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Contemporary Reperfusion Therapy for Cardiogenic Shock: The GUSTO-I Trial Experience

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Objectives. **This study sought to examine the incidence, temporal profile and clinical implications of shock in a large trial of thrombolytic therapy for acute myocardial infarction.**

Background. **Despite advances in the treatment of acute ischemic syndromes, cardiogenic shock remains associated with significant morbidity and mortality.**

Methods. **Patients who presented within 6 h of symptom onset were randomized to four treatment strategies: 1) streptokinase plus subcutaneous heparin; 2) streptokinase plus intravenous heparin; 3) accelerated recombinant tissue-type plasminogen activator (rt-PA) plus intravenous heparin; or 4) streptokinase and rt-PA plus intravenous heparin. The primary end point was 30-day all-cause mortality.**

Results. **Shock occurred in 2,972 patients (7.2%): 315 (11%) had shock on arrival, and 2,657 (89%) developed shock after hospital admission. Reinfarction occurred in 11% of patients who developed shock compared with 3% of patients without shock. The mortality rate was significantly higher in patients who presented with (57%) or developed (55%) shock than in those without shock (3%) ($p < 0.001$). Shock developed significantly less frequently in patients receiving rt-PA. There were fewer deaths in patients who presented with shock and were treated with streptokinase plus intravenous heparin or who developed shock and were treated with streptokinase plus subcutaneous heparin. Patients who developed shock had a significantly lower 30-day mortality rate if angioplasty was performed.**

Conclusions. **Because cardiogenic shock occurred most often after admission and with recurrent ischemia and reinfarction, recognizing signs of incipient shock may improve outcome. Fewer patients treated with rt-PA developed shock, yet those developing shock had the same high mortality rate as those presenting with shock, regardless of treatment. Only angioplasty was associated with a significantly lower mortality rate.**

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Cardiogenic shock is a relatively uncommon complication of acute myocardial infarction, occurring in 5% to 15% of patients in published reports (1–5). The development of shock portends a very poor prognosis, and such patients account for a large proportion of the morbidity and mortality of acute infarction (1,2,4,6–10). Before the era of reperfusion therapy, the mortality rate from cardiogenic shock was ~80%. In a recent longitudinal study of cardiogenic shock from 1975 to 1988 (2), the overall in-hospital case fatality rate was 77.7%. Over the course of the study, the short-term mortality rate actually worsened, increasing from 73.7% in 1975 to 81.7% in 1988.

Early reports of thrombolytic therapy documented a disappointing improvement in mortality in patients with cardiogenic shock (8,11–16). In addition, many thrombolytic trials have systematically excluded patients with shock (17,18). In a recent meta-analysis of 94 reperfusion studies (19), only 22% included patients with cardiogenic shock, and only 3 studies performed subgroup analyses of patients with shock. Advances in reperfusion therapy have continued to occur with more widespread use of adjunctive therapies designed to maximize improvement in these high risk patients. The effect of these strategies on reducing mortality in patients with shock remains unclear.

The multicenter Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) trial (20) of 41,021 patients with acute myocardial infarction included a prospective plan for identifying the subset

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of patients with cardiogenic shock. In the present report, we analyze the GUSTO-I experience with cardiogenic shock and define the incidence, temporal profile and 30-day outcome in patients undergoing contemporary reperfusion treatment.

Methods

Patients. The details and primary end points of the GUSTO-I trial have been previously reported (20). Patients with chest pain lasting ≥20 min but <6 h with typical electrocardiographic (ECG) abnormalities (>0.1-mV ST segment elevation in two or more limb leads or >0.2-mV ST segment elevation in two or more precordial leads) were eligible for randomization to one of four intravenous thrombolytic strategies: 1) streptokinase (Kabikinase, Kabi Vitrum, Stockholm, Sweden), 1.5 million U over 1 h, with subcutaneous heparin, 12,500 U twice daily beginning 4 h after streptokinase; 2) streptokinase, 1.5 million U over 1 h with intravenous heparin (5,000-U bolus and infusion at 1,000 U/h), adjusted to achieve an activated partial thromboplastin time of 60 to 85 s; 3) accelerated recombinant tissue-type plasminogen activator (rt-PA) (Activase, Genentech), 15-mg bolus, 0.75 mg/kg body weight over 30 min (up to 50 mg) and 0.5 mg/kg (up to 35 mg) over the next hour with intravenous heparin; or 4) combined rt-PA (1.0 mg/kg over 60 min, not to exceed 90 mg, with 10% given as a bolus) and streptokinase (1 million U over 60 min) with intravenous heparin. Patient exclusion criteria included previous stroke, active bleeding, allergy to streptokinase and recent trauma or major operation. Cardiogenic shock was not an exclusion criterion; patients with shock were prospectively identified as a subgroup that would be analyzed separately, and a special data collection form was designed and completed for all patients with shock.

