Myocardial Viability in Patients With Q Wave Myocardial Infarction and No Residual Ischemia

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Background. Coronary revascularization in patients with persistent angina after myocardial infarction reduces the incidence of recurrent angina pectoris and myocardial infarction and improves left ventricular function. The results of revascularization after a Q wave myocardial infarction when there is no residual ischemia may depend on myocardial viability.

Methods and Results. To determine whether there was viable myocardium in the infarct area in the absence of clinical and scintigraphic evidence of myocardial ischemia, 15 asymptomatic patients with a Q wave myocardial infarction, no redistribution on stress 201TI test, and single-vessel disease (>70% stenosis) with persistent anterograde blood flow were randomized to percutaneous transluminal coronary artery angioplasty (PTCA) or conservative medical treatment. After 2 months of follow-up, mean coronary blood flow measured by Doppler catheter in the infarct-related artery was higher in the PTCA treatment group (33±6 ml/min, n=8) than in the conservative treatment group (16±4 ml/min, n=7; p<0.05 between groups). The 201TI pathological-to-normal ratios measured on postexercise images did not change in patients treated conservatively during the follow-up period (\(\Delta=+1.1\pm2.2\%\); NS from baseline) but increased significantly in patients treated by PTCA (\(\Delta=+8.5\pm2.3\%\); p<0.01 from baseline; p<0.05 between groups). Segmental wall motion improved on left ventricular angiography 2 months after PTCA (\(\Delta=+11.5\pm2.2\%\); p<0.001 from baseline) significantly more than in the conservative treatment group (\(\Delta=+4.1\pm1.4\%\); p<0.05 between both groups). Improvements of 201TI ratios and segmental wall motion indexes correlated significantly \((r=0.73, p=0.002)\). The mild improvement of global left ventricular ejection fraction measured in the PTCA treatment group did not differ significantly from changes in the conservative treatment group.

Conclusions. Successful angioplasty of the stenotic infarct artery in patients with a Q wave myocardial infarction and no residual ischemia improved coronary flow, 201TI uptake in the infarct area, and regional wall motion. Therefore, myocardial viability may last several weeks, as long as residual blood flow persists in the infarct-related artery. Optimal assessment of viability by imaging techniques should identify patients who are most likely to benefit from revascularization. (Circulation 1992;86:47–55)

Key Words • percutaneous transluminal coronary angioplasty • left ventricular function • segmental wall motion • stress thallium test • hibernating myocardium

Early reperfusion in acute myocardial infarction can limit infarct size and improves left ventricular (LV) function and survival.1–3 However, when a Q wave myocardial infarction has occurred, and coronary angiography has shown severe stenosis of the infarct-related artery, the clinical significance of the residual stenosis is uncertain. A late intervention to remove the obstruction to coronary flow may be useful if viable myocardium persists in the territory depending on the stenotic or occluded artery.

Previous studies have demonstrated percutaneous transluminal coronary artery angioplasty (PTCA) to be effective when performed early for postinfarction angina.4,5 Furthermore, PTCA improves resting LV systolic function in patients with necrosis and myocardial asynergy.5–8 In these studies, PTCA was performed in patients with recurrent angina pectoris and most often within the first 2 weeks after myocardial infarction. In contrast, it is not known whether successful PTCA of the infarct artery can improve LV systolic function in asymptomatic patients with a Q wave myocardial infarction older than 1 month. We hypothesized that some myocardium may remain viable in those patients with a significant stenosis but persistent anterograde blood flow in the infarct-related artery and that PTCA may reveal this viability.

