JACC Vol. 8, No. 3
September 1986:496–503

Prevention of Subsequent Exercise-Induced Periinfarct Ischemia by Emergency Coronary Angioplasty in Acute Myocardial Infarction: Comparison With Intracoronary Streptokinase

ANTHONY Y. FUNG, MD, PETER LAI, MD, JACK E. JUNI, MD,
PATRICK D. V. BOURDILLON, MD, JOSEPH A. WALTON, JR., MD, NATHAN LAUFER, MD,
ANDREW J. BUDA, MD, FACC, BERTRAM PITT, MD, FACC, WILLIAM W. O'NEILL, MD

Ann Arbor, Michigan

To compare the efficacy of emergency percutaneous transluminal coronary angioplasty and intracoronary streptokinase in preventing exercise-induced periinfarct ischemia, 28 patients presenting within 12 hours of the onset of symptoms of acute myocardial infarction were prospectively randomized. Of these, 14 patients were treated with emergency angioplasty and 14 patients received intracoronary streptokinase. Recatheterization and submaximal exercise thallium-201 single photon emission computed tomography were performed before hospital discharge. Periinfarct ischemia was defined as a reversible thallium defect adjacent to a fixed defect assessed qualitatively.

Successful reperfusion was achieved in 86% of patients treated with emergency angioplasty and 86% of patients treated with intracoronary streptokinase (p = NS). Residual stenosis of the infarct-related coronary artery shown at predischarge angiography was 43.8 ± 31.4% for the angioplasty group and 75.0 ± 15.6% for the streptokinase group ($p < 0.05$). Of the angioplasty group, 9% developed exercise-induced periinfarct ischemia compared with 60% of the streptokinase group ($p < 0.05$). Thus, patients with acute myocardial infarction treated with emergency angioplasty had significantly less severe residual coronary stenosis and exercise-induced periinfarct ischemia than did those treated with intracoronary streptokinase. These results suggest further application of coronary angioplasty in the management of acute myocardial infarction.

(J Am Coll Cardiol 1986;8:496–503)

Early coronary reperfusion after acute occlusion has the potential to salvage myocardium (1–3). Reperfusion can be achieved by thrombolytic therapy (4–7), emergency bypass graft surgery (3) or emergency percutaneous transluminal coronary angioplasty (8–9). After thrombolytic therapy with streptokinase, however, the majority of patients have significant residual coronary lesions (10). After maximal dilation by angioplasty, the residual stenosis is usually low grade (11). Thus, patients treated with streptokinase may be more likely than those treated with emergency angioplasty to develop exercise-induced periinfarct ischemia.

Recently, we reported (11) a clinical trial comparing the efficacy of emergency angioplasty versus intracoronary streptokinase in preserving left ventricular function in the setting of acute myocardial infarction. The trial was conducted at the University of Michigan Medical Center, Ann Arbor, and the William Beaumont Hospital, Royal Oak, Michigan from April 1, 1984 to October 31, 1984. To evaluate the efficacy of emergency angioplasty in preventing subsequent exercise-induced periinfarct ischemia, predischarge submaximal exercise thallium-201 tomography was performed in all the patients treated at the University Hospital. The purpose of this study was to compare the incidence of exercise-induced periinfarct ischemia using predischarge submaximal exercise thallium-201 single photon emission computed tomography for the two treatment groups.

Methods

Patient selection. Patients admitted to the University of Michigan Hospitals from April 1, 1984 to October 31, 1984 with suspected acute myocardial infarction who met the following inclusion criteria were eligible for randomization

From the Divisions of Cardiology and Nuclear Medicine, Department of Internal Medicine, University of Michigan Medical Center, Ann Arbor, Michigan.

Manuscript received November 5, 1985; revised manuscript received February 4, 1986, accepted April 8, 1986.

Address for reprints: William W. O'Neill, MD, Room F-245, Cardiology Division, University of Michigan Hospitals, 1500 East Medical Center Drive, Ann Arbor, Michigan 48109.

0735-1097/86/$3.50

to treatment with either intracoronary streptokinase or emergency angioplasty. The inclusion criteria were: 1) characteristic chest pain of more than 30 minutes' duration, unrelieved by nitroglycerin, and arrival at our hospital within 12 hours of symptom onset; 2) electrocardiogram demonstrating 2 mm or more ST segment elevation or new pathologic Q wave, or both, in at least two leads; and 3) informed consent. The exclusion criteria were 1) age over 75 years; 2) contraindication to thrombolytic or anticoagulant therapy; 3) cardiogenic shock, defined as systolic blood pressure of less than 90 mm Hg with left ventricular end-diastolic pressure of more than 15 mm Hg; 4) previous coronary bypass grafting; and 5) angiographic exclusion criteria as defined later. Twenty-eight patients fulfilled the selection criteria (Table 1).