Adjunctive therapy included chewable aspirin, ≥160 mg (Bayer), as soon as possible, followed by 160 to 325 mg/day. Unless contraindicated, intravenous atenolol, 10 mg (ICI Pharmaceuticals), was given in two divided doses followed by daily oral therapy of 50 to 100 mg. Other medications, including nitrates, angiotensin-converting enzyme inhibitors, digitalis and antiarrhythmic and calcium channel blocking agents, were used at the discretion of the attending physician. Angiography, percutaneous transluminal coronary angioplasty, intraaortic balloon pumping and coronary artery bypass graft surgery were also used at the discretion of the investigators. The primary end point of the trial was all-cause mortality at 30 days. Other end points included a composite of death and nonfatal, disabling stroke.

Definitions. *Cardiogenic shock* was defined as systolic blood pressure <90 mm Hg for at least 1 h that was not responsive to fluid administration alone, thought to be secondary to cardiac dysfunction and associated with signs of hypoperfusion or a cardiac index ≤2.2 liters/min per m^2. Patients in whom systolic blood pressure increased to >90 mm Hg within 1 h after administration of positive inotropic agents were still classified as having cardiogenic shock. *Recurrent ischemia* was defined as symptoms (chest discomfort, arm pain, jaw pain, nausea); ECG changes; and new hypotension, pulmonary edema or murmur thought by the physician to represent myocardial ischemia. When the physician determined that a second myocardial infarction had occurred, *reinfarction* was defined on the basis of the presence of two or more of the following criteria: 1) recurrent ischemic symptoms lasting >15 min; 2) new ST-T wave changes or new Q waves; 3) a second elevation in cardiac enzyme levels to over the normal upper limit or by a further 20% if already over the normal upper limit; 4) angiographic reocclusion of a previously patent infarct-related artery.

Data analysis and statistical assessment. Baseline characteristics and clinical outcomes of patients presenting with cardiogenic shock (group A), patients developing shock during the hospital period (group B) and patients without shock (group C) were compared using chi-square tests for discrete variables and the Wilcoxon rank-sum test for continuous characteristics. Odds ratios and 95% confidence intervals were used to compare treatments. Continuous data are summarized as mean value ± SD. Results for categoric variables are given as the number and percent of patients with the specified characteristic. Significance tests were two-tailed, and treatments were compared using intention to treat.

Results

Patients. A total of 41,021 patients were enrolled. Cardiogenic shock was identified in 2,972 patients (7.2%), and data are missing for 285 patients. The majority of patients (2,657 [89%]) developed shock after the initial admission to hospital, and only 315 patients (11%) had cardiogenic shock at their initial assessment. Figure 1 demonstrates the time to development of cardiogenic shock for the treatment groups by intention to treat. The majority of patients who developed shock did so within 48 h of randomization.

There were significant differences between patients without shock and the entire group of those with shock (Table 1). The latter had a significantly higher incidence of risk factors associated with an adverse outcome, including greater age, female gender, previous infarction, anterior myocardial infarction and diabetes mellitus. As expected, systolic blood pressure was significantly lower and heart rate significantly higher in patients with cardiogenic shock.

Thrombolytic therapy. There were differences in time to administration of thrombolytic therapy among the three groups. Patients who developed shock had a significantly longer time to therapy than patients without cardiogenic shock (3.20 ± 1.75 h vs. 3.09 ± 1.62 h, $p = 0.01$). A similar proportion of patients were randomized to each treatment regimen; however, patients treated with accelerated rt-PA therapy were significantly less likely to develop cardiogenic shock than patients treated with the other regimens (Table 2) ($p < 0.001$).