The problem of differentiating viable from nonviable myocardium can be approached by the evaluation of myocardial perfusion and metabolism with positron emission tomography and, more routinely, by 201TI myo-
cardiac perfusion imaging. However, the definitive evidence of viable myocardium and the utility of any therapy purported to save the jeopardized myocardium are ascertained by the changes of ventricular wall motion. In this study, we considered temporal improvement of regional function to be the main determinant of myocardial viability. LV function was assessed by angiography, which was performed before and 2 months after PTCA or conservative medical treatment, according to randomization. Myocardial viability was further assessed by repeated stress $^{201}$TI tests. After 2 months of follow-up, we measured coronary blood flow by a Doppler catheter to determine whether the expected increase of blood flow induced by PTCA was present several weeks later in the artery related to the infarct zone. Coronary reserve was evaluated using the ischemic effects of contrast media, the relation of which to myocardial viability was recently reported. Therefore, we appreciated the effects of revascularization in terms of epicardial coronary blood flow, $^{201}$TI myocardial perfusion, and both regional and global LV function.

Methods

Patient Selection

The patients selected for study had myocardial infarctions with no evidence of residual ischemia and angiographically proven single-vessel disease. The inclusion criteria were 1) myocardial infarction with Q waves in two leads or more; 2) chronic myocardial infarction (i.e., 6 weeks old or more); 3) no chest pain since the occurrence of myocardial infarction; 4) single-vessel disease with a significant stenosis (>70%) but without occlusion on coronary angiography; 5) negative stress test with neither angina, ischemic change of the ECG, nor significant redistribution of $^{201}$TI defects on the initial single photon emission computed tomography (SPECT) study; and 6) informed consent from the patient for the protocol. Exclusion criteria were 1) total occlusion of the infarct-related artery; 2) significant stenosis of another major vessel; 3) any sign of ischemia during exercise test; 4) associated valvular heart disease; 5) severe, uncontrolled hypertension; 6) second- or third-degree atrioventricular block or any uncontrolled cardiac arrhythmia other than sinus arrhythmia or occasional extrasystoles; 7) any condition that might hinder follow-up evaluation; and 8) participation in other trials. Fifteen consecutive patients were included in the study, and each patient was randomized to PTCA or conventional medical treatment without PTCA. Patients of both groups were prescribed aspirin and acebutolol ($\beta$-blocking drug) unless intolerance or contraindication. Physicians in charge of the patients randomized to conventional medical treatment were informed about the study and were free to prescribe any other drug considered to be helpful to the patient. Randomization of patients was stratified with respect to the involved artery.

$^{201}$TI Single Photon Emission Computed Tomography Imaging

After an overnight fast, all patients underwent graded treadmill exercise testing according to the Bruce protocol. A 12-lead ECG and blood pressure were monitored every minute. The test was considered positive when typical chest pain occurred with ST segment depression (>0.1 mV during 0.08 second). Criteria for the termination of the test were severe chest pain, serious arrhythmia, hypotension, or when the patient reached the age-predicted maximal heart rate or had severe fatigue. According to the patient’s weight, 3–3.5 mCi (111–130 MBq) of $^{201}$TI were administered intravenously at peak exercise, and the patient continued to exercise for one additional minute. SPECT imaging was started 5–9 minutes after $^{201}$TI injection and repeated 6 hours later.

SPECT acquisition was performed with a rotating gamma-camera (GammaTome II, Sophia Medical, Buc, France) and was a 180° acquisition, starting from the left posterior oblique 30° position with a circular orbit. Thirty-two projections were acquired on a step-and-shoot mode (40 seconds per projection for stress imaging, 50 seconds for delayed imaging) with a 64x64 matrix. Slices were obtained by filtered-back projection (Hamming-Hann filter) and reoriented along the three axes with a dedicated computer device (Sophy F., Sophy Medical, Buc, France). Background subtraction and attenuation correction were not used. Volume masking excluded noncardiac activity after back-projection when bowel activity was significant.

