IMMEDIATE ANGIOPLASTY COMPARED WITH THE ADMINISTRATION OF A THROMBOLYTIC AGENT FOLLOWED BY CONSERVATIVE TREATMENT FOR MYOCARDIAL INFARCTION

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Abstract *Background.* Immediate angioplasty and the administration of a thrombolytic agent followed by conservative treatment are two approaches to the management of acute myocardial infarction, but these methods have not been compared prospectively.

Methods. We enrolled 108 patients with acute myocardial infarction in a randomized trial designed to test the hypothesis that immediate angioplasty (without previous thrombolytic therapy) may result in greater myocardial salvage than the administration of a thrombolytic agent followed by conservative treatment. The primary end point was the change in the size of the perfusion defect as assessed at admission and discharge by tomographic imaging with technetium-99m sestamibi, a myocardial perfusion agent that can measure myocardium at risk and final infarct size.

Results. End-point data were available for 56 patients randomly assigned to receive tissue plasminogen activator (mean [±SD] time to start of infusion, 232±174 minutes after the onset of chest pain) and 47 patients randomly assigned to receive angioplasty (first balloon inflation at 277±144 minutes). In the case of anterior infarction, myocardial salvage as assessed by imaging with technetium-99m sestamibi was 27±21 percent of the left ventricle for 22 patients in the thrombolysis group, as compared with 31±21 percent for 15 patients in the angioplasty group. For infarcts in all other locations, myocardial salvage was 7±13 percent for 34 patients in the thrombolysis group and 5±10 percent for 32 patients in the angioplasty group. After adjustment for infarct location, the difference in mean salvage between groups was 0 ($P = 0.98$), with a 95 percent confidence interval of ±6 percent of the left ventricle.

Conclusions. In patients with acute myocardial infarction, immediate angioplasty does not appear to result in greater myocardial salvage than the administration of a thrombolytic agent followed by conservative treatment, although a small difference between these two therapeutic approaches cannot be excluded. (N Engl J Med 1993; 328:685-91.)

ALTHOUGH early intravenous thrombolytic therapy reduces mortality and infarct size in patients with acute myocardial infarctions,[1-6] it has several potential limitations. Many patients have contraindications to thrombolytic therapy,[7] and in approximately 25 percent of those so treated reperfusion is not achieved in the short term by thrombolytic therapy alone.[8] Also, even after successful thrombolysis, most patients are left with a high-grade stenosis that may limit flow, impair subsequent myocardial recovery,[9] and increase the risk of reinfarction.

Multiple nonrandomized studies[10-12] have suggested that immediate angioplasty can result in a high rate of reperfusion that is associated with a low rate of in-hospital mortality and an increase in the ejection fraction. The single published randomized trial of immediate angioplasty[13] found that it compared favorably with the use of intracoronary streptokinase.

Studies in laboratory animals have demonstrated that for a given coronary occlusion, the angiographic area at risk is highly variable.[14-16] Technetium-99m sestamibi, a new myocardial perfusion agent, has properties that make it ideal for the assessment of myocardium at risk.[17] The ability of this agent to provide accurate measurements of myocardium at risk and final infarct size has been demonstrated in animal models of permanent coronary occlusion and reperfusion.[18,19] The change in the size of the perfusion defect from the image obtained at the time of hospital admission and that obtained at discharge, which is presumably a measure of myocardial salvage, is substantial in patients treated with either intravenous thrombolysis or immediate angioplasty.[20-22]

This trial was designed to compare the efficacy of immediate angioplasty with that of thrombolysis (using tissue plasminogen activator) followed by conservative treatment as a treatment strategy for acute myocardial infarction. We tested the hypothesis that myocardial salvage may be greater with immediate angioplasty than with thrombolysis, as assessed by serial tomographic imaging with technetium-99m sestamibi.