Procedures performed. The number and type of procedures performed, both diagnostic and therapeutic, differed greatly among the three groups (Table 3). The frequency of cardiac catheterization was significantly higher in the patients without shock, as was performance of balloon angioplasty.

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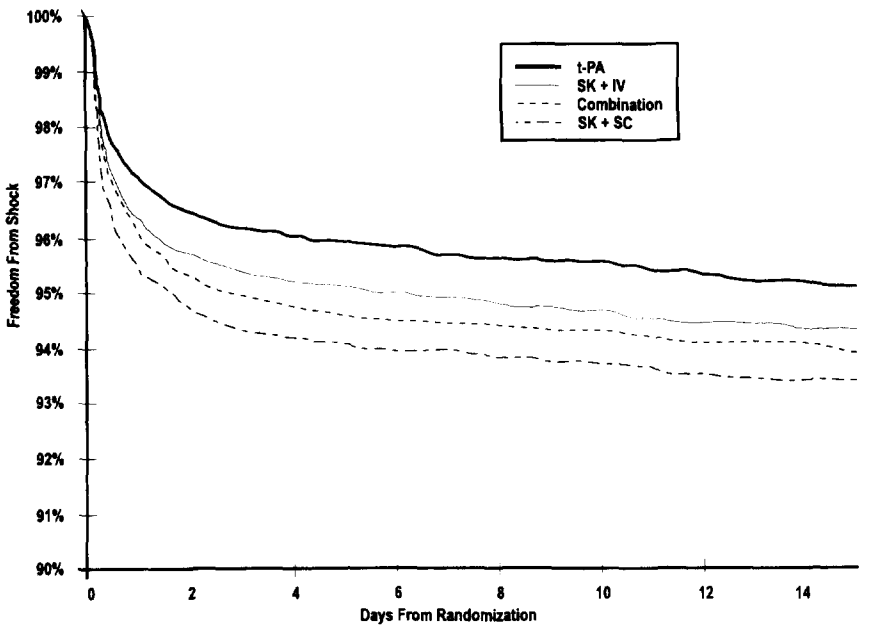

Figure 1. Time to development of cardiogenic shock. Combination = streptokinase and recombinant tissue-type plasminogen activator plus intravenous heparin; SK + SC = streptokinase plus subcutaneous heparin; SK + IV = streptokinase plus intravenous heparin; t-PA = accelerated recombinant tissue-type plasminogen activator.

However, placement of an intraaortic balloon pump was significantly greater in patients with shock (22% and 25% for those with shock on arrival and those who developed shock, respectively, vs. 2% for those who did not develop shock). As expected, the use of pacemakers, Swan-Ganz catheters and intubation for mechanical ventilation were all more frequent in patients with shock. Coronary artery bypass graft surgery was performed more frequently in patients who developed shock (12%) than in those without shock or in those who presented with shock (7%, $p < 0.001$).

Complications and clinical events. In-hospital complications were also significantly more frequent in patients with cardiogenic shock (Table 4), irrespective of when shock occurred. The most frequent arrhythmia was asystole, followed by ventricular fibrillation.

The in-hospital and 30-day mortality rates were significantly different (Table 5). Of the 2,851 patients in the GUSTO-I trial who died at 30 days, 1,647 had had cardiogenic shock. Therefore, cardiogenic shock was present in 58% of all deaths at 30 days in the entire trial. The 30-day mortality rates were 57% and 55%, respectively, in patients with shock on arrival and those who developed shock during the hospital stay compared with only 3% in patients with no shock. The combined end point of death or nonfatal, disabling stroke was similarly increased (59% and 56% vs. 4%, respectively). Patients who developed cardiogenic shock during the hospital stay had markedly increased rates of reinfarction and recurrent ischemia: Reinfarction occurred in 11% compared with 3% in patients with no shock ($p < 0.001$), and recurrent ischemia occurred in 28% and 19%, respectively ($p < 0.001$).