The nature, potential benefits and possible risks of the study were explained to the patients, who gave written informed consent before angiography. The treatment protocol was approved by the Human Ethics Committee of our hospital.

Table 1. Summary of Clinical and Angiographic Data of 28 Patients on Study Entry*

	PTCA	SK
Number of patients	14	14
Age (yr)	55 ± 11	55 ± 8
Sex		
Male	9	12
Female	5	2
Previous MI	1	1
Killip class		
I	11	10
II	2	3
III	1	1
AMI location		
Ant	4	9
Inf	10	5
MI-CA		
LAD	4	9
LCx	2	1
RCA	8	4
Severity of MI-CA lesion		
Total occlusion	14	12
Subtotal occlusion	0	2
Extent of CAD		
One vessel	8	9
Two vessel	1	4
Three vessel	5	1
Peak creatine kinase (IU/liter)	3,415 ± 2,544	3,998 ± 2,333†

*There was no significant difference between the angioplasty and streptokinase groups for any of the variables studied.

†n = 13, one patient died early; peak enzyme level not available. AMI = acute myocardial infarction; Ant = anterior; CAD = coronary artery disease; Inf = inferior; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; MI = myocardial infarction; MI-CA = infarct-related coronary artery; PTCA = percutaneous transluminal coronary angioplasty; RCA = right coronary artery; SK = intracoronary streptokinase.

Cardiac catheterization protocol. Potential candidates for intervention were brought to the cardiac catheterization laboratory as soon as possible. After the placement of vascular sheaths, heparin, 5,000 U, was administered intravenously. Coronary angiography was performed using the Judkins technique with preformed catheters, beginning with opacification of the presumed noninvolved coronary artery. This was followed by left ventriculography. The angiographic exclusion criteria for randomization were: 1) 60% or greater left main coronary artery stenosis; 2) a "left main equivalent" lesion, defined as 70% or greater stenosis of both the proximal left anterior descending artery and proximal circumflex artery; and 3) a tortuous distal circumflex artery occlusion judged to be unsuitable for angioplasty. Patients who fulfilled all the selection criteria were then randomized to receive either emergency angioplasty or intracoronary streptokinase. Blind randomization was accomplished using a closed envelope system. Crossover was not allowed.

Emergency coronary angioplasty protocol. At the initiation of angioplasty, low molecular weight dextran, 10% in 500 ml dextrose solution, was infused intravenously. An additional 5,000 U heparin was given intravenously. Intravenous verapamil, 5 mg, and intracoronary nitroglycerin, 250 μg, were given. Angioplasty of the infarct-related coronary artery was performed using a steerable guide wire system with Grüntzig angioplasty catheters. Successful perforation of thrombus with the guide wire was followed by serial balloon inflations to decrease the translesional pressure gradient to less than 20 mm Hg. Repeat selective coronary angiography was then performed. After the intervention procedure, the 14 patients were taken to the coronary care unit for further medical care. Nifedipine, 30 mg, aspirin, 325 mg, and dipyridamole, 75 mg, orally every 8 hours were given. Heparin was infused intravenously to maintain activated partial thromboplastin time at 2 to 2½ times control value until repeat angiography was performed 7 to 10 days later.

Intracoronary streptokinase therapy protocol. The 14 patients randomized to receive intracoronary streptokinase were given the same dose of intravenous verapamil and intracoronary nitroglycerin as in the angioplasty protocol. Additional heparin and low molecular weight dextran were not administered. Streptokinase infusion of 4,000 U/min was given by ostial infusion. Infusion was continued for 30 minutes after arterial recanalization had occurred or until a total dose of 300,000 U was administered. Repeat opacification of the infarct-related coronary artery was performed every 15 minutes to assess for recanalization. After streptokinase therapy, patients were managed in the coronary care unit and received the same course of nifedipine, aspirin, dipyridamole and heparin as in the angioplasty protocol.

Repeat angiography. Patients who survived and remained in stable condition underwent repeat angiography 7

to 10 days after admission. Standard selective coronary angiography was performed. Each stenosis was examined in multiple projections, including cranial and caudal angulations. The projection in which the stenosis appeared most severe was selected for analysis. Caliper determination of percent diameter reduction was obtained. The same method of measurement was used for the analysis of angiograms obtained before and after initial therapy was administered. Lesions of large diagonal or marginal branches were considered to be lesions of the left anterior descending coronary artery system or the circumflex coronary artery system, respectively.

