has been reported to possess excellent resolution and reproducibility and enables wall thickening to be measured accurately\(^17\)\(^-\)\(^20\) Thus, we hoped to exclude subjective observer variability and to avoid possible misconceptions, which are often detracting factors in echocardiography or radionuclide ventriculography.

**METHODS**

**Controls**

To evaluate observer variability and the accuracy of UFCT, 20 control subjects (12 men, 8 women) were studied. Eighteen of them, who were suspected to have ischemic heart disease due to atypical chest pain, underwent hemodynamic catheterization, coronary angiography and left ventriculography, which disclosed no abnormalities. The other two controls were healthy volunteers. The controls ranged in age from 18 to 78 years with a mean of 44.3 years. The study was performed after informed consent was obtained.

**Patients**

Patients with coronary target lesions in the proximal coronary artery which produced luminal narrowing of more than 75% of the coronary diameter were selected to accurately evaluate the prediction of reversibility of

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wall motion abnormality at the equatorial level and to minimize quantitative errors. From the initial group of 58 de novo patients with 68 target lesions in the present protocol, 8 subjects did not consent to the pharmacological stress test and were eliminated, while one was excluded for arrhythmia which occurred during the pharmacological stress test.

Percutaneous transluminal coronary angioplasty (PTCA) was judged successful when the residual coronary stenosis of the target lesion was less than 50% in diameter, and was judged unsuccessful in 5 target lesions. If restenosis was observed at the follow-up, PTCA was repeated. Six subjects withdrew in the middle of the study. Consequently, 41 patients with 47 successful target lesions (single-vessel: 36, two-vessel: 4, three-vessel: 1, left anterior descending artery: 31, right coronary artery: 9, circumflex artery: 7) were evaluated. All of the patients were afflicted with myocardial infarction. The average duration from the first infarction attack to PTCA in patients with myocardial infarction was 304.4 days. The patients (30 men, 11 women) ranged in age from 35 to 77 years, with a mean of 59.4 years. Thirty five patients were treated with nitrate and calcium antagonist, 3 with nitrate, calcium antagonist and \( \beta \)-blocker, 2 with nitrate and one with calcium antagonist. The \( \beta \)-blocker was generally discontinued 5 days before the day of examination and other oral medication was withdrawn on the morning of the examination. The same medication was administered throughout the entire study.

**Collateral Flow Analysis**

Collateral flow in coronary angiography before PTCA was judged by three experienced cardiologists using Rentrop's classification to investigate the effect of collateral flow on the prediction of wall motion reversibility in this pharmacological stress test. Two groups were evaluated: ie, those which showed collateral-dependent segments with wall motion abnormalities not less than grade 1 Rentrop, and those which showed collateral-independent segments with wall motion abnormalities.

**UFCT Scanning Protocol**

Subjects were scanned with an Imatron C-100 scanner (Imatron, Inc, South San Francisco, California). Patients were placed in the supine position on the scanner couch, tilted 15° axially with their feet down and slewed 25° horizontally to the right to produce an approximate short-axis view of the left ventricle. An intravenous catheter (20 gauge) was inserted into the right antecubital vein for contrast medium and another intravenous catheter (20 gauge) was inserted into the left cephalic or right saphenous vein for administration of DOB and vasodilator injection. The contrast medium was mixed with heparin at a concentration of 10 units /ml. The patient's hands were held above the head during the course of the study. Scans were obtained 28 to 35 sec after the start of a bolus injection of contrast medium (Iohexol 350), administered at 2 ml/sec (total 40–60 ml) followed by a continuous injection at 1 ml/sec (total 10 ml). A multicycle mode protocol, in which one target was scanned repeatedly at a rate of 50 msec/scan at an interval of 8 msec/scan, was employed. The scanning of 10 continuous images in one target was triggered by the R wave of an electrocardiogram, (approximating lead V₃). A preliminary experiment confirmed that 10 images (50×10+8×9=572 msec) were sufficient to cover the systolic phase, since M mode echocardiography revealed that the interval from the R wave to the end-systolic phase ranged from 320 to 410 msec in 6 patients with heart rates of less than 50 beats/min at baseline. Using 4 targets and 2 detectors in sequence, 8 levels were scanned. One scan could cover 76 mm because each level was 8-mm thick and there was a slice gap of 4 mm between each target. Blood pressure was monitored throughout the test.

