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Revisiting Reperfusion Therapy in Inferior Myocardial Infarction

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Although thrombolytic therapy for acute myocardial infarction (MI) is recommended without regard for infarct location, treatment results are less impressive for inferior than for anterior MI because the amount of myocardium at risk is smaller and less strategically located, and the mortality risk is lower. Whereas the risks associated with anterior MI are relatively constant, high risk subsets of patients with an inferior MI can be identified by simple electrocardiographic criteria, including left precordial ST segment depression, complete atrioventricular heart block and right precordial ST segment elevation. Unfortunately, none of the placebo-controlled, randomized trials have analyzed the benefit of thrombolytic therapy for inferior MI in high risk versus low risk subsets.

Thrombolytic therapy should be more successful in reducing infarct size and decreasing mortality in high risk patients with an inferior MI. Thrombolytic therapy may not decrease hospital mortality in low risk patients (baseline risk 2% to 4%) or those with symptom duration >6 h. Whereas it is arguable whether coronary angioplasty is superior to thrombolytic therapy in anterior MI, there are no mortality data to support using angioplasty as a primary or rescue reperfusion strategy instead of thrombolytic therapy in inferior MI, unless thrombolytic contraindications are present or the patient is in cardiogenic shock.

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Five large placebo-controlled multicenter trials (1–5) have convincingly demonstrated that thrombolytic therapy reduces morbidity and mortality in acute myocardial infarction (MI). The benefits have been consistent across almost all subset analyses, justifying appeals to expand restrictive treatment indications so that increasing numbers of patients might have the opportunity for therapeutic benefit (6,7). One of the most important, and still somewhat controversial, subsets is infarct location. Whereas treatment efficacy has been universally accepted for thrombolytic candidates with an anterior MI, skepticism continues to exist regarding treating patients who present with an inferior MI (8–10).

There are several possible explanations for the inconsistency of opinion about treatment recommendations for acute inferior MI: 1) Because mortality with inferior MI is half that of anterior MI, many studies enrolling relatively small numbers of patients have been statistically underpowered to detect a treatment difference; 2) because the amount of myocardium at risk is generally smaller and less strategically located with inferior MI, the window of reperfusion opportunity is shorter and is more likely to be obscured by trials including patients with delayed treatment; 3) patients with inferior MI can be classified by electrocardiographic criteria into high and low risk categories compared with the more homogeneous risk associated with anterior MI. An increased proportion of low risk patients enrolled in a trial will diminish the ability to show a treatment benefit. 4) The critical omission in the early trials of aspirin as adjunctive therapy for streptokinase (SK) has confused the debate.

Mortality Results by Infarct Location

Anterior MI. The 21- to 35-day mortality results from the large placebo-controlled multicenter trials (1–5) for patients with anterior MI treated with intravenous thrombolytic therapy are shown in Figure 1. The control group mortality rate ranged from 14.2% to 20.6% (mean 18.1%) and was significantly reduced with thrombolytic therapy, irrespective of sample size, time to treatment or omission of aspirin as adjunctive therapy for SK. Similar results were found in small placebo-controlled trials from the United States (11) and New Zealand (12). In contrast, German investigators in the ISAM trial (13) could not show a significant reduction in mortality rate with SK therapy (9.2% vs. 9.7%) despite limiting symptom duration to 6 h; treating with aspirin, heparin and a warfarin derivative; and preserving left ventricular function. The control mortality rate in the ISAM trial was much lower than that in the other trials. Either aggressive adjunctive therapy significantly decreased mortality or a lower risk group of patients was enrolled.