Submaximal exercise test with thallium-201 single photon emission computed tomography. A predischarge submaximal exercise test with thallium tomography was performed 7 to 20 days after admission. The test was performed with a motor-driven treadmill 2 or more hours postprandially, and medications were not discontinued. Twelve lead electrocardiograms were recorded at rest and at the end of each stage. Treadmill exercise was conducted in 3 minute uninterrupted stages using a constant speed of 2 miles per hour and a 3% grade increment for each stage. Thallium-201 chloride (3 mCi) was injected at peak exercise, and patients were instructed to continue exercise for an additional minute. Imaging was then performed immediately and 3 hours later. Each patient exercised to a target heart rate of 130 beats/min or until angina, marked ST segment depression, serious ventricular arrhythmia, hypotension or exhaustion occurred. A test was judged positive when the patient either experienced chest pain compatible with angina pectoris or developed diagnostic ischemic electrocardiographic changes ($\geq$1 mm horizontal or downsloping or $\geq$1.5 mm upsloping ST depression).

Imaging was performed using a wide field of view gamma camera (GE 400AT), with a general all-purpose, parallel hole collimator. Imaging data were collected over 180° beginning in the right anterior oblique view and ending in the left posterior oblique view. Sixty-four discrete view images, that is, one image every 2.8°, were acquired. The image format was 64 × 64 with 30,000 to 50,000 counts obtained per image. Each image was acquired for 20 seconds for a total acquisition time of less than 22 minutes. Transaxial tomograms were reconstructed by the filtered back projection method with a ramp-Hanning filter. No attenuation correction was used. Each transaxial tomogram was displayed perpendicular to the long axis of the left ventricle. Each reconstructed slice was 6.25 mm thick. Frontal and sagittal tomograms were also reconstructed from a series of transaxial tomograms, corresponding to transverse and longitudinal sections of the cardiac axis. Postexercise images were compared with 3 hour delayed images.

Two independent experienced observers interpreted the images without knowledge of the patient's coronary anatomy or the mode of therapy. An image defect was considered present if there was a discrete region of absent or decreased activity estimated visually. The defect was anatomically localized as apical, anterior, septal, inferior or lateral, using all views. A defect present immediately after exercise and again at 3 hours delay was considered to indicate a scar, whereas a defect present only immediately after exercise was considered to reflect ischemia. Either partial or complete resolution of a defect was considered evidence of reversible ischemia. If the reversible defect occurred adjacent to the zone of infarction, it was considered to represent periinfarct ischemia; if it occurred at a site removed from the zone of infarction, it was considered to represent distant ischemia.

Statistical evaluation. Averaged data are presented as mean ± SD. The Student t, the Pearson's chi-square and the Fisher exact tests were used for statistical analysis where appropriate. Differences were considered significant at the $p < 0.05$ level.

Results

Clinical data (Tables 1 and 2). Twenty-one male and seven female patients fulfilled the study selection criteria. These patients were randomized so that 14 received intracoronary streptokinase and 14 were treated with emergency coronary angioplasty. The clinical and angiographic data on study entry are summarized in Table 1. The clinical characteristics for each individual patient studied are presented in Table 2.

In two patients in the angioplasty group (Patients 1 and 5) and two patients in the streptokinase group (Patients 17 and 24) reperfusion was not achieved despite interventions. During hospitalization, one patient (Patient 16) died 4 hours after receiving intracoronary streptokinase. She developed severe precordial chest pain in the coronary care unit, and immediate repeat angiography demonstrated reocclusion of the left anterior descending coronary artery. She died in the catheterization laboratory with cardiogenic shock. Two patients (Patients 18 and 25, both in the streptokinase group) had emergency coronary bypass graft surgery performed because of early postinfarct unstable angina. One patient in the angioplasty group (Patient 3) had silent reocclusion documented on predischarge angiography.

Twenty-one patients (11 angioplasty, 10 streptokinase) consented and completed both repeat angiography and predischarge exercise thallium tomographic studies. Three patients in the angioplasty group (Patients 1, 7 and 14), and one patient in the streptokinase group (Patient 21) refused further investigative procedures and only partial data were available.

