Reperfusion after acute ST-segment elevation myocardial infarction (AMI) remains the cornerstone of the initial therapy. Although reperfusion can be accomplished with either primary angioplasty or fibrinolytic therapy, clinical trial data reveal improved outcome with primary angioplasty. Furthermore, primary angioplasty with coronary stenting results in less repeat revascularization than balloon angioplasty. Despite the advantages of mechanical revascularization, primarily with stenting, the majority of patients presenting with AMI will receive pharmacological reperfusion.

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How then should patients with AMI who are initially treated medically subsequently be managed? Medical therapy with platelet inhibition, β-blockade, ACE inhibition, and lipid lowering can all be accomplished according to guidelines. However, what is the role of revascularization after the most acute period? In patients with recurrent ischemia or severe multivessel disease with left ventricular dysfunction, the role of revascularization is relatively clear. However, is there a place for routine percutaneous revascularization (percutaneous coronary intervention [PCI]) in stable, post-AMI patients with 1-vessel disease?

To address this question, Zeymer et al.1 randomized 300 patients with 1-vessel disease 1 to 6 weeks after AMI to angioplasty or medical therapy between 1994 and 1997. Fibrinolytic therapy was used in 180 patients (60%). All patients had significant stenosis or total occlusion (29%) of the infarct-related artery (IRA) and no or mild angina. All but 11 (7.4%) patients in the invasive arm underwent angioplasty, but only 17% received stents. One-year mortality was 0.7% with angioplasty versus 3.3% with medical therapy (P = 0.21), and freedom from death, MI, revascularization, or readmission for severe angina was 90% for angioplasty versus 82% for medical therapy (P = 0.066). At 56 months, mortality was 4.0% with angioplasty versus 11.2% with medical therapy (P = 0.058), and survival free of reinfarction or revascularization was 80% in the interventional group and 66% in the conservative group (P = 0.05). Event rate reduction with angioplasty was similar in patients treated with and without fibrinolysis. Importantly, no reduction in events was noted in patients with totally occluded IRA, although this subgroup was small and underpowered.

This trial is the first to show a trend toward improved long-term outcome with angioplasty in the subacute post-AMI phase for patients who largely do not have an occluded IRA. Furthermore, the study addresses the question in an appropriate group of post-AMI patients, those with 1-vessel disease and mild or no symptoms. However, multiple questions remain. The study reveals a trend toward improved survival, which should be borne out by a larger study. Also, the study was performed largely with balloon angioplasty and is somewhat outdated. Although an ECG stress test before randomization was recommended, it may not have been sufficiently sensitive to evaluate viable myocardium in the infarct region, which may improve in function with revascularization. Furthermore, medical therapy was not well defined. Whether aggressive medical therapy, including statins, ACE inhibitors, and combination antiplatelet therapy would have influenced outcome, is unknown. Most first-year events were rehospitalizations and reinterventions in this unblinded study, which being operator dependent may bias against conservative therapy. There are also questions about subgroups. The data do suggest that late angioplasty may not be beneficial for occluded IRA, the more classic question of the late open artery hypothesis. Although there does not seem to be a differential benefit in patients who did or did not receive fibrinolysis, the study is not sufficiently powered to answer this question. Furthermore, the data do not address concerns about the appropriateness of angioplasty in higher-risk subgroups such as the elderly. Another major concern is generalizability, as outcome with angioplasty was remarkably good, with total mortality just 4% at 4.5 years. Such good results may not be found in the wider community.

Several studies evaluating immediate angioplasty after fibrinolytic therapy failed to show benefit. This approach has largely been abandoned except for failed reperfusion. Topol et al.2 compared immediate coronary angioplasty with subacute elective angioplasty in 197 patients with AMI after reperfusion with recombinant tissue plasminogen activator (rTPA) and found no significant differences in incidence of reocclusion or improvement in left ventricular function. In a larger trial from the Thrombolysis in Myocardial Infarction (TIMI) group,3 3262 patients with AMI treated with rTPA were randomized to a routine invasive strategy 18 to 48 hours after randomization or a conservative strategy with arteriography and angioplasty only for spontaneous or exercise-induced ischemia. Death or reinfarction within 42 days occurred in 10.9% of the invasive group and 9.7% of the conservative group (P = NS). There were no significant differences between the 2 groups in rest or exercise ejection fraction.

The opinions expressed in this editorial are not necessarily those of the editors or of the American Heart Association.