METHODS

Study Group

The study group consisted of 108 patients enrolled between April 1, 1989, and June 30, 1991. These patients represented more than 90 percent of those who were less than 80 years of age; who had severe chest pain lasting for at least 30 minutes and up to 12 hours that was thought to be myocardial in origin; and who met one of the following two criteria: (1) new or presumably new electrocardiographic ST-segment elevation of at least 0.1 mV measured 0.08 second after the J point in at least two of the three inferior leads (II, III, and aVF), in at least two adjacent precordial leads (V_1 to V_6), or in leads I and aVL; or (2) new or presumably new ST depression of at least 0.2 mV measured 0.08 second after the J point in at least two precordial leads from V_1 to V_3. Patients with cardiogenic shock or contraindications to thrombolytic therapy were excluded.

Randomization

Eligible patients were identified promptly on arrival in the emergency room. Once they had given informed consent, they were randomly assigned either to initial therapy with tissue plasminogen

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activator or to immediate angioplasty with a computer-generated randomization schedule that was stratified according to time since the start of chest pain (<4 vs. ≥4 hours) and myocardial site of infarction (anterior vs. all others).

Intravenous Thrombolytic Therapy

Double-chain tissue plasminogen activator (duteplase) was administered as soon as possible in the patients randomly assigned to thrombolysis. The total dose was 0.6 million units per kilogram of body weight over a four-hour period, as described elsewhere.[23]

Heparin therapy was begun immediately with an intravenous bolus of 5000 units. An intravenous infusion was then begun and was adjusted to maintain the partial-thromboplastin time at 2.0 to 2.5 times the control value for the next five days. Each patient subsequently received 12,500 units of heparin subcutaneously every 12 hours for the remainder of the hospitalization.

Immediate Angioplasty

The patients randomly assigned to angioplasty received an intravenous bolus of 5000 units of heparin and were taken to the cardiac catheterization laboratory as soon as possible. After coronary angiography, they received an additional 10,000 units of intravenous heparin before angioplasty of the infarct-related occlusion. Angioplasty was attempted whenever there was occlusion or subtotal occlusion of the infarct-related artery. An intravenous infusion of heparin was maintained for five days as previously described. Successful angioplasty was defined as the restoration of normal flow with less than 50 percent residual stenosis.

Concurrent Therapy

All the patients received 162.5 mg of chewable aspirin in the emergency room and 162.5 mg of aspirin orally each day for the remainder of the hospitalization.

In patients without contraindications, β-blocker agents were administered as soon as possible, as described elsewhere.[24] Standard care for acute myocardial infarction was provided as necessary, but the routine use of calcium-channel blockers was avoided.

Recurrent Ischemia

Recurrent ischemic symptoms that occurred despite pharmacologic therapy and were considered severe or were associated with definite electrocardiographic changes were taken as evidence of clinical instability. Emergency coronary angiography and revascularization by either angioplasty or bypass surgery were performed in patients with such symptoms at the discretion of the staff cardiologist.

Radionuclide Studies

After informed consent was obtained, 20 to 30 mCi of technetium-99m sestamibi was injected before reperfusion therapy. Like thallium-201, technetium-99m sestamibi is taken up by normal myocardium in direct proportion to blood flow.[25,26] Unlike thallium-201, sestamibi has a very slow washout from the myocardium, with minimal redistribution.[25,27] Imaging could therefore be delayed for up to eight hours (mean, six) and still provide information about myocardial perfusion at the time of administration. Imaging was performed with single-photon-emission computed tomography when the patient was clinically stable.[21,22] A repeat injection of sestamibi and similar tomographic imaging were performed 6 to 14 days later, before discharge.

Equilibrium radionuclide angiography was performed with the patient at rest one day after the second sestamibi imaging and approximately six weeks after discharge, as described elsewhere.[28]

Analysis of Imaging Data

All sestamibi images were reconstructed with standard back-projection algorithms and a Ramp–Hanning filter.[21,29] Count profiles were generated for five representative short-axis slices by identifying the highest counts in every 6-degree sector around the circumference of the left ventricle and normalizing this value in relation to the peak counts in the profile. The apical and basal slices were chosen according to predetermined rules.[21,29] The three remaining short-axis slices were spaced equally between the apical and basal slices.