Intervention and recatheterization data (Fig. 1, Table 3). The time from symptom onset to successful reperfusion was 286.7 ± 76.9 minutes for the angioplasty group and 345.0 ± 92.5 minutes for the streptokinase group (p =

Table 2. Clinical Characteristics of 28 Patients

Case	Age (yr) & Sex	Prior MI	Killip Class	AMI Location	MI-CA	Extent of CAD	Time to Reperfusion (min)
			A. Angioplasty Group				
1	62M	0	1	Inf	RCA	1V	U
2	62M	0	1	Inf	RCA	3V	130
3	48M	0	1	Inf	RCA	1V	285
4	70F	0	1	Inf	RCA	1V	240
5	40M	+	1	Inf	RCA	1V	U
6	55M	0	1	Inf	LCx	3V	345
7	34F	0	2	Ant	LAD	2V	315
8	55M	0	1	Ant	LAD	3V	330
9	62F	0	2	Inf	RCA	1V	21
10	55M	0	3	Ant	LAD	1V	315
11	64M	0	1	Inf	RCA	3V	390
12	56F	0	1	Inf	LCx	1V	390
13	37M	0	1	Ant	LAD	1V	225
14	68F	0	1	Inf	RCA	3V	265
			B. Streptokinase Group				
15	59M	0	2	Ant	LAD	1V	195
16	58F	0	1	Ant	LAD	1V	270
17	52M	0	1	Ant	LAD	1V	U
18	61M	+	1	Ant	LAD	3V	450
19	52M	0	1	Inf	RCA	1V	240
20	39M	0	1	Inf	RCA	1V	450
21	58M	0	1	Inf	LCx	2V	330
22	41M	0	1	Inf	RCA	1V	480
23	48M	0	1	Inf	RCA	1V	270
24	62M	0	1	Ant	LAD	1V	U
25	66F	0	1	Ant	LAD	2V	330
26	61M	0	2	Ant	LAD	2V	315
27	55M	0	2	Ant	LAD	2V	390
28	50M	0	3	Ant	LAD	1V	420

F = female; M = male; U = unsuccessful reperfusion; V = vessel; + = present; 0 = absent; other abbreviations as in Table 1.

NS). Initial percent stenosis of the infarct-related coronary artery was 100% for the angioplasty group and 98.2 ± 5.4% for the streptokinase group (p = NS). Immediately after intervention, residual stenosis of the infarct-related coronary artery was 46.4 ± 26.8% for the angioplasty group and 83.6 ± 15.9% for the streptokinase group ($p < 0.01$). At recatheterization 10 days later, the residual stenosis of the infarct-related coronary artery of the angioplasty group was significantly less than that of the streptokinase group (43.8 ± 31.4 versus 75.0 ± 15.6%, $p < 0.05$). The angioplasty group had a significant increase of global left ventricular ejection fraction (from 49.6 ± 11.2 to 57.5 ± 13.5%, $p < 0.01$), but the streptokinase group did not (from 45.5 ± 10.7 to 46.9 ± 12.9%, p = NS).

Submaximal exercise thallium-201 tomographic studies (Fig. 2, Table 4). Exercise duration and pressure-rate product achieved were similar for both groups. One (9%) of the 11 patients (Patient 12) treated with emergency coronary angioplasty developed exercise-induced periinfarct ischemia compared with 6 (60%) of the 10 patients treated

Figure 1. Severity of stenosis of infarct-related coronary artery before intervention, immediately after intervention and at recatheterization 10 days after intervention. NS = not significant; PTCA = percutaneous transluminal coronary angioplasty; STK = intracoronary streptokinase.