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fraction (EF) up to 6 weeks. Simoons et al4 randomized 367 patients with AMI treated with rTPA to an invasive strategy with angioplasty or a noninvasive strategy and noted the clinical course to be more favorable in the noninvasive group. Furthermore, no difference in EF was noted at 10 to 22 days. In the SWIFT trial, 800 patients treated with reperfusion therapy after AMI were randomized to angiography plus revascularization as appropriate or to a conservative strategy.5 Mortality (5.8% in the intervention group and 5.0% in the conservative group) and reinfarction (15.1% and 12.9%) at 12 months were similar in the 2 groups, with no significant differences in angina or EF at follow-up. Although these data are quite consistent, the trials are relatively old, and none included coronary stents. Recent studies suggest that early PCI with stenting after combination therapy with low-dose fibrinolytic therapy and glycoprotein IIb/IIIa inhibitor may be safe and effective in patients with AMI.6 Clinical trials are ongoing to evaluate the efficacy of combination pharmacological therapies that can facilitate PCI in AMI.

The role of subacute angioplasty in patients with AMI without an occluded IRA has been addressed. In a small study, Dakik et al7 randomized 44 stable post-AMI patients with inducible ischemia on a stress test to intensive medical therapy or angioplasty. Stress-induced myocardial ischemia, quantified by nuclear scan 4.5 and 43 days after AMI, was comparably reduced in both groups. Ellis et al8 randomized 87 patients after fibrinolytic therapy with no postinfarct angina and a negative functional test to angioplasty 4 to 14 days after AMI or medical therapy. There was no significant difference in the 6-week resting or exercise EF, and 1-year infarct-free survival was 97.8% in the medical group and 90.5% in the angioplasty group (P = 0.07). The largest trial to consider revascularization in AMI patients with inducible ischemia after fibrinolysis was the DANish trial in Acute Myocardial Infarction (DANAMI) study,9 with 1008 patients randomized to invasive versus conservative strategy. In the invasive arm, 52.9% and 29.2% underwent angioplasty and CABG 2 to 10 weeks after AMI. In the conservative group, only 1.6% were revascularized at 2 months. At 2.4 years follow-up, mortality was 3.6% in the invasive group and 4.4% in the conservative group (P = NS). However, the invasive group had a lower incidence of recurrent MI (5.6% versus 10.5%; P = 0.0038) and admission for unstable angina (17.9% versus 29.5%; P < 0.00001). There was less stable angina at 1 year in the invasive group (21% versus 43%). The study by Zeymer et al10 differs from DANAMI in that all patients had 1- vessel disease, and the majority were without ischemia by stress test; and thus the Zeymer et al10 study more specifically addresses the variant of the late open artery hypothesis in which the IRA is either occluded or has severe residual stenosis.

Routine subacute angioplasty for occluded IRA remains an open question. Sadanandan et al11 reviewed 4 small trials of angioplasty for subacute post-AMI arteries and found the data inconclusive.11-14 Horie et al11 randomized 83 patients with a persistently occluded IRA > 24 hours after a Q-wave anterior AMI to angioplasty or medical therapy. The end-systolic and end-diastolic volume indices were significantly smaller in the angioplasty compared with the medical therapy group at 6 months, and event-free survival was better in the angioplasty group at 5 years. Topol et al12 randomized 71 patients with occluded IRA 12 to 48 hours after AMI to angioplasty or medical therapy. Angioplasty was associated with improved ventricular function at 1 month, but no long-term benefit was noted. Dzavik et al13 randomized 44 patients with an occluded IRA to angioplasty or medical therapy. The initial angioplasty success rate was only 72%, and reocclusion further reduced the patency rates at 4 months (43%). Although by treatment assignment there was no difference in EF, patients with a patent IRA during follow-up had a greater increase in EF. Recently, Yousef et al14 randomized 66 patients with isolated persistent occlusion of the left anterior descending coronary artery to PCI with stent 26 days after AMI or medical therapy. At 12 months, the stent group had worse left ventricular end-systolic and end-diastolic volumes and a higher combined clinical event rate than the medical group. Thus, the available data on routine PCI of an occluded IRA late post-AMI are limited and conflicting. Patients with persistently occluded IRA may be different from those who have patent IRA late after AMI. They may have an altered thrombotic milieu,15 ACE gene polymorphism,16 or severely impaired microvascular perfusion preventing sustained patency. A persistently occluded IRA late after AMI may be a marker for characteristics that explain the long-term adverse outcomes. An absence of beneficial effects of angioplasty in the subgroup of patients with occluded IRA in the present study further supports the need for a large study, which is currently being addressed in the Occluded Artery Trial (OAT).

The current study by Zeymer et al1 thus brings to clinical attention a question of pathophysiologic interest and clinical relevance. These results do suggest that subacute angioplasty, or in the current era coronary stenting, is appropriate for at least some stable patients after AMI, probably including patients who may not have received fibrinolytic therapy. Validation of these findings and proper selection of patients who may derive benefit from such a strategy will require additional, larger studies. If this study is confirmed, it will remain of interest to determine the mechanism of benefit, whether by relieving ischemia, preserving viable myocardium, decreasing remodeling, or improving electrical stability.

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Percutaneous Coronary Intervention in Stable Patients After Acute Myocardial Infarction
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