ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction—Executive Summary

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infarction)

Developed in Collaboration With the Canadian Cardiovascular Society

WRITING COMMITTEE MEMBERS

Elliott M. Antman, MD, FACC, FAHA, Chair; Daniel T. Anbe, MD, FACC, FAHA; Paul Wayne Armstrong, MD, FACC, FAHA; Eric R. Bates, MD, FACC, FAHA; Lee A. Green, MD, MPH; Mary Hand, MSPH, RN, FAHA; Judith S. Hochman, MD, FACC, FAHA; Harlan M. Krumholz, MD, FACC, FAHA; Frederick G. Kushner, MD, FACC, FAHA; Gervasio A. Lamas, MD, FACC; Charles J. Mullany, MB, MS, FACC; Joseph P. Ornato, MD, FACC, FAHA; David L. Pearle, MD, FACC, FAHA; Michael A. Sloan, MD, FACC; Sidney C. Smith, Jr, MD, FACC, FAHA

TASK FORCE MEMBERS

Elliott M. Antman, MD, FACC, FAHA, Chair; Sidney C. Smith, Jr, MD, FACC, FAHA, Vice-chair; Joseph S. Alpert, MD, FACC, FAHA*; Jeffrey L. Anderson, MD, FACC, FAHA; David P. Faxon, MD, FACC, FAHA; Valentin Fuster, MD, PhD, FACC, FAHA; Raymond J. Gibbons, MD, FACC, FAHA*†; Gabriel Gregoratos, MD, FACC, FAHA*; Jonathan L. Halperin, MD, FACC, FAHA; Loren F. Hiratzka, MD, FACC, FAHA; Sharon Ann Hunt, MD, FACC, FAHA; Alice K. Jacobs, MD, FACC, FAHA; Joseph P. Ornato, MD, FACC, FAHA

This document was approved by the American College of Cardiology Foundation Board of Trustees on May 7, 2004 and by the American Heart Association Science Advisory and Coordinating Committee on May 5, 2004.

The ACC/AHA Task Force on Practice Guidelines makes every effort to avoid any actual or potential conflicts of interest that might arise as a result of an outside relationship or personal interest of a member of the writing panel. Specifically, all members of the writing panel are asked to provide disclosure statements of all such relationships that might be perceived as real or potential conflicts of interest. These statements are reviewed by the parent task force, reported orally to all members of the writing panel at the first meeting, and updated as changes occur. The relationship with industry information for the writing committee members is posted on the ACC and AHA World Wide Web sites with the full-length version of the update, along with the names and relationships with industry of the peer reviewers.

When citing this document, the American College of Cardiology Foundation and the American Heart Association would appreciate the following citation format: Antman EM, Anbe DT, Armstrong PW, Bates ER, Green LA, Hand M, Hochman JS, Krumholz HM, Kushner FG, Lamas GA, Mullany CJ, Ornato JP, Pearle DL, Sloan MA, Smith SC Jr. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: executive summary: a report of the ACC/AHA Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines on the Management of Patients With Acute Myocardial Infarction). *Circulation*. 2004;110:588–636.

Copies: This document and the full-text guideline are available on the World Wide Web sites of the American College of Cardiology (www.acc.org), the American Heart Association (www.americanheart.org), and the Canadian Cardiovascular Society (www.ccs.ca). Single copies of this executive summary, published in the August 4, 2004 issue of the *Journal of the American College of Cardiology* or the August 3, 2004 issue of *Circulation* or the companion full-text guideline are available for $10.00 each by calling 1-800-253-4636 or writing to the American College of Cardiology Foundation, Resource Center, 9111 Old Georgetown Road, Bethesda, MD 20814-1699. To purchase bulk reprints (specify version and reprint number: 71-0294 for the executive summary; 71-0293 for the full-text guideline): up to 999 copies, call 1-800-611-6083 (US only) or fax 413-665-2671; 1000 or more copies, call 214-706-1789, fax 214-691-6342, or e-mail pubauth@heart.org.

*Former Task Force member.

†Immediate Past Chair.

(*Circulation*. 2004;110:588-636.)

***Circulation* is available at http://www.circulationaha.org** **DOI: 10.1161/01.CIR.0000134791.68010.FA**

Table of Contents

I. Introduction

Although considerable improvement has occurred in the process of care for patients with ST-elevation myocardial infarction (STEMI), room for improvement exists.[1–3] The purpose of the present guideline is to focus on the numerous advances in the diagnosis and management of patients with STEMI since 1999. This is reflected in the changed name of the guideline: "ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction." The final recommendations for indications for a diagnostic procedure, a particular therapy, or an intervention in patients with STEMI summarize both clinical evidence and expert opinion (Table 1). To provide clinicians with a set of recommendations that can easily be translated into the practice of caring for patients with STEMI, this guideline is organized around the chronology of the interface between the patient and the clinician. The full guideline is available at http://www.acc.org/clinical/guidelines/stemi/index.htm.

II. Pathology

A. Epidemiology

STEMI continues to be a significant public health problem in industrialized countries and is becoming an increasingly significant problem in developing countries.[4] Although the exact incidence is difficult to ascertain, using first-listed and secondary hospital discharge data, there were 1 680 000 unique discharges for ACS in 2001.[5] Applying the conservative estimate of 30% of the ACS patients who have STEMI from the National Registry of Myocardial Infarction-4 [NRMI-4],[5a] we estimate 500 000 STEMI events per year in the U.S. This writing committee strongly endorses several public health campaigns that are likely to contribute to a reduction in the incidence of and fatality from STEMI in the future and additional research of new strategies for the management of STEMI patients in the community.[6–13]

III. Management Before STEMI

A. Identification of Patients at Risk of STEMI

Class I

1. **Primary care providers should evaluate the presence and status of control of major risk factors for coronary heart disease (CHD) for all patients at regular intervals (approximately every 3 to 5 years). (*Level of Evidence: C*)**
2. **Ten-year risk (National Cholesterol Education Program [NCEP] global risk) of developing symptomatic CHD should be calculated for all patients who have 2 or more major risk factors to assess the need for primary prevention strategies.[14] (*Level of Evidence: B*)**
3. **Patients with established CHD should be identified for secondary prevention, and patients with a CHD risk equivalent (eg, diabetes mellitus, chronic kidney disease, or 10-year risk greater than 20% as calculated by Framingham equations) should receive equally intensive risk factor intervention as those with clinically apparent CHD. (*Level of Evidence: A*)**

B. Patient Education for Early Recognition and Response to STEMI

Class I

1. **Patients with symptoms of STEMI (chest discomfort with or without radiation to the arms[s], back, neck, jaw, or epigastrium; shortness of breath; weakness; diaphoresis; nausea; lightheadedness) should be transported to the hospital by ambulance rather than by friends or relatives. (*Level of Evidence: B*)**
2. **Healthcare providers should actively address the following issues regarding STEMI with patients and their families:**
   a. **The patient's heart attack risk (*Level of Evidence: C*)**
   b. **How to recognize symptoms of STEMI (*Level of Evidence: C*)**
   c. **The advisability of calling 9-1-1 if symptoms are unimproved or worsening after 5 minutes, despite feelings of uncertainty about the symptoms and**

TABLE 1. Applying Classification of Recommendations and Level of Evidence

"Estimate of Certainty (Precision) of Treatment of Effect"	"Size of Treatment Effect"			
	Class I	Class IIa	Class IIb	Class III
	Benefit >>> Risk Procedure/Treatment SHOULD be performed/administered	*Benefit >> Risk* *Additional studies with focused objectives needed* IT IS REASONABLE to perform procedure/administer treatment	*Benefit ≥ Risk* *Additional studies with broad objectives needed; additional registry data would be helpful* Procedure/Treatment MAY BE CONSIDERED	*Risk ≥ Benefit* *No additional studies needed* Procedure/Treatment should NOT be performed/administered SINCE IT IS NOT HELPFUL AND MAY BE HARMFUL
Level A *Multiple (3–5) population risk strata evaluated* General consistency of direction and magnitude of effect*	• Recommendation that procedure or treatment is useful/effective • Sufficient evidence from multiple randomized trials or meta-analyses	• Recommendation in favor of treatment or procedure being useful/effective • Some conflicting evidence from multiple randomized trials or meta-analyses	• Recommendation's usefulness/efficacy less well established • Greater conflicting evidence from multiple randomized trials or meta-analyses	• Recommendation that procedure or treatment is not useful/effective and may be harmful • Sufficient evidence from multiple randomized trials or meta-analyses
Level B *Limited (2–3) population risk strata evaluated**	• Recommendation that procedure or treatment is useful/effective • Limited evidence from single randomized trial or nonrandomized studies	• Recommendation in favor of treatment or procedure being useful/effective • Some conflicting evidence from single randomized trial or nonrandomized studies	• Recommendation's usefulness/efficacy less well established • Greater conflicting evidence from single randomized trial or nonrandomized studies	• Recommendation that procedure or treatment is not useful/effective and may be harmful • Limited evidence from single randomized trial or nonrandomized studies
Level C *Very limited (1–2) population risk strata evaluated**	• Recommendation that procedure or treatment is useful/effective • Only expert opinion, case studies, or standard-of-care	• Recommendation in favor of treatment or procedure being useful/effective • Only diverging expert opinion, case studies, or standard-of-care	• Recommendation's usefulness/efficacy less well established • Only diverging expert opinion, case studies, or standard-of-care	• Recommendation that procedure or treatment is not useful/effective and may be harmful • Only expert opinion, case studies, or standard-of-care
Suggested phrases for writing recommendations†	should is recommended is indicated is useful/effective/beneficial	is reasonable can be useful/effective/beneficial is probably recommended or indicated	may/might be considered may/might be reasonable usefulness/effectiveness is unknown/unclear/uncertain or not well established	is not recommended is not indicated should not is not useful/effective/beneficial may be harmful

*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations, such as gender, age, history of diabetes, history of prior MI, history of heart failure, and prior aspirin use.

†The ACC/AHA Task Force on Practice Guidelines recently provided a list of suggested phrases to use when writing recommendations. All recommendations in the STEMI guideline have been written in full sentences that express a complete thought, such that a recommendation, even if separated and presented apart from the rest of the document (including headings above sets of recommendations), would still convey the full intent of the recommendation. It is hoped that this will increase readers' comprehension of the guidelines and will allow queries at the individual recommendation level.

fear of potential embarrassment (*Level of Evidence: C*)

d. A plan for appropriate recognition and response to a potential acute cardiac event that includes the phone number to access emergency medical services (EMS), generally 9-1-1.[15] (*Level of Evidence: C*)

3. Healthcare providers should instruct patients for whom nitroglycerin has been prescribed previously to take ONE nitroglycerin dose sublingually in response to chest discomfort/pain. If chest discomfort/pain is unimproved or worsening 5 minutes after 1 sublingual nitroglycerin dose has been taken, it is recommended that the patient or family member/friend call 9-1-1 immediately to access EMS. (*Level of Evidence: C*)

Morbidity and mortality due to STEMI can be reduced significantly if patients and bystanders recognize symptoms early, activate the EMS system, and thereby shorten the time to definitive treatment. Patients with possible symptoms of STEMI should be transported to the hospital by ambulance rather than by friends or relatives because there is a significant association between arrival at the emergency department (ED) by ambulance and early reperfusion therapy.[16–19] Although the traditional recommendation is for patients to take 1 nitroglycerin dose sublingually, 5 minutes apart, for up to 3 doses before

calling for emergency evaluation, this recommendation has been modified by the writing committee to encourage earlier contacting of EMS by patients with symptoms suggestive of STEMI.[20,21]

IV. Onset of STEMI

A. Out-of-Hospital Cardiac Arrest

Class I

1. **All communities should create and maintain a strong "Chain of Survival" for out-of-hospital cardiac arrest that includes early access (recognition of the problem and activation of the EMS system by a bystander), early cardiopulmonary resuscitation (CPR), early defibrillation for patients who need it, and early advanced cardiac life support (ACLS). *(Level of Evidence: C*)**
2. **Family members of patients experiencing STEMI should be advised to take CPR training and familiarize themselves with the use of an automated external defibrillator (AED). In addition, they should be referred to a CPR training program that has a social support component for family members of post-STEMI patients. (*Level of Evidence: B*)**

The links in the chain include early access (recognition of the problem and activation of the EMS system by a bystander), early CPR, early defibrillation for patients who need it, and early ACLS.

V. Prehospital Issues

A. Emergency Medical Services Systems

Class I

1. **All EMS first responders who respond to patients with chest pain and/or suspected cardiac arrest should be trained and equipped to provide early defibrillation. (*Level of Evidence: A*)**
2. **All public safety first responders who respond to patients with chest pain and/or suspected cardiac arrest should be trained and equipped to provide early defibrillation with AEDs. (Provision of early defibrillation with AEDs by nonpublic safety first responders is a promising new strategy, but further study is needed to determine its safety and efficacy.) (*Level of Evidence: B*)**
3. **Dispatchers staffing 9-1-1 center emergency medical calls should have medical training, should use nationally developed and maintained protocols, and should have a quality-improvement system in place to ensure compliance with protocols. (*Level of Evidence: C*)**

Early access to EMS is promoted by a 9-1-1 system currently available to more than 90% of the US population. To minimize time to treatment, particularly for cardiopulmonary arrest, many communities allow volunteer and/or paid firefighters and other first-aid providers to function as first responders, providing CPR and, increasingly, early defibrillation using automated external defibrillators (AEDs) until emergency medical technicians and paramedics arrive. Most cities and larger suburban areas provide EMS ambulance services with providers from the fire department, a private ambulance company, and/or volunteers.

B. Prehospital Chest Pain Evaluation and Treatment

Class I

1. **Prehospital EMS providers should administer 162 to 325 mg of aspirin (chewed) to chest pain patients suspected of having STEMI unless contraindicated or already taken by patient. Although some trials have used enteric-coated aspirin for initial dosing, more rapid buccal absorption occurs with non–enteric-coated formulations. (*Level of Evidence: C*)**

Class IIa

1. **It is reasonable for all 9-1-1 dispatchers to advise patients without a history of aspirin allergy who have symptoms of STEMI to chew aspirin (162 to 325 mg) while awaiting arrival of prehospital EMS providers. Although some trials have used enteric-coated aspirin for initial dosing, more rapid buccal absorption occurs with non–enteric-coated formulations. *(Level of Evidence: C*)**
2. **It is reasonable that all ACLS providers perform and evaluate 12-lead electrocardiograms (ECGs) routinely on chest pain patients suspected of STEMI. *(Level of Evidence: B)***
3. **If the ECG shows evidence of STEMI, it is reasonable that prehospital ACLS providers review a reperfusion "checklist" and relay the ECG and checklist findings to a predetermined medical control facility and/or receiving hospital. (*Level of Evidence: C*)**

It is reasonable for physicians to encourage the prehospital administration of aspirin via EMS personnel (ie, EMS dispatchers and providers) in patients with symptoms suggestive of STEMI unless its use is contraindicated.[22] For patients who have ECG evidence of STEMI, it is reasonable that paramedics review a reperfusion checklist and relay the ECG and checklist findings to a predetermined medical control facility and/or receiving hospital.

C. Prehospital Fibrinolysis

Class IIa

1. **Establishment of a prehospital fibrinolysis protocol is reasonable in 1) settings in which physicians are present in the ambulance or in 2) well-organized EMS systems with full-time paramedics who have 12-lead ECGs in the field with transmission capability, paramedic initial and ongoing training in ECG interpretation and STEMI treatment, online medical command, a medical director with training/experience in STEMI management, and an ongoing continuous quality-improvement program. (*Level of Evidence: B*)**

Randomized controlled trials of fibrinolytic therapy have demonstrated the benefit of initiating fibrinolytic therapy as early as possible after onset of ischemic-type chest discomfort (Figure 1).[23–25] It appears reasonable to expect that if

fibrinolytic therapy could be started at the time of prehospital evaluation, a greater number of lives could be saved. Prehospital fibrinolysis is reasonable in those settings in which physicians are present in the ambulance or prehospital transport times are more than 60 minutes in high-volume (more than 25,000 runs per year) EMS systems.[26] Other considerations for implementing a prehospital fibrinolytic service include the ability to transmit ECGs, paramedic initial and ongoing training in ECG interpretation and myocardial infarction (MI) treatment, online medical command, a medical director with training/experience in management of STEMI, and full-time paramedics.[27]

D. Prehospital Destination Protocols

Class I

1. **Patients with STEMI who have cardiogenic shock and are less than 75 years of age should be brought immediately or secondarily transferred to facilities capable of cardiac catheterization and rapid revascularization (percutaneous coronary intervention [PCI] or coronary artery bypass graft surgery [CABG]) if it can be performed within 18 hours of onset of shock. (*Level of Evidence: A*)**
2. **Patients with STEMI who have contraindications to fibrinolytic therapy should be brought immediately or secondarily transferred promptly (ie, primary-receiving hospital door-to-departure time less than 30 minutes) to facilities capable of cardiac catheterization and rapid revascularization (PCI or CABG). (*Level of Evidence: B*)**
3. **Every community should have a written protocol that guides EMS system personnel in determining where to take patients with suspected or confirmed STEMI. (*Level of Evidence: C*)**

Class IIa

1. **It is reasonable that patients with STEMI who have cardiogenic shock and are 75 years of age or older be considered for immediate or prompt secondary transfer to facilities capable of cardiac catheterization and rapid revascularization (PCI or CABG) if it can be performed within 18 hours of onset of shock. (*Level of Evidence: B*)**
2. **It is reasonable that patients with STEMI who are at especially high risk of dying, including those with severe congestive heart failure (CHF), be considered for immediate or prompt secondary transfer (ie, primary-receiving hospital door-to-departure time less than 30 minutes) to facilities capable of cardiac catheterization and rapid revascularization (PCI or CABG). (*Level of Evidence: B*)**

Every community should have a written protocol that guides EMS system personnel in determining where to take patients with suspected or confirmed STEMI. Active involvement of local healthcare providers, particularly cardiologists and emergency physicians, is needed to formulate local EMS destination protocols for these patients. In general, patients with suspected STEMI should be taken to the nearest appropriate hospital. However, patients with STEMI and shock are an exception to this general rule. Whenever possible, STEMI patients less than 75 years of age with shock should be transferred to facilities capable of cardiac catheterization and rapid revascularization (PCI or CABG). On the basis of observations in the SHOCK Trial Registry and other registries, it is reasonable to extend such considerations of transfer to invasive centers for elderly patients with shock (see VII.F.5 and Section 7.6.5 of the full-text guidelines). Patients with STEMI who have contraindications to fibrinolytic therapy should be brought immediately or secondarily transferred promptly (ie, primary-receiving hospital door-to-departure time less than 30 minutes) to facilities capable of cardiac catheterization and rapid revascularization (PCI or CABG).

VI. Initial Recognition and Management in the Emergency Department

A. Optimal Strategies for Emergency Department Triage

Class I

1. **Hospitals should establish multidisciplinary teams (including primary care physicians, emergency medicine physicians, cardiologists, nurses, and laboratorians) to develop guideline-based, institution-specific written protocols for triaging and managing patients who are seen in the prehospital setting or present to the ED with symptoms suggestive of STEMI. (*Level of Evidence: B*)**

B. Initial Patient Evaluation

Class I

1. **The delay from patient contact with the healthcare system (typically, arrival at the ED or contact with paramedics) to initiation of fibrinolytic therapy should be less than 30 minutes. Alternatively, if PCI is chosen, the delay from patient contact with the healthcare system (typically, arrival at the ED or contact with paramedics) to balloon inflation should be less than 90 minutes. (*Level of Evidence: B*)**
2. **The choice of initial STEMI treatment should be made by the emergency medicine physician on duty based on a predetermined, institution-specific, written protocol that is a collaborative effort of cardiologists (both those involved in coronary care unit management and interventionalists), emergency physicians, primary care physicians, nurses, and other appropriate personnel. For cases in which the initial diagnosis and treatment plan is unclear to the emergency physician or is not covered directly by the agreed-on protocol, immediate cardiology consultation is advisable. (*Level of Evidence: C*)**

Regardless of the approach used, all patients presenting to the ED with chest discomfort or other symptoms suggestive of STEMI or unstable angina should be considered high-priority triage cases and should be evaluated and treated based on a predetermined, institution-specific chest pain protocol. The goal for patients with STEMI should be to

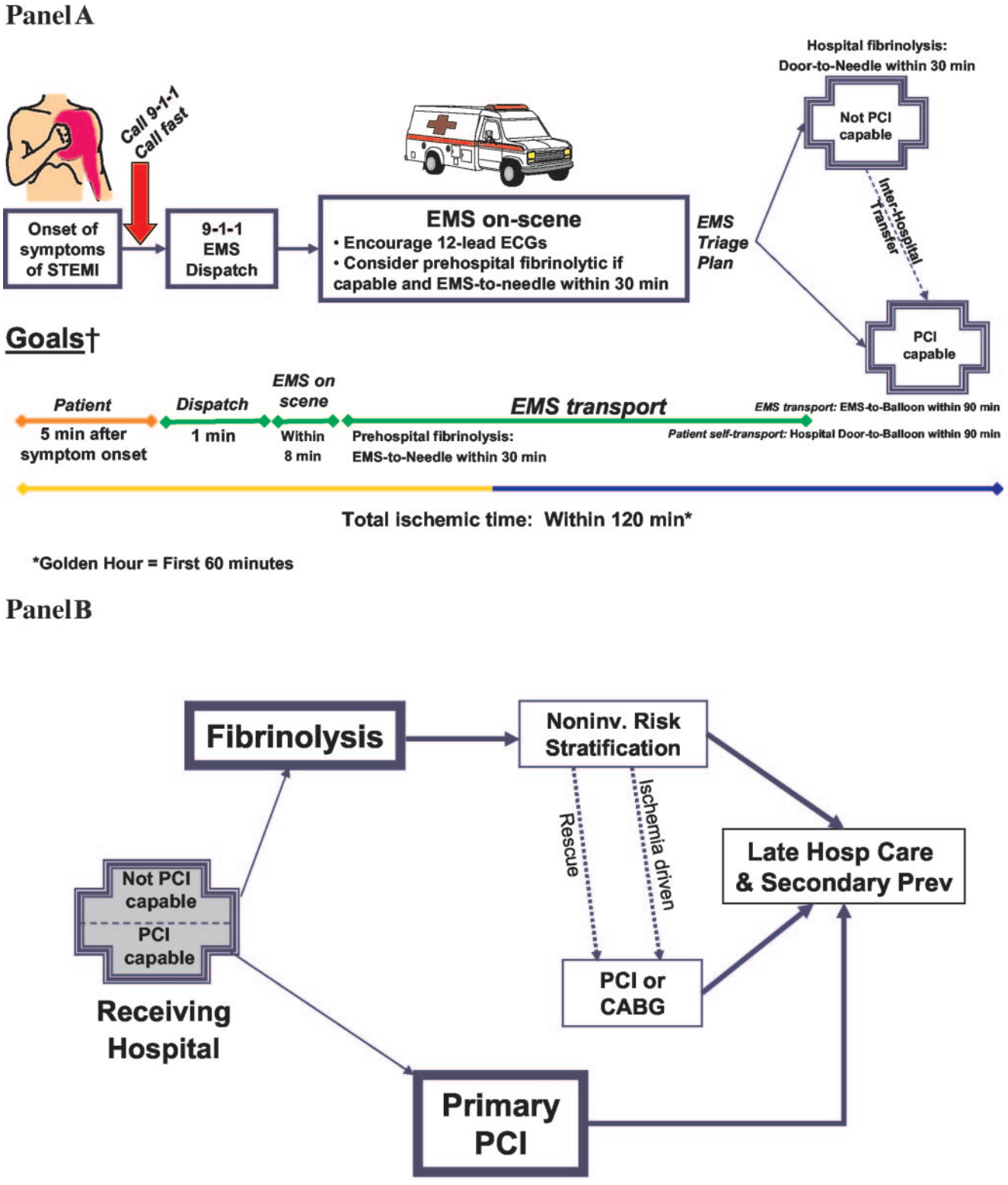

Figure 1. Options for transportation of STEMI patients and initial reperfusion treatment. Panel A, Patient transported by EMS after calling 9-1-1: Reperfusion in patients with STEMI can be accomplished by the pharmacological (fibrinolysis) or catheter-based (primary PCI) approaches. Implementation of these strategies varies based on the mode of transportation of the patient and capabilities at the receiving hospital. Transport time to the hospital is variable from case to case, but the goal is to keep total ischemic time within 120 minutes. There are 3 possibilities: (1) If EMS has fibrinolytic capability and the patient qualifies for therapy, prehospital fibrinolysis should be started within 30 minutes of EMS arrival on scene. (2) If EMS is not capable of administering prehospital fibrinolysis and the patient is transported to a non–PCI-capable hospital, the hospital door-to-needle time should be within 30 minutes for patients in whom fibrinolysis is indicated. (3) If EMS is not capable of administering prehospital fibrinolysis and the patient is transported to a PCI-capable hospital, the hospital door-to-balloon time should be within 90 minutes. Interhospital transfer: It is also appropriate to consider emergency interhospital transfer of the patient to a PCI-capable hospital for mechanical revascularization if (1) there is a contraindication to fibrinolysis; (2) PCI can be initiated promptly

achieve a door-to-needle time within 30 minutes and a door-to-balloon time within 90 minutes (Figure 1).[25]

1. History

Class I

1. **The targeted history of STEMI patients taken in the ED should ascertain whether the patient has had prior episodes of myocardial ischemia such as stable or unstable angina, MI, CABG, or PCI. Evaluation of the patient's complaints should focus on chest discomfort, associated symptoms, sex- and age-related differences in presentation, hypertension, diabetes mellitus, possibility of aortic dissection, risk of bleeding, and clinical cerebrovascular disease (amaurosis fugax, face/limb weakness or clumsiness, face/limb numbness or sensory loss, ataxia, or vertigo). (*Level of Evidence: C*)**

2. Physical Examination

Class I

1. **A physical examination should be performed to aid in the diagnosis and assessment of the extent, location, and presence of complications of STEMI. (*Level of Evidence: C*)**
2. **A brief, focused, and limited neurological examination to look for evidence of prior stroke or cognitive deficits should be performed on STEMI patients before administration of fibrinolytic therapy. (*Level of Evidence: C*)**

A brief physical examination may promote rapid triage, whereas a more detailed physical examination aids in the differential diagnosis and is useful for assessing the extent, location, and presence of complications of STEMI.

Figure 1 (continued). (within 90 minutes after the patient presented to the initial receiving hospital or within 60 minutes compared to when fibrinolysis with a fibrin-specific agent could be initiated at the initial receiving hospital); or (3) fibrinolysis is administered and is unsuccessful (ie, "rescue PCI"). Secondary nonemergency interhospital transfer can be considered for recurrent ischemia. Patient self-transport: Patient self-transportation is discouraged. If the patient arrives at a non–PCI-capable hospital, the door-to-needle time should be within 30 minutes. If the patient arrives at a PCI-capable hospital, the door-to-balloon time should be within 90 minutes. The treatment options and time recommendations after first hospital arrival are the same. Panel B, For patients who receive fibrinolysis, noninvasive risk stratification is recommended to identify the need for rescue PCI (failed fibrinolysis) or ischemia-driven PCI. See Sections 6.3.1.6.4.5. and 6.3.1.6.7. in the full-text guidelines. Regardless of the initial method of reperfusion treatment, all patients should receive late hospital care and secondary prevention of STEMI. EMS indicates Emergency Medical System; PCI, percutaneous coronary intervention; CABG, coronary artery bypass graft surgery; Hosp, hospital; Noninv., Noninvasive. * Golden hour = First 60 minutes;† The medical system goal is to facilitate rapid recognition and treatment of patients with STEMI such that door-to-needle (or medical contact–to-needle) time for initiation of fibrinolytic therapy is within 30 minutes or that door-to-balloon (or medical contact–to-balloon) time for PCI is within 90 minutes. These goals should not be understood as ideal times but rather as the longest times that should be considered acceptable for a given system. Systems that are able to achieve even more rapid times for treatment of patients with STEMI should be encouraged. Modified with permission from Armstrong et al. *Circulation*. 2003;107:2533–7.[25]

3. Electrocardiogram

Class I

1. **A 12-lead ECG should be performed and shown to an experienced emergency physician within 10 minutes of ED arrival for all patients with chest discomfort (or anginal equivalent) or other symptoms suggestive of STEMI. (*Level of Evidence: C*)**
2. **If the initial ECG is not diagnostic of STEMI but the patient remains symptomatic, and there is a high clinical suspicion for STEMI, serial ECGs at 5- to 10-minute intervals or continuous 12-lead ST-segment monitoring should be performed to detect the potential development of ST elevation. (*Level of Evidence: C*)**
3. **In patients with inferior STEMI, right-sided ECG leads should be obtained to screen for ST elevation suggestive of right ventricular (RV) infarction. (See Section 7.6.6 of the full-text guidelines and the ACC/AHA/ASE 2003 Guideline Update for the Clinical Application of Echocardiography.) (*Level of Evidence: B*)**

The 12-lead ECG in the ED is at the center of the therapeutic decision pathway because of the strong evidence that ST-segment elevation identifies patients who benefit from reperfusion therapy.[28]

4. Laboratory Examinations

Class I

1. **Laboratory examinations should be performed as part of the management of STEMI patients but should not delay the implementation of reperfusion therapy. (*Level of Evidence: C*)**

In addition to serum cardiac biomarkers for cardiac damage, several routine evaluations have important implications for management of patients with STEMI. Although these studies should be ordered when the patient is first seen, therapeutic decisions should not be delayed until results are obtained because of the crucial role of time to therapy in STEMI.

5. Biomarkers of Cardiac Damage

Class I

1. **Cardiac-specific troponins should be used as the optimum biomarkers for the evaluation of patients with STEMI who have coexistent skeletal muscle injury. (*Level of Evidence: C*)**
2. **For patients with ST elevation on the 12-lead ECG and symptoms of STEMI, reperfusion therapy should be initiated as soon as possible and is not contingent on a biomarker assay. (*Level of Evidence: C*)**

Class IIa

1. **Serial biomarker measurements can be useful to provide supportive noninvasive evidence of reperfusion of the infarct artery after fibrinolytic therapy in patients**

not undergoing angiography within the first 24 hours after fibrinolytic therapy. (*Level of Evidence: B*)

Class III

1. Serial biomarker measurements should not be relied on to diagnose reinfarction within the first 18 hours after the onset of STEMI. (*Level of Evidence: C*)

For patients with ST-segment elevation, the diagnosis of STEMI is secure; initiation of reperfusion therapy should not be delayed to wait for the results of a cardiac biomarker assay.[29] Quantitative analysis of cardiac biomarker measurements provides prognostic information and a noninvasive assessment of the likelihood that the patient has undergone successful reperfusion when fibrinolytic therapy is administered.

a. Bedside Testing for Serum Cardiac Biomarkers

Class I

1. Although handheld bedside (point-of-care) assays may be used for a qualitative assessment of the presence of an elevated level of a serum cardiac biomarker, subsequent measurements of cardiac biomarker levels should be performed with a quantitative test. (*Level of Evidence: B*)
2. For patients with ST elevation on the 12-lead ECG and symptoms of STEMI, reperfusion therapy should be initiated as soon as possible and is not contingent on a bedside biomarker assay. (*Level of Evidence: C*)

A positive bedside test should be confirmed by a conventional quantitative test. However, reperfusion therapy should not be delayed to wait for the results of a quantitative assay.

6. Imaging

Class I

1. Patients with STEMI should have a portable chest X-ray, but this should not delay implementation of reperfusion therapy (unless a potential contraindication, such as aortic dissection, is suspected). (*Level of Evidence: C*)
2. Imaging studies such as a high-quality portable chest X-ray, transthoracic and/or transesophageal echocardiography, and a contrast chest computed tomographic scan or a MRI scan should be used to differentiate STEMI from aortic dissection in patients for whom this distinction is initially unclear. (*Level of Evidence: B*)

Class IIa

1. Portable echocardiography is reasonable to clarify the diagnosis of STEMI and allow risk stratification of patients with chest pain on arrival at the ED, especially if the diagnosis of STEMI is confounded by left bundle-branch block (LBBB) or pacing, or there is suspicion of posterior STEMI with anterior ST depressions. (See Section 7.6.7 Mechanical Causes of Heart Failure/Low Output Syndrome of the full-text guidelines.) (*Level of Evidence: B*)

Class III

1. Single-photon emission computed tomography (SPECT) radionuclide imaging should not be performed to diagnose STEMI in patients for whom the diagnosis of STEMI is evident on the ECG. (*Level of Evidence: B*)

C. Management

1. Routine Measures

a. Oxygen

Class I

1. Supplemental oxygen should be administered to patients with arterial oxygen desaturation (SaO_2 less than 90%). (*Level of Evidence: B*)

Class IIa

1. It is reasonable to administer supplemental oxygen to all patients with uncomplicated STEMI during the first 6 hours. (*Level of Evidence: C*)

b. Nitroglycerin

Class I

1. Patients with ongoing ischemic discomfort should receive sublingual nitroglycerin (0.4 mg) every 5 minutes for a total of 3 doses, after which an assessment should be made about the need for intravenous nitroglycerin. (*Level of Evidence: C*)
2. Intravenous nitroglycerin is indicated for relief of ongoing ischemic discomfort, control of hypertension, or management of pulmonary congestion. (*Level of Evidence: C*)

Class III

1. Nitrates should not be administered to patients with systolic blood pressure less than 90 mm Hg or greater than or equal to 30 mm Hg below baseline, severe bradycardia (less than 50 bpm), tachycardia (more than 100 bpm), or suspected RV infarction. (*Level of Evidence: C*)
2. Nitrates should not be administered to patients who have received a phosphodiesterase inhibitor for erectile dysfunction within the last 24 hours (48 hours for tadalafil). (*Level of Evidence: B*)

Nitroglycerin may be administered to relieve ischemic pain and is clearly indicated as a vasodilator in patients with STEMI associated with left ventricular (LV) failure. Nitrates in all forms should be avoided in patients with initial systolic blood pressures less than 90 mm Hg or greater than or equal to 30 mm Hg below baseline, in patients with marked bradycardia or tachycardia,[30] and in patients with known or suspected RV infarction. In view of their marginal treatment benefits, nitrates should not be used if hypotension limits the administration of beta-blockers, which have more powerful salutary effects.

c. Analgesia

Class I

1. Morphine sulfate (2 to 4 mg IV with increments of 2 to 8 mg IV repeated at 5- to 15-minute intervals) is the

analgesic of choice for management of pain associated with STEMI. (*Level of Evidence: C*)

d. Aspirin

Class I

1. Aspirin should be chewed by patients who have not taken aspirin before presentation with STEMI. The initial dose should be 162 mg (*Level of Evidence: A*) to 325 mg *(Level of Evidence: C*). Although some trials have used enteric-coated aspirin for initial dosing, more rapid buccal absorption occurs with non–enteric-coated aspirin formulations.

In a dose of 162 mg or more, aspirin produces a rapid clinical antithrombotic effect caused by immediate and near-total inhibition of thromboxane A2 production. Aspirin now forms part of the early management of all patients with suspected STEMI and should be given promptly, and certainly within the first 24 hours, at a dose between 162 and 325 mg and continued indefinitely at a daily dose of 75 to 162 mg.[31] Although some trials have used enteric-coated aspirin for initial dosing, more rapid buccal absorption occurs with non–enteric-coated formulations.[32]

e. Beta-Blockers

Class I

1. Oral beta-blocker therapy should be administered promptly to those patients without a contraindication, irrespective of concomitant fibrinolytic therapy or performance of primary PCI. (*Level of Evidence: A*)

Class IIa

1. It is reasonable to administer IV beta-blockers promptly to STEMI patients without contraindications, especially if a tachyarrhythmia or hypertension is present. (*Level of Evidence: B*)

Immediate beta-blocker therapy appears to reduce the magnitude of infarction and incidence of associated complications in subjects not receiving concomitant fibrinolytic therapy, the rate of reinfarction in patients receiving fibrinolytic therapy, and the frequency of life-threatening ventricular tachyarrhythmias.

f. Reperfusion

GENERAL CONCEPTS.

Class I

1. All STEMI patients should undergo rapid evaluation for reperfusion therapy and have a reperfusion strategy implemented promptly after contact with the medical system. (*Level of Evidence: A*)

Evidence exists that expeditious restoration of flow in the obstructed infarct artery after the onset of symptoms in STEMI patients is a key determinant of short- and long-term outcomes regardless of whether reperfusion is accomplished by fibrinolysis or PCI.[33–35] As discussed previously (also see Section 4.1 of the full-text guidelines), efforts should be made to shorten the time from recognition of symptoms by the patient to contact with the medical system. All healthcare providers caring for STEMI patients from the point of entry into the medical system must recognize the need for rapid triage and implementation of care in a fashion analogous to the handling of trauma patients. When considering recommendations for timely reperfusion of STEMI patients, the Writing Committee reviewed data from clinical trials, focusing particular attention on enrollment criteria for selection of patients for randomization, actual times reported in the trial report rather than simply the allowable window specified in the trial protocol, treatment effect of the reperfusion strategy on individual components of a composite primary end point (eg, mortality, recurrent nonfatal infarction), ancillary therapies (eg, antithrombin and antiplatelet agents), and the interface between fibrinolysis and referral for angiography and revascularization. When available, data from registries were also reviewed to assess the generalizability of observations from clinical trials of reperfusion to routine practice. Despite the wealth of reports on reperfusion for STEMI, it is not possible to produce a simple algorithm, given the heterogeneity of patient profiles and availability of resources in various clinical settings at various times of day. This section introduces the recommendations for an aggressive attempt to minimize the time from entry into the medical system to implementation of a reperfusion strategy using the concept of medical system goals. More detailed discussion of these goals and the issues to be considered in selecting the type of reperfusion therapy are discussed in the Selection of Reperfusion Therapy section of VI.C.1.f (Section 6.3.1.6.2 of the full-text guidelines), followed by a discussion of available resources.

The medical system goal is to facilitate rapid recognition and treatment of patients with STEMI such that door-to-needle (or medical contact–to-needle) time for initiation of fibrinolytic therapy can be achieved within 30 minutes or that door-to-balloon (or medical contact–to-balloon) time for PCI can be kept under 90 minutes. These goals may not be relevant for the patients with an appropriate reason for delay, such as uncertainty about the diagnosis (particularly for the use of fibrinolytic therapy), need for the evaluation and treatment of other life-threatening conditions (eg, respiratory failure), or delays associated with the patient's informed choice to have more time to consider the decision. In the absence of such types of circumstances, the emphasis is on having a system in place such that when a patient with STEMI presents for medical care, reperfusion therapy is able to be provided as soon as possible within these time periods. Because there is not considered to be a threshold effect for the benefit of shorter times to reperfusion, these goals should not be understood as "ideal" times but the longest times that should be considered acceptable. Systems that are able to achieve even more rapid times for patients should be encouraged. Also, this goal should not be perceived as an average performance standard but a goal of an early treatment system that every hospital should seek for every appropriate patient.

SELECTION OF REPERFUSION STRATEGY. Several issues should be considered in selecting the type of reperfusion therapy:

- *Time From Onset of Symptoms*. Time from onset of symptoms to fibrinolytic therapy is an important predictor of MI size and patient outcome.[36] The efficacy of fibrinolytic agents in lysing thrombus diminishes with the passage of time.[37] Fibrinolytic therapy administered within the first 2 hours (especially the first hour) can occasionally abort MI and dramatically reduce mortality.[23,38]

  In contrast, the ability to produce a patent infarct artery is much less dependent on symptom duration in patients undergoing PCI. Several reports claim no influence of time

delay on mortality rates when PCI is performed after 2 to 3 hours of symptom duration.[39,40] Importantly, after adjustment for baseline characteristics, time from symptom onset to balloon inflation is significantly correlated with 1-year mortality in patients undergoing primary PCI for STEMI.[41] The Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology[42] and this Committee both recommend a target of medical contact–to-balloon or door-to-balloon time within 90 minutes.

- *Risk of STEMI*. Several models have been developed that assist clinicians in estimating the risk of mortality in patients with STEMI.[43–47] Although these models vary somewhat in the factors loaded into the risk-prediction tool and also vary with respect to statistical measures of their discriminative power (eg, C statistic), all the models provide clinicians with a means to assess the continuum of risk from STEMI. When the estimated mortality with fibrinolysis is extremely high, as is the case in patients with cardiogenic shock, compelling evidence exists that favors a PCI strategy.
- *Risk of Bleeding*. Choice of reperfusion therapy is also affected by the patient's risk of bleeding. When both types of reperfusion are available, the higher the patient's risk of bleeding with fibrinolytic therapy, the more strongly the decision should favor PCI. If PCI is unavailable, then the benefit of pharmacological reperfusion therapy should be balanced against the risk.
- *Time Required for Transport to a Skilled PCI Laboratory*. The availability of interventional cardiology facilities is a key determinant of whether PCI can be provided. For facilities that can offer PCI, the literature suggests that this approach is superior to pharmacological reperfusion.[48] The trials comparing pharmacological and PCI strategies, however, were conducted before the advent of more recent pharmacological and PCI strategies. When a composite end point of death, nonfatal recurrent MI, or stroke is analyzed, much of the superiority of a PCI strategy is driven by a reduction in the rate of nonfatal recurrent MI (Figure 2).[36] The rate of nonfatal recurrent MI can be influenced both by the adjunctive therapy used and by the proportion of patients who are referred for PCI when the initial attempt at fibrinolysis fails or myocardial ischemia recurs after initially successful pharmacological reperfusion.

The experience and location of the PCI laboratory also plays a role in the choice of therapy. Not all laboratories can provide prompt, high-quality primary PCI. Even centers with interventional cardiology facilities may not be able to provide the staffing required for 24-hour coverage of the catheterization laboratory. Despite staffing availability, the volume of cases in the laboratory may be insufficient for the team to acquire and maintain skills required for rapid PCI reperfusion strategies.

A decision must be made when a STEMI patient presents to a center without interventional cardiology facilities. Fibrinolytic therapy can generally be provided sooner than primary PCI. As the time delay for performing PCI increases, the mortality benefit associated with expeditiously performed primary PCI over fibrinolysis decreases.[49] Compared with a fibrin-specific lytic agent, a PCI strategy may not reduce mortality when a delay greater than 60 minutes is anticipated versus immediate administration of a lytic.

Given the current literature, it is not possible to say definitively that a particular reperfusion approach is superior for all patients, in all clinical settings, at all times of day (Danchin N; oral presentation at American Heart Association Scientific Sessions 2003, Orlando, FL, November 2003).[50–52] The main point is that some type of reperfusion therapy should be selected for all appropriate patients with suspected STEMI. The appropriate and timely use of some reperfusion therapy is likely more important than the choice of therapy, given the current literature and the expanding array of options. Clinical circumstances in which fibrinolytic therapy is generally preferred or an invasive strategy is generally preferred are shown in Figure 3.

Available Resources

Class I

1. STEMI patients presenting to a facility without the capability for expert, prompt intervention with primary PCI within 90 minutes of first medical contact should undergo fibrinolysis unless contraindicated. (*Level of Evidence: A*)

Pharmacological Reperfusion.

Indications for Fibrinolytic Therapy

Class I

1. In the absence of contraindications, fibrinolytic therapy should be administered to STEMI patients with symptom onset within the prior 12 hours and ST elevation greater than 0.1 mV in at least 2 contiguous precordial leads or at least 2 adjacent limb leads. (*Level of Evidence: A*)

2. In the absence of contraindications, fibrinolytic therapy should be administered to STEMI patients with symptom onset within the prior 12 hours and new or presumably new LBBB. (*Level of Evidence: A*)

Class IIa

1. In the absence of contraindications, it is reasonable to administer fibrinolytic therapy to STEMI patients with symptom onset within the prior 12 hours and 12-lead ECG findings consistent with a true posterior MI. (*Level of Evidence: C*)

2. In the absence of contraindications, it is reasonable to administer fibrinolytic therapy to patients with symptoms of STEMI beginning within the prior 12 to 24 hours who have continuing ischemic symptoms and ST elevation greater than 0.1 mV in at least 2 contiguous precordial leads or at least 2 adjacent limb leads. (*Level of Evidence: B*)

Class III

1. Fibrinolytic therapy should not be administered to asymptomatic patients whose initial symptoms of STEMI began more than 24 hours earlier. (*Level of Evidence: C*)

2. Fibrinolytic therapy should not be administered to patients whose 12-lead ECG shows only ST-segment depression except if a true posterior MI is suspected. (*Level of Evidence: A*)

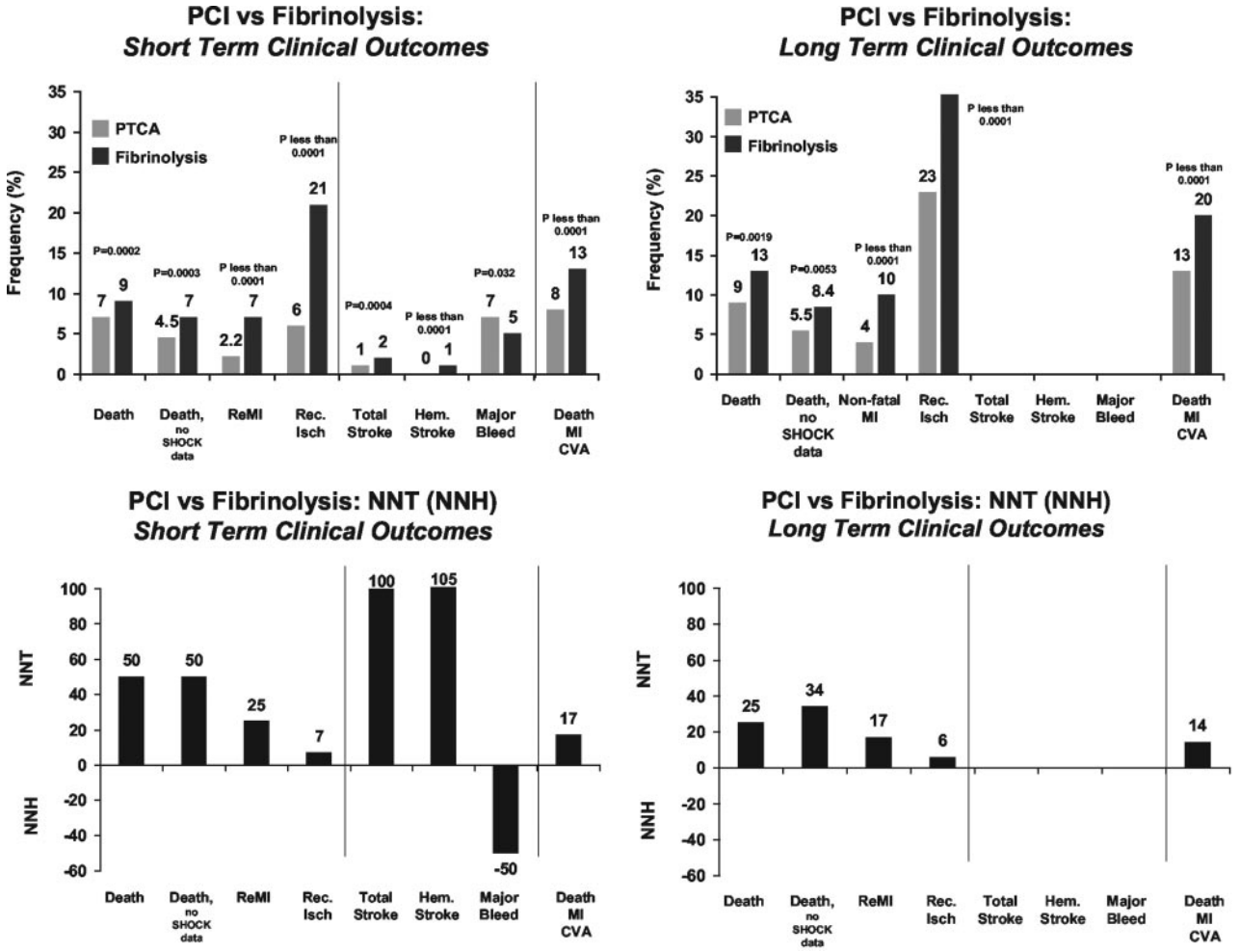

Figure 2. PCI vs fibrinolysis for STEMI. Short-term (4 to 6 weeks; top left) and long-term (top right) outcomes for various end points shown are plotted for STEMI patients randomized to PCI or fibrinolysis for reperfusion in 23 trials (n=7739). Given the frequency of events for each end point in the 2 treatment groups, the number needed to treat (NNT) or number needed to harm (NNH) is shown for the short-term (bottom left) and long-term (bottom right) outcomes. The magnitude of treatment differences for death, nonfatal reinfarction, and stroke varies depending on whether PCI is compared with streptokinase or a fibrin-specific lytic. For example, when primary PCI is compared with alteplase and the SHOCK trial is excluded, the mortality rate is 5.5% vs 6.7% (odds ratio 0.81, 95% confidence interval 0.64 to 1.03, $P=0.081$).[76a] See references 76 and 76a for additional discussion. Modified with permission from Elsevier (Keeley et al. The Lancet. 2003;361:13–20)[76] ReMI indicates recurrent MI; Rec. Isch, recurrent ischemia; Hem. Stroke, hemorrhagic stroke; and CVA, cerebrovascular accident.

Because the benefit of fibrinolytic therapy is directly related to the time from symptom onset, treatment benefit is maximized by the earliest possible application of therapy. The constellation of clinical features that must be present (although not necessarily at the same time) to serve as an indication for fibrinolysis includes symptoms of myocardial ischemia and ST elevation greater than 0.1 mV, in at least 2 contiguous leads, or new or presumably new LBBB on the presenting ECG.[23,54]

Contraindications/Cautions

Class I

1. **Healthcare providers should ascertain whether the patient has neurological contraindications to fibrinolytic therapy, including any history of intracranial hemorrhage (ICH), significant closed head or facial trauma within the past 3 months, uncontrolled hypertension, or ischemic stroke within the past 3 months. (See Table 2 for a comprehensive list.) (*Level of Evidence: A*)**
2. **STEMI patients at substantial (greater than or equal to 4%) risk of ICH should be treated with PCI rather than with fibrinolytic therapy. (See Figure 3 for further management considerations.) (*Level of Evidence: A*)**

A detailed list of contraindications and cautions for the use of fibrinolytic therapy is shown in Table 2.

Complications of Fibrinolytic Therapy: Neurological and Other

Class I

1. **The occurrence of a change in neurological status during or after reperfusion therapy, particularly within the first 24 hours after initiation of treatment, is**

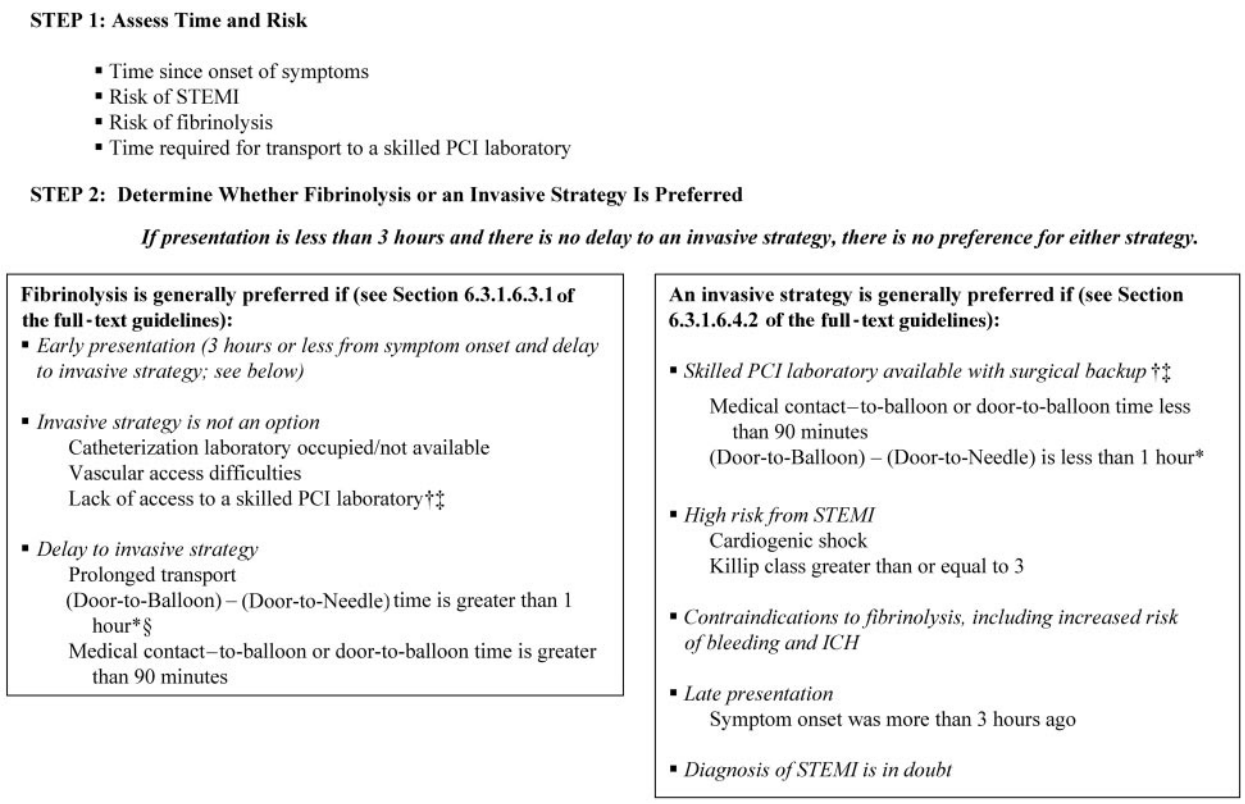

Figure 3. Assessment of reperfusion options for patients with STEMI. STEMI indicates ST-elevation myocardial infarction; PCI, percutaneous coronary intervention; ICH, intracranial hemorrhage. *Applies to fibrin-specific agents (see Figure 15 in the full-text STEMI guidelines). †Operator experience greater than a total of 75 primary PCI cases per year. ‡Team experience greater than a total of 36 primary PCI cases per year. §This calculation implies that the estimated delay to the implementation of the invasive strategy is greater than 1 hour vs initiation of fibrinolytic therapy immediately with a fibrin-specific agent.

considered to be due to ICH until proven otherwise. Fibrinolytic, antiplatelet, and anticoagulant therapies should be discontinued until brain imaging scan shows no evidence of ICH. (*Level of Evidence: A*)

2. Neurology and/or neurosurgery or hematology consultations should be obtained for STEMI patients who have ICH as dictated by clinical circumstances. (*Level of Evidence: C*)

3. In patients with ICH, infusions of cryoprecipitate, fresh frozen plasma, protamine, and platelets should be given, as dictated by clinical circumstances. (*Level of Evidence: C*)

Class IIa

1. In patients with ICH, it is reasonable to:

a. Optimize blood pressure and blood glucose levels. (*Level of Evidence: C*)

b. Reduce intracranial pressure with an infusion of mannitol, endotracheal intubation, and hyperventilation. (*Level of Evidence: C*)

c. Consider neurosurgical evacuation of ICH. (*Level of Evidence: C*)

Combination Therapy With Glycoprotein IIb/IIIa Inhibitors

Class IIb

1. Combination pharmacological reperfusion with abciximab and half-dose reteplase or tenecteplase may be considered for prevention of reinfarction (*Level of Evidence: A*) and other complications of STEMI in selected patients: anterior location of MI, age less than 75 years, and no risk factors for bleeding. In two clinical trials of combination reperfusion, the prevention of reinfarction did not translate into a survival benefit at either 30 days or 1 year.[54a] (*Level of Evidence: B*)

2. Combination pharmacological reperfusion with abciximab and half-dose reteplase or tenecteplase may be considered for prevention of reinfarction and other complications of STEMI in selected patients (anterior location of MI, age less than 75 years, and no risk factors for bleeding) in whom an early referral for angiography and PCI (ie, facilitated PCI) is planned. (*Level of Evidence: C*)

Class III

1. Combination pharmacological reperfusion with abciximab and half-dose reteplase or tenecteplase should not be given to patients aged greater than 75 years because of an increased risk of ICH. (*Level of Evidence: B*)

PERCUTANEOUS CORONARY INTERVENTION

Coronary Angiography

Class I

1. Diagnostic coronary angiography should be performed:

TABLE 2. Contraindications and Cautions for Fibrinolysis Use in ST-Elevation Myocardial Infarction*

Absolute contraindications

- Any prior ICH
- Known structural cerebral vascular lesion (eg, AVM)
- Known malignant intracranial neoplasm (primary or metastatic)
- Ischemic stroke within 3 months EXCEPT acute ischemic stroke within 3 hours
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed head or facial trauma within 3 months

Relative contraindications

- History of chronic severe, poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (SBP greater than 180 mm Hg or DBP greater than 110 mm Hg)†
- History of prior ischemic stroke greater than 3 months, dementia, or known intracranial pathology not covered in contraindications
- Traumatic or prolonged (greater than 10 minutes) CPR or major surgery (less than 3 weeks)
- Recent (within 2 to 4 weeks) internal bleeding
- Noncompressible vascular punctures
- For streptokinase/anistreplase: prior exposure (more than 5 days ago) or prior allergic reaction to these agents
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR, the higher the risk of bleeding

AVM indicates arteriovenous malformation; SBP, systolic blood pressure; DBP, diastolic blood pressure; ICH, intracranial hemorrhage; CPR, cardiopulmonary resuscitation.

*Viewed as advisory for clinical decision making and may not be all-inclusive or definitive.