A two-dimensional mapping of SPECT data was automatically performed using three-dimensional radial sampling. A bull’s-eye polar map of the maximal value of the myocardial pixels (count-rate density) was displayed to compare postexercise and delayed slices demonstrating the absence of significant redistribution of $^{201}$TI defects on the initial SPECT study. Bull’s-eye analysis is an automatic process with good reproducibility for the comparison of similar regions on two serial scans. Regions of interest, avoiding the border zones, were drawn in normal and pathological areas on the first postexercise bull’s-eye image. Normal areas were easily determined because all patients had single-vessel disease. Average values contained in the pixels allowed automatic calculation of a pathological-to-normal ratio. The same regions of interest were used to calculate a second pathological-to-normal ratio on the second stress $^{201}$TI SPECT performed after 2 months of follow-up.

Cardiac Catheterization

Contrast ventriculography was filmed in the 30° right anterior oblique projection on 35-mm cinefilm at 50 frames per second. Contrast ventriculograms were reviewed on a frame-by-frame basis. End-diastolic and end-systolic silhouettes were traced by two experienced angiographers independently of each other, and only sinus beats, which were not postextrasystolic beats, were analyzed. LV ejection fraction was calculated with the area–length method. Regional wall motion was determined by the area-based method, as previously described. Briefly, a computer program was used to define 11 areas of motion around the superimposed ventricular silhouettes. The most basal inferior area corresponding to the mitral valve location was not analyzed, and no correction was made for displacement. For each segment, the ratio of the segmental area to the sum of areas of all segments determined the segmental area ejection fraction index. Abnormal con-
tractility of a segment was defined as motion exceeding 2 SDs from the normal mean determined from normal patients in our laboratory. The infarct area included adjacent segments with abnormal wall motion. Regional wall motion of this infarct area was calculated as the sum of segmental area ejection fraction indexes of the adjacent segments. Therefore, this value of the infarct zone was a percentage of the global area ejection fraction. In each patient, we compared the value obtained on the first LV angiogram with the value calculated in the same segments on the 2-month follow-up angiogram.

Coronary angioplasty was performed with three or more inflations across the lesion, using sufficient pressure to achieve full balloon expansion. The angioplasty procedure was successful in all patients (residual stenosis <40% in all views and no major procedural complications). Postangioplasty protocol included continuous heparin infusion for 24 hours and aspirin (250 mg daily).

Computer-assisted measurements of coronary artery diameters were made by videodensitometry. The cineframe was digitized, and the image was calibrated on the basis of the known size of the catheter. We used this program to determine the coronary diameter at the site of Doppler flow measurements. Results of PTCA in terms of epicardial blood flow and coronary reserve were assessed only at late follow-up, as early measurements after PTCA are depressed. Similar measurements were also systematically obtained in patients randomized to conventional medical therapy; however, because the stenosis was still present in the infarct artery, it did not allow comparisons of coronary reserve data between the two groups. Coronary flow velocities (V) were measured by a Doppler flow-velocity catheter (20 MHz pulsed Doppler catheter, Millar, Millar Instruments Inc., Houston, Tex.) connected to a velocimeter (Model DC-101, Millar Instruments) and then to a multichannel recorder (Gould ES 1000, Gould Inc., Cleveland, Ohio). Phasic and mean velocity signals were displayed simultaneously with the ECG and blood pressure tracings. After administration of heparin and nitrates, the Doppler catheter was positioned into the infarct-related artery through an 8F guiding catheter over a 0.014-in. steerable guide wire. The position of the Doppler catheter and the range gate control were adjusted to obtain the best audio flow velocity signal and phasic flow velocity waveform. Coronary flow velocities were recorded at rest and during the injection of 6 ml of ioxaglic acid (Hexabrix, Guerbet, Aulnay-sous-Bois, France) at a rate of 4 ml/sec with a power injector, as we described previously. The coronary flow reserve was calculated as the quotient of the mean peak blood flow velocity and mean resting blood flow velocity. The coronary artery diameter was measured at the level of the tip of the Doppler catheter. Cross-sectional area was calculated by the formula: \( \text{CSA} = D^2 \times \pi/4 \), and coronary blood flow was calculated as \( V \times \text{CSA} \).

