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Effects of Severity of the Residual Stenosis of the Infarct-Related Coronary Artery on Left Ventricular Dilation and Function After Acute Myocardial Infarction

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Objectives. **This study was designed to evaluate the relation between the severity of the residual stenosis of the infarct-related artery and changes in left ventricular volume and function after a first anterior myocardial infarction.**

Background. **Although thrombolytic therapy improves clinical outcome after acute myocardial infarction, the relations between the severity of the residual stenosis of the infarct-related artery and postinfarction left ventricular remodeling and function are unclear.**

Methods. **Fifty-eight patients with a first anterior myocardial infarction and significant disease only in the left anterior descending coronary artery on arteriography performed after 7 to 10 days were evaluated. All patients received thrombolytic therapy. Residual stenosis of the infarct-related artery was measured with quantitative coronary arteriography. Left ventricular volumes and ejection fraction were measured by echocardiography and radionuclide angiography, respectively, 7 to 10 days, 6 months and 1 year after infarction. End-diastolic and end-systolic left ventricular volumes were measured by two-dimensional echocardiography and normalized to body surface area. Patients were classified into three groups according to baseline residual stenosis severity: total occlusion (Group I), minimal lesion diameter <1.5 mm (Group II) and minimal diameter ≥1.5 mm (Group III).**

Results. **Group I patients had significantly greater left ventricular end-diastolic and end-systolic volumes at 6 months and 1 year than did the other groups. Group II patients had greater end-diastolic and end-systolic volumes than did Group III patients at 1 year. In addition, Group I patients had a lower ejection fraction at 1 year than that of the other groups. The minimal lesion diameter was significantly correlated with percent change in end-diastolic volume at 1 year.**

Conclusions. **The severity of the baseline residual stenosis of the infarct-related artery is an important predictor of change in left ventricular volumes in the 1st year after infarction. Total occlusion of the infarct-related artery is associated with greater left ventricular dilation and functional impairment.**

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The presence of a patent infarct-related vessel at the time of hospital discharge has been shown to be one of the most important independent predictors of late survival in patients with acute myocardial infarction (1,2). However, the relation between vessel patency and postinfarction left ventricular remodeling is less understood. Previous studies (3–5) have shown that, apart from infarct size, the presence of a patent infarct-related artery is an important predictor of subsequent left ventricular dilation. However, these studies were performed in patients who did not receive thrombolytic therapy. Because thrombolytic therapy frequently causes reperfusion in the reversibly injured myocardium, probably limiting infarct size and left ventricular dysfunction, the course of subsequent left ventricular remodeling may be modified. In addition, none of these studies employed quantitative angiographic measurements to assess the severity of the residual stenosis of the infarct-related artery.

The aim of the present study was to evaluate the relation between the severity of the residual narrowing of the infarct-related artery assessed by quantitative angiography and changes in left ventricular volume and function in the year after a first anterior myocardial infarction in patients who received early thrombolytic therapy.

Methods

Patient selection. Patients admitted with the diagnosis of a first acute myocardial infarction were considered. Study inclusion criteria were 1) diagnosis of a first acute transmural anterior myocardial infarction with characteristic electrocardiographic (ECG) ST segment elevation and subsequent confirmation by characteristic creatine kinase MB isoenzyme elevation; 2) patient receipt of intravenous thrombolytic treatment within 5 h of the onset of symptoms (recombinant tissue-type plasminogen activator [rt-PA] bolus injection of 10 mg, followed by an additional 50 mg

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during 1 h and 40 mg during the next 2 h); 3) no contraindications to thrombolytic therapy; and 4) significant disease present only in the left anterior descending coronary artery on coronary arteriography (patients with >50% diameter stenosis in vessels other than the infarct-related artery were excluded).

The exclusion criteria were 1) history of previous myocardial infarction; 2) presence of significant valvular heart disease, cardiomyopathy or atrial arrhythmias; 3) inadequate echocardiographic recordings; and 4) subsequent coronary intervention (for example, coronary bypass surgery or angioplasty) before completion of the study protocol.

Protocol. Blood was sampled for serum creatine kinase immediately after admission, every 4 h over the next 24 h and then every 8 to 12 h over the next 48 h. Peak serum creatine kinase was taken as the estimate of infarct size. Between 7 and 10 days after admission, all patients underwent coronary arteriography, echocardiography and radionuclide angiography. Echocardiographic and radionuclide studies were repeated at 6 months and 1 year. All patients had given written informed consent for the study, which was approved by the Institutional Ethical Committee.

Patients were considered to have acute (<24 h) reperfusion based on the following noninvasive clinical criteria (6–9): 1) early peak creatine kinase level ≤12 h after the start of thrombolysis; 2) ≥50% reduction in ST segment elevation 90 to 120 min after the start of thrombolysis; 3) reperfusion arrhythmias within the 1st 90 min of thrombolytic therapy; and 4) abrupt lessening of chest pain after thrombolytic therapy.