†Could be an absolute contraindication in low-risk patients with ST-elevation myocardial infarction (see Section 6.3.1.6.3.2 of the full-text guidelines).

a. **In candidates for primary or rescue PCI. (*Level of Evidence: A*)**
b. **In patients with cardiogenic shock who are candidates for revascularization. (*Level of Evidence: A*)**
c. **In candidates for surgical repair of ventricular septal rupture or severe mitral regurgitation (MR). (*Level of Evidence: B*)**
d. **In patients with persistent hemodynamic and/or electrical instability. (*Level of Evidence: C*)**

Class III

1. **Coronary angiography should not be performed in patients with extensive comorbidities in whom the risks of revascularization are likely to outweigh the benefits. (*Level of Evidence: C*)**

Primary PCI

Class I

1. **General considerations: If immediately available, primary PCI should be performed in patients with STEMI (including true posterior MI) or MI with new or presumably new LBBB who can undergo PCI of the infarct artery within 12 hours of symptom onset, if performed in a timely fashion (balloon inflation within 90 minutes of presentation) by persons skilled in the procedure (individuals who perform more than 75 PCI procedures per year). The procedure should be supported by experienced personnel in an appropriate laboratory environment (performs more than 200 PCI procedures per year, of which at least 36 are primary PCI for STEMI, and has cardiac surgery capability). (*Level of Evidence: A*)**
2. **Specific considerations:**
   a. **Primary PCI should be performed as quickly as possible, with a goal of a medical contact–to-balloon or door-to-balloon time of within 90 minutes. (*Level of Evidence: B*)**
   b. **If the symptom duration is within 3 hours and the expected door-to-balloon time minus the expected door-to-needle time is:**
      i) **within 1 hour, primary PCI is generally preferred. (*Level of Evidence: B*)**
      ii) **greater than 1 hour, fibrinolytic therapy (fibrin-specific agents) is generally preferred. (*Level of Evidence: B*)**
   c. **If symptom duration is greater than 3 hours, primary PCI is generally preferred and should be performed with a medical contact–to-balloon or door-to-balloon time as brief as possible, with a goal of within 90 minutes. (*Level of Evidence: B*)**
   d. **Primary PCI should be performed for patients younger than 75 years old with ST elevation or LBBB who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock, unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: A*)**
   e. **Primary PCI should be performed in patients with severe CHF and/or pulmonary edema (Killip class 3) and onset of symptoms within 12 hours. The medical contact–to-balloon or door-to-balloon time should be as short as possible (ie, goal within 90 min). (*Level of Evidence: B*)**

Class IIa

1. **Primary PCI is reasonable for selected patients 75 years or older with ST elevation or LBBB or who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock. Patients with good prior functional status who are suitable for revascularization and agree to invasive care may be selected for such an invasive strategy. (*Level of Evidence: B*)**
2. **It is reasonable to perform primary PCI for patients with onset of symptoms within the prior 12 to 24 hours and 1 or more of the following:**
   a. **Severe CHF (*Level of Evidence: C*)**
   b. **Hemodynamic or electrical instability (*Level of Evidence: C*)**
   c. **Persistent ischemic symptoms. (*Level of Evidence: C*)**

Class IIb

1. **The benefit of primary PCI for STEMI patients eligible for fibrinolysis is not well established when performed by an operator who performs fewer than 75 PCI procedures per year. (*Level of Evidence: C*)**

Class III

1. **PCI should not be performed in a noninfarct artery at the time of primary PCI in patients without hemodynamic compromise. (*Level of Evidence: C*)**
2. **Primary PCI should not be performed in asymptomatic patients more than 12 hours after onset of STEMI if they are hemodynamically and electrically stable. (*Level of Evidence: C*)**

Primary PCI has been compared with fibrinolytic therapy in 22 randomized clinical trials.[50,52,55–74] An additional trial, SHOCK (SHould we emergently revascularize Occluded Coronaries for cardiogenic shocK?),[75] that compared medical stabilization with immediate revascularization for cardiogenic shock was included along with the above 22 trials in an overview of primary PCI versus fibrinolysis.[76] These investigations demonstrate that PCI-treated patients experience lower short-term mortality rates, less nonfatal reinfarction, and less hemorrhagic stroke than those treated by fibrinolysis but have an increased risk for major bleeding.[76] These results have been achieved in medical centers with experienced providers and under circumstances in which PCI can be performed promptly after patient presentation (Figure 2).[76]

Additional considerations that affect the magnitude of the difference between PCI- and fibrinolysis-treated patients include the fact that unfractionated heparin (UFH) was used as the antithrombin with fibrinolytics (as opposed to other antithrombins such as enoxaparin [see Ancillary Therapy in Section VI.C.1.f and also Section 6.3.1.6.8.1.1 of the full-text guidelines] or bivalirudin [see Section 6.3.1.6.8.1.2 of the full-text guidelines] that are associated with a reduction in the rate of recurrent MI after fibrinolysis), a smaller but still statistically significant advantage for PCI compared with a fibrin-specific lytic versus streptokinase, and variation among the PCI arms as to whether a stent was implanted or glycoprotein (GP) IIb/IIIa antagonists were administered. Figure 2 shows the short- and long-term outcomes of patients with STEMI treated by fibrinolysis versus PCI and the number of patients who need to be treated to prevent 1 event or cause 1 harmful complication when selecting PCI instead of fibrinolysis as the reperfusion strategy (Figure 2).[76] Of note, when primary PCI is compared with tissue plasminogen activator (tPA) and the SHOCK trial is excluded, the mortality rate is 5.5% versus 6.7% (odds ratio 0.81%, 95% confidence interval [CI] 0.64 to 1.03, *P* equals 0.081.[76a]

There is serious and legitimate concern that a routine policy of primary PCI for patients with STEMI will result in unacceptable delays in achieving reperfusion in a substantial number of cases and produce less than optimal outcomes if performed by less-experienced operators. The mean time delay for PCI instead of fibrinolysis in the randomized studies was approximately 40 minutes.[76] Strict performance criteria must be mandated for primary PCI programs so that long door-to-balloon times and performance by low-volume or poor-outcome operators/laboratories do not occur. Interventional cardiologists and centers should strive for outcomes to include (1) medical contact–to-balloon or door-to-balloon times less than 90 minutes; (2) TIMI (Thrombolysis In Myocardial Infarction) 2/3 flow rates obtained in more than 90% of patients; (3) emergency CABG rate less than 2% among all patients undergoing the procedure; (4) actual performance of PCI in a high percentage of patients (85%) brought to the laboratory; and (5) risk-adjusted in-hospital mortality rate less than 7% in patients without cardiogenic shock. This would result in a risk-adjusted mortality rate with PCI comparable to that reported for fibrinolytic therapy in fibrinolytic-eligible patients[76] and would be consistent with previously reported registry experience.[77–80] Otherwise, the focus of treatment should be the early use of fibrinolytic therapy (Figure 2).[76]

PCI appears to have its greatest mortality benefit in high-risk patients. In patients with cardiogenic shock, an absolute 9% reduction in 30-day mortality with coronary revascularization instead of immediate medical stabilization was reported in the SHOCK trial.[75]

Time from symptom onset to reperfusion is an important predictor of patient outcome. Two studies[81,82] have reported increasing mortality rates with increasing door-to-balloon times. Other studies have shown smaller infarct size, better LV function, and fewer complications when reperfusion occurs before PCI.[83–85] An analysis of the randomized controlled trials comparing fibrinolysis with a fibrin-specific agent versus primary PCI suggests that the mortality benefit with PCI exists when treatment is delayed by no more than 60 minutes. Mortality increases significantly with each 15-minute delay in the time between arrival and restoration of TIMI-3 flow (door-to–TIMI-3 flow time), which further underscores the importance of timely reperfusion in patients who undergo primary PCI.[86] Importantly, after adjustment for baseline characteristics, time from symptom onset to balloon inflation is significantly correlated with 1-year mortality in patients undergoing primary PCI for STEMI (relative risk equals 1.08 for each 30-minute delay from symptom onset to balloon inflation; *P* equals 0.04).[35,41] Given that the medical contact–to-needle time goal within 30 minutes, this Writing Committee joins the Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology in lowering the medical contact–to-balloon or door-to-balloon time goal from within 120 minutes to within 90 minutes in an attempt to maximize the benefits for reperfusion by PCI.[42]

If the expected door-to-balloon time exceeds the expected door-to-needle time by more than 60 minutes, fibrinolytic treatment with a fibrin-specific agent should be considered unless it is contraindicated. This is particularly important when symptom duration is less than 3 hours but is less important with longer symptom duration, when less ischemic myocardium can be salvaged.

Primary PCI in fibrinolytic-ineligible patients

Class I

1. **Primary PCI should be performed in fibrinolytic-ineligible patients who present with STEMI within 12 hours of symptom onset. (*Level of Evidence: C*)**

Class IIa

1. **It is reasonable to perform primary PCI for fibrinolytic-ineligible patients with onset of symptoms within the prior 12 to 24 hours and 1 or more of the following:**
   a. **Severe CHF (*Level of Evidence: C*)**
   b. **Hemodynamic or electrical instability (*Level of Evidence: C*)**
   c. **Persistent ischemic symptoms. (*Level of Evidence: C*)**

Randomized controlled trials evaluating the outcome of PCI for patients who present with STEMI but who are ineligible for fibrinolytic therapy have not been performed. Few data are available to characterize the value of primary PCI for this subset of STEMI patients; however, the recommendations in Section IV.A (and Section 4.2 of the full-text guidelines) are applicable to these patients. Nevertheless, these patients are at increased risk for mortality,[87] and there is a general consensus that PCI is an appropriate means for achieving reperfusion in those who cannot receive fibrinolytics because of increased risk of bleeding.[88–91]

Primary PCI Without On-Site Cardiac Surgery

Class IIb

1. **Primary PCI might be considered in hospitals without on-site cardiac surgery, provided that there exists a proven plan for rapid transport to a cardiac surgery operating room in a nearby hospital with appropriate hemodynamic support capability for transfer. The procedure should be limited to patients with STEMI or MI with new, or presumably new, LBBB on ECG, and should be done in a timely fashion (balloon inflation within 90 minutes of presentation) by persons skilled in the procedure (at least 75 PCIs per year) and at hospitals that perform a minimum of 36 primary PCI procedures per year. (*Level of Evidence: B*)**

Class III

1. **Primary PCI should not be performed in hospitals without on-site cardiac surgery and without a proven plan for rapid transport to a cardiac surgery operating room in a nearby hospital or without appropriate hemodynamic support capability for transfer. (*Level of Evidence: C*)**

From clinical data and expert consensus, the Committee recommends that primary PCI for acute STEMI performed at hospitals without established elective PCI programs should be restricted to those institutions capable of performing a requisite minimum number of primary PCI procedures (36 per year) with a proven plan for rapid and effective PCI and rapid access to cardiac surgery in a nearby hospital. The benefit of primary PCI is not well established for operators who perform fewer than 75 PCIs per year or in a hospital that performs fewer than 36 primary PCI procedures per year. In addition, the benefit of timely reperfusion of the infarct artery by primary PCI at sites without on-site surgery must be weighed against the small but finite risk of harm to the patient related to the time required to transfer the patient to a site with CABG surgery capabilities.[92,93]

Interhospital Transfer for Primary PCI

To achieve optimal results, time from the first hospital door to the balloon inflation in the second hospital should be as short as possible, with a goal of within 90 minutes. Significant reductions in door-to-balloon times might be achieved by directly transporting patients to PCI centers rather than transporting them to the nearest hospital, if interhospital transfer will subsequently be required to obtain primary PCI.

Primary Stenting

Primary stenting has been compared with primary angioplasty in 9 studies.[94–103] There were no differences in mortality (3.0% versus 2.8%) or reinfarction (1.8% versus 2.1%) rates. However, major adverse cardiac events were reduced, driven by the reduction in subsequent target-vessel revascularization with stenting.

Preliminary reports suggest that compared with conventional bare metal stents, drug-eluting stents are not associated with increased risk when used for primary PCI in STEMI patients.[104] Postprocedure vessel patency, biomarker release, and the incidence of short-term adverse events were similar in patients receiving sirolimus (n equals 186) or bare metal (n equals 183) stents. Thirty-day event rates of death, reinfarction, or revascularization were 7.5% versus 10.4%, respectively (*P* equals 0.4).[104]

Facilitated PCI

Class IIb

1. **Facilitated PCI might be performed as a reperfusion strategy in higher-risk patients when PCI is not immediately available and bleeding risk is low. (*Level of Evidence: B*)**

Facilitated PCI refers to a strategy of planned immediate PCI after an initial pharmacological regimen such as full-dose fibrinolysis, half-dose fibrinolysis, a GP IIb/IIIa inhibitor, or a combination of reduced-dose fibrinolytic therapy and a platelet GP IIb/IIIa inhibitor. A strategy of facilitated PCI holds promise in higher-risk patients when PCI is not immediately available. Potential risks include increased bleeding complications, especially in patients who are at least 75 years of age (see Pharmacological Reperfusion in Section VI.C.1.f and Section 6.3.1.6.3.8. of the full-text guidelines), and potential limitations include added cost. Several randomized trials of facilitated PCI with a variety of pharmacological regimens are in progress.

Rescue PCI

Class I

1. **Rescue PCI should be performed in patients less than 75 years old with ST elevation or LBBB who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock, unless further support is futile because**

of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: B*)
2. Rescue PCI should be performed in patients with severe CHF and/or pulmonary edema (Killip class 3) and onset of symptoms within 12 hours. (*Level of Evidence: B*)

Class IIa

1. Rescue PCI is reasonable for selected patients 75 years or older with ST elevation or LBBB or who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock. Patients with good prior functional status who are suitable for revascularization and agree to invasive care may be selected for such an invasive strategy. (*Level of Evidence: B*)
2. It is reasonable to perform rescue PCI for patients with 1 or more of the following:
a. Hemodynamic or electrical instability (*Level of Evidence: C*)
b. Persistent ischemic symptoms. (*Level of Evidence: C*)

Rescue PCI refers to PCI within 12 hours after failed fibrinolysis for patients with continuing or recurrent myocardial ischemia.

A major problem in adopting a strategy of rescue PCI lies in the limitation of accurate identification of patients for whom fibrinolytic therapy has not restored antegrade coronary flow. In a prior era in which the practice of PCI was less mature, immediate catheterization of all patients after fibrinolytic therapy to identify those with an occluded infarct artery was found to be impractical, costly, and often associated with bleeding complications.[105,106] This strategy is being reevaluated in clinical trials testing facilitated PCI in the contemporary PCI setting.

There are no convincing data to support the routine use of late adjuvant PCI days after failed fibrinolysis or for patients who do not receive reperfusion therapy. Nevertheless, this is being done in some STEMI patients as an extension of the invasive strategy for non-STEMI patients. The Occluded Artery Trial (OAT) is currently randomizing patients to test whether routine PCI days to weeks after MI improves long-term clinical outcomes in asymptomatic high-risk patients with an occluded infarct artery.[107]

PCI for Cardiogenic Shock

Class I

1. Primary PCI is recommended for patients less than 75 years old with ST elevation or LBBB who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock, unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: A*)

Class IIa

1. Primary PCI is reasonable for selected patients aged 75 years or older with ST elevation or LBBB who develop shock within 36 hours of MI and who are suitable for revascularization that can be performed within 18 hours of shock. Patients with good prior functional status who are suitable for revascularization and agree to invasive care may be selected for such an invasive strategy. (*Level of Evidence: B*)

Observational studies support the value of PCI for patients who develop cardiogenic shock in the early hours of STEMI. In the SHOCK trial,[75] the survival curves continued to progressively diverge such that at 6 months and 1 year, there was a significant mortality reduction with emergency revascularization (53% versus 66%, P less than 0.03).[108] The prespecified subgroup analysis of patients less than 75 years old showed an absolute 15% reduction in 30-day mortality (P less than 0.02), whereas there was no apparent benefit for the small cohort (n equals 56) of patients more than 75 years old. These data strongly support the approach that patients younger than 75 years with STEMI complicated by cardiogenic shock should undergo emergency revascularization and support measures. Three registries[109–111] have demonstrated a marked survival benefit for elderly patients who are clinically selected for revascularization (approximately 1 of 5 patients), so age alone should not disqualify a patient from early revascularization. (See Section VII.F.5 and also Section 7.6.5 of the full-text guidelines.)

Percutaneous Coronary Intervention After Fibrinolysis

Class I

1. In patients whose anatomy is suitable, PCI should be performed when there is objective evidence of recurrent MI. (*Level of Evidence: C*)
2. In patients whose anatomy is suitable, PCI should be performed for moderate or severe spontaneous or provocable myocardial ischemia during recovery from STEMI. (*Level of Evidence: B*)
3. In patients whose anatomy is suitable, PCI should be performed for cardiogenic shock or hemodynamic instability. (See section on PCI for Cardiogenic Shock in Section VI.C.1.f.) (*Level of Evidence: B*)

Class IIa

1. It is reasonable to perform routine PCI in patients with LV ejection fraction (LVEF) less than or equal to 0.40, CHF, or serious ventricular arrhythmias. (*Level of Evidence: C*)
2. It is reasonable to perform PCI when there is documented clinical heart failure during the acute episode, even though subsequent evaluation shows preserved LV function (LVEF greater than 0.40). (*Level of Evidence: C*)

Class IIb

1. Routine PCI might be considered as part of an invasive strategy after fibrinolytic therapy. (*Level of Evidence: B*)

Immediately After Successful Fibrinolysis. Randomized prospective trials examined the efficacy and safety of immediate PCI after fibrinolysis.[105,106,112] These trials showed no benefit of routine PCI of the stenotic infarct artery immediately after fibrinolytic therapy. The strategy did not appear to salvage myocardium, improve LVEF, or prevent reinfarction

or death. Those subjected to this approach appeared to have an increased incidence of adverse events, including bleeding, recurrent ischemia, emergency CABG, and death. These studies have not been repeated in the modern interventional era with improved equipment, improved antiplatelet and anticoagulant strategies, and coronary stents, thus leaving the question of routine PCI early after successful fibrinolysis unresolved in contemporary practice. Studies of facilitated PCI are enrolling patients.[113–116]

Hours to Days After Successful Fibrinolysis. Great improvements in equipment, operator experience, and adjunctive pharmacotherapy have increased PCI success rates and decreased complications. More recently, the invasive strategy for NSTEMI patients has been given a Class I recommendation by the ACC/AHA Guidelines for the Management of Patients With Unstable Angina/Non-STEMI.[117] STEMI patients are increasingly being treated similarly as an extension of this approach. Although 6 published reports[115,118–121,123] and 1 preliminary report[122] support this strategy, randomized studies similar to those in NSTEMI need to be performed.

Acute Surgical Reperfusion

Class I

1. Emergency or urgent CABG in patients with STEMI should be undertaken in the following circumstances:

- **a. Failed PCI with persistent pain or hemodynamic instability in patients with coronary anatomy suitable for surgery. (*Level of Evidence: B*)**
- **b. Persistent or recurrent ischemia refractory to medical therapy in patients who have coronary anatomy suitable for surgery, have a significant area of myocardium at risk, and are not candidates for PCI or fibrinolytic therapy. (*Level of Evidence: B*)**
- **c. At the time of surgical repair of postinfarction ventricular septal rupture (VSR) or mitral valve insufficiency. (*Level of Evidence: B*)**
- **d. Cardiogenic shock in patients less than 75 years old with ST elevation, LBBB, or posterior MI who develop shock within 36 hours of STEMI, have severe multivessel or left main disease, and are suitable for revascularization that can be performed within 18 hours of shock, unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: A*)**
- **e. Life-threatening ventricular arrhythmias in the presence of greater than or equal to 50% left main stenosis and/or triple-vessel disease. (*Level of Evidence: B*)**

Class IIa

1. Emergency CABG can be useful as the primary reperfusion strategy in patients who have suitable anatomy, who are not candidates for fibrinolysis or PCI, and who are in the early hours (6 to 12 hours) of an evolving STEMI, especially if severe multivessel or left main disease is present. (*Level of Evidence: B*)

2. Emergency CABG can be effective in selected patients 75 years or older with ST elevation, LBBB, or posterior MI who develop shock within 36 hours of STEMI, have severe triple-vessel or left main disease, and are suitable for revascularization that can be performed within 18 hours of shock. Patients with good prior functional status who are suitable for revascularization and agree to invasive care may be selected for such an invasive strategy. (*Level of Evidence: B*)

Class III

1. Emergency CABG should not be performed in patients with persistent angina and a small area of risk if they are hemodynamically stable. (*Level of Evidence: C*)

2. Emergency CABG should not be performed in patients with successful epicardial reperfusion but unsuccessful microvascular reperfusion. (*Level of Evidence: C*)

Patients With STEMI Not Receiving Reperfusion

Guideline-based recommendations for nonreperfusion treatments should not vary whether or not patients received reperfusion therapy. The major difference is that patients not receiving reperfusion therapy are considered to have a higher risk for future adverse events.[124]

Assessment of Reperfusion

Class IIa

1. It is reasonable to monitor the pattern of ST elevation, cardiac rhythm, and clinical symptoms over the 60 to 180 minutes after initiation of fibrinolytic therapy. Noninvasive findings suggestive of reperfusion include relief of symptoms, maintenance or restoration of hemodynamic and or electrical stability, and a reduction of at least 50% of the initial ST-segment elevation injury pattern on a follow-up ECG 60 to 90 minutes after initiation of therapy. (*Level of Evidence: B*)

Persistence of unrelenting ischemic chest pain, absence of resolution of the qualifying ST-segment elevation, and hemodynamic and/or electrical instability are generally indicators of failed pharmacological reperfusion and the need to consider rescue PCI. Aggressive medical support may be necessary in the interim. (See Rescue PCI in Section in VI.C.I.f.)

Ancillary Therapy

Antithrombins as Ancillary Therapy to Reperfusion Therapy

Unfractionated heparin as ancillary therapy to reperfusion therapy

Class I

1. Patients undergoing percutaneous or surgical revascularization should be given UFH. (*Level of Evidence: C*)

2. UFH should be given intravenously to patients undergoing reperfusion therapy with alteplase, reteplase, or tenecteplase, with dosing as follows: bolus of 60 U/kg

(maximum 4000 U) followed by an initial infusion of 12 U/kg per hour (maximum 1000 U/hr) adjusted to maintain activated partial thromboplastin time (aPTT) at 1.5 to 2.0 times control (approximately 50 to 70 seconds). (*Level of Evidence: C*)

3. **UFH should be given intravenously to patients treated with nonselective fibrinolytic agents (streptokinase, anistreplase, or urokinase) who are at high risk for systemic emboli (large or anterior MI, atrial fibrillation, previous embolus, or known LV thrombus). (*Level of Evidence: B*)**
4. **Platelet counts should be monitored daily in patients given UFH. (*Level of Evidence: C*)**

Class IIb

1. **It may be reasonable to administer UFH intravenously to patients undergoing reperfusion therapy with streptokinase. (*Level of Evidence: B*)**

Because of the evidence that the measured effect of UFH on the aPTT is important for patient outcome and that the predominant variable mediating the effect of a given dose of heparin is weight,[125] it is important to administer the initial doses of UFH as a weight-adjusted bolus.[126] For fibrin-specific (alteplase, reteplase, and tenecteplase) fibrinolytic-treated patients, a 60 U/kg bolus followed by a maintenance infusion of 12 U/kg per hour (with a maximum of 4000 U bolus and 1000 U/h initial infusion for patients weighing greater than 70 kg) is recommended. The recommended weight-adjusted dose of UFH, when it is administered without fibrinolytics, is 60 to 70 U/kg IV bolus and 12 to 15 U/kg per hour infusion.[117]

Low-Molecular-Weight Heparin as Ancillary Therapy to Reperfusion Therapy

Class IIb

1. **LMWH might be considered an acceptable alternative to UFH as ancillary therapy for patients less than 75 years of age who are receiving fibrinolytic therapy, provided that significant renal dysfunction (serum creatinine greater than 2.5 mg/dL in men or 2.0 mg/dL in women) is not present. Enoxaparin (30 mg IV bolus followed by 1.0 mg/kg subcutaneous injection every 12 hours until hospital discharge) used in combination with full-dose tenecteplase is the most comprehensively studied regimen in patients less than 75 years of age. (*Level of Evidence: B*)**

Class III

1. **LMWH should not be used as an alternative to UFH as ancillary therapy in patients over 75 years of age who are receiving fibrinolytic therapy. (*Level of Evidence: B*)**
2. **LMWH should not be used as an alternative to UFH as ancillary therapy in patients less than 75 years of age who are receiving fibrinolytic therapy but have significant renal dysfunction (serum creatinine greater than 2.5 mg/dL in men or 2.0 mg/dL in women). (*Level of Evidence: B*)**

The available data suggest that the rate of early (60 to 90 minutes) reperfusion of the infarct artery, either assessed angiographically or by noninvasive means, is not enhanced by administration of an LMWH. However, a generally consistent theme of a lower rate of reocclusion of the infarct artery, reinfarction, or recurrent ischemic events emerges in patients receiving LMWH regardless of whether the control group was given placebo or UFH.

Direct Antithrombins as Ancillary Therapy to Reperfusion Therapy

Class IIa

1. **In patients with known heparin-induced thrombocytopenia, it is reasonable to consider bivalirudin as a useful alternative to heparin to be used in conjunction with streptokinase. Dosing according to the HERO (Hirulog and Early Reperfusion or Occlusion)-2 regimen (a bolus of 0.25 mg/kg followed by an intravenous infusion of 0.5 mg/kg per hour for the first 12 hours and 0.25 mg/kg per hour for the subsequent 36 hours)[127] is recommended but with a reduction in the infusion rate if the PTT is above 75 seconds within the first 12 hours. (*Level of Evidence: B*)**

On the basis of the data in the HERO-2 trial, the Writing Committee believed that bivalirudin could be considered an acceptable alternative to UFH in those STEMI patients who receive fibrinolysis with streptokinase, have heparin-induced thrombocytopenia, and who, in the opinion of the treating physician, would benefit from anticoagulation.

Antiplatelets

Aspirin

Class I

1. **A daily dose of aspirin (initial dose of 162 to 325 mg orally; maintenance dose of 75 to 162 mg) should be given indefinitely after STEMI to all patients without a true aspirin allergy. (*Level of Evidence: A*)**

As discussed, aspirin should be given to the patient with suspected STEMI as early as possible and should be continued indefinitely, regardless of the strategy for reperfusion and regardless of whether additional antiplatelet agents are administered. True aspirin allergy is the only exception to this recommendation.

Thienopyridines

Class I

1. **In patients who have undergone diagnostic cardiac catheterization and for whom PCI is planned, clopidogrel should be started and continued for at least 1 month after bare metal stent implantation, for several months after drug-eluting stent implantation (3 months for sirolimus, 6 months for paclitaxel), and for up to 12 months in patients who are not at high risk for bleeding. (*Level of Evidence: B*)**
2. **In patients taking clopidogrel in whom CABG is planned, the drug should be withheld for at least 5 days, and preferably for 7, unless the urgency for**

revascularization outweighs the risks of excess bleeding. (*Level of Evidence: B*)

Class IIa

1. Clopidogrel is probably indicated in patients receiving fibrinolytic therapy who are unable to take aspirin because of hypersensitivity or major gastrointestinal intolerance. (*Level of Evidence: C*)

Clopidogrel combined with aspirin is recommended for STEMI patients who undergo coronary stent implantation.[128–132] There are no safety data available regarding the combination of fibrinolytic agents and clopidogrel, but ongoing trials will provide this information in the future. However, in patients in whom aspirin is contraindicated because of aspirin sensitivity, clopidogrel is probably useful as a substitute for aspirin to reduce the risk of occlusion.[133] There are no safety data comparing 300 and 600 mg as loading doses for clopidogrel. We do not recommend routine administration of clopidogrel as pretreatment in patients who have not yet undergone diagnostic cardiac catheterization and in whom CABG surgery would be performed within 5 to 7 days if warranted.[134]

Glycoprotein IIb/IIIa inhibitors

Class IIa

1. It is reasonable to start treatment with abciximab as early as possible before primary PCI (with or without stenting) in patients with STEMI. (*Level of Evidence: B*)

Class IIb

1. Treatment with tirofiban or eptifibatide may be considered before primary PCI (with or without stenting) in patients with STEMI. (*Level of Evidence: C*)

The Writing Committee believes that it is reasonable to start treatment with abciximab as early as possible in patients undergoing primary PCI (with or without stenting) but, given the size and limitations of the available data set, assigned a Class IIa recommendation to this treatment. The data on tirofiban and eptifibatide in primary PCI are far more limited than for abciximab. However, given the common mode of action of the agents, a modest amount of angiographic data,[135] and general clinical experience to date, tirofiban or eptifibatide may be useful as antiplatelet therapy to support primary PCI for STEMI (with or without stenting) (Class IIb recommendation).

Other Pharmacological Measures

Inhibition of Renin-Angiotensin-Aldosterone System

Class I

1. An angiotensin converting enzyme (ACE) inhibitor should be administered orally within the first 24 hours of STEMI to patients with anterior infarction, pulmonary congestion, or LVEF less than 0.40, in the absence of hypotension (systolic blood pressure less than 100 mm Hg or less than 30 mm Hg below baseline) or known contraindications to that Class of medications. (*Level of Evidence: A*)

2. An angiotensin receptor blocker (ARB) should be administered to STEMI patients who are intolerant of ACE inhibitors and who have either clinical or radiological signs of heart failure or LVEF less than 0.40. Valsartan and candesartan have established efficacy for this recommendation. (*Level of Evidence: C*)

Class IIa

1. An ACE inhibitor administered orally within the first 24 hours of STEMI can be useful in patients without anterior infarction, pulmonary congestion, or LVEF less than 0.40 in the absence of hypotension (systolic blood pressure less than 100 mm Hg or less than 30 mm Hg below baseline) or known contraindications to that class of medications. The expected treatment benefit in such patients is less (5 lives saved per 1000 patients treated) than for patients with LV dysfunction. (*Level of Evidence: B*)

Class III

1. An intravenous ACE inhibitor should not be given to patients within the first 24 hours of STEMI because of the risk of hypotension. (A possible exception may be patients with refractory hypertension.) (*Level of Evidence: B*)

A number of large, randomized clinical trials have assessed the role of ACE inhibitors early in the course of acute MI. All trials with oral ACE inhibitors have shown benefit from their early use, including those in which early entry criteria included clinical suspicion of acute infarctions. Data from these trials indicate that ACE inhibitors should generally be started within the first 24 hours, ideally after fibrinolytic therapy has been completed and blood pressure has stabilized. ACE inhibitors should not be used if systolic blood pressure is less than 100 mm Hg or less than 30 mm Hg below baseline, if clinically relevant renal failure is present, if there is a history of bilateral stenosis of the renal arteries, or if there is known allergy to ACE inhibitors.

The use of ARBs has not been explored as thoroughly as ACE inhibitors in STEMI patients. However, clinical experience in the management of patients with heart failure and data from clinical trials in STEMI patients (see Sections 7.4.3 and 7.6.4 of the full-text guidelines) suggest that ARBs may be useful in patients with depressed LV function or clinical heart failure but who are intolerant of an ACE inhibitor. Use of aldosterone antagonists in STEMI patients is discussed in Sections 7.4.3 and 7.6.4 of the full-text guidelines.

Metabolic Modulation of the Glucose-Insulin Axis

Strict Glucose Control During STEMI

Class I

1. An insulin infusion to normalize blood glucose is recommended for patients with STEMI and complicated courses. (*Level of Evidence: B*)

Class IIa

1. **During the acute phase (first 24 to 48 hours) of the management of STEMI in patients with hyperglycemia, it is reasonable to administer an insulin infusion to normalize blood glucose, even in patients with an uncomplicated course. (*Level of Evidence: B*)**
2. **After the acute phase of STEMI, it is reasonable to individualize treatment of diabetics, selecting from a combination of insulin, insulin analogs, and oral hypoglycemic agents that achieve the best glycemic control and are well tolerated. (*Level of Evidence: C*)**

Compelling evidence for tight glucose control in patients in the intensive care unit (a large proportion of whom were there after cardiac surgery) supports the importance of intensive insulin therapy to achieve a normal blood glucose level in critically ill patients.[136,136a]

Magnesium

Class IIa

1. **It is reasonable that documented magnesium deficits be corrected, especially in patients receiving diuretics before the onset of STEMI. (*Level of Evidence: C*)**
2. **It is reasonable that episodes of torsade de pointes-type ventricular tachycardia (VT) associated with a prolonged QT interval be treated with 1 to 2 g of magnesium administered as an intravenous bolus over 5 minutes. (*Level of Evidence: C*)**

Class III

1. **In the absence of documented electrolyte deficits or torsade de pointes-type VT, routine intravenous magnesium should not be administered to STEMI patients at any level of risk. (*Level of Evidence: A*)**

Calcium Channel Blockers

Class IIa

1. **It is reasonable to give verapamil or diltiazem to patients in whom beta-blockers are ineffective or contraindicated (eg, bronchospastic disease) for relief of ongoing ischemia or control of a rapid ventricular response with atrial fibrillation or flutter after STEMI in the absence of CHF, LV dysfunction, or atrioventricular (AV) block. (*Level of Evidence: C*)**

Class III

1. **Diltiazem and verapamil are contraindicated in patients with STEMI and associated systolic LV dysfunction and CHF. (*Level of Evidence: A*)**
2. **Nifedipine (immediate-release form) is contraindicated in treatment of STEMI because of the reflex sympathetic activation, tachycardia, and hypotension associated with its use. (*Level of Evidence: B*)**

See the full-text guidelines for further explanation.

VII. Hospital Management

A. Location

1. Coronary Care Unit

Class I

1. **STEMI patients should be admitted to a quiet and comfortable environment that provides for continuous monitoring of the ECG and pulse oximetry and has ready access to facilities for hemodynamic monitoring and defibrillation. (*Level of Evidence: C*)**
2. **The patient's medication regimen should be reviewed to confirm the administration of aspirin and beta-blockers in an adequate dose to control heart rate and to assess the need for intravenous nitroglycerin for control of angina, hypertension, or heart failure. (*Level of Evidence: A*)**
3. **The ongoing need for supplemental oxygen should be assessed by monitoring arterial oxygen saturation. When stable for 6 hours, the patient should be reassessed for oxygen need (ie, O_2 saturation of less than 90%), and discontinuation of supplemental oxygen should be considered. (*Level of Evidence: C*)**
4. **Nursing care should be provided by individuals certified in critical care, with staffing based on the specific needs of patients and provider competencies, as well as organizational priorities. (*Level of Evidence: C*)**
5. **Care of STEMI patients in the critical care unit (CCU) should be structured around protocols derived from practice guidelines. (*Level of Evidence: C*)**
6. **Electrocardiographic monitoring leads should be based on the location and rhythm to optimize detection of ST deviation, axis shift, conduction defects, and dysrhythmias. (*Level of Evidence: B*)**

Class III

1. **It is not an effective use of the CCU environment to admit terminally ill, "do not resuscitate" patients with STEMI, because clinical and comfort needs can be provided outside of a critical care environment. (*Level of Evidence: C*)**

2. Stepdown Unit

Class I

1. **It is a useful triage strategy to admit low-risk STEMI patients who have undergone successful PCI directly to the stepdown unit for post-PCI care rather than to the CCU. (*Level of Evidence: C*)**
2. **STEMI patients originally admitted to the CCU who demonstrate 12 to 24 hours of clinical stability (absence of recurrent ischemia, heart failure, or hemodynamically compromising dysrhythmias) should be transferred to the stepdown unit. (*Level of Evidence: C*)**

Class IIa

1. **It is reasonable for patients recovering from STEMI who have clinically symptomatic heart failure to be managed on the stepdown unit, provided that facilities for continuous monitoring of pulse oximetry and appropriately skilled nurses are available. (*Level of Evidence: C*)**

2. **It is reasonable for patients recovering from STEMI who have arrhythmias that are hemodynamically well-tolerated (eg, atrial fibrillation with a controlled ventricular response; paroxysms of nonsustained VT lasting less than 30 seconds) to be managed on the stepdown unit, provided that facilities for continuous monitoring of the ECG, defibrillators, and appropriately skilled nurses are available. (*Level of Evidence: C*)**

Class IIb

1. **Patients recovering from STEMI who have clinically significant pulmonary disease requiring high-flow supplemental oxygen or noninvasive mask ventilation/bilevel positive airway pressure (BIPAP)/continuous positive airway pressure (CPAP) may be considered for care on a stepdown unit provided that facilities for continuous monitoring of pulse oximetry and appropriately skilled nurses with a sufficient nurse:patient ratio are available. (*Level of Evidence: C*)**

B. Early, General Measures

1. Level of Activity

Class IIa

1. **After 12 to 24 hours, it is reasonable to allow patients with hemodynamic instability or continued ischemia to have bedside commode privileges. (*Level of Evidence: C*)**

Class III

1. **Patients with STEMI who are free of recurrent ischemic discomfort, symptoms of heart failure, or serious disturbances of heart rhythm should not be on bed rest for more than 12 to 24 hours. (*Level of Evidence: C*)**

2. Diet

Class I

1. **Patients with STEMI should be prescribed the NCEP Adult Treatment Panel III (ATP III) Therapeutic Lifestyle Changes (TLC) diet, which focuses on reduced intake of fats and cholesterol, less than 7% of total calories as saturated fats, less than 200 mg of cholesterol per day, increased consumption of omega-3 fatty acids, and appropriate caloric intake for energy needs. (*Level of Evidence: C*)**
2. **Diabetic patients with STEMI should have an appropriate food group balance and caloric intake. (*Level of Evidence: B*)**
3. **Sodium intake should be restricted in STEMI patients with hypertension or heart failure. (*Level of Evidence: B*)**

STEMI patients should receive a reduced saturated fat and cholesterol diet per the ATP III TLC approach.[137] (See VII.L.2 and Section 7.12.2 of the full-text guidelines.)

3. Patient Education in the Hospital Setting

Class I

1. **Patient counseling to maximize adherence to evidence-based post-STEMI treatments (eg, compliance with taking medication, exercise prescription, and smoking cessation) should begin during the early phase of hospitalization, occur intensively at discharge, and continue at follow-up visits with providers and through cardiac rehabilitation programs and community support groups, as appropriate. (*Level of Evidence: C*)**
2. **Critical pathways and protocols and other quality-improvement tools (eg, the ACC "Guidelines Applied in Practice" and the AHA's "Get with the Guidelines") should be used to improve the application of evidence-based treatments by patients with STEMI, caregivers, and institutions. (*Level of Evidence: C*)**

Patient education should be viewed as a continuous process that should to be part of every patient encounter (ie, on hospital arrival, inpatient admission, discharge, and at follow-up visits).

4. Analgesia/Anxiolytics

Class IIa

1. **It is reasonable to use anxiolytic medications in STEMI patients to alleviate short-term anxiety or altered behavior related to hospitalization for STEMI. (*Level of Evidence: C*)**
2. **It is reasonable to routinely assess the patient's anxiety level and manage it with behavioral interventions and referral for counseling. (*Level of Evidence: C*)**

Anxiety and depression are prevalent in patients hospitalized for STEMI because patients are confronted with a diagnosis that is major, both psychologically and physically.[138,139] Anxiety has been demonstrated to predict in-hospital recurrent ischemia and arrhythmias[140] and cardiac events during the first year after an MI.[141]

C. Risk Stratification During Early Hospital Course

Risk stratification is a continuous process and requires the updating of initial assessments with data obtained during the hospital stay. Indicators of failed reperfusion (eg, recurrence of chest pain and persistence of ECG findings indicating infarction) identify a patient who should undergo coronary angiography. Similarly, findings consistent with mechanical complications (eg, sudden onset of heart failure or presence of a new murmur) herald increased risk and suggest the need for rapid intervention. For patients who did not undergo primary reperfusion, changes in clinical status (eg, development of shock) may herald a worsening clinical status and are an indication for coronary angiography. Patients with a low risk of complications may be candidates for early discharge. The lowest-risk patients are those who did not have STEMI despite the initial suspicions. Clinicians should strive to identify such patients within 8 to 12 hours of onset of symptoms. Serial sampling of serum cardiac biomarkers and use of 12-lead ECGs and their interpretation in the context of the number of hours that have elapsed since onset of the patient's symptoms can determine the presence of STEMI better than adherence to a rigid protocol that requires that a specified number of samples be drawn in the hospital.

D. Medication Assessment

1. Beta-Blockers

Class I

1. **Patients receiving beta-blockers within the first 24 hours of STEMI without adverse effects should continue to receive them during the early convalescent phase of STEMI. (*Level of Evidence: A*)**
2. **Patients without contraindications to beta-blockers who did not receive them within the first 24 hours after STEMI should have them started in the early convalescent phase. (*Level of Evidence: A*)**
3. **Patients with early contraindications within the first 24 hours of STEMI should be reevaluated for candidacy for beta-blocker therapy. (*Level of Evidence: C*)**

There is overwhelming evidence for the benefits of early beta-blockade in patients with STEMI and without contraindications to their use (see Section 6.3.1.5 of the full-text guidelines). Benefits have been demonstrated for patients with and without concomitant fibrinolytic therapy, both early and late after STEMI. Meta-analysis of trials from the prefibrinolytic era involving more than 24 000 patients receiving beta-blockers have shown a 14% relative risk reduction in mortality through 7 days and a 23% reduction in long-term mortality.[142]

2. Nitroglycerin

Class I

1. **Intravenous nitroglycerin is indicated in the first 48 hours after STEMI for treatment of persistent ischemia, CHF, or hypertension. The decision to administer intravenous nitroglycerin and the dose used should not preclude therapy with other proven mortality-reducing interventions, such as beta-blockers or ACE inhibitors. (*Level of Evidence: B*)**
2. **Intravenous, oral, or topical nitrates are useful beyond the first 48 hours after STEMI for treatment of recurrent angina or persistent CHF if their use does not preclude therapy with beta-blockers or ACE inhibitors. (*Level of Evidence: B*)**

Class IIb

1. **The continued use of nitrate therapy beyond the first 24 to 48 hours in the absence of continued or recurrent angina or CHF may be helpful, although the benefit is likely to be small and is not well established in contemporary practice. (*Level of Evidence: B*)**

Class III

1. **Nitrates should not be administered to patients with systolic pressure less than 90 mm Hg or greater than or equal to 30 mm Hg below baseline, severe bradycardia (less than 50 bpm), tachycardia (more than 100 bpm) or RV infarction. (*Level of Evidence: C*)**

3. Inhibition of the Renin-Angiotensin-Aldosterone System

Class I

1. **An ACE inhibitor should be administered orally during convalescence from STEMI in patients who tolerate this class of medication, and it should be continued over the long term. (*Level of Evidence: A*)**
2. **An ARB should be administered to STEMI patients who are intolerant of ACE inhibitors and have either clinical or radiological signs of heart failure or LVEF less than 0.40. Valsartan and candesartan have demonstrated efficacy for this recommendation. (*Level of Evidence: B*)**
3. **Long-term aldosterone blockade should be prescribed for post-STEMI patients without significant renal dysfunction (creatinine should be less than or equal to 2.5 mg/dL in men and less than or equal to 2.0 mg/dL in women) or hyperkalemia (potassium should be less than or equal to 5.0 mEq/L) who are already receiving therapeutic doses of an ACE inhibitor, have an LVEF less than or equal to 0.40, and have either symptomatic heart failure or diabetes. (*Level of Evidence: A*)**

Class IIa

1. **In STEMI patients who tolerate ACE inhibitors, an ARB can be useful as an alternative to ACE inhibitors provided there are either clinical or radiological signs of heart failure or LVEF is less than 0.40. Valsartan and candesartan have established efficacy for this recommendation. (*Level of Evidence: B*)**

The use of ACE inhibitors in the initial management of the STEMI patient was reviewed previously. The proportional benefit of ACE inhibitor therapy is largest in higher-risk subgroups, including those with previous infarction, heart failure, depressed LVEF, and tachycardia.[143–145] Survival benefit for patients more than 75 years old and for a low-risk subgroup without the features noted above is equivocal.[144,145]

Aldosterone blockade is another means of inhibiting the renin-angiotensin-aldosterone system that has been applied to patients in the post-STEMI setting. RALES (Randomized Aldactone Evaluation Study) and EPHESUS (Eplerenone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study) support the long-term use of an aldosterone blocker in STEMI patients with heart failure, an ejection fraction of 0.40 or less, or both, provided the serum creatinine is less than or equal to 2.5 mg/dL in men and less than or equal to 2.0 mg/dL in women and serum potassium concentration is less than or equal to 5.0 mEq/L.[146,147]

The use of ARBs after STEMI has not been explored as thoroughly as ACE inhibitors in STEMI patients.[148,149] Given the extensive randomized trial and routine clinical experience with ACE inhibitors, they remain the logical first agent for inhibition of the renin-angiotensin-aldosterone system in patients convalescing from STEMI.[150] Valsartan monotherapy (target dose 160 mg twice daily) should be administered to STEMI patients who are intolerant of ACE inhibitors and have evidence of LV dysfunction. Valsartan monotherapy can be a useful alternative to ACE inhibitors; the decision in individual patients may be influenced by physician and patient preference, cost, and anticipated side-effect profile.

4. Antiplatelets

Class I

1. **Aspirin 162 to 325 mg should be given on day 1 of STEMI and in the absence of contraindications should be continued indefinitely on a daily basis thereafter at a dose of 75 to 162 mg. (*Level of Evidence: A*)**
2. **A thienopyridine (preferably clopidogrel) should be administered to patients who are unable to take aspirin because of hypersensitivity or major gastrointestinal intolerance. (*Level of Evidence: C*)**
3. **For patients taking clopidogrel for whom CABG is planned, if possible, the drug should be withheld for at least 5 days, and preferably for 7, unless the urgency for revascularization outweighs the risks of bleeding. (*Level of Evidence: B*)**
4. **For patients who have undergone diagnostic cardiac catheterization and for whom PCI is planned, clopidogrel should be started and continued for at least 1 month after bare metal stent implantation and for several months after drug-eluting stent implantation (3 months for sirolimus, 6 months for paclitaxel) and up to 12 months in patients who are not at high risk for bleeding. (*Level of Evidence: B*)**

5. Antithrombotics

Class I

1. **Intravenous UFH (bolus of 60 U/kg, maximum 4000 U IV; initial infusion 12 U/kg per hour, maximum of 1000 U/h) or LMWH should be used in patients after STEMI who are at high risk for systemic emboli (large or anterior MI, atrial fibrillation, previous embolus, known LV thrombus, or cardiogenic shock). (*Level of Evidence: C*)**

Class IIa

1. **It is reasonable that STEMI patients not undergoing reperfusion therapy who do not have a contraindication to anticoagulation be treated with intravenous or subcutaneous UFH or with subcutaneous LMWH for at least 48 hours. In patients whose clinical condition necessitates prolonged bedrest and/or minimized activities, it is reasonable that treatment be continued until the patient is ambulatory. (*Level of Evidence: C*)**

Class IIb

1. **Prophylaxis for deep venous thrombosis (DVT) with subcutaneous LMWH (dosed appropriately for specific agent) or with subcutaneous UFH, 7500 U to 12 500 U twice per day until completely ambulatory, may be useful, but the effectiveness of such a strategy is not well established in the contemporary era of routine aspirin use and early mobilization. (*Level of Evidence: C*)**

6. Oxygen

Class I

1. **Supplemental oxygen therapy should be continued beyond the first 6 hours in STEMI patients with arterial oxygen desaturation (SaO_2 less than 90%) or overt pulmonary congestion. (*Level of Evidence: C*)**

E. Estimation of Infarct Size

Measurement of infarct size is an important element in the overall care of patients with STEMI. There are 5 major modalities that can be applied to sizing MI.

1. Electrocardiographic Techniques

Class I

1. **All patients with STEMI should have follow-up ECGs at 24 hours and at hospital discharge to assess the success of reperfusion and/or the extent of infarction, defined in part by the presence or absence of new Q waves. (*Level of Evidence: B*)**

2. Cardiac Biomarker Methods

The most widely accepted method for quantifying infarction has been the use of serial creatine kinase and the creatine kinase-MB isoenzyme.

3. Radionuclide Imaging

The most comprehensive assessment of STEMI with radionuclide imaging was developed with the technetium sestamibi SPECT approach.[151] This approach is well delineated in the ACC/AHA/ASNC Guidelines for the Clinical Use of Cardiac Radionuclide Imaging.[152]

4. Echocardiography

Global and regional LV function provides an assessment of the functional consequences of STEMI and ischemia. Readers are referred to the ACC/AHA/ASE 2003 Guideline Update for the Clinical Application of Echocardiography[153] and to Section 7.11.1.2 of the full-text STEMI guidelines.

5. Magnetic Resonance Imaging

Measurement of infarct size by MRI is a promising new technique that affords enhanced spatial resolution, thereby permitting more accurate assessment of both the transmural and circumferential extent of infarction.[154] However, additional experience and comparison with other methods of assessing infarct size are required before any clinical recommendations can be provided.

F. Hemodynamic Disturbances

1. Hemodynamic Assessment

Class I

1. **Pulmonary artery catheter monitoring should be performed for the following:**
   a. **Progressive hypotension, when unresponsive to fluid administration or when fluid administration may be contraindicated. (*Level of Evidence: C*)**
   b. **Suspected mechanical complications of STEMI, (ie, VSR, papillary muscle rupture, or free wall rupture with pericardial tamponade) if an echocardiogram has not been performed. (*Level of Evidence: C*)**
2. **Intra-arterial pressure monitoring should be performed for the following:**

a. Patients with severe hypotension (systolic arterial pressure less than 80 mm Hg). (*Level of Evidence: C*)
b. Patients receiving vasopressor/inotropic agents. (*Level of Evidence: C*)
c. Cardiogenic shock. (*Level of Evidence: C*)

Class IIa

1. Pulmonary artery catheter monitoring can be useful for the following:
   a. Hypotension in a patient without pulmonary congestion who has not responded to an initial trial of fluid administration. (*Level of Evidence: C*)
   b. Cardiogenic shock. (*Level of Evidence: C*)
   c. Severe or progressive CHF or pulmonary edema that does not respond rapidly to therapy. (*Level of Evidence: C*)
   d. Persistent signs of hypoperfusion without hypotension or pulmonary congestion. (*Level of Evidence: C*)
   e. Patients receiving vasopressor/inotropic agents. (*Level of Evidence: C*)
2. Intra-arterial pressure monitoring can be useful for patients receiving intravenous sodium nitroprusside or other potent vasodilators. (*Level of Evidence: C*)

Class IIb

1. Intra-arterial pressure monitoring might be considered in patients receiving intravenous inotropic agents. (*Level of Evidence: C*)

Class III

1. Pulmonary artery catheter monitoring is not recommended in patients with STEMI without evidence of hemodynamic instability or respiratory compromise. (*Level of Evidence: C*)
2. Intra-arterial pressure monitoring is not recommended for patients with STEMI who have no pulmonary congestion and have adequate tissue perfusion without use of circulatory support measures. (*Level of Evidence: C*)

2. Hypotension

Class I

1. Rapid volume loading with an IV infusion should be administered to patients without clinical evidence for volume overload. (*Level of Evidence: C*)
2. Rhythm disturbances or conduction abnormalities causing hypotension should be corrected. (*Level of Evidence: C*)
3. Intra-aortic balloon counterpulsation should be performed in patients who do not respond to other interventions, unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: B*)
4. Vasopressor support should be given for hypotension that does not resolve after volume loading. (*Level of Evidence: C*)
5. Echocardiography should be used to evaluate mechanical complications unless these are assessed by invasive measures. (*Level of Evidence: C*)

3. Low-Output State

Class I

1. LV function and potential presence of a mechanical complication should be assessed by echocardiography if these have not been evaluated by invasive measures. (*Level of Evidence: C*)
2. Recommended treatments for low-output states include:
   a. Inotropic support. (*Level of Evidence: B*)
   b. Intra-aortic counterpulsation. (*Level of Evidence: B*)
   c. Mechanical reperfusion with PCI or CABG. (*Level of Evidence: B*)
   d. Surgical correction of mechanical complications. (*Level of Evidence: B*)

Class III

1. Beta-blockers or calcium channel antagonists should not be administered to patients in a low-output state due to pump failure. (*Level of Evidence: B*)

A preshock state of hypoperfusion with normal blood pressure may develop before circulatory collapse and is manifested by cold extremities, cyanosis, oliguria, or decreased mentation.[155] Hospital mortality is high, so these patients should be aggressively diagnosed and treated as though they had cardiogenic shock. The initial pharmacological intervention for low cardiac output is often a dobutamine infusion. Intra-aortic counterpulsation therapy may be required to improve coronary artery perfusion pressure if hypotension is present. If the blood pressure permits, afterload-reducing agents should be added to decrease cardiac work and pulmonary congestion. Coronary artery revascularization of ischemic myocardium with either PCI or CABG has been shown to decrease mortality in patients with cardiogenic shock and is strongly recommended in suitable candidates.[75,108] Likewise, patients with VSR, papillary muscle rupture, or free wall rupture with pericardial tamponade may benefit from emergency surgical repair.

4. Pulmonary Congestion

Class I

1. Oxygen supplementation to arterial saturation greater than 90% is recommended for patients with pulmonary congestion. (*Level of Evidence: C*)
2. Morphine sulfate should be given to patients with pulmonary congestion. (*Level of Evidence: C*)
3. ACE inhibitors, beginning with titration of a short-acting ACE inhibitor with a low initial dose (eg, 1 to 6.25 mg of captopril) should be given to patients with pulmonary edema unless the systolic blood pressure is less than 100 mm Hg or more than 30 mm Hg below baseline. Patients with pulmonary congestion and marginal or low blood pressure often need circulatory support with inotropic and vasopressor agents and/or intra-aortic balloon counterpulsation to relieve pulmonary congestion and maintain adequate perfusion. (*Level of Evidence: A*)

4. **Nitrates should be administered to patients with pulmonary congestion unless the systolic blood pressure is less than 100 mm Hg or more than 30 mm Hg below baseline. Patients with pulmonary congestion and marginal or low blood pressure often need circulatory support with inotropic and vasopressor agents and/or intra-aortic balloon counterpulsation to relieve pulmonary congestion and maintain adequate perfusion. (*Level of Evidence: C*)**
5. **A diuretic (low- to intermediate-dose furosemide, or torsemide or bumetanide) should be administered to patients with pulmonary congestion if there is associated volume overload. Caution is advised for patients who have not received volume expansion. (*Level of Evidence: C*)**
6. **Beta-blockade should be initiated before discharge for secondary prevention. For those who remain in heart failure throughout the hospitalization, low doses should be initiated, with gradual titration on an outpatient basis. (*Level of Evidence: B*)**
7. **Long-term aldosterone blockade should be prescribed for post-STEMI patients without significant renal dysfunction (creatinine should be less than or equal to 2.5 mg/dL in men and less than or equal to 2.0 mg/dL in women) or hyperkalemia (potassium should be less than or equal to 5.0 mEq/L) who are already receiving therapeutic doses of an ACE inhibitor, have an LVEF less than or equal to 0.40, and have either symptomatic heart failure or diabetes. (*Level of Evidence: A*)**
8. **Echocardiography should be performed urgently to estimate LV and RV function and to exclude a mechanical complication. (*Level of Evidence: C*)**

Class IIb

1. **It may be reasonable to insert an intra-aortic balloon pump (IABP) for the management of patients with refractory pulmonary congestion. (*Level of Evidence: C*)**

Class III

1. **Beta-blockers or calcium channel blockers should not be administered acutely to STEMI patients with frank cardiac failure evidenced by pulmonary congestion or signs of a low-output state. (*Level of Evidence: B*)**

The immediate management goals include adequate oxygenation and preload reduction to relieve pulmonary congestion. Because of sympathetic stimulation, the blood pressure should be elevated in the presence of pulmonary edema. Patients with this appropriate response can typically tolerate the required medications, all of which lower blood pressure. However, iatrogenic cardiogenic shock may result from aggressive simultaneous use of agents that cause hypotension, initiating a cycle of hypoperfusion-ischemia. If acute pulmonary edema is not associated with elevation of the systemic blood pressure, impending cardiogenic shock must be suspected. If pulmonary edema is associated with hypotension, cardiogenic shock is diagnosed. Those patients often need circulatory support with inotropic and vasopressor agents and/or intra-aortic balloon counterpulsation to relieve pulmonary congestion and maintain adequate perfusion (Figure 4) (See Section VII.F.5, and see Section 7.6.5 of the full-text guidelines).

5. Cardiogenic Shock

Class I

1. **Intra-aortic balloon counterpulsation is recommended for STEMI patients when cardiogenic shock is not quickly reversed with pharmacological therapy. The IABP is a stabilizing measure for angiography and prompt revascularization. (*Level of Evidence: B*)**
2. **Intra-arterial monitoring is recommended for the management of STEMI patients with cardiogenic shock. (*Level of Evidence: C*)**
3. **Early revascularization, either PCI or CABG, is recommended for patients less than 75 years old with ST elevation or LBBB who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock, unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: A*)**
4. **Fibrinolytic therapy should be administered to STEMI patients with cardiogenic shock who are unsuitable for further invasive care and do not have contraindications to fibrinolysis. (*Level of Evidence: B*)**
5. **Echocardiography should be used to evaluate mechanical complications unless these are assessed by invasive measures. (*Level of Evidence: C*)**

Class IIa

1. **Pulmonary artery catheter monitoring can be useful for the management of STEMI patients with cardiogenic shock. (*Level of Evidence: C*)**
2. **Early revascularization, either PCI or CABG, is reasonable for selected patients 75 years or older with ST elevation or LBBB who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock. Patients with good prior functional status who agree to invasive care may be selected for such an invasive strategy. *(Level of Evidence: B*)**

Given the large overall treatment benefit of 13 lives saved per 100 patients treated in the SHOCK trial, early revascularization is recommended for those less than 75 years who are suitable for revascularization.[75,108,156] Two other large registries reported a substantial survival benefit for elderly patients who were selected clinically on the basis of physician judgment.

Interventions should be performed as soon as possible. It is recommended that patients who arrive at the hospital in cardiogenic shock (15% of cases) or who develop it after hospital arrival (85%) should be transferred to a regional tertiary care center with revascularization facilities experienced with these patients. When shock has resolved, ACE inhibitors and beta-blockers, initiated in low doses with progressive increases as recommended in the CHF guidelines, should be administered before discharge.[157] (See Section 7.6.7.6 of the full-text guidelines for discussion of mechanical support for the failing heart.)

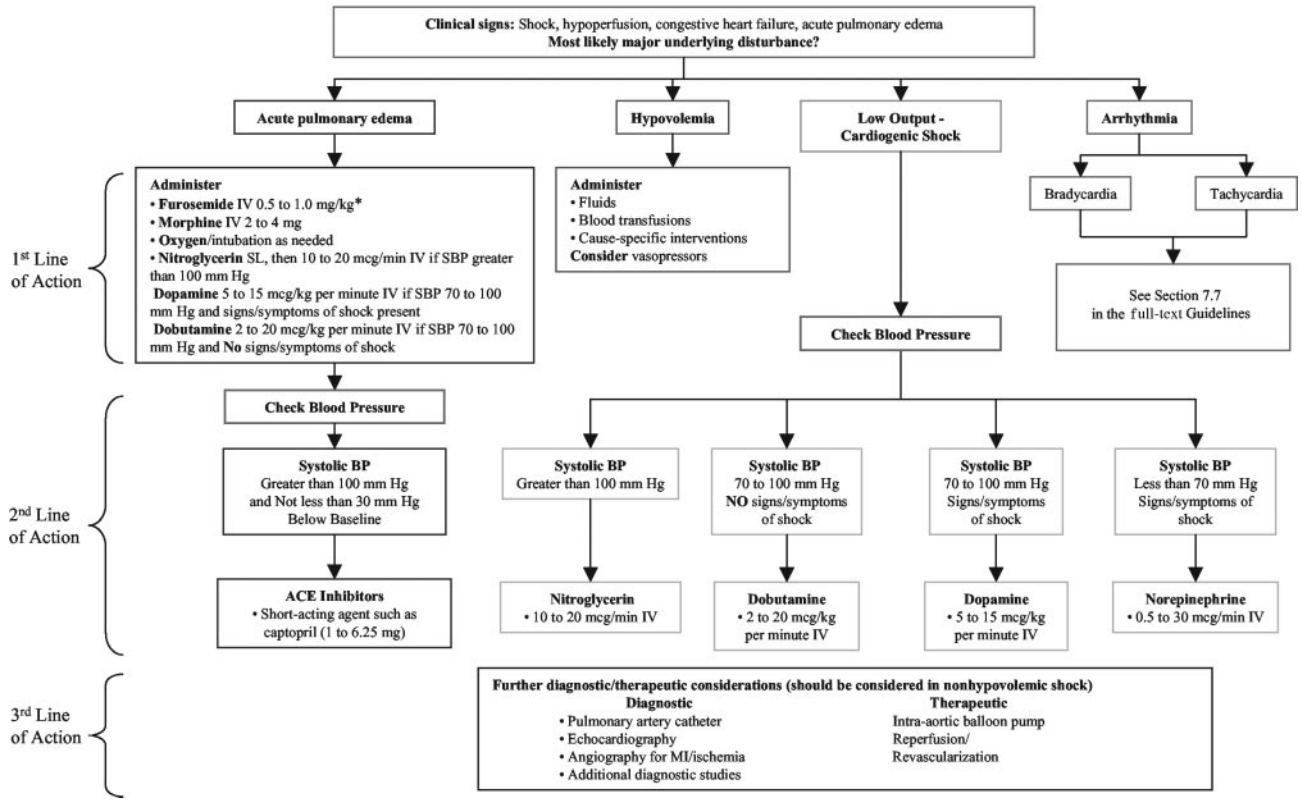

Figure 4. Emergency management of complicated ST-elevation myocardial infarction. The emergency management of patients with cardiogenic shock, acute pulmonary edema, or both is outlined. SBP indicates systolic blood pressure; IV, intravenous; BP, blood pressure; ACE, angiotensin converting enzyme; MI, myocardial infarction. *Furosemide less than 0.5 mg/kg for new-onset acute pulmonary edema without hypovolemia; 1 mg/kg for acute or chronic volume overload, renal insufficiency. Nesiritide has not been studied adequately in patients with STEMI. Combinations of medications, eg, dobutamine and dopamine, may be used. Modified with permission from Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: Part 7: The Era of Reperfusion. Section 1: Acute Coronary Syndromes (Acute Myocardial Infarction). *Circulation*. 2000;102(suppl 1):I-172–I-216.[26]

6. Right Ventricular Infarction

Class I

1. **Patients with inferior STEMI and hemodynamic compromise should be assessed with a right precordial V_4R lead to detect ST-segment elevation and an echocardiogram to screen for RV infarction. (See the ACC/AHA/ASE 2003 Guideline Update for the Clinical Application of Echocardiography.) (*Level of Evidence: B*)**
2. **The following principles apply to therapy of patients with STEMI and RV infarction and ischemic dysfunction:**
   a. **Early reperfusion should be achieved if possible. (*Level of Evidence: C*)**
   b. **AV synchrony should be achieved, and bradycardia should be corrected. (*Level of Evidence: C*)**
   c. **RV preload should be optimized, which usually requires initial volume challenge in patients with hemodynamic instability provided the jugular venous pressure is normal or low. (*Level of Evidence: C*)**
   d. **RV afterload should be optimized, which usually requires therapy for concomitant LV dysfunction. (*Level of Evidence: C*)**
   e. **Inotropic support should be used for hemodynamic instability not responsive to volume challenge. (*Level of Evidence: C*)**

Class IIa

1. **After infarction that leads to clinically significant RV dysfunction, it is reasonable to delay CABG surgery for 4 weeks to allow recovery of contractile performance. (*Level of Evidence: C*)**

Treatment of RV ischemia/infarction includes early maintenance of RV preload, reduction of RV afterload, inotropic support of the dysfunctional RV, early reperfusion,[158] and maintenance of AV synchrony.