With respect to the four different thrombolytic strategies, there were nonsignificant differences in major clinical events (Table 6, Fig. 2). In patients with shock on arrival, those given streptokinase and intravenous heparin tended to have a lower in-hospital and 30-day mortality rate and combined end point of 30-day mortality or nonfatal, disabling stroke. In the larger group of patients who developed shock during the initial hospital stay, the streptokinase and subcutaneous heparin strategy was associated with a lower mortality rate (51% vs. 57% for rt-PA at 30 days) and combined end point of mortality or nonfatal, disabling stroke (52% vs. 57% for rt-PA).

Performance of balloon angioplasty. There was a relation between performance of dilation and outcome in the patients with shock (Table 7). Although angioplasty was performed in only a minority of patients in GUSTO-I (22%), there was a statistically significant and clinically important difference in patients with shock. The 30-day mortality rate in patients with cardiogenic shock on arrival who underwent angioplasty was 43% compared with 61% ($p = 0.028$) in patients with shock on arrival who did not undergo this procedure. Even more striking were the results of angioplasty in the larger group of patients who developed shock during the hospital stay. When angioplasty was performed in this group, the 30-day mortality rate was 32% versus 61% in patients who did not undergo dilation ($p < 0.001$).

Discussion

Results of clinical trials. The GUSTO-I trial, a large multicenter randomized trial of thrombolytic therapy for acute myocardial infarction, documented that cardiogenic shock remains an extremely serious complication. Death in association with cardiogenic shock accounted for 58% of all deaths in this 41,021-patient trial. The overall 30-day mortality rate in patients with cardiogenic shock was 55%. In this series, the paradox exists that, whereas cardiogenic shock is less common in patients treated with accelerated rt-PA, rt-PA appears to be less effective in the setting of shock that is present on arrival.

Table 1. Baseline Clinical Characteristics

	Shock on Arrival (n = 315)	Developed Shock (n = 2,657)	No Shock (n = 37,746)
Age (yr)	66.3 ± 10.7	67.1 ± 11.2	60.4 ± 11.8*
Male gender (%)	63	63	76
Hypertension (%)	39	44	38
Diabetes (%)	23	18	14
Previous MI (%)	28	25	16
Previous bypass surgery (%)	5	7	4
Infarction location (%)			
Anterior	54	50	38
Inferior	42	47	58
Other	4	3	3
Killip class (%)			
I	0	70	87
II	0	24	12
III	0	6	1
IV	100	0	0
Time to therapy (h)	2.97 ± 1.47	3.20 ± 1.75	3.09 ± 1.62†
Systolic BP (mm Hg)	94.1 ± 28.2	117.7 ± 25.6	130.1 ± 22.7
Heart rate (beats/min)	88.8 ± 30.7	81.4 ± 20.7	74.9 ± 17.0
Treatment randomization (%)			
SK + SC heparin	23	27	24
SK + IV heparin	25	27	25
rt-PA + IV heparin	25	22	26
Combination therapy	27	24	25

*All $p < 0.001$, unless otherwise indicated, for any shock versus no shock. †$p = 0.02$ for any shock versus no shock. Data presented are percent of patients or mean value ± SD. BP = blood pressure; Combination therapy = streptokinase (SK) and recombinant tissue-type plasminogen activator (rt-PA) plus intravenous (IV) heparin; MI = myocardial infarction; SC = subcutaneous.

Although angioplasty was used in only a minority of patients, it was associated with improved outcome.

Information from clinical trials of thrombolytic therapy and cardiogenic shock has been limited (8,19). In many thrombolytic trials, cardiogenic shock has been a specific exclusion criterion. In a meta-analysis of 94 randomized clinical trials of thrombolytic therapy through 1991 (19), only 3 performed subgroup analyses of patients with cardiogenic shock on arrival. Two of these three trials, which in aggregate included 636 patients, compared thrombolytic therapy with a control group; both failed to demonstrate a benefit in terms of reduced in-hospital mortality (11,16,19). Two other placebo-controlled trials documented 25% and 43% reductions in the incidence of cardiogenic shock after thrombolytic treatment compared with placebo, although when shock developed, mortality remained substantially increased (17,18).