Echocardiography. Two-dimensional echocardiographic examination was performed by means of a Hewlett-Packard model 77020A echocardiographic system with a 2.5-MHz transducer. Left ventricular end-diastolic and end-systolic volumes were computed according to an algorithm that related left ventricular volume to a half-ellipsoid and half-cylinder geometry (10). An apical four-chamber ventricular long-axis view and perpendicular mid-ventricular septum to lateral wall distance were used, assuming a circular left ventricular cross-sectional configuration. End-diastole was defined as the frame coincident with the onset of the QRS complex on the ECG and end-systole as the subsequent frame with the smallest ventricular cavity area. All volumes were normalized to body surface area. The examinations were reviewed and analyzed by two independent observers. Concordance of analysis was achieved in 95% of cases. If discrepancies arose, these studies were reviewed and agreement was obtained.

Radionuclide angiography. High temporal resolution radionuclide ventriculography was performed with the patient at rest in the supine position. Red blood cells were labeled in vivo with 25 mCi of technetium-99m. Imaging was performed with a small field of view Anger camera equipped with a low energy, general purpose, parallel hole collimator oriented in the 45° left anterior oblique position, with a 15° caudal tilt. Data were acquired in frame mode by computer-based ECG gating, with 2× digital zoom. Ejection fraction was measured on the raw time-activity curve by a standard technique and expressed as a percent (11).

Cardiac catheterization and quantitative coronary arteriography. Selective coronary arteriography was performed with the percutaneous femoral approach. Coronary angiograms were obtained after the administration of intracoronary nitroglycerin. Nitroglycerin was employed to minimize the effect of varying vasomotor tone on vessel lumen diameter. All angiograms were reviewed as suitable for analysis by quantitative coronary arteriography. Multiple projections including cranial- and caudal-angulated views were obtained for all patients. The infarct-related artery was identified by analysis of the acute ECG ST segment changes, the site of regional wall motion abnormalities on two-dimensional echocardiography and the coronary angiogram.

Coronary cineangiographic films were analyzed by a previously described (12) computer-assisted edge detection coronary quantitation system. End-diastolic cine frames that showed the narrowest stenotic diameter and clearly demonstrated the stenotic segment were selected and magnified (×4). Coronary segments were centered in the image field. The image was digitized and then processed by an Epix 4 Meg video processor board in a Hewlett-Packard host computer. The user was then asked to place several points along each edge of the segment to be measured. These points were used to guide the edge-searching process. The user-specified points were connected by a curve generated by cubic spline interpolation. At each point along this curve, a perpendicular line of 24 pixels centered on the curve was constructed. This line was perpendicular to and centered on the edge of the coronary artery segment. The image was sampled at each point along the perpendicular line. These 24-pixel values form a brightness function that had a low value outside the opacified coronary artery and increased to a high value within the artery. The edge was defined by using an algorithm of the weighted sum of first and second derivatives of the brightness function. Detected edges were presented to the user for (optional) editing and approval. At no time was the length of an edited margin >20% of the total length of the quantitated segment. Once the edges were approved, a center line was constructed. The diameter was computed as the distance between the edges perpendicular to the center line. The diameter function was filtered by a 5-point median filter to remove artifacts occasionally introduced by unusual segment geometry. The user then indicated the segment fiducial (starting) and end points. The minimal lesion diameter was used for subsequent analysis. Catheters of known diameter were used for calibration (13).

Patients were stratified into three groups according to whether the minimal lesion diameter was 0 (total occlusion), <1.5 or ≥1.5 mm.

Statistical analysis. All data are expressed as mean value ± SD unless otherwise stated. Analysis of variance was used to assess serial measurements and the differences among groups. Correlations between variables were assessed using

Table 1. Baseline Clinical Characteristics of the Three Patient Groups

	Group I (total occlusion) (n = 14)	Group II (<1.5-mm stenosis) (n = 24)	Group III (≥1.5-mm stenosis) (n = 20)
Age (yr)	58 ± 10	56 ± 9	58 ± 7
Male/female (no.)	10/4	21/3	17/3
Peak CK (U/liter)	2,645 ± 1,452	2,214 ± 1,322	2,295 ± 1,296
Minimal lesion diameter (mm)	0.0 ± 0.0	0.9 ± 0.4	1.7 ± 0.6
EDV (ml/m^2)	74.3 ± 8.5	70.5 ± 8.3	68.7 ± 7.9
ESV (ml/m^2)	50.5 ± 7.5	45.6 ± 8.1	42.7 ± 7.8
Rest EF (%)	43.7 ± 9.2	42.5 ± 8.4	44.8 ± 7.6

CK = serum creatine kinase; EDV = left ventricular end-diastolic volume; EF = left ventricular ejection fraction; ESV = left ventricular end-systolic volume.

linear regression analysis and Pearson's correlation coefficient. Multivariate regression analysis was performed to test dependency of change in left ventricular volumes on minimal lesion diameter and peak creatine kinase. Differences were considered significant when the confidence limits were >95% ($p < 0.05$).