7. Mechanical Causes of Heart Failure/Low-Output Syndrome

a. Diagnosis

On physical examination, the presence of a new cardiac murmur indicates the possibility of either a VSR or MR. A precise diagnosis can usually be established with transthoracic or transesophageal echocardiography.

b. Mitral Valve Regurgitation

Class I

1. **Patients with acute papillary muscle rupture should be considered for urgent cardiac surgical repair, unless further support is considered futile because of the**

patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: B*)
2. CABG surgery should be undertaken at the same time as mitral valve surgery. (*Level of Evidence: B*)

The patient should be stabilized with an IABP, inotropic support, and afterload reduction (to reduce regurgitant volume and pulmonary congestion) while emergency surgery is arranged.

c. Ventricular Septal Rupture After STEMI

Class I

1. Patients with STEMI complicated by the development of a VSR should be considered for urgent cardiac surgical repair, unless further support is considered futile because of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: B*)
2. CABG should be undertaken at the same time as repair of the VSR. (*Level of Evidence: B*)

Insertion of an IABP and prompt surgical referral are recommended for almost every patient with an acute VSR. Invasive monitoring is recommended in all patients, together with judicious use of inotropes and a vasodilator to maintain optimal hemodynamics. Surgical repair usually involves excision of all necrotic tissue and patch repair of the VSR, together with coronary artery grafting.

d. Left Ventricular Free-Wall Rupture

Class I

1. Patients with free-wall rupture should be considered for urgent cardiac surgical repair, unless further support is considered futile because of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: B*)
2. CABG should be undertaken at the same time as repair of free-wall rupture. (*Level of Evidence: C*)

Surgery includes repair of the ventricle by a direct suture technique or patch to cover the ventricular perforation[159] in addition to CABG as needed.

e. Left Ventricular Aneurysm

Class IIa

1. It is reasonable that patients with STEMI who develop a ventricular aneurysm associated with intractable ventricular tachyarrhythmias and/or pump failure unresponsive to medical and catheter-based therapy be considered for LV aneurysmectomy and CABG surgery. (*Level of Evidence: B*)

f. Mechanical Support of the Failing Heart

INTRA-AORTIC BALLOON COUNTERPULSATION

Class I

1. Intra-aortic balloon counterpulsation should be used in STEMI patients with hypotension (systolic blood pressure less than 90 mm Hg or 30 mm Hg below baseline mean arterial pressure) who do not respond to other interventions, unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care. See Section 7.6.2 of the full-text guidelines. (*Level of Evidence: B*)
2. Intra-aortic balloon counterpulsation is recommended for STEMI patients with low-output state. See Section 7.6.3 of the full-text guidelines. (*Level of Evidence: B*)
3. Intra-aortic balloon counterpulsation is recommended for STEMI patients when cardiogenic shock is not quickly reversed with pharmacological therapy. IABP is a stabilizing measure for angiography and prompt revascularization. See Section 7.6.5 of the full-text guidelines. (*Level of Evidence: B*)
4. Intra-aortic balloon counterpulsation should be used in addition to medical therapy for STEMI patients with recurrent ischemic-type chest discomfort and signs of hemodynamic instability, poor LV function, or a large area of myocardium at risk. Such patients should be referred urgently for cardiac catheterization and should undergo revascularization as needed. See Section 7.8.2 of the full-text guidelines. (*Level of Evidence: C*)

Class IIa

1. It is reasonable to manage STEMI patients with refractory polymorphic VT with intra-aortic balloon counterpulsation to reduce myocardial ischemia. See Section 7.7.1.2 of the full-text guidelines. (*Level of Evidence: B*)

Class IIb

1. It may be reasonable to use intra-aortic balloon counterpulsation in the management of STEMI patients with refractory pulmonary congestion. See Section 7.6.4 of the full-text guidelines. (*Level of Evidence: C*)

Selected patients with cardiogenic shock after STEMI, especially if not candidates for revascularization, may be considered for either a short- or long-term mechanical support device to serve as a bridge to recovery or to subsequent cardiac transplantation.

G. Arrhythmias After STEMI

1. Ventricular Arrhythmias

a. Ventricular Fibrillation

Class I

1. Ventricular fibrillation (VF) or pulseless VT should be treated with an unsynchronized electric shock with an initial monophasic shock energy of 200 J; if unsuccessful, a second shock of 200 to 300 J should be given, and then, if necessary, a third shock of 360 J. (*Level of Evidence: B*)

Class IIa

1. It is reasonable that VF or pulseless VT that is refractory to electrical shock be treated with amiodarone (300 mg or 5 mg/kg, IV bolus) followed by a repeat unsynchronized electric shock. (*Level of Evidence: B*)

2. It is reasonable to correct electrolyte and acid-base disturbances (potassium greater than 4.0 mEq/L and magnesium greater than 2.0 mg/dL) to prevent recurrent episodes of VF once an initial episode of VF has been treated. (*Level of Evidence: C*)

Class IIb

1. It may be reasonable to treat VT or shock-refractory VF with boluses of intravenous procainamide. However, this has limited value owing to the length of time required for administration. (*Level of Evidence: C*)

Class III

1. Prophylactic administration of antiarrhythmic therapy is not recommended when using fibrinolytic agents. (*Level of Evidence: B*)

There is no convincing evidence that the prophylactic use of lidocaine reduces mortality, and the prior practice of routine (prophylactic) administration of lidocaine to all patients with known or suspected STEMI has been largely abandoned. VF should be treated with an unsynchronized electric shock using an initial monophasic shock energy of 200 J. If this is unsuccessful, a second shock using 200 to 300 J and, if necessary, a third shock using 360 J are indicated.[160]

b. Ventricular Tachycardia

Class I

1. Sustained (more than 30 seconds or causing hemodynamic collapse) polymorphic VT should be treated with an unsynchronized electric shock with an initial monophasic shock energy of 200 J; if unsuccessful, a second shock of 200 to 300 J should be given, and, if necessary, a third shock of 360 J. (*Level of Evidence: B*)
2. Episodes of sustained monomorphic VT associated with angina, pulmonary edema, or hypotension (blood pressure less than 90 mm Hg) should be treated with a synchronized electric shock of 100 J initial monophasic shock energy. Increasing energies may be used if not initially successful. Brief anesthesia is desirable if hemodynamically tolerable. (*Level of Evidence: B*)
3. Sustained monomorphic VT not associated with angina, pulmonary edema, or hypotension (blood pressure less than 90 mm Hg) should be treated with:
   a. Amiodarone: 150 mg infused over 10 minutes (alternative dose 5 mg/kg); repeat 150 mg every 10 to 15 minutes as needed. Alternative infusion: 360 mg over 6 hours (1 mg/min), then 540 mg over the next 18 hours (0.5 mg/min). The total cumulative dose, including additional doses given during cardiac arrest, must not exceed 2.2 g over 24 hours. (*Level of Evidence: B*)
   b. Synchronized electrical cardioversion starting at monophasic energies of 50 J (brief anesthesia is necessary). (*Level of Evidence: B*)

Class IIa

1. It is reasonable to manage refractory polymorphic VT by:
   a. Aggressive attempts to reduce myocardial ischemia and adrenergic stimulation, including therapies such as beta-adrenoceptor blockade, IABP use, and consideration of emergency PCI/CABG surgery. (*Level of Evidence: B*)
   b. Aggressive normalization of serum potassium to greater than 4.0 mEq/L and of magnesium to greater than 2.0 mg/dL. (*Level of Evidence: C*)
   c. If the patient has bradycardia to a rate less than 60 beats per minute or long QTc, temporary pacing at a higher rate may be instituted. (*Level of Evidence: C*)

Class IIb

1. It is may be useful to treat sustained monomorphic VT not associated with angina, pulmonary edema, or hypotension (blood pressure less than 90 mm Hg) with a procainamide bolus and infusion. (*Level of Evidence: C*)

Class III

1. The routine use of prophylactic antiarrhythmic drugs (ie, lidocaine) is not indicated for suppression of isolated ventricular premature beats, couplets, runs of accelerated idioventricular rhythm, or nonsustained VT. (*Level of Evidence: B*)
2. The routine use of prophylactic antiarrhythmic therapy is not indicated when fibrinolytic agents are administered. (*Level of Evidence: B*)

Management Strategies for VT. Cardioversion is always indicated for episodes of sustained hemodynamically compromising VT.[161] Episodes of sustained VT that are somewhat better tolerated hemodynamically may initially be treated with drug regimens, including amiodarone or procainamide.

c. Ventricular Premature Beats

Class III

1. Treatment of isolated ventricular premature beats, couplets, and nonsustained VT is not recommended unless they lead to hemodynamic compromise. (*Level of Evidence: A*)

Before the present era of care of the STEMI patient with antiplatelet therapy, beta-blockade, ACE inhibitors, and, above all, reperfusion strategies, it was thought that ventricular warning arrhythmias preceded VF. Careful monitoring has refuted this concept, and treatment of these rhythm disturbances is not recommended unless they lead to hemodynamic compromise.

d. Accelerated Idioventricular Rhythms and Accelerated Junctional Rhythms

Class III

1. Antiarrhythmic therapy is not indicated for accelerated idioventricular rhythm. (*Level of Evidence: C*)

2. **Antiarrhythmic therapy is not indicated for accelerated junctional rhythm. (*Level of Evidence: C*)**

e. Implantable Cardioverter Defibrillator Implantation in Patients After STEMI

Class I

1. **An implantable cardioverter-defibrillator (ICD) is indicated for patients with VF or hemodynamically significant sustained VT more than 2 days after STEMI, provided the arrhythmia is not judged to be due to transient or reversible ischemia or reinfarction. (*Level of Evidence: A*)**
2. **An ICD is indicated for patients without spontaneous VF or sustained VT more than 48 hours after STEMI whose STEMI occurred at least 1 month previously, who have an LVEF between 0.31 and 0.40, demonstrate additional evidence of electrical instability (eg, nonsustained VT), and have inducible VF or sustained VT on electrophysiological testing. (*Level of Evidence: B*)**

Class IIa

1. **If there is reduced LVEF (0.30 or less), at least 1 month after STEMI and 3 months after coronary artery revascularization, it is reasonable to implant an ICD in post STEMI patients without spontaneous VF or sustained VT more than 48 hours after STEMI. (*Level of Evidence: B*)**

Class IIb

1. **The usefulness of an ICD is not well established in STEMI patients without spontaneous VF or sustained VT more than 48 hours after STEMI who have a reduced LVEF (0.31 to 0.40) at least 1 month after STEMI but who have no additional evidence of electrical instability (eg, nonsustained VT). (*Level of Evidence: B*)**
2. **The usefulness of an ICD is not well established in STEMI patients without spontaneous VF or sustained VT more than 48 hours after STEMI who have a reduced LVEF (0.31 to 0.40) at least 1 month after STEMI and additional evidence of electrical instability (eg, nonsustained VT) but who do not have inducible VF or sustained VT on electrophysiological testing. (*Level of Evidence: B*)**

Class III

1. **An ICD is not indicated in STEMI patients who do not experience spontaneous VF or sustained VT more than 48 hours after STEMI and in whom the LVEF is greater than 0.40 at least 1 month after STEMI. (*Level of Evidence: C*)**

See the full-text guidelines for discussion.

2. ***Supraventricular Arrhythmias/Atrial Fibrillation***

Class I

1. **Sustained atrial fibrillation and atrial flutter in patients with hemodynamic compromise or ongoing ischemia should be treated with one or more of the following:**
   a. **Synchronized cardioversion with an initial monophasic shock of 200 J for atrial fibrillation and 50 J for flutter, preceded by brief general anesthesia or conscious sedation whenever possible. (*Level of Evidence: C*)**
   b. **For episodes of atrial fibrillation that do not respond to electrical cardioversion or recur after a brief period of sinus rhythm, the use of antiarrhythmic therapy aimed at slowing the ventricular response is indicated. One or more of these pharmacological agents may be used:**
      i. **Intravenous amiodarone.[162] (*Level of Evidence: C*)**
      ii. **Intravenous digoxin for rate control principally for patients with severe LV dysfunction and heart failure. (*Level of Evidence: C*)**
2. **Sustained atrial fibrillation and atrial flutter in patients with ongoing ischemia but without hemodynamic compromise should be treated with one or more of the following:**
   a. **Beta-adrenergic blockade is preferred, unless contraindicated. (*Level of Evidence: C*)**
   b. **Intravenous diltiazem or verapamil. (*Level of Evidence: C*)**
   c. **Synchronized cardioversion with an initial monophasic shock of 200 J for atrial fibrillation and 50 J for flutter, preceded by brief general anesthesia or conscious sedation whenever possible. (*Level of Evidence: C*)**
3. **For episodes of sustained atrial fibrillation or flutter without hemodynamic compromise or ischemia, rate control is indicated. In addition, patients with sustained atrial fibrillation or flutter should be given anticoagulant therapy. Consideration should be given to cardioversion to sinus rhythm in patients with a history of atrial fibrillation or flutter prior to STEMI. (*Level of Evidence: C*)**
4. **Reentrant paroxysmal supraventricular tachycardia, because of its rapid rate, should be treated with the following in the sequence shown:**
   a. **Carotid sinus massage. (*Level of Evidence: C*)**
   b. **Intravenous adenosine (6 mg × 1 over 1 to 2 seconds; if no response, 12 mg IV after 1 to 2 minutes may be given; repeat 12 mg dose if needed. (*Level of Evidence: C*)**
   c. **Intravenous beta-adrenergic blockade with metoprolol (2.5 to 5.0 mg every 2 to 5 minutes to a total of 15 mg over 10 to 15 minutes) or atenolol (2.5 to 5.0 mg over 2 minutes to a total of 10 mg in 10 to 15 minutes). (*Level of Evidence: C*)**
   d. **Intravenous diltiazem (20 mg [0.25 mg/kg]) over 2 minutes followed by an infusion of 10 mg/h). (*Level of Evidence: C*)**
   e. **Intravenous digoxin, recognizing that there may be a delay of at least 1 hour before pharmacological effects appear (8 to 15 mcg/kg [0.6 to 1.0 mg in a person weighing 70 kg]). (*Level of Evidence: C*)**

Class III

1. **Treatment of atrial premature beats is not indicated. (*Level of Evidence: C*)**

See the full-text guidelines for discussion.

3. Bradyarrhythmias
See Table 3 for recommendations.

a. Acute Treatment of Conduction Disturbances and Bradyarrhythmias

VENTRICULAR ASYSTOLE

Class I

1. **Prompt resuscitative measures, including chest compressions, atropine, vasopressin, epinephrine, and temporary pacing, should be administered to treat ventricular asystole. (*Level of Evidence: B*)**

b. Use of Permanent Pacemakers

PERMANENT PACING FOR BRADYCARDIA OR CONDUCTION BLOCKS ASSOCIATED WITH STEMI

Class I

1. **Permanent ventricular pacing is indicated for persistent second-degree AV block in the His-Purkinje system with bilateral bundle-branch block or third-degree AV block within or below the His-Purkinje system after STEMI. (*Level of Evidence: B*)**
2. **Permanent ventricular pacing is indicated for transient advanced second- or third-degree infranodal AV block and associated bundle-branch block. If the site of block is uncertain, an electrophysiological study may be necessary. (*Level of Evidence: B*)**
3. **Permanent ventricular pacing is indicated for persistent and symptomatic second- or third-degree AV block. (*Level of Evidence: C*)**

Class IIb

1. **Permanent ventricular pacing may be considered for persistent second- or third-degree AV block at the AV node level. (*Level of Evidence: B*)**

Class III

1. **Permanent ventricular pacing is not recommended for transient AV block in the absence of intraventricular conduction defects. (*Level of Evidence: B*)**
2. **Permanent ventricular pacing is not recommended for transient AV block in the presence of isolated left anterior fascicular block. (*Level of Evidence: B*)**
3. **Permanent ventricular pacing is not recommended for acquired left anterior fascicular block in the absence of AV block. (*Level of Evidence: B*)**
4. **Permanent ventricular pacing is not recommended for persistent first-degree AV block in the presence of bundle-branch block that is old or of indeterminate age. (*Level of Evidence: B*)**

Indications for permanent pacing after STEMI in patients experiencing AV block are related in large measure to the presence of intraventricular conduction defects (Table 3). Unlike some other indications for permanent pacing, the criteria for patients with STEMI and AV block do not necessarily depend on the presence of symptoms. Furthermore, the requirement for temporary pacing in STEMI does not by itself constitute an indication for permanent pacing.[163]

SINUS NODE DYSFUNCTION AFTER STEMI

Class I

1. **Symptomatic sinus bradycardia, sinus pauses greater than 3 seconds, or sinus bradycardia with a heart rate less than 40 bpm and associated hypotension or signs of systemic hemodynamic compromise should be treated with an intravenous bolus of atropine 0.6 to 1.0 mg. If bradycardia is persistent and maximal (2 mg) doses of atropine have been used, transcutaneous or transvenous (preferably atrial) temporary pacing should be instituted. (*Level of Evidence: C*)**

The published ACC/AHA Guidelines[164] for Implantation of Pacemakers should be used to guide therapy in STEMI patients with persistent sinus node dysfunction.

PACING MODE SELECTION IN STEMI PATIENTS

Class I

1. **All patients who have an indication for permanent pacing after STEMI should be evaluated for ICD indications. (*Level of Evidence: C*)**

Class IIa

1. **It is reasonable to implant a permanent dual-chamber pacing system in STEMI patients who need permanent pacing and are in sinus rhythm. It is reasonable that patients in permanent atrial fibrillation or flutter receive a single-chamber ventricular device. (*Level of Evidence: C*)**
2. **It is reasonable to evaluate all patients who have an indication for permanent pacing after STEMI for biventricular pacing (cardiac resynchronization therapy). (*Level of Evidence: C*)**

When a permanent pacemaker is being considered for a post-STEMI patient, the clinician should address 2 additional questions regarding the patient: is there an indication for biventricular pacing, and is there an indication for ICD use?[165] The algorithm to define whether an ICD is indicated is contained in Figure 5.

H. Recurrent Chest Pain After STEMI

1. Pericarditis

Class I

1. **Aspirin is recommended for treatment of pericarditis after STEMI. Doses as high as 650 mg orally (enteric) every 4 to 6 hours may be needed. (*Level of Evidence: B*)**
2. **Anticoagulation should be immediately discontinued if pericardial effusion develops or increases. (*Level of Evidence: C*)**

TABLE 3. Recommendations for Treatment of Atrioventricular and Intraventricular Conduction Disturbances During STEMI

	Atrioventricular Conduction													
			First-Degree AV Block				Mobitz I Second-Degree AV Block				Mobitz II Second-Degree AV Block			
Intraventricular Conduction	Normal		Anterior MI		Nonanterior MI		Anterior MI		Nonanterior MI		Anterior MI		Nonanterior MI	
Normal	**Action**	**Class**	**Action**	**Class**	**Action**	**Class**	**Action**	**Class**	**Action**	**Class**	**Action**	**Class**	**Action**	**Class**
	Observe	I	Observe	I	Observe	I	Observe	IIb	Observe	IIa	Observe	III	Observe	III
	A	III	A	III	A	III	A*	III	A	III	A	III	A	III
	TC	III	TC	IIb	TC	IIb	TC	I	TC	I	TC	I	TC	I
	TV	III	TV	III	TV	III	TV	III	TV	III	TV	IIa	TV	IIa
Old or new fascicular block (LAFB or LPFB)	Observe	I	Observe	IIb	Observe	IIb	Observe	IIb	Observe	IIb	Observe	III	Observe	III
	A	III	A	III	A	III	A*	III	A	III	A	III	A	III
	TC	IIb	TC	I	TC	IIa	TC	I	TC	I	TC	I	TC	I
	TV	III	TV	III	TV	III	TV	III	TV	III	TV	IIa	TV	IIb
Old bundle-branch block	Observe	I	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III
	A	III	A	III	A	III	A*	III	A	III	A	III	A	III
	TC	IIb	TC	I	TC	I	TC	I	TC	I	TC	I	TC	I
	TV	III	TV	IIb	TV	IIb	TV	IIb	TV	IIb	TV	IIa	TV	IIa
New bundle-branch block	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III
	A	III	A	III	A	III	A*	III	A	III	A	III	A	III
	TC	I	TC	I	TC	I	TC	I	TC	I	TC	IIb	TC	IIb
	TV	IIb	TV	IIa	TV	IIa	TV	IIa	TV	IIa	TV	I	TV	I
Fascicular block + RBBB	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III
	A	III	A	III	A	III	A*	III	A	III	A	III	A	III
	TC	I	TC	I	TC	I	TC	I	TC	I	TC	IIb	TC	IIb
	TV	IIb	TV	IIa	TV	IIa	TV	IIa	TV	IIa	TV	I	TV	I
Alternating left and right bundle-branch block	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III
	A	III	A	III	A	III	A*	III	A	III	A	III	A	III
	TC	IIb	TC	IIb	TC	IIb	TC	IIb	TC	IIb	TC	IIb	TC	IIb
	TV	I	TV	I	TV	I	TV	I	TV	I	TV	I	TV	I

This table is designed to summarize the atrioventricular (column headings) and intraventricular (row headings) conduction disturbances that may occur during acute anterior or nonanterior STEMI, the possible treatment options, and the indications for each possible therapeutic option.

LAFB indicates left anterior fascicular block; LPFB, left posterior fascicular block; RBBB, right bundle-branch block; A, atropine; TC, transcutaneous pacing; TV, temporary transvenous pacing; STEMI, ST elevation myocardial infarction; AV, atrioventricular; and MI, myocardial infarction.

Action

There are 4 possible actions, or therapeutic options, listed and classified for each bradyarrhythmia or conduction problem:

1. Observe: continued ECG monitoring, no further action planned.
2. A, and A*: atropine administered at 0.6 to 1.0 mg IV every 5 minutes to up to 0.04 mg/kg. In general, because the increase in sinus rate with atropine is unpredictable, this is to be avoided unless there is symptomatic bradycardia that will likely respond to a vagolytic agent, such as sinus bradycardia or Mobitz I, as denoted by the asterisk, above.
3. TC: application of transcutaneous pads and standby transcutaneous pacing with no further progression to transvenous pacing imminently planned.
4. TV: temporary transvenous pacing. It is assumed, but not specified in the table, that at the discretion of the clinician, transcutaneous pads will be applied and standby transcutaneous pacing will be in effect as the patient is transferred to the fluoroscopy unit for temporary transvenous pacing.

Class

Each possible therapeutic option is further classified according to ACC/AHA criteria as I, IIa, IIb, and III.

Level of Evidence

This table was developed from (1) published observational case reports and case series, (2) published summaries, not meta-analyses, of these data; and (3) expert opinion, largely from the prereperfusion era. There are no published randomized trials comparing different strategies of managing conduction disturbances after STEMI. Thus, the level of evidence for the recommendations in this table is C.

How to Use the Table

Example: 54-year-old man is admitted with an anterior STEMI and a narrow QRS on admission. On day 1, he develops a right bundle-branch block (RBBB), with a PR interval of 0.28 seconds.

1. RBBB is an intraventricular conduction disturbance, so look at row 'New bundle-branch block.'
2. Find the column for 'First-Degree AV Block.'
3. Find the 'Action' and 'Class' cells at the convergence.
4. Note that "Observe" and "Atropine" are class III, not indicated; transcutaneous pacing (TC) is class I. Temporary transvenous pacing (TV) is class IIa.

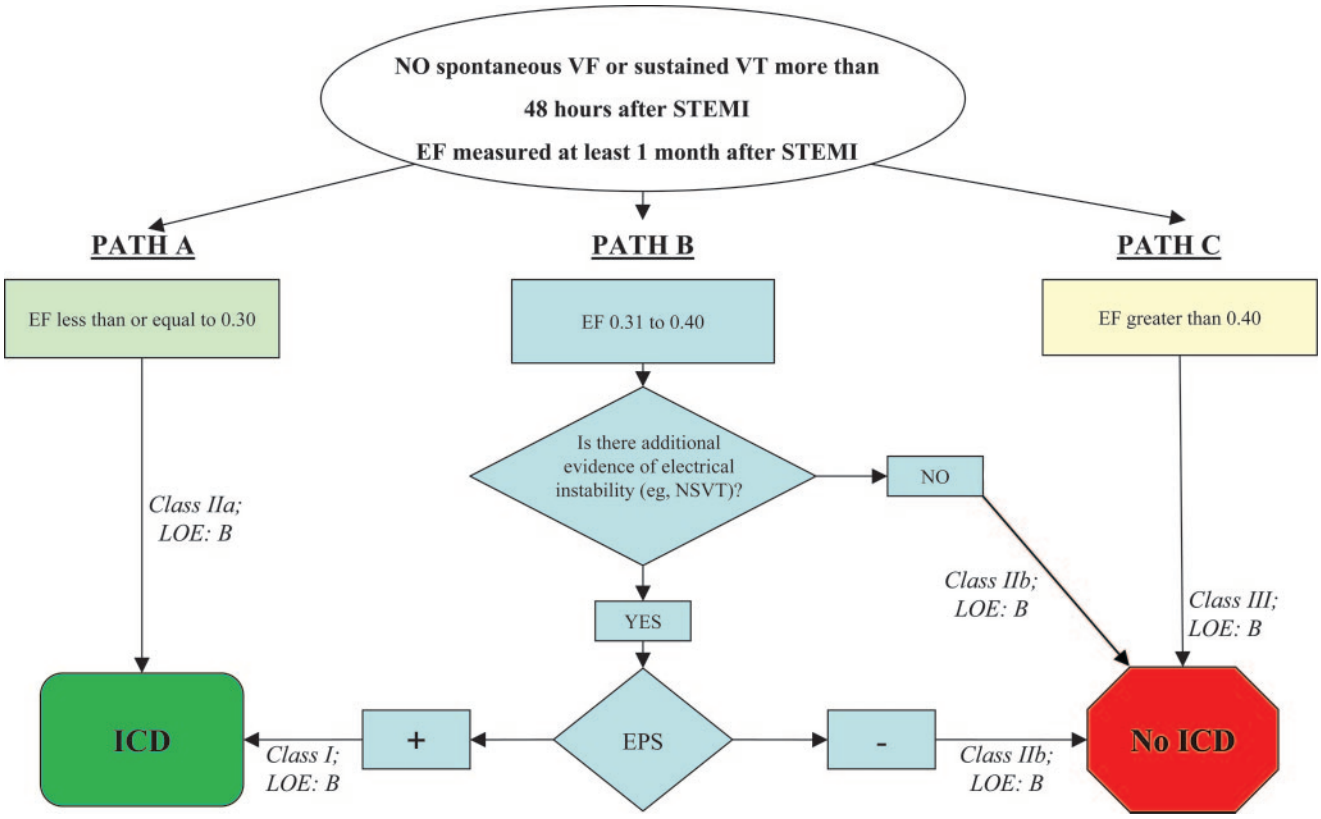

Figure 5. Algorithm to aid in selection of ICD in patients with STEMI and diminished ejection fraction (EF). Appropriate management path is selected based on LVEF measured at least 1 month after STEMI. These criteria, which are based on published data, form the basis for the full-text guidelines in Section 7.7.1.5. All patients, whether an ICD is implanted or not, should receive medical therapy as outlined in the guidelines. VF indicates ventricular fibrillation; VII, ventricular tachycardia; STEMI, ST-elevation myocardial infarction; NSVT, nonsustained VT; LOE, level of evidence; EPS, electrophysiological studies; LVEF, left ventricular EF.

Class IIa

1. **For episodes of pericarditis after STEMI that are not adequately controlled with aspirin, it is reasonable to administer 1 or more of the following:**
   a. **Colchicine 0.6 mg every 12 hours orally. (*Level of Evidence: B*)**
   b. **Acetaminophen 500 mg orally every 6 hours. (*Level of Evidence: C*)**

Class IIb

1. **Nonsteroidal anti-inflammatory drugs may be considered for pain relief; however, they should not be used for extended periods because of their continuous effect on platelet function, an increased risk of myocardial scar thinning, and infarct expansion. (*Level of Evidence: B*)**
2. **Corticosteroids might be considered only as a last resort in patients with pericarditis refractory to aspirin or nonsteroidal drugs. Although corticosteroids are effective for pain relief, their use is associated with an increased risk of scar thinning and myocardial rupture. (*Level of Evidence: C*)**

Class III

1. **Ibuprofen should not be used for pain relief because it blocks the antiplatelet effect of aspirin and can cause myocardial scar thinning and infarct expansion. (*Level of Evidence: B*)**

2. Recurrent Ischemia/Infarction

Class I

1. **Patients with recurrent ischemic-type chest discomfort after initial reperfusion therapy for STEMI should undergo escalation of medical therapy with nitrates and beta-blockers to decrease myocardial oxygen demand and reduce ischemia. Intravenous anticoagulation should be initiated if not already accomplished. (*Level of Evidence: B*)**
2. **In addition to escalation of medical therapy, patients with recurrent ischemic-type chest discomfort and signs of hemodynamic instability, poor LV function, or a large area of myocardium at risk should be referred urgently for cardiac catheterization and undergo revascularization as needed. Insertion of an IABP should also be considered. (*Level of Evidence: C*)**
3. **Patients with recurrent ischemic-type chest discomfort who are considered candidates for revascularization should undergo coronary arteriography and PCI or CABG as dictated by coronary anatomy. (*Level of Evidence: B*)**

Class IIa

1. **It is reasonable to (re)administer fibrinolytic therapy to patients with recurrent ST elevation and ischemic-type chest discomfort who are not considered candi-**

dates for revascularization or for whom coronary angiography and PCI cannot be rapidly (ideally within 60 minutes from the onset of recurrent discomfort) implemented. (*Level of Evidence: C*)

Class III

1. **Streptokinase should not be readministered to treat recurrent ischemia/infarction in patients who received a non–fibrin-specific fibrinolytic agent more than 5 days previously to treat the acute STEMI event. (*Level of Evidence: C*)**

Patients with recurrent ischemic-type chest discomfort should undergo escalation of medical therapy that includes beta-blockers (intravenously and then orally) and nitrates (sublingually and then intravenously); consideration should be given to initiation of intravenous anticoagulation if the patient is not already therapeutically anticoagulated. Secondary causes of recurrent ischemia, such as poorly controlled heart failure, anemia, and arrhythmias, should be corrected.

I. Other Complications

1. *Ischemic Stroke*

Class I

1. **Neurological consultation should be obtained in STEMI patients who have an acute ischemic stroke. (*Level of Evidence: C*)**
2. **STEMI patients who have an acute ischemic stroke should be evaluated with echocardiography, neuroimaging, and vascular imaging studies to determine the cause of the stroke. (*Level of Evidence: C*)**
3. **STEMI patients with acute ischemic stroke and persistent atrial fibrillation should receive lifelong moderate-intensity (international normalized ratio [INR] 2 to 3) warfarin therapy. (*Level of Evidence: A*)**
4. **STEMI patients with or without acute ischemic stroke who have a cardiac source of embolism (atrial fibrillation, mural thrombus, or akinetic segment) should receive moderate-intensity (INR 2 to 3) warfarin therapy (in addition to aspirin). The duration of warfarin therapy should be dictated by clinical circumstances (eg, at least 3 months for patients with an LV mural thrombus or akinetic segment and indefinitely in patients with persistent atrial fibrillation). The patient should receive LMWH or UFH until adequately anticoagulated with warfarin. (*Level of Evidence: B*)**

Class IIa

1. **It is reasonable to assess the risk of ischemic stroke in patients with STEMI. (*Level of Evidence: A*)**
2. **It is reasonable that STEMI patients with nonfatal acute ischemic stroke receive supportive care to minimize complications and maximize functional outcome. (*Level of Evidence: C*)**

Class IIb

1. **Carotid angioplasty/stenting, 4 to 6 weeks after ischemic stroke, might be considered in STEMI patients who have an acute ischemic stroke attributable to an internal carotid artery–origin stenosis of at least 50% and who have a high surgical risk of morbidity/mortality early after STEMI. (*Level of Evidence: C*)**

An algorithm for evaluation and antithrombotic therapy for ischemic stroke is shown in Figure 35 of the full-text guideline.

2. *DVT and Pulmonary Embolism*

Class I

1. **DVT or pulmonary embolism after STEMI should be treated with full-dose LMWH for a minimum of 5 days and until the patient is adequately anticoagulated with warfarin. Start warfarin concurrently with LMWH and titrate to INR of 2 to 3. (*Level of Evidence: A*)**
2. **Patients with CHF after STEMI who are hospitalized for prolonged periods, unable to ambulate, or considered at high risk for DVT and are not otherwise anticoagulated should receive low-dose heparin prophylaxis, preferably with LMWH. (*Level of Evidence: A*)**

J. CABG Surgery After STEMI

1. *Timing of Surgery*

Class IIa

1. **In patients who have had a STEMI, CABG mortality is elevated for the first 3 to 7 days after infarction, and the benefit of revascularization must be balanced against this increased risk. Patients who have been stabilized (no ongoing ischemia, hemodynamic compromise, or life-threatening arrhythmia) after STEMI and who have incurred a significant fall in LV function should have their surgery delayed to allow myocardial recovery to occur. If critical anatomy exists, revascularization should be undertaken during the index hospitalization. (*Level of Evidence: B*)**

The Writing Committee believes that if stable STEMI patients with preserved LV function require surgical revascularization, then CABG can be undertaken within several days of the infarction without an increased risk.

2. *Arterial Grafting*

Class I

1. **An internal mammary artery graft to a significantly stenosed left anterior descending coronary artery should be used whenever possible in patients undergoing CABG after STEMI. (*Level of Evidence: B*)**

3. *CABG for Recurrent Ischemia After STEMI*

Class I

1. **Urgent CABG is indicated if the coronary angiogram reveals anatomy that is unsuitable for PCI. (*Level of Evidence: B*)**

4. Elective CABG Surgery After STEMI in Patients With Angina

Class I

1. **CABG is recommended for patients with stable angina who have significant left main coronary artery stenosis. (*Level of Evidence: A*)**
2. **CABG is recommended for patients with stable angina who have left main equivalent disease: significant (at least 70%) stenosis of the proximal left anterior descending coronary artery and proximal left circumflex artery. (*Level of Evidence: A*)**
3. **CABG is recommended for patients with stable angina who have 3-vessel disease (Survival benefit is greater when LVEF is less than 0.50). (*Level of Evidence: A*)**
4. **CABG is beneficial for patients with stable angina who have 1- or 2-vessel coronary disease without significant proximal left anterior descending coronary artery stenosis but with a large area of viable myocardium and high-risk criteria on noninvasive testing. (*Level of Evidence: B*)**
5. **CABG is recommended in patients with stable angina who have 2-vessel disease with significant proximal left anterior descending coronary artery stenosis and either ejection fraction less than 0.50 or demonstrable ischemia on noninvasive testing. (*Level of Evidence: A*)**

The role of surgical revascularization has been reviewed extensively in the ACC/AHA Guidelines for CABG Surgery.[166] Consideration for revascularization after STEMI includes PCI and CABG. Providers should individualize patient management on the basis of clinical circumstances, available revascularization options, and patient preference.

5. CABG Surgery After STEMI and Antiplatelet Agents

Class I

1. **Aspirin should not be withheld before elective or nonelective CABG after STEMI. (*Level of Evidence: C*)**
2. **Aspirin (75 to 325 mg daily) should be prescribed as soon as possible (within 24 hours) after CABG unless contraindicated. (*Level of Evidence: B*)**
3. **In patients taking clopidogrel in whom elective CABG is planned, the drug should be withheld for 5 to 7 days. (*Level of Evidence: B*)**

STEMI patients undergoing revascularization frequently receive 1 or more antiplatelet agents and heparin, all of which may increase risk of serious bleeding during and after cardiac surgery. Delaying surgery until platelet function has recovered may not be feasible in many circumstances. In patients treated with the small-molecule GP IIb/IIIa receptor antagonists, tirofiban and eptifibatide, platelet function returns toward normal within 4 hours of stopping treatment. Platelet aggregation does not return toward normal for more than 48 hours in patients treated with abciximab. Management strategies, other than delaying surgery, include platelet transfusions for patients who were recently treated with abciximab, reduced heparin dosing during cardiopulmonary bypass, and possible use of antifibrinolytic agents such as aprotinin or tranexamic acid.[167] Because clopidogrel, when added to aspirin, increases the risk of bleeding during major surgery in patients who are scheduled for elective CABG, clopidogrel should be withheld for at least 5 days[168] and preferably for 7 days before surgery.[169]

K. Convalescence, Discharge, and Post-MI Care

1. Risk Stratification at Hospital Discharge

The risk stratification approach for decision-making about catheterization is described in Figure 6. The suggested algorithm for electrophysiological testing and ICD placement is shown in Figure 5.

a. Role of Exercise Testing

Class I

1. **Exercise testing should be performed either in the hospital or early after discharge in STEMI patients not selected for cardiac catheterization and without high-risk features to assess the presence and extent of inducible ischemia. (*Level of Evidence: B*)**
2. **In patients with baseline abnormalities that compromise ECG interpretation, echocardiography or myocardial perfusion imaging should be added to standard exercise testing. (*Level of Evidence: B*)**

Class IIb

1. **Exercise testing might be considered before discharge of patients recovering from STEMI to guide the post-discharge exercise prescription or to evaluate the functional significance of a coronary lesion previously identified at angiography. (*Level of Evidence: C*)**

Class III

1. **Exercise testing should not be performed within 2 to 3 days of STEMI in patients who have not undergone successful reperfusion. (*Level of Evidence: C*)**
2. **Exercise testing should not be performed to evaluate patients with STEMI who have unstable postinfarction angina, decompensated CHF, life-threatening cardiac arrhythmias, noncardiac conditions that severely limit their ability to exercise, or other absolute contraindications to exercise testing.[170] (*Level of Evidence: C*)**
3. **Exercise testing should not be used for risk stratification in patients with STEMI who have already been selected for cardiac catheterization. (*Level of Evidence: C*)**

Exercise testing after STEMI may be performed to (1) assess functional capacity and the patient's ability to perform tasks at home and at work; (2) establish exercise parameters for cardiac rehabilitation; (3) evaluate the efficacy of the patient's current medical regimen; (4) risk-stratify the post-STEMI patient according to the likelihood of a subsequent cardiac event;[171–175] (5) evaluate chest pain symptoms after STEMI; and (6) provide reassurance to patients regarding their functional capacity after STEMI as a guide to returning to work.

b. Role of Echocardiography

Noninvasive imaging in patients recovering from STEMI includes echocardiography and radionuclide imaging. This section discusses the role of echocardiography. (See Sections

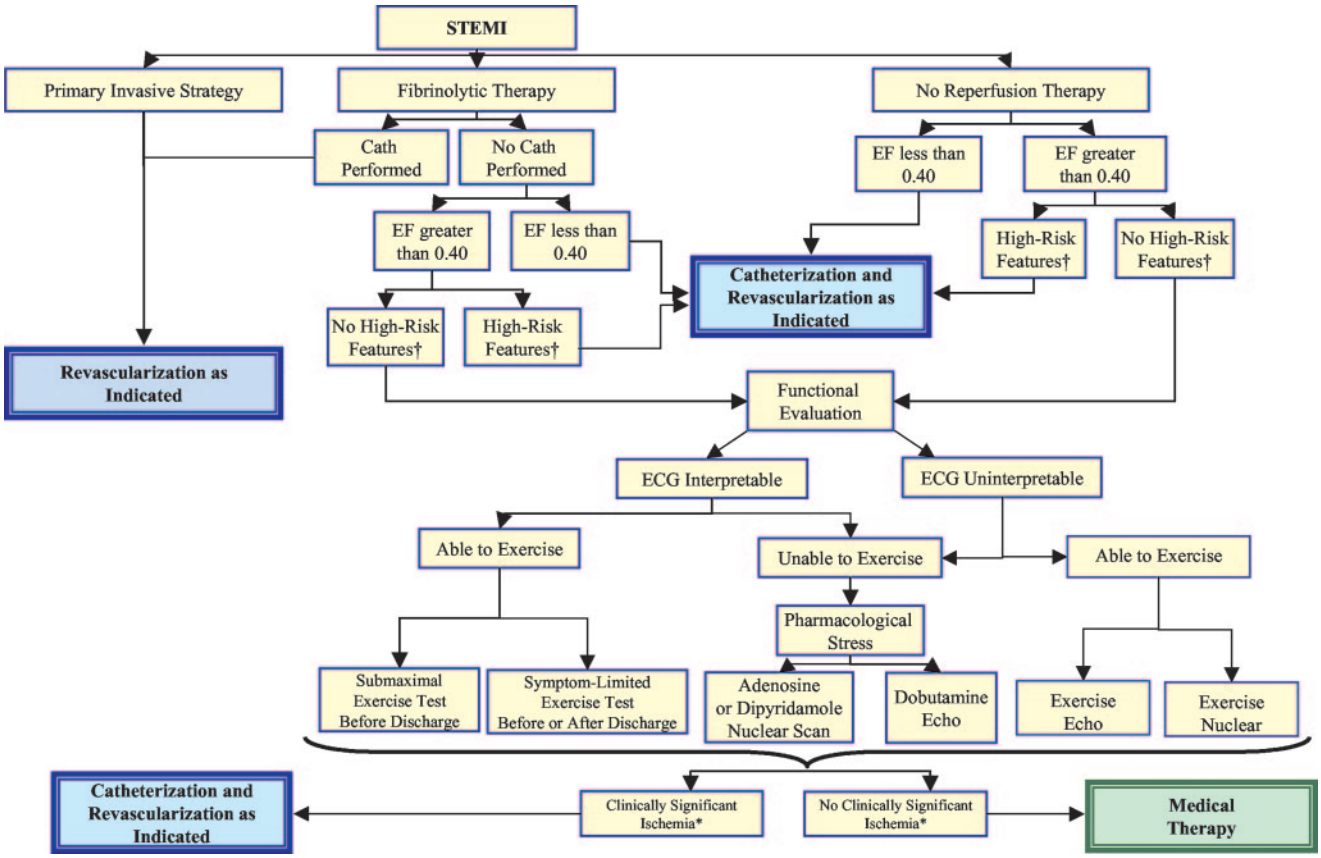

Figure 6. Evidence-based approach to need for catheterization (cath) and revascularization after STEMI. This algorithm shows treatment paths for patients who initially undergo a primary invasive strategy, receive fibrinolytic therapy, or do not undergo reperfusion therapy for STEMI. Patients who have not undergone a primary invasive strategy and have no high-risk features should undergo functional evaluation with one of the noninvasive tests shown. When clinically significant ischemia is detected, patients should undergo catheterization and revascularization as indicated; if no clinically significant ischemia is detected, medical therapy is prescribed after STEMI. *Please see Table 23 of the ACC/AHA Guidelines for the Management of Patients With Chronic Stable Angina for further definition. †Please see Table 3, Section 6.3.1.6.2., and Section 7.3. in the full-text STEMI guidelines for further discussion. STEMI indicates ST-elevation myocardial infarction; EF, ejection fraction; ECG, electrocardiography.

7.11.1.3, 7.11.1.4, and 7.11.1.5 of the full-text guidelines for additional discussion on imaging considerations.)

Class I

1. **Echocardiography should be used in patients with STEMI not undergoing LV angiography to assess baseline LV function, especially if the patient is hemodynamically unstable. (*Level of Evidence: C*)**
2. **Echocardiography should be used to evaluate patients with inferior STEMI, clinical instability, and clinical suspicion of RV infarction. (See ACC/AHA Guidelines for Clinical Application of Echocardiography.[153]) (*Level of Evidence: C*)**
3. **Echocardiography should be used in patients with STEMI to evaluate suspected complications, including acute MR, cardiogenic shock, infarct expansion, VSR, intracardiac thrombus, and pericardial effusion. (*Level of Evidence: C*)**
4. **Stress echocardiography (or myocardial perfusion imaging) should be used in patients with STEMI for in-hospital or early postdischarge assessment for inducible ischemia when baseline abnormalities are expected to compromise ECG interpretation. (*Level of Evidence: C*)**

Class IIa

1. **Echocardiography is reasonable in patients with STEMI to re-evaluate ventricular function during recovery when results are used to guide therapy. (*Level of Evidence: C*)**
2. **Dobutamine echocardiography (or myocardial perfusion imaging) is reasonable in hemodynamically and electrically stable patients 4 or more days after STEMI to assess myocardial viability when required to define the potential efficacy of revascularization. (*Level of Evidence: C*)**
3. **In STEMI patients who have not undergone contrast ventriculography, echocardiography is reasonable to assess ventricular function after revascularization. (*Level of Evidence: C*)**

Class III

1. **Echocardiography should not be used for early routine reevaluation in patients with STEMI in the absence of any change in clinical status or revascularization procedure. Reassessment of LV function 30 to 90 days later may be reasonable. (*Level of Evidence: C*)**

The use of echocardiography in STEMI is discussed in detail in the ACC/AHA/ASE 2003 Guideline Update for the Clinical Application of Echocardiography.[153]

c. Exercise Myocardial Perfusion Imaging

Noninvasive imaging in patients recovering from STEMI includes echocardiography and radionuclide imaging. This section discusses the role of exercise myocardial perfusion imaging. (See Sections 7.11.1.2, 7.11.1.4, and 7.11.1.5 of the full-text guidelines for additional discussion on imaging considerations.)

Class I

1. **Dipyridamole or adenosine stress perfusion nuclear scintigraphy or dobutamine echocardiography before or early after discharge should be used in patients with STEMI who are not undergoing cardiac catheterization to look for inducible ischemia in patients judged to be unable to exercise. (*Level of Evidence: B*)**

Class IIa

1. **Myocardial perfusion imaging or dobutamine echocardiography is reasonable in hemodynamically and electrically stable patients 4 to 10 days after STEMI to assess myocardial viability when required to define the potential efficacy of revascularization. (*Level of Evidence: C*)**

Recommended strategies for exercise test evaluations after STEMI are presented in Figure 6. These strategies and the data on which they are based are reviewed in more detail in the ACC/AHA 2002 Guideline Update for Exercise Testing.[170]

d. LV Function

Noninvasive imaging in patients recovering from STEMI includes echocardiography and radionuclide imaging. This section discusses the importance of measurement of LV function. Either of the above imaging techniques can provide clinically useful information.

Class I

1. **LVEF should be measured in all STEMI patients. (*Level of Evidence: B*)**

Assessment of LV function after STEMI has been shown to be one of the most accurate predictors of future cardiac events in both the prereperfusion[176] and the reperfusion eras.[177,178] Multiple techniques for assessing LV function of patients after STEMI have important prognostic value. Because of the dynamic nature of LV function recovery after STEMI, clinicians should consider the timing of the imaging study relative to the index event when assessing LV function. (See Table 6 of the ACC/AHA/ASE 2003 Guideline Update on the Clinical Application of Echocardiography for further discussion of the impact of timing on assessment of LV function and inducible ischemia.)[153]

e. Invasive Evaluation

Class I

1. **Coronary arteriography should be performed in patients with spontaneous episodes of myocardial ischemia or episodes of myocardial ischemia provoked by minimal exertion during recovery from STEMI. (*Level of Evidence: A*)**
2. **Coronary arteriography should be performed for intermediate- or high-risk findings on noninvasive testing after STEMI (see Table 23 of the ACC/AHA 2002 Guideline Update for the Management of Patients With Chronic Stable Angina).[179] (*Level of Evidence: B*)**
3. **Coronary arteriography should be performed if the patient is sufficiently stable before definitive therapy of a mechanical complication of STEMI, such as acute MR, VSR, pseudoaneurysm, or LV aneurysm. (*Level of Evidence: B*)**
4. **Coronary arteriography should be performed in patients with persistent hemodynamic instability. (*Level of Evidence: B*)**
5. **Coronary arteriography should be performed in survivors of STEMI who had clinical heart failure during the acute episode but subsequently demonstrated well-preserved LV function. (*Level of Evidence: C*)**

Class IIa

1. **It is reasonable to perform coronary arteriography when STEMI is suspected to have occurred by a mechanism other than thrombotic occlusion of an atherosclerotic plaque. This would include coronary embolism, certain metabolic or hematological diseases, or coronary artery spasm. (*Level of Evidence: C*)**
2. **Coronary arteriography is reasonable in STEMI patients with any of the following: diabetes mellitus, LVEF less than 0.40, CHF, prior revascularization, or life-threatening ventricular arrhythmias. (*Level of Evidence: C*)**

Class IIb

1. **Catheterization and revascularization may be considered as part of a strategy of routine coronary arteriography for risk assessment after fibrinolytic therapy (See Section 6.3.1.6.4.7 of the full-text guidelines). (*Level of Evidence: B*)**

Class III

1. **Coronary arteriography should not be performed in survivors of STEMI who are thought not to be candidates for coronary revascularization. (*Level of Evidence: A*)**

The Writing Committee encourages contemporary research into the benefit of routine catheterization versus watchful waiting after fibrinolytic therapy in the contemporary era.[180] (See Section 6.3.1.6.4.7 of the full-text guidelines)

f. Assessment of Ventricular Arrhythmias

Class IIb

1. **Noninvasive assessment of the risk of ventricular arrhythmias may be considered (including signal-averaged ECG, 24-hour ambulatory monitoring, heart rate variability, micro T-wave alternans, and T-wave variability) in patients recovering from STEMI. (*Level of Evidence: B*)**

The clinical applicability of these tests to the post-STEMI patient is in a state of evolution. Until these issues are resolved, use these tests are used only to support routine management and risk assessment.

L. Secondary Prevention

Class I

1. **Patients who survive the acute phase of STEMI should have plans initiated for secondary prevention therapies. (*Level of Evidence: A*)**

Secondary prevention therapies, unless contraindicated, are an essential part of the management of all patients with STEMI (Table 4),[181] regardless of sex.[182,183] Inasmuch as atherosclerotic vascular disease is frequently found in multiple vascular beds, the physician should search for symptoms or signs of peripheral vascular disease or cerebrovascular disease in patients presenting with STEMI.

1. Patient Education Before Discharge

Class I

1. **Before hospital discharge, all STEMI patients should be educated about and actively involved in planning for adherence to the lifestyle changes and drug therapies that are important for the secondary prevention of cardiovascular disease. (*Level of Evidence: B*)**
2. **Post-STEMI patients and their family members should receive discharge instructions about recognizing acute cardiac symptoms and appropriate actions to take in response (ie, calling 9-1-1 if symptoms are unimproved or worsening 5 minutes after onset, or if symptoms are unimproved or worsening 5 minutes after 1 sublingual nitroglycerin dose) to ensure early evaluation and treatment should symptoms recur. (*Level of Evidence: C*)**
3. **Family members of STEMI patients should be advised to learn about AEDs and CPR and be referred to a CPR training program. Ideally, such training programs would have a social support component targeting family members of high-risk patients. (*Level of Evidence: C*)**

2. Lipid Management

Class I

1. **Dietary therapy that is low in saturated fat and cholesterol (less than 7% of total calories as saturated fat and less than 200 mg/d cholesterol) should be started on discharge after recovery from STEMI. Increased consumption of the following should be encouraged: omega–3 fatty acids, fruits, vegetables, soluble (viscous) fiber, and whole grains. Calorie intake should be balanced with energy output to achieve and maintain a healthy weight. (*Level of Evidence: A*)**
2. **A lipid profile should be obtained from past records, but if not available, it should be performed in all patients with STEMI, preferably after they have fasted and within 24 hours of admission. (*Level of Evidence: C*)**
3. **The target LDL-C level after STEMI should be *substantially* less than 100 mg/dL. (*Level of Evidence: A*)**
   a. **Patients with LDL-C 100 mg/dl or above should be prescribed drug therapy on hospital discharge, with preference given to statins. (*Level of Evidence: A*)**
   b. **Patients with LDL-C less than 100 mg/dL or unknown LDL-C levels should be prescribed statin therapy on hospital discharge. (*Level of Evidence: B*)**
4. **Patients with non–high-density lipoprotein cholesterol (non HDL-C) levels less than 130 mg/dL who have an HDL-C level less than 40 mg/dL should receive special emphasis on nonpharmacological therapy (eg, exercise, weight loss, and smoking cessation) to increase HDL-C. (*Level of Evidence: B*)**

Class IIa

1. **It is reasonable to prescribe drug therapy at discharge to patients with non–HDL-C greater than or equal to 130 mg/dL, with a goal of reducing non–HDL-C to *substantially* less than 130 mg/dL. (*Level of Evidence: B*)**
2. **It is reasonable to prescribe drug therapy such as niacin or fibrate therapy to raise HDL-C levels in patients with LDL-C less than 100 mg/dL and non–HDL-C less than 130 mg/dL but HDL-C less than 40 mg/dL despite dietary and other nonpharmacological therapy. (*Level of Evidence: B*) Dietary-supplement niacin must not be used as a substitute for prescription niacin, and over-the-counter niacin should be used only if approved and monitored by a physician.**
3. **It is reasonable to add drug therapy with either niacin or a fibrate to diet regardless of LDL-C and HDL-C levels when triglyceride levels are greater than 500 mg/dL. In this setting, non–HDL-C (goal *substantially* less than 130 mg/dL) should be the cholesterol target rather than LDL-C. (*Level of Evidence: B*) Dietary-supplement niacin must not be used as a substitute for prescription niacin, and over-the-counter niacin should be used only if approved and monitored by a physician.**

Early secondary prevention trials conducted before the use of statin therapy, which used then-available drugs and diet to lower cholesterol, demonstrated significant reductions of 25% in nonfatal MIs and 14% in fatal MIs.[14] Subsequently, a growing body of evidence, mainly from large randomized clinical trials of statin therapy, has firmly established the desirability of lowering atherogenic serum lipids in patients who have recovered from a STEMI. See Table 4 for additional discussion of recommendations.

3. Weight Management

Class I

1. **Measurement of waist circumference and calculation of body mass index are recommended. Desirable body mass index range is 18.5 to 24.9 kg/m^2. A waist circumference greater than 40 inches in men and 35 inches in women would result in evaluation for metabolic syndrome and implementation of weight-reduction strategies. (*Level of Evidence: B*)**
2. **Patients should be advised about appropriate strategies for weight management and physical activity**

TABLE 4. Secondary Prevention for STEMI Patients

Goals	Intervention Recommendations
Smoking: Goal complete cessation	Assess tobacco use. Strongly encourage patient and family to stop smoking and to avoid secondhand smoke. Provide counseling, pharmacological therapy (including nicotine replacement and bupropion), and formal smoking cessation programs as appropriate.
Blood pressure control: Goal Less than 140/90 mm Hg or Less than 130/80 mm Hg if chronic kidney disease or diabetes	If blood pressure is 120/80 mm Hg or greater: • Initiate lifestyle modification (weight control, physical activity, alcohol moderation, moderate sodium restriction, and emphasis on fruits, vegetables, and low-fat dairy products) in all patients. If blood pressure is 140/90 mm Hg or greater or 130/80 mm Hg or greater for individuals with chronic kidney disease or diabetes: • Add blood pressure medications, emphasizing the use of beta-blockers and inhibition of the renin-angiotensin-aldosterone system.
Lipid management: (TG less than 200 mg/dL) Primary goal LDL-C *substantially* less than 100 mg/dL	Start dietary therapy in all patients (less than 7% of total calories as saturated fat and less than 200 mg/d cholesterol). Promote physical activity and weight management. Encourage increased consumption of omega-3 fatty acids. Assess fasting lipid profile in all patients, preferably within 24 hours of STEMI. Add drug therapy according to the following guide: LDL-C *substantially* less than 100 mg/dL (baseline or on-treatment): • Statins should be used to lower LDL-C. LDL-C greater than or equal to 100 mg/dL (baseline or on-treatment): • Intensify LDL-C–lowering therapy with drug treatment, giving preference to statins.
Lipid management: (TG 200 mg/dL or greater) Primary goal Non–HDL-C* *substantially* less than 130 mg/dL	If TG is greater than or equal to 150 mg/dL or HDL-C is less than 40 mg/dL: • Emphasize weight management and physical activity. Advise smoking cessation. If TG is 200 to 499 mg/dL: • After LDL-C–lowering therapy,† consider adding fibrate or niacin.‡ If TG is greater than or equal to 500 mg/dL: • Consider fibrate or niacin‡ before LDL-C–lowering therapy.† • Consider omega-3 fatty acids as adjunct for high TG.
Physical activity: Minimum goal 30 minutes 3 to 4 days per week; Optimal daily	Assess risk, preferably with exercise test, to guide prescription. Encourage minimum of 30 to 60 minutes of activity, preferably daily, or at least 3 or 4 times weekly (walking, jogging, cycling, or other aerobic activity) supplemented by an increase in daily lifestyle activities (eg, walking breaks at work, gardening, household work). Cardiac rehabilitation/secondary prevention programs, when available, are recommended for patients with STEMI, particularly those with multiple modifiable risk factors and/or those moderate- to high-risk patients in whom supervised exercise training is warranted.
Weight management: Goal BMI 18.5–24.9 kg/m^2 Waist circumference: Women: Less than 35 inches Men: Less than 40 inches	Calculate BMI and measure waist circumference as part of evaluation. Monitor response of BMI and waist circumference to therapy. Start weight management and physical activity as appropriate. Desirable BMI range is 18.5 to 24.9 kg/m^2. If waist circumference is greater than or equal to 35 inches in women or greater than or equal to 40 inches in men, initiate lifestyle changes and treatment strategies for metabolic syndrome.
Diabetes management: Goal HbA1c less than 7%	Appropriate hypoglycemic therapy to achieve near-normal fasting plasma glucose, as indicated by HbA1c. Treatment of other risks (eg, physical activity, weight management, blood pressure, and cholesterol management).
Antiplatelet agents/anticoagulants:	Start and continue indefinitely aspirin 75 to 162 mg/d if not contraindicated. Consider clopidogrel 75 mg/d or warfarin if aspirin is contraindicated. Manage warfarin to INR of 2.5 to 3.5 in post-STEMI patients when clinically indicated or for those not able to take aspirin or clopidogrel (Figure 7).
Renin-Angiotensin-Aldosterone System Blockers:	ACE inhibitors in all patients indefinitely; start, early in stable high-risk patients (anterior MI, previous MI, Killip class greater than or equal to II [S_3 gallop, rales, radiographic CHF], LVEF less than 0.40). ARBs in patients who are intolerant of ACE inhibitors and who have either clinical or radiological signs of heart failure or LVEF less than 0.40. Aldosterone blockade in patients without significant renal dysfunction§ or hyperkalemia who are already receiving therapeutic doses of an ACE inhibitor, have an LVEF less than or equal to 0.40, and have either diabetes or heart failure.
Beta-Blockers:	Start in all patients. Continue indefinitely. Observe usual contraindications.

BMI indicates body mass index; in, inches; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; BMI, body mass index; INR, international normalization ratio; ACE, angiotensin converting enzyme; CHF, congestive heart failure; LVEF, left ventricular ejection fraction; ARB, angiotensin receptor blocker and TG, triglycerides.

*Non–HDL cholesterol equals total cholesterol minus HDL cholesterol.

†Treat to a goal of non–HDL-C *substantially* less than 130 mg/dL.

‡Dietary-supplement niacin must not be used as a substitute for prescription niacin, and over-the-counter niacin should be used only if approved and monitored by a physician.

§Creatinine should be less than or equal to 2.5 mg/dL in men or less than or equal to 2.0 mg/dL in women.

||Potassium should be less than or equal to 5.0 mEq/L.