Inferior MI. The 21- to 35-day mortality results for patients with inferior MI (1–5) are shown in Figure 2. The control group mortality rate ranged from 7.8% to 10.2% (mean 8.6%). SK therapy without adjunctive aspirin had only a small impact on mortality reduction (1,3). These poor results may be due to lower patency rates and higher reocclusion rates than have been documented for other treatment strategies. Several

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Abbreviations and Acronyms

AV = atrioventricular
ECG = electrocardiogram, electrocardiographic
HBDH = alpha-hydroxybutyrate dehydrogenase
LVEF = left ventricular ejection fraction
MI = myocardial infarction
MR = mitral regurgitation
PTCA = percutaneous transluminal coronary angioplasty
RCA = right coronary artery
RV = right ventricle, right ventricular
SK = streptokinase

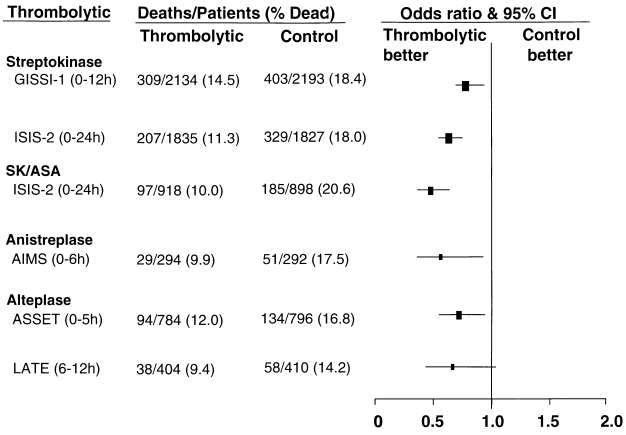

Figure 1. Anterior MI. Odds ratios and 95% confidence intervals (CI) for reduction of 21- to 35-day mortality in the groups assigned to thrombolytic therapy versus placebo. AIMS = APSAC Intervention Mortality Study; ASSET = Anglo-Scandinavian Study of Early Thrombolysis; GISSI-1 = Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico; ISIS-2 = Second International Study of Infarct Survival; LATE = Late Assessment of Thrombolytic Efficacy; SK/ASA = streptokinase/aspirin.

(14,15) but not all (16) studies have found that reperfusion rates with SK are lower for inferior than for anterior MI. In The Netherlands Interuniversity trial (8), where aspirin was not given, the 1-year reinfarction rate after inferior MI was 21% versus 8% after anterior MI.

In contrast, when aspirin was added to SK (3), or when anistreplase (2) or alteplase (4) was given, mortality was significantly reduced in placebo-controlled trials. Results from the LATE trial (5) suggest that the treatment window might be 6 h for inferior MI compared with 12 h for anterior MI. The lack of treatment benefit seen in the Western Washington Intravenous Streptokinase trial (11) can be explained by small sample size (n = 234), failure to use adjunctive aspirin therapy and low mortality risk (1.8% in control patients). A reduced mortality rate was demonstrated in the treatment group in the New Zealand trial (3.0% vs. 8.7%) (12). The ISAM study (13) demonstrated the same insignificant 0.5% mortality benefit (4.2% vs. 4.7%) for inferior as for anterior MI. However, at 7 months the mortality rate was improved (10.2% vs. 14.2%) in the ISAM trial (17), as it was after 6 months in the ASSET trial (7.7% vs 12.8%) (4) and at 1 year in the AIMS trial (7.6% vs. 11.9%) (2). When the Fibrinolytic Therapy Trialists (FTT) collaborative group (18) pooled results from nine placebo-controlled studies enrolling >1,000 patients, the mortality benefit was only 7.5% vs. 8.4%. The inclusion of patients with low risk, uncertain indications and symptom duration >12 h dilutes the potential mortality benefit that can be obtained when patients with ongoing ischemic chest pain, ST segment elevation and symptom duration <6 h are treated.

ECG High Risk Subsets in Inferior MI

Whereas the risks associated with anterior MI are relatively consistent, high risk subgroups of patients with inferior MI can be identified by simple ECG criteria (19). These include patients with left precordial ST segment depression, third-degree atrioventricular (AV) block and right precordial ST segment elevation.