The perfusion defect was quantified from these five slices by previously published methods,[21,29] which included measuring the radius of each slice in a geometric model of the left ventricle. The defect was identified with a threshold value of 60 percent of peak counts. The validity of this approach to the measurement of myocardium at risk and final infarct size has been demonstrated in phantoms[29] and in animal models of permanent occlusion[18] and reperfusion.[30]

Direct and Indirect Costs

Hospital and professional charges related to cardiac care during the initial and all subsequent hospitalizations up to six months after presentation were recorded. Costs were calculated as 80 percent of charges. Indirect measures of cost included the total number of days in the hospital and the coronary care unit, readmission to the hospital within six months, and return to work. Return to work was expressed as the proportion of patients returning to employment who had been employed before their myocardial infarction. A cost–effectiveness analysis was performed by determining the ratio of the mean cumulative six-month cost to the mean myocardial salvage for each treatment group.

The adjustment of costs included the addition of a charge for 100 mg of commercially available tissue plasminogen activator in the thrombolysis group, because the study drug was supplied free of charge by the manufacturer. Charges for the sestamibi studies were excluded from the analysis, since these studies were primarily used for the determination of end-point data rather than for patient care. Because of the short duration of the study, no adjustments for inflation were made.

Statistical Analysis

The primary end point of the trial was the change in the size of the perfusion defect from the image obtained with sestamibi at the time of admission and that obtained before discharge. The patients were analyzed on an intention-to-treat basis with an unpaired t-test. The study was designed to detect a difference between groups of 8 percent in the mean change in size of the perfusion defect in the left ventricle, which is approximately equivalent to a difference of 0.04 in the ejection fraction.[31]

By design, patients who died before predischarge imaging were assigned a value for the change in perfusion-defect size that was equivalent to the worst result measured in any patient. Patients who were referred for emergency coronary-artery bypass grafting before the initial imaging because reperfusion was inadequate after initial therapy were assigned a value of 0.

Additional analyses were performed by adjusting for the clinical features shown in Table 1, by omitting the patients with imputed values, by studying patients on the basis of treatment received rather than intention to treat, by using analysis of covariance (with infarct size as the dependent variable and myocardium at risk and other clinical variables as covariates), and by using nonparametric methods (with death assigned the worst rank).[32]

The protocol was approved by the institutional review board of the Mayo Clinic.

Results

General

A total of 108 patients were randomized. End-point data were not available for five patients because the radiopharmaceutical was not administered at the time of admission (three patients) or because the patient was unable or unwilling to undergo imaging (two patients).

There were four in-hospital deaths, two in each group, for an overall in-hospital mortality of 4 percent

Table 1. Clinical Features of 103 Patients for Whom Data on the Primary End Point Were Available, According to Randomization Group.

CHARACTERISTIC	THROMBOLYSIS	ANGIOPLASTY
No. of patients	56	47
Sex (M/F)	40/16	37/10
Age (yr)	62±13	60±11
No. with previous surgery or angioplasty	2	1
No. with previous infarct	7	2
ST elevation/depression	54/2	46/1
Infarct location		
Anterior	22	15
Inferior	30	31
Other	4	1
Infarct-related artery		
Left anterior descending	15	15
Left circumflex	7	3
Right coronary	19	29
Indeterminate or no angiography	15	—
Hours from chest pain to randomization		
<4	43	35
≥4	13	12
Minutes to treatment*		
All patients	232±174	277±144
Randomized in <4 hr†	158±56	210±76
Randomized in ≥4 hr	477±208	461±128

*Defined as the start of intravenous thrombolysis in the thrombolysis group and the first balloon inflation in the angioplasty group. Plus–minus values are means ±SD.