Results

Clinical characteristics (Table 1). Of 66 patients initially enrolled in the study, 8 were subsequently excluded because 4 died and 4 underwent either surgical coronary bypass or coronary angioplasty during the follow-up period. The final study group consisted of 58 patients, 48 men and 10 women with a mean age of 56 ± 9 years.

There were 14 patients in Group I (total occlusion), 24 in Group II (minimal lesion diameter <1.5 mm) and 20 in Group III (minimal lesion diameter >1.5 mm). The mean lumen diameter was 0.9 ± 0.4 mm in Group II and 1.7 ± 0.6 mm in Group III. There were no significant differences in age, gender distribution, medications and hemodynamic variables among the three groups. Patients in Group I had a higher peak creatine kinase level than did the other groups, although the difference was not significant (Group I, 2,645 ± 1,452 U/liter; Group II, 2,214 ± 1,322 U/liter and Group III, 2,295 ± 1,296 U/liter; p = NS). No patient was taking any angiotensin-converting enzyme inhibitor during the study.

Changes in Left Ventricular Volumes and Ejection Fraction (Fig. 1 and 2)

At baseline, there were no significant differences in left ventricular end-diastolic volume, end-systolic volume and ejection fraction among the three groups (Table 1). Group I and Group II patients showed a progressive increase in left ventricular end-diastolic and end-systolic volumes during the follow-up period.

End-diastolic volume. In Group I, end-diastolic volume increased from 74.3 ± 8.5 to 94.1 ± 9.3 ml/m^2 at 6 months ($p < 0.01$) and to 104.2 ± 10.1 ml/m^2 at 1 year ($p < 0.01$ vs. baseline and $p < 0.01$ vs. 6 months). In Group II, end-diastolic volume increased from 70.5 ± 8.3 to 79.6 ± 10.2 ml/m^2 at 6 months (p = NS) and to 84.8 ± 8.9 ml/m^2 at 1 year ($p < 0.01$ vs. baseline and $p < 0.02$ vs. 6 months). However, there were no significant differences in end-diastolic volume in Group III at baseline (68.7 ± 7.9 ml/m^2), 6 months (71.3 ± 9.8 ml/m^2) and 1 year (73.2 ± 8.3 ml/m^2). Comparisons among groups revealed that Group I had a

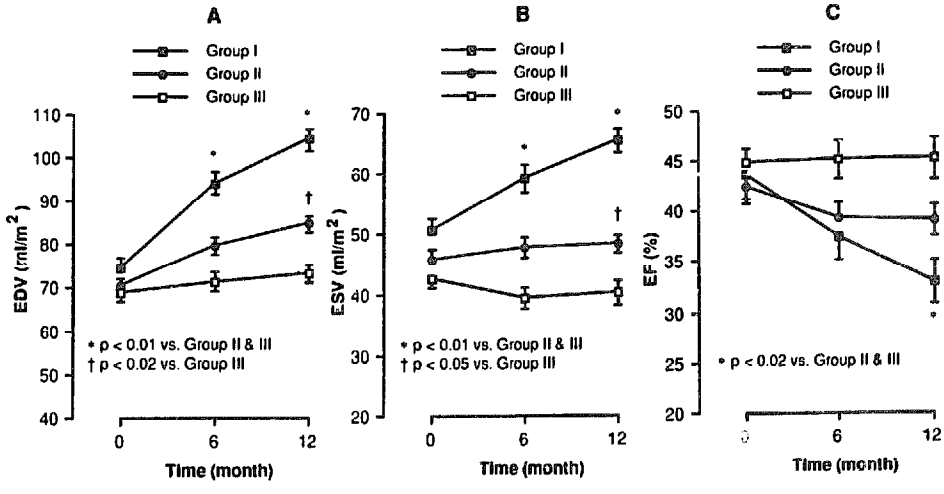


Figure 1. Graphs showing the serial changes in left ventricular end-diastolic volume (EDV) (A), end-systolic volume (ESV) (B) and ejection fraction (EF) (C) in the three study groups in the 1-year follow-up period. Data are expressed as the mean value ± SEM.