Table 2. Incidence of Shock for Each Assigned Therapy

	rt-PA (n = 10,376)	SK + IV (n = 10,393)	Combination (n = 10,346)	SK + SC (n = 9,820)
Arrived in shock	0.8	0.8	0.8	0.7
Developed shock*	5.5	6.9	6.3	7.4
No shock*	93.7	92.4	92.9	91.8

*$p < 0.001$ for patients who developed shock versus those who did not and for rt-PA versus other thrombolytic agents. Data presented are percent of patients. Abbreviations as in Table 1.

Table 3. Procedures Performed

	Shock on Arrival (n = 315)	Developed Shock (n = 2,657)	No Shock (n = 37,764)
Cardiac catheterization	123 (40)	1,228 (46)	21,201 (56)*
PTCA	48 (15)	519 (20)	8,305 (22)
Intraaortic balloon	68 (22)	666 (25)	745 (2)
Bypass surgery	23 (7)	317 (12)	3,161 (8)
Cardioversion/defibrillation	113 (37)	928 (35)	2,763 (7)
Intubation	176 (56)	1,250 (47)	3,217 (9)
Swan-Ganz catheter	127 (41)	1,183 (45)	3,720 (10)
Pacemaker insertion	79 (25)	721 (27)	2,054 (5)

*All $p < 0.001$ for any shock versus no shock. Data presented are number (%) of patients. PTCA = percutaneous transluminal coronary angioplasty.

The International Study Group (21) documented lower mortality in patients with shock treated with streptokinase than in those treated with rt-PA. In that study, 64.9% of the patients treated with streptokinase died compared with 78.1% of the rt-PA–treated patients ($p = 0.04$). A trend toward improved mortality with streptokinase was also found in the present study, where patients who developed shock and who had been treated with streptokinase and subcutaneous heparin had a 30-day mortality of 51% compared with 57% for patients treated with rt-PA ($p = 0.061$). The mechanism for this reduction is not clear. Other possible explanations include afterload reduction related to hypotension with streptokinase (which would not seem beneficial in this setting) or decreased viscosity and attendant improved microcirculatory flow. Alternatively, these results could have occurred by chance; however, the fact that the same trend was present in another study makes this less likely. Although there was a trend toward improved mortality with streptokinase once shock developed, shock subsequently developed less frequently in patients randomized to rt-PA than streptokinase. This may be the result of the improved initial reperfusion rates with rt-PA. For prevention of shock, therefore, rt-PA is the preferred strategy.

Table 4. In-Hospital Complications

	Shock on Arrival (n = 315)	Developed Shock (n = 2,657)	No Shock (n = 37,764)
AV block	78 (25)	751 (28)	2,530 (7)*
Asystole	129 (42)	1,062 (40)	1,130 (3)*
Sustained VT	87 (28)	693 (26)	1,724 (5)*
Ventricular fibrillation	100 (32)	687 (26)	1,930 (5)*
Acute mitral regurgitation	12 (4)	176 (7)	383 (1)
Ventricular septal defect	3 (1)	65 (2)	127 (0.3)

*$p < 0.001$ for any shock versus no shock. Data presented are number (%) of patients (patients who developed multiple complications are counted more than once; therefore, the percent of patients may exceed 100). AV = atrioventricular; VT = ventricular tachycardia.

Table 5. Clinical Events

	Shock on Arrival (n = 315)	Developed Shock (n = 2,657)	No Shock (n = 37,764)
Reinfarction	5 (2)	302 (11)	1,312 (3)*
Recurrent ischemia	35 (11)	740 (28)	7,323 (19)*
Mortality			
In-hospital	181 (57)	1,477 (56)	1,019 (3)*
30-day	181 (57)	1,466 (55)†	1,164 (3)*
Stroke	8 (3)	72 (3)	510 (1)
Hemorrhagic	1 (0.3)	23 (0.9)	242 (0.6)
Nonhemorrhagic	5 (2.0)	33 (1.0)	32 (0.1)
30-day mortality or nonfatal, disabling stroke	186 (59)	1,496 (56)	1,477 (4)*

*$p < 0.001$ for any shock versus no shock. †Value is lower than that of in-hospital mortality because 11 patients died in hospital after stays >30 days. Data presented are number (%) of patients.