Left precordial ST segment depression. The significance of concomitant ST segment depression in patients with inferior ST segment elevation from acute inferior MI has been extensively investigated. Although some have suggested that these are electrical reciprocal changes without anatomic or physiologic implications (20), and others have implicated anterior wall ischemia due to left anterior descending coronary artery disease (21), overwhelming evidence supports a larger mass of ischemic myocardium perfused by a more extensive infarct-related artery as the usual cause of the ECG abnormality (22). In a consecutive series of patients undergoing acute coronary arteriography, the infarct-related artery was more often the right coronary artery (RCA) than the circumflex artery (79% vs. 21%), but the precordial changes were more frequent with circumflex infarct-related arteries (71% vs. 40%) (23). Precordial ST segment depression occurs in ~50% of patients with a first inferior MI (23). However, the changes can be attenuated by right ventricular (RV) ischemia (24), preexisting precordial ST segment elevation or reperfusion and usually resolve within 24 h (25). The magnitude of precordial ST segment depression is positively correlated with the magnitude of inferior ST segment elevation (26). The presence of these changes is prognostically important because they are associated with larger infarct size (22,25,27), lower left ventricular ejection fraction (LVEF) (22,25) and more complications (21), including death (22,25). Persistent precordial ST segment depression is particularly important in this regard (27). Precordial ST segment depression on hospital admission has also been associated with a higher mortality rate during follow-up after hospital discharge for up to 5 years (27,28).

Two placebo-controlled studies (29,30) have evaluated the significance of precordial ST segment depression in patients treated with thrombolytic therapy. The Netherlands Interuniversity trial (29) studied the value of the initial ECG in predicting infarct size limitation in 488 patients by measuring cumulative 72-h release of myocardial alpha-hydroxybutyrate dehydrogenase (HBDH). Compared with conventionally

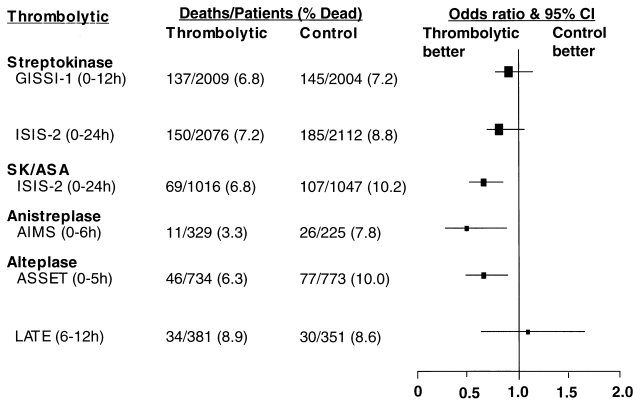

Figure 2. Inferior MI. Odds ratios and confidence intervals for reduction of 21- to 35-day mortality in the groups assigned to thrombolytic therapy versus placebo. Abbreviations as in Figure 1.

treated patients, infarct size limitation was shown with SK (no aspirin) in both anterior MI (480 U/liter of HBDH) and inferior MI (330 U/liter of HBDH). In inferior MI, >6 mm versus ≤6 mm of summed ST segment elevation in leads II, III and aVF was associated with better infarct size limitation (460 vs. 240 U/liter of HBDH), as was >4 mm versus ≤4 mm of summed ST segment depression in leads I, aVL and V_1 to V_6 (430 vs. 260 U/liter of HBDH). Note that the benefit in patients with an inferior MI and more extensive ECG abnormalities was similar to the median infarct size limitation in patients with anterior MI.

The European Cooperative Study Group (30) performed a similar study in 655 patients treated with aspirin, intravenous heparin and either alteplase or placebo. The summed amount of both ST segment elevation and depression measured 60 ms after the J point had a linear relation with cumulative HBDH release, LVEF and hospital mortality. In inferior MI, treatment benefit when summed precordial ST segment depression was >5.5 mm versus ≤5.5 mm was associated with greater HBDH reduction (234 vs. 137 U/liter of HBDH) and better LVEF preservation (3.6% vs. 2.5%). Importantly, the hospital mortality rate was reduced from 6.8% to 1.9% by alteplase in patients with a precordial ST segment depression sum >5.5 mm; the mortality rate was reduced only from 1.7% to 1.1% when the sum was ≤5.5 mm.