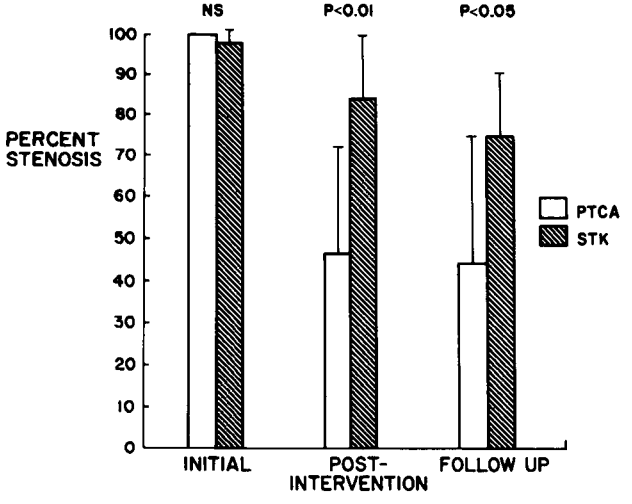


Table 3. Catheterization and Follow-Up Data in 28 Patients

Case	Initial MI-CA % Stenosis	Postintervention % Stenosis	Follow-Up MI-CA % Stenosis	Initial EF %	Follow-Up EF %	Δ EF %	Collaterals	Intervention Related Complications
				A. Angioplasty Group				
1	100	100	R	56	R	NA	0	
2	100	40	40	36	55	+19	0	
3	100	40	100	53	48	−5	0	Reperfusion VT
4	100	30	30	62	80	+18	0	AV fistula
5	100	100	100	57	55	−2	+	
6	100	20	10	54	72	+18	0	
7	100	40	R	52	R	NA	+	Hematoma; transfused
8	100	60	45	35	45	+10	0	GI bleeding; transfused
9	100	20	10	69	75	+6	0	Reperfusion VF
10	100	40	50	36	35	−1	+	
11	100	60	60	NA	29	NA	+	Reperfusion VF
12	100	50	50	54	56	+2	+	
13	100	40	20	46	58	+12	0	VF; catheter in LV
14	100	10	10	44	53	+9	+	
				B. Streptokinase Group				
15	100	90	90	24	25	+1	0	
16	100	90	NA	55	NA	NA	0	Death after 4 hours
17	95	100	50	34	33	−1	0	
18	100	95	NA	22	NA	NA	+	Angina, CABG
19	100	80	80	47	49	+2	+	
20	100	60	70	58	47	−11	+	
21	100	95	R	47	R	NA	0	Hematoma; transfused
22	80	60	60	53	64	+11	0	
23	100	50	50	49	51	+2	0	
24	100	100	80	56	51	−5	0	
25	100	90	95	55	69	+14	0	Angina, IABP, CABG
26	100	90	90	48	51	+3	0	Reperfusion VF
27	100	80	80	39	38	−1	+	
28	100	90	70	37	38	+1	0	

AV = arteriovenous; CABG = coronary artery bypass graft; EF = ejection fraction; GI = gastrointestinal; IABP = intraaortic balloon pump; LV = left ventricle; NA = not available; R = refused to participate; VF = ventricular fibrillation; VT = ventricular tachycardia; Δ = difference; other abbreviations as in Tables 1 and 2.

with intracoronary streptokinase ($p < 0.05$). In addition, all of the seven patients who developed exercise-induced periinfarct ischemia had 50% or more residual stenosis of the infarct-related vessel (Fig. 3).

Discussion

Limitations of thrombolytic therapy. Several recent studies (12–14) have provided evidence that early reperfusion by thrombolytic therapy may improve survival after acute myocardial infarction. However, as emphasized by Laffel and Braunwald (15), "thrombolytic therapy can at best only reestablish antegrade flow in the infarct-related coronary artery. It cannot be expected to reverse factors responsible for initiation of the thrombus, such as advanced atherosclerotic plaques, intimal rupture, enhanced platelet adhesiveness or coronary spasm." Indeed, it is our experience (11) as well as that of others (16) that after intracoronary streptokinase therapy, residual stenosis of the infarct-related coronary artery is usually significant and frequently high grade. These patients are undoubtedly exposed to the risk of rethrombosis and probably further myocardial necrosis. The incidence of in-hospital reocclusion has been reported to be between 15 and 35% (16–18).

Harrison et al. (19) noted that streptokinase-treated patients with residual luminal cross-sectional area less than 0.4 mm^2 had an incidence of rethrombosis of 54%. In contrast, patients with less severe residual stenosis did not develop rethrombosis. Gold et al. (20) found that recurrent in-hospital ischemic events were recorded in 76% of patients with high grade stenotic lesions of 90% or greater after streptokinase infusion, whereas ischemic complications in patients with less severe lesions were substantially less frequent.

Although many of the patients successfully treated with streptokinase do not develop reocclusion, it is conceivable

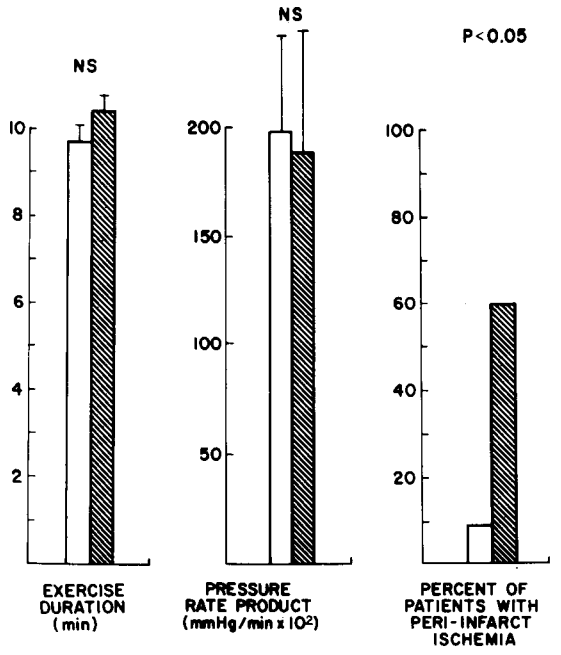


Figure 2. Results of predischarge submaximal exercise thallium-201 tomography. **Open columns**, percutaneous transluminal coronary angioplasty; **hatched columns**, intracoronary streptokinase. NS = not significant.