Study Protocol

Fifteen patients met the inclusion criteria after clinical evaluation, stress \( ^{201}\text{Tl} \) SPECT, and coronary angiography. The patients randomized for PTCA underwent this procedure within the same week. The second evaluation was scheduled between 2 and 3 months later to allow eventual recovery of regional wall motion after revascularization. A stress \( ^{201}\text{Tl} \) SPECT was performed under the same medical treatment as initially. Cardiac catheterization was repeated including coronary angiography and LV angiography. In all patients, coronary blood flow and coronary reserve were measured only at the 2-month follow-up evaluation.

Statistical Analysis

All values are expressed as mean±SEM. Comparisons were made by paired or unpaired \( t \) test where appropriate. Association between variables was determined by linear regression analysis, and a correlation coefficient was calculated between these variables. Statistical significance level was declared at a value of \( p=0.05 \).

Results

Baseline Clinical Characteristics

The patients participating in this study were three women and 12 men (Table 1). Mean age was 48 ±4 years and was comparable in both groups. The infarct-related artery was the right coronary artery in eight patients and the left anterior descending (LAD) coronary artery in seven patients. The LAD coronary artery was involved in four of eight patients in the PTCA group and in three of seven patients in the conservative treatment group. All patients had single-vessel disease with a significant stenosis and a Q wave myocardial infarction at least 6 weeks old. They were free of angina, and there was no evidence of recurrent ischemia on the ECG or stress \( ^{201}\text{Tl} \) SPECT. Mean global LV ejection fraction was 48 ±3% in the PTCA group and 51 ±3% in the conservative treatment group (NS between groups). All patients received aspirin and acebutolol with the exception of three patients who did not tolerate \( \beta \)-blockers (patients 5, 13, and 15). Among patients treated conservatively, four received nitrates, one received calcium channel blockers, and two had converting enzyme inhibitors (two patients received a combination of two of these drugs).

Coronary Hemodynamics

One patient had a significant restenosis on the follow-up angiogram (defined as a stenosis >50%) and was redilated. All data from this patient were kept in the PTCA group for analysis.

Mean Doppler flow velocity measured in the dilated vessels 2 months after PTCA was 6.4 ±0.8 cm/sec, and the calculated mean coronary blood flow was 33 ±6 ml/min. Mean coronary flow was significantly lower in nondilated arteries (16 ±4 ml/min, \( p<0.05 \)). The very low values of coronary reserve (<1.3) were measured in four patients treated conservatively with a persistent severe stenosis of the infarct artery. Coronary blood flow reserve was 2.2 ±0.3 in the PTCA group. Although patients in the PTCA group with high coronary reserve values increased both \( ^{201}\text{Tl} \) score and LV function, there were no significant correlations between improvements of \( ^{201}\text{Tl} \) ratios or segmental wall motion and coronary reserve values.

Myocardial Imaging

During baseline stress \( ^{201}\text{Tl} \) SPECT, 12 of 15 patients achieved 75% or more of the predicted maximal heart
rate. In all patients, the effort level was comparable, and there were no differences in mean maximal heart rate between the first and second stress tests in either group. Baseline pathological-to-normal ratios did not differ significantly between groups. Individual changes of myocardial $^{201}$Tl uptake are shown in Figures 1 and 2. $^{201}$Tl pathological-to-normal ratios improved significantly after PTCA ($\Delta = +8.5 \pm 2.3\%$; $p<0.01$ from baseline) but did not change in patients with conservative treatment ($\Delta = +1.1 \pm 2.2\%$; NS from baseline). Mean increments differed significantly between the two groups ($p<0.05$).

**Left Ventricular Function**

Regional wall motion of the infarct area was expressed as a percentage of the global area ejection fraction (GAE). In all cases, the exercise test produced a comparable level of effort. There were no significant differences in mean maximal heart rates between the first and second stress tests in either group. Baseline pathological-to-normal ratios did not differ significantly between groups. Individual changes of myocardial $^{201}$Tl uptake are shown in Figures 1 and 2. $^{201}$Tl pathological-to-normal ratios improved significantly after PTCA ($\Delta = +8.5 \pm 2.3\%$; $p<0.01$ from baseline) but did not change in patients with conservative treatment ($\Delta = +1.1 \pm 2.2\%$; NS from baseline). Mean increments differed significantly between the two groups ($p<0.05$).