The Thrombolysis and Angioplasty in Myocardial Infarction (TAMI) study group (15) noted a higher complication rate and a worse ventriculographic outcome in inferior MI despite reperfusion therapy when precordial changes were present. These results have been confirmed by the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) study in >16,000 patients (31). Moreover, hospital and 1-year mortality rates were higher when precordial changes were present in the GUSTO-I study (31).

Thus, it appears that thrombolytic therapy is most likely to reduce infarct size and mortality in patients with inferior MI when precordial ST segment depression is present. A similar benefit would also be expected when concomitant ST segment elevation or depression is present in the lateral leads. Patients without precordial or lateral changes have such a good prognosis (hospital mortality rate 2% to 4%) that it is unlikely that thrombolytic therapy will alter their 30-day mortality rate.

AV heart block. Berger and Ryan (19) reviewed the published data before 1989 and documented third-degree AV block in 12% of patients with acute inferior MI, with an additional 7% having second-degree AV block. More recent reports (32,33) with equivalent sample sizes have noted a similar incidence of third-degree AV block. The onset is gradual in 50% of patients, according to Berger and Ryan (19), and abrupt in the other 50%. Two-thirds of the patients who develop AV block do so within the first 24 h (19,34). Mortality rates are high for third-degree AV block (29% to 37% vs. 6% to 14% without AV block (19,32,33) but are not elevated with second-degree AV block (35) compared with patients without AV block. Mortality appears to be due to larger infarct size rather than directly related to the electrical abnormality or to concomitant multivessel disease (19,34). Unfortunately, only a few studies have addressed the issue, which contrasts with published data on precordial ST segment depression.

Nicod et al. (34) noted greater creatine kinase enzyme release (1,840 vs. 1,322 IU/liter) in patients with AV block. A lower LVEF was documented by Kaul et al. (36) (49% vs. 55%) and Strasberg et al. (37) (51% vs. 58%). Importantly, ~50% of patients with third-degree AV block have RV MI (36,37), which may contribute to their poor prognosis. Others have demonstrated higher rates of atrial and ventricular arrhythmias, congestive heart failure and cardiogenic shock (32,33). However, hospital survivors have the same LVEF as patients who do not develop third-degree AV block (34), and there is no increased risk for subsequent mortality (32–34). The explanation for this paradox may be that whereas precordial ST segment depression predicts a larger ischemic area at risk for infarction, third-degree AV block may be a common marker of the larger infarctions that result in pulmonary edema, cardiogenic shock and acute mortality. A recent report (38) demonstrating rapid and lasting restoration of normal sinus rhythm after injection of the adenosine antagonist theophylline (100 mg/min to a maximum of 250 mg) suggests that third-degree AV block is a metabolic complication rather than an ischemic complication.

Four reports (39–42) have evaluated the impact of thrombolytic therapy in patients with AV block. Clemmenson et al. (39) noted a 13% (50 of 373) incidence of third-degree AV block in patients with a inferior MI; the mortality rate was 20% compared with 4% in patients without third-degree AV block despite thrombolytic therapy. Berger et al. (40) found a 12% (214 of 1,796) incidence of second-degree or third-degree AV block in a lower risk group of patients with inferior MI. The mortality rate was 7.1% versus 2.7% when AV block was absent. The relative risk for mortality for patients with AV block receiving thrombolytic therapy in each study was equivalent to that from the prethrombolytic era (19,32,33), and LVEF values in survivors again were not influenced by the

development of AV block. This finding supports the concept that AV block is a complication of the larger infarction rather than predictive of a larger ischemic area, where reperfusion might be expected to salvage myocardium and reduce mortality. Alternatively, thrombolytic therapy may be ineffective in reducing mortality in the large number of patients who present with third-degree AV block and have associated hypotension (43) or shock (44) or who develop third-degree AV block after thrombolytic therapy due to failure to reperfuse or because of infarct-related artery reocclusion.