Modified with permission from Smith et al. *Circulation.* 2001;104:1577–1579.[181]

(usually accomplished in conjunction with cardiac rehabilitation). (*Level of Evidence: B*)

3. **A plan should be established to monitor the response of body mass index and waist circumference to therapy (usually accomplished in conjunction with cardiac rehabilitation). (*Level of Evidence: B*)**

4. Smoking Cessation

Class I

1. **Patients recovering from STEMI who have a history of cigarette smoking should be strongly encouraged to stop smoking and to avoid secondhand smoke. Counseling should be provided to the patient and family, along with pharmacological therapy (including nicotine replacement and bupropion) and formal smoking-cessation programs as appropriate. (*Level of Evidence: B*)**
2. **All STEMI patients should be assessed for a history of cigarette smoking. (*Level of Evidence: A*)**

5. Antiplatelet Therapy

Class I

1. **A daily dose of aspirin 75 to 162 mg orally should be given indefinitely to patients recovering from STEMI. (*Level of Evidence: A*)**
2. **If true aspirin allergy is present, preferably clopidogrel (75 mg orally per day) or, alternatively, ticlopidine (250 mg orally twice daily) should be substituted. (*Level of Evidence: C*)**
3. **If true aspirin allergy is present, warfarin therapy with a target INR of 2.5 to 3.5 is a useful alternative to clopidogrel in patients less than 75 years of age who are at low risk for bleeding and who can be monitored adequately for dose adjustment to maintain a target INR range. (*Level of Evidence: C*)**

Class III

1. **Ibuprofen should not be used because it blocks the antiplatelet effects of aspirin. (*Level of Evidence: C*)**

On the basis of 12 randomized trials in 18 788 patients with prior infarction, the Antiplatelet Trialists' Collaboration reported a 25% reduction in the risk of recurrent infarction, stroke, or vascular death in patients receiving prolonged antiplatelet therapy (36 fewer events for every 1000 patients treated).[31] No antiplatelet therapy has proved superior to aspirin in this population, and daily doses of aspirin between 80 and 325 mg appear to be effective.[184] The CAPRIE (Clopidogrel versus Aspirin in Patients at Risk of Ischemic Events) trial, which compared aspirin with clopidogrel in 19 185 patients at high risk for vascular events, demonstrated a modest but significant (8.6%, *P* equals 0.043) reduction in serious vascular events with clopidogrel compared with aspirin.[185] These data suggest clopidogrel as the best alternative to aspirin in patients with true aspirin allergy.

The use of warfarin therapy for secondary prevention of vascular events in patients after STEMI is discussed in Section 7.12.11 of the full-text guidelines. Large randomized trials have demonstrated that oral anticoagulants, when given in adequate doses, reduce the rates of adverse outcomes, at the cost of a small increase in hemorrhagic events.[186–188] In the Warfarin, Aspirin, Reinfarction Study (WARIS II), warfarin without aspirin in a dose intended to achieve an INR of 2.8 to 4.2 resulted in a significant reduction in a composite end point (death, nonfatal reinfarction, or thromboembolic stroke) compared with therapy with aspirin alone (16.7% versus 20.0%).[186] Warfarin therapy resulted in a small but significant increase in major, nonfatal bleeding compared with therapy with aspirin alone (0.62% versus 0.17% per year). Chronic therapy with warfarin after STEMI presents an alternative to clopidogrel in patients with aspirin allergy.

6. Inhibition of Renin-Angiotensin-Aldosterone-System

Class I

1. **An ACE inhibitor should be prescribed at discharge for all patients without contraindications after STEMI. (*Level of Evidence: A*)**
2. **Long-term aldosterone blockade should be prescribed for post-STEMI patients without significant renal dysfunction (creatinine should be less than or equal to 2.5 mg/dL in men and less than or equal to 2.0 mg/dL in women) or hyperkalemia (potassium should be less than or equal to 5.0 mEq/L) who are already receiving therapeutic doses of an ACE inhibitor, have an LVEF less than or equal to 0.40, and have either symptomatic heart failure or diabetes. (*Level of Evidence: A*)**
3. **An ARB should be prescribed at discharge in those STEMI patients who are intolerant of an ACE inhibitor and have either clinical or radiological signs of heart failure and LVEF less than 0.40. Valsartan and candesartan have established efficacy for this recommendation. (*Level of Evidence: B*)**

Class IIa

1. **In STEMI patients who tolerate ACE inhibitors, an ARB can be useful as an alternative to ACE inhibitors in the long-term management of STEMI patients, provided there are either clinical or radiological signs of heart failure or LVEF less than 0.40. Valsartan and candesartan have established efficacy for this recommendation. (*Level of Evidence: B*)**

Class IIb

1. **The combination of an ACE inhibitor and an ARB may be considered in the long-term management of STEMI patients with persistent symptomatic heart failure and LVEF less than 0.40. (*Level of Evidence: B*)**

The use of ACE inhibitors early in the acute phase of STEMI and in the hospital management phase has been described earlier.

Compelling evidence now supports the broad long-term use of ACE inhibitors after STEMI.[189,190] The results of the VALIANT study (Valsartan in Acute Myocardial Infarction Trial) evaluating valsartan are discussed in Section 7.4.3 of the full-text guidelines. The series of CHARM studies (Candesartan in Heart Failure Assessment in Reduction of Mortality), although focusing on the evaluation of candesartan in

patients with chronic heart failure, provides information that can be extrapolated to the long-term management of the STEMI patient, because 50% to 60% of the patients studied had ischemic heart disease as the cause of heart failure.[191–193]

Given the extensive randomized trial and routine clinical experience with ACE inhibitors, they remain the logical first agent for inhibition of the renin-angiotensin-aldosterone system in the long-term management of patients with STEMI.[150,194] The ARBs valsartan and candesartan should be administered over the long term to STEMI patients with symptomatic heart failure who are intolerant of ACE inhibitors. As described in Section 7.4.3 of the full-text guidelines, the choice between an ACE inhibitor and an ARB over the long term in patients who are tolerant of ACE inhibitors will vary with individual physician and patient preference, as well as cost and anticipated side-effect profile.[150,194]

The results of the most relevant clinical trials that tested combinations of ACE inhibitors and ARBs have been subtly different, but clinically relevant. Whereas the CHARM-Added[192] trial demonstrated a reduction in the combined end point of heart failure hospitalization and death over ACE inhibition alone, the VALIANT study[149] reported that the combination of captopril and valsartan was equivalent to either alone, but with a greater number of adverse effects. Thus, when combination ACE inhibition and angiotensin receptor blockade is considered necessary, the preferred ARB is candesartan. Although there is evidence that the combination of an ACE inhibitor and an aldosterone inhibitor is effective at reducing mortality and is well tolerated in patients with a serum creatinine level of 2.5 mg/dL or less and a serum potassium concentration of 5.0 mEq/L or less (see Section 7.4.3 of the full-text guidelines), much less experience exists with the combination of an ARB and aldosterone inhibitor (24% of 2028 patients in the CHARM-Alternative trial)[191] and the triple combination of an ACE inhibitor, ARB, and an aldosterone antagonist (17% of 2548 patients in the CHARM-Added trial).[192]

The combination of an ACE inhibitor and an ARB (valsartan 20 mg/d orally initially; titrated up to 160 mg orally twice per day, or candesartan 4 to 8 mg/d orally initially; titrated up to 32 mg/d orally) or an ACE inhibitor and an aldosterone inhibitor may be considered for the long-term management of STEMI patients with symptomatic heart failure and LVEF less than 0.40, provided the serum creatinine level is less than or equal to 2.5 mg/dL in men and less than or equal to 2.0 mg/dL in women and the serum potassium concentration is less than or equal to 5.0 mEq/L (See Sections 7.4.3 and 7.6.4 of the full-text guidelines.)

7. Beta-Blockers

Class I

1. **All patients after STEMI except those at low risk (normal or near-normal ventricular function, successful reperfusion, and absence of significant ventricular arrhythmias) and those with contraindications should receive beta-blocker therapy. Treatment should begin within a few days of the event, if not initiated acutely, and continue indefinitely. (*Level of Evidence: A*)**
2. **Patients with moderate or severe LV failure should receive beta-blocker therapy with a gradual titration scheme. (*Level of Evidence: B*)**

Class IIa

1. **It is reasonable to prescribe beta-blockers to low-risk patients after STEMI who have no contraindications to that class of medications. (*Level of Evidence: A*)**

The use of beta-blockers in the early phase of STEMI and in hospital management is reviewed in Sections 6.3.1.6 and 7.4.1 of the full-text guidelines. The benefits of beta-blocker therapy in patients without contraindications have been demonstrated with or without reperfusion, initiated early or later in the clinical course, and for all age groups. The benefits of beta-blocker therapy for secondary prevention are well established.[142,196] In patients with moderate or severe LV failure, beta-blocker therapy should be administered with a gradual titration scheme.[197] Long-term beta-blocker therapy should be administered to survivors of STEMI who have subsequently undergone revascularization, because there is evidence of a mortality benefit from their use despite revascularization with either CABG surgery or PCI.[198]

8. Blood Pressure Control

Class I

1. **Blood pressure should be treated with drug therapy to a target level of less than 140/90 mm Hg and to less than 130/80 mm Hg for patients with diabetes or chronic kidney disease. (*Level of Evidence: B*)**
2. **Lifestyle modification (weight control, dietary changes, physical activity, and sodium restriction) should be initiated in all patients with blood pressure greater than or equal to 120/80 mm Hg. (*Level of Evidence: B*)**

Class IIb

1. **A target blood pressure goal of 120/80 mm Hg for post-STEMI patients may be reasonable. (*Level of Evidence: C*)**

Class III

1. **Short-acting dihydropyridine calcium channel blocking agents should not be used for the treatment of hypertension. (*Level of Evidence: B*)**

The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC-7)[199] recommends that patients be treated after MI with ACE inhibitors, beta-blockers, and, if necessary, aldosterone antagonists to a target blood pressure of less than 140/90 mm Hg, or less than 130/80 mm Hg for those with chronic kidney disease or diabetes.[199] Most patients will require 2 or more drugs to reach this goal, and when the blood pressure is greater than 20/10 mm Hg above goal, 2 drugs should usually be used from the outset.

JNC-7 emphasizes the importance of lifestyle modifications for all patients with blood pressure of 120/80 mm Hg or

greater.[199] These modifications include weight reduction if overweight or obese, consumption of a diet rich in fruits and vegetables and low in total fat and saturated fat, and reduction of sodium to no more than 2.4 g/d.[199]

9. Diabetes Management

Class I

1. **Hypoglycemic therapy should be initiated to achieve HbA1c less than 7%. (*Level of Evidence: B*)**

Class III

1. **Thiazolidinediones should not be used in patients recovering from STEMI who have New York Heart Association class III or IV heart failure. (*Level of Evidence: B*)**

10. Hormone Therapy

Class III

1. **Hormone therapy with estrogen plus progestin should not be given de novo to postmenopausal women after STEMI for secondary prevention of coronary events. (*Level of Evidence: A*)**
2. **Postmenopausal women who are already taking estrogen plus progestin at the time of a STEMI should not continue hormone therapy. However, women who are beyond 1 to 2 years after initiation of hormone therapy who wish to continue hormone therapy for another compelling indication should weigh the risks and benefits, recognizing a greater risk of cardiovascular events. However, hormone therapy should not be continued while patients are on bedrest in the hospital. (*Level of Evidence: B*)**

On the basis of the Heart and Estrogen/progestin Replacement Study (HERS),[200] the Heart and Estrogen/progestin Replacement Study Follow-up (HERS-2),[201] and the Women's Health Initiative,[202] postmenopausal women should not receive combination estrogen and progestin therapy for primary or secondary prevention of CHD. It is recommended that the use of hormone therapy be discontinued in women who have STEMI.[200–202]

11. Warfarin Therapy

Class I

1. **Warfarin should be given to aspirin-allergic post-STEMI patients with indications for anticoagulation as follows:**
   a. **Without stent implanted (INR 2.5 to 3.5). (*Level of Evidence: B*)**
   b. **With stent implanted and clopidogrel 75 mg/d administered concurrently (INR 2.0 to 3.0). (*Level of Evidence: C*)**
2. **Warfarin (INR 2.5 to 3.5) is a useful alternative to clopidogrel in aspirin-allergic patients after STEMI who do not have a stent implanted. (*Level of Evidence: B*)**
3. **Warfarin (INR 2.0 to 3.0) should be prescribed for post-STEMI patients with either persistent or paroxysmal atrial fibrillation. (*Level of Evidence: A*)**
4. **In post-STEMI patients with LV thrombus noted on an imaging study, warfarin should be prescribed for at least 3 months (*Level of Evidence: B*) and indefinitely in patients without an increased risk of bleeding (*Level of Evidence: C*).**
5. **Warfarin alone (INR 2.5 to 3.5) or warfarin (INR 2.0 to 3.0) in combination with aspirin (75 to 162 mg) should be prescribed in post-STEMI patients who have no stent implanted and who have indications for anticoagulation. (*Level of Evidence: B*)**

Class IIa

1. **In post-STEMI patients less than 75 years of age without specific indications for anticoagulation who can have their level of anticoagulation monitored reliably, warfarin alone (INR 2.5 to 3.5) or warfarin (INR 2.0 to 3.0) in combination with aspirin (75 to 162 mg) can be useful for secondary prevention. (*Level of Evidence: B*)**
2. **It is reasonable to prescribe warfarin to post-STEMI patients with LV dysfunction and extensive regional wall-motion abnormalities. (*Level of Evidence: A*)**

Class IIb

1. **Warfarin may be considered in patients with severe LV dysfunction, with or without CHF. (*Level of Evidence: C*)**

The indications for long-term anticoagulation after STEMI remain controversial and are evolving. Although the use of warfarin has been demonstrated to be cost-effective compared with standard therapy without aspirin, the superior safety, efficacy and cost-effectiveness of aspirin has made it the antithrombotic agent of choice for secondary prevention[203] (Figure 7).

12. Physical Activity

Class I

1. **On the basis of assessment of risk, ideally with an exercise test to guide the prescription, all patients recovering from STEMI should be encouraged to exercise for a minimum of 30 minutes, preferably daily but at least 3 or 4 times per week (walking, jogging, cycling, or other aerobic activity), supplemented by an increase in daily lifestyle activities (eg, walking breaks at work, gardening, and household work). (*Level of Evidence: B*)**
2. **Cardiac rehabilitation/secondary prevention programs, when available, are recommended for patients with STEMI, particularly those with multiple modifiable risk factors and/or those moderate- to high-risk patients in whom supervised exercise training is warranted. (*Level of Evidence: C*)**

13. Antioxidants

Class III

1. **Antioxidant vitamins such as vitamin E and/or vitamin C supplements should not be prescribed to patients**

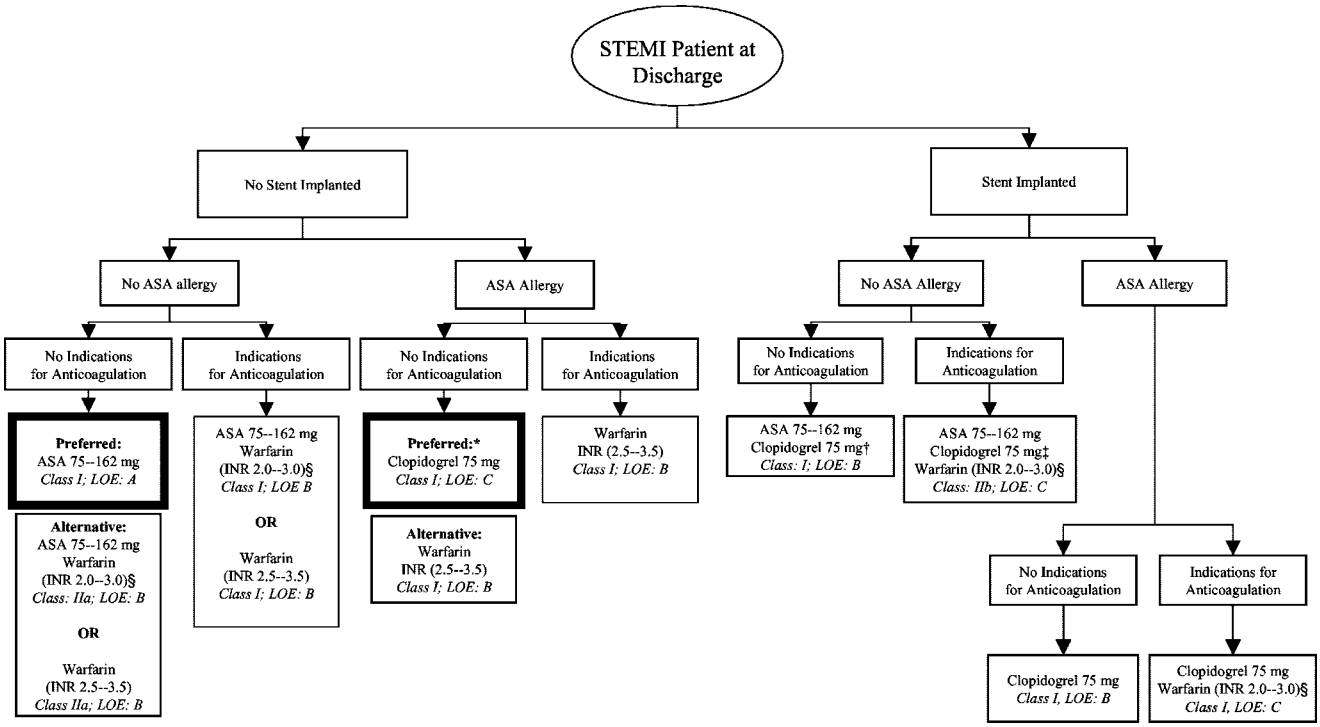

Figure 7. Long-term antithrombotic therapy at hospital discharge after STEMI. ASA indicates aspirin; LOE, level of evidence LV, left ventricular; and INR, international normalized ratio. *Clopidogrel is preferred over warfarin because of increased risk of bleeding and low patient compliance in warfarin trials. †For 12 months. ‡Discontinue clopidogrel 1 month after implantation of a bare metal stent or several months after implantation of a drug-eluting stent (3 months after sirolimus and 6 months after paclitaxel) because of the potential increased risk of bleeding with warfarin and 2 antiplatelet agents. Continue aspirin and warfarin long term if warfarin is indicated for other reasons such as atrial fibrillation, LV thrombus, cerebral emboli, or extensive regional wall-motion abnormality. §An INR of 2.0 to 3.0 is acceptable with tight control, but the lower end of this range is preferable. The combination of antiplatelet therapy and warfarin may be considered in patients aged less than 75 years with low bleeding risk who can be monitored reliably.

recovering from STEMI to prevent cardiovascular disease. (*Level of Evidence: A*)

There is no convincing evidence to support lipid- or water-soluble antioxidant supplementation in patients after STEMI or patients with or without established coronary disease.

VIII. Long-Term Management

A. Psychosocial Impact of STEMI

Class I

1. The psychosocial status of the patient should be evaluated, including inquiries regarding symptoms of depression, anxiety, or sleep disorders and the social support environment. (*Level of Evidence: C*)

Class IIa

1. Treatment with cognitive-behavioral therapy and selective serotonin reuptake inhibitors can be useful for STEMI patients with depression that occurs in the year after hospital discharge. (*Level of Evidence: A*)

Treatment of depression with combined cognitive-behavioral therapy and selective serotonin reuptake inhibitors improves outcome in terms of depression symptoms and social function.[204–206] It appears prudent to assess STEMI patients for depression during hospitalization and during the first month after STEMI and to intervene and reassess yearly in the first 5 years, as appropriate. There is evidence that the STEMI experience, with its sudden and unexpected onset, dramatic changes in lifestyle, and the additive effort of comorbid life events, is a relatively traumatic event and may produce impaired coping during subsequent ischemic events.[207]

B. Cardiac Rehabilitation

Class I

1. Cardiac rehabilitation/secondary prevention programs, when available, are recommended for patients with STEMI, particularly those with multiple modifiable risk factors and/or those moderate- to high-risk patients in whom supervised exercise training is warranted. (*Level of Evidence: C*)

C. Follow-Up Visit With Medical Provider

Class I

1. A follow-up visit should delineate the presence or absence of cardiovascular symptoms and functional class. (*Level of Evidence: C*)

2. **The patient's list of current medications should be reevaluated in a follow-up visit, and appropriate titration of ACE inhibitors, beta-blockers, and statins should be undertaken. (*Level of Evidence: C*)**
3. **The predischarge risk assessment and planned workup should be reviewed and continued (Figure 6). This should include a check of LV function and possibly Holter monitoring for those patients whose early post-STEMI ejection fraction was 0.31 to 0.40 or lower, in consideration of possible ICD use (Figure 5). (*Level of Evidence: C*)**
4. **The healthcare provider should review and emphasize the principles of secondary prevention with the patient and family members (Table 4).[181] (*Level of Evidence: C*)**
5. **The psychosocial status of the patient should be evaluated in follow-up, including inquiries regarding symptoms of depression, anxiety, or sleep disorders and the social support environment. (*Level of Evidence: C*)**
6. **In a follow-up visit, the healthcare provider should discuss in detail issues of physical activity, return to work, resumption of sexual activity, and travel, including driving and flying. The metabolic equivalent values for various activities are provided as a resource in Table 34 of the full-text guideline. (*Level of Evidence: C*)**
7. **Patients and their families should be asked if they are interested in CPR training after the patient is discharged from the hospital. (*Level of Evidence: C*)**
8. **Providers should actively review the following issues with patients and their families:**
   a. **The patient's heart attack risk. (*Level of Evidence: C*)**
   b. **How to recognize symptoms of STEMI. (*Level of Evidence: C*)**
   c. **The advisability of calling 9-1-1 if symptoms are unimproved or worsening after 5 minutes, despite feelings of uncertainty about the symptoms and fear of potential embarrassment. (*Level of Evidence: C*)**
   d. **A plan for appropriate recognition and response to a potential acute cardiac event, including the phone number to access EMS, generally 9-1-1.[15] (*Level of Evidence: C*)**
9. **Cardiac rehabilitation/secondary prevention programs, when available, are recommended for patients with STEMI, particularly those with multiple modifiable risk factors and/or those moderate- to high-risk patients in whom supervised exercise training is warranted. (*Level of Evidence: C*)**

References

1. Mehta RH, Montoye CK, Gallogly M, et al., for the GAP Steering Committee of the American College of Cardiology. Improving quality of care for acute myocardial infarction: The Guidelines Applied in Practice (GAP) Initiative. *JAMA*. 2002;287:1269–1276.
2. Jencks SF, Huff ED, Cuerdon T. Change in the quality of care delivered to medicare beneficiaries, 1998–1999 to 2000–2001. *JAMA*. 2003;289:305–312.
3. Schneider EC, Zaslavsky AM, Epstein AM. Racial disparities in the quality of care for enrollees in medicare managed care. *JAMA*. 2002;287:1288–1294.
4. Rogers WJ, Canto JG, Lambrew CT, et al. Temporal trends in the treatment of over 1.5 million patients with myocardial infarction in the US from 1990 through 1999: the National Registry of Myocardial Infarction 1, 2 and 3. *J Am Coll Cardiol*. 2000;36:2056–2063.
5. American Heart Association. *Heart Disease and Stroke Statistics—2004 Update.* Dallas, TX: American Heart Association 2003.
5a. Wiviott SD, Morrow DA, Giugliano RP, et al. Performance of the thrombolysis in myocardial infarction risk index for early acute coronary syndrome in the National Registry of Myocardial Infarction: a simple risk index predicts mortality in both ST and non-ST elevation myocardial infarction. *J Am Coll Cardiol* 2003;41:365A–366A.
6. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA*. 2001;285:2486–2497.
7. Levey AS, Coresh J, Balk E, et al. National Kidney Foundation practice guidelines for chronic kidney disease: evaluation, classification, and stratification. *Ann Intern Med*. 2003;139:137–147.
8. National, Heart, Lung, and Blood Institute. *Act in Time to Heart Attack.* Available at: http://:www.nhlbi.nih.gov/actintime, 2003. Accessed May 15, 2003.
9. American College of Cardiology. *Guidelines Applied in Practice.* Available at: http://www.acc.org/gap/gap.htm. Accessed May 15, 2003.
10. American Heart Association. *Get With the Guidelines.* Available at http://www.americanheart.org/presenter.jhtml?identifier=1165. Accessed May 15, 2003.
11. Topol EJ, Kereiakes DJ. Regionalization of care for acute ischemic heart disease: a call for specialized centers. *Circulation*. 2003;107:1463–1466.
12. Califf RM, Faxon DP. Need for centers to care for patients with acute coronary syndromes. *Circulation*. 2003;107:1467–1470.
13. Willerson JT. Editor's commentary: centers of excellence. *Circulation*. 2003;107:1471–1472.
14. National Cholesterol Education Program. *Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III)*. NIH publication No. 02–5125. Bethesda, Md: National Heart, Lung, and Blood Institute, 2002. Guidelines, Related Tools, and Patient Information, available at: http://www.nhlbi.nih.gov/guidelines/cholesterol/index.htm. Accessed April 12, 2003.
15. Dracup K, Alonzo AA, Atkins JM, et al. The physician's role in minimizing prehospital delay in patients at high risk for acute myocardial infarction: recommendations from the National Heart Attack Alert Program. Working Group on Educational Strategies to Prevent Prehospital Delay in Patients at High Risk for Acute Myocardial Infarction. *Ann Intern Med*. 1997;126:645–651.
16. Hedges JR, Feldman HA, Bittner V, et al, for the REACT Study Group. Impact of community intervention to reduce patient delay time on use of reperfusion therapy for acute myocardial infarction: Rapid Early Action for Coronary Treatment (REACT) trial. *Acad Emerg Med*. 2000;7:862–872.
17. Canto JG, Zalenski RJ, Ornato JP, et al, for the National Registry of Myocardial Infarction 2 Investigators. Use of emergency medical services in acute myocardial infarction and subsequent quality of care: observations from the National Registry of Myocardial Infarction 2. *Circulation*. 2002;106:3018–3023.
18. Goldberg R, Goff D, Cooper L, et al. Age and sex differences in presentation of symptoms among patients with acute coronary disease: the Trial: Rapid Early Action for Coronary Treatment (REACT). *Coron Artery Dis*. 2000;11:399–407.
19. Hutchings CB, Mann NC, Daya M, et al, for the Rapid Early Action for Coronary Treatment Study. Patients with chest pain calling 9-1-1 or self-transporting to reach definitive care: which mode is quicker? *Am Heart J*. 2004;147:35–41.
20. Faxon D, Lenfant C. Timing is everything: motivating patients to call 9–1–1 at onset of acute myocardial infarction. *Circulation*. 2001;104:1210–1211.
21. US Department of Health and Human Services, Public Health Service, National Institutes of Health, National Heart, Lung, and Blood Institute. NIH publication No. 01–3313. September 2001. Available at: http://www.nhlbi.nih.gov/health/prof/heart/mi/provider.pdf. Accessed December 16, 2002.
22. Eisenberg MJ, Topol EJ. Prehospital administration of aspirin in patients with unstable angina and acute myocardial infarction. *Arch Intern Med*. 1996;156:1506–1510.
23. Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. *Lancet*. 1994;343:311–322.
24. Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI). Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. *Lancet*. 1986;1:397–402.

25. Armstrong PW, Collen D, Antman E. Fibrinolysis for acute myocardial infarction: the future is here and now. *Circulation*. 2003;107: 2533–2537.
26. The American Heart Association in collaboration with the International Liaison Committee on Resuscitation. Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: Part 7: the era of reperfusion: section 1: acute coronary syndromes (acute myocardial infarction). *Circulation*. 2000;102(suppl I):I-172–I-203.
27. Pedley DK, Bissett K, Connolly EM, et al. Prospective observational cohort study of time saved by prehospital thrombolysis for ST elevation myocardial infarction delivered by paramedics. *BMJ*. 2003;327:22–26.
28. Menown IB, Mackenzie G, Adgey AA. Optimizing the initial 12-lead electrocardiographic diagnosis of acute myocardial infarction. *Eur Heart J*. 2000;21:275–283.
29. Case definitions for acute coronary heart disease in epidemiology and clinical research studies: a statement from the AHA Council on Epidemiology and Prevention; AHA Statistics Committee; World Heart Federation Council on Epidemiology and Prevention; the European Society of Cardiology Working Group on Epidemiology and Prevention; Centers for Disease Control and Prevention; and the National Heart, Lung, and Blood Institute. *Circulation*. 2003;108:2543–2549.
30. Come PC, Pitt B. Nitroglycerin-induced severe hypotension and bradycardia in patients with acute myocardial infarction. *Circulation*. 1976; 54:624–628.
31. Antithrombotic Trialists' Collaboration. Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. *BMJ*. 2002;324:71–86.
32. Sagar KA, Smyth MR. A comparative bioavailability study of different aspirin formulations using on-line multidimensional chromatography. *J Pharm Biomed Anal*. 1999;21:383–392.
33. De Luca G, Suryapranata H, Zijlstra F, et al, for the ZWOLLE Myocardial Infarction Study Group. Symptom-onset-to-balloon time and mortality in patients with acute myocardial infarction treated by primary angioplasty. *J Am Coll Cardiol*. 2003;42:991–997.
34. Boersma E, Mercado N, Poldermans D, et al. Acute myocardial infarction. *Lancet*. 2003;361:847–858.
35. De Luca G, Suryapranata H, Ottervanger JP, et al. Time delay to treatment and mortality in primary angioplasty for acute myocardial infarction: every minute of delay counts. *Circulation*. 2004;109: 1223–1225.
36. Boersma E, Maas AC, Deckers JW, et al. Early thrombolytic treatment in acute myocardial infarction: reappraisal of the golden hour. *Lancet*. 1996;348:771–775.
37. Zeymer U, Tebbe U, Essen R, et al, for the ALKK-Study Group. Influence of time to treatment on early infarct-related artery patency after different thrombolytic regimens. *Am Heart J*. 1999;137:34–38.
38. Weaver WD, Cerqueira M, Hallstrom AP, et al. Prehospital-initiated vs hospital-initiated thrombolytic therapy: the Myocardial Infarction Triage and Intervention Trial. *JAMA*. 1993;270:1211–1216.
39. Brodie BR, Stuckey TD, Wall TC, et al. Importance of time to reperfusion for 30-day and late survival and recovery of left ventricular function after primary angioplasty for acute myocardial infarction. *J Am Coll Cardiol*. 1998;32:1312–1319.
40. Brodie BR, Stuckey TD, Wall TC, et al. Importance of time to reperfusion on outcomes with primary coronary angioplasty for acute myocardial infarction (results from the Stent Primary Angioplasty in Myocardial Infarction Trial). *Am J Cardiol*. 2001;88:1085–1090.
41. Williams DO. Treatment delayed is treatment denied. *Circulation*. 2004; 109:1806-1808.
42. The Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology. Management of acute myocardial infarction in patients presenting with ST-segment elevation. *Eur Heart J*. 2003;24:28–66.
43. Morrow DA, Antman EM, Charlesworth A, et al. TIMI risk score for ST-elevation myocardial infarction: a convenient, bedside, clinical score for risk assessment at presentation: an intravenous nPA for treatment of infarcting myocardium early II trial substudy. *Circulation*. 2000;102: 2031–2037.
44. Lee KL, Woodlief LH, Topol EJ, et al, for the GUSTO-I Investigators. Predictors of 30-day mortality in the era of reperfusion for acute myocardial infarction: results from an international trial of 41,021 patients. *Circulation*. 1995;91:1659–1668.
45. Morrow DA, Antman EM, Giugliano RP, et al. A simple risk index for rapid initial triage of patients with ST-elevation myocardial infarction: an InTIME II substudy. *Lancet*. 2001;358:1571–1575.
46. Krumholz HM, Chen J, Wang Y, et al. Comparing AMI mortality among hospitals in patients 65 years of age and older: evaluating methods of risk adjustment. *Circulation*. 1999;99:2986–2992.
47. Granger CB, Goldberg RJ, Dabbous O, et al, for the Global Registry of Acute Coronary Events Investigators. Predictors of hospital mortality in the Global Registry of Acute Coronary Events. *Arch Intern Med*. 2003; 163:2345–2353.
48. Magid DJ, Calonge BN, Rumsfeld JS, et al, for the National Registry of Myocardial Infarction 2 and 3 Investigators. Relation between hospital primary angioplasty volume and mortality for patients with acute MI treated with primary angioplasty vs thrombolytic therapy. *JAMA*. 2000; 284:3131–3138.
49. Nallamothu BK, Bates ER. Percutaneous coronary intervention versus fibrinolytic therapy in acute myocardial infarction: is timing (almost) everything? *Am J Cardiol*. 2003;92:824–826.
50. Bonnefoy E, Lapostolle F, Leizorovicz A, et al., for the Comparison of Angioplasty and Prehospital Thrombolysis in Acute Myocardial Infarction study group. Primary angioplasty versus prehospital fibrinolysis in acute myocardial infarction: a randomised study. *Lancet*. 2002; 360:825–829.
51. Steg PG, Bonnefoy E, Chabaud S, et al. Impact of time to treatment on mortality after prehospital fibrinolysis or primary angioplasty: data from the CAPTIM randomized clinical trial. *Circulation*. 2003;108:2851–2856.
52. Widimsky P, Budesinsky T, Vorac D, et al. Long distance transport for primary angioplasty vs immediate thrombolysis in acute myocardial infarction: final results of the randomized national multicentre trial: PRAGUE-2. *Eur Heart J*. 2003;24:94–104.
53. Deleted in press.
54. Antman EM. General hospital management. In: Julian DG, Braunwald E, eds. *Management of Acute Myocardial Infarction.* London, England: WB Saunders 1994;42–44.
54a. Lincoff AM, Califf RM, Van de Werf F, et al, for the Global Use of Strategies To Open Coronary Arteries (GUSTO) Investigators. Mortality at 1 year with combination platelet glycoprotein IIb/IIIa inhibition and reduced-dose fibrinolytic therapy vs conventional fibrinolytic therapy for acute myocardial infarction: GUSTO V randomized trial. *JAMA*. 2002;288:2130-2135.
55. Zijlstra F, Beukema WP, van't Hof AW, et al. Randomized comparison of primary coronary angioplasty with thrombolytic therapy in low risk patients with acute myocardial infarction. *J Am Coll Cardiol*. 1997;29:908–912.
56. Ribeiro EE, Silva LA, Carneiro R, et al. Randomized trial of direct coronary angioplasty versus intravenous streptokinase in acute myocardial infarction. *J Am Coll Cardiol*. 1993;22:376–380.
57. Grinfield L, Berrocal D, Bellardi J, et al. Fibrinolytics versus primary angioplasty in acute myocardial infarction (FAP): a randomized trial in a community hospital in Argentina. *J Am Coll Cardiol*. 1996;27:A222. Abstract.
58. Zijlstra F, de Boer MJ, Hoorntje JC, et al. A comparison of immediate coronary angioplasty with intravenous streptokinase in acute myocardial infarction. *N Engl J Med*. 1993;328:680–684.
59. Widimsky P, Groch L, Zelízko M, et al. Multicentre randomized trial comparing transport to primary angioplasty vs immediate thrombolysis vs combined strategy for patients with acute myocardial infarction presenting to a community hospital without a catheterization laboratory: the PRAGUE study. *Eur Heart J*. 2000;21:823–831.
60. de Boer MJ, Ottervanger JP, van't Hof AW, et al, for the Zwolle Myocardial Infarction Study Group. Reperfusion therapy in elderly patients with acute myocardial infarction: a randomized comparison of primary angioplasty and thrombolytic therapy. *J Am Coll Cardiol*. 2002; 39:1723–1728.
61. Akhras F, Ousa AA, Swann G, et al. Primary coronary angioplasty or intravenous thrombolysis for patients with acute myocardial infarction? Acute and late follow-up results in a new cardiac unit. *J Am Coll Cardiol*. 1997;29:A235–A236. Abstract.
62. Deleted in press.
63. Grines CL, Browne KF, Marco J, et al, for the Primary Angioplasty in Myocardial Infarction Study Group. A comparison of immediate angioplasty with thrombolytic therapy for acute myocardial infarction. *N Engl J Med*. 1993;328:673–679.
64. Gibbons RJ, Holmes DR, Reeder GS, et al, for the Mayo Coronary Care Unit and Catheterization Laboratory Groups. Immediate angioplasty compared with the administration of a thrombolytic agent followed by conservative treatment for myocardial infarction. *N Engl J Med*. 1993; 328:685–691.

65. Ribichini F, Steffenino G, Dellavalle A, et al. Comparison of thrombolytic therapy and primary coronary angioplasty with liberal stenting for inferior myocardial infarction with precordial ST-segment depression: immediate and long-term results of a randomized study. *J Am Coll Cardiol*. 1998;32:1687–1694.
66. García E, Elízaga J, Pérez-Castellano N, et al. Primary angioplasty versus systemic thrombolysis in anterior myocardial infarction. *J Am Coll Cardiol*. 1999;33:605–611.
67. The Global Use of Strategies to Open Occluded Coronary Arteries in Acute Coronary Syndromes (GUSTO IIb) Angioplasty Substudy Investigators. A clinical trial comparing primary coronary angioplasty with tissue plasminogen activator for acute myocardial infarction. *N Engl J Med*. 1997;336:1621–1628.
68. Le May MR, Labinaz M, Davies RF, et al. Stenting versus thrombolysis in acute myocardial infarction trial (STAT). *J Am Coll Cardiol*. 2001; 37:985–991.
69. Schömig A, Kastrati A, Dirschinger J, et al, for the Stent versus Thrombolysis for Occluded Coronary Arteries in Patients with Acute Myocardial Infarction Study Investigators. Coronary stenting plus platelet glycoprotein IIb/IIIa blockade compared with tissue plasminogen activator in acute myocardial infarction. *N Engl J Med*. 2000;343:385–391.
70. Vermeer F, Oude Ophuis AJ, vd Berg EJ et al. Prospective randomised comparison between thrombolysis, rescue PTCA, and primary PTCA in patients with extensive myocardial infarction admitted to a hospital without PTCA facilities: a safety and feasibility study. *Heart*. 1999;82: 426–431.
71. Kastrati A, Mehilli J, Dirschinger J, et al, for the Stent versus Thrombolysis for Occluded Coronary Arteries in Patients with Acute Myocardial Infarction (STOPAMI-2) Study. Myocardial salvage after coronary stenting plus abciximab versus fibrinolysis plus abciximab in patients with acute myocardial infarction: a randomised trial. *Lancet*. 2002;359:920–925.
72. Andersen HR, Nielsen TT, Rasmussen K, et al, for the DANAMI-2 Investigators. Thrombolytic therapy vs primary percutaneous coronary intervention for myocardial infarction in patients presenting to hospitals without on-site cardiac surgery: a randomized controlled trial. *JAMA*. 2002;287:1943–1951.
73. Kastrati A, Mehilli J, Dirschinger J, et al, for the Stent versus Thrombolysis for Occluded Coronary Arteries in Patients With Acute Myocardial Infarction (STOPAMI-2) Study. A randomized trial of transfer for primary angioplasty versus on-site thrombolysis in patients with high-risk myocardial infarction: the Air Primary Angioplasty in Myocardial Infarction study. *J Am Coll Cardiol*. 2002;39:1713–1719.
74. Andersen HR, Nielsen TT, Rasmussen K, et al, for the DANAMI-2 Investigators. A comparison of coronary angioplasty with fibrinolytic therapy in acute myocardial infarction. *N Engl J Med*. 2003;349: 733–742.
75. Hochman JS, Sleeper LA, Webb JG, et al, for the Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock (SHOCK) Investigators. Early revascularization in acute myocardial infarction complicated by cardiogenic shock. *N Engl J Med*. 1999;341: 625–634.
76. Keeley EC, Boura JA, Grines CL. Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials. *Lancet*. 2003;361:13–20.
76a. Melandri G. The obsession with primary angioplasty. *Circulation*. 2003; 108:e1620.
77. Rogers WJ, Dean LS, Moore PB, et al, for the Alabama Registry of Myocardial Ischemia Investigators. Comparison of primary angioplasty versus thrombolytic therapy for acute myocardial infarction. *Am J Cardiol*. 1994;74:111–118.
78. Every NR, Parsons LS, Hlatky M, et al, for the Myocardial Infarction Triage and Intervention Investigators. A comparison of thrombolytic therapy with primary coronary angioplasty for acute myocardial infarction. *N Engl J Med*. 1996;335:1253–1260.
79. Tiefenbrunn AJ, Chandra NC, French WJ, et al. Clinical experience with primary percutaneous transluminal coronary angioplasty compared with alteplase (recombinant tissue-type plasminogen activator) in patients with acute myocardial infarction: a report from the Second National Registry of Myocardial Infarction (NRMI-2). *J Am Coll Cardiol*. 1998; 31:1240–1245.
80. Danchin N, Vaur L, Genès N, et al. Treatment of acute myocardial infarction by primary coronary angioplasty or intravenous thrombolysis in the "real world": one-year results from a nationwide French survey. *Circulation*. 1999;99:2639–2644.
81. Berger PB, Ellis SG, Holmes DR, et al. Relationship between delay in performing direct coronary angioplasty and early clinical outcome in patients with acute myocardial infarction: results from the Global Use of Strategies To Open Occluded Arteries in Acute Coronary Syndromes (GUSTO-IIb) trial. *Circulation*. 1999;100:14–20.
82. Cannon CP, Gibson CM, Lambrew CT, et al. Relationship of symptom-onset-to-balloon time and door-to-balloon time with mortality in patients undergoing angioplasty for acute myocardial infarction. *JAMA*. 2000; 283:2941–2947.
83. Brodie BR, Stuckey TD, Hansen C, et al. Benefit of coronary reperfusion before intervention on outcomes after primary angioplasty for acute myocardial infarction. *Am J Cardiol*. 2000;85:13–18.
84. Stone GW, Cox D, Garcia E, et al. Normal flow (TIMI-3) before mechanical reperfusion therapy is an independent determinant of survival in acute myocardial infarction: analysis from the primary angioplasty in myocardial infarction trials. *Circulation*. 2001;104:636–641.
85. Clements IP, Christian TF, Higano ST, et al. Residual flow to the infarct zone as a determinant of infarct size after direct angioplasty. *Circulation*. 1993;88:1527–1533.
86. Juliard JM, Feldman LJ, Golmard JL, et al. Relation of mortality of primary angioplasty during acute myocardial infarction to door-to-Thrombolysis In Myocardial Infarction (TIMI) time. *Am J Cardiol*. 2003;91:1401–1405.
87. Cragg DR, Friedman HZ, Bonema JD, et al. Outcome of patients with acute myocardial infarction who are ineligible for thrombolytic therapy. *Ann Intern Med*. 1991;115:173–177.
88. Brodie BR, Weintraub RA, Stuckey TD, et al. Outcomes of direct coronary angioplasty for acute myocardial infarction in candidates and non-candidates for thrombolytic therapy. *Am J Cardiol*. 1991;67:7–12.
89. Himbert D, Juliard JM, Steg PG, et al. Primary coronary angioplasty for acute myocardial infarction with contraindication to thrombolysis. *Am J Cardiol*. 1993;71:377–381.
90. Zahn R, Schuster S, Schiele R, et al, for the Maximal Individual Therapy in Acute Myocardial Infarction (MITRA) Study Group. Comparison of primary angioplasty with conservative therapy in patients with acute myocardial infarction and contraindications for thrombolytic therapy. *Cathet Cardiovasc Interv*. 1999;46:127–133.
91. Grzybowski M, Clements EA, Parsons L, et al. Mortality benefit of immediate revascularization of acute ST-segment elevation myocardial infarction in patients with contraindications to thrombolytic therapy: a propensity analysis. *JAMA*. 2003;290:1891–1898.
92. Lotfi M, Mackie K, Dzavik V, et al. Impact of delays to cardiac surgery after failed angioplasty and stenting. *J Am Coll Cardiol*. 2004;43: 337–342.
93. Dehmer GJ, Gantt DS. Coronary intervention at hospitals without on-site cardiac surgery: are we pushing the envelope too far? *J Am Coll Cardiol*. 2004;43:343–345.
94. Antoniucci D, Santoro GM, Bolognese L, et al. A clinical trial comparing primary stenting of the infarct-related artery with optimal primary angioplasty for acute myocardial infarction: results from the Florence Randomized Elective Stenting in Acute Coronary Occlusions (FRESCO) trial. *J Am Coll Cardiol*. 1998;31:1234–1239.
95. Rodríguez A, Bernardi V, Fernández M, et al. In-hospital and late results of coronary stents versus conventional balloon angioplasty in acute myocardial infarction: Gianturco-Roubin in Acute Myocardial Infarction (GRAMI trial). *Am J Cardiol*. 1998;81:1286–1291.
96. Suryapranata H, Ottervanger JP, Nibbering E, et al. Long term outcome and cost-effectiveness of stenting versus balloon angioplasty for acute myocardial infarction. *Heart*. 2001;85:667–671.
97. Saito S, Hosokawa G, Tanaka S, et al, for the PASTA Trial Investigators. Primary stent implantation is superior to balloon angioplasty in acute myocardial infarction: final results of the primary angioplasty versus stent implantation in acute myocardial infarction (PASTA) trial. *Cathet Cardiovasc Interv*. 1999;48:262–268.
98. Grines CL, Cox DA, Stone GW, et al, for the Stent Primary Angioplasty in Myocardial Infarction Study Group. Coronary angioplasty with or without stent implantation for acute myocardial infarction. *N Engl J Med*. 1999;341:1949–1956.
99. Kawashima A, Ueda K, Nishida Y, et al. Quantitative angiographic analysis of restenosis of primary stenting using Wiktor stent for acute myocardial infarction: results from a multicenter randomized PRISAM study. *Circulation*. 1999;100(suppl I):I-856. Abstract.
100. Maillard L, Hamon M, Khalife K, et al, for the STENTIM-2 Investigators. A comparison of systematic stenting and conventional balloon angioplasty during primary percutaneous transluminal coronary angio-

plasty for acute myocardial infarction. *J Am Coll Cardiol*. 2000;35:1729–1736.
101. Scheller B, Hennen B, Severin-Kneib S, et al. Long-term follow-up of a randomized study of primary stenting versus angioplasty in acute myocardial infarction. *Am J Med*. 2001;110:1–6.
102. Zhu MM, Feit A, Chadow H, et al. Primary stent implantation compared with primary balloon angioplasty for acute myocardial infarction: a meta-analysis of randomized clinical trials. *Am J Cardiol*. 2001;88:297–301.
103. Stone GW, Grines CL, Cox DA, et al, for the Controlled Abciximab and Device Investigation to Lower Late Angioplasty Complications (CADILLAC) Investigators. Comparison of angioplasty with stenting, with or without abciximab, in acute myocardial infarction. *N Engl J Med*. 2002;346:957–966.
104. Lemos PA, Saia F, Hofma SH, et al. Short- and long-term clinical benefit of sirolimus-eluting stents compared to conventional bare stents for patients with acute myocardial infarction. *J Am Coll Cardiol*. 2004;43:704–708.
104a. Ellis SG, Armstrong P, Betriu A, et al. Facilitated percutaneous coronary intervention versus primary percutaneous coronary intervention: design and rationale of the Facilitated Intervention with Enhanced Reperfusion Speed to Stop Events (FINESSE) trial. *Am Heart J*. 2004;147:E16.
105. The TIMI Research Group. Immediate vs delayed catheterization and angioplasty following thrombolytic therapy for acute myocardial infarction: TIMI II A results. *JAMA*. 1988;260:2849–2858.
106. Topol EJ, Califf RM, George BS, et al. A randomized trial of immediate versus delayed elective angioplasty after intravenous tissue plasminogen activator in acute myocardial infarction. *N Engl J Med*. 1987;317:581–588.
107. Sadanandan S, Buller C, Menon V, et al. The late open artery hypothesis: a decade later. *Am Heart J*. 2001;142:411–421.
108. Hochman JS, Sleeper LA, White HD, et al, for the SHOCK Investigators: Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock: one-year survival following early revascularization for cardiogenic shock. *JAMA*. 2001;285:190–192.
109. Dzavik V, Sleeper LA, Cocke TP, et al, for the SHOCK Investigators. Early revascularization is associated with improved survival in elderly patients with acute myocardial infarction complicated by cardiogenic shock: a report from the SHOCK Trial Registry. *Eur Heart J*. 2003;24:828–837.
110. Dauerman HL, Goldberg RJ, Malinski M, et al. Outcomes and early revascularization for patients greater than or equal to 65 years of age with cardiogenic shock. *Am J Cardiol*. 2001;87:844–848.
111. Dauerman HL, Ryan TJ, Piper WD, et al. Outcomes of percutaneous coronary intervention among elderly patients in cardiogenic shock: a multicenter, decade-long experience. *J Invasive Cardiol*. 2003;15:380–384.
112. Simoons ML, Arnold AE, Betriu A, et al. Thrombolysis with tissue plasminogen activator in acute myocardial infarction: no additional benefit from immediate percutaneous coronary angioplasty. *Lancet*. 1988;1:197–203.
113. Ross AM, Coyne KS, Reiner JS, et al, for the PACT (Plasminogen-activator Angioplasty Compatibility Trial). Investigators: A randomized trial comparing primary angioplasty with a strategy of short-acting thrombolysis and immediate planned rescue angioplasty in acute myocardial infarction: the PACT trial. *J Am Coll Cardiol*. 1999;34:1954–1962.
114. Montalescot G, Barragan P, Wittenberg O, et al, for the ADMIRAL (Abciximab before Direct Angioplasty and Stenting in Myocardial Infarction Regarding Acute and Long-Term Follow-up). Investigators. Platelet glycoprotein IIb/IIIa inhibition with coronary stenting for acute myocardial infarction. *N Engl J Med*. 2001;344:1895–1903.
115. Zeymer U, Uebis R, Vogt A, et al, for the ALKK-Study Group. Randomized comparison of percutaneous transluminal coronary angioplasty and medical therapy in stable survivors of acute myocardial infarction with single-vessel disease: a study of the Arbeitsgemeinschaft Leitende Kardiologische Krankenhausärzte. *Circulation*. 2003;108:1324–1328.
116. Scheller B, Hennen B, Hammer B, et al, for the SIAM III Study Group. Beneficial effects of immediate stenting after thrombolysis in acute myocardial infarction. *J Am Coll Cardiol*. 2003;42:634–641.
117. Braunwald E, Antman E, Beasley J, et al. ACC/AHA 2002 guideline update for the management of patients with unstable angina and non–ST-segment elevation myocardial infarction: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients With Unstable Angina). *J Am Coll Cardiol*. 2002;40:1366.
118. Stenestrand U, Wallentin L. Early revascularisation and 1-year survival in 14-day survivors of acute myocardial infarction: a prospective cohort study. *Lancet*. 2002;359:1805–1811.
119. Alter DA, Tu JV, Austin PC, et al. Waiting times, revascularization modality, and outcomes after acute myocardial infarction at hospitals with and without on-site revascularization facilities in Canada. *J Am Coll Cardiol*. 2003;42:410–419.
120. Gupta M, Chang WC, Van de Werf F, et al, for the ASSENT II Investigators. International differences in in-hospital revascularization and outcomes following acute myocardial infarction: a multilevel analysis of patients in ASSENT-2. *Eur Heart J*. 2003;24:1640–1650.
121. Gibson CM, Karha J, Murphy SA, et al, for the TIMI Study Group. Early and long-term clinical outcomes associated with reinfarction following fibrinolytic administration in the thrombolysis in myocardial infarction trials. *J Am Coll Cardiol*. 2003;42:7–16.
122. Lablanche JM, Gueret P, Blanchard D, et al. Beneficial effects of very early PTCA in patients with intravenous thrombolysis for acute myocardial infarction: data from a nation-wide registry in France. *Circulation*. 2002;106(suppl II):II-630. Abstract.
123. Fernandez-Aviles F, Alonso JJ, Castro-Beiras A, et al. Prospective randomized trial comparing a routine invasive strategy within 24 hours to thrombolysis versus an ischemia-guided conservative approach to acute myocardial infarction with ST-segment elevation: the Gracia-1 trial. *Lancet*. 2004. In press.
124. ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. Randomised trial of intravenous streptokinase, oral aspirin, both, or neither among 17,187 cases of suspected acute myocardial infarction: ISIS-2. *Lancet*. 1988;2:349–360.
125. Granger CB, Hirsch J, Califf RM, et al. Activated partial thromboplastin time and outcome after thrombolytic therapy for acute myocardial infarction: results from the GUSTO-I trial. *Circulation*. 1996;93:870–878.
126. White HD, Yusuf S. Issues regarding the use of heparin following streptokinase therapy. *J Thromb Thrombolysis*. 1995;2:5–10.
127. Thrombin-specific anticoagulation with bivalirudin versus heparin in patients receiving fibrinolytic therapy for acute myocardial infarction: the HERO-2 randomised trial. *Lancet*. 2001;358:1855–1863.
128. Bertrand ME, Rupprecht HJ, Urban P, et al. Double-blind study of the safety of clopidogrel with and without a loading dose in combination with aspirin compared with ticlopidine in combination with aspirin after coronary stenting : the Clopidogrel Aspirin Stent International Cooperative Study (CLASSICS). *Circulation*. 2000;102:624–629.
129. Mehta SR, Yusuf S, Peters RJ, et al, for the Clopidogrel in Unstable angina to prevent Recurrent Events trial (CURE) Investigators. Effects of pretreatment with clopidogrel and aspirin followed by long-term therapy in patients undergoing percutaneous coronary intervention: the PCI-CURE study. *Lancet*. 2001;358:527–533.
130. Steinhubl SR, Berger PB, Mann JT III, et al, for the CREDO (Clopidogrel for the Reduction of Events During Observation) Investigators. Early and sustained dual oral antiplatelet therapy following percutaneous coronary intervention: a randomized controlled trial. *JAMA*. 2002;288:2411–2420.
131. CYPHER™ sirolimus-eluting coronary stent on RAPTORTM over-the-wire delivery system and CYPHER™ sirolimus-eluting coronary stent on RAPTORRAIL® rapid exchange delivery system [instructions for use]. Miami, FL; Cordis, a Johnson & Johnson Company; April 2003.
132. TAXUS: paclitaxel-eluting coronary stent system Monorail and over the wire coronary stent delivery system [directions for use]. Natick, MA: Boston Scientific; March 2004.
133. Patrono C, Bachmann F, Baigent C, et al. Expert consensus document on the use of antiplatelet agents: The Task Force on the Use of Antiplatelet Agents in Patients With Atherosclerotic Cardiovascular Disease of the European Society of Cardiology. *Eur Heart J*. 2004;25:166–181.
134. Levine GN, Kern MJ, Berger PB, et al, for the American Heart Association Diagnostic and Interventional Catheterization Committee and Council on Clinical Cardiology. Management of patients undergoing percutaneous coronary revascularization. *Ann Intern Med*. 2003;139:123–136.
135. Lee DP, Herity NA, Hiatt BL, et al, for the TIrofiban Given in the Emergency Room before Primary Angioplasty. Adjunctive platelet glycoprotein IIb/IIIa receptor inhibition with tirofiban before primary angioplasty improves angiographic outcomes: results of the TIrofiban Given in the Emergency Room before Primary Angioplasty (TIGER-PA) pilot trial. *Circulation*. 2003;107:1497–1501.
136. Finney SJ, Zekveld C, Elia A, et al. Glucose control and mortality in critically ill patients. *JAMA*. 2003;290:2041–2047.

136a. Clement S, Braithwaite SS, Magee MF, et al. Management of diabetes and hyperglycemia in hospitals. *Diabetes Care*. 2004;27:553-597.
137. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106:3143–3421.
138. Malan SS. Psychosocial adjustment following MI: current views and nursing implications. *J Cardiovasc Nurs*. 1992;6:57–70.
139. Havik OE, Maeland JG. Patterns of emotional reactions after a myocardial infarction. *J Psychosom Res*. 1990;34:271–285.
140. Moser DK, Dracup K. Is anxiety early after myocardial infarction associated with subsequent ischemic and arrhythmic events? *Psychosom Med*. 1996;58:395–401.
141. Frasure-Smith N, Lespérance F, Talajic M. The impact of negative emotions on prognosis following myocardial infarction: is it more than depression? *Health Psychol*. 1995;14:388–398.
142. Chae Cu, Hennekens CH. Beta blockers. In: Hennekens CH, ed. *Clinical Trials in Cardiovascular Disease: A Companion to Braunwald's Heart Disease.* Philadelphia, Pa: WB Saunders 1999;79–94.
143. Latini R, Maggioni AP, Flather M, et al. ACE inhibitor use in patients with myocardial infarction: summary of evidence from clinical trials. *Circulation*. 1995;92:3132–3137.
144. ACE Inhibitor Myocardial Infarction Collaborative Group. Indications for ACE inhibitors in the early treatment of acute myocardial infarction: systematic overview of individual data from 100,000 patients in randomized trials. *Circulation*. 1998;97:2202–2212.
145. Flather MD, Yusuf S, Køber L, et al, for the ACE-Inhibitor Myocardial Infarction Collaborative Group. Long-term ACE-inhibitor therapy in patients with heart failure or left-ventricular dysfunction: a systematic overview of data from individual patients. *Lancet*. 2000;355:1575–1581.
146. Pitt B, Zannad F, Remme WJ, et al, for the Randomized Aldactone Evaluation Study Investigators. The effect of spironolactone on morbidity and mortality in patients with severe heart failure. *N Engl J Med*. 1999;341:709–717.
147. Pitt B, Remme W, Zannad F, et al, for the Eplerenone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study Investigators. Eplerenone, a selective aldosterone blocker, in patients with left ventricular dysfunction after myocardial infarction. *N Engl J Med*. 2003;348:1309–1321.
148. Dickstein K, Kjekshus J, for the OPTIMAAL Steering Committee of the Optimal Trial in Myocardial Infarction with Angiotensin II Antagonist Losartan (OPTIMAAL) Study Group. Effects of losartan and captopril on mortality and morbidity in high-risk patients after acute myocardial infarction: the OPTIMAAL randomised trial: Optimal Trial in Myocardial Infarction with Angiotensin II Antagonist Losartan. *Lancet*. 2002;360:752–760.
149. Pfeffer MA, McMurray JJ, Velazquez EJ, et al, for the Valsartan in Acute Myocardial Infarction Trial Investigators. Valsartan, captopril, or both in myocardial infarction complicated by heart failure, left ventricular dysfunction, or both. *N Engl J Med*. 2003;349:1893–1906.
150. Mann DL, Deswal A. Angiotensin-receptor blockade in acute myocardial infarction: a matter of dose. *N Engl J Med*. 2003;349:1963–1965.
151. Gibbons RJ, Miller TD, Christian TF. Infarct size measured by single photon emission computed tomographic imaging with (99m) Tc-sestamibi: a measure of the efficacy of therapy in acute myocardial infarction. *Circulation*. 2000;101:101–108.
152. Klocke FJ, Baird MG, Bateman TM, et al. ACC/AHA/ASNC guidelines for the clinical use of cardiac radionuclide imaging: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/ASNC Committee to Revise the 1995 Guidelines for the Clinical Use of Radionuclide Imaging). Available at: http://www.acc.org/clinical/guidelines/radio/rni_fulltext.pdf, 2003. Accessed January 15, 2004.
153. Cheitlin MD, Armstrong WF, Aurigemma GP, et al. ACC/AHA/ASE 2003 guideline update for the clinical application of echocardiography: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/ASE Committee to Update the 1997 Guidelines for the Clinical Application of Echocardiography). Available at: www.acc.org/clinical/guidelines/echo/index.pdf, 2003. Accessed January 15, 2004.
154. Mahrholdt H, Wagner A, Holly TA, et al. Reproducibility of chronic infarct size measurement by contrast-enhanced magnetic resonance imaging. *Circulation*. 2002;106:2322–2327.
155. Menon V, Slater JN, White HD, et al. Acute myocardial infarction complicated by systemic hypoperfusion without hypotension: report of the SHOCK trial registry. *Am J Med*. 2000;108:374–380.
156. Hochman JS, Boland J, Sleeper LA, et al, for the SHOCK Registry Investigators. Current spectrum of cardiogenic shock and effect of early revascularization on mortality: results of an International Registry. *Circulation*. 1995;91:873–881.
157. Hunt SA, Baker DW, Chin MH, et al. ACC/AHA guidelines for the evaluation and management of chronic heart failure in the adult: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1995 Guidelines for the Evaluation and Management of Heart Failure). Available at: http://www.acc.org/clinical/guidelines/failure/hf_index.htm, 2001. Accessed June 5, 2004.
158. Kinch JW, Ryan TJ. Right ventricular infarction. *N Engl J Med*. 1994;330:1211–1217.
159. Núñez L, de la Llana R, López Sendón J, et al. Diagnosis and treatment of subacute free wall ventricular rupture after infarction. *Ann Thorac Surg*. 1983;35:525–529.
160. Guidelines for cardiopulmonary resuscitation and emergency cardiac care: Emergency Cardiac Care Committee and Subcommittees, American Heart Association: Part III: adult advanced cardiac life support. *JAMA*. 1992;268:2199–2241.
161. Campbell RWF. Arrhythmias. In: Julian DG. Braunwald E, eds. *Management of Acute Myocardial Infarction.* London, England: WB Saunders 1994;223–240.
162. Kontoyannis DA, Anastasiou-Nana MI, Kontoyannis SA, et al. Intravenous amiodarone decreases the duration of atrial fibrillation associated with acute myocardial infarction. *Cardiovasc Drugs Ther*. 2001;15:155–160.
163. Ryan TJ, Antman EM, Brooks NH, et al. ACC/AHA guidelines for the management of patients with acute myocardial infarction: 1999 guideline update: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). *J Am Coll Cardiol*. 1999;34:890–911.
164. Gregoratos G, Abrams J, Epstein AE, et al. ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/NASPE Committee to Update the 1998 Pacemaker Guidelines). *J Am Coll Cardiol*. 2002;40:1703–1719.
165. Lamas GA, Ellenbogen KA. Evidence base for pacemaker mode selection: from physiology to randomized trials. *Circulation*. 2004;109:443–451.
166. Eagle KA, Guyton RA, Davidoff R, et al. ACC/AHA guidelines for coronary artery bypass graft surgery: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1991 Guidelines for Coronary Artery Bypass Graft Surgery). American College of Cardiology/American Heart Association. *J Am Coll Cardiol*. 1999;34:1262–1347.
167. Dyke C, Bhatia D. Inhibitors of the platelet receptor glycoprotein IIb-IIIa and complications during percutaneous coronary revascularization: management strategies for the cardiac surgeon. *J Cardiovasc Surg (Torino)*. 1999;40:505–516.
168. Yusuf S, Zhao F, Mehta SR, et al, for the Clopidogrel in Unstable Angina to Prevent Recurrent Events Trial Investigators. Effects of clopidogrel in addition to aspirin in patients with acute coronary syndromes without ST-segment elevation. *N Engl J Med*. 2001;345:494–502.
169. *Physician's Desk Reference.* Clopidogrel package insert. Montvale, NJ: Medical Economics Co; 2756.
170. Gibbons RJ, Balady GJ, Bricker JT, et al. ACC/AHA 2002 guideline update for exercise testing: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Exercise Testing). Available at: http://www.acc.org/clinical/guidelines/exercise/exercise_clean.pdf, 2002. Accessed February 12, 2004.
171. Théroux P, Waters DD, Halphen C, et al. Prognostic value of exercise testing soon after myocardial infarction. *N Engl J Med*. 1979;301:341–345.
172. DeBusk RF, Kraemer HC, Nash E, et al. Stepwise risk stratification soon after acute myocardial infarction. *Am J Cardiol*. 1983;52:1161–1166.
173. Krone RJ, Gillespie JA, Weld FM, et al. Low-level exercise testing after myocardial infarction: usefulness in enhancing clinical risk stratification. *Circulation*. 1985;71:80–89.