**Pharmacological Stress Test**

The first scan was performed at rest to establish a baseline in both control subjects and patients. The second scan was performed only in patients after DOB was administered intravenously at a rate of 4 \( \mu \)g/kg per min for 5 min with either nitroglycerin (22 patients; 0.3 mg sublingually) or isosorbide dinitrate (19 patients; 2.5 mg bolus intravenous injection). The protocol was to be discontinued if a patient complained of discomfort, if systolic blood pressure increased to over 200 mmHg or decreased 30
Thickening

End-diastole

Maximum-systole

Coronary perfusion area

Fig. 2. Wall motion analysis.

Thickening = (Maximum-systolic thickness /end-diastolic thickness − 1) × 100.

Maximum-systolic thickness = Maximum-systolic distance from the endocardial boundary to the epicardial boundary

End-diastolic thickness = End-diastolic distance from the endocardial boundary to the epicardial boundary

A reference point (0°) was selected at the 12:00 o'clock position. Coronary perfusion areas were calculated from 310°−40° for the area of the left anterior descending artery (LAD), from 130°−220° for the area of the right coronary artery (RCA) and from 220°−310° for the area of the circumflex artery (CX).

Fig. 3. Observer variability in wall thickening in control subjects

Tracing error = 1.15 ± 8.32%, coefficient of variation = 12.6% SEE = standard error of estimate.

during systole when the wall thickness was maximum (maximum-systolic phase images) were traced manually with a trackball cursor by an experienced observer without prior knowledge of the patients' data. The maximum-systolic phase was selected because it has been reported that early systolic thickening and late systolic thinning occur following DOB infusion. Generally, level and window settings were selected from ranges of 20−90 and 250−500, respectively, to identify the clearest boundary between the blood pool and the left ventricular wall. The endocardial boundary was traced excluding papillary muscles and trabeculae. Particular care was taken to ensure that the endocardial line was smooth by tracing the maximum outline of the blood pool while watching a cine mode on a monitor. Thickening of the left ventricular wall was calculated only at the equatorial level of the left ventricle to minimize quantitative errors. A reference point (0°) was selected at the 12:00 o'clock position (Fig. 2). In the measurement of wall thickening, the end-diastolic endocardial center of gravity was used in the end-diastolic phase and the maximum-systolic endocardial center of gravity was used in the maximum-systolic phase to measure the wall thickness perpendicular to the endocardial boundary as accurately as possible. The lengths of 36 radii, each 10° apart, from the center of gravity to the endocardial and

Wall Motion Analysis

The field of view of the reconstructed image was 26 cm, and the size of the matrix was 360 × 360. Therefore, each pixel was 0.72 mm. Endocardial and epicardial boundaries in end-diastolic images and in those
TABLE 1 HEMODYNAMIC CHANGES AND CARDIAC FUNCTION AT THE PHARMACOLOGICAL STRESS TEST