McNeill et al. (41) initiated very early thrombolytic treatment in 20 patients, and no deaths occurred. Duration of AV block was much less than in their prethrombolytic era experience, confirming the same observation by Clemmenson et al. (39). In a preliminary report, Maggioni et al. (42) found a 10.4% incidence of third-degree AV block in 4,194 patients treated with thrombolytic therapy in the GISSI-2 trial. These patients had a 15-day mortality rate of 12.8% versus 4.5% in patients without third-degree AV block.

Thus, although third-degree AV block is associated with larger infarct size and increased risk for in-hospital morbidity and mortality, the present data do not clearly demonstrate that thrombolytic therapy reduces the relative risk of mortality. The absolute mortality reduction with thrombolytic therapy is not known because thrombolytic trials exclude a large number of high risk patients who are included in observational studies.

Right precordial ST segment elevation. Right coronary artery thrombosis proximal to the RV branches or proximal occlusion of a dominant circumflex artery produces right precordial ST segment elevation and RV dysfunction in ~40% of patients with inferior MI (45). Fifty percent of these patients have no clinical abnormalities, despite right precordial ECG ST segment elevation or RV echocardiographic abnormalities, whereas 50% have hemodynamic perturbations. The most serious hemodynamic complication is cardiogenic shock, which occurs in 3% to 8% of patients with inferior MI (46–48). The mortality rate in eight studies reporting on 58 patients with RV myocardial infarction and shock was 34% (46,48–54), approximately half that reported for left ventricular myocardial infarction with shock. Other complications of RV myocardial infarction include high degree AV block in as many as 50% of patients, rupture of the interventricular septum, thrombus formation and subsequent pulmonary embolism, refractory hypoxemia due to right to left interatrial shunting through a patent foramen ovale or atrial septal defect and tricuspid regurgitation (45). In-hospital mortality for RV myocardial infarction is rare, unless cardiogenic shock is present. Long-term prognosis is predominantly determined by left ventricular function but appears to be worsened by the combination of both right and left ventricular dysfunction (55,56).

Isolated RV free wall ischemia seldom disturbs cardiac function, but several associated factors can lead to hemodynamic impairment (57,58). Occlusion of the RCA branches to the right atrium produces ischemic right atrial dilation and dysfunction. Right atrial volume and pressure overload impede venous return to the atrium and optimal filling of the RV. Similarly, loss of atrial function due to atrial fibrillation or AV dysynchrony are electrical complications that compromise the preload-dependent RV. RV stroke volume is also dependent on heart rate such that bradyarrhythmias due to sinus node dysfunction or the Bezold-Jarisch reflex decrease cardiac output. Finally, ischemic dysfunction of global left ventricular contraction, interventricular contraction or the crista supraventricularis muscle bundle in the RV diminish systolic ventricular interactions that compensate for RV dysfunction. The hemodynamic abnormalities resulting from RV dysfunction include disproportionate elevation of right-sided filling pressures, systemic hypotension and low cardiac output.

RV dysfunction in the setting of inferior MI is predominantly due to ischemia rather than infarction. In the absence of reperfusion therapy, many (59–60) but not all (61–62) studies have shown substantial improvement in RV ejection fraction in the majority of patients over a few days to several weeks. The improvement is probably due to resolution of ischemic dysfunction secondary to spontaneous reperfusion of the RCA or to the development of collateral circulation. The ability of the RV to tolerate prolonged ischemia better than the left ventricle was demonstrated in an elegant study by Schofer et al. (63). Intracoronary thallium scintigraphy was performed before and after thrombolysis in 11 patients. RV thallium defects detected before thrombolysis were reversible. The determination of myocardial necrosis by technetium-99m accumulation in the RV was seen in three of eight patients versus left ventricular accumulation in seven of eight patients.