174. Valentine PA, Frew JL, Mashford ML, et al. Lidocaine in the prevention of sudden death in the pre-hospital phase of acute infarction: a double-blind study. *N Engl J Med*. 1974;291:1327–1331.
175. Ross J, Gilpin EA, Madsen EB, et al. A decision scheme for coronary angiography after acute myocardial infarction. *Circulation*. 1989;79: 292–303.
176. Risk stratification and survival after myocardial infarction. *N Engl J Med*. 1983;309:331–336.
177. Zaret BL, Wackers TH, Terrin M, et al. Does left ventricular ejection fraction following thrombolytic therapy have the same prognostic impact described in the prethrombolytic era? Results of the TIMI II Trial. *J Am Coll Cardiol*. 1991;17:214A. Abstract.
178. Roig E, Magriñá J, Garcia A, et al. Prognostic value of exercise radionuclide angiography in low-risk acute myocardial infarction survivors. *Eur Heart J*. 1993;14:213–218.
179. Gibbons RJ, Abrams J, Chatterjee K, et al. ACC/AHA 2002 guideline update for the management of patients with chronic stable angina: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1999 Guidelines for the Management of Patients with Chronic Stable Angina). Available at: www.acc.org/clinical/guidelines/stable/stable.pdf 2002. Accessed March 18, 2004.
180. O'Neill WW. "Watchful waiting" after thrombolysis: it's time for a re-evaluation. *J Am Coll Cardiol*. 2003;42:17–19.
181. Smith SC, Blair SN, Bonow RO, et al. AHA/ACC scientific statement: AHA/ACC guidelines for preventing heart attack and death in patients with atherosclerotic cardiovascular disease: 2001 update: a statement for healthcare professionals from the American Heart Association and the American College of Cardiology. *Circulation*. 2001;104:1577–1579.
182. Mosca L, Appel LJ, Benjamin EJ, et al. Evidence-based guidelines for cardiovascular disease prevention in women. *Circulation*. 2004;109: 672–693.
183. Dalal H, Evans PH, Campbell JL. Recent developments in secondary prevention and cardiac rehabilitation after acute myocardial infarction. *BMJ*. 2004;328:693–697.
184. Gutstein DE, Fuster V. Pathophysiologic bases for adjunctive therapies in the treatment and secondary prevention of acute myocardial infarction. *Clin Cardiol*. 1998;21:161–168.
185. CAPRIE Steering Committee. A randomised, blinded, trial of clopidogrel versus aspirin in patients at risk of ischaemic events (CAPRIE). *Lancet*. 1996;348:1329–1339.
186. Hurlen M, Smith P, Arnesen H. Effects of warfarin, aspirin and the two combined, on mortality and thromboembolic morbidity after myocardial infarction: the WARIS-II (Warfarin-Aspirin Reinfarction Study) design. *Scand Cardiovasc J*. 2000;34:168–171.
187. van Es RF, Jonker JJ, Verheugt FW, et al, for the Antithrombotics in the Secondary Prevention of Events in Coronary Thrombosis-2 (ASPECT-2) Research Group. Aspirin and coumadin after acute coronary syndromes (the ASPECT-2 study): a randomised controlled trial. *Lancet*. 2002;360:109–113.
188. Brouwer MA, van den Bergh PJ, Aengevaeren WR, et al. Aspirin plus coumarin versus aspirin alone in the prevention of reocclusion after fibrinolysis for acute myocardial infarction: results of the Antithrombotics in the Prevention of Reocclusion In Coronary Thrombolysis (APRICOT)-2 Trial. *Circulation*. 2002;106:659–665.
189. Yusuf S, Sleight P, Pogue J, et al, for the Heart Outcomes Prevention Evaluation Study Investigators. Effects of an angiotensin-converting-enzyme inhibitor, ramipril, on cardiovascular events in high-risk patients. *N Engl J Med*. 2000;342:145–153.
190. Fox KM, for the EURopean trial On reduction of cardiac events with Perindopril in stable coronary Artery disease Investigators. Efficacy of perindopril in reduction of cardiovascular events among patients with stable coronary artery disease: randomised, double-blind, placebo-controlled, multicentre trial (the EUROPA study). *Lancet*. 2003;362: 782–788.
191. Granger CB, McMurray JJ, Yusuf S, et al, for the CHARM Investigators and Committees. Effects of candesartan in patients with chronic heart failure and reduced left-ventricular systolic function intolerant to angiotensin-converting-enzyme inhibitors: the CHARM-Alternative trial. *Lancet*. 2003;362:772–776.
192. McMurray JJ, Ostergren J, Swedberg K, et al, for the CHARM Investigators and Committees. Effects of candesartan in patients with chronic heart failure and reduced left-ventricular systolic function taking angiotensin-converting-enzyme inhibitors: the CHARM-Added trial. *Lancet*. 2003;362:767–771.
193. Yusuf S, Pfeffer MA, Swedberg K, et al, for the CHARM Investigators and Committees. Effects of candesartan in patients with chronic heart failure and preserved left-ventricular ejection fraction: the CHARM-Preserved trial. *Lancet*. 2003;362:777–781.
194. Executive Council of the Heart Failure Society of America. Implications of recent clinical trials for heart failure performance measures. *J Card Fail*. 2004;10:4–5.
195. Deleted in press.
196. Antman E, Braunwald E. Acute myocardial infarction. In: Braunwald E, Zipes DP, Libby P, eds. *Heart Disease: A Textbook of Cardiovascular Medicine*. 6th ed. Philadelphia, Pa: WB Saunders;2001:1114–1251. Chapter 5.
197. Gheorghiade M, Colucci WS, Swedberg K. Beta-blockers in chronic heart failure. *Circulation*. 2003;107:1570–1575.
198. Chen J, Radford MJ, Wang Y, et al. Are beta-blockers effective in elderly patients who undergo coronary revascularization after acute myocardial infarction? *Arch Intern Med*. 2000;160:947–952.
199. Seventh report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7): resetting the hypertension sails. *Hypertension*. 2003;41: 1178–1179.
200. Hulley S, Grady D, Bush T, et al, for the Heart and Estrogen/progestin Replacement Study (HERS) Research Group. Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women. *JAMA*. 1998;280:605–613.
201. Grady D, Herrington D, Bittner V, et al, for the HERS Research Group. Cardiovascular disease outcomes during 6.8 years of hormone therapy: Heart and Estrogen/progestin Replacement Study follow-up (HERS II). *JAMA*. 2002;288:49–57.
202. Rossouw JE, Anderson GL, Prentice RL, et al, for the Writing Group for the Women's Health Initiative Investigators. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women's Health Initiative randomized controlled trial. *JAMA*. 2002;288:321–333.
203. Cairns JA, Markham BA. Economics and efficacy in choosing oral anticoagulants or aspirin after myocardial infarction. *JAMA*. 1995;273:965–967.
203a. Kris-Etherton PM, Lichtenstein AH, Howard BV, Steinberg D, Witzman JL. Antioxidant vitamin supplements and cardiovascular disease. *Circulation*. In press.
204. Glassman AH, O'Connor CM, Califf RM, et al, for the Sertraline Antidepressant Heart Attack Randomized Trial (SADHEART) Group. Sertraline treatment of major depression in patients with acute MI or unstable angina. *JAMA*. 2002;288:701–709.
205. Swenson JR, O'Connor CM, Barton D, et al, for the Sertraline Antidepressant Heart Attack Randomized Trial (SADHART) Group. Influence of depression and effect of treatment with sertraline on quality of life after hospitalization for acute coronary syndrome. *Am J Cardiol*. 2003; 92:1271–1276.
206. Berkman LF, Blumenthal J, Burg M, et al, for the Enhancing Recovery in Coronary Heart Disease Patients Investigators (ENRICHD). Effects of treating depression and low perceived social support on clinical events after myocardial infarction: the Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD) Randomized Trial. *JAMA*. 2003; 289:3106–3116.
207. Alonzo AA. Acute myocardial infarction and posttraumatic stress disorder: the consequences of cumulative adversity. *J Cardiovasc Nurs*. 1999;13:33–45.

ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction—Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infarction)

Writing Committee Members, Elliott M. Antman, Daniel T. Anbe, Paul Wayne Armstrong, Eric R. Bates, Lee A. Green, Mary Hand, Judith S. Hochman, Harlan M. Krumholz, Frederick G. Kushner, Gervasio A. Lamas, Charles J. Mullany, Joseph P. Ornato, David L. Pearle, Michael A. Sloan, Sidney C. Smith, Jr, Elliott M. Antman, Sidney C. Smith, Jr, Joseph S. Alpert, Jeffrey L. Anderson, David P. Faxon, Valentin Fuster, Raymond J. Gibbons, Gabriel Gregoratos, Jonathan L. Halperin, Loren F. Hiratzka, Sharon Ann Hunt, Alice K. Jacobs and Joseph P. Ornato

Circulation. 2004;110:588-636
doi: 10.1161/01.CIR.0000134791.68010.FA
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0009-7322. Online ISSN: 1524-4539

Corrections

In the article by Antman et al, "ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction—Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infarction)," which appeared in the August 3, 2004, issue of the journal (*Circulation*. 2004:110:588–636), the following errors occur:

- Table 3 (p 619): In the footnote entry "How to Use the Table," the example text states that Temporary transvenous pacing (TV) is Class IIb, whereas the table shows TV to be Class IIa. This should be Class IIa in both instances.
- Table 4 (p 626): The footnote entry listing the source of the data is incorrect. The correct citation should be "Smith et al. *Circulation*. 2001;104:1577–1579."
- Page 630: The recommendation for Cardiac Rehabilitation programs should be a Class I recommendation, not a Class IIa recommendation.

The corrected version of this article is available online at http://circ.ahajournals.org/cgi/content/full/110/5/588. (The previous version, if needed, can be accessed by selecting the "Previous Version of This Article" link.)

DOI: 10.1161/01.CIR.0000163465.65091.7A

In the article by Antman et al, "ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infarction)," which appeared in the August 31, 2004, issue of the journal (*Circulation*. 2004;110:e82–e292), the following errors occur:

- Table 12 (p e127): The entry "Traumatic or prolonged (greater than 10 minutes) CPR or major surgery (within less than 3 weeks)" should read "Traumatic or prolonged (greater than 10 minutes) CPR or major surgery (less than 3 weeks)."
- Table 13 (p e129): In the Model column for CCP and for InTIME-2, the SBP values on admission of 170 mm Hg or greater=1 entries should be deleted.
- Table 23 (p e166): Row 2 is incorrect. It should read "2. IV or D5W to keep the vein open. Start a second IV if IV medication is being given. This may be a heparin lock."
- Table 23 (p e166): Row 3 is incorrect. It should read "3. Vital signs: every 30 min until stable, then every 4 h as needed. Notify physician if HR is less than 60 bpm or greater than 100 bpm, systolic BP is less than 100 mm Hg or greater than 150 mm Hg, respiratory rate is less than 8 breaths per minute or greater than 22 breaths per minute."
- Table 23 (p e166): Row 5 is incorrect. It should read "5. Diet: NPO except for sips of water until stable. Then start 2 gram sodium/day, low saturated fat (less than 7% total calories/day), low cholesterol (less than 200 mg/d) diet, such as Therapeutic Lifestyle Changes (TLC) diet."
- Table 23 (p e166): Row 6 is incorrect. It should read "6. Activity: Bedrest and bedside commode and light activity when stable."
- Table 29 (p e198): Column 1 is missing a heading. It should read "Normal."
- Table 29 (p e199): The "How to Use the Table" footnote line 4 states that Temporary transvenous pacing (TV) is Class IIb, whereas the table according to the instructions shows that TV should be Class IIa. The footnote should read Class IIa.
- Page e237: The entry for Class I number 9 is incorrect. It should read "Class I. Cardiac

(*Circulation.* 2005;111:2013–2014.)

***Corrections* is available at http://www.circulationaha.org**

rehabilitation/secondary prevention programs, when available, are recommended for patients with STEMI, particularly those with multiple modifiable risk factors and/or those moderate- to high-risk patients for whom supervised exercise training is warranted. (Level of Evidence: C)"

- Page e239: Under the staff listing for the American Heart Association, the name of Fernando Costa, MD, FAHA, Staff Scientist, was omitted.
- Page e247: The entry for Peer Reviewer Dr Frans Van de Werf under the column Stock Ownership should read "None."

The corrected version of this article is available online at http://circ.ahajournals.org/cgi/reprint/110/9/e82. (The previous version, if needed, can be accessed by selecting the "Previous Version of This Article" link.)

DOI: 10.1161/01.CIR.0000163471.93598.4A

In the article by Eagle and Guyton et al, "ACC/AHA 2004 Guideline Update for Coronary Artery Bypass Graft Surgery: Summary Article," which appeared in the August 31, 2004, issue of the journal (*Circulation*. 2004;110:1168–1176), the following error occurred:

On page 1175, the Class IIa recommendation in Section 9.2.4, "ST-Elevation MI," should read as follows: "CABG may be performed as primary reperfusion in patients who have suitable anatomy and who are not candidates for or who have had failed fibrinolysis/PCI and who are in the early hours (6 to 12 hours) of evolving STEMI (Level of Evidence: B)."

The corrected version of this article is available online at http://circ.ahajournals.org/cgi/content/full/110/9/1168. (The previous version, if needed, can be accessed by selecting the "Previous Version of This Article" link.)

DOI: 10.1161/01.CIR.0000163473.82675.77

In the article by Eagle and Guyton et al, "ACC/AHA 2004 Guideline Update for Coronary Artery Bypass Graft Surgery—Full Text," which appeared in the October 5, 2004, issue of the journal (*Circulation*. 2004;110:e340–e437), the following errors occurred:

- Page e348: The footnote to Table 3 should read as follows: "**Calculation of Mortality Risk**: An 80-year old female, with an EF less than 40% who is having elective CABG surgery, has had no prior CABG surgery and has no other risk factors. Her total score=6.5 (age greater than or equal to 80) +2 (female sex) +2 (EF less than 40%)=10.5. Since her total score equals 10.5, round up to 11; her predicted risk of mortality=4.0%."
- Page e371: In Table 13, the value for the Class Indication for Preoperative Carotid screening should be IIa, not I as it currently shows.

The corrected version of this article is available online at http://circ.ahajournals.org/cgi/reprint/110/14/e340. (The previous version, if needed, can be accessed by selecting the "Previous Version of This Article" link.)

DOI: 10.1161/01.CIR.0000163478.50217.83

ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction—Executive Summary

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infarction)

Developed in Collaboration With the Canadian Cardiovascular Society

WRITING COMMITTEE MEMBERS

Elliott M. Antman, MD, FACC, FAHA, Chair; Daniel T. Anbe, MD, FACC, FAHA; Paul Wayne Armstrong, MD, FACC, FAHA; Eric R. Bates, MD, FACC, FAHA; Lee A. Green, MD, MPH; Mary Hand, MSPH, RN, FAHA; Judith S. Hochman, MD, FACC, FAHA; Harlan M. Krumholz, MD, FACC, FAHA; Frederick G. Kushner, MD, FACC, FAHA; Gervasio A. Lamas, MD, FACC; Charles J. Mullany, MB, MS, FACC; Joseph P. Ornato, MD, FACC, FAHA; David L. Pearle, MD, FACC, FAHA; Michael A. Sloan, MD, FACC; Sidney C. Smith, Jr, MD, FACC, FAHA

TASK FORCE MEMBERS

Elliott M. Antman, MD, FACC, FAHA, Chair; Sidney C. Smith, Jr, MD, FACC, FAHA, Vice-chair; Joseph S. Alpert, MD, FACC, FAHA*; Jeffrey L. Anderson, MD, FACC, FAHA; David P. Faxon, MD, FACC, FAHA; Valentin Fuster, MD, PhD, FACC, FAHA; Raymond J. Gibbons, MD, FACC, FAHA*†; Gabriel Gregoratos, MD, FACC, FAHA*; Jonathan L. Halperin, MD, FACC, FAHA; Loren F. Hiratzka, MD, FACC, FAHA; Sharon Ann Hunt, MD, FACC, FAHA; Alice K. Jacobs, MD, FACC, FAHA; Joseph P. Ornato, MD, FACC, FAHA

This document was approved by the American College of Cardiology Foundation Board of Trustees on May 7, 2004 and by the American Heart Association Science Advisory and Coordinating Committee on May 5, 2004.

The ACC/AHA Task Force on Practice Guidelines makes every effort to avoid any actual or potential conflicts of interest that might arise as a result of an outside relationship or personal interest of a member of the writing panel. Specifically, all members of the writing panel are asked to provide disclosure statements of all such relationships that might be perceived as real or potential conflicts of interest. These statements are reviewed by the parent task force, reported orally to all members of the writing panel at the first meeting, and updated as changes occur. The relationship with industry information for the writing committee members is posted on the ACC and AHA World Wide Web sites with the full-length version of the update, along with the names and relationships with industry of the peer reviewers.

When citing this document, the American College of Cardiology Foundation and the American Heart Association would appreciate the following citation format: Antman EM, Anbe DT, Armstrong PW, Bates ER, Green LA, Hand M, Hochman JS, Krumholz HM, Kushner FG, Lamas GA, Mullany CJ, Ornato JP, Pearle DL, Sloan MA, Smith SC Jr. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: executive summary: a report of the ACC/AHA Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines on the Management of Patients With Acute Myocardial Infarction). *Circulation*. 2004;110:588–636.

Copies: This document and the full-text guideline are available on the World Wide Web sites of the American College of Cardiology (www.acc.org), the American Heart Association (www.americanheart.org), and the Canadian Cardiovascular Society (www.ccs.ca). Single copies of this executive summary, published in the August 4, 2004 issue of the *Journal of the American College of Cardiology* or the August 3, 2004 issue of *Circulation* or the companion full-text guideline are available for $10.00 each by calling 1-800-253-4636 or writing to the American College of Cardiology Foundation, Resource Center, 9111 Old Georgetown Road, Bethesda, MD 20814-1699. To purchase bulk reprints (specify version and reprint number: 71-0294 for the executive summary; 71-0293 for the full-text guideline): up to 999 copies, call 1-800-611-6083 (US only) or fax 413-665-2671; 1000 or more copies, call 214-706-1789, fax 214-691-6342, or e-mail pubauth@heart.org.

*Former Task Force member.

†Immediate Past Chair.

(*Circulation*. 2004;110:588-636.)

***Circulation* is available at http://www.circulationaha.org** **DOI: 10.1161/01.CIR.0000134791.68010.FA**

Table of Contents

I. Introduction

Although considerable improvement has occurred in the process of care for patients with ST-elevation myocardial infarction (STEMI), room for improvement exists.[1–3] The purpose of the present guideline is to focus on the numerous advances in the diagnosis and management of patients with STEMI since 1999. This is reflected in the changed name of the guideline: "ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction." The final recommendations for indications for a diagnostic procedure, a particular therapy, or an intervention in patients with STEMI summarize both clinical evidence and expert opinion (Table 1). To provide clinicians with a set of recommendations that can easily be translated into the practice of caring for patients with STEMI, this guideline is organized around the chronology of the interface between the patient and the clinician. The full guideline is available at http://www.acc.org/clinical/guidelines/stemi/index.htm.

II. Pathology

A. Epidemiology

STEMI continues to be a significant public health problem in industrialized countries and is becoming an increasingly significant problem in developing countries.[4] Although the exact incidence is difficult to ascertain, using first-listed and secondary hospital discharge data, there were 1 680 000 unique discharges for ACS in 2001.[5] Applying the conservative estimate of 30% of the ACS patients who have STEMI from the National Registry of Myocardial Infarction-4 [NRMI-4],[5a] we estimate 500 000 STEMI events per year in the U.S. This writing committee strongly endorses several public health campaigns that are likely to contribute to a reduction in the incidence of and fatality from STEMI in the future and additional research of new strategies for the management of STEMI patients in the community.[6–13]

III. Management Before STEMI

A. Identification of Patients at Risk of STEMI

Class I

1. **Primary care providers should evaluate the presence and status of control of major risk factors for coronary heart disease (CHD) for all patients at regular intervals (approximately every 3 to 5 years). (*Level of Evidence: C*)**
2. **Ten-year risk (National Cholesterol Education Program [NCEP] global risk) of developing symptomatic CHD should be calculated for all patients who have 2 or more major risk factors to assess the need for primary prevention strategies.[14] (*Level of Evidence: B*)**
3. **Patients with established CHD should be identified for secondary prevention, and patients with a CHD risk equivalent (eg, diabetes mellitus, chronic kidney disease, or 10-year risk greater than 20% as calculated by Framingham equations) should receive equally intensive risk factor intervention as those with clinically apparent CHD. (*Level of Evidence: A*)**

B. Patient Education for Early Recognition and Response to STEMI

Class I

1. **Patients with symptoms of STEMI (chest discomfort with or without radiation to the arms[s], back, neck, jaw, or epigastrium; shortness of breath; weakness; diaphoresis; nausea; lightheadedness) should be transported to the hospital by ambulance rather than by friends or relatives. (*Level of Evidence: B*)**
2. **Healthcare providers should actively address the following issues regarding STEMI with patients and their families:**
   a. **The patient's heart attack risk (*Level of Evidence: C*)**
   b. **How to recognize symptoms of STEMI (*Level of Evidence: C*)**
   c. **The advisability of calling 9-1-1 if symptoms are unimproved or worsening after 5 minutes, despite feelings of uncertainty about the symptoms and**

TABLE 1. Applying Classification of Recommendations and Level of Evidence

"Estimate of Certainty (Precision) of Treatment of Effect"	"Size of Treatment Effect"			
	Class I	Class IIa	Class IIb	Class III
	Benefit >>> Risk Procedure/Treatment SHOULD be performed/administered	*Benefit >> Risk* *Additional studies with focused objectives needed* IT IS REASONABLE to perform procedure/administer treatment	*Benefit ≥ Risk* *Additional studies with broad objectives needed; additional registry data would be helpful* Procedure/Treatment MAY BE CONSIDERED	*Risk ≥ Benefit* *No additional studies needed* Procedure/Treatment should NOT be performed/administered SINCE IT IS NOT HELPFUL AND MAY BE HARMFUL
Level A *Multiple (3–5) population risk strata evaluated** *General consistency of direction and magnitude of effect*	• Recommendation that procedure or treatment is useful/effective • Sufficient evidence from multiple randomized trials or meta-analyses	• Recommendation in favor of treatment or procedure being useful/effective • Some conflicting evidence from multiple randomized trials or meta-analyses	• Recommendation's usefulness/efficacy less well established • Greater conflicting evidence from multiple randomized trials or meta-analyses	• Recommendation that procedure or treatment is not useful/effective and may be harmful • Sufficient evidence from multiple randomized trials or meta-analyses
Level B *Limited (2–3) population risk strata evaluated**	• Recommendation that procedure or treatment is useful/effective • Limited evidence from single randomized trial or nonrandomized studies	• Recommendation in favor of treatment or procedure being useful/effective • Some conflicting evidence from single randomized trial or nonrandomized studies	• Recommendation's usefulness/efficacy less well established • Greater conflicting evidence from single randomized trial or nonrandomized studies	• Recommendation that procedure or treatment is not useful/effective and may be harmful • Limited evidence from single randomized trial or nonrandomized studies
Level C *Very limited (1–2) population risk strata evaluated**	• Recommendation that procedure or treatment is useful/effective • Only expert opinion, case studies, or standard-of-care	• Recommendation in favor of treatment or procedure being useful/effective • Only diverging expert opinion, case studies, or standard-of-care	• Recommendation's usefulness/efficacy less well established • Only diverging expert opinion, case studies, or standard-of-care	• Recommendation that procedure or treatment is not useful/effective and may be harmful • Only expert opinion, case studies, or standard-of-care
Suggested phrases for writing recommendations†	should is recommended is indicated is useful/effective/beneficial	is reasonable can be useful/effective/beneficial is probably recommended or indicated	may/might be considered may/might be reasonable usefulness/effectiveness is unknown/unclear/uncertain or not well established	is not recommended is not indicated should not is not useful/effective/beneficial may be harmful

*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations, such as gender, age, history of diabetes, history of prior MI, history of heart failure, and prior aspirin use.

†The ACC/AHA Task Force on Practice Guidelines recently provided a list of suggested phrases to use when writing recommendations. All recommendations in the STEMI guideline have been written in full sentences that express a complete thought, such that a recommendation, even if separated and presented apart from the rest of the document (including headings above sets of recommendations), would still convey the full intent of the recommendation. It is hoped that this will increase readers' comprehension of the guidelines and will allow queries at the individual recommendation level.

fear of potential embarrassment (*Level of Evidence: C*)

d. A plan for appropriate recognition and response to a potential acute cardiac event that includes the phone number to access emergency medical services (EMS), generally 9-1-1.[15] (*Level of Evidence: C*)

3. Healthcare providers should instruct patients for whom nitroglycerin has been prescribed previously to take ONE nitroglycerin dose sublingually in response to chest discomfort/pain. If chest discomfort/pain is unimproved or worsening 5 minutes after 1 sublingual nitroglycerin dose has been taken, it is recommended that the patient or family member/friend call 9-1-1 immediately to access EMS. (*Level of Evidence: C*)

Morbidity and mortality due to STEMI can be reduced significantly if patients and bystanders recognize symptoms early, activate the EMS system, and thereby shorten the time to definitive treatment. Patients with possible symptoms of STEMI should be transported to the hospital by ambulance rather than by friends or relatives because there is a significant association between arrival at the emergency department (ED) by ambulance and early reperfusion therapy.[16–19] Although the traditional recommendation is for patients to take 1 nitroglycerin dose sublingually, 5 minutes apart, for up to 3 doses before

calling for emergency evaluation, this recommendation has been modified by the writing committee to encourage earlier contacting of EMS by patients with symptoms suggestive of STEMI.[20,21]

IV. Onset of STEMI

A. Out-of-Hospital Cardiac Arrest

Class I

1. **All communities should create and maintain a strong "Chain of Survival" for out-of-hospital cardiac arrest that includes early access (recognition of the problem and activation of the EMS system by a bystander), early cardiopulmonary resuscitation (CPR), early defibrillation for patients who need it, and early advanced cardiac life support (ACLS). *(Level of Evidence: C*)**
2. **Family members of patients experiencing STEMI should be advised to take CPR training and familiarize themselves with the use of an automated external defibrillator (AED). In addition, they should be referred to a CPR training program that has a social support component for family members of post-STEMI patients. (*Level of Evidence: B*)**

The links in the chain include early access (recognition of the problem and activation of the EMS system by a bystander), early CPR, early defibrillation for patients who need it, and early ACLS.

V. Prehospital Issues

A. Emergency Medical Services Systems

Class I

1. **All EMS first responders who respond to patients with chest pain and/or suspected cardiac arrest should be trained and equipped to provide early defibrillation. (*Level of Evidence: A*)**
2. **All public safety first responders who respond to patients with chest pain and/or suspected cardiac arrest should be trained and equipped to provide early defibrillation with AEDs. (Provision of early defibrillation with AEDs by nonpublic safety first responders is a promising new strategy, but further study is needed to determine its safety and efficacy.) (*Level of Evidence: B*)**
3. **Dispatchers staffing 9-1-1 center emergency medical calls should have medical training, should use nationally developed and maintained protocols, and should have a quality-improvement system in place to ensure compliance with protocols. (*Level of Evidence: C*)**

Early access to EMS is promoted by a 9-1-1 system currently available to more than 90% of the US population. To minimize time to treatment, particularly for cardiopulmonary arrest, many communities allow volunteer and/or paid firefighters and other first-aid providers to function as first responders, providing CPR and, increasingly, early defibrillation using automated external defibrillators (AEDs) until emergency medical technicians and paramedics arrive. Most cities and larger suburban areas provide EMS ambulance services with providers from the fire department, a private ambulance company, and/or volunteers.

B. Prehospital Chest Pain Evaluation and Treatment

Class I

1. **Prehospital EMS providers should administer 162 to 325 mg of aspirin (chewed) to chest pain patients suspected of having STEMI unless contraindicated or already taken by patient. Although some trials have used enteric-coated aspirin for initial dosing, more rapid buccal absorption occurs with non–enteric-coated formulations. (*Level of Evidence: C*)**

Class IIa

1. **It is reasonable for all 9-1-1 dispatchers to advise patients without a history of aspirin allergy who have symptoms of STEMI to chew aspirin (162 to 325 mg) while awaiting arrival of prehospital EMS providers. Although some trials have used enteric-coated aspirin for initial dosing, more rapid buccal absorption occurs with non–enteric-coated formulations. *(Level of Evidence: C*)**
2. **It is reasonable that all ACLS providers perform and evaluate 12-lead electrocardiograms (ECGs) routinely on chest pain patients suspected of STEMI. *(Level of Evidence: B)***
3. **If the ECG shows evidence of STEMI, it is reasonable that prehospital ACLS providers review a reperfusion "checklist" and relay the ECG and checklist findings to a predetermined medical control facility and/or receiving hospital. (*Level of Evidence: C*)**

It is reasonable for physicians to encourage the prehospital administration of aspirin via EMS personnel (ie, EMS dispatchers and providers) in patients with symptoms suggestive of STEMI unless its use is contraindicated.[22] For patients who have ECG evidence of STEMI, it is reasonable that paramedics review a reperfusion checklist and relay the ECG and checklist findings to a predetermined medical control facility and/or receiving hospital.

C. Prehospital Fibrinolysis

Class IIa

1. **Establishment of a prehospital fibrinolysis protocol is reasonable in 1) settings in which physicians are present in the ambulance or in 2) well-organized EMS systems with full-time paramedics who have 12-lead ECGs in the field with transmission capability, paramedic initial and ongoing training in ECG interpretation and STEMI treatment, online medical command, a medical director with training/experience in STEMI management, and an ongoing continuous quality-improvement program. (*Level of Evidence: B*)**

Randomized controlled trials of fibrinolytic therapy have demonstrated the benefit of initiating fibrinolytic therapy as early as possible after onset of ischemic-type chest discomfort (Figure 1).[23–25] It appears reasonable to expect that if

fibrinolytic therapy could be started at the time of prehospital evaluation, a greater number of lives could be saved. Prehospital fibrinolysis is reasonable in those settings in which physicians are present in the ambulance or prehospital transport times are more than 60 minutes in high-volume (more than 25,000 runs per year) EMS systems.[26] Other considerations for implementing a prehospital fibrinolytic service include the ability to transmit ECGs, paramedic initial and ongoing training in ECG interpretation and myocardial infarction (MI) treatment, online medical command, a medical director with training/experience in management of STEMI, and full-time paramedics.[27]

D. Prehospital Destination Protocols

Class I

1. **Patients with STEMI who have cardiogenic shock and are less than 75 years of age should be brought immediately or secondarily transferred to facilities capable of cardiac catheterization and rapid revascularization (percutaneous coronary intervention [PCI] or coronary artery bypass graft surgery [CABG]) if it can be performed within 18 hours of onset of shock. (*Level of Evidence: A*)**
2. **Patients with STEMI who have contraindications to fibrinolytic therapy should be brought immediately or secondarily transferred promptly (ie, primary-receiving hospital door-to-departure time less than 30 minutes) to facilities capable of cardiac catheterization and rapid revascularization (PCI or CABG). (*Level of Evidence: B*)**
3. **Every community should have a written protocol that guides EMS system personnel in determining where to take patients with suspected or confirmed STEMI. (*Level of Evidence: C*)**

Class IIa

1. **It is reasonable that patients with STEMI who have cardiogenic shock and are 75 years of age or older be considered for immediate or prompt secondary transfer to facilities capable of cardiac catheterization and rapid revascularization (PCI or CABG) if it can be performed within 18 hours of onset of shock. (*Level of Evidence: B*)**
2. **It is reasonable that patients with STEMI who are at especially high risk of dying, including those with severe congestive heart failure (CHF), be considered for immediate or prompt secondary transfer (ie, primary-receiving hospital door-to-departure time less than 30 minutes) to facilities capable of cardiac catheterization and rapid revascularization (PCI or CABG). (*Level of Evidence: B*)**

Every community should have a written protocol that guides EMS system personnel in determining where to take patients with suspected or confirmed STEMI. Active involvement of local healthcare providers, particularly cardiologists and emergency physicians, is needed to formulate local EMS destination protocols for these patients. In general, patients with suspected STEMI should be taken to the nearest appropriate hospital. However, patients with STEMI and shock are an exception to this general rule. Whenever possible, STEMI patients less than 75 years of age with shock should be transferred to facilities capable of cardiac catheterization and rapid revascularization (PCI or CABG). On the basis of observations in the SHOCK Trial Registry and other registries, it is reasonable to extend such considerations of transfer to invasive centers for elderly patients with shock (see VII.F.5 and Section 7.6.5 of the full-text guidelines). Patients with STEMI who have contraindications to fibrinolytic therapy should be brought immediately or secondarily transferred promptly (ie, primary-receiving hospital door-to-departure time less than 30 minutes) to facilities capable of cardiac catheterization and rapid revascularization (PCI or CABG).

VI. Initial Recognition and Management in the Emergency Department

A. Optimal Strategies for Emergency Department Triage

Class I

1. **Hospitals should establish multidisciplinary teams (including primary care physicians, emergency medicine physicians, cardiologists, nurses, and laboratorians) to develop guideline-based, institution-specific written protocols for triaging and managing patients who are seen in the prehospital setting or present to the ED with symptoms suggestive of STEMI. (*Level of Evidence: B*)**

B. Initial Patient Evaluation

Class I

1. **The delay from patient contact with the healthcare system (typically, arrival at the ED or contact with paramedics) to initiation of fibrinolytic therapy should be less than 30 minutes. Alternatively, if PCI is chosen, the delay from patient contact with the healthcare system (typically, arrival at the ED or contact with paramedics) to balloon inflation should be less than 90 minutes. (*Level of Evidence: B*)**
2. **The choice of initial STEMI treatment should be made by the emergency medicine physician on duty based on a predetermined, institution-specific, written protocol that is a collaborative effort of cardiologists (both those involved in coronary care unit management and interventionalists), emergency physicians, primary care physicians, nurses, and other appropriate personnel. For cases in which the initial diagnosis and treatment plan is unclear to the emergency physician or is not covered directly by the agreed-on protocol, immediate cardiology consultation is advisable. (*Level of Evidence: C*)**

Regardless of the approach used, all patients presenting to the ED with chest discomfort or other symptoms suggestive of STEMI or unstable angina should be considered high-priority triage cases and should be evaluated and treated based on a predetermined, institution-specific chest pain protocol. The goal for patients with STEMI should be to

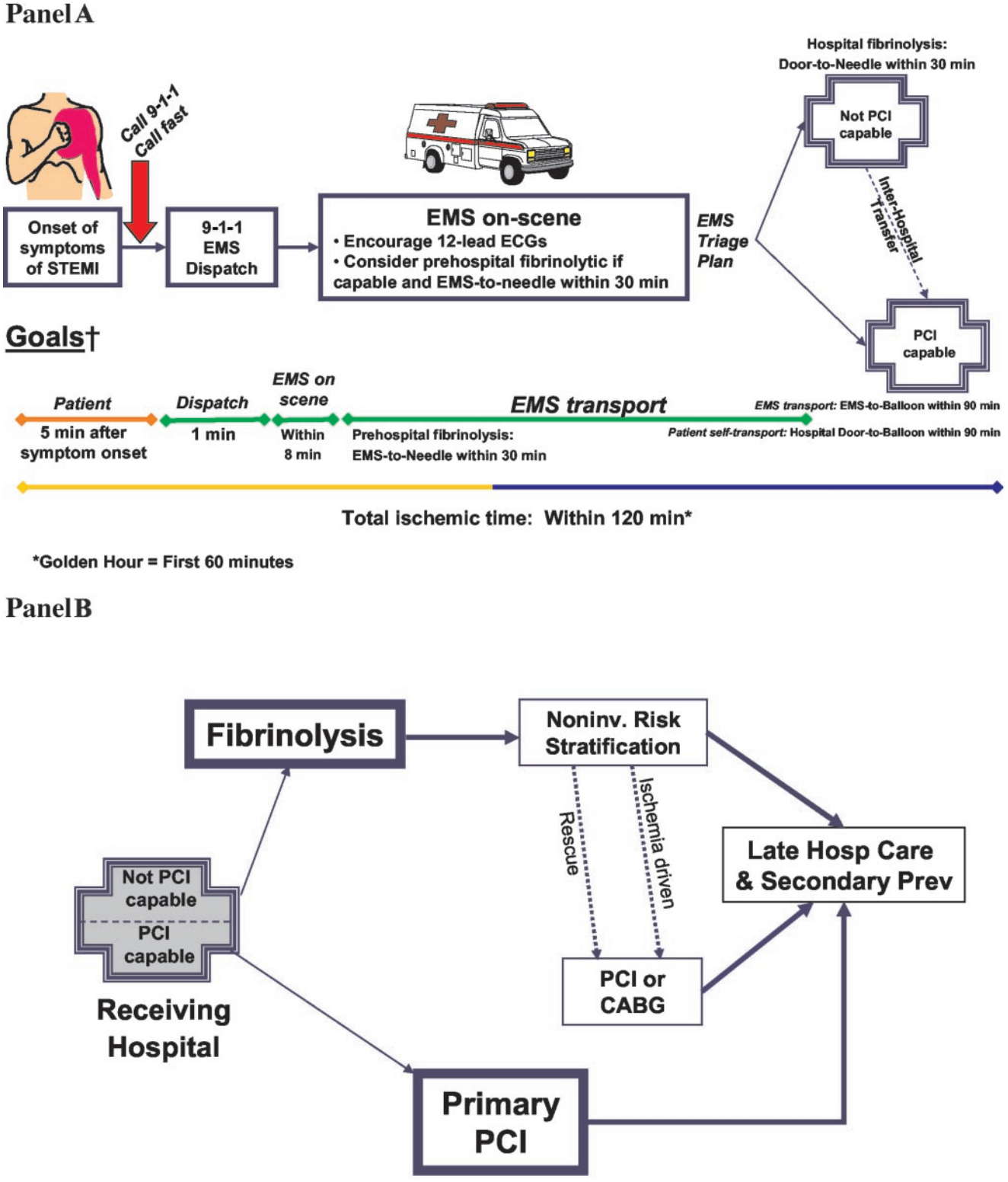

Figure 1. Options for transportation of STEMI patients and initial reperfusion treatment. Panel A, Patient transported by EMS after calling 9-1-1: Reperfusion in patients with STEMI can be accomplished by the pharmacological (fibrinolysis) or catheter-based (primary PCI) approaches. Implementation of these strategies varies based on the mode of transportation of the patient and capabilities at the receiving hospital. Transport time to the hospital is variable from case to case, but the goal is to keep total ischemic time within 120 minutes. There are 3 possibilities: (1) If EMS has fibrinolytic capability and the patient qualifies for therapy, prehospital fibrinolysis should be started within 30 minutes of EMS arrival on scene. (2) If EMS is not capable of administering prehospital fibrinolysis and the patient is transported to a non–PCI-capable hospital, the hospital door-to-needle time should be within 30 minutes for patients in whom fibrinolysis is indicated. (3) If EMS is not capable of administering prehospital fibrinolysis and the patient is transported to a PCI-capable hospital, the hospital door-to-balloon time should be within 90 minutes. Interhospital transfer: It is also appropriate to consider emergency interhospital transfer of the patient to a PCI-capable hospital for mechanical revascularization if (1) there is a contraindication to fibrinolysis; (2) PCI can be initiated promptly

achieve a door-to-needle time within 30 minutes and a door-to-balloon time within 90 minutes (Figure 1).[25]

1. History

Class I

1. **The targeted history of STEMI patients taken in the ED should ascertain whether the patient has had prior episodes of myocardial ischemia such as stable or unstable angina, MI, CABG, or PCI. Evaluation of the patient's complaints should focus on chest discomfort, associated symptoms, sex- and age-related differences in presentation, hypertension, diabetes mellitus, possibility of aortic dissection, risk of bleeding, and clinical cerebrovascular disease (amaurosis fugax, face/limb weakness or clumsiness, face/limb numbness or sensory loss, ataxia, or vertigo). (*Level of Evidence: C*)**

2. Physical Examination

Class I

1. **A physical examination should be performed to aid in the diagnosis and assessment of the extent, location, and presence of complications of STEMI. (*Level of Evidence: C*)**
2. **A brief, focused, and limited neurological examination to look for evidence of prior stroke or cognitive deficits should be performed on STEMI patients before administration of fibrinolytic therapy. (*Level of Evidence: C*)**

A brief physical examination may promote rapid triage, whereas a more detailed physical examination aids in the differential diagnosis and is useful for assessing the extent, location, and presence of complications of STEMI.

3. Electrocardiogram

Class I

1. **A 12-lead ECG should be performed and shown to an experienced emergency physician within 10 minutes of ED arrival for all patients with chest discomfort (or anginal equivalent) or other symptoms suggestive of STEMI. (*Level of Evidence: C*)**
2. **If the initial ECG is not diagnostic of STEMI but the patient remains symptomatic, and there is a high clinical suspicion for STEMI, serial ECGs at 5- to 10-minute intervals or continuous 12-lead ST-segment monitoring should be performed to detect the potential development of ST elevation. (*Level of Evidence: C*)**
3. **In patients with inferior STEMI, right-sided ECG leads should be obtained to screen for ST elevation suggestive of right ventricular (RV) infarction. (See Section 7.6.6 of the full-text guidelines and the ACC/AHA/ASE 2003 Guideline Update for the Clinical Application of Echocardiography.) (*Level of Evidence: B*)**

The 12-lead ECG in the ED is at the center of the therapeutic decision pathway because of the strong evidence that ST-segment elevation identifies patients who benefit from reperfusion therapy.[28]

4. Laboratory Examinations

Class I

1. **Laboratory examinations should be performed as part of the management of STEMI patients but should not delay the implementation of reperfusion therapy. (*Level of Evidence: C*)**

In addition to serum cardiac biomarkers for cardiac damage, several routine evaluations have important implications for management of patients with STEMI. Although these studies should be ordered when the patient is first seen, therapeutic decisions should not be delayed until results are obtained because of the crucial role of time to therapy in STEMI.

5. Biomarkers of Cardiac Damage

Class I

1. **Cardiac-specific troponins should be used as the optimum biomarkers for the evaluation of patients with STEMI who have coexistent skeletal muscle injury. (*Level of Evidence: C*)**
2. **For patients with ST elevation on the 12-lead ECG and symptoms of STEMI, reperfusion therapy should be initiated as soon as possible and is not contingent on a biomarker assay. (*Level of Evidence: C*)**

Class IIa

1. **Serial biomarker measurements can be useful to provide supportive noninvasive evidence of reperfusion of the infarct artery after fibrinolytic therapy in patients**

Figure 1 (continued). (within 90 minutes after the patient presented to the initial receiving hospital or within 60 minutes compared to when fibrinolysis with a fibrin-specific agent could be initiated at the initial receiving hospital); or (3) fibrinolysis is administered and is unsuccessful (ie, "rescue PCI"). Secondary nonemergency interhospital transfer can be considered for recurrent ischemia. Patient self-transport: Patient self-transportation is discouraged. If the patient arrives at a non–PCI-capable hospital, the door-to-needle time should be within 30 minutes. If the patient arrives at a PCI-capable hospital, the door-to-balloon time should be within 90 minutes. The treatment options and time recommendations after first hospital arrival are the same. Panel B, For patients who receive fibrinolysis, noninvasive risk stratification is recommended to identify the need for rescue PCI (failed fibrinolysis) or ischemia-driven PCI. See Sections 6.3.1.6.4.5. and 6.3.1.6.7. in the full-text guidelines. Regardless of the initial method of reperfusion treatment, all patients should receive late hospital care and secondary prevention of STEMI. EMS indicates Emergency Medical System; PCI, percutaneous coronary intervention; CABG, coronary artery bypass graft surgery; Hosp, hospital; Noninv., Noninvasive. * Golden hour = First 60 minutes;† The medical system goal is to facilitate rapid recognition and treatment of patients with STEMI such that door-to-needle (or medical contact–to-needle) time for initiation of fibrinolytic therapy is within 30 minutes or that door-to-balloon (or medical contact–to-balloon) time for PCI is within 90 minutes. These goals should not be understood as ideal times but rather as the longest times that should be considered acceptable for a given system. Systems that are able to achieve even more rapid times for treatment of patients with STEMI should be encouraged. Modified with permission from Armstrong et al. *Circulation*. 2003;107:2533–7.[25]

not undergoing angiography within the first 24 hours after fibrinolytic therapy. (*Level of Evidence: B*)

Class III

1. Serial biomarker measurements should not be relied on to diagnose reinfarction within the first 18 hours after the onset of STEMI. (*Level of Evidence: C*)

For patients with ST-segment elevation, the diagnosis of STEMI is secure; initiation of reperfusion therapy should not be delayed to wait for the results of a cardiac biomarker assay.[29] Quantitative analysis of cardiac biomarker measurements provides prognostic information and a noninvasive assessment of the likelihood that the patient has undergone successful reperfusion when fibrinolytic therapy is administered.

a. Bedside Testing for Serum Cardiac Biomarkers

Class I

1. Although handheld bedside (point-of-care) assays may be used for a qualitative assessment of the presence of an elevated level of a serum cardiac biomarker, subsequent measurements of cardiac biomarker levels should be performed with a quantitative test. (*Level of Evidence: B*)
2. For patients with ST elevation on the 12-lead ECG and symptoms of STEMI, reperfusion therapy should be initiated as soon as possible and is not contingent on a bedside biomarker assay. (*Level of Evidence: C*)

A positive bedside test should be confirmed by a conventional quantitative test. However, reperfusion therapy should not be delayed to wait for the results of a quantitative assay.

6. Imaging

Class I

1. Patients with STEMI should have a portable chest X-ray, but this should not delay implementation of reperfusion therapy (unless a potential contraindication, such as aortic dissection, is suspected). (*Level of Evidence: C*)
2. Imaging studies such as a high-quality portable chest X-ray, transthoracic and/or transesophageal echocardiography, and a contrast chest computed tomographic scan or a MRI scan should be used to differentiate STEMI from aortic dissection in patients for whom this distinction is initially unclear. (*Level of Evidence: B*)

Class IIa

1. Portable echocardiography is reasonable to clarify the diagnosis of STEMI and allow risk stratification of patients with chest pain on arrival at the ED, especially if the diagnosis of STEMI is confounded by left bundle-branch block (LBBB) or pacing, or there is suspicion of posterior STEMI with anterior ST depressions. (See Section 7.6.7 Mechanical Causes of Heart Failure/Low Output Syndrome of the full-text guidelines.) (*Level of Evidence: B*)

Class III

1. Single-photon emission computed tomography (SPECT) radionuclide imaging should not be performed to diagnose STEMI in patients for whom the diagnosis of STEMI is evident on the ECG. (*Level of Evidence: B*)

C. Management

1. Routine Measures

a. Oxygen

Class I

1. Supplemental oxygen should be administered to patients with arterial oxygen desaturation (SaO_2 less than 90%). (*Level of Evidence: B*)

Class IIa

1. It is reasonable to administer supplemental oxygen to all patients with uncomplicated STEMI during the first 6 hours. (*Level of Evidence: C*)

b. Nitroglycerin

Class I

1. Patients with ongoing ischemic discomfort should receive sublingual nitroglycerin (0.4 mg) every 5 minutes for a total of 3 doses, after which an assessment should be made about the need for intravenous nitroglycerin. (*Level of Evidence: C*)
2. Intravenous nitroglycerin is indicated for relief of ongoing ischemic discomfort, control of hypertension, or management of pulmonary congestion. (*Level of Evidence: C*)

Class III

1. Nitrates should not be administered to patients with systolic blood pressure less than 90 mm Hg or greater than or equal to 30 mm Hg below baseline, severe bradycardia (less than 50 bpm), tachycardia (more than 100 bpm), or suspected RV infarction. (*Level of Evidence: C*)
2. Nitrates should not be administered to patients who have received a phosphodiesterase inhibitor for erectile dysfunction within the last 24 hours (48 hours for tadalafil). (*Level of Evidence: B*)

Nitroglycerin may be administered to relieve ischemic pain and is clearly indicated as a vasodilator in patients with STEMI associated with left ventricular (LV) failure. Nitrates in all forms should be avoided in patients with initial systolic blood pressures less than 90 mm Hg or greater than or equal to 30 mm Hg below baseline, in patients with marked bradycardia or tachycardia,[30] and in patients with known or suspected RV infarction. In view of their marginal treatment benefits, nitrates should not be used if hypotension limits the administration of beta-blockers, which have more powerful salutary effects.

c. Analgesia

Class I

1. Morphine sulfate (2 to 4 mg IV with increments of 2 to 8 mg IV repeated at 5- to 15-minute intervals) is the

analgesic of choice for management of pain associated with STEMI. (*Level of Evidence: C*)

d. Aspirin

Class I

1. Aspirin should be chewed by patients who have not taken aspirin before presentation with STEMI. The initial dose should be 162 mg (*Level of Evidence: A*) to 325 mg *(Level of Evidence: C*). Although some trials have used enteric-coated aspirin for initial dosing, more rapid buccal absorption occurs with non–enteric-coated aspirin formulations.

In a dose of 162 mg or more, aspirin produces a rapid clinical antithrombotic effect caused by immediate and near-total inhibition of thromboxane A2 production. Aspirin now forms part of the early management of all patients with suspected STEMI and should be given promptly, and certainly within the first 24 hours, at a dose between 162 and 325 mg and continued indefinitely at a daily dose of 75 to 162 mg.[31] Although some trials have used enteric-coated aspirin for initial dosing, more rapid buccal absorption occurs with non–enteric-coated formulations.[32]

e. Beta-Blockers

Class I

1. Oral beta-blocker therapy should be administered promptly to those patients without a contraindication, irrespective of concomitant fibrinolytic therapy or performance of primary PCI. (*Level of Evidence: A*)

Class IIa

1. It is reasonable to administer IV beta-blockers promptly to STEMI patients without contraindications, especially if a tachyarrhythmia or hypertension is present. (*Level of Evidence: B*)

Immediate beta-blocker therapy appears to reduce the magnitude of infarction and incidence of associated complications in subjects not receiving concomitant fibrinolytic therapy, the rate of reinfarction in patients receiving fibrinolytic therapy, and the frequency of life-threatening ventricular tachyarrhythmias.

f. Reperfusion

GENERAL CONCEPTS.

Class I

1. All STEMI patients should undergo rapid evaluation for reperfusion therapy and have a reperfusion strategy implemented promptly after contact with the medical system. (*Level of Evidence: A*)

Evidence exists that expeditious restoration of flow in the obstructed infarct artery after the onset of symptoms in STEMI patients is a key determinant of short- and long-term outcomes regardless of whether reperfusion is accomplished by fibrinolysis or PCI.[33–35] As discussed previously (also see Section 4.1 of the full-text guidelines), efforts should be made to shorten the time from recognition of symptoms by the patient to contact with the medical system. All healthcare providers caring for STEMI patients from the point of entry into the medical system must recognize the need for rapid triage and implementation of care in a fashion analogous to the handling of trauma patients. When considering recommendations for timely reperfusion of STEMI patients, the Writing Committee reviewed data from clinical trials, focusing particular attention on enrollment criteria for selection of patients for randomization, actual times reported in the trial report rather than simply the allowable window specified in the trial protocol, treatment effect of the reperfusion strategy on individual components of a composite primary end point (eg, mortality, recurrent nonfatal infarction), ancillary therapies (eg, antithrombin and antiplatelet agents), and the interface between fibrinolysis and referral for angiography and revascularization. When available, data from registries were also reviewed to assess the generalizability of observations from clinical trials of reperfusion to routine practice. Despite the wealth of reports on reperfusion for STEMI, it is not possible to produce a simple algorithm, given the heterogeneity of patient profiles and availability of resources in various clinical settings at various times of day. This section introduces the recommendations for an aggressive attempt to minimize the time from entry into the medical system to implementation of a reperfusion strategy using the concept of medical system goals. More detailed discussion of these goals and the issues to be considered in selecting the type of reperfusion therapy are discussed in the Selection of Reperfusion Therapy section of VI.C.1.f (Section 6.3.1.6.2 of the full-text guidelines), followed by a discussion of available resources.

The medical system goal is to facilitate rapid recognition and treatment of patients with STEMI such that door-to-needle (or medical contact–to-needle) time for initiation of fibrinolytic therapy can be achieved within 30 minutes or that door-to-balloon (or medical contact–to-balloon) time for PCI can be kept under 90 minutes. These goals may not be relevant for the patients with an appropriate reason for delay, such as uncertainty about the diagnosis (particularly for the use of fibrinolytic therapy), need for the evaluation and treatment of other life-threatening conditions (eg, respiratory failure), or delays associated with the patient's informed choice to have more time to consider the decision. In the absence of such types of circumstances, the emphasis is on having a system in place such that when a patient with STEMI presents for medical care, reperfusion therapy is able to be provided as soon as possible within these time periods. Because there is not considered to be a threshold effect for the benefit of shorter times to reperfusion, these goals should not be understood as "ideal" times but the longest times that should be considered acceptable. Systems that are able to achieve even more rapid times for patients should be encouraged. Also, this goal should not be perceived as an average performance standard but a goal of an early treatment system that every hospital should seek for every appropriate patient.

SELECTION OF REPERFUSION STRATEGY. Several issues should be considered in selecting the type of reperfusion therapy:

- *Time From Onset of Symptoms*. Time from onset of symptoms to fibrinolytic therapy is an important predictor of MI size and patient outcome.[36] The efficacy of fibrinolytic agents in lysing thrombus diminishes with the passage of time.[37] Fibrinolytic therapy administered within the first 2 hours (especially the first hour) can occasionally abort MI and dramatically reduce mortality.[23,38]

  In contrast, the ability to produce a patent infarct artery is much less dependent on symptom duration in patients undergoing PCI. Several reports claim no influence of time

delay on mortality rates when PCI is performed after 2 to 3 hours of symptom duration.[39,40] Importantly, after adjustment for baseline characteristics, time from symptom onset to balloon inflation is significantly correlated with 1-year mortality in patients undergoing primary PCI for STEMI.[41] The Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology[42] and this Committee both recommend a target of medical contact–to-balloon or door-to-balloon time within 90 minutes.

- *Risk of STEMI*. Several models have been developed that assist clinicians in estimating the risk of mortality in patients with STEMI.[43–47] Although these models vary somewhat in the factors loaded into the risk-prediction tool and also vary with respect to statistical measures of their discriminative power (eg, C statistic), all the models provide clinicians with a means to assess the continuum of risk from STEMI. When the estimated mortality with fibrinolysis is extremely high, as is the case in patients with cardiogenic shock, compelling evidence exists that favors a PCI strategy.
- *Risk of Bleeding*. Choice of reperfusion therapy is also affected by the patient's risk of bleeding. When both types of reperfusion are available, the higher the patient's risk of bleeding with fibrinolytic therapy, the more strongly the decision should favor PCI. If PCI is unavailable, then the benefit of pharmacological reperfusion therapy should be balanced against the risk.
- *Time Required for Transport to a Skilled PCI Laboratory*. The availability of interventional cardiology facilities is a key determinant of whether PCI can be provided. For facilities that can offer PCI, the literature suggests that this approach is superior to pharmacological reperfusion.[48] The trials comparing pharmacological and PCI strategies, however, were conducted before the advent of more recent pharmacological and PCI strategies. When a composite end point of death, nonfatal recurrent MI, or stroke is analyzed, much of the superiority of a PCI strategy is driven by a reduction in the rate of nonfatal recurrent MI (Figure 2).[36] The rate of nonfatal recurrent MI can be influenced both by the adjunctive therapy used and by the proportion of patients who are referred for PCI when the initial attempt at fibrinolysis fails or myocardial ischemia recurs after initially successful pharmacological reperfusion.

The experience and location of the PCI laboratory also plays a role in the choice of therapy. Not all laboratories can provide prompt, high-quality primary PCI. Even centers with interventional cardiology facilities may not be able to provide the staffing required for 24-hour coverage of the catheterization laboratory. Despite staffing availability, the volume of cases in the laboratory may be insufficient for the team to acquire and maintain skills required for rapid PCI reperfusion strategies.

A decision must be made when a STEMI patient presents to a center without interventional cardiology facilities. Fibrinolytic therapy can generally be provided sooner than primary PCI. As the time delay for performing PCI increases, the mortality benefit associated with expeditiously performed primary PCI over fibrinolysis decreases.[49] Compared with a fibrin-specific lytic agent, a PCI strategy may not reduce mortality when a delay greater than 60 minutes is anticipated versus immediate administration of a lytic.

Given the current literature, it is not possible to say definitively that a particular reperfusion approach is superior for all patients, in all clinical settings, at all times of day (Danchin N; oral presentation at American Heart Association Scientific Sessions 2003, Orlando, FL, November 2003).[50–52] The main point is that some type of reperfusion therapy should be selected for all appropriate patients with suspected STEMI. The appropriate and timely use of some reperfusion therapy is likely more important than the choice of therapy, given the current literature and the expanding array of options. Clinical circumstances in which fibrinolytic therapy is generally preferred or an invasive strategy is generally preferred are shown in Figure 3.

Available Resources

Class I

1. STEMI patients presenting to a facility without the capability for expert, prompt intervention with primary PCI within 90 minutes of first medical contact should undergo fibrinolysis unless contraindicated. (*Level of Evidence: A*)

PHARMACOLOGICAL REPERFUSION.