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fraction, and individual changes of segmental area ejection fraction indexes are reported in Figure 3. After PTCA, wall motion of the infarct area improved significantly (Δ = +11.5±2.2% after the follow-up period; p<0.001) (Figure 4). The segmental wall motion of patients allocated to conservative medical treatment increased only slightly (Δ = +4.1±1.4%, p=0.03). Improvement of regional wall motion was significantly greater in the PTCA group compared with the conservative treatment group (p<0.05).

Global LV function increased slightly from 48±3% baseline to 54±3% 2 months after PTCA (p=0.04) but did not change in patients who were not dilated (from 51±3% baseline to 52±4% at 2 months follow-up, p=NS). Mean increments in the two groups were not statistically different (p=NS). Changes of LV volumes in either group were not significant (Table 2).

The individual changes of segmental area ejection fraction indexes are plotted against the changes of 201TI pathological-to-normal ratios for all patients in Figure 5. A relation between improvements of 201TI uptake and regional wall motion of the infarct area is evident (r=0.73, p=0.002).

**Figure 2. Bar graph showing individual changes of 201TI pathological-to-normal ratios (%) in patients treated conservatively (patients 1 to 7) and by revascularization (patients 8 to 15).**

**Figure 3. Bar graph showing individual changes of segmental area ejection fraction indexes (expressed as a percentage of global area ejection fraction) in patients treated conservatively (patients 1 to 7) and by revascularization (patients 8 to 15). EF, ejection fraction; LV angiography, left ventricular angiography.**

**Discussion**

Myocardial asynergy does not imply anatomic fibrosis and loss of myocardial viability. Therefore, this criterion alone, obtained at LV angiography or echocardiography, should not be used for decisions concerning myocardial revascularization even in patients with previous myocardial infarction. Patients with an old Q wave myocardial infarction, a regional contractile dysfunction, and a stenotic infarct-related artery with neither clinical nor scintigraphic evidence of myocardial ischemia would ordinarily be managed conservatively. In this study, however, successful PTCA of the infarct-related vessel improved blood flow, 201TI uptake, and LV function, suggesting persistent viable myocardium. Another important finding of this work was that individual changes of regional LV function correlated significantly with the changes in myocardial 201TI uptake on stress test. Our results demonstrate that in patients with a Q wave myocardial infarction and residual anterograde blood flow in the infarct artery, some viable tissue persists weeks after the acute phase of necrosis despite the absence of clinical ischemia. Coronary blood flow was significantly increased 2 months after PTCA, demonstrating a beneficial effect on epicardial blood flow late after dilatation of the infarct artery. Coronary reserve is commonly measured with drugs causing maximal arterial vasodilatation, which is not adapted to the myocardial metabolic needs. Hyperemia caused by contrast medium injection, although not maximal, is related to the induced transient ischemia, and this effect can be used for assessment of both coronary reserve and myocardial viability.14 In this study, coronary reserve was measured in dilated patients to test another parameter of myocardial viability, and we used contrast medium injection instead of a vasodilating drug to avoid inappropriate pharmacological vasodilatation. Coronary reserve measurements were obtained only 2 months after angioplasty, as early measurements after PTCA underestimate the real value of coronary reserve.21,22 The lack of correlation between the coronary reserve and the recovery of segmental wall motion after PTCA may imply that coronary reserve measured in the infarct artery does not indicate the amount of viable myocardium within the infarct zone.