†P<0.001 for the difference between groups.

(Table 2). The size of the perfusion defect increased from the time of admission to discharge by 25 percent of the left ventricle (suggesting an extension of the infarct) in one patient assigned to angioplasty who died. The remaining three patients who died were assigned this value. Emergency bypass surgery was performed before the first set of images was obtained in two patients in the angioplasty group, who were assigned values of 0. The primary end point was measured directly in the remaining 98 patients. Imputed or measured data on 103 of the 108 patients enrolled are presented throughout this paper.

Base-Line Characteristics

Fifty-six patients were randomly assigned to thrombolysis, and 47 patients to angioplasty (Table 1). A small number of patients in each group had a history of previous myocardial infarction, angioplasty, or bypass surgery. Thirty-seven patients (36 percent) had anterior infarctions, including 22 in the thrombolysis group and 15 in the angioplasty group. Seventy-eight patients (76 percent) were randomized within four hours of the onset of chest pain, including 43 in the thrombolysis group and 35 in the angioplasty group.

In-Hospital Therapy

Immediate angioplasty was attempted in 45 of the 47 patients assigned to angioplasty; in the remaining 2 patients, the infarct-related artery was widely patent at the time of initial angiography. Angioplasty was successful in 42 of the 45 patients in whom it was attempted, for an overall success rate of 93 percent. Seven patients assigned to angioplasty (15 percent) had recurrent ischemia during hospitalization that required additional revascularization. One patient had successful repeat angioplasty; the remaining six underwent coronary-artery bypass grafting.

Thrombolytic therapy was administered to 51 of the 56 patients assigned to this therapy. Of the five patients who did not receive thrombolysis, two required prolonged resuscitation after randomization, two became severely hypertensive, and one was found to have a history of lymphoma; all five underwent successful immediate angioplasty. During hospitalization, recurrent ischemia requiring subsequent revascularization developed in 20 patients (36 percent) randomly assigned to thrombolysis. Angioplasty was attempted in 16 patients and was successful in 13 (81 percent). Bypass surgery was performed in seven patients, including the three in whom angioplasty was unsuccessful.

Measurements Using Sestamibi

The values for myocardium at risk, final infarct size, and myocardial salvage were slightly but not significantly larger in the thrombolysis group, reflecting the greater number of anterior infarcts in this group (Table 2). After adjustment for infarct location, the difference in mean salvage between the two groups was 0 (P = 0.98), with a 95 percent confidence interval of ±6 percent of the left ventricle.

In patients with anterior infarcts, the mean (±SD) amount of myocardial salvage was 27±21 percent of the left ventricle in the thrombolysis group and 31±21 percent of the left ventricle in the angioplasty group. There was a wide range of individual values within each group (Fig. 1).

In patients with inferior infarcts, myocardial salvage was 7±13 percent of the left ventricle in the

Table 2. Measurements with Sestamibi in 103 Randomized Patients for Whom Data on the Primary End Point Were Available.*

MEASURE	ALL PATIENTS			PATIENTS WITH ANTERIOR MI			PATIENTS WITH NON-ANTERIOR MI		
	THROMBOLYSIS	ANGIOPLASTY	P VALUE	THROMBOLYSIS	ANGIOPLASTY	P VALUE	THROMBOLYSIS	ANGIOPLASTY	P VALUE
Myocardium at risk	31±20	27±21	0.25	47±19	49±16	0.74	21±12	15±11	0.05
Final infarct size	15±17	13±16	0.48	20±22	18±20	0.83	12±10	10±14	0.53
Myocardial salvage	15±19	13±19	0.64	27±21	31±21	0.61	7±13	5±10	0.47
95% confidence interval	10–20	8–18	—	18–36	20–42	—	3–11	2–9	—

*All measurements are given as percentages of the left ventricle. Plus–minus values are means ±SD. MI denotes myocardial infarction.