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Pharmacological stress test before PTCA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Rate (beats/min)</td>
<td>64.3±1.8</td>
<td>73.8±2.2**</td>
</tr>
<tr>
<td>Systolic Blood Pressure (mmHg)</td>
<td>137.8±3.1</td>
<td>147.4±3.7**</td>
</tr>
<tr>
<td>Diastolic Blood Pressure (mmHg)</td>
<td>78.1±2.0</td>
<td>76.9±1.8</td>
</tr>
<tr>
<td>End-diastolic Volume (ml)</td>
<td>153.2±5.3</td>
<td>152.0±5.5</td>
</tr>
<tr>
<td>Stroke Volume (ml)</td>
<td>62.4±1.9</td>
<td>73.3±2.1**</td>
</tr>
<tr>
<td>End-systolic Volume (ml)</td>
<td>80.5±6.1</td>
<td>63.2±5.3**</td>
</tr>
<tr>
<td>Ejection Fraction (%)</td>
<td>42.0±1.5</td>
<td>51.5±1.5**</td>
</tr>
<tr>
<td>Cardiac Output (l/min)</td>
<td>3.97±0.12</td>
<td>5.38±0.17**</td>
</tr>
</tbody>
</table>

Mean ± SE

**: p<0.01 vs baseline

For baseline, at the pharmacological stress test and after PTCA. In control subjects, to evaluate both observer variability and the accuracy of UFCT, two measurements, at an interval of approximately one month, were taken by the same observer.

In wall motion analysis, since the mean ± standard deviation (SD) of the wall thickening of the first tracing in control subjects at rest was 69.4±13.7%, wall thickening of less than 2 SD below the mean (mean – 2×SD = 69.4 – 2×13.7 = 42.0%) of control subjects was considered wall motion abnormality. Observer variability was shown to have a SD of 8.3% based on the difference between the first and second tracings in control subjects. The coefficient of variation in tracing wall thickening was 12.6% (Fig. 3). In addition, an increase in the thickness of the ventricular wall of more than 2 SD of the observer variability (2×8.3 = 16.6%) beyond the thickening at baseline was considered to indicate reversibility of wall motion at the pharmacological stress test or improvement in wall motion after PTCA. Wall motion deterioration was judged when the thickening of the ventricular wall decreased more than 2 SD (16.6%), and the deteriorated segment was defined as irreversible at the pharmacological stress test.

Statistical Analysis

Data regarding hemodynamics, cardiac function and wall thickening are presented as the mean±SE. Cardiac function was calculated by summing the left ventricular value in 8 levels. Statistical comparisons of hemodynamics and cardiac function were performed using Student’s paired t-test. Statistical comparisons of wall thickening were performed using the unpaired t-test. A p value of less than 0.05 was considered statistically significant.

RESULTS

Clinical Effects of the Pharmacological Stress Test and UFCT Scanning

Arrhythmia (premature ventricular contraction) occurred in one patient during DOB infusion. Side-effects due to the contrast medium related to renal insufficiency were not observed, although 2 patients demonstrated urticaria after scanning. In addi-
TABLE II THE ACCURACY OF THE PHARMACOLOGICAL STRESS TEST AS A PREDICTOR OF WALL MOTION REVERSIBILITY

<table>
<thead>
<tr>
<th></th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wall motion abnormalities</td>
<td>12/20 = 60.0%</td>
<td>25/27 = 92.6%</td>
<td>12/14 = 85.7%</td>
<td>25/33 = 75.8%</td>
</tr>
<tr>
<td>n = 47</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Collateral-dependent segments</td>
<td>7/8 = 87.5%</td>
<td>5/6 = 83.3%</td>
<td>7/8 = 87.5%</td>
<td>5/6 = 83.3%</td>
</tr>
<tr>
<td>with wall motion abnormalities</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n = 14</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Collateral-independent segments</td>
<td>5/12 = 41.7%</td>
<td>20/21 = 95.2%</td>
<td>5/6 = 83.3%</td>
<td>20/27 = 74.1%</td>
</tr>
<tr>
<td>with wall motion abnormalities</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n = 33</td>
<td></td>
<td></td>
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</tbody>
</table>

PPV = positive predictive value, NPV = negative predictive value.

...tion, hypotension was noted in one patient at scanning after PTCA, but blood pressure soon returned to normal.