Indications for Fibrinolytic Therapy

Class I

1. In the absence of contraindications, fibrinolytic therapy should be administered to STEMI patients with symptom onset within the prior 12 hours and ST elevation greater than 0.1 mV in at least 2 contiguous precordial leads or at least 2 adjacent limb leads. (*Level of Evidence: A*)
2. In the absence of contraindications, fibrinolytic therapy should be administered to STEMI patients with symptom onset within the prior 12 hours and new or presumably new LBBB. (*Level of Evidence: A*)

Class IIa

1. In the absence of contraindications, it is reasonable to administer fibrinolytic therapy to STEMI patients with symptom onset within the prior 12 hours and 12-lead ECG findings consistent with a true posterior MI. (*Level of Evidence: C*)
2. In the absence of contraindications, it is reasonable to administer fibrinolytic therapy to patients with symptoms of STEMI beginning within the prior 12 to 24 hours who have continuing ischemic symptoms and ST elevation greater than 0.1 mV in at least 2 contiguous precordial leads or at least 2 adjacent limb leads. (*Level of Evidence: B*)

Class III

1. Fibrinolytic therapy should not be administered to asymptomatic patients whose initial symptoms of STEMI began more than 24 hours earlier. (*Level of Evidence: C*)
2. Fibrinolytic therapy should not be administered to patients whose 12-lead ECG shows only ST-segment depression except if a true posterior MI is suspected. (*Level of Evidence: A*)

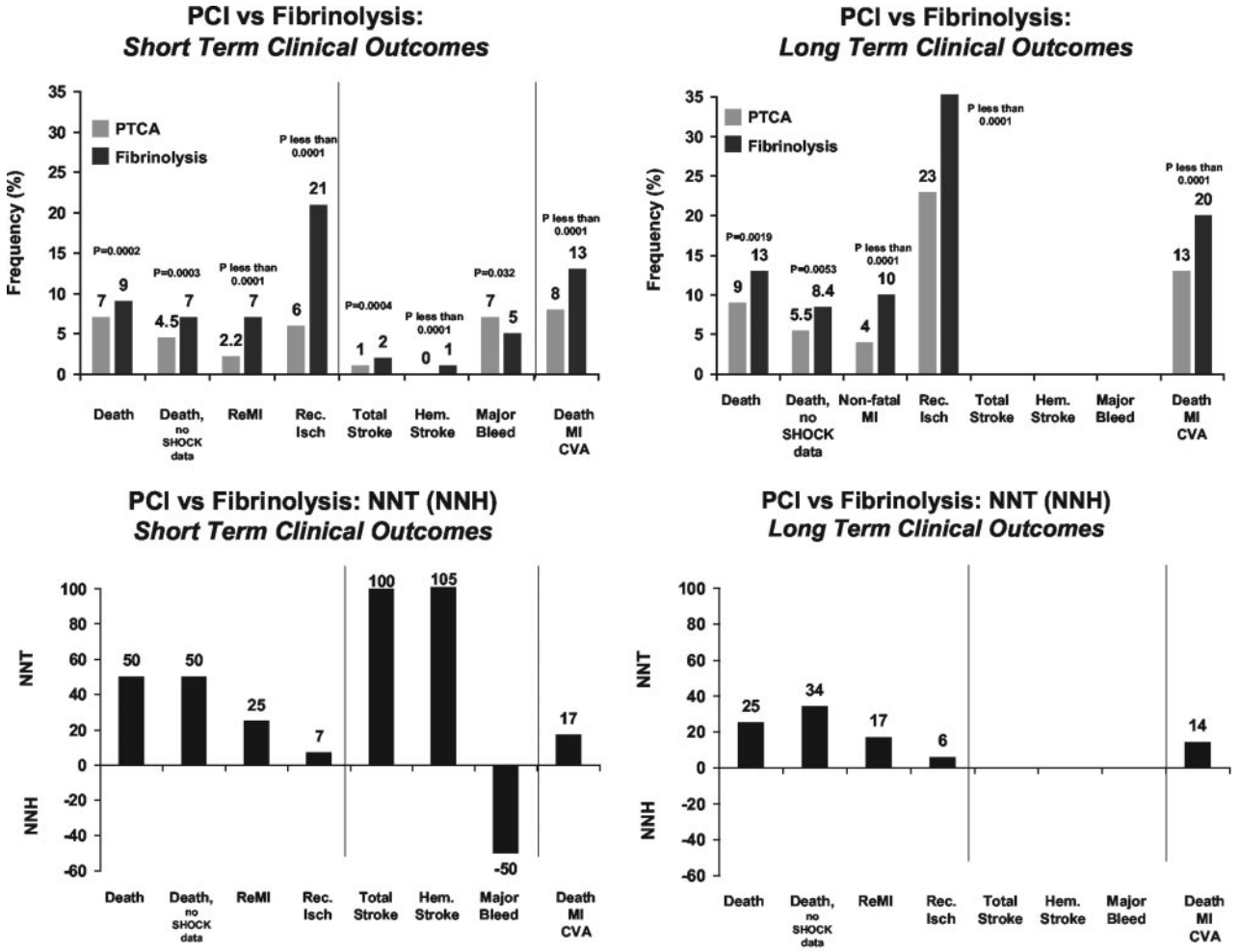

Figure 2. PCI vs fibrinolysis for STEMI. Short-term (4 to 6 weeks; top left) and long-term (top right) outcomes for various end points shown are plotted for STEMI patients randomized to PCI or fibrinolysis for reperfusion in 23 trials (n=7739). Given the frequency of events for each end point in the 2 treatment groups, the number needed to treat (NNT) or number needed to harm (NNH) is shown for the short-term (bottom left) and long-term (bottom right) outcomes. The magnitude of treatment differences for death, nonfatal reinfarction, and stroke varies depending on whether PCI is compared with streptokinase or a fibrin-specific lytic. For example, when primary PCI is compared with alteplase and the SHOCK trial is excluded, the mortality rate is 5.5% vs 6.7% (odds ratio 0.81, 95% confidence interval 0.64 to 1.03, $P=0.081$).[76a] See references 76 and 76a for additional discussion. Modified with permission from Elsevier (Keeley et al. The Lancet. 2003;361:13–20)[76] ReMI indicates recurrent MI; Rec. Isch, recurrent ischemia; Hem. Stroke, hemorrhagic stroke; and CVA, cerebrovascular accident.

Because the benefit of fibrinolytic therapy is directly related to the time from symptom onset, treatment benefit is maximized by the earliest possible application of therapy. The constellation of clinical features that must be present (although not necessarily at the same time) to serve as an indication for fibrinolysis includes symptoms of myocardial ischemia and ST elevation greater than 0.1 mV, in at least 2 contiguous leads, or new or presumably new LBBB on the presenting ECG.[23,54]

Contraindications/Cautions

Class I

1. **Healthcare providers should ascertain whether the patient has neurological contraindications to fibrinolytic therapy, including any history of intracranial hemorrhage (ICH), significant closed head or facial trauma within the past 3 months, uncontrolled hypertension, or ischemic stroke within the past 3 months. (See Table 2 for a comprehensive list.) (*Level of Evidence: A*)**
2. **STEMI patients at substantial (greater than or equal to 4%) risk of ICH should be treated with PCI rather than with fibrinolytic therapy. (See Figure 3 for further management considerations.) (*Level of Evidence: A*)**

A detailed list of contraindications and cautions for the use of fibrinolytic therapy is shown in Table 2.

Complications of Fibrinolytic Therapy: Neurological and Other

Class I

1. **The occurrence of a change in neurological status during or after reperfusion therapy, particularly within the first 24 hours after initiation of treatment, is**

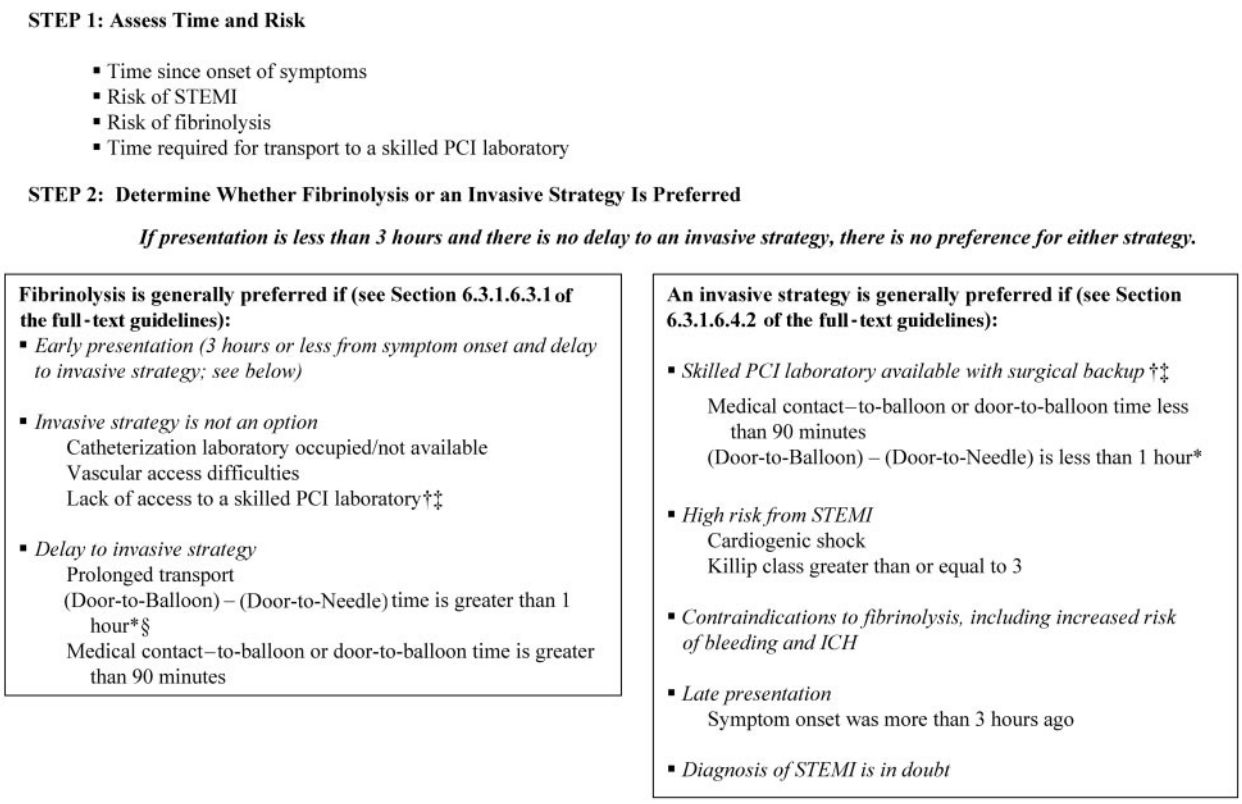

Figure 3. Assessment of reperfusion options for patients with STEMI. STEMI indicates ST-elevation myocardial infarction; PCI, percutaneous coronary intervention; ICH, intracranial hemorrhage. *Applies to fibrin-specific agents (see Figure 15 in the full-text STEMI guidelines). †Operator experience greater than a total of 75 primary PCI cases per year. ‡Team experience greater than a total of 36 primary PCI cases per year. §This calculation implies that the estimated delay to the implementation of the invasive strategy is greater than 1 hour vs initiation of fibrinolytic therapy immediately with a fibrin-specific agent.

considered to be due to ICH until proven otherwise. Fibrinolytic, antiplatelet, and anticoagulant therapies should be discontinued until brain imaging scan shows no evidence of ICH. (*Level of Evidence: A*)

2. **Neurology and/or neurosurgery or hematology consultations should be obtained for STEMI patients who have ICH as dictated by clinical circumstances. (*Level of Evidence: C*)**
3. **In patients with ICH, infusions of cryoprecipitate, fresh frozen plasma, protamine, and platelets should be given, as dictated by clinical circumstances. (*Level of Evidence: C*)**

Class IIa

1. **In patients with ICH, it is reasonable to:**
   a. **Optimize blood pressure and blood glucose levels. (*Level of Evidence: C*)**
   b. **Reduce intracranial pressure with an infusion of mannitol, endotracheal intubation, and hyperventilation. (*Level of Evidence: C*)**
   c. **Consider neurosurgical evacuation of ICH. (*Level of Evidence: C*)**

Combination Therapy With Glycoprotein IIb/IIIa Inhibitors

Class IIb

1. **Combination pharmacological reperfusion with abciximab and half-dose reteplase or tenecteplase may be considered for prevention of reinfarction (*Level of Evidence: A*) and other complications of STEMI in selected patients: anterior location of MI, age less than 75 years, and no risk factors for bleeding. In two clinical trials of combination reperfusion, the prevention of reinfarction did not translate into a survival benefit at either 30 days or 1 year.[54a] (*Level of Evidence: B*)**
2. **Combination pharmacological reperfusion with abciximab and half-dose reteplase or tenecteplase may be considered for prevention of reinfarction and other complications of STEMI in selected patients (anterior location of MI, age less than 75 years, and no risk factors for bleeding) in whom an early referral for angiography and PCI (ie, facilitated PCI) is planned. (*Level of Evidence: C*)**

Class III

1. **Combination pharmacological reperfusion with abciximab and half-dose reteplase or tenecteplase should not be given to patients aged greater than 75 years because of an increased risk of ICH. (*Level of Evidence: B*)**

PERCUTANEOUS CORONARY INTERVENTION

Coronary Angiography

Class I

1. **Diagnostic coronary angiography should be performed:**

TABLE 2. Contraindications and Cautions for Fibrinolysis Use in ST-Elevation Myocardial Infarction*

Absolute contraindications
• Any prior ICH
• Known structural cerebral vascular lesion (eg, AVM)
• Known malignant intracranial neoplasm (primary or metastatic)
• Ischemic stroke within 3 months EXCEPT acute ischemic stroke within 3 hours
• Suspected aortic dissection
• Active bleeding or bleeding diathesis (excluding menses)
• Significant closed head or facial trauma within 3 months
Relative contraindications
• History of chronic severe, poorly controlled hypertension
• Severe uncontrolled hypertension on presentation (SBP greater than 180 mm Hg or DBP greater than 110 mm Hg)†
• History of prior ischemic stroke greater than 3 months, dementia, or known intracranial pathology not covered in contraindications
• Traumatic or prolonged (greater than 10 minutes) CPR or major surgery (less than 3 weeks)
• Recent (within 2 to 4 weeks) internal bleeding
• Noncompressible vascular punctures
• For streptokinase/anistreplase: prior exposure (more than 5 days ago) or prior allergic reaction to these agents
• Pregnancy
• Active peptic ulcer
• Current use of anticoagulants: the higher the INR, the higher the risk of bleeding

AVM indicates arteriovenous malformation; SBP, systolic blood pressure; DBP, diastolic blood pressure; ICH, intracranial hemorrhage; CPR, cardiopulmonary resuscitation.

*Viewed as advisory for clinical decision making and may not be all-inclusive or definitive.

†Could be an absolute contraindication in low-risk patients with ST-elevation myocardial infarction (see Section 6.3.1.6.3.2 of the full-text guidelines).

a. In candidates for primary or rescue PCI. (*Level of Evidence: A*)

b. In patients with cardiogenic shock who are candidates for revascularization. (*Level of Evidence: A*)

c. In candidates for surgical repair of ventricular septal rupture or severe mitral regurgitation (MR). (*Level of Evidence: B*)

d. In patients with persistent hemodynamic and/or electrical instability. (*Level of Evidence: C*)

Class III

1. Coronary angiography should not be performed in patients with extensive comorbidities in whom the risks of revascularization are likely to outweigh the benefits. (*Level of Evidence: C*)

Primary PCI

Class I

1. General considerations: If immediately available, primary PCI should be performed in patients with STEMI (including true posterior MI) or MI with new or presumably new LBBB who can undergo PCI of the infarct artery within 12 hours of symptom onset, if performed in a timely fashion (balloon inflation within 90 minutes of presentation) by persons skilled in the procedure (individuals who perform more than 75 PCI procedures per year). The procedure should be supported by experienced personnel in an appropriate laboratory environment (performs more than 200 PCI procedures per year, of which at least 36 are primary PCI for STEMI, and has cardiac surgery capability). (*Level of Evidence: A*)

2. Specific considerations:

a. Primary PCI should be performed as quickly as possible, with a goal of a medical contact–to-balloon or door-to-balloon time of within 90 minutes. (*Level of Evidence: B*)

b. If the symptom duration is within 3 hours and the expected door-to-balloon time minus the expected door-to-needle time is:

i) within 1 hour, primary PCI is generally preferred. (*Level of Evidence: B*)

ii) greater than 1 hour, fibrinolytic therapy (fibrin-specific agents) is generally preferred. (*Level of Evidence: B*)

c. If symptom duration is greater than 3 hours, primary PCI is generally preferred and should be performed with a medical contact–to-balloon or door-to-balloon time as brief as possible, with a goal of within 90 minutes. (*Level of Evidence: B*)

d. Primary PCI should be performed for patients younger than 75 years old with ST elevation or LBBB who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock, unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: A*)

e. Primary PCI should be performed in patients with severe CHF and/or pulmonary edema (Killip class 3) and onset of symptoms within 12 hours. The medical contact–to-balloon or door-to-balloon time should be as short as possible (ie, goal within 90 min). (*Level of Evidence: B*)

Class IIa

1. Primary PCI is reasonable for selected patients 75 years or older with ST elevation or LBBB or who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock. Patients with good prior functional status who are suitable for revascularization and agree to invasive care may be selected for such an invasive strategy. (*Level of Evidence: B*)

2. It is reasonable to perform primary PCI for patients with onset of symptoms within the prior 12 to 24 hours and 1 or more of the following:

a. Severe CHF (*Level of Evidence: C*)

b. Hemodynamic or electrical instability (*Level of Evidence: C*)

c. Persistent ischemic symptoms. (*Level of Evidence: C*)

Class IIb

1. **The benefit of primary PCI for STEMI patients eligible for fibrinolysis is not well established when performed by an operator who performs fewer than 75 PCI procedures per year. (*Level of Evidence: C*)**

Class III

1. **PCI should not be performed in a noninfarct artery at the time of primary PCI in patients without hemodynamic compromise. (*Level of Evidence: C*)**
2. **Primary PCI should not be performed in asymptomatic patients more than 12 hours after onset of STEMI if they are hemodynamically and electrically stable. (*Level of Evidence: C*)**

Primary PCI has been compared with fibrinolytic therapy in 22 randomized clinical trials.[50,52,55–74] An additional trial, SHOCK (SHould we emergently revascularize Occluded Coronaries for cardiogenic shocK?),[75] that compared medical stabilization with immediate revascularization for cardiogenic shock was included along with the above 22 trials in an overview of primary PCI versus fibrinolysis.[76] These investigations demonstrate that PCI-treated patients experience lower short-term mortality rates, less nonfatal reinfarction, and less hemorrhagic stroke than those treated by fibrinolysis but have an increased risk for major bleeding.[76] These results have been achieved in medical centers with experienced providers and under circumstances in which PCI can be performed promptly after patient presentation (Figure 2).[76]

Additional considerations that affect the magnitude of the difference between PCI- and fibrinolysis-treated patients include the fact that unfractionated heparin (UFH) was used as the antithrombin with fibrinolytics (as opposed to other antithrombins such as enoxaparin [see Ancillary Therapy in Section VI.C.1.f and also Section 6.3.1.6.8.1.1 of the full-text guidelines] or bivalirudin [see Section 6.3.1.6.8.1.2 of the full-text guidelines] that are associated with a reduction in the rate of recurrent MI after fibrinolysis), a smaller but still statistically significant advantage for PCI compared with a fibrin-specific lytic versus streptokinase, and variation among the PCI arms as to whether a stent was implanted or glycoprotein (GP) IIb/IIIa antagonists were administered. Figure 2 shows the short- and long-term outcomes of patients with STEMI treated by fibrinolysis versus PCI and the number of patients who need to be treated to prevent 1 event or cause 1 harmful complication when selecting PCI instead of fibrinolysis as the reperfusion strategy (Figure 2).[76] Of note, when primary PCI is compared with tissue plasminogen activator (tPA) and the SHOCK trial is excluded, the mortality rate is 5.5% versus 6.7% (odds ratio 0.81%, 95% confidence interval [CI] 0.64 to 1.03, *P* equals 0.081.[76a]

There is serious and legitimate concern that a routine policy of primary PCI for patients with STEMI will result in unacceptable delays in achieving reperfusion in a substantial number of cases and produce less than optimal outcomes if performed by less-experienced operators. The mean time delay for PCI instead of fibrinolysis in the randomized studies was approximately 40 minutes.[76] Strict performance criteria must be mandated for primary PCI programs so that long door-to-balloon times and performance by low-volume or poor-outcome operators/laboratories do not occur. Interventional cardiologists and centers should strive for outcomes to include (1) medical contact–to-balloon or door-to-balloon times less than 90 minutes; (2) TIMI (Thrombolysis In Myocardial Infarction) 2/3 flow rates obtained in more than 90% of patients; (3) emergency CABG rate less than 2% among all patients undergoing the procedure; (4) actual performance of PCI in a high percentage of patients (85%) brought to the laboratory; and (5) risk-adjusted in-hospital mortality rate less than 7% in patients without cardiogenic shock. This would result in a risk-adjusted mortality rate with PCI comparable to that reported for fibrinolytic therapy in fibrinolytic-eligible patients[76] and would be consistent with previously reported registry experience.[77–80] Otherwise, the focus of treatment should be the early use of fibrinolytic therapy (Figure 2).[76]

PCI appears to have its greatest mortality benefit in high-risk patients. In patients with cardiogenic shock, an absolute 9% reduction in 30-day mortality with coronary revascularization instead of immediate medical stabilization was reported in the SHOCK trial.[75]

Time from symptom onset to reperfusion is an important predictor of patient outcome. Two studies[81,82] have reported increasing mortality rates with increasing door-to-balloon times. Other studies have shown smaller infarct size, better LV function, and fewer complications when reperfusion occurs before PCI.[83–85] An analysis of the randomized controlled trials comparing fibrinolysis with a fibrin-specific agent versus primary PCI suggests that the mortality benefit with PCI exists when treatment is delayed by no more than 60 minutes. Mortality increases significantly with each 15-minute delay in the time between arrival and restoration of TIMI-3 flow (door-to–TIMI-3 flow time), which further underscores the importance of timely reperfusion in patients who undergo primary PCI.[86] Importantly, after adjustment for baseline characteristics, time from symptom onset to balloon inflation is significantly correlated with 1-year mortality in patients undergoing primary PCI for STEMI (relative risk equals 1.08 for each 30-minute delay from symptom onset to balloon inflation; *P* equals 0.04).[35,41] Given that the medical contact–to-needle time goal within 30 minutes, this Writing Committee joins the Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology in lowering the medical contact–to-balloon or door-to-balloon time goal from within 120 minutes to within 90 minutes in an attempt to maximize the benefits for reperfusion by PCI.[42]

If the expected door-to-balloon time exceeds the expected door-to-needle time by more than 60 minutes, fibrinolytic treatment with a fibrin-specific agent should be considered unless it is contraindicated. This is particularly important when symptom duration is less than 3 hours but is less important with longer symptom duration, when less ischemic myocardium can be salvaged.

Primary PCI in fibrinolytic-ineligible patients

Class I

1. **Primary PCI should be performed in fibrinolytic-ineligible patients who present with STEMI within 12 hours of symptom onset. (*Level of Evidence: C*)**

Class IIa

1. **It is reasonable to perform primary PCI for fibrinolytic-ineligible patients with onset of symptoms within the prior 12 to 24 hours and 1 or more of the following:**
   a. **Severe CHF (*Level of Evidence: C*)**
   b. **Hemodynamic or electrical instability (*Level of Evidence: C*)**
   c. **Persistent ischemic symptoms. (*Level of Evidence: C*)**

Randomized controlled trials evaluating the outcome of PCI for patients who present with STEMI but who are ineligible for fibrinolytic therapy have not been performed. Few data are available to characterize the value of primary PCI for this subset of STEMI patients; however, the recommendations in Section IV.A (and Section 4.2 of the full-text guidelines) are applicable to these patients. Nevertheless, these patients are at increased risk for mortality,[87] and there is a general consensus that PCI is an appropriate means for achieving reperfusion in those who cannot receive fibrinolytics because of increased risk of bleeding.[88–91]

Primary PCI Without On-Site Cardiac Surgery

Class IIb

1. **Primary PCI might be considered in hospitals without on-site cardiac surgery, provided that there exists a proven plan for rapid transport to a cardiac surgery operating room in a nearby hospital with appropriate hemodynamic support capability for transfer. The procedure should be limited to patients with STEMI or MI with new, or presumably new, LBBB on ECG, and should be done in a timely fashion (balloon inflation within 90 minutes of presentation) by persons skilled in the procedure (at least 75 PCIs per year) and at hospitals that perform a minimum of 36 primary PCI procedures per year. (*Level of Evidence: B*)**

Class III

1. **Primary PCI should not be performed in hospitals without on-site cardiac surgery and without a proven plan for rapid transport to a cardiac surgery operating room in a nearby hospital or without appropriate hemodynamic support capability for transfer. (*Level of Evidence: C*)**

From clinical data and expert consensus, the Committee recommends that primary PCI for acute STEMI performed at hospitals without established elective PCI programs should be restricted to those institutions capable of performing a requisite minimum number of primary PCI procedures (36 per year) with a proven plan for rapid and effective PCI and rapid access to cardiac surgery in a nearby hospital. The benefit of primary PCI is not well established for operators who perform fewer than 75 PCIs per year or in a hospital that performs fewer than 36 primary PCI procedures per year. In addition, the benefit of timely reperfusion of the infarct artery by primary PCI at sites without on-site surgery must be weighed against the small but finite risk of harm to the patient related to the time required to transfer the patient to a site with CABG surgery capabilities.[92,93]

Interhospital Transfer for Primary PCI

To achieve optimal results, time from the first hospital door to the balloon inflation in the second hospital should be as short as possible, with a goal of within 90 minutes. Significant reductions in door-to-balloon times might be achieved by directly transporting patients to PCI centers rather than transporting them to the nearest hospital, if interhospital transfer will subsequently be required to obtain primary PCI.

Primary Stenting

Primary stenting has been compared with primary angioplasty in 9 studies.[94–103] There were no differences in mortality (3.0% versus 2.8%) or reinfarction (1.8% versus 2.1%) rates. However, major adverse cardiac events were reduced, driven by the reduction in subsequent target-vessel revascularization with stenting.

Preliminary reports suggest that compared with conventional bare metal stents, drug-eluting stents are not associated with increased risk when used for primary PCI in STEMI patients.[104] Postprocedure vessel patency, biomarker release, and the incidence of short-term adverse events were similar in patients receiving sirolimus (n equals 186) or bare metal (n equals 183) stents. Thirty-day event rates of death, reinfarction, or revascularization were 7.5% versus 10.4%, respectively (*P* equals 0.4).[104]

Facilitated PCI

Class IIb

1. **Facilitated PCI might be performed as a reperfusion strategy in higher-risk patients when PCI is not immediately available and bleeding risk is low. (*Level of Evidence: B*)**

Facilitated PCI refers to a strategy of planned immediate PCI after an initial pharmacological regimen such as full-dose fibrinolysis, half-dose fibrinolysis, a GP IIb/IIIa inhibitor, or a combination of reduced-dose fibrinolytic therapy and a platelet GP IIb/IIIa inhibitor. A strategy of facilitated PCI holds promise in higher-risk patients when PCI is not immediately available. Potential risks include increased bleeding complications, especially in patients who are at least 75 years of age (see Pharmacological Reperfusion in Section VI.C.1.f and Section 6.3.1.6.3.8. of the full-text guidelines), and potential limitations include added cost. Several randomized trials of facilitated PCI with a variety of pharmacological regimens are in progress.

Rescue PCI

Class I

1. **Rescue PCI should be performed in patients less than 75 years old with ST elevation or LBBB who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock, unless further support is futile because**

of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: B*)
2. Rescue PCI should be performed in patients with severe CHF and/or pulmonary edema (Killip class 3) and onset of symptoms within 12 hours. (*Level of Evidence: B*)

Class IIa

1. Rescue PCI is reasonable for selected patients 75 years or older with ST elevation or LBBB or who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock. Patients with good prior functional status who are suitable for revascularization and agree to invasive care may be selected for such an invasive strategy. (*Level of Evidence: B*)
2. It is reasonable to perform rescue PCI for patients with 1 or more of the following:
a. Hemodynamic or electrical instability (*Level of Evidence: C*)
b. Persistent ischemic symptoms. (*Level of Evidence: C*)

Rescue PCI refers to PCI within 12 hours after failed fibrinolysis for patients with continuing or recurrent myocardial ischemia.

A major problem in adopting a strategy of rescue PCI lies in the limitation of accurate identification of patients for whom fibrinolytic therapy has not restored antegrade coronary flow. In a prior era in which the practice of PCI was less mature, immediate catheterization of all patients after fibrinolytic therapy to identify those with an occluded infarct artery was found to be impractical, costly, and often associated with bleeding complications.[105,106] This strategy is being reevaluated in clinical trials testing facilitated PCI in the contemporary PCI setting.

There are no convincing data to support the routine use of late adjuvant PCI days after failed fibrinolysis or for patients who do not receive reperfusion therapy. Nevertheless, this is being done in some STEMI patients as an extension of the invasive strategy for non-STEMI patients. The Occluded Artery Trial (OAT) is currently randomizing patients to test whether routine PCI days to weeks after MI improves long-term clinical outcomes in asymptomatic high-risk patients with an occluded infarct artery.[107]

PCI for Cardiogenic Shock

Class I

1. Primary PCI is recommended for patients less than 75 years old with ST elevation or LBBB who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock, unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: A*)

Class IIa

1. Primary PCI is reasonable for selected patients aged 75 years or older with ST elevation or LBBB who develop shock within 36 hours of MI and who are suitable for revascularization that can be performed within 18 hours of shock. Patients with good prior functional status who are suitable for revascularization and agree to invasive care may be selected for such an invasive strategy. (*Level of Evidence: B*)

Observational studies support the value of PCI for patients who develop cardiogenic shock in the early hours of STEMI. In the SHOCK trial,[75] the survival curves continued to progressively diverge such that at 6 months and 1 year, there was a significant mortality reduction with emergency revascularization (53% versus 66%, *P* less than 0.03).[108] The prespecified subgroup analysis of patients less than 75 years old showed an absolute 15% reduction in 30-day mortality (*P* less than 0.02), whereas there was no apparent benefit for the small cohort (n equals 56) of patients more than 75 years old. These data strongly support the approach that patients younger than 75 years with STEMI complicated by cardiogenic shock should undergo emergency revascularization and support measures. Three registries[109–111] have demonstrated a marked survival benefit for elderly patients who are clinically selected for revascularization (approximately 1 of 5 patients), so age alone should not disqualify a patient from early revascularization. (See Section VII.F.5 and also Section 7.6.5 of the full-text guidelines.)

Percutaneous Coronary Intervention After Fibrinolysis

Class I

1. In patients whose anatomy is suitable, PCI should be performed when there is objective evidence of recurrent MI. (*Level of Evidence: C*)
2. In patients whose anatomy is suitable, PCI should be performed for moderate or severe spontaneous or provocable myocardial ischemia during recovery from STEMI. (*Level of Evidence: B*)
3. In patients whose anatomy is suitable, PCI should be performed for cardiogenic shock or hemodynamic instability. (See section on PCI for Cardiogenic Shock in Section VI.C.1.f.) (*Level of Evidence: B*)

Class IIa

1. It is reasonable to perform routine PCI in patients with LV ejection fraction (LVEF) less than or equal to 0.40, CHF, or serious ventricular arrhythmias. (*Level of Evidence: C*)
2. It is reasonable to perform PCI when there is documented clinical heart failure during the acute episode, even though subsequent evaluation shows preserved LV function (LVEF greater than 0.40). (*Level of Evidence: C*)

Class IIb

1. Routine PCI might be considered as part of an invasive strategy after fibrinolytic therapy. (*Level of Evidence: B*)

Immediately After Successful Fibrinolysis. Randomized prospective trials examined the efficacy and safety of immediate PCI after fibrinolysis.[105,106,112] These trials showed no benefit of routine PCI of the stenotic infarct artery immediately after fibrinolytic therapy. The strategy did not appear to salvage myocardium, improve LVEF, or prevent reinfarction

or death. Those subjected to this approach appeared to have an increased incidence of adverse events, including bleeding, recurrent ischemia, emergency CABG, and death. These studies have not been repeated in the modern interventional era with improved equipment, improved antiplatelet and anticoagulant strategies, and coronary stents, thus leaving the question of routine PCI early after successful fibrinolysis unresolved in contemporary practice. Studies of facilitated PCI are enrolling patients.[113–116]

Hours to Days After Successful Fibrinolysis. Great improvements in equipment, operator experience, and adjunctive pharmacotherapy have increased PCI success rates and decreased complications. More recently, the invasive strategy for NSTEMI patients has been given a Class I recommendation by the ACC/AHA Guidelines for the Management of Patients With Unstable Angina/Non-STEMI.[117] STEMI patients are increasingly being treated similarly as an extension of this approach. Although 6 published reports[115,118–121,123] and 1 preliminary report[122] support this strategy, randomized studies similar to those in NSTEMI need to be performed.

Acute Surgical Reperfusion

Class I

1. Emergency or urgent CABG in patients with STEMI should be undertaken in the following circumstances:

- **a. Failed PCI with persistent pain or hemodynamic instability in patients with coronary anatomy suitable for surgery. (*Level of Evidence: B*)**
- **b. Persistent or recurrent ischemia refractory to medical therapy in patients who have coronary anatomy suitable for surgery, have a significant area of myocardium at risk, and are not candidates for PCI or fibrinolytic therapy. (*Level of Evidence: B*)**
- **c. At the time of surgical repair of postinfarction ventricular septal rupture (VSR) or mitral valve insufficiency. (*Level of Evidence: B*)**
- **d. Cardiogenic shock in patients less than 75 years old with ST elevation, LBBB, or posterior MI who develop shock within 36 hours of STEMI, have severe multivessel or left main disease, and are suitable for revascularization that can be performed within 18 hours of shock, unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: A*)**
- **e. Life-threatening ventricular arrhythmias in the presence of greater than or equal to 50% left main stenosis and/or triple-vessel disease. (*Level of Evidence: B*)**

Class IIa

1. Emergency CABG can be useful as the primary reperfusion strategy in patients who have suitable anatomy, who are not candidates for fibrinolysis or PCI, and who are in the early hours (6 to 12 hours) of an evolving STEMI, especially if severe multivessel or left main disease is present. (*Level of Evidence: B*)

2. Emergency CABG can be effective in selected patients 75 years or older with ST elevation, LBBB, or posterior MI who develop shock within 36 hours of STEMI, have severe triple-vessel or left main disease, and are suitable for revascularization that can be performed within 18 hours of shock. Patients with good prior functional status who are suitable for revascularization and agree to invasive care may be selected for such an invasive strategy. (*Level of Evidence: B*)

Class III

1. Emergency CABG should not be performed in patients with persistent angina and a small area of risk if they are hemodynamically stable. (*Level of Evidence: C*)

2. Emergency CABG should not be performed in patients with successful epicardial reperfusion but unsuccessful microvascular reperfusion. (*Level of Evidence: C*)

Patients With STEMI Not Receiving Reperfusion

Guideline-based recommendations for nonreperfusion treatments should not vary whether or not patients received reperfusion therapy. The major difference is that patients not receiving reperfusion therapy are considered to have a higher risk for future adverse events.[124]

Assessment of Reperfusion

Class IIa

1. It is reasonable to monitor the pattern of ST elevation, cardiac rhythm, and clinical symptoms over the 60 to 180 minutes after initiation of fibrinolytic therapy. Noninvasive findings suggestive of reperfusion include relief of symptoms, maintenance or restoration of hemodynamic and or electrical stability, and a reduction of at least 50% of the initial ST-segment elevation injury pattern on a follow-up ECG 60 to 90 minutes after initiation of therapy. (*Level of Evidence: B*)

Persistence of unrelenting ischemic chest pain, absence of resolution of the qualifying ST-segment elevation, and hemodynamic and/or electrical instability are generally indicators of failed pharmacological reperfusion and the need to consider rescue PCI. Aggressive medical support may be necessary in the interim. (See Rescue PCI in Section in VI.C.I.f.)

Ancillary Therapy

Antithrombins as Ancillary Therapy to Reperfusion Therapy

Unfractionated heparin as ancillary therapy to reperfusion therapy

Class I

1. Patients undergoing percutaneous or surgical revascularization should be given UFH. (*Level of Evidence: C*)

2. UFH should be given intravenously to patients undergoing reperfusion therapy with alteplase, reteplase, or tenecteplase, with dosing as follows: bolus of 60 U/kg

(maximum 4000 U) followed by an initial infusion of 12 U/kg per hour (maximum 1000 U/hr) adjusted to maintain activated partial thromboplastin time (aPTT) at 1.5 to 2.0 times control (approximately 50 to 70 seconds). (*Level of Evidence: C*)

3. **UFH should be given intravenously to patients treated with nonselective fibrinolytic agents (streptokinase, anistreplase, or urokinase) who are at high risk for systemic emboli (large or anterior MI, atrial fibrillation, previous embolus, or known LV thrombus). (*Level of Evidence: B*)**
4. **Platelet counts should be monitored daily in patients given UFH. (*Level of Evidence: C*)**

Class IIb

1. **It may be reasonable to administer UFH intravenously to patients undergoing reperfusion therapy with streptokinase. (*Level of Evidence: B*)**

Because of the evidence that the measured effect of UFH on the aPTT is important for patient outcome and that the predominant variable mediating the effect of a given dose of heparin is weight,[125] it is important to administer the initial doses of UFH as a weight-adjusted bolus.[126] For fibrin-specific (alteplase, reteplase, and tenecteplase) fibrinolytic-treated patients, a 60 U/kg bolus followed by a maintenance infusion of 12 U/kg per hour (with a maximum of 4000 U bolus and 1000 U/h initial infusion for patients weighing greater than 70 kg) is recommended. The recommended weight-adjusted dose of UFH, when it is administered without fibrinolytics, is 60 to 70 U/kg IV bolus and 12 to 15 U/kg per hour infusion.[117]

Low-Molecular-Weight Heparin as Ancillary Therapy to Reperfusion Therapy

Class IIb

1. **LMWH might be considered an acceptable alternative to UFH as ancillary therapy for patients less than 75 years of age who are receiving fibrinolytic therapy, provided that significant renal dysfunction (serum creatinine greater than 2.5 mg/dL in men or 2.0 mg/dL in women) is not present. Enoxaparin (30 mg IV bolus followed by 1.0 mg/kg subcutaneous injection every 12 hours until hospital discharge) used in combination with full-dose tenecteplase is the most comprehensively studied regimen in patients less than 75 years of age. (*Level of Evidence: B*)**

Class III

1. **LMWH should not be used as an alternative to UFH as ancillary therapy in patients over 75 years of age who are receiving fibrinolytic therapy. (*Level of Evidence: B*)**
2. **LMWH should not be used as an alternative to UFH as ancillary therapy in patients less than 75 years of age who are receiving fibrinolytic therapy but have significant renal dysfunction (serum creatinine greater than 2.5 mg/dL in men or 2.0 mg/dL in women). (*Level of Evidence: B*)**

The available data suggest that the rate of early (60 to 90 minutes) reperfusion of the infarct artery, either assessed angiographically or by noninvasive means, is not enhanced by administration of an LMWH. However, a generally consistent theme of a lower rate of reocclusion of the infarct artery, reinfarction, or recurrent ischemic events emerges in patients receiving LMWH regardless of whether the control group was given placebo or UFH.

Direct Antithrombins as Ancillary Therapy to Reperfusion Therapy

Class IIa

1. **In patients with known heparin-induced thrombocytopenia, it is reasonable to consider bivalirudin as a useful alternative to heparin to be used in conjunction with streptokinase. Dosing according to the HERO (Hirulog and Early Reperfusion or Occlusion)-2 regimen (a bolus of 0.25 mg/kg followed by an intravenous infusion of 0.5 mg/kg per hour for the first 12 hours and 0.25 mg/kg per hour for the subsequent 36 hours)[127] is recommended but with a reduction in the infusion rate if the PTT is above 75 seconds within the first 12 hours. (*Level of Evidence: B*)**

On the basis of the data in the HERO-2 trial, the Writing Committee believed that bivalirudin could be considered an acceptable alternative to UFH in those STEMI patients who receive fibrinolysis with streptokinase, have heparin-induced thrombocytopenia, and who, in the opinion of the treating physician, would benefit from anticoagulation.

Antiplatelets

Aspirin

Class I

1. **A daily dose of aspirin (initial dose of 162 to 325 mg orally; maintenance dose of 75 to 162 mg) should be given indefinitely after STEMI to all patients without a true aspirin allergy. (*Level of Evidence: A*)**

As discussed, aspirin should be given to the patient with suspected STEMI as early as possible and should be continued indefinitely, regardless of the strategy for reperfusion and regardless of whether additional antiplatelet agents are administered. True aspirin allergy is the only exception to this recommendation.

Thienopyridines

Class I

1. **In patients who have undergone diagnostic cardiac catheterization and for whom PCI is planned, clopidogrel should be started and continued for at least 1 month after bare metal stent implantation, for several months after drug-eluting stent implantation (3 months for sirolimus, 6 months for paclitaxel), and for up to 12 months in patients who are not at high risk for bleeding. (*Level of Evidence: B*)**
2. **In patients taking clopidogrel in whom CABG is planned, the drug should be withheld for at least 5 days, and preferably for 7, unless the urgency for**

revascularization outweighs the risks of excess bleeding. (*Level of Evidence: B*)

Class IIa

1. Clopidogrel is probably indicated in patients receiving fibrinolytic therapy who are unable to take aspirin because of hypersensitivity or major gastrointestinal intolerance. (*Level of Evidence: C*)

Clopidogrel combined with aspirin is recommended for STEMI patients who undergo coronary stent implantation.[128–132] There are no safety data available regarding the combination of fibrinolytic agents and clopidogrel, but ongoing trials will provide this information in the future. However, in patients in whom aspirin is contraindicated because of aspirin sensitivity, clopidogrel is probably useful as a substitute for aspirin to reduce the risk of occlusion.[133] There are no safety data comparing 300 and 600 mg as loading doses for clopidogrel. We do not recommend routine administration of clopidogrel as pretreatment in patients who have not yet undergone diagnostic cardiac catheterization and in whom CABG surgery would be performed within 5 to 7 days if warranted.[134]

Glycoprotein IIb/IIIa inhibitors

Class IIa

1. It is reasonable to start treatment with abciximab as early as possible before primary PCI (with or without stenting) in patients with STEMI. (*Level of Evidence: B*)

Class IIb

1. Treatment with tirofiban or eptifibatide may be considered before primary PCI (with or without stenting) in patients with STEMI. (*Level of Evidence: C*)

The Writing Committee believes that it is reasonable to start treatment with abciximab as early as possible in patients undergoing primary PCI (with or without stenting) but, given the size and limitations of the available data set, assigned a Class IIa recommendation to this treatment. The data on tirofiban and eptifibatide in primary PCI are far more limited than for abciximab. However, given the common mode of action of the agents, a modest amount of angiographic data,[135] and general clinical experience to date, tirofiban or eptifibatide may be useful as antiplatelet therapy to support primary PCI for STEMI (with or without stenting) (Class IIb recommendation).

Other Pharmacological Measures

Inhibition of Renin-Angiotensin-Aldosterone System

Class I

1. An angiotensin converting enzyme (ACE) inhibitor should be administered orally within the first 24 hours of STEMI to patients with anterior infarction, pulmonary congestion, or LVEF less than 0.40, in the absence of hypotension (systolic blood pressure less than 100 mm Hg or less than 30 mm Hg below baseline) or known contraindications to that Class of medications. (*Level of Evidence: A*)

2. An angiotensin receptor blocker (ARB) should be administered to STEMI patients who are intolerant of ACE inhibitors and who have either clinical or radiological signs of heart failure or LVEF less than 0.40. Valsartan and candesartan have established efficacy for this recommendation. (*Level of Evidence: C*)

Class IIa

1. An ACE inhibitor administered orally within the first 24 hours of STEMI can be useful in patients without anterior infarction, pulmonary congestion, or LVEF less than 0.40 in the absence of hypotension (systolic blood pressure less than 100 mm Hg or less than 30 mm Hg below baseline) or known contraindications to that class of medications. The expected treatment benefit in such patients is less (5 lives saved per 1000 patients treated) than for patients with LV dysfunction. (*Level of Evidence: B*)

Class III

1. An intravenous ACE inhibitor should not be given to patients within the first 24 hours of STEMI because of the risk of hypotension. (A possible exception may be patients with refractory hypertension.) (*Level of Evidence: B*)

A number of large, randomized clinical trials have assessed the role of ACE inhibitors early in the course of acute MI. All trials with oral ACE inhibitors have shown benefit from their early use, including those in which early entry criteria included clinical suspicion of acute infarctions. Data from these trials indicate that ACE inhibitors should generally be started within the first 24 hours, ideally after fibrinolytic therapy has been completed and blood pressure has stabilized. ACE inhibitors should not be used if systolic blood pressure is less than 100 mm Hg or less than 30 mm Hg below baseline, if clinically relevant renal failure is present, if there is a history of bilateral stenosis of the renal arteries, or if there is known allergy to ACE inhibitors.

The use of ARBs has not been explored as thoroughly as ACE inhibitors in STEMI patients. However, clinical experience in the management of patients with heart failure and data from clinical trials in STEMI patients (see Sections 7.4.3 and 7.6.4 of the full-text guidelines) suggest that ARBs may be useful in patients with depressed LV function or clinical heart failure but who are intolerant of an ACE inhibitor. Use of aldosterone antagonists in STEMI patients is discussed in Sections 7.4.3 and 7.6.4 of the full-text guidelines.

Metabolic Modulation of the Glucose-Insulin Axis

Strict Glucose Control During STEMI

Class I

1. An insulin infusion to normalize blood glucose is recommended for patients with STEMI and complicated courses. (*Level of Evidence: B*)

Class IIa

1. **During the acute phase (first 24 to 48 hours) of the management of STEMI in patients with hyperglycemia, it is reasonable to administer an insulin infusion to normalize blood glucose, even in patients with an uncomplicated course. (*Level of Evidence: B*)**
2. **After the acute phase of STEMI, it is reasonable to individualize treatment of diabetics, selecting from a combination of insulin, insulin analogs, and oral hypoglycemic agents that achieve the best glycemic control and are well tolerated. (*Level of Evidence: C*)**

Compelling evidence for tight glucose control in patients in the intensive care unit (a large proportion of whom were there after cardiac surgery) supports the importance of intensive insulin therapy to achieve a normal blood glucose level in critically ill patients.[136,136a]

Magnesium

Class IIa

1. **It is reasonable that documented magnesium deficits be corrected, especially in patients receiving diuretics before the onset of STEMI. (*Level of Evidence: C*)**
2. **It is reasonable that episodes of torsade de pointes-type ventricular tachycardia (VT) associated with a prolonged QT interval be treated with 1 to 2 g of magnesium administered as an intravenous bolus over 5 minutes. (*Level of Evidence: C*)**

Class III

1. **In the absence of documented electrolyte deficits or torsade de pointes-type VT, routine intravenous magnesium should not be administered to STEMI patients at any level of risk. (*Level of Evidence: A*)**

Calcium Channel Blockers

Class IIa

1. **It is reasonable to give verapamil or diltiazem to patients in whom beta-blockers are ineffective or contraindicated (eg, bronchospastic disease) for relief of ongoing ischemia or control of a rapid ventricular response with atrial fibrillation or flutter after STEMI in the absence of CHF, LV dysfunction, or atrioventricular (AV) block. (*Level of Evidence: C*)**

Class III

1. **Diltiazem and verapamil are contraindicated in patients with STEMI and associated systolic LV dysfunction and CHF. (*Level of Evidence: A*)**
2. **Nifedipine (immediate-release form) is contraindicated in treatment of STEMI because of the reflex sympathetic activation, tachycardia, and hypotension associated with its use. (*Level of Evidence: B*)**

See the full-text guidelines for further explanation.

VII. Hospital Management

A. Location

1. Coronary Care Unit

Class I

1. **STEMI patients should be admitted to a quiet and comfortable environment that provides for continuous monitoring of the ECG and pulse oximetry and has ready access to facilities for hemodynamic monitoring and defibrillation. (*Level of Evidence: C*)**
2. **The patient's medication regimen should be reviewed to confirm the administration of aspirin and beta-blockers in an adequate dose to control heart rate and to assess the need for intravenous nitroglycerin for control of angina, hypertension, or heart failure. (*Level of Evidence: A*)**
3. **The ongoing need for supplemental oxygen should be assessed by monitoring arterial oxygen saturation. When stable for 6 hours, the patient should be reassessed for oxygen need (ie, O_2 saturation of less than 90%), and discontinuation of supplemental oxygen should be considered. (*Level of Evidence: C*)**
4. **Nursing care should be provided by individuals certified in critical care, with staffing based on the specific needs of patients and provider competencies, as well as organizational priorities. (*Level of Evidence: C*)**
5. **Care of STEMI patients in the critical care unit (CCU) should be structured around protocols derived from practice guidelines. (*Level of Evidence: C*)**
6. **Electrocardiographic monitoring leads should be based on the location and rhythm to optimize detection of ST deviation, axis shift, conduction defects, and dysrhythmias. (*Level of Evidence: B*)**

Class III

1. **It is not an effective use of the CCU environment to admit terminally ill, "do not resuscitate" patients with STEMI, because clinical and comfort needs can be provided outside of a critical care environment. (*Level of Evidence: C*)**

2. Stepdown Unit

Class I

1. **It is a useful triage strategy to admit low-risk STEMI patients who have undergone successful PCI directly to the stepdown unit for post-PCI care rather than to the CCU. (*Level of Evidence: C*)**
2. **STEMI patients originally admitted to the CCU who demonstrate 12 to 24 hours of clinical stability (absence of recurrent ischemia, heart failure, or hemodynamically compromising dysrhythmias) should be transferred to the stepdown unit. (*Level of Evidence: C*)**

Class IIa

1. **It is reasonable for patients recovering from STEMI who have clinically symptomatic heart failure to be managed on the stepdown unit, provided that facilities for continuous monitoring of pulse oximetry and appropriately skilled nurses are available. (*Level of Evidence: C*)**

2. **It is reasonable for patients recovering from STEMI who have arrhythmias that are hemodynamically well-tolerated (eg, atrial fibrillation with a controlled ventricular response; paroxysms of nonsustained VT lasting less than 30 seconds) to be managed on the stepdown unit, provided that facilities for continuous monitoring of the ECG, defibrillators, and appropriately skilled nurses are available. (*Level of Evidence: C*)**

Class IIb

1. **Patients recovering from STEMI who have clinically significant pulmonary disease requiring high-flow supplemental oxygen or noninvasive mask ventilation/bilevel positive airway pressure (BIPAP)/continuous positive airway pressure (CPAP) may be considered for care on a stepdown unit provided that facilities for continuous monitoring of pulse oximetry and appropriately skilled nurses with a sufficient nurse:patient ratio are available. (*Level of Evidence: C*)**

B. Early, General Measures

1. Level of Activity

Class IIa

1. **After 12 to 24 hours, it is reasonable to allow patients with hemodynamic instability or continued ischemia to have bedside commode privileges. (*Level of Evidence: C*)**

Class III

1. **Patients with STEMI who are free of recurrent ischemic discomfort, symptoms of heart failure, or serious disturbances of heart rhythm should not be on bed rest for more than 12 to 24 hours. (*Level of Evidence: C*)**

2. Diet

Class I

1. **Patients with STEMI should be prescribed the NCEP Adult Treatment Panel III (ATP III) Therapeutic Lifestyle Changes (TLC) diet, which focuses on reduced intake of fats and cholesterol, less than 7% of total calories as saturated fats, less than 200 mg of cholesterol per day, increased consumption of omega-3 fatty acids, and appropriate caloric intake for energy needs. (*Level of Evidence: C*)**
2. **Diabetic patients with STEMI should have an appropriate food group balance and caloric intake. (*Level of Evidence: B*)**
3. **Sodium intake should be restricted in STEMI patients with hypertension or heart failure. (*Level of Evidence: B*)**

STEMI patients should receive a reduced saturated fat and cholesterol diet per the ATP III TLC approach.[137] (See VII.L.2 and Section 7.12.2 of the full-text guidelines.)

3. Patient Education in the Hospital Setting

Class I

1. **Patient counseling to maximize adherence to evidence-based post-STEMI treatments (eg, compliance with taking medication, exercise prescription, and smoking cessation) should begin during the early phase of hospitalization, occur intensively at discharge, and continue at follow-up visits with providers and through cardiac rehabilitation programs and community support groups, as appropriate. (*Level of Evidence: C*)**
2. **Critical pathways and protocols and other quality-improvement tools (eg, the ACC "Guidelines Applied in Practice" and the AHA's "Get with the Guidelines") should be used to improve the application of evidence-based treatments by patients with STEMI, caregivers, and institutions. (*Level of Evidence: C*)**

Patient education should be viewed as a continuous process that should to be part of every patient encounter (ie, on hospital arrival, inpatient admission, discharge, and at follow-up visits).

4. Analgesia/Anxiolytics

Class IIa

1. **It is reasonable to use anxiolytic medications in STEMI patients to alleviate short-term anxiety or altered behavior related to hospitalization for STEMI. (*Level of Evidence: C*)**
2. **It is reasonable to routinely assess the patient's anxiety level and manage it with behavioral interventions and referral for counseling. (*Level of Evidence: C*)**

Anxiety and depression are prevalent in patients hospitalized for STEMI because patients are confronted with a diagnosis that is major, both psychologically and physically.[138,139] Anxiety has been demonstrated to predict in-hospital recurrent ischemia and arrhythmias[140] and cardiac events during the first year after an MI.[141]

C. Risk Stratification During Early Hospital Course

Risk stratification is a continuous process and requires the updating of initial assessments with data obtained during the hospital stay. Indicators of failed reperfusion (eg, recurrence of chest pain and persistence of ECG findings indicating infarction) identify a patient who should undergo coronary angiography. Similarly, findings consistent with mechanical complications (eg, sudden onset of heart failure or presence of a new murmur) herald increased risk and suggest the need for rapid intervention. For patients who did not undergo primary reperfusion, changes in clinical status (eg, development of shock) may herald a worsening clinical status and are an indication for coronary angiography. Patients with a low risk of complications may be candidates for early discharge. The lowest-risk patients are those who did not have STEMI despite the initial suspicions. Clinicians should strive to identify such patients within 8 to 12 hours of onset of symptoms. Serial sampling of serum cardiac biomarkers and use of 12-lead ECGs and their interpretation in the context of the number of hours that have elapsed since onset of the patient's symptoms can determine the presence of STEMI better than adherence to a rigid protocol that requires that a specified number of samples be drawn in the hospital.

D. Medication Assessment

1. Beta-Blockers

Class I

1. **Patients receiving beta-blockers within the first 24 hours of STEMI without adverse effects should continue to receive them during the early convalescent phase of STEMI. (*Level of Evidence: A*)**
2. **Patients without contraindications to beta-blockers who did not receive them within the first 24 hours after STEMI should have them started in the early convalescent phase. (*Level of Evidence: A*)**
3. **Patients with early contraindications within the first 24 hours of STEMI should be reevaluated for candidacy for beta-blocker therapy. (*Level of Evidence: C*)**

There is overwhelming evidence for the benefits of early beta-blockade in patients with STEMI and without contraindications to their use (see Section 6.3.1.5 of the full-text guidelines). Benefits have been demonstrated for patients with and without concomitant fibrinolytic therapy, both early and late after STEMI. Meta-analysis of trials from the prefibrinolytic era involving more than 24 000 patients receiving beta-blockers have shown a 14% relative risk reduction in mortality through 7 days and a 23% reduction in long-term mortality.[142]

2. Nitroglycerin

Class I

1. **Intravenous nitroglycerin is indicated in the first 48 hours after STEMI for treatment of persistent ischemia, CHF, or hypertension. The decision to administer intravenous nitroglycerin and the dose used should not preclude therapy with other proven mortality-reducing interventions, such as beta-blockers or ACE inhibitors. (*Level of Evidence: B*)**
2. **Intravenous, oral, or topical nitrates are useful beyond the first 48 hours after STEMI for treatment of recurrent angina or persistent CHF if their use does not preclude therapy with beta-blockers or ACE inhibitors. (*Level of Evidence: B*)**

Class IIb

1. **The continued use of nitrate therapy beyond the first 24 to 48 hours in the absence of continued or recurrent angina or CHF may be helpful, although the benefit is likely to be small and is not well established in contemporary practice. (*Level of Evidence: B*)**

Class III

1. **Nitrates should not be administered to patients with systolic pressure less than 90 mm Hg or greater than or equal to 30 mm Hg below baseline, severe bradycardia (less than 50 bpm), tachycardia (more than 100 bpm) or RV infarction. (*Level of Evidence: C*)**

3. Inhibition of the Renin-Angiotensin-Aldosterone System

Class I

1. **An ACE inhibitor should be administered orally during convalescence from STEMI in patients who tolerate this class of medication, and it should be continued over the long term. (*Level of Evidence: A*)**
2. **An ARB should be administered to STEMI patients who are intolerant of ACE inhibitors and have either clinical or radiological signs of heart failure or LVEF less than 0.40. Valsartan and candesartan have demonstrated efficacy for this recommendation. (*Level of Evidence: B*)**
3. **Long-term aldosterone blockade should be prescribed for post-STEMI patients without significant renal dysfunction (creatinine should be less than or equal to 2.5 mg/dL in men and less than or equal to 2.0 mg/dL in women) or hyperkalemia (potassium should be less than or equal to 5.0 mEq/L) who are already receiving therapeutic doses of an ACE inhibitor, have an LVEF less than or equal to 0.40, and have either symptomatic heart failure or diabetes. (*Level of Evidence: A*)**

Class IIa

1. **In STEMI patients who tolerate ACE inhibitors, an ARB can be useful as an alternative to ACE inhibitors provided there are either clinical or radiological signs of heart failure or LVEF is less than 0.40. Valsartan and candesartan have established efficacy for this recommendation. (*Level of Evidence: B*)**

The use of ACE inhibitors in the initial management of the STEMI patient was reviewed previously. The proportional benefit of ACE inhibitor therapy is largest in higher-risk subgroups, including those with previous infarction, heart failure, depressed LVEF, and tachycardia.[143–145] Survival benefit for patients more than 75 years old and for a low-risk subgroup without the features noted above is equivocal.[144,145]

Aldosterone blockade is another means of inhibiting the renin-angiotensin-aldosterone system that has been applied to patients in the post-STEMI setting. RALES (Randomized Aldactone Evaluation Study) and EPHESUS (Eplerenone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study) support the long-term use of an aldosterone blocker in STEMI patients with heart failure, an ejection fraction of 0.40 or less, or both, provided the serum creatinine is less than or equal to 2.5 mg/dL in men and less than or equal to 2.0 mg/dL in women and serum potassium concentration is less than or equal to 5.0 mEq/L.[146,147]

The use of ARBs after STEMI has not been explored as thoroughly as ACE inhibitors in STEMI patients.[148,149] Given the extensive randomized trial and routine clinical experience with ACE inhibitors, they remain the logical first agent for inhibition of the renin-angiotensin-aldosterone system in patients convalescing from STEMI.[150] Valsartan monotherapy (target dose 160 mg twice daily) should be administered to STEMI patients who are intolerant of ACE inhibitors and have evidence of LV dysfunction. Valsartan monotherapy can be a useful alternative to ACE inhibitors; the decision in individual patients may be influenced by physician and patient preference, cost, and anticipated side-effect profile.