The RV is less prone to infarction than the left ventricle because of a more favorable oxygen supply–demand relation. Several factors increase oxygen supply: 1) The thin-walled RV has a lower coronary vascular resistance, with minimal compression of the microvasculature during systole. This and a relatively higher coronary artery/RV end-diastolic pressure gradient result in both systolic and diastolic coronary blood flow in the RV as opposed to predominantly diastolic flow in the left ventricle (64). 2) The left anterior descending coronary artery contributes 25% of the right ventricular free wall blood flow (65). 3) RV transmural perfusion is more homogeneous, whereas left ventricular ischemia causes disproportionate subendocardial ischemia. 4) Potential collateral flow from the left main coronary artery to the RCA is about three times that available in the opposite direction (66). 5) Direct perfusion from the RV cavity through the thebesian venous system is possible (67), but the concept has been challenged (68). Oxygen demand is reduced in the RV compared with the left ventricle because it has one-sixth the muscle mass, performs one-fourth the stroke work and ejects against one-tenth the afterload (69).

The ability to tolerate prolonged RV ischemia without necrosis should make these patients attractive candidates for reperfusion. Unfortunately, only scant data exist from small studies (60–62,70). In patients achieving reperfusion, the RV ejection fraction was improved by 24 to 48 h and improved further at later time points. The presence of RV wall motion abnormalities on predischarge radionuclide ventriculography

Table 1. Infarct Size Reduction After Thrombolytic Therapy

Study (ref no.)	No. of Pts	Time	Method of Detection	Infarct Size Reduction	
				Ant MI	Inf MI
van der Laarse et al. (72)	495	3 days	Enzymes	34%	31%
Ritchie et al. (73)	100	8 wk	SPECT	17%	31%
Ritchie et al. (74)	207	8 wk	SPECT	15%	19%
Schroder et al. (14)					
<3 h	389	2 days	Enzymes	16%	14%
<6 h	651	2 days	Enzymes	10%	4%
Bassand et al. (75)	231	3 wk	SPECT	33%	16%

Ant = anterior; Inf = inferior; MI = myocardial infarction; Pts = patients; ref = reference; SPECT = single-photon emission computed tomography.

in the TIMI-II study (71) was strongly associated with an occluded infarct-related artery (42% vs. 13% with a patent infarct-related artery), suggesting that early patency facilitates early recovery of RV function. The influence of thrombolytic therapy on mortality in patients with RV MI was not examined in the placebo-controlled thrombolytic trials.

Left Ventricular Function

The effect of thrombolytic therapy on left ventricular function can be assessed by evaluating infarct size, end-systolic volumes and LVEF.

Infarct size. The measurement of myocardial infarct size has been accomplished using enzymatic and scintigraphic techniques (Table 1). Thrombolytic therapy produces a reduction in infarct size in both anterior and inferior MI (14,72–75).

End-systolic volume. End-systolic volume has been proven to be an important prognostic variable (76). In the Interuniversity Trial (77), SK therapy resulted in significantly lower end-systolic volumes in both anterior (45 vs. 60 ml/m^2) and inferior MI (37 vs. 48 ml/m^2). Similarly, White et al. (12) documented significantly lower end-systolic volumes in both anterior (61 vs. 85 ml) and inferior MI (53 vs. 64 ml) with SK therapy.

LVEF. Several placebo-controlled trials (12,73–75,77–81) have reported LVEF results by infarct location using either radionuclide or contrast ventriculography (Table 2). LVEF in inferior MI is higher than in anterior MI, in keeping with the concept that better LVEF is associated with lower mortality risk. Preservation of LVEF occurs in both anterior and inferior MI, although the benefit is somewhat greater for anterior MI, presumably because of the larger amount of ischemic myocardium at risk.

Thus, although the risk of mortality from inferior MI is half that of anterior MI, and the mortality reduction with thrombolytic therapy in inferior MI is approximately half that in anterior MI, the salutary effects on ventricular function due to thrombolytic therapy are more equivalent.