JACC Vol. 20, No. 2
August 1992:307–13

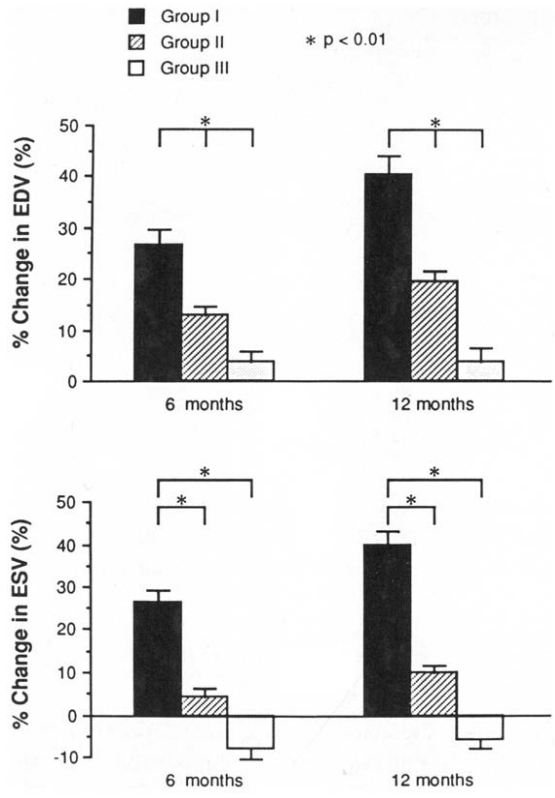


Figure 2. Graphs showing percent changes in end-diastolic volume (EDV) (**upper**) and end-systolic volume (ESV) (**lower**) in the three study groups at 6 and 12 months after myocardial infarction. Data are expressed as mean value ± SEM.

significantly larger end-diastolic volume than that in Groups II and III at both 6 months and 1 year (6 months, 94.1 ± 9.3 vs. 79.6 ± 10.2 and 71.3 ± 9.8 ml/m^2, respectively, $p < 0.01$; 1 year, 104.2 ± 10.1 vs. 84.8 ± 8.9 and 73.2 ± 8.3 ml/m^2, respectively, $p < 0.01$). Group II also had a significantly higher end-diastolic volume at 1 year compared with that in Group III (84.8 ± 8.9 vs. 73.2 ± 8.3 ml/m^2, $p < 0.02$) (Fig. 1A).

End-systolic volume. In Group I, end-systolic volume increased from 50.5 ± 7.5 to 59.1 ± 8.7 ml/m^2 at 6 months ($p < 0.02$) and to 65.4 ± 7.4 ml/m^2 at 1 year ($p < 0.01$ vs. baseline and $p < 0.02$ vs. 6 months). In Group II, end-systolic volume increased from 45.6 ± 8.1 to 47.7 ± 8.8 ml/m^2 at 6 months (p = NS) and to 50.2 ± 7.1 ml/m^2 at 1 year ($p < 0.02$ vs. baseline and p = NS vs. 6 months). However, there were no significant differences in end-systolic volume in Group III at baseline (42.7 ± 7.8 ml/m^2), 6 months (39.4 ± 7.9 ml/m^2) and 1 year (40.3 ± 8.3 ml/m^2). Comparisons among groups revealed that Group I had a significantly larger end-systolic volume than did Groups II and III at both 6 months and 1 year (6 months, 59.1 ± 8.7 vs. 47.7 ± 8.8 and 39.4 ± 7.9 ml/m^2, respectively, $p < 0.01$; 1 year, 65.4 ± 7.4 vs. 50.2 ± 7.1 and 40.3 ± 8.3 ml/m^2, respectively, $p < 0.01$). Group II also had a significantly higher end-systolic volume at 1 year compared with that in Group III (50.2 ± 7.1 vs. 40.3 ± 8.3 ml/m^2, $p < 0.05$) (Fig 1B).

Ejection fraction. Left ventricular ejection fraction in Group I decreased from 43.7 ± 9.2% to 37.5 ± 8.7% at 6 months (p = NS) and to 33.1 ± 7.7% at 1 year ($p < 0.01$ vs. baseline), so that it was significantly lower than that of Groups II and III at 1 year (33.1 ± 7.7% vs. 39.2 ± 7.6% and 46.4 ± 9.1%, respectively, $p < 0.02$). Ejection fraction in Groups II and III showed no significant serial changes (Fig. 1C). Figure 2 shows the percent changes in end-diastolic volume and end-systolic volume in the three groups at 6 and 12 months.

Effect of acute reperfusion (<24 h) on left ventricular volumes and function (Table 2). There were 31 patients (4 in Group I, 14 in Group II and 13 in Group III) who showed clinical criteria of acute reperfusion (<24 h). There were no significant differences between patients with or without acute reperfusion in terms of serial end-systolic volume, end-diastolic volume and ejection fraction in all three groups.

Relation of minimal lesion diameter to change in left ventricular end-diastolic volume (Fig. 3). The relation of minimal lesion diameter and peak creatine kinase (an estimation of infarct size) to the change in left ventricular volumes at 1 year was analyzed by multivariate regression analysis. Both variables were found to be significant independent predictors of end-diastolic volume change at 1 year ($r = 0.71$, $p < 0.001$). Of the two variables, minimal lesion diameter had a higher partial correlation coefficient ($r = 0.66$, $p < 0.001$) than did peak creatine kinase ($r = 0.38$, $p < 0.01$).