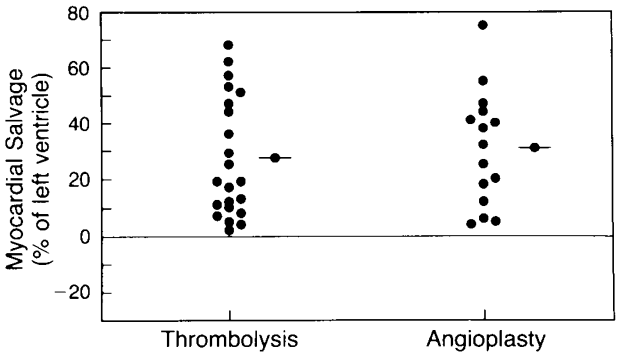

Figure 1. Myocardial Salvage as a Percentage of the Left Ventricle in Patients with Anterior Infarctions.

The mean values (horizontal lines) were 27 percent in the thrombolysis group and 31 percent in the angioplasty group. There was a wide range in each group, with no significant difference between the groups.

thrombolysis group and 5±10 percent of the left ventricle in the angioplasty group. There was a wide range of individual values in each group (Fig. 2), with less salvage than in anterior infarcts. The mean values in each treatment group were reduced by the inclusion of the patients who died and two additional patients (one in each group) with markedly negative values suggesting infarct extension.

Additional Analyses

There was no significant difference between thrombolysis and angioplasty when any of the alternative analyses were used. Myocardial salvage was measured as 14±20 percent of the left ventricle in the 31 patients treated with thrombolysis alone, 18±19 percent in the 20 patients treated with thrombolysis plus subsequent angioplasty or surgery, and 13±18 percent in the 50 patients treated with angioplasty alone.

Secondary End Points

There was no significant difference between the two groups in the ejection fraction at discharge or at six weeks, recurrent infarction, or death during six months of follow-up (Table 3). There were 12 late revascularization procedures in the thrombolysis group and 4 in the angioplasty group ($P = 0.075$).

Direct and Indirect Costs

The results of the cost analysis are shown in Table 4, studied according to the intention to treat. No significant difference in hospital cost or overall cost was found between the two treatment strategies, although there was a trend toward lower cost in the angioplasty group. Significantly lower six-month follow-up costs, numbers of initial hospital days, and numbers of readmissions were observed in the angioplasty group. The percentages of patients returning to work were not significantly different. The cost–effectiveness analysis did not demonstrate any significant difference between the two strategies. Analysis of cost data according to treatment received and according to the exclusion of patients who did not receive assigned treatment revealed no significant differences in monetary or indirect measures of cost and virtually identical cost–effectiveness ratios.

Discussion

The results of this trial indicate that in patients with acute myocardial infarction there is little difference in myocardial salvage between a strategy of immediate angioplasty and a strategy of thrombolysis followed by conservative treatment. Myocardial salvage was highly variable in both groups, presumably reflecting the variability in the time to reperfusion and collateral blood flow in the infarct zone. Patients in both treatment groups were treated with subsequent revascularization for episodes of recurrent ischemia. Thirty-six percent of the patients randomly assigned to initial therapy with intravenous thrombolysis underwent subsequent angioplasty or bypass surgery, whereas 15 percent of patients assigned to immediate angioplasty were so treated. Thus, these results apply to a treatment strategy that incorporates initial therapy with either thrombolysis or angioplasty and subsequent additional revascularization when clinically appropriate. Both initial therapies were associated with a low in-hospital mortality and salvage of approximately half the myocardium at risk. The method by which reperfusion is achieved in patients treated a mean of four hours after the onset of symptoms does not appear to be a major determinant of the efficacy of treatment. These results may not apply to therapy administered sooner after the onset of symptoms.