Effects of the Pharmacological Stress Test on Hemodynamics and Cardiac Function

Heart rate and systolic blood pressure increased at the pharmacological stress test. However, diastolic blood pressure did not change significantly (Table I). End-diastolic volume also did not change significantly from the baseline value during the pharmacological stress test, while end-systolic volume decreased. Stroke volume and ejection fraction both significantly increased.

Effect of the Pharmacological Stress Test and PTCA on Wall Motion

In the 47 segments that showed wall motion abnormalities before PTCA, wall motion improved in 14 segments at the pharmacological stress test, which were therefore judged reversible, while the 33 segments which did not improve, including one deteriorated segment, were judged irreversible. Twenty segments were improved by PTCA and 27 segments remained unchanged after PTCA. No deteriorated segments were observed after PTCA. Wall thickening in the 20 segments that were improved after PTCA was 19.4 ± 3.9% at baseline, 45.7 ± 8.7% at the pharmacological stress test, and 49.5 ± 4.6% after PTCA. Wall thickening in the 27 segments that were unchanged after PTCA was 18.5 ± 3.6%, 24.0 ± 5.7%, and 15.9 ± 4.9%, respectively. There was no significant difference in wall thickening at baseline between improved and unchanged segments after PTCA.

Twelve of the 20 segments that were improved after PTCA were judged reversible at the pharmacological stress test to yield a sensitivity of 12/20 = 60.0% and a positive predictive value of 12/14 = 85.7%. Twenty-five of the 27 segments that were unchanged after PTCA were judged irreversible. Thus, the specificity was 25/27 = 92.6%, the negative predictive value was 25/33 = 75.8%, and the predictive accuracy was 37/47 = 78.7% (Table II).

Effect of Collateral Flow on the Predictive Value of the Pharmacological Stress Test

There were 14 collateral-dependent segments with wall motion abnormalities before PTCA (Rentrop's classification grade 1: 5, grade 2: 7, grade 3: 2) and 33 collateral-independent segments. The grades of proximal stenosis in collateral-dependent segments and collateral-independent segments were 98.4 ± 1.0% (100%; 10, 99%; 2, 90%; 2) and 89.9 ± 1.4% (100%; 4, 99%; 5, 90%; 18, 75%; 6), respectively. The grades of proximal stenosis in collateral-dependent segments were significantly higher than those in collateral-independent segments (p < 0.05).

Among the 14 collateral-dependent segments, 8 were judged reversible and 6 were judged irreversible at the pharmacological stress test. Seven of the 8 segments that were improved after PTCA had also improved at the pharmacological stress test to yield a sensitivity of 7/8 = 87.5% and a positive predictive value of 7/8 = 87.5%. Five of the 6 segments which remained unchanged after PTCA were judged irreversible. Thus,
Collateral Flow in the Prediction of Wall Reversibility

Fig. 4. Wall motion changes in collateral-dependent segments with wall motion abnormalities before percutaneous transluminal coronary angioplasty (PTCA), at the pharmacological stress test and after PTCA, as compared with baseline values before PTCA.

Fig. 5. This patient underwent PTCA in segment 6. The target coronary artery was dilated from total occlusion to 25%. The reversibility of anterior wall motion with grade 3 Rentrop collateral flow was predicted by the pharmacological stress test.

Left column = equatorial level at baseline before PTCA.
Middle column = equatorial level at the pharmacological stress test.
Right column = equatorial level after PTCA.
Upper row = end-diastolic phase at the equatorial level.
Lower row = maximum-systolic phase at the equatorial level.
PTCA = percutaneous transluminal coronary angioplasty.

the specificity was $5/6 = 83.3\%$, the negative predictive value was $5/6 = 83.3\%$ and the predictive accuracy was $12/14 = 85.7\%$ (Figs. 4, 5, Table II).