In Figure 3, the percent change in left ventricular end-diastolic volume at 1 year was plotted against minimal lesion diameter. Although there was a significant linear relation between them ($r = 0.66$, $p < 0.001$), the distribution of the data points suggested a nonlinear pattern. Accordingly, polynomial curve-fitting analysis was performed. Data points were best represented by a second degree polynomial function, with a correlation coefficient of 0.77 ($p < 0.001$).

Discussion

The present study showed that left ventricular dilation occurs after thrombolytic therapy in patients with acute anterior myocardial infarction. The degree of left ventricular dilation is related to the severity of the residual stenosis of the infarct-related artery and is independent of final infarct size. This suggests that the degree of continuing perfusion of the infarct area is important for the subsequent left ventricular remodeling process even after early reperfusion.

Infarct expansion and left ventricular remodeling after myocardial infarction. Left ventricular volume is a well recognized prognostic factor in patients recovering from myocardial infarction (14). In fact, in patients with coronary artery disease, left ventricular volume, even more than the

Table 2. Effects of Clinical Acute Reperfusion (<24 h) on Serial Left Ventricular Dilation and Ejection Fraction in the Three Patient Groups

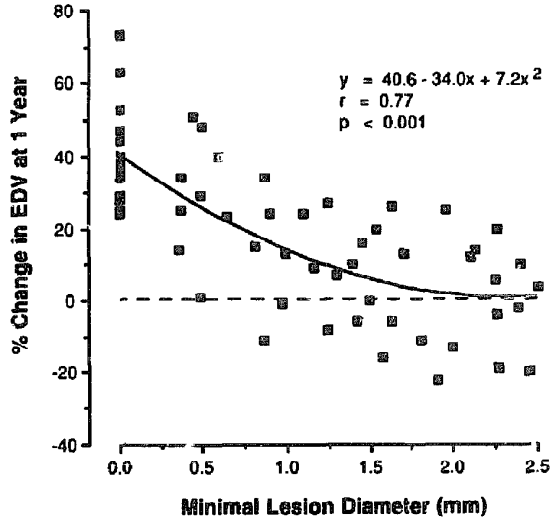
	Group I (total occlusion) (n = 14)		Group II (<1.5-mm stenosis) (n = 24)		Group III (≥1.5-mm stenosis) (n = 20)	
	Reperfusion (n = 4)	No Reperfusion (n = 10)	Reperfusion (n = 14)	No Reperfusion (n = 10)	Reperfusion (n = 13)	No Reperfusion (n = 7)
ESV (ml/m²)						
Baseline	49.4 ± 7.9	50.9 ± 8.4	46.2 ± 8.7	44.8 ± 8.9	43.1 ± 7.3	42.0 ± 7.7
6 Months	60.2 ± 9.1	58.7 ± 7.8	47.1 ± 8.7	48.5 ± 8.3	38.6 ± 8.9	40.9 ± 8.2
1 Year	64.2 ± 8.6	65.9 ± 8.2	49.6 ± 8.9	51.0 ± 7.4	39.4 ± 7.4	42.0 ± 8.7
EDV (ml/m²)						
Baseline	72.5 ± 8.1	75.0 ± 7.3	68.9 ± 7.5	72.7 ± 7.9	68.5 ± 8.5	69.1 ± 9.5
6 Months	92.5 ± 9.2	94.7 ± 8.8	80.2 ± 8.4	78.8 ± 9.0	70.5 ± 7.2	72.8 ± 8.9
1 Year	102.7 ± 8.7	104.8 ± 7.5	83.9 ± 8.5	86.1 ± 7.5	72.2 ± 8.1	75.1 ± 8.0
EF (%)						
Baseline	44.5 ± 8.9	43.4 ± 7.7	43.2 ± 8.4	41.5 ± 7.6	45.6 ± 7.2	43.3 ± 7.6
6 Months	38.2 ± 7.9	37.2 ± 7.6	39.8 ± 8.8	40.8 ± 8.9	44.5 ± 8.5	46.5 ± 9.1
1 Year	34.5 ± 8.2	32.5 ± 9.3	38.2 ± 8.1	40.6 ± 7.3	45.3 ± 8.9	48.4 ± 7.8

There were no significant differences between patients with or without acute reperfusion for serial volumes and ejection fraction in all three groups. Abbreviations as in Table 1.

extent of coronary artery disease, represents the most potent predictor of subsequent death (15).