4. Antiplatelets

Class I

1. **Aspirin 162 to 325 mg should be given on day 1 of STEMI and in the absence of contraindications should be continued indefinitely on a daily basis thereafter at a dose of 75 to 162 mg. (*Level of Evidence: A*)**
2. **A thienopyridine (preferably clopidogrel) should be administered to patients who are unable to take aspirin because of hypersensitivity or major gastrointestinal intolerance. (*Level of Evidence: C*)**
3. **For patients taking clopidogrel for whom CABG is planned, if possible, the drug should be withheld for at least 5 days, and preferably for 7, unless the urgency for revascularization outweighs the risks of bleeding. (*Level of Evidence: B*)**
4. **For patients who have undergone diagnostic cardiac catheterization and for whom PCI is planned, clopidogrel should be started and continued for at least 1 month after bare metal stent implantation and for several months after drug-eluting stent implantation (3 months for sirolimus, 6 months for paclitaxel) and up to 12 months in patients who are not at high risk for bleeding. (*Level of Evidence: B*)**

5. Antithrombotics

Class I

1. **Intravenous UFH (bolus of 60 U/kg, maximum 4000 U IV; initial infusion 12 U/kg per hour, maximum of 1000 U/h) or LMWH should be used in patients after STEMI who are at high risk for systemic emboli (large or anterior MI, atrial fibrillation, previous embolus, known LV thrombus, or cardiogenic shock). (*Level of Evidence: C*)**

Class IIa

1. **It is reasonable that STEMI patients not undergoing reperfusion therapy who do not have a contraindication to anticoagulation be treated with intravenous or subcutaneous UFH or with subcutaneous LMWH for at least 48 hours. In patients whose clinical condition necessitates prolonged bedrest and/or minimized activities, it is reasonable that treatment be continued until the patient is ambulatory. (*Level of Evidence: C*)**

Class IIb

1. **Prophylaxis for deep venous thrombosis (DVT) with subcutaneous LMWH (dosed appropriately for specific agent) or with subcutaneous UFH, 7500 U to 12 500 U twice per day until completely ambulatory, may be useful, but the effectiveness of such a strategy is not well established in the contemporary era of routine aspirin use and early mobilization. (*Level of Evidence: C*)**

6. Oxygen

Class I

1. **Supplemental oxygen therapy should be continued beyond the first 6 hours in STEMI patients with arterial oxygen desaturation (SaO_2 less than 90%) or overt pulmonary congestion. (*Level of Evidence: C*)**

E. Estimation of Infarct Size

Measurement of infarct size is an important element in the overall care of patients with STEMI. There are 5 major modalities that can be applied to sizing MI.

1. Electrocardiographic Techniques

Class I

1. **All patients with STEMI should have follow-up ECGs at 24 hours and at hospital discharge to assess the success of reperfusion and/or the extent of infarction, defined in part by the presence or absence of new Q waves. (*Level of Evidence: B*)**

2. Cardiac Biomarker Methods

The most widely accepted method for quantifying infarction has been the use of serial creatine kinase and the creatine kinase-MB isoenzyme.

3. Radionuclide Imaging

The most comprehensive assessment of STEMI with radionuclide imaging was developed with the technetium sestamibi SPECT approach.[151] This approach is well delineated in the ACC/AHA/ASNC Guidelines for the Clinical Use of Cardiac Radionuclide Imaging.[152]

4. Echocardiography

Global and regional LV function provides an assessment of the functional consequences of STEMI and ischemia. Readers are referred to the ACC/AHA/ASE 2003 Guideline Update for the Clinical Application of Echocardiography[153] and to Section 7.11.1.2 of the full-text STEMI guidelines.

5. Magnetic Resonance Imaging

Measurement of infarct size by MRI is a promising new technique that affords enhanced spatial resolution, thereby permitting more accurate assessment of both the transmural and circumferential extent of infarction.[154] However, additional experience and comparison with other methods of assessing infarct size are required before any clinical recommendations can be provided.

F. Hemodynamic Disturbances

1. Hemodynamic Assessment

Class I

1. Pulmonary artery catheter monitoring should be performed for the following:

- **a. Progressive hypotension, when unresponsive to fluid administration or when fluid administration may be contraindicated. (*Level of Evidence: C*)**
- **b. Suspected mechanical complications of STEMI, (ie, VSR, papillary muscle rupture, or free wall rupture with pericardial tamponade) if an echocardiogram has not been performed. (*Level of Evidence: C*)**

2. Intra-arterial pressure monitoring should be performed for the following:

a. Patients with severe hypotension (systolic arterial pressure less than 80 mm Hg). (*Level of Evidence: C*)
b. Patients receiving vasopressor/inotropic agents. (*Level of Evidence: C*)
c. Cardiogenic shock. (*Level of Evidence: C*)

Class IIa

1. Pulmonary artery catheter monitoring can be useful for the following:
   a. Hypotension in a patient without pulmonary congestion who has not responded to an initial trial of fluid administration. (*Level of Evidence: C*)
   b. Cardiogenic shock. (*Level of Evidence: C*)
   c. Severe or progressive CHF or pulmonary edema that does not respond rapidly to therapy. (*Level of Evidence: C*)
   d. Persistent signs of hypoperfusion without hypotension or pulmonary congestion. (*Level of Evidence: C*)
   e. Patients receiving vasopressor/inotropic agents. (*Level of Evidence: C*)
2. Intra-arterial pressure monitoring can be useful for patients receiving intravenous sodium nitroprusside or other potent vasodilators. (*Level of Evidence: C*)

Class IIb

1. Intra-arterial pressure monitoring might be considered in patients receiving intravenous inotropic agents. (*Level of Evidence: C*)

Class III

1. Pulmonary artery catheter monitoring is not recommended in patients with STEMI without evidence of hemodynamic instability or respiratory compromise. (*Level of Evidence: C*)
2. Intra-arterial pressure monitoring is not recommended for patients with STEMI who have no pulmonary congestion and have adequate tissue perfusion without use of circulatory support measures. (*Level of Evidence: C*)

2. Hypotension

Class I

1. Rapid volume loading with an IV infusion should be administered to patients without clinical evidence for volume overload. (*Level of Evidence: C*)
2. Rhythm disturbances or conduction abnormalities causing hypotension should be corrected. (*Level of Evidence: C*)
3. Intra-aortic balloon counterpulsation should be performed in patients who do not respond to other interventions, unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: B*)
4. Vasopressor support should be given for hypotension that does not resolve after volume loading. (*Level of Evidence: C*)
5. Echocardiography should be used to evaluate mechanical complications unless these are assessed by invasive measures. (*Level of Evidence: C*)

3. Low-Output State

Class I

1. LV function and potential presence of a mechanical complication should be assessed by echocardiography if these have not been evaluated by invasive measures. (*Level of Evidence: C*)
2. Recommended treatments for low-output states include:
   a. Inotropic support. (*Level of Evidence: B*)
   b. Intra-aortic counterpulsation. (*Level of Evidence: B*)
   c. Mechanical reperfusion with PCI or CABG. (*Level of Evidence: B*)
   d. Surgical correction of mechanical complications. (*Level of Evidence: B*)

Class III

1. Beta-blockers or calcium channel antagonists should not be administered to patients in a low-output state due to pump failure. (*Level of Evidence: B*)

A preshock state of hypoperfusion with normal blood pressure may develop before circulatory collapse and is manifested by cold extremities, cyanosis, oliguria, or decreased mentation.[155] Hospital mortality is high, so these patients should be aggressively diagnosed and treated as though they had cardiogenic shock. The initial pharmacological intervention for low cardiac output is often a dobutamine infusion. Intra-aortic counterpulsation therapy may be required to improve coronary artery perfusion pressure if hypotension is present. If the blood pressure permits, afterload-reducing agents should be added to decrease cardiac work and pulmonary congestion. Coronary artery revascularization of ischemic myocardium with either PCI or CABG has been shown to decrease mortality in patients with cardiogenic shock and is strongly recommended in suitable candidates.[75,108] Likewise, patients with VSR, papillary muscle rupture, or free wall rupture with pericardial tamponade may benefit from emergency surgical repair.

4. Pulmonary Congestion

Class I

1. Oxygen supplementation to arterial saturation greater than 90% is recommended for patients with pulmonary congestion. (*Level of Evidence: C*)
2. Morphine sulfate should be given to patients with pulmonary congestion. (*Level of Evidence: C*)
3. ACE inhibitors, beginning with titration of a short-acting ACE inhibitor with a low initial dose (eg, 1 to 6.25 mg of captopril) should be given to patients with pulmonary edema unless the systolic blood pressure is less than 100 mm Hg or more than 30 mm Hg below baseline. Patients with pulmonary congestion and marginal or low blood pressure often need circulatory support with inotropic and vasopressor agents and/or intra-aortic balloon counterpulsation to relieve pulmonary congestion and maintain adequate perfusion. (*Level of Evidence: A*)

4. **Nitrates should be administered to patients with pulmonary congestion unless the systolic blood pressure is less than 100 mm Hg or more than 30 mm Hg below baseline. Patients with pulmonary congestion and marginal or low blood pressure often need circulatory support with inotropic and vasopressor agents and/or intra-aortic balloon counterpulsation to relieve pulmonary congestion and maintain adequate perfusion. (*Level of Evidence: C*)**
5. **A diuretic (low- to intermediate-dose furosemide, or torsemide or bumetanide) should be administered to patients with pulmonary congestion if there is associated volume overload. Caution is advised for patients who have not received volume expansion. (*Level of Evidence: C*)**
6. **Beta-blockade should be initiated before discharge for secondary prevention. For those who remain in heart failure throughout the hospitalization, low doses should be initiated, with gradual titration on an outpatient basis. (*Level of Evidence: B*)**
7. **Long-term aldosterone blockade should be prescribed for post-STEMI patients without significant renal dysfunction (creatinine should be less than or equal to 2.5 mg/dL in men and less than or equal to 2.0 mg/dL in women) or hyperkalemia (potassium should be less than or equal to 5.0 mEq/L) who are already receiving therapeutic doses of an ACE inhibitor, have an LVEF less than or equal to 0.40, and have either symptomatic heart failure or diabetes. (*Level of Evidence: A*)**
8. **Echocardiography should be performed urgently to estimate LV and RV function and to exclude a mechanical complication. (*Level of Evidence: C*)**

Class IIb

1. **It may be reasonable to insert an intra-aortic balloon pump (IABP) for the management of patients with refractory pulmonary congestion. (*Level of Evidence: C*)**

Class III

1. **Beta-blockers or calcium channel blockers should not be administered acutely to STEMI patients with frank cardiac failure evidenced by pulmonary congestion or signs of a low-output state. (*Level of Evidence: B*)**

The immediate management goals include adequate oxygenation and preload reduction to relieve pulmonary congestion. Because of sympathetic stimulation, the blood pressure should be elevated in the presence of pulmonary edema. Patients with this appropriate response can typically tolerate the required medications, all of which lower blood pressure. However, iatrogenic cardiogenic shock may result from aggressive simultaneous use of agents that cause hypotension, initiating a cycle of hypoperfusion-ischemia. If acute pulmonary edema is not associated with elevation of the systemic blood pressure, impending cardiogenic shock must be suspected. If pulmonary edema is associated with hypotension, cardiogenic shock is diagnosed. Those patients often need circulatory support with inotropic and vasopressor agents and/or intra-aortic balloon counterpulsation to relieve pulmonary congestion and maintain adequate perfusion (Figure 4) (See Section VII.F.5, and see Section 7.6.5 of the full-text guidelines).

5. Cardiogenic Shock

Class I

1. **Intra-aortic balloon counterpulsation is recommended for STEMI patients when cardiogenic shock is not quickly reversed with pharmacological therapy. The IABP is a stabilizing measure for angiography and prompt revascularization. (*Level of Evidence: B*)**
2. **Intra-arterial monitoring is recommended for the management of STEMI patients with cardiogenic shock. (*Level of Evidence: C*)**
3. **Early revascularization, either PCI or CABG, is recommended for patients less than 75 years old with ST elevation or LBBB who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock, unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: A*)**
4. **Fibrinolytic therapy should be administered to STEMI patients with cardiogenic shock who are unsuitable for further invasive care and do not have contraindications to fibrinolysis. (*Level of Evidence: B*)**
5. **Echocardiography should be used to evaluate mechanical complications unless these are assessed by invasive measures. (*Level of Evidence: C*)**

Class IIa

1. **Pulmonary artery catheter monitoring can be useful for the management of STEMI patients with cardiogenic shock. (*Level of Evidence: C*)**
2. **Early revascularization, either PCI or CABG, is reasonable for selected patients 75 years or older with ST elevation or LBBB who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock. Patients with good prior functional status who agree to invasive care may be selected for such an invasive strategy. *(Level of Evidence: B*)**

Given the large overall treatment benefit of 13 lives saved per 100 patients treated in the SHOCK trial, early revascularization is recommended for those less than 75 years who are suitable for revascularization.[75,108,156] Two other large registries reported a substantial survival benefit for elderly patients who were selected clinically on the basis of physician judgment.

Interventions should be performed as soon as possible. It is recommended that patients who arrive at the hospital in cardiogenic shock (15% of cases) or who develop it after hospital arrival (85%) should be transferred to a regional tertiary care center with revascularization facilities experienced with these patients. When shock has resolved, ACE inhibitors and beta-blockers, initiated in low doses with progressive increases as recommended in the CHF guidelines, should be administered before discharge.[157] (See Section 7.6.7.6 of the full-text guidelines for discussion of mechanical support for the failing heart.)

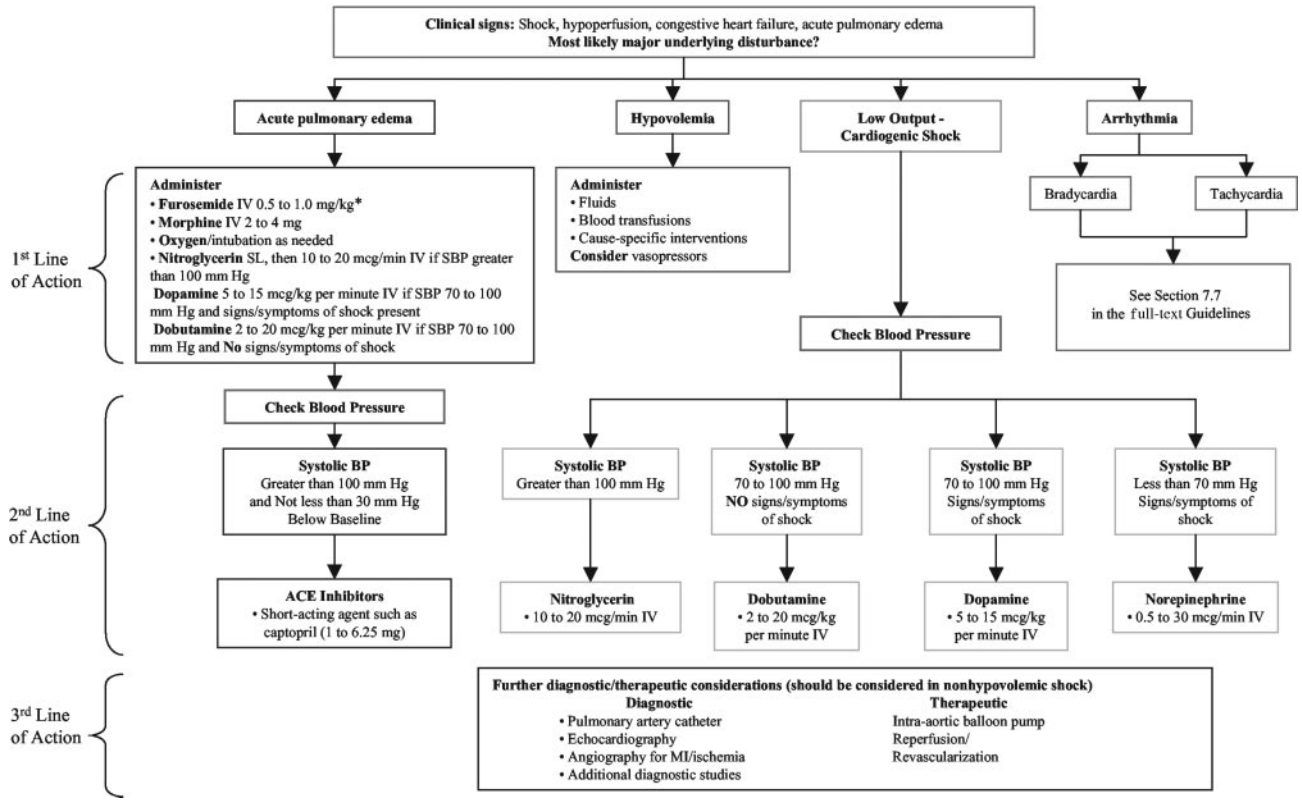

Figure 4. Emergency management of complicated ST-elevation myocardial infarction. The emergency management of patients with cardiogenic shock, acute pulmonary edema, or both is outlined. SBP indicates systolic blood pressure; IV, intravenous; BP, blood pressure; ACE, angiotensin converting enzyme; MI, myocardial infarction. *Furosemide less than 0.5 mg/kg for new-onset acute pulmonary edema without hypovolemia; 1 mg/kg for acute or chronic volume overload, renal insufficiency. Nesiritide has not been studied adequately in patients with STEMI. Combinations of medications, eg, dobutamine and dopamine, may be used. Modified with permission from Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: Part 7: The Era of Reperfusion. Section 1: Acute Coronary Syndromes (Acute Myocardial Infarction). *Circulation*. 2000;102(suppl 1):I-172–I-216.[26]

6. Right Ventricular Infarction

Class I

1. **Patients with inferior STEMI and hemodynamic compromise should be assessed with a right precordial V_4R lead to detect ST-segment elevation and an echocardiogram to screen for RV infarction. (See the ACC/AHA/ASE 2003 Guideline Update for the Clinical Application of Echocardiography.) (*Level of Evidence: B*)**
2. **The following principles apply to therapy of patients with STEMI and RV infarction and ischemic dysfunction:**
   a. **Early reperfusion should be achieved if possible. (*Level of Evidence: C*)**
   b. **AV synchrony should be achieved, and bradycardia should be corrected. (*Level of Evidence: C*)**
   c. **RV preload should be optimized, which usually requires initial volume challenge in patients with hemodynamic instability provided the jugular venous pressure is normal or low. (*Level of Evidence: C*)**
   d. **RV afterload should be optimized, which usually requires therapy for concomitant LV dysfunction. (*Level of Evidence: C*)**
   e. **Inotropic support should be used for hemodynamic instability not responsive to volume challenge. (*Level of Evidence: C*)**

Class IIa

1. **After infarction that leads to clinically significant RV dysfunction, it is reasonable to delay CABG surgery for 4 weeks to allow recovery of contractile performance. (*Level of Evidence: C*)**

Treatment of RV ischemia/infarction includes early maintenance of RV preload, reduction of RV afterload, inotropic support of the dysfunctional RV, early reperfusion,[158] and maintenance of AV synchrony.

7. Mechanical Causes of Heart Failure/Low-Output Syndrome

a. Diagnosis

On physical examination, the presence of a new cardiac murmur indicates the possibility of either a VSR or MR. A precise diagnosis can usually be established with transthoracic or transesophageal echocardiography.

b. Mitral Valve Regurgitation

Class I

1. **Patients with acute papillary muscle rupture should be considered for urgent cardiac surgical repair, unless further support is considered futile because of the**

patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: B*)
2. CABG surgery should be undertaken at the same time as mitral valve surgery. (*Level of Evidence: B*)

The patient should be stabilized with an IABP, inotropic support, and afterload reduction (to reduce regurgitant volume and pulmonary congestion) while emergency surgery is arranged.

c. Ventricular Septal Rupture After STEMI

Class I

1. Patients with STEMI complicated by the development of a VSR should be considered for urgent cardiac surgical repair, unless further support is considered futile because of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: B*)
2. CABG should be undertaken at the same time as repair of the VSR. (*Level of Evidence: B*)

Insertion of an IABP and prompt surgical referral are recommended for almost every patient with an acute VSR. Invasive monitoring is recommended in all patients, together with judicious use of inotropes and a vasodilator to maintain optimal hemodynamics. Surgical repair usually involves excision of all necrotic tissue and patch repair of the VSR, together with coronary artery grafting.

d. Left Ventricular Free-Wall Rupture

Class I

1. Patients with free-wall rupture should be considered for urgent cardiac surgical repair, unless further support is considered futile because of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: B*)
2. CABG should be undertaken at the same time as repair of free-wall rupture. (*Level of Evidence: C*)

Surgery includes repair of the ventricle by a direct suture technique or patch to cover the ventricular perforation[159] in addition to CABG as needed.

e. Left Ventricular Aneurysm

Class IIa

1. It is reasonable that patients with STEMI who develop a ventricular aneurysm associated with intractable ventricular tachyarrhythmias and/or pump failure unresponsive to medical and catheter-based therapy be considered for LV aneurysmectomy and CABG surgery. (*Level of Evidence: B*)

f. Mechanical Support of the Failing Heart

INTRA-AORTIC BALLOON COUNTERPULSATION

Class I

1. Intra-aortic balloon counterpulsation should be used in STEMI patients with hypotension (systolic blood pressure less than 90 mm Hg or 30 mm Hg below baseline mean arterial pressure) who do not respond to other interventions, unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care. See Section 7.6.2 of the full-text guidelines. (*Level of Evidence: B*)
2. Intra-aortic balloon counterpulsation is recommended for STEMI patients with low-output state. See Section 7.6.3 of the full-text guidelines. (*Level of Evidence: B*)
3. Intra-aortic balloon counterpulsation is recommended for STEMI patients when cardiogenic shock is not quickly reversed with pharmacological therapy. IABP is a stabilizing measure for angiography and prompt revascularization. See Section 7.6.5 of the full-text guidelines. (*Level of Evidence: B*)
4. Intra-aortic balloon counterpulsation should be used in addition to medical therapy for STEMI patients with recurrent ischemic-type chest discomfort and signs of hemodynamic instability, poor LV function, or a large area of myocardium at risk. Such patients should be referred urgently for cardiac catheterization and should undergo revascularization as needed. See Section 7.8.2 of the full-text guidelines. (*Level of Evidence: C*)

Class IIa

1. It is reasonable to manage STEMI patients with refractory polymorphic VT with intra-aortic balloon counterpulsation to reduce myocardial ischemia. See Section 7.7.1.2 of the full-text guidelines. (*Level of Evidence: B*)

Class IIb

1. It may be reasonable to use intra-aortic balloon counterpulsation in the management of STEMI patients with refractory pulmonary congestion. See Section 7.6.4 of the full-text guidelines. (*Level of Evidence: C*)

Selected patients with cardiogenic shock after STEMI, especially if not candidates for revascularization, may be considered for either a short- or long-term mechanical support device to serve as a bridge to recovery or to subsequent cardiac transplantation.

G. Arrhythmias After STEMI

1. Ventricular Arrhythmias

a. Ventricular Fibrillation

Class I

1. Ventricular fibrillation (VF) or pulseless VT should be treated with an unsynchronized electric shock with an initial monophasic shock energy of 200 J; if unsuccessful, a second shock of 200 to 300 J should be given, and then, if necessary, a third shock of 360 J. (*Level of Evidence: B*)

Class IIa

1. It is reasonable that VF or pulseless VT that is refractory to electrical shock be treated with amiodarone (300 mg or 5 mg/kg, IV bolus) followed by a repeat unsynchronized electric shock. (*Level of Evidence: B*)

2. **It is reasonable to correct electrolyte and acid-base disturbances (potassium greater than 4.0 mEq/L and magnesium greater than 2.0 mg/dL) to prevent recurrent episodes of VF once an initial episode of VF has been treated. (*Level of Evidence: C*)**

Class IIb

1. **It may be reasonable to treat VT or shock-refractory VF with boluses of intravenous procainamide. However, this has limited value owing to the length of time required for administration. (*Level of Evidence: C*)**

Class III

1. **Prophylactic administration of antiarrhythmic therapy is not recommended when using fibrinolytic agents. (*Level of Evidence: B*)**

There is no convincing evidence that the prophylactic use of lidocaine reduces mortality, and the prior practice of routine (prophylactic) administration of lidocaine to all patients with known or suspected STEMI has been largely abandoned. VF should be treated with an unsynchronized electric shock using an initial monophasic shock energy of 200 J. If this is unsuccessful, a second shock using 200 to 300 J and, if necessary, a third shock using 360 J are indicated.[160]

b. Ventricular Tachycardia

Class I

1. **Sustained (more than 30 seconds or causing hemodynamic collapse) polymorphic VT should be treated with an unsynchronized electric shock with an initial monophasic shock energy of 200 J; if unsuccessful, a second shock of 200 to 300 J should be given, and, if necessary, a third shock of 360 J. (*Level of Evidence: B*)**
2. **Episodes of sustained monomorphic VT associated with angina, pulmonary edema, or hypotension (blood pressure less than 90 mm Hg) should be treated with a synchronized electric shock of 100 J initial monophasic shock energy. Increasing energies may be used if not initially successful. Brief anesthesia is desirable if hemodynamically tolerable. (*Level of Evidence: B*)**
3. **Sustained monomorphic VT not associated with angina, pulmonary edema, or hypotension (blood pressure less than 90 mm Hg) should be treated with:**
   a. **Amiodarone: 150 mg infused over 10 minutes (alternative dose 5 mg/kg); repeat 150 mg every 10 to 15 minutes as needed. Alternative infusion: 360 mg over 6 hours (1 mg/min), then 540 mg over the next 18 hours (0.5 mg/min). The total cumulative dose, including additional doses given during cardiac arrest, must not exceed 2.2 g over 24 hours. (*Level of Evidence: B*)**
   b. **Synchronized electrical cardioversion starting at monophasic energies of 50 J (brief anesthesia is necessary). (*Level of Evidence: B*)**

Class IIa

1. **It is reasonable to manage refractory polymorphic VT by:**
   a. **Aggressive attempts to reduce myocardial ischemia and adrenergic stimulation, including therapies such as beta-adrenoceptor blockade, IABP use, and consideration of emergency PCI/CABG surgery. (*Level of Evidence: B*)**
   b. **Aggressive normalization of serum potassium to greater than 4.0 mEq/L and of magnesium to greater than 2.0 mg/dL. (*Level of Evidence: C*)**
   c. **If the patient has bradycardia to a rate less than 60 beats per minute or long QTc, temporary pacing at a higher rate may be instituted. (*Level of Evidence: C*)**

Class IIb

1. **It is may be useful to treat sustained monomorphic VT not associated with angina, pulmonary edema, or hypotension (blood pressure less than 90 mm Hg) with a procainamide bolus and infusion. (*Level of Evidence: C*)**

Class III

1. **The routine use of prophylactic antiarrhythmic drugs (ie, lidocaine) is not indicated for suppression of isolated ventricular premature beats, couplets, runs of accelerated idioventricular rhythm, or nonsustained VT. (*Level of Evidence: B*)**
2. **The routine use of prophylactic antiarrhythmic therapy is not indicated when fibrinolytic agents are administered. (*Level of Evidence: B*)**

Management Strategies for VT. Cardioversion is always indicated for episodes of sustained hemodynamically compromising VT.[161] Episodes of sustained VT that are somewhat better tolerated hemodynamically may initially be treated with drug regimens, including amiodarone or procainamide.

c. Ventricular Premature Beats

Class III

1. **Treatment of isolated ventricular premature beats, couplets, and nonsustained VT is not recommended unless they lead to hemodynamic compromise. (*Level of Evidence: A*)**

Before the present era of care of the STEMI patient with antiplatelet therapy, beta-blockade, ACE inhibitors, and, above all, reperfusion strategies, it was thought that ventricular warning arrhythmias preceded VF. Careful monitoring has refuted this concept, and treatment of these rhythm disturbances is not recommended unless they lead to hemodynamic compromise.

d. Accelerated Idioventricular Rhythms and Accelerated Junctional Rhythms

Class III

1. **Antiarrhythmic therapy is not indicated for accelerated idioventricular rhythm. (*Level of Evidence: C*)**

2. **Antiarrhythmic therapy is not indicated for accelerated junctional rhythm. (*Level of Evidence: C*)**

e. Implantable Cardioverter Defibrillator Implantation in Patients After STEMI

Class I

1. **An implantable cardioverter-defibrillator (ICD) is indicated for patients with VF or hemodynamically significant sustained VT more than 2 days after STEMI, provided the arrhythmia is not judged to be due to transient or reversible ischemia or reinfarction. (*Level of Evidence: A*)**
2. **An ICD is indicated for patients without spontaneous VF or sustained VT more than 48 hours after STEMI whose STEMI occurred at least 1 month previously, who have an LVEF between 0.31 and 0.40, demonstrate additional evidence of electrical instability (eg, nonsustained VT), and have inducible VF or sustained VT on electrophysiological testing. (*Level of Evidence: B*)**

Class IIa

1. **If there is reduced LVEF (0.30 or less), at least 1 month after STEMI and 3 months after coronary artery revascularization, it is reasonable to implant an ICD in post STEMI patients without spontaneous VF or sustained VT more than 48 hours after STEMI. (*Level of Evidence: B*)**

Class IIb

1. **The usefulness of an ICD is not well established in STEMI patients without spontaneous VF or sustained VT more than 48 hours after STEMI who have a reduced LVEF (0.31 to 0.40) at least 1 month after STEMI but who have no additional evidence of electrical instability (eg, nonsustained VT). (*Level of Evidence: B*)**
2. **The usefulness of an ICD is not well established in STEMI patients without spontaneous VF or sustained VT more than 48 hours after STEMI who have a reduced LVEF (0.31 to 0.40) at least 1 month after STEMI and additional evidence of electrical instability (eg, nonsustained VT) but who do not have inducible VF or sustained VT on electrophysiological testing. (*Level of Evidence: B*)**

Class III

1. **An ICD is not indicated in STEMI patients who do not experience spontaneous VF or sustained VT more than 48 hours after STEMI and in whom the LVEF is greater than 0.40 at least 1 month after STEMI. (*Level of Evidence: C*)**

See the full-text guidelines for discussion.

2. ***Supraventricular Arrhythmias/Atrial Fibrillation***

Class I

1. **Sustained atrial fibrillation and atrial flutter in patients with hemodynamic compromise or ongoing ischemia should be treated with one or more of the following:**
   a. **Synchronized cardioversion with an initial monophasic shock of 200 J for atrial fibrillation and 50 J for flutter, preceded by brief general anesthesia or conscious sedation whenever possible. (*Level of Evidence: C*)**
   b. **For episodes of atrial fibrillation that do not respond to electrical cardioversion or recur after a brief period of sinus rhythm, the use of antiarrhythmic therapy aimed at slowing the ventricular response is indicated. One or more of these pharmacological agents may be used:**
      i. **Intravenous amiodarone.[162] (*Level of Evidence: C*)**
      ii. **Intravenous digoxin for rate control principally for patients with severe LV dysfunction and heart failure. (*Level of Evidence: C*)**
2. **Sustained atrial fibrillation and atrial flutter in patients with ongoing ischemia but without hemodynamic compromise should be treated with one or more of the following:**
   a. **Beta-adrenergic blockade is preferred, unless contraindicated. (*Level of Evidence: C*)**
   b. **Intravenous diltiazem or verapamil. (*Level of Evidence: C*)**
   c. **Synchronized cardioversion with an initial monophasic shock of 200 J for atrial fibrillation and 50 J for flutter, preceded by brief general anesthesia or conscious sedation whenever possible. (*Level of Evidence: C*)**
3. **For episodes of sustained atrial fibrillation or flutter without hemodynamic compromise or ischemia, rate control is indicated. In addition, patients with sustained atrial fibrillation or flutter should be given anticoagulant therapy. Consideration should be given to cardioversion to sinus rhythm in patients with a history of atrial fibrillation or flutter prior to STEMI. (*Level of Evidence: C*)**
4. **Reentrant paroxysmal supraventricular tachycardia, because of its rapid rate, should be treated with the following in the sequence shown:**
   a. **Carotid sinus massage. (*Level of Evidence: C*)**
   b. **Intravenous adenosine (6 mg × 1 over 1 to 2 seconds; if no response, 12 mg IV after 1 to 2 minutes may be given; repeat 12 mg dose if needed. (*Level of Evidence: C*)**
   c. **Intravenous beta-adrenergic blockade with metoprolol (2.5 to 5.0 mg every 2 to 5 minutes to a total of 15 mg over 10 to 15 minutes) or atenolol (2.5 to 5.0 mg over 2 minutes to a total of 10 mg in 10 to 15 minutes). (*Level of Evidence: C*)**
   d. **Intravenous diltiazem (20 mg [0.25 mg/kg]) over 2 minutes followed by an infusion of 10 mg/h). (*Level of Evidence: C*)**
   e. **Intravenous digoxin, recognizing that there may be a delay of at least 1 hour before pharmacological effects appear (8 to 15 mcg/kg [0.6 to 1.0 mg in a person weighing 70 kg]). (*Level of Evidence: C*)**

Class III

1. **Treatment of atrial premature beats is not indicated. (*Level of Evidence: C*)**

See the full-text guidelines for discussion.

3. Bradyarrhythmias

See Table 3 for recommendations.

a. Acute Treatment of Conduction Disturbances and Bradyarrhythmias

VENTRICULAR ASYSTOLE

Class I

1. **Prompt resuscitative measures, including chest compressions, atropine, vasopressin, epinephrine, and temporary pacing, should be administered to treat ventricular asystole. (*Level of Evidence: B*)**

b. Use of Permanent Pacemakers

PERMANENT PACING FOR BRADYCARDIA OR CONDUCTION BLOCKS ASSOCIATED WITH STEMI

Class I

1. **Permanent ventricular pacing is indicated for persistent second-degree AV block in the His-Purkinje system with bilateral bundle-branch block or third-degree AV block within or below the His-Purkinje system after STEMI. (*Level of Evidence: B*)**
2. **Permanent ventricular pacing is indicated for transient advanced second- or third-degree infranodal AV block and associated bundle-branch block. If the site of block is uncertain, an electrophysiological study may be necessary. (*Level of Evidence: B*)**
3. **Permanent ventricular pacing is indicated for persistent and symptomatic second- or third-degree AV block. (*Level of Evidence: C*)**

Class IIb

1. **Permanent ventricular pacing may be considered for persistent second- or third-degree AV block at the AV node level. (*Level of Evidence: B*)**

Class III

1. **Permanent ventricular pacing is not recommended for transient AV block in the absence of intraventricular conduction defects. (*Level of Evidence: B*)**
2. **Permanent ventricular pacing is not recommended for transient AV block in the presence of isolated left anterior fascicular block. (*Level of Evidence: B*)**
3. **Permanent ventricular pacing is not recommended for acquired left anterior fascicular block in the absence of AV block. (*Level of Evidence: B*)**
4. **Permanent ventricular pacing is not recommended for persistent first-degree AV block in the presence of bundle-branch block that is old or of indeterminate age. (*Level of Evidence: B*)**

Indications for permanent pacing after STEMI in patients experiencing AV block are related in large measure to the presence of intraventricular conduction defects (Table 3). Unlike some other indications for permanent pacing, the criteria for patients with STEMI and AV block do not necessarily depend on the presence of symptoms. Furthermore, the requirement for temporary pacing in STEMI does not by itself constitute an indication for permanent pacing.[163]

SINUS NODE DYSFUNCTION AFTER STEMI

Class I

1. **Symptomatic sinus bradycardia, sinus pauses greater than 3 seconds, or sinus bradycardia with a heart rate less than 40 bpm and associated hypotension or signs of systemic hemodynamic compromise should be treated with an intravenous bolus of atropine 0.6 to 1.0 mg. If bradycardia is persistent and maximal (2 mg) doses of atropine have been used, transcutaneous or transvenous (preferably atrial) temporary pacing should be instituted. (*Level of Evidence: C*)**

The published ACC/AHA Guidelines[164] for Implantation of Pacemakers should be used to guide therapy in STEMI patients with persistent sinus node dysfunction.

PACING MODE SELECTION IN STEMI PATIENTS

Class I

1. **All patients who have an indication for permanent pacing after STEMI should be evaluated for ICD indications. (*Level of Evidence: C*)**

Class IIa

1. **It is reasonable to implant a permanent dual-chamber pacing system in STEMI patients who need permanent pacing and are in sinus rhythm. It is reasonable that patients in permanent atrial fibrillation or flutter receive a single-chamber ventricular device. (*Level of Evidence: C*)**
2. **It is reasonable to evaluate all patients who have an indication for permanent pacing after STEMI for biventricular pacing (cardiac resynchronization therapy). (*Level of Evidence: C*)**

When a permanent pacemaker is being considered for a post-STEMI patient, the clinician should address 2 additional questions regarding the patient: is there an indication for biventricular pacing, and is there an indication for ICD use?[165] The algorithm to define whether an ICD is indicated is contained in Figure 5.

H. Recurrent Chest Pain After STEMI

1. Pericarditis

Class I

1. **Aspirin is recommended for treatment of pericarditis after STEMI. Doses as high as 650 mg orally (enteric) every 4 to 6 hours may be needed. (*Level of Evidence: B*)**
2. **Anticoagulation should be immediately discontinued if pericardial effusion develops or increases. (*Level of Evidence: C*)**

TABLE 3. Recommendations for Treatment of Atrioventricular and Intraventricular Conduction Disturbances During STEMI

	Atrioventricular Conduction													
			First-Degree AV Block				Mobitz I Second-Degree AV Block				Mobitz II Second-Degree AV Block			
Intraventricular Conduction	Normal		Anterior MI		Nonanterior MI		Anterior MI		Nonanterior MI		Anterior MI		Nonanterior MI	
Normal	**Action**	**Class**	**Action**	**Class**	**Action**	**Class**	**Action**	**Class**	**Action**	**Class**	**Action**	**Class**	**Action**	**Class**
	Observe	I	Observe	I	Observe	I	Observe	IIb	Observe	IIa	Observe	III	Observe	III
	A	III	A	III	A	III	A*	III	A	III	A	III	A	III
	TC	III	TC	IIb	TC	IIb	TC	I	TC	I	TC	I	TC	I
	TV	III	TV	III	TV	III	TV	III	TV	III	TV	IIa	TV	IIa
Old or new fascicular block (LAFB or LPFB)	Observe	I	Observe	IIb	Observe	IIb	Observe	IIb	Observe	IIb	Observe	III	Observe	III
	A	III	A	III	A	III	A*	III	A	III	A	III	A	III
	TC	IIb	TC	I	TC	IIa	TC	I	TC	I	TC	I	TC	I
	TV	III	TV	III	TV	III	TV	III	TV	III	TV	IIa	TV	IIb
Old bundle-branch block	Observe	I	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III
	A	III	A	III	A	III	A*	III	A	III	A	III	A	III
	TC	IIb	TC	I	TC	I	TC	I	TC	I	TC	I	TC	I
	TV	III	TV	IIb	TV	IIb	TV	IIb	TV	IIb	TV	IIa	TV	IIa
New bundle-branch block	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III
	A	III	A	III	A	III	A*	III	A	III	A	III	A	III
	TC	I	TC	I	TC	I	TC	I	TC	I	TC	IIb	TC	IIb
	TV	IIb	TV	IIa	TV	IIa	TV	IIa	TV	IIa	TV	I	TV	I
Fascicular block + RBBB	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III
	A	III	A	III	A	III	A*	III	A	III	A	III	A	III
	TC	I	TC	I	TC	I	TC	I	TC	I	TC	IIb	TC	IIb
	TV	IIb	TV	IIa	TV	IIa	TV	IIa	TV	IIa	TV	I	TV	I
Alternating left and right bundle-branch block	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III	Observe	III
	A	III	A	III	A	III	A*	III	A	III	A	III	A	III
	TC	IIb	TC	IIb	TC	IIb	TC	IIb	TC	IIb	TC	IIb	TC	IIb
	TV	I	TV	I	TV	I	TV	I	TV	I	TV	I	TV	I

This table is designed to summarize the atrioventricular (column headings) and intraventricular (row headings) conduction disturbances that may occur during acute anterior or nonanterior STEMI, the possible treatment options, and the indications for each possible therapeutic option.

LAFB indicates left anterior fascicular block; LPFB, left posterior fascicular block; RBBB, right bundle-branch block; A, atropine; TC, transcutaneous pacing; TV, temporary transvenous pacing; STEMI, ST elevation myocardial infarction; AV, atrioventricular; and MI, myocardial infarction.

Action

There are 4 possible actions, or therapeutic options, listed and classified for each bradyarrhythmia or conduction problem:

1. Observe: continued ECG monitoring, no further action planned.

2. A, and A*: atropine administered at 0.6 to 1.0 mg IV every 5 minutes to up to 0.04 mg/kg. In general, because the increase in sinus rate with atropine is unpredictable, this is to be avoided unless there is symptomatic bradycardia that will likely respond to a vagolytic agent, such as sinus bradycardia or Mobitz I, as denoted by the asterisk, above.

3. TC: application of transcutaneous pads and standby transcutaneous pacing with no further progression to transvenous pacing imminently planned.

4. TV: temporary transvenous pacing. It is assumed, but not specified in the table, that at the discretion of the clinician, transcutaneous pads will be applied and standby transcutaneous pacing will be in effect as the patient is transferred to the fluoroscopy unit for temporary transvenous pacing.

Class

Each possible therapeutic option is further classified according to ACC/AHA criteria as I, IIa, IIb, and III.

Level of Evidence

This table was developed from (1) published observational case reports and case series, (2) published summaries, not meta-analyses, of these data; and (3) expert opinion, largely from the prereperfusion era. There are no published randomized trials comparing different strategies of managing conduction disturbances after STEMI. Thus, the level of evidence for the recommendations in this table is C.

How to Use the Table

Example: 54-year-old man is admitted with an anterior STEMI and a narrow QRS on admission. On day 1, he develops a right bundle-branch block (RBBB), with a PR interval of 0.28 seconds.

1. RBBB is an intraventricular conduction disturbance, so look at row 'New bundle-branch block.'

2. Find the column for 'First-Degree AV Block.'

3. Find the 'Action' and 'Class' cells at the convergence.

4. Note that "Observe" and "Atropine" are class III, not indicated; transcutaneous pacing (TC) is class I. Temporary transvenous pacing (TV) is class IIb.

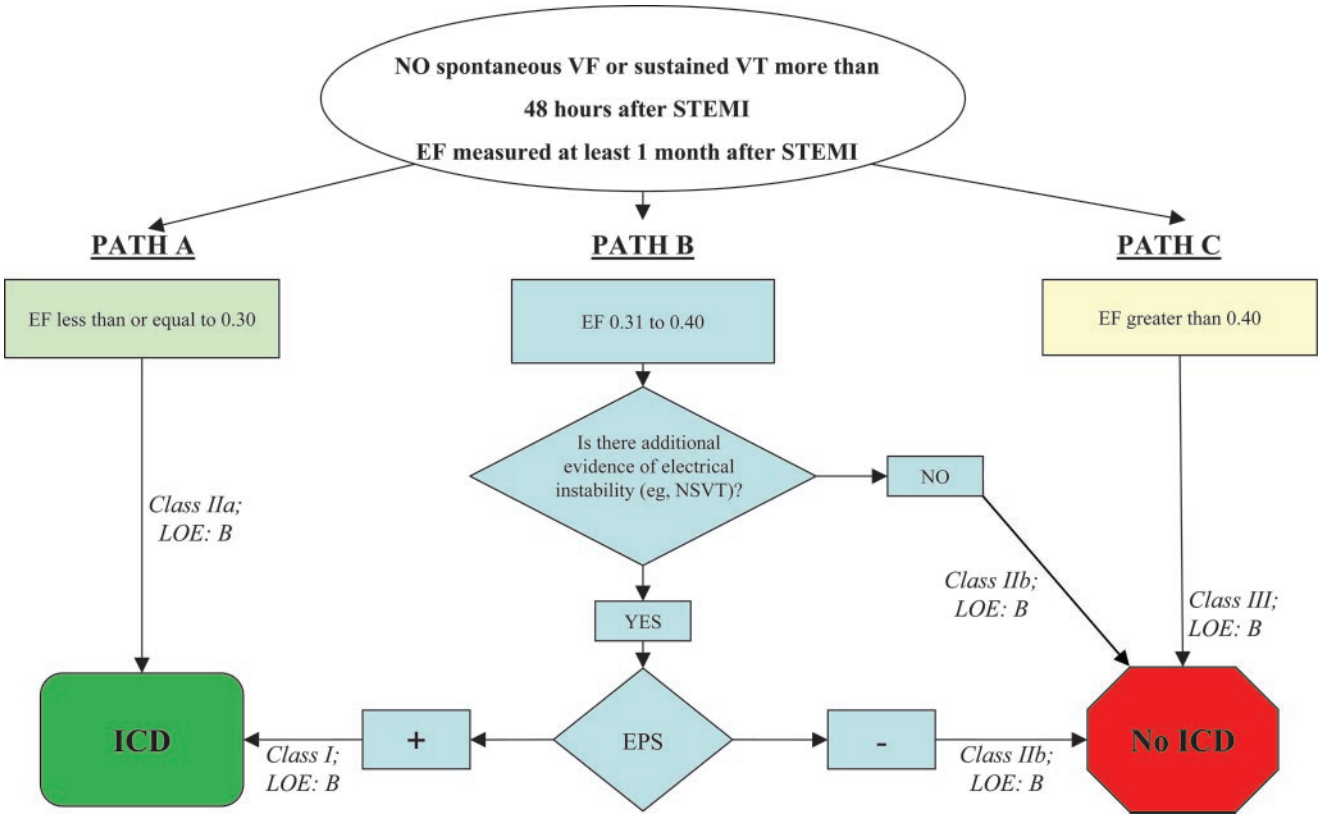

Figure 5. Algorithm to aid in selection of ICD in patients with STEMI and diminished ejection fraction (EF). Appropriate management path is selected based on LVEF measured at least 1 month after STEMI. These criteria, which are based on published data, form the basis for the full-text guidelines in Section 7.7.1.5. All patients, whether an ICD is implanted or not, should receive medical therapy as outlined in the guidelines. VF indicates ventricular fibrillation; VII, ventricular tachycardia; STEMI, ST-elevation myocardial infarction; NSVT, nonsustained VT; LOE, level of evidence; EPS, electrophysiological studies; LVEF, left ventricular EF.

Class IIa

1. **For episodes of pericarditis after STEMI that are not adequately controlled with aspirin, it is reasonable to administer 1 or more of the following:**
   a. **Colchicine 0.6 mg every 12 hours orally. (*Level of Evidence: B*)**
   b. **Acetaminophen 500 mg orally every 6 hours. (*Level of Evidence: C*)**

Class IIb

1. **Nonsteroidal anti-inflammatory drugs may be considered for pain relief; however, they should not be used for extended periods because of their continuous effect on platelet function, an increased risk of myocardial scar thinning, and infarct expansion. (*Level of Evidence: B*)**
2. **Corticosteroids might be considered only as a last resort in patients with pericarditis refractory to aspirin or nonsteroidal drugs. Although corticosteroids are effective for pain relief, their use is associated with an increased risk of scar thinning and myocardial rupture. (*Level of Evidence: C*)**

Class III

1. **Ibuprofen should not be used for pain relief because it blocks the antiplatelet effect of aspirin and can cause myocardial scar thinning and infarct expansion. (*Level of Evidence: B*)**

2. Recurrent Ischemia/Infarction

Class I

1. **Patients with recurrent ischemic-type chest discomfort after initial reperfusion therapy for STEMI should undergo escalation of medical therapy with nitrates and beta-blockers to decrease myocardial oxygen demand and reduce ischemia. Intravenous anticoagulation should be initiated if not already accomplished. (*Level of Evidence: B*)**
2. **In addition to escalation of medical therapy, patients with recurrent ischemic-type chest discomfort and signs of hemodynamic instability, poor LV function, or a large area of myocardium at risk should be referred urgently for cardiac catheterization and undergo revascularization as needed. Insertion of an IABP should also be considered. (*Level of Evidence: C*)**
3. **Patients with recurrent ischemic-type chest discomfort who are considered candidates for revascularization should undergo coronary arteriography and PCI or CABG as dictated by coronary anatomy. (*Level of Evidence: B*)**

Class IIa

1. **It is reasonable to (re)administer fibrinolytic therapy to patients with recurrent ST elevation and ischemic-type chest discomfort who are not considered candi-**

dates for revascularization or for whom coronary angiography and PCI cannot be rapidly (ideally within 60 minutes from the onset of recurrent discomfort) implemented. (*Level of Evidence: C*)

Class III

1. **Streptokinase should not be readministered to treat recurrent ischemia/infarction in patients who received a non–fibrin-specific fibrinolytic agent more than 5 days previously to treat the acute STEMI event. (*Level of Evidence: C*)**

Patients with recurrent ischemic-type chest discomfort should undergo escalation of medical therapy that includes beta-blockers (intravenously and then orally) and nitrates (sublingually and then intravenously); consideration should be given to initiation of intravenous anticoagulation if the patient is not already therapeutically anticoagulated. Secondary causes of recurrent ischemia, such as poorly controlled heart failure, anemia, and arrhythmias, should be corrected.

I. Other Complications

1. *Ischemic Stroke*

Class I

1. **Neurological consultation should be obtained in STEMI patients who have an acute ischemic stroke. (*Level of Evidence: C*)**
2. **STEMI patients who have an acute ischemic stroke should be evaluated with echocardiography, neuroimaging, and vascular imaging studies to determine the cause of the stroke. (*Level of Evidence: C*)**
3. **STEMI patients with acute ischemic stroke and persistent atrial fibrillation should receive lifelong moderate-intensity (international normalized ratio [INR] 2 to 3) warfarin therapy. (*Level of Evidence: A*)**
4. **STEMI patients with or without acute ischemic stroke who have a cardiac source of embolism (atrial fibrillation, mural thrombus, or akinetic segment) should receive moderate-intensity (INR 2 to 3) warfarin therapy (in addition to aspirin). The duration of warfarin therapy should be dictated by clinical circumstances (eg, at least 3 months for patients with an LV mural thrombus or akinetic segment and indefinitely in patients with persistent atrial fibrillation). The patient should receive LMWH or UFH until adequately anticoagulated with warfarin. (*Level of Evidence: B*)**

Class IIa

1. **It is reasonable to assess the risk of ischemic stroke in patients with STEMI. (*Level of Evidence: A*)**
2. **It is reasonable that STEMI patients with nonfatal acute ischemic stroke receive supportive care to minimize complications and maximize functional outcome. *(Level of Evidence: C*)**

Class IIb

1. **Carotid angioplasty/stenting, 4 to 6 weeks after ischemic stroke, might be considered in STEMI patients who have an acute ischemic stroke attributable to an internal carotid artery–origin stenosis of at least 50% and who have a high surgical risk of morbidity/mortality early after STEMI. (*Level of Evidence: C*)**

An algorithm for evaluation and antithrombotic therapy for ischemic stroke is shown in Figure 35 of the full-text guideline.

2. *DVT and Pulmonary Embolism*

Class I

1. **DVT or pulmonary embolism after STEMI should be treated with full-dose LMWH for a minimum of 5 days and until the patient is adequately anticoagulated with warfarin. Start warfarin concurrently with LMWH and titrate to INR of 2 to 3. (*Level of Evidence: A*)**
2. **Patients with CHF after STEMI who are hospitalized for prolonged periods, unable to ambulate, or considered at high risk for DVT and are not otherwise anticoagulated should receive low-dose heparin prophylaxis, preferably with LMWH. (*Level of Evidence: A*)**

J. CABG Surgery After STEMI

1. *Timing of Surgery*

Class IIa

1. **In patients who have had a STEMI, CABG mortality is elevated for the first 3 to 7 days after infarction, and the benefit of revascularization must be balanced against this increased risk. Patients who have been stabilized (no ongoing ischemia, hemodynamic compromise, or life-threatening arrhythmia) after STEMI and who have incurred a significant fall in LV function should have their surgery delayed to allow myocardial recovery to occur. If critical anatomy exists, revascularization should be undertaken during the index hospitalization. (*Level of Evidence: B*)**

The Writing Committee believes that if stable STEMI patients with preserved LV function require surgical revascularization, then CABG can be undertaken within several days of the infarction without an increased risk.

2. *Arterial Grafting*

Class I

1. **An internal mammary artery graft to a significantly stenosed left anterior descending coronary artery should be used whenever possible in patients undergoing CABG after STEMI. (*Level of Evidence: B*)**

3. *CABG for Recurrent Ischemia After STEMI*

Class I

1. **Urgent CABG is indicated if the coronary angiogram reveals anatomy that is unsuitable for PCI. (*Level of Evidence: B*)**

4. Elective CABG Surgery After STEMI in Patients With Angina

Class I

1. **CABG is recommended for patients with stable angina who have significant left main coronary artery stenosis. (*Level of Evidence: A*)**
2. **CABG is recommended for patients with stable angina who have left main equivalent disease: significant (at least 70%) stenosis of the proximal left anterior descending coronary artery and proximal left circumflex artery. (*Level of Evidence: A*)**
3. **CABG is recommended for patients with stable angina who have 3-vessel disease (Survival benefit is greater when LVEF is less than 0.50). (*Level of Evidence: A*)**
4. **CABG is beneficial for patients with stable angina who have 1- or 2-vessel coronary disease without significant proximal left anterior descending coronary artery stenosis but with a large area of viable myocardium and high-risk criteria on noninvasive testing. (*Level of Evidence: B*)**
5. **CABG is recommended in patients with stable angina who have 2-vessel disease with significant proximal left anterior descending coronary artery stenosis and either ejection fraction less than 0.50 or demonstrable ischemia on noninvasive testing. (*Level of Evidence: A*)**

The role of surgical revascularization has been reviewed extensively in the ACC/AHA Guidelines for CABG Surgery.[166] Consideration for revascularization after STEMI includes PCI and CABG. Providers should individualize patient management on the basis of clinical circumstances, available revascularization options, and patient preference.

5. CABG Surgery After STEMI and Antiplatelet Agents

Class I

1. **Aspirin should not be withheld before elective or nonelective CABG after STEMI. (*Level of Evidence: C*)**
2. **Aspirin (75 to 325 mg daily) should be prescribed as soon as possible (within 24 hours) after CABG unless contraindicated. (*Level of Evidence: B*)**
3. **In patients taking clopidogrel in whom elective CABG is planned, the drug should be withheld for 5 to 7 days. (*Level of Evidence: B*)**

STEMI patients undergoing revascularization frequently receive 1 or more antiplatelet agents and heparin, all of which may increase risk of serious bleeding during and after cardiac surgery. Delaying surgery until platelet function has recovered may not be feasible in many circumstances. In patients treated with the small-molecule GP IIb/IIIa receptor antagonists, tirofiban and eptifibatide, platelet function returns toward normal within 4 hours of stopping treatment. Platelet aggregation does not return toward normal for more than 48 hours in patients treated with abciximab. Management strategies, other than delaying surgery, include platelet transfusions for patients who were recently treated with abciximab, reduced heparin dosing during cardiopulmonary bypass, and possible use of antifibrinolytic agents such as aprotinin or tranexamic acid.[167] Because clopidogrel, when added to aspirin, increases the risk of bleeding during major surgery in patients who are scheduled for elective CABG, clopidogrel should be withheld for at least 5 days[168] and preferably for 7 days before surgery.[169]

K. Convalescence, Discharge, and Post-MI Care

1. Risk Stratification at Hospital Discharge

The risk stratification approach for decision-making about catheterization is described in Figure 6. The suggested algorithm for electrophysiological testing and ICD placement is shown in Figure 5.

a. Role of Exercise Testing

Class I

1. **Exercise testing should be performed either in the hospital or early after discharge in STEMI patients not selected for cardiac catheterization and without high-risk features to assess the presence and extent of inducible ischemia. (*Level of Evidence: B*)**
2. **In patients with baseline abnormalities that compromise ECG interpretation, echocardiography or myocardial perfusion imaging should be added to standard exercise testing. (*Level of Evidence: B*)**

Class IIb

1. **Exercise testing might be considered before discharge of patients recovering from STEMI to guide the post-discharge exercise prescription or to evaluate the functional significance of a coronary lesion previously identified at angiography. (*Level of Evidence: C*)**

Class III

1. **Exercise testing should not be performed within 2 to 3 days of STEMI in patients who have not undergone successful reperfusion. (*Level of Evidence: C*)**
2. **Exercise testing should not be performed to evaluate patients with STEMI who have unstable postinfarction angina, decompensated CHF, life-threatening cardiac arrhythmias, noncardiac conditions that severely limit their ability to exercise, or other absolute contraindications to exercise testing.[170] (*Level of Evidence: C*)**
3. **Exercise testing should not be used for risk stratification in patients with STEMI who have already been selected for cardiac catheterization. (*Level of Evidence: C*)**

Exercise testing after STEMI may be performed to (1) assess functional capacity and the patient's ability to perform tasks at home and at work; (2) establish exercise parameters for cardiac rehabilitation; (3) evaluate the efficacy of the patient's current medical regimen; (4) risk-stratify the post-STEMI patient according to the likelihood of a subsequent cardiac event;[171–175] (5) evaluate chest pain symptoms after STEMI; and (6) provide reassurance to patients regarding their functional capacity after STEMI as a guide to returning to work.

b. Role of Echocardiography

Noninvasive imaging in patients recovering from STEMI includes echocardiography and radionuclide imaging. This section discusses the role of echocardiography. (See Sections

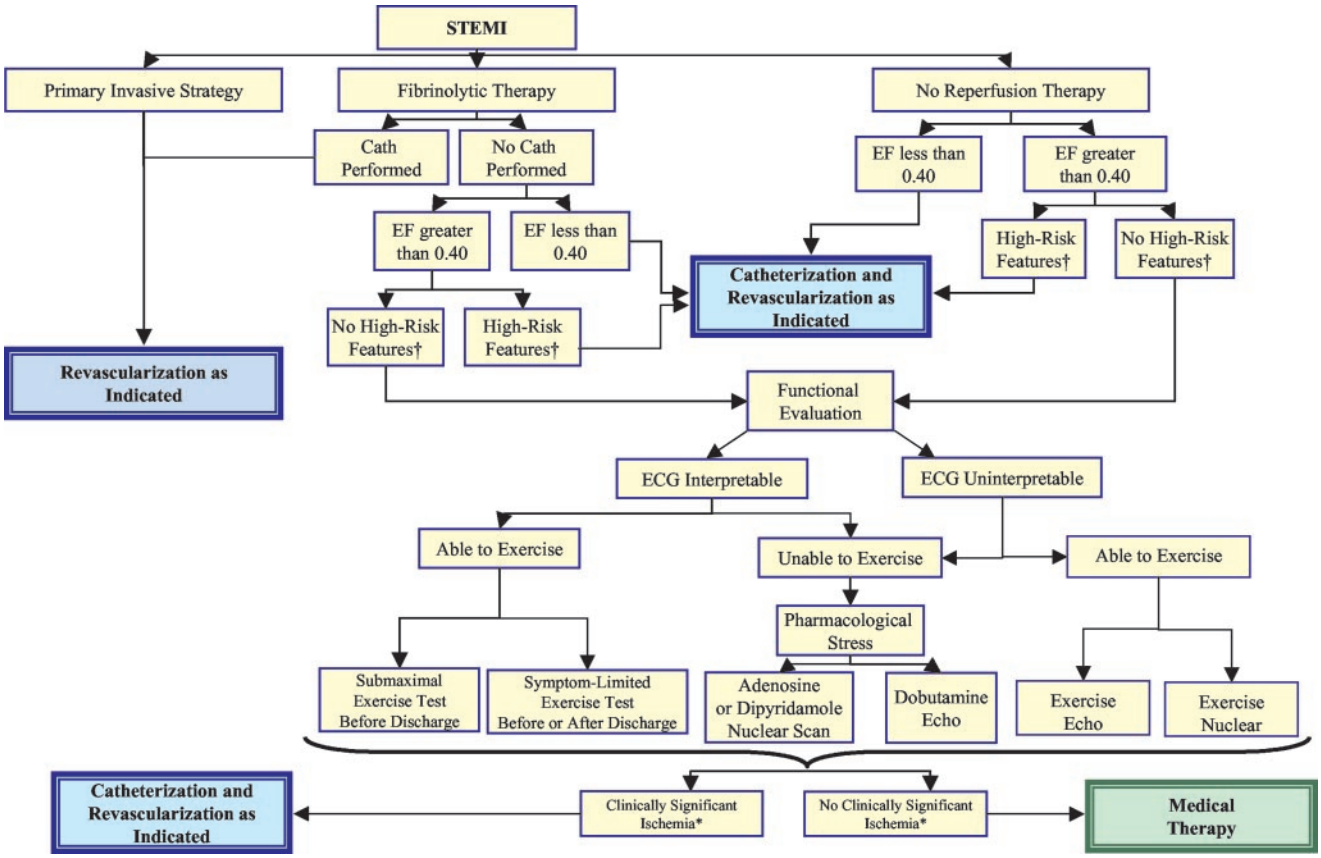

Figure 6. Evidence-based approach to need for catheterization (cath) and revascularization after STEMI. This algorithm shows treatment paths for patients who initially undergo a primary invasive strategy, receive fibrinolytic therapy, or do not undergo reperfusion therapy for STEMI. Patients who have not undergone a primary invasive strategy and have no high-risk features should undergo functional evaluation with one of the noninvasive tests shown. When clinically significant ischemia is detected, patients should undergo catheterization and revascularization as indicated; if no clinically significant ischemia is detected, medical therapy is prescribed after STEMI. *Please see Table 23 of the ACC/AHA Guidelines for the Management of Patients With Chronic Stable Angina for further definition. †Please see Table 3, Section 6.3.1.6.2., and Section 7.3. in the full-text STEMI guidelines for further discussion. STEMI indicates ST-elevation myocardial infarction; EF, ejection fraction; ECG, electrocardiography.