Timing of shock. The temporal profile of cardiogenic shock in patients with acute myocardial infarction is of substantial interest in view of the high mortality rate. In the present study, only a minority of patients presented with cardiogenic shock on admission; 89% developed shock after admission, the majority during the first 48 h after randomization. Other studies have documented similar findings (15). The preponderance of shock developing during the hospital stay may be a consequence of excluding patients presenting with shock from enrollment in clinical trials. Col et al. (19) have suggested that this may relate to difficulty in obtaining informed consent in this high risk population, urgency in treating them versus taking time for randomization protocols or physician perception of the appropriateness of the specific approach in these patients.

In the GUSTO-I trial, patients who developed cardiogenic shock were more likely to have reinfarction. Whether these patients had a larger index infarction or developed infarct extension cannot be determined.

Characteristics of patients who developed shock. Other studies with smaller patient numbers have documented factors associated with development of shock. The Secondary Prevention Reinfarction Israeli Nifedipine Trial (SPRINT) study group (15) identified several factors that were independent predictors of shock, including increasing age, female gender, history of angina pectoris, history of stroke, peripheral vascular disease, peak lactate dehydrogenase levels and hyperglycemia. Patients who had all factors, excluding lactate dehydrogenase elevation, had a 35% probability of developing in-hospital shock. The predictive value in a large population of patients remains to be determined.

The extent of myocardial loss required for the development of cardiogenic shock is thought to be at least 40% (22,23). These data are based on autopsy series; more recent nuclear scintigraphy using technetium-99m sestamibi (24) has shown that some patients with >40% infarction of the left ventricle may survive without congestive heart failure. Certainly larger infarctions have the greater propensity toward shock unless a specific mechanical factor is the etiology (e.g., ventricular septal defect or mitral regurgitation). Identification of patients with recurrent ischemia or recurrent infarction should alert the clinician to an increased potential for the development of cardiogenic shock.

A central finding of the present study is that the patients who developed cardiogenic shock during the hospital stay, under medical treatment, had the same high mortality as those with shock on arrival. This finding underlines the importance of developing algorithms for identification of high risk patients so that preventive strategies can be undertaken.

Adjunctive therapies. Despite the finding that mortality in GUSTO-I patients treated with thrombolytic therapy appeared to improve compared with that of historical cohorts, it remained very high. Therapy to interrupt the process of infarct extension or the natural progression of large infarcts during shock is currently being evaluated, including adenosine, L-carnitine, Rheothrix, P selectin inhibitors and fructose diphosphate (25–27).

Other adjunctive therapy has focused on restoration of patency in the infarct-related artery. In addition to infarct size, patency of the infarct-related artery is one of the most important predictors of in-hospital mortality. In a series of 200 patients with cardiogenic shock (6), the mortality of patients with a patent infarct-related artery was 33% versus 75% in those with an occluded artery. In selected series, the use of balloon angioplasty appears to be associated with improved outcome (28–33). Although angioplasty was performed in only 19% of GUSTO-I patients with shock, those who developed

Table 6. Outcome by Treatment Received

Outcome	rt-PA	SK + IV	Combination	SK + SC	p Value
Shock on arrival (n = 315)					
In-hospital mortality	47 (59)	43 (54)	50 (60)	41 (57)	0.82
30-day mortality	49 (61)	43 (54)	48 (57)	41 (59)	0.85
30-day mortality or nonfatal, disabling stroke	49 (61)	44 (56)	52 (62)	41 (57)	0.92
Developed shock (n = 2,657)					
In-hospital mortality	326 (57)	404 (57)	371 (57)	376 (52)	0.10
30-day mortality	323 (57)	399 (56)	370 (57)	374 (51)	0.12
30-day mortality or nonfatal, disabling stroke	325 (57)	404 (57)	375 (58)	380 (52)	0.15

Data are number (%) of patients. Abbreviations as in Table 1.