201TI pathological-to-normal ratios increased after PTCA of the infarct-related artery and were unchanged in patients with persistent underperfused necrotic areas. Because all patients had single-vessel coronary artery disease, normal and pathological areas were easily discriminated, and any increase of pathological-to-normal ratios over the follow-up period was caused by improvement of 201TI uptake in the pathological area. For inclusion in the study, all patients had patent infarct-related arteries, and perfusion of the infarct zone, although severely decreased, never depended on collaterals but only on the infarct-related vessel. Subsequently, the increased anterograde epicardial blood flow obtained by PTCA could explain improvements of 201TI uptake and ventricular function of the infarcted wall. 201TI uptake depends on both coronary flow and tissue viability, and results in the PTCA group demonstrate that both conditions are necessary to improve uptake in the infarct zone. Finally, changes of 201TI uptake correlated well with changes of the regional...
systolic function, and temporal improvements of both parameters appear to be good retrospective markers of myocardial viability. These data suggest that some tissue remains viable for a long time in the zone of earlier infarction when limited anterograde coronary flow is present.

The concept of hibernating myocardium, defined as reduced myocardial blood flow and regional LV function, requires that abnormal contractile function is at least partially reversible when blood flow is restored. Moreover, Rahimtoola pointed out that hibernating myocardium is often painless and does not represent

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<th>No PTCA</th>
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<tr>
<td>Ejection fraction (%)</td>
<td>51±3</td>
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<td>End-diastolic volume index (ml/m²)</td>
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<td>End-systolic volume index (ml/m²)</td>
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<td>Stroke volume (ml/m²)</td>
<td>41±3</td>
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*p<0.05 from baseline.
true myocardial ischemia. The latter has not been considered in previous studies evaluating hibernating myocardium after myocardial infarction, and revascularization in patients with recurrent ischemia after myocardial infarction is certainly beneficial in terms of clinical improvement and recovery of contractile function.4,5,7,8 Our patients had no chest pain at rest and during exercise and had persistent $^{201}$Tl defects on the 6-hour delayed images; this may suggest that standard baseline evaluation by combined clinical, invasive, and noninvasive nuclear studies cannot identify viable myocardium within a zone of Q wave infarction. One limitation is that the stress $^{201}$Tl test used in our protocol is not sensitive enough to detect viability. Patients with persistent defects 3–4 hours after an exercise $^{201}$Tl test can improve $^{201}$Tl uptake and wall motion after revascularization surgery, which demonstrates that standard $^{201}$Tl scintigraphy is imprecise in detecting myocardial viability.28,29 This remark may be extended to our patients, although permanent $^{201}$Tl defects were also associated with a medical history of myocardial infarction with Q waves on the ECG. The selection criteria of patients with persistent defects must be considered, and investigators have reported normalization of 75% of stable defects after PTCA in a patient population with only 8% of ECG evidence of myocardial infarction.30 Redistribution is usually performed 3–4 hours after the injection of $^{201}$Tl and was studied at 6 hours in our patients, but several investigations have demonstrated that late redistribution imaging (performed until 72 hours after injection) can detect $^{201}$Tl uptake in regions with permanent defects at 3–4 hours.31,32 However, delayed $^{201}$Tl imaging yields poor image quality, which may alter the reliability of the results. Further evidence that standard $^{201}$Tl redistribution imaging at 3–4 hours has limited sensitivity is provided by studies with reinjection of $^{201}$Tl, which causes improved uptake in a large number of regions with apparently irreversible $^{201}$Tl defects.33 Enhanced $^{201}$Tl uptake after reinjection represents viable myocardium in regions with persistent defects on standard $^{201}$Tl scintigraphy.33–35 Gibson et al demonstrated with planar scintigraphy that the degree of decreased $^{201}$Tl activity within stable defects was useful in predicting the response to revascularization surgery. A mild reduction in $^{201}$Tl activity (pathological-to-normal ratio between 50% and 75%) in persistent defects was associated more frequently with improvement of segmental wall motion than defects with severe decrease of $^{201}$Tl activity (pathological-to-normal ratio <50%). Similar data were reported with $^{201}$Tl SPECT, which suggests that the amount of $^{201}$Tl activity in irreversible defects is an indicator of viable myocardium.34 Although our study population was different from patients recruited in these studies, the degree of $^{201}$Tl uptake in the Q wave area of our patients may be related to the recovery of segmental wall motion we measured.