that a considerable portion of them may develop periinfarct ischemia during stress. This is illustrated by the results of this study. Of the 10 patients treated with intracoronary streptokinase alone, 6 (60%) had exercise-induced periinfarct ischemia and the degree of residual stenosis was 75.0 ± 15.6%. Because thrombolytic therapy cannot be expected to alter the underlying atheromatous lesions and because it is associated with a significant incidence of subsequent exercise-induced periinfarct ischemia, an alternative reperfusion strategy may be necessary to reduce this vulnerability. In this study, we are able to show that patients treated with emergency coronary angioplasty had a substantially lower incidence of exercise-induced periinfarct ischemia than did patients treated with intracoronary streptokinase. Only 1 (9%) of the 11 patients in the angioplasty group had exercise-induced periinfarct reversible defects on thallium tomography. This observable difference may be explained by the finding that residual stenosis of the infarct artery was considerably less severe in the angioplasty group, being only 43.8 ± 31.4%.

Residual stenosis and exercise-induced periinfarct ischemia. Our data show that neither of the two patients with failed reperfusion or reocclusion had exercise-induced periinfarct ischemia on thallium tomography (Fig. 3). It is quite possible that in these patients, the infarct progressed to completion, thus eliminating the development of subsequent periinfarct ischemia. Similarly, all six patients with less than 50% residual stenosis of the infarct-related artery did not develop such scintigraphic patterns, albeit for presumably obvious and different reasons. It is not purely coincidental that all patients in the latter category were successfully treated by coronary angioplasty.

In contrast, 7 (54%) of the remaining 13 patients with 50% or more residual stenosis had exercise-induced reversible periinfarct defects on thallium scintigraphy. Some or all of the following possibilities may be postulated to explain the lack of thallium defects during exercise in some patients despite the presence of significant angiographic residual stenosis. First, periinfarct ischemia may actually be present but not detected by thallium tomography. This may be because the ischemic regions are too small or the method of detection is insufficiently sensitive, or both. Second, exercise-induced periinfarct ischemic events may in fact be absent. This may be because of either inadequate exercise stress relative to the coronary lesion, the presence of extensive collateral blood flow or the failure of interventional therapy to salvage myocardium. Regardless of the explanation, the majority of the exercise-induced periinfarct ischemic events were observed in the streptokinase group.

Infarct location and time to reperfusion. Although statistically insignificant, two baseline clinical characteristics appear unevenly in the treated groups, and therefore deserve further comment. First, a larger proportion of patients in the angioplasty group had inferior myocardial infarction. One (11%) of the nine tested patients in the angioplasty group with inferior infarction developed exercise-induced periinfarct ischemia whereas four (80%) of the five patients in the streptokinase group had a similar scintigraphic pattern ($p < 0.01$). In this subgroup, the statistical significance of the difference between the treated groups is demonstrated. In contrast, among tested patients with anterior infarction, none of the three patients in the angioplasty group and two (33%) of the six patients in the streptokinase group had exercise-induced periinfarct ischemia (p = NS). It may appear that patients with anterior myocardial infarction are less likely to develop exercise-induced periinfarct ischemia. However, as mentioned earlier, development of serious in-hospital ischemic events precluded subsequent thallium tomographic evaluation in three patients with anterior infarction who were treated with streptokinase. Obviously, these three patients developed ischemia even without exercise provocation. Thus, infarct location probably was not an important factor in the subsequent development of ischemia.

The second uneven baseline variable is the longer time interval to reperfusion in the streptokinase-treated group of patients (Fig. 4). The longer delay could theoretically result in a bias in the incidence of subsequent exercise-induced ischemic events against this treatment group. However, the occurrence of these ischemic events is quite evenly distributed and appears to be independent of the time intervals to reperfusion in the streptokinase-treated patients. All 10 tested patients in the angioplasty group had a time interval to

Table 4. Submaximal Exercise Test and Thallium Scintigraphy Data in 28 Patients

Case	Exercise Duration	PRP	Chest Pain	ECG STΔ	Scar	Periinfarct Ischemia	Distant Ischemia
			A. Angioplasty Group				
1	8 min	110	0	0	+	0	0
2	7 min	171	+	+	+	0	+
3	6 min	149	0	0	+	0	0
4	6 min	196	0	0	+	0	0
5	11 min	224	0	0	+	0	0
6	12 min	217	0	0	+	0	0
7			Refused				
8	9 min	138	0	+	+	0	0
9	8 min	199	0	0	+	0	0
10	21 min	218	0	0	+	0	0
11	8 min	177	0	0	+	0	0
12	6 min 30 seconds	312	+	+	+	+	0
13	12 min 30 seconds	272	0	0	+	0	0
14			Refused				
			B. Streptokinase Group				
15	6 min	73	0	0	+	+	0
16			Died				
17	14 min 23 seconds	224	0	+	+	0	0
18			Angina, CABG				
19	14 min	190	0	0	+	+	0
20	6 min	90	0	0	+	+	0
21	9 min	224	0	0	+	0	0
22	12 min	290	0	0	+	+	0
23	16 min	198	0	0	+	+	0
24	15 min	221	0	0	+	0	0
25			Angina, IABP, CABG				
26	9 min	247	0	0	+	0	0
27	6 min 50 seconds	200	0	0	+	0	0
28	6 min	122	0	0	+	+	0

ECG = electrocardiographic; PRP = pressure-rate product (mm Hg/min × 10^2); other abbreviations as in Tables 2 and 3.