Among the 33 collateral-independent segments, 6 were judged reversible and 27 were judged irreversible, including one deteriorated segment, at the pharmacological stress test. Five of the 12 segments that were improved after PTCA had also im-
Fig. 6. Wall motion changes in collateral-independent segments with wall motion abnormalities before percutaneous transluminal coronary angioplasty (PTCA), at the pharmacological stress test and after PTCA, as compared with baseline values before PTCA.

Fig. 7. This patient underwent PTCA in segment 6. The target coronary artery was dilated from total occlusion to 25%. However, the reversibility of anterior wall motion without collateral flow could not be predicted by the pharmacological stress test.

Improved: 12, Improved: 5, Unchanged: 21, Deteriorated: 0, Deteriorated: 0

Improved: 6, Unchanged: 26, Deteriorated: 1

Improved at the pharmacological stress test to yield a sensitivity of 5/12 = 41.7% and a positive predictive value of 5/6 = 83.3%. Twenty of the 21 segments that were unchanged after PTCA were judged irreversible. Thus, the specificity was 20/21 = 95.2%, the negative predictive value was 20/27 = 74.1% and the predictive accuracy was 25/33 = 75.8% (Figs. 6, 7, Table II).

The 7 collateral-independent segments which were not predicted by the pharmacological stress test showed a high degree of stenosis (100%: 4, 99%: 3).

DISCUSSION

Recently, the evaluation of hibernating myocardium by low-dose DOB has been...
studied using echocardiography. However, the mechanism of the effect of DOB on hibernating myocardium has not yet been clarified and several questions remain to be answered. Tamaki et al reported that DOB infusion decreased metabolic reserve in patients with severe coronary artery disease using C-11 palmitate in positron emission tomography. Schulz et al have reported that glycogen and phosphocreatine decreased and lactate increased following simple regional DOB infusion in hypoperfused myocardium in a short-term hibernation model. Moreover, high-dose DOB has been shown to produce new wall motion abnormalities due to ischemia. Eventually, DOB infusion makes ischemia worse in patients with coronary artery disease, and even low-dose DOB may produce new wall motion abnormalities in severe coronary artery disease. Smart et al reported that low-dose DOB (4 μg/kg per min) was more sensitive than high-dose DOB (20–40 μg/kg per min) in detecting stunned myocardium. These findings conflict with the result that gradual administration of DOB (5–20 μg/kg per min) improved wall motion in reversible myocardium. Moreover, inotropic stimulation causes normal myocardium to have a tethering effect on akinetic segments, and the functional border zone can be overestimated. Therefore, the observer’s subjective visual judgment may yield misconceptions. In this study, a combination of low-dose DOB and a vasodilator (nitroglycerin or isosorbide dinitrate) was analyzed quantitatively by UFCT to exclude these problems. We hypothesized that this combination was beneficial because it not only dilated the epicardial coronary artery, collateral artery and coronary stenosis, but also prevented distal coronary artery or collateral vasoconstriction because of the vasodilator, and increased the oxygen supply, which enhanced the contraction by the catecholamine (Fig. 1). Moreover, this combination improved the sensitivity of the pharmacological stress test beyond that of a test which used only low-dose DOB, especially in collateral-dependent segments with wall motion abnormalities. Therefore, the role of collateral flow was investigated in 2 groups; ie, those which showed collateral-dependent segments with wall motion abnormalities and those which showed collateral-independent segments with wall motion abnormalities.

The prediction of wall motion reversibility in collateral-dependent segments with wall motion abnormalities before PTCA yielded satisfactory results, while the prediction of wall motion reversibility in collateral-independent segments with wall motion abnormalities tended to underestimate such reversibility, especially in cases with high levels of stenosis.