The implications of these results for clinical practice are clear. Since intravenous thrombolysis is easier to administer and more widely available, it remains the treatment of choice for initial therapy in most pa-

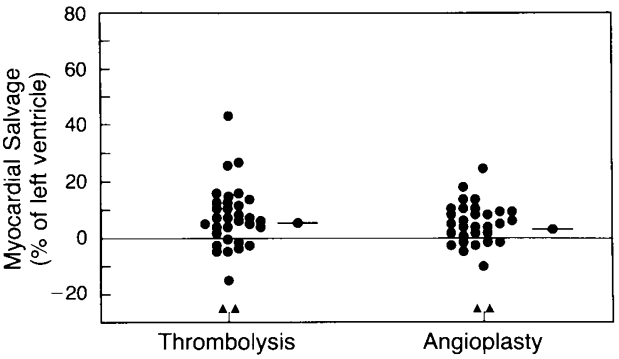

Figure 2. Myocardial Salvage as a Percentage of the Left Ventricle in Patients with Inferior Myocardial Infarctions.

The four in-hospital deaths are shown as triangles. Several patients had markedly negative values for myocardial salvage, indicating substantial extension of the infarct during the hospitalization. One patient with infarct extension involving 25 percent of the left ventricle died before discharge. Three other patients who died before discharge were assigned values of −25 percent because of the study design. The mean values for myocardial salvage (horizontal lines) were 7 percent in the thrombolysis group and 5 percent in the angioplasty group.

Table 3. Secondary End Points in the Patients Studied.*

END POINT	THROMBOLYSIS	ANGIOPLASTY	P VALUE
Ejection fraction on radionuclide angiography			
At discharge	0.50±0.11	0.53±0.12	0.22
After 6 wk	0.50±0.10	0.53±0.11	0.21
Events during 6-mo follow-up			
Death	0	1	0.68
Recurrent infarction	2	0	
Bypass grafting	8	2	0.075
Angioplasty	4	2	

*Plus–minus values are means ±SD.

tients eligible for thrombolytic therapy. Although subsequent angioplasty was frequently performed for recurrent ischemia in the patients treated with initial thrombolysis, more than half the patients assigned to initial thrombolysis did not require subsequent revascularization procedures. In patients with contraindications to thrombolysis, immediate angioplasty should be considered as an alternative therapy, because from the standpoint of myocardial salvage, its efficacy is equal to that of thrombolytic therapy. Much larger trials will be needed to determine whether it is equally efficacious in reducing early mortality.

The primary end point was the change in the size of the perfusion defect from admission to discharge on imaging with technetium-99m sestamibi. Studies in laboratory animals indicate that the injection of this radiolabeled agent during coronary occlusion permits assessment of myocardium at risk[19,20] and that its injection after reperfusion permits measurement of final infarct size.[20] Clinical studies have demonstrated that the final size of the perfusion defect as measured by imaging with sestamibi correlates closely with peak release of creatine kinase[22]; ejection fraction and regional wall motion at the time of discharge[21]; ejection fraction and regional wall motion at six weeks[31]; and ejection fraction, end-diastolic volume index, and end-systolic volume index at one year.[33] The change in the size of the perfusion defect on imaging between admission and discharge has been related to arterial patency[34] and to late recovery of regional wall motion as assessed by echocardiography.[35] The advantage of this end point for clinical trials is that it takes into account the known wide variability in myocardium at risk for a given coronary occlusion, which has been demonstrated in both animal models and clinical studies. In animal models of reperfusion, the size of myocardium at risk is responsible for 66 percent of the variability in final infarct size.[16]

The mean amount of myocardium salvaged in the patients treated with initial thrombolysis in this study is very similar to the amount previously reported by this laboratory for patients enrolled in the Thrombolysis in Myocardial Infarction trial.[21] The mean amount of myocardium salvaged in the patients treated with initial angioplasty in this study is very similar to the value that we reported previously for a nonrandomized series of patients undergoing successful primary angioplasty.[22] The mean final size of both anterior and inferior infarcts in the group treated with initial thrombolysis (20±22 and 12±10, respectively) was very similar to those reported previously from the Western Washington trial, which used tomographic thallium-201 imaging and a different quantitative method.[36]