Figure 3. Relation between residual stenosis of infarct-related coronary artery at recatheterization and exercise-induced periinfarct ischemia shown on thallium tomography in 11 patients who underwent angioplasty (PTCA) and 10 patients who received streptokinase (STK).

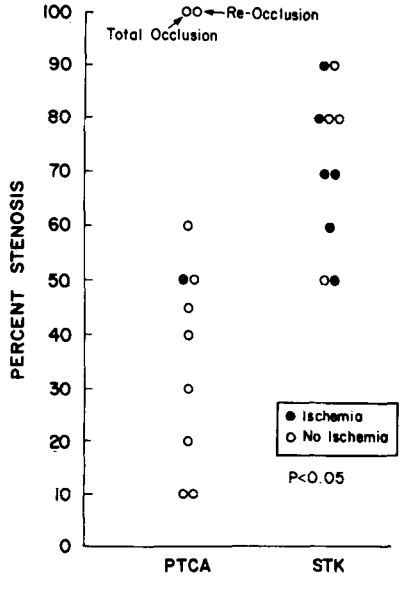
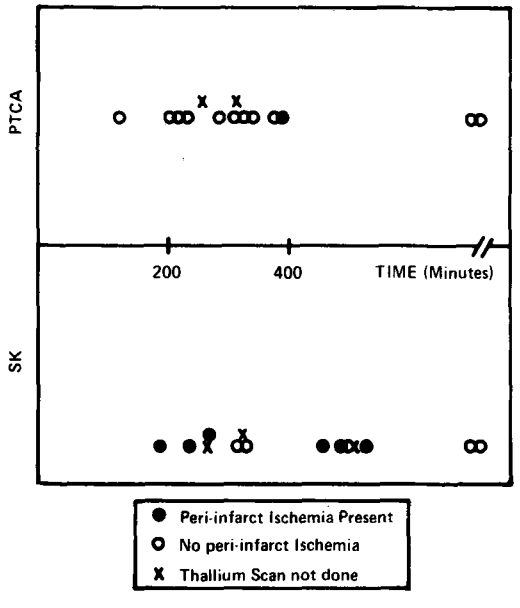


Figure 4. The time from symptom onset to reperfusion is plotted against the occurrence of exercise-induced periinfarct ischemia in 28 patients. PTCA = percutaneous transluminal coronary angioplasty; SK = intracoronary streptokinase.



reperfusion shorter than 400 minutes and only 1 developed subsequent exercise-induced periinfarct ischemia. In contrast, three of the five tested patients in the streptokinase group had a similar scintigraphic pattern when the time to reperfusion was shorter than 400 minutes ($p < 0.05$). Thus, it may be concluded that the difference in delay in achieving reperfusion in the two treatment groups probably did not significantly influence the outcome of our conclusions.

Clinical implications. A delayed redistribution pattern on thallium scintigraphy signifies, in addition to reversible ischemia, the presence of viable myocardial tissue in the corresponding region (21,22). Gibson et al. (23) showed that such a scintigraphic pattern seen in patients after acute myocardial infarction is strongly predictive of subsequent clinical cardiac events. In this study, we have shown that a significant proportion of patients have residual viable myocardium at risk after successful acute interventional therapy. This is most likely related to the degree of residual stenosis of the involved coronary artery and is considerably more prevalent in the patients treated with intracoronary streptokinase. These findings suggest that after acute myocardial infarction, patients treated with intracoronary streptokinase may be at greater risk than those treated with emergency coronary angioplasty. Reperfusion by emergency coronary angioplasty would therefore appear to be superior to intracoronary streptokinase in this respect. Whether angioplasty alone or in combination with thrombolytic therapy is superior in salvaging myocardium and improving long-term survival, must await future studies.

We gratefully acknowledge the statistical assistance of Carl Dmuchowski and the secretarial help of Vi Rhodes in preparing this manuscript.