Mitral Regurgitation

Significant mitral regurgitation (MR) is an important complication of acute MI because it is associated with an increased risk of heart failure and death (82). MR has been found by auscultation in 17% to 55%, echocardiography in 20% to 50% and contrast ventriculography in 5% to 20% of patients with an acute MI, but severe MR occurs in <5% (82). Normally, systolic contraction of the papillary muscles maintains coapta-

Table 2. Left Ventricular Ejection Fraction After Thrombolytic Therapy

Study (ref no.)	No. of Pts		Lytic Agent	Route	Time	Method	LVEF (%)					
							Ant MI			Inf MI		
	Ant MI	Inf MI					Lytic	Control	Δ	Lytic	Control	Δ
Ritchie et al. (73)	89	118	SK	IC	8 wk	RNA	41	37	+4	52	49	+3
Ritchie et al. (74)	64	141	SK	IV	8 wk	RNA	43	37	+6	54	52	+2
Serruys et al. (77)	148	184	SK	IC/IV	2 wk	Contrast	50	43	+7	57	49	+8
White et al. (12)	64	91	SK	IV	3 wk	Contrast	57	49	+8	60	55	+5
Guerci et al. (78)	53	80	rt-PA	IV	10 days	RNA	45	33	+12	59	54	+5
O'Rourke et al. (79)	57	69	rt-PA	IV	3 wk	Contrast	57	47	+10	66	59	+7
NHFA (80)	40	61	rt-PA	IV	1 wk	Contrast	53	40	+13	62	57	+5
Bassand et al. (75)	88	121	APSAC	IV	4 days	Contrast	47	40	+7	56	51	+5
Meinertz et al. (81)	144	169	APSAC	IV	2–3 wk	Contrast	53	54	−1	60	61	−1

APSAC = anisoylated plasminogen–streptokinase (SK) activator complex; IC = intracoronary; IV = intravenous; LVEF = left ventricular ejection fraction; NHFA = National Heart Foundation of Australia; RNA = radionuclide angiogram; SK = streptokinase; rt-PA = recombinant tissue-type plasminogen activator; Δ = change; other abbreviations as in Table 1.

tion of the leaflets and prevents prolapse of the valve into the left atrium. Ischemia of the papillary muscle can cause diastolic lengthening or decreased systolic shortening, leading to leaflet prolapse. Alternatively, regional dyssynergy of the left ventricular segment at the base of a papillary muscle or alteration of the normal spatial geometry of the papillary muscles and the chordae tendineae due to left ventricular dilation can compromise leaflet coaptation.

Whereas the anterolateral papillary muscle is perfused by the diagonal branches of the left anterior descending coronary artery and the obtuse marginal branches of the circumflex artery, the posteromedial papillary muscle is perfused only by the posterior descending artery and is thus more vulnerable to ischemic dysfunction or rupture. Therefore, posterolateral MI is more common in patients with acute ischemic MR. Clinical predictors of developing acute ischemic MR include female gender, older age, previous MI, multivessel disease, a large MI and an occluded infarct-related artery (82–85).

The impact of reperfusion therapy on preventing ischemic MR has not been clearly elucidated. Lehmann et al. (83) reported no benefit in the TIMI trial, where 206 patients initially had angiographic occlusion of the infarct-related artery before thrombolytic therapy. However, they excluded 13 of 27 patients with initial MR because those patients did not have repeat contrast ventriculography before discharge, and they added 7 patients to the analysis who had late, but not early, MR. In contrast to the mild MR studied by Lehmann et al. (83), Tcheng et al. (82) described 50 patients with moderately severe to severe MR from 1,480 consecutive patients undergoing acute cardiac catheterization at Duke University. They also described no benefit with reperfusion therapy, but only 23 patients received early thrombolytic therapy, and only 22 of the remaining 32 patients underwent percutaneous transluminal coronary angioplasty (PTCA).

The most rigorous report comes from Leor et al. (86) who studied 104 patients with a first inferior MI. Thrombolytic therapy was given to 55 patients; the 49 patients ineligible for thrombolytic therapy formed the control group. Treated patients had a marked reduction in moderate to severe MR measured by pulsed Doppler echocardiography at 24 h (4% vs. 16%), 7 to 10 days (11% vs. 24%) and 28 to 30 days (7% vs. 15%). Severe MR developed in five patients in the control group but in no patients in the treatment group. In a follow-up study (87), improvement in posterobasal segment function was associated with a decreased incidence of significant MR.

Although Tcheng et al. (82) reported no benefit with PTCA in a high risk group of patients, anecdotal resolution of acute MR after PTCA has been documented (84,86–89).