Myocardial expansion has been observed within the first few days after acute myocardial infarction and continues over a period of 6 to 30 months (3,16–19). Myocardial expansion is accompanied by regional deformation of ventricular shape and marked abnormality of the infarct-related segment, leading to a significant increase in left ventricular cavity size. McKay et al. (20) developed a model of left ventricular remodeling after acute myocardial infarction. In this model, acute infarction induces left ventricular systolic and diastolic dysfunction, leading to an increase in systolic and diastolic wall stress in both infarcted and noninfarcted segments. In the infarcted segments, dilation and thinning progress until the healing of injured myocardium increases the ability of the zone to resist wall stress. In noninfarcted segments, diastolic stress provides the stimulus for volume overload hypertrophy that tends to return systolic and diastolic wall stress toward normal. These remodeling changes that occur in some patients after acute myocardial infarction result in an increase in left ventricular volume and seem to be associated with hemodynamic improvement, including lower left ventricular filling pressure and increased cardiac output.

Figure 3. Plot of relation between minimal lesion diameter and percent change in left ventricular end-diastolic volume (EDV) at 1 year in 58 patients. In view of the nonlinear appearance of the data points, a second-degree polynomial curve-fitting approach was used.



Role of patency of the infarct-related artery. Lamas et al. (21) found that the characteristics of the infarct-related vessel were important in determining the severity of infarct expansion and left ventricular distortion early after infarction. In patients with severe left ventricular shape distortion, the incidence of a totally occluded infarct-related vessel at catheterization approximately 2 weeks after acute myocardial infarction was >80%.

The presence of a patent infarct-related vessel at the time of hospital discharge has been shown to be one of the most important independent predictors of late survival (1,2). Jeremy et al. (4) found that left ventricular end-diastolic volume increased in patients without spontaneous reperfusion of the infarct-related artery and only rarely in patients with spontaneous reperfusion. The only coronary anatomic predictor of progressive left ventricular dilation was an occluded infarct-related vessel without collateral filling at the time of predischarge catheterization. A similar finding was described by Pfeffer et al. (22). Patients with an occluded infarct-related vessel showed chronic left ventricular en-

JACC Vol. 20, No. 2
August 1992:307–13

largement. In a study of serial changes in left ventricular volumes after thrombolytic therapy for acute myocardial infarction, Lavie et al. (23), also revealed that patients with a nonreperfused artery had greater left ventricular dilation than did patients with a reperfused artery. Blanke et al. (24) noted that spontaneous reperfusion of the infarct-related artery was associated with improved left ventricular function. Previous animal studies (25,26) also showed that relatively late coronary reperfusion, performed after the time required to salvage significant quantities of myocardium, is associated with a decrease in wall thinning, dilation and aneurysm formation. The findings in the present study are consistent with these previous reports in showing that patients with total occlusion have significantly more left ventricular dilation and reduced ejection fraction in the year after infarction.

Correlation of change in left ventricular end-diastolic volume and severity of residual stenosis. The present study suggests that the degree of perfusion of the infarct-related artery after the initial insult is important in the process of continuing infarct expansion and subsequent left ventricular dilation. The association between persistent occlusion of the left anterior descending artery and aneurysm formation in patients with anterior myocardial infarction (27) and the observations in animal studies (26) that an unperfused infarct is more likely to expand than a perfused infarct are consistent with our findings.

These observations suggest that perfusion of the infarct-related artery after the acute myocardial insult may influence the myocardial infarct healing and remodeling processes. Improvement in myocardial healing and preservation of an epicardial rim of tissue by continuing perfusion of the patent infarct-related artery may result in preserved left ventricular cavity size and avoidance of aneurysm formation (28). This may have multiple secondary beneficial effects, including less predisposition to arrhythmias, fewer thromboembolic complications of mural thrombus, decreased likelihood of cardiac rupture and improved diastolic and to a lesser extent systolic function by virtue of preservation of the cavity geometry. These effects may contribute to the improved survival observed in patients with a patent infarct-related vessel after infarction.

Histologic differences between unperfused and reperfused infarcts have been observed and this may be responsible for the difference in propensity to ventricular dilation. Absence of perfusion is associated with coagulation necrosis, whereas reperfusion causes contraction band necrosis (29). Connelly et al. (30) suggested that the tensile strength of unperfused and reperfused infarct scars is different, although there is doubt as to whether this is significant at physiologic pressures. It is possible that reperfusion during the early healing phase may prevent the process of myocyte slippage and infarct thinning, which results in infarct expansion and may lead to continuing ventricular dilation. Another possibility is that reperfusion may prevent expansion and dilation by preserving islands of epicardium. It may also be that the diastolic properties of the infarct area or surrounding ischemic myocardium may be altered and favor ventricular dilation (31).