References

1. Anderson JL, Marshall HW, Bray BE, et al. A randomized trial of intracoronary streptokinase in the treatment of acute myocardial infarction. N Engl J Med 1983;308:1312–8.
2. Sobel BE, Geltman EM, Tiefenbrunn AJ, et al. Improvement of regional myocardial metabolism after coronary thrombolysis induced with tissue-type plasminogen activator or streptokinase. Circulation 1984;69:983–90.
3. DeWood MA, Heit J, Spores J, et al. Anterior transmural myocardial infarction: effects of surgical coronary reperfusion on global and regional left ventricular function. J Am Coll Cardiol 1983;1:1223–34.
4. Rentrop KP, Blanke H, Karsch KR, Kaiser H, Kostering H, Leitz K. Selective intracoronary thrombolysis in acute myocardial infarction and unstable angina pectoris. Circulation 1981;63:307–17.
5. Ganz W, Buckbinder N, Marcus H, et al. Intracoronary thrombolysis in evolving myocardial infarction. Am Heart J 1981;101:4–13.
6. Kennedy JW, Ritchie JL, Davis KB, Fritz JK. The Western Washington randomized trial of intracoronary streptokinase in acute myocardial infarction trial. N Engl J Med 1983;309:1477–82.
7. Khaja F, Walton JA, Brymer JF, et al. Intracoronary fibrinolytic therapy in acute myocardial infarction—report of a prospective randomized trial. N Engl J Med 1983;308:1306–11.
8. Meyer J, Merx W, Schmitz H, et al. Percutaneous transluminal coronary angioplasty immediately after intracoronary streptolysis of transmural myocardial infarction. Circulation 1982;66:905–13.
9. Hartzler GO, Rutherford BD, McConahay DR. Percutaneous transluminal coronary angioplasty: application for acute myocardial infarction. Am J Cardiol 1984;53:117C–21C.
10. Kennedy JW, Stewart DK. Interventional coronary arteriography. Annu Rev Med 1984;35:513–34.
11. O'Neill WW, Lai P, Vellappillil G, et al. Preliminary report of a randomized, prospective clinical trial of intracoronary streptokinase versus coronary angioplasty therapy of acute myocardial infarction (abstr). J Am Coll Cardiol 1985;5:494
12. Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI). Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. Lancet 1986;1:397–401.
13. Simoons ML, v/d Brand M, de Zwaan C, et al. Improved survival after early thrombolysis in acute myocardial infarction: a randomised trial by the Interuniversity Cardiology Institute in Netherlands. Lancet 1985;2:578–82.
14. Kennedy JW, Ritchie JL, Davis KB, Stadius ML, Maynard C, Fritz JK. The Western Washington randomized trial of intracoronary streptokinase in acute myocardial infarction: a 12-month follow-up report. N Engl J Med 1985;312:1073–8.
15. Laffel GL, Braunwald E. Thrombolytic therapy: a new strategy for the treatment of acute myocardial infarction (second of two parts). N Engl J Med 1984;311:770–6.
16. Dodge HT, Sheehan FH, Mathey DG, Brown BG, Kennedy JW. Usefulness of coronary artery bypass graft surgery or percutaneous transluminal angioplasty after thrombolytic therapy. Circulation 1985;72(suppl V):V-39–45.
17. Gold HK, Leinbach RC, Palacios IF, et al. Coronary reocclusion after selective administration of streptokinase. Circulation 1983;68(suppl I):I-50–4.
18. Lee G, Low RI, Takeda P, et al. Importance of follow-up medical and surgical approaches to prevent reinfarction, reocclusion, and recurrent angina following intracoronary thrombolysis with streptokinase in acute myocardial infarction. Am Heart J 1982;104:921–4.
19. Harrison DG, Ferguson DW, Collins SM, et al. Rethrombosis after reperfusion with streptokinase: importance of geometry of residual lesions. Circulation 1984;69:991–9.
20. Gold HK, Cowley MJ, Palacios IF, et al. Combined intracoronary streptokinase infusion and coronary angioplasty during acute myocardial infarction. Am J Cardiol 1984;53:122C–5C.
21. Rozanski A, Berman DS, Gray R, et al. Use of thallium-201 redistribution scintigraphy in the preoperative differentiation of reversible and nonreversible myocardial asynergy. Circulation 1981;64:936–44.
22. Gibson RS, Watson DD, Taylor GJ, et al. Prospective assessment of regional myocardial perfusion before and after coronary revascularization surgery by quantitative thallium-201 scintigraphy. J Am Coll Cardiol 1983;1:804–15.
23. Gibson RS, Watson DD, Craddock GB, et al. Prediction of cardiac events after uncomplicated myocardial infarction: a prospective study comparing pre-discharge exercise thallium-201 scintigraphy and coronary angiography. Circulation 1983;68:321–36.