Percutaneous Transluminal Coronary Angioplasty

Primary PTCA. PTCA is an attractive reperfusion strategy relative to thrombolysis because of high patency rates, excellent restoration of normal coronary flow characteristics, absence of contraindications, decreased recurrent ischemic events and freedom from intracerebral hemorrhage (90). Patency rates do not appear to be influenced by arterial distribution (91), although as with thrombolytic therapy, reocclusion rates may be slightly higher in inferior MI (91). Transient hypotension or bradycardia, or both, at the time of reperfusion (the Bezold-Jarish reflex) can occur during RCA reperfusion. Whereas atropine is often unsuccessful in overcoming this reflex, an intravenous injection of 1 to 2 mg of metaraminal quickly restores hemodynamic stability.

The major limitation of direct PTCA is immediate access to an excellent cardiac catheterization laboratory with expert interventionalists. Although it can be claimed that PTCA in anterior MI produces lower mortality rates than thrombolysis, no survival benefit has been seen in inferior MI (92). However, rapid hemodynamic improvement has been noted after successful PTCA in patients with RV MI (93), and arrhythmias and AV block frequently resolve (94). PTCA appears to be superior to thrombolysis in patients with cardiogenic shock (44).

Rescue PTCA. Lower success rates and higher reocclusion rates result from rescue PTCA than with primary PTCA (95). This finding undoubtedly is due to more complicated index lesions in which thrombolysis has already failed. An increased incidence of ventricular fibrillation, AV block and bradycardia/hypotension complicate procedures in the RCA but can easily be treated if the interventionalist is prepared to address them quickly (96,97). There are no data suggesting that mortality rates are reduced by rescue PTCA in inferior MI. In fact, the Randomized Evaluation of Salvage Angioplasty With Combined Utilization of Endpoints (RESCUE-I) trial (98) enrolled only patients with a moderate or large anterior MI in an attempt to perform a positive study.

Conclusions

Thrombolytic therapy reduces infarct size, preserves left ventricular function and decreases mortality in patients with an inferior MI. The ECG can be used to stratify patients into high risk (precordial ST segment depression, third-degree AV block, RV MI) or low risk (isolated ST segment elevation in leads II, III, aVF) subsets. Unfortunately, none of the placebo-controlled trials have analyzed the benefit of thrombolytic therapy in high risk versus low risk subjects. Data from other studies suggest that thrombolytic therapy should be most useful in patients at higher clinical risk and with larger potential infarct size (precordial ST segment depression [99], ST segment elevation in more than three leads [100], RV MI), but it is not clear that there is therapeutic benefit in patients with isolated ST segment elevation in leads II, III and aVF or symptoms >6 h.

Whereas it is arguable whether PTCA is superior to thrombolytic therapy in anterior MI, there are no mortality data to support using PTCA as a primary or rescue reperfusion strategy in inferior MI instead of thrombolytic therapy, unless

thrombolytic contraindications are present or the patient is in cardiogenic shock or perhaps congestive heart failure.

The GUSTO-I trial (101) demonstrated a superior survival benefit for alteplase compared with SK that was more important for anterior MI (19 lives saved/1,000 patients) than inferior MI (6 lives saved/1,000 patients). Assuming that patients with high risk or a complicated inferior MI have a mortality rate more similar to those with an anterior MI, alteplase would seem to be the favored thrombolytic strategy. Conversely, because mortality rates with uncomplicated inferior MI are low, it may not matter which thrombolytic agent is used from a mortality standpoint, although earlier reperfusion with alteplase would more likely salvage ischemic myocardium. Although thrombolytic therapy for patients with a low risk inferior MI may decrease infarct size and preserve left ventricular function, it may not decrease hospital mortality rates because the baseline risk is only 2% to 4%. With the risk of intracerebral hemorrhage constant and increasing scrutiny directed toward therapeutic cost-effectiveness, the risk/benefit (102) and cost/benefit ratios of administering thrombolytic therapy to low risk patients with inferior MI should be revisited.

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