The nonlinear relation between left ventricular end-diastolic volume change and minimal lesion diameter (Fig. 3) shows that left ventricular dilation decreases dramatically with an increase in minimal lesion diameter. Given the same infarct size, when the minimal lesion diameter is >1.5 mm, subsequent left ventricular dilation is minimal. Previous studies (31–33) have shown that minimal lumen dimension (either linear diameter or cross-sectional area) is important in predicting the functional impairment of the left ventricle associated with coronary stenosis. Tobis et al. (33) showed that a minimal lesion diameter <1.5 mm in the proximal left anterior descending artery identified patients likely to have functional impairment during atrial pacing as assessed by either global ejection fraction or segmental wall motion defects. Therefore, when the minimal diameter of the infarct-related artery is >1.5 mm, perfusion of the infarct area may be adequate, even during periods of left ventricular stress. This may prevent subsequent left ventricular dilation.

Role of therapy. Medical therapies have recently been shown to reduce infarct expansion and left ventricular dilation after acute myocardial infarction. Captopril has been shown to reduce ventricular dilation and mortality after experimental infarction in animals (34) and to decrease ventricular dilation in asymptomatic patients after acute anterior myocardial infarction (35). The effect of other drugs (for example, nitrates and calcium channel blocking agents) on left ventricular dilation is not well established (36,37). This study suggests that more aggressive therapy (for example, angiotensin-converting enzyme inhibiting agents) should be given to patients with an occluded vessel or severe residual stenosis after infarction to minimize subsequent left ventricular dilation. Whether coronary angioplasty or other interventional procedures should be performed to improve perfusion of the infarct-related vessel to minimize left ventricular dilation remains to be addressed by future studies. An appropriate patient study group would be those patients with acute myocardial infarction who have variable degrees of residual stenosis after early treatment with angioplasty.

References

1. Stadius ML, Davis K, Maynard C, Ritchie JL, Kennedy JW. Risk stratification for 1 year survival based on characteristics identified in the early hours of acute myocardial infarction. Circulation 1986;74:703–11.
2. Dalen JE, Gore JM, Braunwald E, et al. Six- and twelve-month follow-up of the phase I Thrombolysis in Myocardial Infarction (TIMI) trial. Am J Cardiol 1988;62:179–85.
3. Hochman JS, Bulkley BH. Expansion of acute myocardial infarction: an experimental study. Circulation 1982;65:1446–50.
4. Jeremy RW, Hackworthy RA, Bautovich G, Hutton BF, Harris PJ. Infarct artery perfusion and changes in left ventricular volume in the month after acute myocardial infarction. J Am Coll Cardiol 1987;9:989–95.
5. Jeremy RW, Allman KC, Bautovitch G, Harris PJ. Patterns of left

ventricular dilation during the six months after myocardial infarction. J Am Coll Cardiol 1989;13:304–10.