Recently, Vanoverschelde et al reported that myocardial blood flow was similar among collateral-dependent segments with and without segmental dysfunction in patients with coronary total occlusion without a history of infarction, but flow reserve decreased in collateral-dependent segments with dysfunction. Therefore, the presence of reversible segments with wall motion abnormalities can be explained by at least two mechanisms: ie, hypoperfusion and some disturbance in the contraction mechanism, even though blood flow is normal at rest due to collateral flow. Low-dose DOB together with a vasodilator may affect this disturbance in the contraction mechanism and improve the wall motion of collateral-dependent segments if those segments are reversible. However, this treatment has limitations as a predictor for collateral-independent segments with high-grade stenosis produced by hypoperfusion.

**Duration and Dose of the Pharmacological Stress Test**

Schulz and co-workers have reported in a short-term hibernation model that the work performed during contraction of hypoperfused myocardium improved temporarily through the use of energy from accumulated phosphocreatine and anaerobic glycolysis following local DOB infusion. Consequently, contraction work may decrease and new wall motion abnormalities may appear if accumulated phosphocreatine is no longer available, since the adenosine triphosphate that is produced by anaerobic glycolysis is insufficient to maintain contraction work. Although the above experimental model does not apply in every aspect to humans, it does indicate the importance of stimulating myocardium by prescribing the most suitable dosage for the optimum period of time.
Schulz and co-workers have reported that hemodynamics stabilized within 5 min, which may be the best time for measurement. Accordingly, when administering a vasodilator, it is important to produce the maximum effect within 5 min.

We previously performed a low-dose DOB stress test using UFCT in control subjects, and it was possible to detect changes in cardiac function and wall motion by this method. We reported that the percent increase in thickening with 4 \( \mu \)g/kg per min of DOB was 70% of that with 8 \( \mu \)g/kg per min in control subjects. As mentioned above, Smart et al. reported that low-dose DOB (4 \( \mu \)g/kg per min) was more sensitive than high-dose DOB because high-dose DOB affects heart rate and systolic pressure. Therefore, 4 \( \mu \)g/kg per min of DOB was used in this study.

Choice of Modalities and Parameters

UFCT possesses rapid scanning, excellent spatial resolution, contrast resolution and reproducibility, and is not invasive! In addition, special technical experience and skill are not needed in UFCT and scanning is not greatly influenced by the patient’s condition, such as obesity or lung disease, as in echocardiography. Furthermore, UFCT images are calculated by a computer and measurements can be obtained quite easily. These attributes make UFCT suitable for quantitative analysis, and for the purposes of the present study.

The thickening of the left ventricular wall, which is a relatively individual parameter rather than an absolute value, is not influenced by heart size, volume or the tethering effect of contraction of normal myocardium following inotropic stimulation. Therefore, wall thickening was chosen as the parameter of quantitative analysis in this study to exclude observer variability and to avoid possible misconceptions. UFCT, but not ventriculography, can be used to measure wall thickening.

Limitations

Only one of 8 ventricular levels was analyzed, which made it impossible to detect improvement in wall thickening in adjacent levels. This is the most important limitation of this study. Naturally, it would have been possible to evaluate wall motion in all 8 levels using an observer’s subjective visual judgment, similar to the wall motion scoring system in echocardiography. However, the purpose of this study was to accurately evaluate the prediction of wall motion reversibility without a subjective observer’s misconceptions, rather than to detect improvement in wall thickening in adjacent levels. Therefore, to increase the accuracy of wall measurement in this study, only the equatorial levels in patients with proximal coronary target lesions were selected, since images of the apical levels were too small to be properly evaluated by quantitative analysis.

In this study, collateral flow was evaluated only by angiography and was not quantified. Therefore, it is questionable whether the vasodilator had some influence on collateral flow beyond that caused by the increase in myocardial oxygen requirements induced by DOB. Consequently, collateral flow should be quantified in a future study.

CONCLUSIONS

The pharmacological stress test in this study (a combination of low-dose dobutamine and a vasodilator) satisfactorily predicted wall motion reversibility in collateral-dependent segments. Therefore, collateral flow may be an important factor in accurately predicting wall motion reversibility using this pharmacological stress test.

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