Most infarct areas contain both dead and viable myocytes, and positron emission tomography demonstrated a concordant reduction in regional perfusion and glucose usage in only 32% of Q wave regions; the majority of chronic Q wave regions had persistent metabolic activity.37 Stable $^{201}$Tl defects frequently manifest $^{18}$F-2-fluoro-deoxyglucose uptake, which confirms that permanent defects contain viable myocardium.38,39 Nevertheless, $^{201}$Tl imaging with reinjection and interpretation of the level of $^{201}$Tl activity within persistent defects identifies the same viable regions as does positron emission tomography with $^{18}$F-2-fluoro-deoxyglucose.34 Reliable evaluation of viability by $^{201}$Tl SPECT is important to prospectively identify patients in whom it is probable that wall motion will improve after revascularization. We believe that some $^{201}$Tl defects in our patients would have been reversible after $^{201}$Tl reinjection or after late redistribution imaging. Several studies have reported the important prognostic value of redistribution defects after a myocardial infarction, which allows a rational decision as to the probable benefits of revascularization.40,41 However, the objective of this study was not to prospectively identify jeopardized myocardium by $^{201}$Tl SPECT for the purpose of guiding PTCA but to determine whether this procedure could reveal myocardial viability through changes of $^{201}$Tl uptake and LV function.

Reversal of regional dysfunction by revascularization in patients with stable coronary artery disease has been described after days and months of reduced coronary blood flow.8,23,25 However, revascularization in patients with myocardial infarction and persistent underperfusion of the infarct area has been studied mostly within days of the acute episode when stunning, which is another reversible state of contractile dysfunction, is important.8,42,43 To minimize the role of stunned myocardium, we selected patients 6 weeks or more after the acute episode. The spontaneous improvement of wall motion was minimal in patients not treated by angioplasty but might have been better after revascularization when considering baseline $^{201}$Tl ratios. In contrast, successful PTCA clearly increased coronary blood flow and LV function, which suggests that hibernating myocardium was present in these asymptomatic patients with old Q wave myocardial infarctions and critical stenoses on the infarct-related arteries. Most patients treated conservatively were prescribed nitrates and/or converting enzyme inhibitors, which might have played a role in the trend toward decreased end-diastolic
volumes and the mild improvement of segmental wall motion measured in this group.

The decreased contractile function may represent an adaptive response of the myocardium to prolonged underperfusion. This state of chronic perfusion–contraction matching with reduced myocardial energy expenditure has been defined as "chronic hibernation." A metabolic adaptation may occur with decreased aerobic metabolism and increased anaerobic metabolism to keep the myocardium viable. Recovery of oxidative metabolism after recanalization may be the key determinant of functional recovery. Persistence of tissue viability long after a myocardial infarction may depend not only on patency of the infarct-related artery but also on development of collaterals to the infarct area.

Residual myocardial viability several months after acute myocardial infarction has recently been demonstrated by positron emission tomography. A good antegrade flow in the infarct-related artery may be of clinical relevance not only with respect to improved LV function but also to the prevention of severe arrhythmias and reduction of mortality.

We conclude that viable myocardium without concomitant clinical ischemia may exist in patients with myocardial infarction and severe stenosis in the infarct-related coronary artery. Subsequently, myocardial viability is probably present as long as the residual blood flow allows perfusion–contraction matching with a minimal energy demand. Nuclear imaging studies should help evaluate the presence and extent of jeopardized myocardium after a myocardial infarction and identify prospectively the asymptomatic patients who are most likely to benefit from angioplasty of the stenotic infarct-related artery.

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Circulation. 1992;86:47-55
doi: 10.1161/01.CIR.86.1.47

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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