6. Hohnloser SH, Zabel M, Kasper W, Meinertz T, Just H. Assessment of coronary artery patency after thrombolytic therapy: accurate prediction utilizing the combined analysis of three noninvasive markers. J Am Coll Cardiol 1991;18:44–9.
7. Hogg KJ, Hornung RS, Howie CA, et al. Electrocardiographic prediction of coronary artery patency after thrombolytic therapy after acute myocardial infarction: use of the ST-segment as a non-invasive marker. Br Heart J 1988;60:275–80.
8. Goldberg S, Greenspon A, Urban P, et al. Reperfusion arrhythmia: a marker of restoration of antegrade flow during intracoronary thrombolysis for acute myocardial infarction. Am Heart J 1983;105:26–32.
9. Kircher BJ, Topol EJ, O'Neil W, Pitt B. Prediction of infarct coronary artery recanalization after intravenous thrombolytic therapy. Am J Cardiol 1987;59:513–5.
10. Wyatt HL, Heng MK, Meerbaum S, et al. Cross-sectional echocardiography. II. Analysis of mathematic models for quantifying volume of the formalin-fixed left ventricle. Circulation 1980;61:1119–25.
11. Borer JS, Bacharach SL, Green MV, Kent KM, Epstein SE, Johnston GS. Real time radionuclide cine angiography in the noninvasive evaluation of global and regional left ventricular function at rest and during exercise in patients with coronary artery disease. N Engl J Med 1977;296: 839–44.
12. Leung WH, Sanders W, Alderman EL. Coronary artery quantitation and data management system for paired cine angiograms. Cathet Cardiovasc Diagn 1991;24:121–34.
13. Leung WH, Demopulos PA, Alderman EL, Sanders W, Stadius ML. Evaluation of catheters and metallic catheter markers as calibration standard for measurement of coronary dimension. Cathet Cardiovasc Diagn 1990;21:148–53.
14. Henning H, Gilpin EA, Covell JW, Swan EA, O'Rourke RA, Ross J Jr. Prognosis after acute myocardial infarction: a multivariate analysis of mortality and survival. Circulation 1979;59:1124–33.
15. White HD, Norris RM, Brown MA, Brandt PWT, Whitlock RML, Wild CJ. Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction. Circulation 1987;76:44–51.
16. Erlebacher JA, Weiss JL, Eaton LW, Kallmann C, Weisfeldt ML, Bulkley BH. Late effects of acute infarct dilation on heart size: a two-dimensional echocardiographic study. Am J Cardiol 1982;49:1120–6.
17. Eaton LW, Weiss JL, Bulkley BH, Garrison JB, Weisfeldt ML. Regional cardiac dilatation after acute myocardial infarction: recognition by two-dimensional echocardiography. N Engl J Med 1979;300:57–62.
18. Hutchins GM, Bulkley BH. Infarct expansion versus extension: two different complications of acute myocardial infarction. J Am Coll Cardiol 1978;41:1127–32.
19. Weisman HF, Bush DE, Mannisi JA, Bulkley BH. Global cardiac remodeling after acute myocardial infarction: a study in the rat model. J Am Coll Cardiol 1985;5:1355–62.
20. McKay RG, Pfeffer MA, Pasternak RC, et al. Left ventricular remodeling after myocardial infarction: a corollary to infarct expansion. Circulation 1986;74:693–702.
21. Lamas GA, Vaughan DE, Pfeffer MA. Left ventricular thrombus formation after first anterior wall acute myocardial infarction. Am J Cardiol 1988;62:31–5.
22. Pfeffer MA, Lamas GA, Vaughan DE, Parisi AF, Braunwald E. Effect of captopril on progressive ventricular dilatation after anterior myocardial infarction. N Engl J Med 1988;319:80–6.
23. Lavie CJ, O'Keefe JH, Chesebro JH, Clements IP, Gibbons RJ. Prevention of late ventricular dilatation after acute myocardial infarction by successful thrombolytic reperfusion. Am J Cardiol 1990;66:31–6.
24. Blanke H, Cohen M, Karsch KR, Fagerstrom R, Rentrop KP. Prevalence and significance of residual flow to the infarct zone during acute myocardial infarction. J Am Coll Cardiol 1985;5:827–31.
25. Force T, Kemper A, Leavitt M, Parisi AF. Acute reduction in functional infarct expansion with late coronary reperfusion: assessment with quantitative two-dimensional echocardiography. J Am Coll Cardiol 1988;11. 192–200.
26. Hochman JS, Choo H. Limitation of myocardial infarct expansion by reperfusion independent of myocardial salvage. Circulation 1987;75:299–306.
27. Forman MB, Kopelman HA, Collins HW, et al. Poor collaterals and single vessel disease predispose to aneurysm formation after anterior myocardial infarction (abstr). Circulation 1985;72(suppl III):III–439.
28. Hale SL, Kloner RA. Left ventricular topographic alterations in the completely healed rat infarct caused by early and late coronary artery reperfusion. Am Heart J 1988;116:1508–13.
29. Hutchins GM, Bulkley BH. Correlation of myocardial contraction band necrosis and vascular patency: a study of coronary artery bypass graft anastomoses at branch points. Lab Invest 1977;36:642–8.
30. Connelly CM, Vogel WM, Wiegner AW, et al. Effects of reperfusion after coronary artery occlusion of post-infarction scar tissue. Circ Res 1985; 57:562–77.
31. Leung WH, Lau CP. Correlation of quantitative angiographic parameters and changes in left ventricular diastolic function after angioplasty of the left anterior descending artery. Am J Cardiol 1991;67:1061–6.
32. Harrison DG, White CW, Hiratzka LF, et al. The value of lesion cross-sectional area determined by quantitative coronary angiography in assessing the physiologic significance of proximal left anterior descending coronary arterial stenoses. Circulation 1984;69:1111–9.
33. Tobis J, Sato D, Nalcioglu O, et al. Correlation of minimum coronary lumen diameter with left ventricular functional impairment induced by atrial pacing. Am J Cardiol 1988;61:697–703.
34. Pfeffer JM, Pfeffer MA, Braunwald E. Influence of chronic captopril therapy on the infarcted left ventricle of the rat. Circ Res 1985:57:84–95.
35. Pfeffer MA, Lamas GA, Vaughan DE, Parisi AF, Braunwald E. Effect of captopril on progressive ventricular dilatation after anterior myocardial infarction. N Engl J Med 1988;319:80–6.
36. Judgutt BI, Warnica JW. Intravenous nitroglycerin therapy to limit myocardial infarct size, expansion, and complications: effect of timing, dosage, and infarct location. Circulation 1988;78:906–19.
37. Gottlieb SO, Becker LC, Weiss JL, et al. Nifedipine in acute myocardial infarction: an assessment of left ventricular function, infarct size, and infarct expansion: a double blind, randomised, placebo controlled trial. Br Heart J 1988;59:411–8.