When an intention-to-treat analysis was done, a trend toward lower costs was observed in the angioplasty group. Although the difference of approximately $4,500 in initial hospital costs did not reach statistical significance, primarily because of the large standard deviation, these results fit with a hospital stay for the angioplasty group that is a mean of three days shorter. Similarly, the significantly lower six-month follow-up costs correlate with substantially fewer readmissions in the angioplasty group. The savings of $378 per 1 percent of left ventricular myocardium salvaged in the angioplasty group and a difference of approximately $6,800 in the cumulative six-month cost, although tending to favor initial angioplasty, did not reach statistical significance. Although this cost analysis suggests an advantage of initial angioplasty, there is a major caveat about this conclusion. The analysis of cost data according to treatment received and according to the exclusion of patients who did not receive the assigned treatment showed no significant differences in costs. The higher monetary costs and number of hospital days in the more rigorous intention-to-treat analysis relate primarily to costs incurred by the five patients who had a crossover of treatment — i.e., who were initially randomly assigned to thrombolysis but who received immediate angioplasty because of medical contraindications to thrombolysis. This subgroup had the highest hospital charges and the most hospital days.

Several limitations of this study should be recog-

Table 4. Costs of Treatment Provided, According to the Intention-to-Treat Analysis.

VARIABLE	THROMBOLYSIS	ANGIOPLASTY	P VALUE
Hospital cost* ($)	21,400±14,806	16,811±8,827	0.09
Six-month follow-up cost ($)	2,738±7,666	480±3,069	0.03
Total six-month cost ($)	24,129±18,806	17,292±8,967	0.09
Hospital days	10.6±8.1	7.7±2.9	0.01
Coronary care unit days	4.3±3.6	4.0±2.6	0.6
Readmissions (% of group)	18	4	0.04
Resumption of work (% of group)	73	86	0.22
Cost ($)/1% LV myocardium†	1,491	1,113	0.28
95% Confidence interval	1,051–2,115	733–1,689	

*Charges for cardiac care are corrected by an average ratio of costs to charges of 80 percent; all values for costs and number of hospital days are means ±SD.

†LV denotes left ventricle. Ninety-five percent confidence intervals are based on asymptotic calculation of standard error for the logarithm of the cost-to-salvage ratio.

nized. Data on the primary end point were missing for five patients for technical reasons. Values for the primary end point were imputed in five other patients according to the prospective trial design. The value of −25 that was assigned to the three patients who died before the discharge imaging appears to be quite extreme for patients with inferior myocardial infarction. The results were similar, however, if the patients with imputed values were omitted from the analysis or if a somewhat less extreme value was assigned to the three patients who died before the final imaging.

The 95 percent confidence intervals for the primary end point did include a difference of 6 percent of the left ventricle between the two therapies, which could represent a difference of 4 percent of the left ventricle in the patients with inferior infarctions and a difference of 8 percent of the left ventricle in those with anterior infarctions. A difference in salvage of 8 percent of the left ventricle in the patients with anterior infarctions therefore remains possible, and could be evaluated only in a trial of 196 patients with anterior myocardial infarctions.

Finally, because many patients underwent subsequent revascularization for recurrent ischemia during their hospital course, this trial represents a comparison of two therapeutic strategies, rather than a study of the use of intravenous thrombolysis alone. The thrombolytic strategy requires that episodes of recurrent ischemia be treated according to the conservative strategy of the Thrombolysis in Myocardial Infarction trial.[24] A failure to treat episodes of recurrent ischemia might lead to a higher rate of reinfarction and therefore to lesser degrees of myocardial salvage in the thrombolysis group.

Despite these limitations, these data demonstrate that immediate angioplasty and thrombolysis followed by conservative treatment have equal rates of myocardial salvage in the treatment of patients with acute myocardial infarction without cardiogenic shock.

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