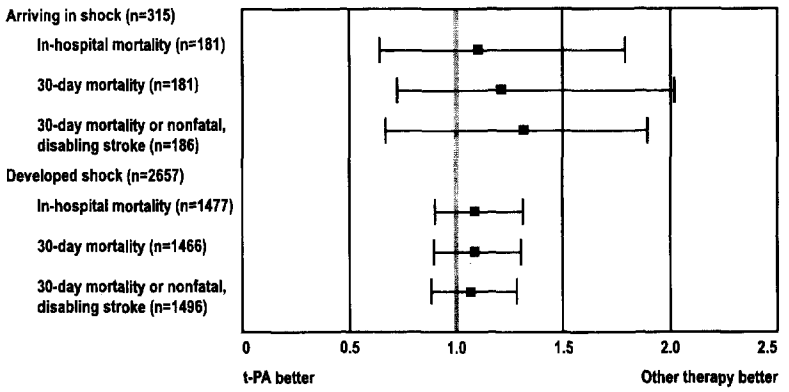

Figure 2. Odds ratios: outcomes according to treatment received. t-PA = recombinant tissue-type plasminogen activator. **Squares** = odds of end point occurring in individual patients; **horizontal bars** = 95% confidence interval.

shock during the hospital stay and underwent dilation had a mortality rate of 31% compared with 61% for patients who did not. These findings must be interpreted cautiously because of the potential risk of confounding in this analysis: Patient selection criteria for angiography and dilation, in addition to actual treatment received, could have had a major impact on the observed outcomes. Hochman et al. (10), in a pilot study of cardiogenic shock, documented that patients who underwent angiography had an improved outcome compared with patients who did not, irrespective of whether they had angioplasty. The most plausible hypothesis is that patients who are too ill to undergo angiography are at increased risk for a fatal outcome; these patients would have been counted in the "no angioplasty" group, leading to a substantial bias. The same considerations apply for the finding in the present study of improved outcomes in patients who underwent dilation. Patients who are at very high risk may not survive long enough to undergo angioplasty. However, because of the important relation between patency and improved left ventricular function and survival in the GUSTO-I angiographic substudy, it is reasonable to maximize reperfusion in these high risk patients.

Other adjunctive therapies are also important (34–40). Intraaortic balloon counterpulsation has been shown to enhance lysis and perfusion and to prevent reocclusion (35–40). It has also recently been shown to improve the outcome in patients with cardiogenic shock (40). In view of the available data, intraaortic balloon pumping may have been underutilized in the GUSTO-I trial because it was used in only 22% of patients with shock on arrival and in 25% of patients who developed shock during the hospital stay. However, many centers did not have on-site facilities for placement of a balloon pump. The combination of mechanical reperfusion and intraaortic balloon pumping may optimize outcome. Finally, surgical revascularization may also play a role, although the logistics are daunting and mortality is high.

Conclusions. Cardiogenic shock in association with acute myocardial infarction resulted in markedly increased mortality and was present in a majority of the patients in the GUSTO-I trial who died. This high mortality was seen whether the patient presented with or developed shock. The rt-PA treatment was associated with a reduction in the subsequent development of cardiogenic shock compared with streptokinase; however, if shock was present on arrival, patients randomized to streptokinase tended to have improved survival. Streptokinase treatment was associated with a somewhat lower mortality, probably related to its relative lack of dependency on tissue perfusion for clot lysis. However, irrespective of the specific thrombolytic regimen used, the mortality rates for patients with shock remained very high. Most commonly, shock occurred after admission and in the setting of recurrent ischemia or reinfarction. Patients with cardiogenic shock who underwent dilation showed substantial improvement in survival compared with those who did not. These data support an intense effort at recognizing patients at risk for shock and an aggressive approach to reperfusion in these high risk patients. Patients who developed shock during the hospital stay had the same prognosis as those with shock on arrival. Development of a means of recognizing incipient signs of shock may lead to methods of further enhancing outcomes.

Table 7. Relation Between Procedure and Outcome

	30-Day Mortality				p
	Cath Only	PTCA	CABG	None	Value
Shock on arrival	10 (20)	20 (43)	5 (22)	143 (77)	< 0.001
Developed shock	160 (35)	145 (32)	93 (29)	1,057 (75)	< 0.001
No shock	119 (1)	62 (1)	45 (1)	934 (6)	< 0.001

Data presented are number (%) of patients. CABG = coronary artery bypass graft surgery; Cath = catheterization; PTCA = percutaneous transluminal coronary angioplasty.

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