7.11.1.3, 7.11.1.4, and 7.11.1.5 of the full-text guidelines for additional discussion on imaging considerations.)

Class I

1. **Echocardiography should be used in patients with STEMI not undergoing LV angiography to assess baseline LV function, especially if the patient is hemodynamically unstable. (*Level of Evidence: C*)**
2. **Echocardiography should be used to evaluate patients with inferior STEMI, clinical instability, and clinical suspicion of RV infarction. (See ACC/AHA Guidelines for Clinical Application of Echocardiography.[153]) (*Level of Evidence: C*)**
3. **Echocardiography should be used in patients with STEMI to evaluate suspected complications, including acute MR, cardiogenic shock, infarct expansion, VSR, intracardiac thrombus, and pericardial effusion. (*Level of Evidence: C*)**
4. **Stress echocardiography (or myocardial perfusion imaging) should be used in patients with STEMI for in-hospital or early postdischarge assessment for inducible ischemia when baseline abnormalities are expected to compromise ECG interpretation. (*Level of Evidence: C*)**

Class IIa

1. **Echocardiography is reasonable in patients with STEMI to re-evaluate ventricular function during recovery when results are used to guide therapy. (*Level of Evidence: C*)**
2. **Dobutamine echocardiography (or myocardial perfusion imaging) is reasonable in hemodynamically and electrically stable patients 4 or more days after STEMI to assess myocardial viability when required to define the potential efficacy of revascularization. *(Level of Evidence: C*)**
3. **In STEMI patients who have not undergone contrast ventriculography, echocardiography is reasonable to assess ventricular function after revascularization. *(Level of Evidence: C*)**

Class III

1. **Echocardiography should not be used for early routine reevaluation in patients with STEMI in the absence of any change in clinical status or revascularization procedure. Reassessment of LV function 30 to 90 days later may be reasonable. (*Level of Evidence: C*)**

The use of echocardiography in STEMI is discussed in detail in the ACC/AHA/ASE 2003 Guideline Update for the Clinical Application of Echocardiography.[153]

c. Exercise Myocardial Perfusion Imaging

Noninvasive imaging in patients recovering from STEMI includes echocardiography and radionuclide imaging. This section discusses the role of exercise myocardial perfusion imaging. (See Sections 7.11.1.2, 7.11.1.4, and 7.11.1.5 of the full-text guidelines for additional discussion on imaging considerations.)

Class I

1. **Dipyridamole or adenosine stress perfusion nuclear scintigraphy or dobutamine echocardiography before or early after discharge should be used in patients with STEMI who are not undergoing cardiac catheterization to look for inducible ischemia in patients judged to be unable to exercise. (*Level of Evidence: B*)**

Class IIa

1. **Myocardial perfusion imaging or dobutamine echocardiography is reasonable in hemodynamically and electrically stable patients 4 to 10 days after STEMI to assess myocardial viability when required to define the potential efficacy of revascularization. (*Level of Evidence: C*)**

Recommended strategies for exercise test evaluations after STEMI are presented in Figure 6. These strategies and the data on which they are based are reviewed in more detail in the ACC/AHA 2002 Guideline Update for Exercise Testing.[170]

d. LV Function

Noninvasive imaging in patients recovering from STEMI includes echocardiography and radionuclide imaging. This section discusses the importance of measurement of LV function. Either of the above imaging techniques can provide clinically useful information.

Class I

1. **LVEF should be measured in all STEMI patients. (*Level of Evidence: B*)**

Assessment of LV function after STEMI has been shown to be one of the most accurate predictors of future cardiac events in both the prereperfusion[176] and the reperfusion eras.[177,178] Multiple techniques for assessing LV function of patients after STEMI have important prognostic value. Because of the dynamic nature of LV function recovery after STEMI, clinicians should consider the timing of the imaging study relative to the index event when assessing LV function. (See Table 6 of the ACC/AHA/ASE 2003 Guideline Update on the Clinical Application of Echocardiography for further discussion of the impact of timing on assessment of LV function and inducible ischemia.)[153]

e. Invasive Evaluation

Class I

1. **Coronary arteriography should be performed in patients with spontaneous episodes of myocardial ischemia or episodes of myocardial ischemia provoked by minimal exertion during recovery from STEMI. (*Level of Evidence: A*)**
2. **Coronary arteriography should be performed for intermediate- or high-risk findings on noninvasive testing after STEMI (see Table 23 of the ACC/AHA 2002 Guideline Update for the Management of Patients With Chronic Stable Angina).[179] (*Level of Evidence: B*)**
3. **Coronary arteriography should be performed if the patient is sufficiently stable before definitive therapy of a mechanical complication of STEMI, such as acute MR, VSR, pseudoaneurysm, or LV aneurysm. (*Level of Evidence: B*)**
4. **Coronary arteriography should be performed in patients with persistent hemodynamic instability. (*Level of Evidence: B*)**
5. **Coronary arteriography should be performed in survivors of STEMI who had clinical heart failure during the acute episode but subsequently demonstrated well-preserved LV function. (*Level of Evidence: C*)**

Class IIa

1. **It is reasonable to perform coronary arteriography when STEMI is suspected to have occurred by a mechanism other than thrombotic occlusion of an atherosclerotic plaque. This would include coronary embolism, certain metabolic or hematological diseases, or coronary artery spasm. (*Level of Evidence: C*)**
2. **Coronary arteriography is reasonable in STEMI patients with any of the following: diabetes mellitus, LVEF less than 0.40, CHF, prior revascularization, or life-threatening ventricular arrhythmias. (*Level of Evidence: C*)**

Class IIb

1. **Catheterization and revascularization may be considered as part of a strategy of routine coronary arteriography for risk assessment after fibrinolytic therapy (See Section 6.3.1.6.4.7 of the full-text guidelines). (*Level of Evidence: B*)**

Class III

1. **Coronary arteriography should not be performed in survivors of STEMI who are thought not to be candidates for coronary revascularization. (*Level of Evidence: A*)**

The Writing Committee encourages contemporary research into the benefit of routine catheterization versus watchful waiting after fibrinolytic therapy in the contemporary era.[180] (See Section 6.3.1.6.4.7 of the full-text guidelines)

f. Assessment of Ventricular Arrhythmias

Class IIb

1. **Noninvasive assessment of the risk of ventricular arrhythmias may be considered (including signal-averaged ECG, 24-hour ambulatory monitoring, heart rate variability, micro T-wave alternans, and T-wave variability) in patients recovering from STEMI. (*Level of Evidence: B*)**

The clinical applicability of these tests to the post-STEMI patient is in a state of evolution. Until these issues are resolved, use these tests are used only to support routine management and risk assessment.

L. Secondary Prevention

Class I

1. **Patients who survive the acute phase of STEMI should have plans initiated for secondary prevention therapies. (*Level of Evidence: A*)**

Secondary prevention therapies, unless contraindicated, are an essential part of the management of all patients with STEMI (Table 4),[181] regardless of sex.[182,183] Inasmuch as atherosclerotic vascular disease is frequently found in multiple vascular beds, the physician should search for symptoms or signs of peripheral vascular disease or cerebrovascular disease in patients presenting with STEMI.

1. Patient Education Before Discharge

Class I

1. **Before hospital discharge, all STEMI patients should be educated about and actively involved in planning for adherence to the lifestyle changes and drug therapies that are important for the secondary prevention of cardiovascular disease. (*Level of Evidence: B*)**
2. **Post-STEMI patients and their family members should receive discharge instructions about recognizing acute cardiac symptoms and appropriate actions to take in response (ie, calling 9-1-1 if symptoms are unimproved or worsening 5 minutes after onset, or if symptoms are unimproved or worsening 5 minutes after 1 sublingual nitroglycerin dose) to ensure early evaluation and treatment should symptoms recur. (*Level of Evidence: C*)**
3. **Family members of STEMI patients should be advised to learn about AEDs and CPR and be referred to a CPR training program. Ideally, such training programs would have a social support component targeting family members of high-risk patients. (*Level of Evidence: C*)**

2. Lipid Management

Class I

1. **Dietary therapy that is low in saturated fat and cholesterol (less than 7% of total calories as saturated fat and less than 200 mg/d cholesterol) should be started on discharge after recovery from STEMI. Increased consumption of the following should be encouraged: omega–3 fatty acids, fruits, vegetables, soluble (viscous) fiber, and whole grains. Calorie intake should be balanced with energy output to achieve and maintain a healthy weight. (*Level of Evidence: A*)**
2. **A lipid profile should be obtained from past records, but if not available, it should be performed in all patients with STEMI, preferably after they have fasted and within 24 hours of admission. (*Level of Evidence: C*)**
3. **The target LDL-C level after STEMI should be *substantially* less than 100 mg/dL. (*Level of Evidence: A*)**
   a. **Patients with LDL-C 100 mg/dl or above should be prescribed drug therapy on hospital discharge, with preference given to statins. (*Level of Evidence: A*)**
   b. **Patients with LDL-C less than 100 mg/dL or unknown LDL-C levels should be prescribed statin therapy on hospital discharge. (*Level of Evidence: B*)**
4. **Patients with non–high-density lipoprotein cholesterol (non HDL-C) levels less than 130 mg/dL who have an HDL-C level less than 40 mg/dL should receive special emphasis on nonpharmacological therapy (eg, exercise, weight loss, and smoking cessation) to increase HDL-C. (*Level of Evidence: B*)**

Class IIa

1. **It is reasonable to prescribe drug therapy at discharge to patients with non–HDL-C greater than or equal to 130 mg/dL, with a goal of reducing non–HDL-C to *substantially* less than 130 mg/dL. (*Level of Evidence: B*)**
2. **It is reasonable to prescribe drug therapy such as niacin or fibrate therapy to raise HDL-C levels in patients with LDL-C less than 100 mg/dL and non–HDL-C less than 130 mg/dL but HDL-C less than 40 mg/dL despite dietary and other nonpharmacological therapy. (*Level of Evidence: B*) Dietary-supplement niacin must not be used as a substitute for prescription niacin, and over-the-counter niacin should be used only if approved and monitored by a physician.**
3. **It is reasonable to add drug therapy with either niacin or a fibrate to diet regardless of LDL-C and HDL-C levels when triglyceride levels are greater than 500 mg/dL. In this setting, non–HDL-C (goal *substantially* less than 130 mg/dL) should be the cholesterol target rather than LDL-C. (*Level of Evidence: B*) Dietary-supplement niacin must not be used as a substitute for prescription niacin, and over-the-counter niacin should be used only if approved and monitored by a physician.**

Early secondary prevention trials conducted before the use of statin therapy, which used then-available drugs and diet to lower cholesterol, demonstrated significant reductions of 25% in nonfatal MIs and 14% in fatal MIs.[14] Subsequently, a growing body of evidence, mainly from large randomized clinical trials of statin therapy, has firmly established the desirability of lowering atherogenic serum lipids in patients who have recovered from a STEMI. See Table 4 for additional discussion of recommendations.

3. Weight Management

Class I

1. **Measurement of waist circumference and calculation of body mass index are recommended. Desirable body mass index range is 18.5 to 24.9 kg/m^2. A waist circumference greater than 40 inches in men and 35 inches in women would result in evaluation for metabolic syndrome and implementation of weight-reduction strategies. (*Level of Evidence: B*)**
2. **Patients should be advised about appropriate strategies for weight management and physical activity**

TABLE 4. Secondary Prevention for STEMI Patients

Goals	Intervention Recommendations
Smoking: Goal complete cessation	Assess tobacco use. Strongly encourage patient and family to stop smoking and to avoid secondhand smoke. Provide counseling, pharmacological therapy (including nicotine replacement and bupropion), and formal smoking cessation programs as appropriate.
Blood pressure control: Goal Less than 140/90 mm Hg or Less than 130/80 mm Hg if chronic kidney disease or diabetes	If blood pressure is 120/80 mm Hg or greater: • Initiate lifestyle modification (weight control, physical activity, alcohol moderation, moderate sodium restriction, and emphasis on fruits, vegetables, and low-fat dairy products) in all patients. If blood pressure is 140/90 mm Hg or greater or 130/80 mm Hg or greater for individuals with chronic kidney disease or diabetes: • Add blood pressure medications, emphasizing the use of beta-blockers and inhibition of the renin-angiotensin-aldosterone system.
Lipid management: (TG less than 200 mg/dL) Primary goal LDL-C *substantially* less than 100 mg/dL	Start dietary therapy in all patients (less than 7% of total calories as saturated fat and less than 200 mg/d cholesterol). Promote physical activity and weight management. Encourage increased consumption of omega-3 fatty acids. Assess fasting lipid profile in all patients, preferably within 24 hours of STEMI. Add drug therapy according to the following guide: LDL-C *substantially* less than 100 mg/dL (baseline or on-treatment): • Statins should be used to lower LDL-C. LDL-C greater than or equal to 100 mg/dL (baseline or on-treatment): • Intensify LDL-C–lowering therapy with drug treatment, giving preference to statins.
Lipid management: (TG 200 mg/dL or greater) Primary goal Non–HDL-C* *substantially* less than 130 mg/dL	If TG is greater than or equal to 150 mg/dL or HDL-C is less than 40 mg/dL: • Emphasize weight management and physical activity. Advise smoking cessation. If TG is 200 to 499 mg/dL: • After LDL-C–lowering therapy,† consider adding fibrate or niacin.‡ If TG is greater than or equal to 500 mg/dL: • Consider fibrate or niacin‡ before LDL-C–lowering therapy.† • Consider omega-3 fatty acids as adjunct for high TG.
Physical activity: Minimum goal 30 minutes 3 to 4 days per week; Optimal daily	Assess risk, preferably with exercise test, to guide prescription. Encourage minimum of 30 to 60 minutes of activity, preferably daily, or at least 3 or 4 times weekly (walking, jogging, cycling, or other aerobic activity) supplemented by an increase in daily lifestyle activities (eg, walking breaks at work, gardening, household work). Cardiac rehabilitation/secondary prevention programs, when available, are recommended for patients with STEMI, particularly those with multiple modifiable risk factors and/or those moderate- to high-risk patients in whom supervised exercise training is warranted.
Weight management: Goal BMI 18.5–24.9 kg/m^2 Waist circumference: Women: Less than 35 inches Men: Less than 40 inches	Calculate BMI and measure waist circumference as part of evaluation. Monitor response of BMI and waist circumference to therapy. Start weight management and physical activity as appropriate. Desirable BMI range is 18.5 to 24.9 kg/m^2. If waist circumference is greater than or equal to 35 inches in women or greater than or equal to 40 inches in men, initiate lifestyle changes and treatment strategies for metabolic syndrome.
Diabetes management: Goal HbA1c less than 7%	Appropriate hypoglycemic therapy to achieve near-normal fasting plasma glucose, as indicated by HbA1c. Treatment of other risks (eg, physical activity, weight management, blood pressure, and cholesterol management).
Antiplatelet agents/anticoagulants:	Start and continue indefinitely aspirin 75 to 162 mg/d if not contraindicated. Consider clopidogrel 75 mg/d or warfarin if aspirin is contraindicated. Manage warfarin to INR of 2.5 to 3.5 in post-STEMI patients when clinically indicated or for those not able to take aspirin or clopidogrel (Figure 7).
Renin-Angiotensin-Aldosterone System Blockers:	ACE inhibitors in all patients indefinitely; start, early in stable high-risk patients (anterior MI, previous MI, Killip class greater than or equal to II [S_3 gallop, rales, radiographic CHF], LVEF less than 0.40). ARBs in patients who are intolerant of ACE inhibitors and who have either clinical or radiological signs of heart failure or LVEF less than 0.40. Aldosterone blockade in patients without significant renal dysfunction§ or hyperkalemia who are already receiving therapeutic doses of an ACE inhibitor, have an LVEF less than or equal to 0.40, and have either diabetes or heart failure.
Beta-Blockers:	Start in all patients. Continue indefinitely. Observe usual contraindications.

BMI indicates body mass index; in, inches; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; BMI, body mass index; INR, international normalization ratio; ACE, angiotensin converting enzyme; CHF, congestive heart failure; LVEF, left ventricular ejection fraction; ARB, angiotensin receptor blocker and TG, triglycerides.

*Non–HDL cholesterol equals total cholesterol minus HDL cholesterol.

†Treat to a goal of non–HDL-C *substantially* less than 130 mg/dL.

‡Dietary-supplement niacin must not be used as a substitute for prescription niacin, and over-the-counter niacin should be used only if approved and monitored by a physician.

§Creatinine should be less than or equal to 2.5 mg/dL in men or less than or equal to 2.0 mg/dL in women.

||Potassium should be less than or equal to 5.0 mEq/L.

Modified with permission from Smith et al. *Circulation.* 2004;109:672–93.[181]

(usually accomplished in conjunction with cardiac rehabilitation). (*Level of Evidence: B*)
3. **A plan should be established to monitor the response of body mass index and waist circumference to therapy (usually accomplished in conjunction with cardiac rehabilitation). (*Level of Evidence: B*)**

4. Smoking Cessation

Class I

1. **Patients recovering from STEMI who have a history of cigarette smoking should be strongly encouraged to stop smoking and to avoid secondhand smoke. Counseling should be provided to the patient and family, along with pharmacological therapy (including nicotine replacement and bupropion) and formal smoking-cessation programs as appropriate. (*Level of Evidence: B*)**
2. **All STEMI patients should be assessed for a history of cigarette smoking. (*Level of Evidence: A*)**

5. Antiplatelet Therapy

Class I

1. **A daily dose of aspirin 75 to 162 mg orally should be given indefinitely to patients recovering from STEMI. (*Level of Evidence: A*)**
2. **If true aspirin allergy is present, preferably clopidogrel (75 mg orally per day) or, alternatively, ticlopidine (250 mg orally twice daily) should be substituted. (*Level of Evidence: C*)**
3. **If true aspirin allergy is present, warfarin therapy with a target INR of 2.5 to 3.5 is a useful alternative to clopidogrel in patients less than 75 years of age who are at low risk for bleeding and who can be monitored adequately for dose adjustment to maintain a target INR range. (*Level of Evidence: C*)**

Class III

1. **Ibuprofen should not be used because it blocks the antiplatelet effects of aspirin. (*Level of Evidence: C*)**

On the basis of 12 randomized trials in 18 788 patients with prior infarction, the Antiplatelet Trialists' Collaboration reported a 25% reduction in the risk of recurrent infarction, stroke, or vascular death in patients receiving prolonged antiplatelet therapy (36 fewer events for every 1000 patients treated).[31] No antiplatelet therapy has proved superior to aspirin in this population, and daily doses of aspirin between 80 and 325 mg appear to be effective.[184] The CAPRIE (Clopidogrel versus Aspirin in Patients at Risk of Ischemic Events) trial, which compared aspirin with clopidogrel in 19 185 patients at high risk for vascular events, demonstrated a modest but significant (8.6%, *P* equals 0.043) reduction in serious vascular events with clopidogrel compared with aspirin.[185] These data suggest clopidogrel as the best alternative to aspirin in patients with true aspirin allergy.

The use of warfarin therapy for secondary prevention of vascular events in patients after STEMI is discussed in Section 7.12.11 of the full-text guidelines. Large randomized trials have demonstrated that oral anticoagulants, when given in adequate doses, reduce the rates of adverse outcomes, at the cost of a small increase in hemorrhagic events.[186–188] In the Warfarin, Aspirin, Reinfarction Study (WARIS II), warfarin without aspirin in a dose intended to achieve an INR of 2.8 to 4.2 resulted in a significant reduction in a composite end point (death, nonfatal reinfarction, or thromboembolic stroke) compared with therapy with aspirin alone (16.7% versus 20.0%).[186] Warfarin therapy resulted in a small but significant increase in major, nonfatal bleeding compared with therapy with aspirin alone (0.62% versus 0.17% per year). Chronic therapy with warfarin after STEMI presents an alternative to clopidogrel in patients with aspirin allergy.

6. Inhibition of Renin-Angiotensin-Aldosterone-System

Class I

1. **An ACE inhibitor should be prescribed at discharge for all patients without contraindications after STEMI. (*Level of Evidence: A*)**
2. **Long-term aldosterone blockade should be prescribed for post-STEMI patients without significant renal dysfunction (creatinine should be less than or equal to 2.5 mg/dL in men and less than or equal to 2.0 mg/dL in women) or hyperkalemia (potassium should be less than or equal to 5.0 mEq/L) who are already receiving therapeutic doses of an ACE inhibitor, have an LVEF less than or equal to 0.40, and have either symptomatic heart failure or diabetes. (*Level of Evidence: A*)**
3. **An ARB should be prescribed at discharge in those STEMI patients who are intolerant of an ACE inhibitor and have either clinical or radiological signs of heart failure and LVEF less than 0.40. Valsartan and candesartan have established efficacy for this recommendation. (*Level of Evidence: B*)**

Class IIa

1. **In STEMI patients who tolerate ACE inhibitors, an ARB can be useful as an alternative to ACE inhibitors in the long-term management of STEMI patients, provided there are either clinical or radiological signs of heart failure or LVEF less than 0.40. Valsartan and candesartan have established efficacy for this recommendation. (*Level of Evidence: B*)**

Class IIb

1. **The combination of an ACE inhibitor and an ARB may be considered in the long-term management of STEMI patients with persistent symptomatic heart failure and LVEF less than 0.40. (*Level of Evidence: B*)**

The use of ACE inhibitors early in the acute phase of STEMI and in the hospital management phase has been described earlier.

Compelling evidence now supports the broad long-term use of ACE inhibitors after STEMI.[189,190] The results of the VALIANT study (Valsartan in Acute Myocardial Infarction Trial) evaluating valsartan are discussed in Section 7.4.3 of the full-text guidelines. The series of CHARM studies (Candesartan in Heart Failure Assessment in Reduction of Mortality), although focusing on the evaluation of candesartan in

patients with chronic heart failure, provides information that can be extrapolated to the long-term management of the STEMI patient, because 50% to 60% of the patients studied had ischemic heart disease as the cause of heart failure.[191–193]

Given the extensive randomized trial and routine clinical experience with ACE inhibitors, they remain the logical first agent for inhibition of the renin-angiotensin-aldosterone system in the long-term management of patients with STEMI.[150,194] The ARBs valsartan and candesartan should be administered over the long term to STEMI patients with symptomatic heart failure who are intolerant of ACE inhibitors. As described in Section 7.4.3 of the full-text guidelines, the choice between an ACE inhibitor and an ARB over the long term in patients who are tolerant of ACE inhibitors will vary with individual physician and patient preference, as well as cost and anticipated side-effect profile.[150,194]

The results of the most relevant clinical trials that tested combinations of ACE inhibitors and ARBs have been subtly different, but clinically relevant. Whereas the CHARM-Added[192] trial demonstrated a reduction in the combined end point of heart failure hospitalization and death over ACE inhibition alone, the VALIANT study[149] reported that the combination of captopril and valsartan was equivalent to either alone, but with a greater number of adverse effects. Thus, when combination ACE inhibition and angiotensin receptor blockade is considered necessary, the preferred ARB is candesartan. Although there is evidence that the combination of an ACE inhibitor and an aldosterone inhibitor is effective at reducing mortality and is well tolerated in patients with a serum creatinine level of 2.5 mg/dL or less and a serum potassium concentration of 5.0 mEq/L or less (see Section 7.4.3 of the full-text guidelines), much less experience exists with the combination of an ARB and aldosterone inhibitor (24% of 2028 patients in the CHARM-Alternative trial)[191] and the triple combination of an ACE inhibitor, ARB, and an aldosterone antagonist (17% of 2548 patients in the CHARM-Added trial).[192]

The combination of an ACE inhibitor and an ARB (valsartan 20 mg/d orally initially; titrated up to 160 mg orally twice per day, or candesartan 4 to 8 mg/d orally initially; titrated up to 32 mg/d orally) or an ACE inhibitor and an aldosterone inhibitor may be considered for the long-term management of STEMI patients with symptomatic heart failure and LVEF less than 0.40, provided the serum creatinine level is less than or equal to 2.5 mg/dL in men and less than or equal to 2.0 mg/dL in women and the serum potassium concentration is less than or equal to 5.0 mEq/L (See Sections 7.4.3 and 7.6.4 of the full-text guidelines.)

7. Beta-Blockers

Class I

1. **All patients after STEMI except those at low risk (normal or near-normal ventricular function, successful reperfusion, and absence of significant ventricular arrhythmias) and those with contraindications should receive beta-blocker therapy. Treatment should begin within a few days of the event, if not initiated acutely, and continue indefinitely. (*Level of Evidence: A*)**
2. **Patients with moderate or severe LV failure should receive beta-blocker therapy with a gradual titration scheme. (*Level of Evidence: B*)**

Class IIa

1. **It is reasonable to prescribe beta-blockers to low-risk patients after STEMI who have no contraindications to that class of medications. (*Level of Evidence: A*)**

The use of beta-blockers in the early phase of STEMI and in hospital management is reviewed in Sections 6.3.1.6 and 7.4.1 of the full-text guidelines. The benefits of beta-blocker therapy in patients without contraindications have been demonstrated with or without reperfusion, initiated early or later in the clinical course, and for all age groups. The benefits of beta-blocker therapy for secondary prevention are well established.[142,196] In patients with moderate or severe LV failure, beta-blocker therapy should be administered with a gradual titration scheme.[197] Long-term beta-blocker therapy should be administered to survivors of STEMI who have subsequently undergone revascularization, because there is evidence of a mortality benefit from their use despite revascularization with either CABG surgery or PCI.[198]

8. Blood Pressure Control

Class I

1. **Blood pressure should be treated with drug therapy to a target level of less than 140/90 mm Hg and to less than 130/80 mm Hg for patients with diabetes or chronic kidney disease. (*Level of Evidence: B*)**
2. **Lifestyle modification (weight control, dietary changes, physical activity, and sodium restriction) should be initiated in all patients with blood pressure greater than or equal to 120/80 mm Hg. (*Level of Evidence: B*)**

Class IIb

1. **A target blood pressure goal of 120/80 mm Hg for post-STEMI patients may be reasonable. (*Level of Evidence: C*)**

Class III

1. **Short-acting dihydropyridine calcium channel blocking agents should not be used for the treatment of hypertension. (*Level of Evidence: B*)**

The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC-7)[199] recommends that patients be treated after MI with ACE inhibitors, beta-blockers, and, if necessary, aldosterone antagonists to a target blood pressure of less than 140/90 mm Hg, or less than 130/80 mm Hg for those with chronic kidney disease or diabetes.[199] Most patients will require 2 or more drugs to reach this goal, and when the blood pressure is greater than 20/10 mm Hg above goal, 2 drugs should usually be used from the outset.

JNC-7 emphasizes the importance of lifestyle modifications for all patients with blood pressure of 120/80 mm Hg or

greater.[199] These modifications include weight reduction if overweight or obese, consumption of a diet rich in fruits and vegetables and low in total fat and saturated fat, and reduction of sodium to no more than 2.4 g/d.[199]

9. Diabetes Management

Class I

1. **Hypoglycemic therapy should be initiated to achieve HbA1c less than 7%. (*Level of Evidence: B*)**

Class III

1. **Thiazolidinediones should not be used in patients recovering from STEMI who have New York Heart Association class III or IV heart failure. (*Level of Evidence: B*)**

10. Hormone Therapy

Class III

1. **Hormone therapy with estrogen plus progestin should not be given de novo to postmenopausal women after STEMI for secondary prevention of coronary events. (*Level of Evidence: A*)**
2. **Postmenopausal women who are already taking estrogen plus progestin at the time of a STEMI should not continue hormone therapy. However, women who are beyond 1 to 2 years after initiation of hormone therapy who wish to continue hormone therapy for another compelling indication should weigh the risks and benefits, recognizing a greater risk of cardiovascular events. However, hormone therapy should not be continued while patients are on bedrest in the hospital. (*Level of Evidence: B*)**

On the basis of the Heart and Estrogen/progestin Replacement Study (HERS),[200] the Heart and Estrogen/progestin Replacement Study Follow-up (HERS-2),[201] and the Women's Health Initiative,[202] postmenopausal women should not receive combination estrogen and progestin therapy for primary or secondary prevention of CHD. It is recommended that the use of hormone therapy be discontinued in women who have STEMI.[200–202]

11. Warfarin Therapy

Class I

1. **Warfarin should be given to aspirin-allergic post-STEMI patients with indications for anticoagulation as follows:**
   a. **Without stent implanted (INR 2.5 to 3.5). (*Level of Evidence: B*)**
   b. **With stent implanted and clopidogrel 75 mg/d administered concurrently (INR 2.0 to 3.0). (*Level of Evidence: C*)**
2. **Warfarin (INR 2.5 to 3.5) is a useful alternative to clopidogrel in aspirin-allergic patients after STEMI who do not have a stent implanted. (*Level of Evidence: B*)**
3. **Warfarin (INR 2.0 to 3.0) should be prescribed for post-STEMI patients with either persistent or paroxysmal atrial fibrillation. (*Level of Evidence: A*)**
4. **In post-STEMI patients with LV thrombus noted on an imaging study, warfarin should be prescribed for at least 3 months (*Level of Evidence: B*) and indefinitely in patients without an increased risk of bleeding (*Level of Evidence: C*).**
5. **Warfarin alone (INR 2.5 to 3.5) or warfarin (INR 2.0 to 3.0) in combination with aspirin (75 to 162 mg) should be prescribed in post-STEMI patients who have no stent implanted and who have indications for anticoagulation. (*Level of Evidence: B*)**

Class IIa

1. **In post-STEMI patients less than 75 years of age without specific indications for anticoagulation who can have their level of anticoagulation monitored reliably, warfarin alone (INR 2.5 to 3.5) or warfarin (INR 2.0 to 3.0) in combination with aspirin (75 to 162 mg) can be useful for secondary prevention. (*Level of Evidence: B*)**
2. **It is reasonable to prescribe warfarin to post-STEMI patients with LV dysfunction and extensive regional wall-motion abnormalities. (*Level of Evidence: A*)**

Class IIb

1. **Warfarin may be considered in patients with severe LV dysfunction, with or without CHF. (*Level of Evidence: C*)**

The indications for long-term anticoagulation after STEMI remain controversial and are evolving. Although the use of warfarin has been demonstrated to be cost-effective compared with standard therapy without aspirin, the superior safety, efficacy and cost-effectiveness of aspirin has made it the antithrombotic agent of choice for secondary prevention[203] (Figure 7).

12. Physical Activity

Class I

1. **On the basis of assessment of risk, ideally with an exercise test to guide the prescription, all patients recovering from STEMI should be encouraged to exercise for a minimum of 30 minutes, preferably daily but at least 3 or 4 times per week (walking, jogging, cycling, or other aerobic activity), supplemented by an increase in daily lifestyle activities (eg, walking breaks at work, gardening, and household work). (*Level of Evidence: B*)**
2. **Cardiac rehabilitation/secondary prevention programs, when available, are recommended for patients with STEMI, particularly those with multiple modifiable risk factors and/or those moderate- to high-risk patients in whom supervised exercise training is warranted. (*Level of Evidence: C*)**

13. Antioxidants

Class III

1. **Antioxidant vitamins such as vitamin E and/or vitamin C supplements should not be prescribed to patients**

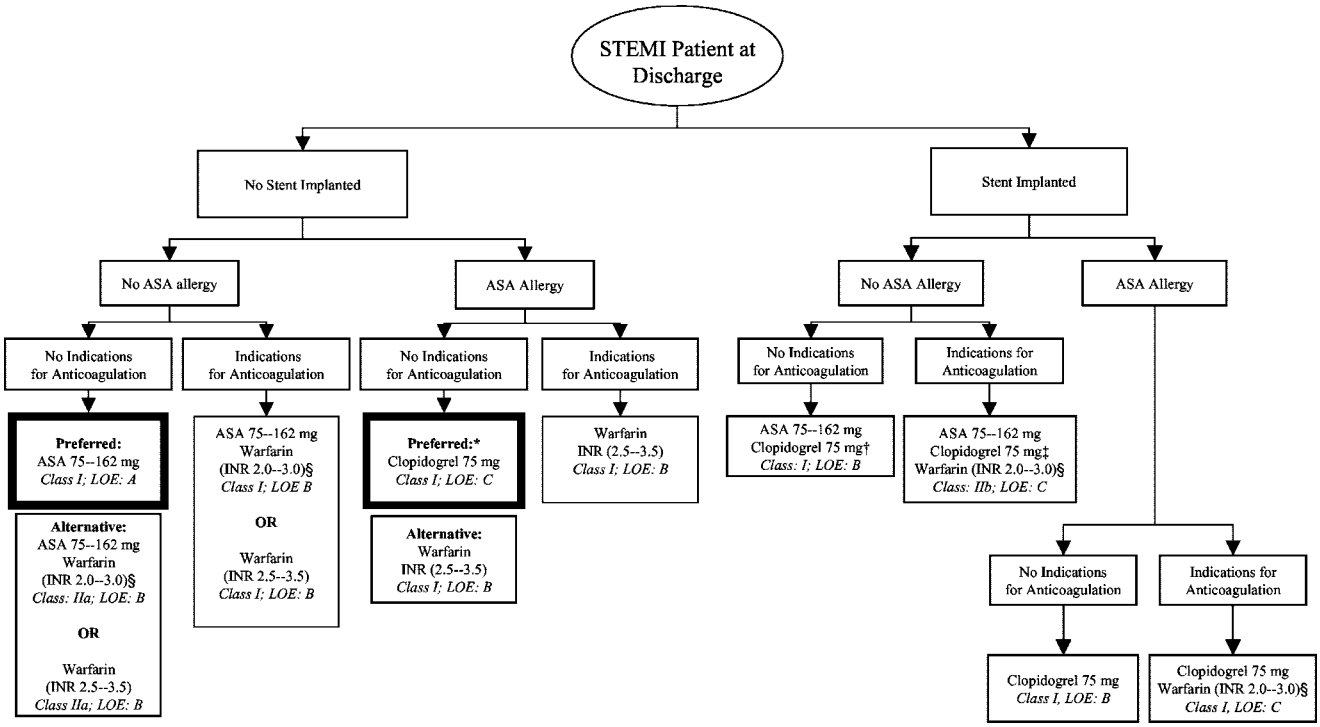

Figure 7. Long-term antithrombotic therapy at hospital discharge after STEMI. ASA indicates aspirin; LOE, level of evidence LV, left ventricular; and INR, international normalized ratio. *Clopidogrel is preferred over warfarin because of increased risk of bleeding and low patient compliance in warfarin trials. †For 12 months. ‡Discontinue clopidogrel 1 month after implantation of a bare metal stent or several months after implantation of a drug-eluting stent (3 months after sirolimus and 6 months after paclitaxel) because of the potential increased risk of bleeding with warfarin and 2 antiplatelet agents. Continue aspirin and warfarin long term if warfarin is indicated for other reasons such as atrial fibrillation, LV thrombus, cerebral emboli, or extensive regional wall-motion abnormality. §An INR of 2.0 to 3.0 is acceptable with tight control, but the lower end of this range is preferable. The combination of antiplatelet therapy and warfarin may be considered in patients aged less than 75 years with low bleeding risk who can be monitored reliably.

recovering from STEMI to prevent cardiovascular disease. (*Level of Evidence: A*)

There is no convincing evidence to support lipid- or water-soluble antioxidant supplementation in patients after STEMI or patients with or without established coronary disease.

VIII. Long-Term Management

A. Psychosocial Impact of STEMI

Class I

1. The psychosocial status of the patient should be evaluated, including inquiries regarding symptoms of depression, anxiety, or sleep disorders and the social support environment. (*Level of Evidence: C*)

Class IIa

1. Treatment with cognitive-behavioral therapy and selective serotonin reuptake inhibitors can be useful for STEMI patients with depression that occurs in the year after hospital discharge. (*Level of Evidence: A*)

Treatment of depression with combined cognitive-behavioral therapy and selective serotonin reuptake inhibitors improves outcome in terms of depression symptoms and social function.[204–206] It appears prudent to assess STEMI patients for depression during hospitalization and during the first month after STEMI and to intervene and reassess yearly in the first 5 years, as appropriate. There is evidence that the STEMI experience, with its sudden and unexpected onset, dramatic changes in lifestyle, and the additive effort of comorbid life events, is a relatively traumatic event and may produce impaired coping during subsequent ischemic events.[207]

B. Cardiac Rehabilitation

Class IIa

1. Cardiac rehabilitation/secondary prevention programs, when available, are recommended for patients with STEMI, particularly those with multiple modifiable risk factors and/or those moderate- to high-risk patients in whom supervised exercise training is warranted. (*Level of Evidence: C*)

C. Follow-Up Visit With Medical Provider

Class I

1. A follow-up visit should delineate the presence or absence of cardiovascular symptoms and functional class. (*Level of Evidence: C*)

2. **The patient's list of current medications should be reevaluated in a follow-up visit, and appropriate titration of ACE inhibitors, beta-blockers, and statins should be undertaken. (*Level of Evidence: C*)**
3. **The predischarge risk assessment and planned workup should be reviewed and continued (Figure 6). This should include a check of LV function and possibly Holter monitoring for those patients whose early post-STEMI ejection fraction was 0.31 to 0.40 or lower, in consideration of possible ICD use (Figure 5). (*Level of Evidence: C*)**
4. **The healthcare provider should review and emphasize the principles of secondary prevention with the patient and family members (Table 4).[181] (*Level of Evidence: C*)**
5. **The psychosocial status of the patient should be evaluated in follow-up, including inquiries regarding symptoms of depression, anxiety, or sleep disorders and the social support environment. (*Level of Evidence: C*)**
6. **In a follow-up visit, the healthcare provider should discuss in detail issues of physical activity, return to work, resumption of sexual activity, and travel, including driving and flying. The metabolic equivalent values for various activities are provided as a resource in Table 34 of the full-text guideline. (*Level of Evidence: C*)**
7. **Patients and their families should be asked if they are interested in CPR training after the patient is discharged from the hospital. (*Level of Evidence: C*)**
8. **Providers should actively review the following issues with patients and their families:**
   a. **The patient's heart attack risk. (*Level of Evidence: C*)**
   b. **How to recognize symptoms of STEMI. (*Level of Evidence: C*)**
   c. **The advisability of calling 9-1-1 if symptoms are unimproved or worsening after 5 minutes, despite feelings of uncertainty about the symptoms and fear of potential embarrassment. (*Level of Evidence: C*)**
   d. **A plan for appropriate recognition and response to a potential acute cardiac event, including the phone number to access EMS, generally 9-1-1.[15] (*Level of Evidence: C*)**
9. **Cardiac rehabilitation/secondary prevention programs, when available, are recommended for patients with STEMI, particularly those with multiple modifiable risk factors and/or those moderate- to high-risk patients in whom supervised exercise training is warranted. (*Level of Evidence: C*)**

References

1. Mehta RH, Montoye CK, Gallogly M, et al., for the GAP Steering Committee of the American College of Cardiology. Improving quality of care for acute myocardial infarction: The Guidelines Applied in Practice (GAP) Initiative. *JAMA*. 2002;287:1269–1276.
2. Jencks SF, Huff ED, Cuerdon T. Change in the quality of care delivered to medicare beneficiaries, 1998–1999 to 2000–2001. *JAMA*. 2003;289: 305–312.
3. Schneider EC, Zaslavsky AM, Epstein AM. Racial disparities in the quality of care for enrollees in medicare managed care. *JAMA*. 2002; 287:1288–1294.
4. Rogers WJ, Canto JG, Lambrew CT, et al. Temporal trends in the treatment of over 1.5 million patients with myocardial infarction in the US from 1990 through 1999: the National Registry of Myocardial Infarction 1, 2 and 3. *J Am Coll Cardiol*. 2000;36:2056–2063.
5. American Heart Association. *Heart Disease and Stroke Statistics—2004 Update*. Dallas, TX: American Heart Association 2003.

5a. Wiviott SD, Morrow DA, Giugliano RP, et al. Performance of the thrombolysis in myocardial infarction risk index for early acute coronary syndrome in the National Registry of Myocardial Infarction: a simple risk index predicts mortality in both ST and non-ST elevation myocardial infarction. *J Am Coll Cardiol* 2003;41:365A–366A.

6. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA*. 2001;285:2486–2497.
7. Levey AS, Coresh J, Balk E, et al. National Kidney Foundation practice guidelines for chronic kidney disease: evaluation, classification, and stratification. *Ann Intern Med*. 2003;139:137–147.
8. National, Heart, Lung, and Blood Institute. *Act in Time to Heart Attack*. Available at: http://:www.nhlbi.nih.gov/actintime, 2003. Accessed May 15, 2003.
9. American College of Cardiology. *Guidelines Applied in Practice*. Available at: http://www.acc.org/gap/gap.htm. Accessed May 15, 2003.
10. American Heart Association. *Get With the Guidelines*. Available at http://www.americanheart.org/presenter.jhtml?identifier=1165. Accessed May 15, 2003.
11. Topol EJ, Kereiakes DJ. Regionalization of care for acute ischemic heart disease: a call for specialized centers. *Circulation*. 2003;107:1463–1466.
12. Califf RM, Faxon DP. Need for centers to care for patients with acute coronary syndromes. *Circulation*. 2003;107:1467–1470.
13. Willerson JT. Editor's commentary: centers of excellence. *Circulation*. 2003;107:1471–1472.
14. National Cholesterol Education Program. *Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III)*. NIH publication No. 02–5125. Bethesda, Md: National Heart, Lung, and Blood Institute, 2002. Guidelines, Related Tools, and Patient Information, available at: http://www.nhlbi.nih.gov/guidelines/cholesterol/index.htm. Accessed April 12, 2003.
15. Dracup K, Alonzo AA, Atkins JM, et al. The physician's role in minimizing prehospital delay in patients at high risk for acute myocardial infarction: recommendations from the National Heart Attack Alert Program. Working Group on Educational Strategies to Prevent Prehospital Delay in Patients at High Risk for Acute Myocardial Infarction. *Ann Intern Med*. 1997;126:645–651.
16. Hedges JR, Feldman HA, Bittner V, et al, for the REACT Study Group. Impact of community intervention to reduce patient delay time on use of reperfusion therapy for acute myocardial infarction: Rapid Early Action for Coronary Treatment (REACT) trial. *Acad Emerg Med*. 2000;7: 862–872.
17. Canto JG, Zalenski RJ, Ornato JP, et al, for the National Registry of Myocardial Infarction 2 Investigators. Use of emergency medical services in acute myocardial infarction and subsequent quality of care: observations from the National Registry of Myocardial Infarction 2. *Circulation*. 2002;106:3018–3023.
18. Goldberg R, Goff D, Cooper L, et al. Age and sex differences in presentation of symptoms among patients with acute coronary disease: the Trial: Rapid Early Action for Coronary Treatment (REACT). *Coron Artery Dis*. 2000;11:399–407.
19. Hutchings CB, Mann NC, Daya M, et al, for the Rapid Early Action for Coronary Treatment Study. Patients with chest pain calling 9-1-1 or self-transporting to reach definitive care: which mode is quicker? *Am Heart J*. 2004;147:35–41.
20. Faxon D, Lenfant C. Timing is everything: motivating patients to call 9–1–1 at onset of acute myocardial infarction. *Circulation*. 2001;104: 1210–1211.
21. US Department of Health and Human Services, Public Health Service, National Institutes of Health, National Heart, Lung, and Blood Institute. NIH publication No. 01–3313. September 2001. Available at: http://www.nhlbi.nih.gov/health/prof/heart/mi/provider.pdf. Accessed December 16, 2002.
22. Eisenberg MJ, Topol EJ. Prehospital administration of aspirin in patients with unstable angina and acute myocardial infarction. *Arch Intern Med*. 1996;156:1506–1510.
23. Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. *Lancet*. 1994;343:311–322.
24. Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI). Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. *Lancet*. 1986;1:397–402.

25. Armstrong PW, Collen D, Antman E. Fibrinolysis for acute myocardial infarction: the future is here and now. *Circulation*. 2003;107: 2533–2537.
26. The American Heart Association in collaboration with the International Liaison Committee on Resuscitation. Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: Part 7: the era of reperfusion: section 1: acute coronary syndromes (acute myocardial infarction). *Circulation*. 2000;102(suppl I):I-172–I-203.
27. Pedley DK, Bissett K, Connolly EM, et al. Prospective observational cohort study of time saved by prehospital thrombolysis for ST elevation myocardial infarction delivered by paramedics. *BMJ*. 2003;327:22–26.
28. Menown IB, Mackenzie G, Adgey AA. Optimizing the initial 12-lead electrocardiographic diagnosis of acute myocardial infarction. *Eur Heart J*. 2000;21:275–283.
29. Case definitions for acute coronary heart disease in epidemiology and clinical research studies: a statement from the AHA Council on Epidemiology and Prevention; AHA Statistics Committee; World Heart Federation Council on Epidemiology and Prevention; the European Society of Cardiology Working Group on Epidemiology and Prevention; Centers for Disease Control and Prevention; and the National Heart, Lung, and Blood Institute. *Circulation*. 2003;108:2543–2549.
30. Come PC, Pitt B. Nitroglycerin-induced severe hypotension and bradycardia in patients with acute myocardial infarction. *Circulation*. 1976; 54:624–628.
31. Antithrombotic Trialists' Collaboration. Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. *BMJ*. 2002;324:71–86.
32. Sagar KA, Smyth MR. A comparative bioavailability study of different aspirin formulations using on-line multidimensional chromatography. *J Pharm Biomed Anal*. 1999;21:383–392.
33. De Luca G, Suryapranata H, Zijlstra F, et al, for the ZWOLLE Myocardial Infarction Study Group. Symptom-onset-to-balloon time and mortality in patients with acute myocardial infarction treated by primary angioplasty. *J Am Coll Cardiol*. 2003;42:991–997.
34. Boersma E, Mercado N, Poldermans D, et al. Acute myocardial infarction. *Lancet*. 2003;361:847–858.
35. De Luca G, Suryapranata H, Ottervanger JP, et al. Time delay to treatment and mortality in primary angioplasty for acute myocardial infarction: every minute of delay counts. *Circulation*. 2004;109: 1223–1225.
36. Boersma E, Maas AC, Deckers JW, et al. Early thrombolytic treatment in acute myocardial infarction: reappraisal of the golden hour. *Lancet*. 1996;348:771–775.
37. Zeymer U, Tebbe U, Essen R, et al, for the ALKK-Study Group. Influence of time to treatment on early infarct-related artery patency after different thrombolytic regimens. *Am Heart J*. 1999;137:34–38.
38. Weaver WD, Cerqueira M, Hallstrom AP, et al. Prehospital-initiated vs hospital-initiated thrombolytic therapy: the Myocardial Infarction Triage and Intervention Trial. *JAMA*. 1993;270:1211–1216.
39. Brodie BR, Stuckey TD, Wall TC, et al. Importance of time to reperfusion for 30-day and late survival and recovery of left ventricular function after primary angioplasty for acute myocardial infarction. *J Am Coll Cardiol*. 1998;32:1312–1319.
40. Brodie BR, Stuckey TD, Wall TC, et al. Importance of time to reperfusion on outcomes with primary coronary angioplasty for acute myocardial infarction (results from the Stent Primary Angioplasty in Myocardial Infarction Trial). *Am J Cardiol*. 2001;88:1085–1090.
41. Williams DO. Treatment delayed is treatment denied. *Circulation*. 2004; 109:1806-1808.
42. The Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology. Management of acute myocardial infarction in patients presenting with ST-segment elevation. *Eur Heart J*. 2003;24:28–66.
43. Morrow DA, Antman EM, Charlesworth A, et al. TIMI risk score for ST-elevation myocardial infarction: a convenient, bedside, clinical score for risk assessment at presentation: an intravenous nPA for treatment of infarcting myocardium early II trial substudy. *Circulation*. 2000;102: 2031–2037.
44. Lee KL, Woodlief LH, Topol EJ, et al, for the GUSTO-I Investigators. Predictors of 30-day mortality in the era of reperfusion for acute myocardial infarction: results from an international trial of 41,021 patients. *Circulation*. 1995;91:1659–1668.
45. Morrow DA, Antman EM, Giugliano RP, et al. A simple risk index for rapid initial triage of patients with ST-elevation myocardial infarction: an InTIME II substudy. *Lancet*. 2001;358:1571–1575.
46. Krumholz HM, Chen J, Wang Y, et al. Comparing AMI mortality among hospitals in patients 65 years of age and older: evaluating methods of risk adjustment. *Circulation*. 1999;99:2986–2992.
47. Granger CB, Goldberg RJ, Dabbous O, et al, for the Global Registry of Acute Coronary Events Investigators. Predictors of hospital mortality in the Global Registry of Acute Coronary Events. *Arch Intern Med*. 2003; 163:2345–2353.
48. Magid DJ, Calonge BN, Rumsfeld JS, et al, for the National Registry of Myocardial Infarction 2 and 3 Investigators. Relation between hospital primary angioplasty volume and mortality for patients with acute MI treated with primary angioplasty vs thrombolytic therapy. *JAMA*. 2000; 284:3131–3138.
49. Nallamothu BK, Bates ER. Percutaneous coronary intervention versus fibrinolytic therapy in acute myocardial infarction: is timing (almost) everything? *Am J Cardiol*. 2003;92:824–826.
50. Bonnefoy E, Lapostolle F, Leizorovicz A, et al., for the Comparison of Angioplasty and Prehospital Thrombolysis in Acute Myocardial Infarction study group. Primary angioplasty versus prehospital fibrinolysis in acute myocardial infarction: a randomised study. *Lancet*. 2002; 360:825–829.
51. Steg PG, Bonnefoy E, Chabaud S, et al. Impact of time to treatment on mortality after prehospital fibrinolysis or primary angioplasty: data from the CAPTIM randomized clinical trial. *Circulation*. 2003;108:2851–2856.
52. Widimsky P, Budesinsky T, Vorac D, et al. Long distance transport for primary angioplasty vs immediate thrombolysis in acute myocardial infarction: final results of the randomized national multicentre trial: PRAGUE-2. *Eur Heart J*. 2003;24:94–104.
53. Deleted in press.
54. Antman EM. General hospital management. In: Julian DG, Braunwald E, eds. *Management of Acute Myocardial Infarction*. London, England: WB Saunders 1994;42–44.

54a. Lincoff AM, Califf RM, Van de Werf F, et al, for the Global Use of Strategies To Open Coronary Arteries (GUSTO) Investigators. Mortality at 1 year with combination platelet glycoprotein IIb/IIIa inhibition and reduced-dose fibrinolytic therapy vs conventional fibrinolytic therapy for acute myocardial infarction: GUSTO V randomized trial. *JAMA*. 2002;288:2130-2135.

55. Zijlstra F, Beukema WP, van't Hof AW, et al. Randomized comparison of primary coronary angioplasty with thrombolytic therapy in low risk patients with acute myocardial infarction. *J Am Coll Cardiol*. 1997;29:908–912.
56. Ribeiro EE, Silva LA, Carneiro R, et al. Randomized trial of direct coronary angioplasty versus intravenous streptokinase in acute myocardial infarction. *J Am Coll Cardiol*. 1993;22:376–380.
57. Grinfield L, Berrocal D, Bellardi J, et al. Fibrinolytics versus primary angioplasty in acute myocardial infarction (FAP): a randomized trial in a community hospital in Argentina. *J Am Coll Cardiol*. 1996;27:A222. Abstract.
58. Zijlstra F, de Boer MJ, Hoorntje JC, et al. A comparison of immediate coronary angioplasty with intravenous streptokinase in acute myocardial infarction. *N Engl J Med*. 1993;328:680–684.
59. Widimsky P, Groch L, Zelízko M, et al. Multicentre randomized trial comparing transport to primary angioplasty vs immediate thrombolysis vs combined strategy for patients with acute myocardial infarction presenting to a community hospital without a catheterization laboratory: the PRAGUE study. *Eur Heart J*. 2000;21:823–831.
60. de Boer MJ, Ottervanger JP, van't Hof AW, et al, for the Zwolle Myocardial Infarction Study Group. Reperfusion therapy in elderly patients with acute myocardial infarction: a randomized comparison of primary angioplasty and thrombolytic therapy. *J Am Coll Cardiol*. 2002; 39:1723–1728.
61. Akhras F, Ousa AA, Swann G, et al. Primary coronary angioplasty or intravenous thrombolysis for patients with acute myocardial infarction? Acute and late follow-up results in a new cardiac unit. *J Am Coll Cardiol*. 1997;29:A235–A236. Abstract.
62. Deleted in press.
63. Grines CL, Browne KF, Marco J, et al, for the Primary Angioplasty in Myocardial Infarction Study Group. A comparison of immediate angioplasty with thrombolytic therapy for acute myocardial infarction. *N Engl J Med*. 1993;328:673–679.
64. Gibbons RJ, Holmes DR, Reeder GS, et al, for the Mayo Coronary Care Unit and Catheterization Laboratory Groups. Immediate angioplasty compared with the administration of a thrombolytic agent followed by conservative treatment for myocardial infarction. *N Engl J Med*. 1993; 328:685–691.

65. Ribichini F, Steffenino G, Dellavalle A, et al. Comparison of thrombolytic therapy and primary coronary angioplasty with liberal stenting for inferior myocardial infarction with precordial ST-segment depression: immediate and long-term results of a randomized study. *J Am Coll Cardiol*. 1998;32:1687–1694.
66. García E, Elízaga J, Pérez-Castellano N, et al. Primary angioplasty versus systemic thrombolysis in anterior myocardial infarction. *J Am Coll Cardiol*. 1999;33:605–611.
67. The Global Use of Strategies to Open Occluded Coronary Arteries in Acute Coronary Syndromes (GUSTO IIb) Angioplasty Substudy Investigators. A clinical trial comparing primary coronary angioplasty with tissue plasminogen activator for acute myocardial infarction. *N Engl J Med*. 1997;336:1621–1628.
68. Le May MR, Labinaz M, Davies RF, et al. Stenting versus thrombolysis in acute myocardial infarction trial (STAT). *J Am Coll Cardiol*. 2001; 37:985–991.
69. Schömig A, Kastrati A, Dirschinger J, et al, for the Stent versus Thrombolysis for Occluded Coronary Arteries in Patients with Acute Myocardial Infarction Study Investigators. Coronary stenting plus platelet glycoprotein IIb/IIIa blockade compared with tissue plasminogen activator in acute myocardial infarction. *N Engl J Med*. 2000;343:385–391.
70. Vermeer F, Oude Ophuis AJ, vd Berg EJ et al. Prospective randomised comparison between thrombolysis, rescue PTCA, and primary PTCA in patients with extensive myocardial infarction admitted to a hospital without PTCA facilities: a safety and feasibility study. *Heart*. 1999;82: 426–431.
71. Kastrati A, Mehilli J, Dirschinger J, et al, for the Stent versus Thrombolysis for Occluded Coronary Arteries in Patients with Acute Myocardial Infarction (STOPAMI-2) Study. Myocardial salvage after coronary stenting plus abciximab versus fibrinolysis plus abciximab in patients with acute myocardial infarction: a randomised trial. *Lancet*. 2002;359:920–925.
72. Andersen HR, Nielsen TT, Rasmussen K, et al, for the DANAMI-2 Investigators. Thrombolytic therapy vs primary percutaneous coronary intervention for myocardial infarction in patients presenting to hospitals without on-site cardiac surgery: a randomized controlled trial. *JAMA*. 2002;287:1943–1951.
73. Kastrati A, Mehilli J, Dirschinger J, et al, for the Stent versus Thrombolysis for Occluded Coronary Arteries in Patients With Acute Myocardial Infarction (STOPAMI-2) Study. A randomized trial of transfer for primary angioplasty versus on-site thrombolysis in patients with high-risk myocardial infarction: the Air Primary Angioplasty in Myocardial Infarction study. *J Am Coll Cardiol*. 2002;39:1713–1719.
74. Andersen HR, Nielsen TT, Rasmussen K, et al, for the DANAMI-2 Investigators. A comparison of coronary angioplasty with fibrinolytic therapy in acute myocardial infarction. *N Engl J Med*. 2003;349: 733–742.
75. Hochman JS, Sleeper LA, Webb JG, et al, for the Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock (SHOCK) Investigators. Early revascularization in acute myocardial infarction complicated by cardiogenic shock. *N Engl J Med*. 1999;341: 625–634.
76. Keeley EC, Boura JA, Grines CL. Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials. *Lancet*. 2003;361:13–20.
76a. Melandri G. The obsession with primary angioplasty. *Circulation*. 2003; 108:e1620.
77. Rogers WJ, Dean LS, Moore PB, et al, for the Alabama Registry of Myocardial Ischemia Investigators. Comparison of primary angioplasty versus thrombolytic therapy for acute myocardial infarction. *Am J Cardiol*. 1994;74:111–118.
78. Every NR, Parsons LS, Hlatky M, et al, for the Myocardial Infarction Triage and Intervention Investigators. A comparison of thrombolytic therapy with primary coronary angioplasty for acute myocardial infarction. *N Engl J Med*. 1996;335:1253–1260.
79. Tiefenbrunn AJ, Chandra NC, French WJ, et al. Clinical experience with primary percutaneous transluminal coronary angioplasty compared with alteplase (recombinant tissue-type plasminogen activator) in patients with acute myocardial infarction: a report from the Second National Registry of Myocardial Infarction (NRMI-2). *J Am Coll Cardiol*. 1998; 31:1240–1245.
80. Danchin N, Vaur L, Genès N, et al. Treatment of acute myocardial infarction by primary coronary angioplasty or intravenous thrombolysis in the "real world": one-year results from a nationwide French survey. *Circulation*. 1999;99:2639–2644.
81. Berger PB, Ellis SG, Holmes DR, et al. Relationship between delay in performing direct coronary angioplasty and early clinical outcome in patients with acute myocardial infarction: results from the Global Use of Strategies To Open Occluded Arteries in Acute Coronary Syndromes (GUSTO-IIb) trial. *Circulation*. 1999;100:14–20.
82. Cannon CP, Gibson CM, Lambrew CT, et al. Relationship of symptom-onset-to-balloon time and door-to-balloon time with mortality in patients undergoing angioplasty for acute myocardial infarction. *JAMA*. 2000; 283:2941–2947.
83. Brodie BR, Stuckey TD, Hansen C, et al. Benefit of coronary reperfusion before intervention on outcomes after primary angioplasty for acute myocardial infarction. *Am J Cardiol*. 2000;85:13–18.
84. Stone GW, Cox D, Garcia E, et al. Normal flow (TIMI-3) before mechanical reperfusion therapy is an independent determinant of survival in acute myocardial infarction: analysis from the primary angioplasty in myocardial infarction trials. *Circulation*. 2001;104:636–641.
85. Clements IP, Christian TF, Higano ST, et al. Residual flow to the infarct zone as a determinant of infarct size after direct angioplasty. *Circulation*. 1993;88:1527–1533.
86. Juliard JM, Feldman LJ, Golmard JL, et al. Relation of mortality of primary angioplasty during acute myocardial infarction to door-to-Thrombolysis In Myocardial Infarction (TIMI) time. *Am J Cardiol*. 2003;91:1401–1405.
87. Cragg DR, Friedman HZ, Bonema JD, et al. Outcome of patients with acute myocardial infarction who are ineligible for thrombolytic therapy. *Ann Intern Med*. 1991;115:173–177.
88. Brodie BR, Weintraub RA, Stuckey TD, et al. Outcomes of direct coronary angioplasty for acute myocardial infarction in candidates and non-candidates for thrombolytic therapy. *Am J Cardiol*. 1991;67:7–12.
89. Himbert D, Juliard JM, Steg PG, et al. Primary coronary angioplasty for acute myocardial infarction with contraindication to thrombolysis. *Am J Cardiol*. 1993;71:377–381.
90. Zahn R, Schuster S, Schiele R, et al, for the Maximal Individual Therapy in Acute Myocardial Infarction (MITRA) Study Group. Comparison of primary angioplasty with conservative therapy in patients with acute myocardial infarction and contraindications for thrombolytic therapy. *Cathet Cardiovasc Interv*. 1999;46:127–133.
91. Grzybowski M, Clements EA, Parsons L, et al. Mortality benefit of immediate revascularization of acute ST-segment elevation myocardial infarction in patients with contraindications to thrombolytic therapy: a propensity analysis. *JAMA*. 2003;290:1891–1898.
92. Lotfi M, Mackie K, Dzavik V, et al. Impact of delays to cardiac surgery after failed angioplasty and stenting. *J Am Coll Cardiol*. 2004;43: 337–342.
93. Dehmer GJ, Gantt DS. Coronary intervention at hospitals without on-site cardiac surgery: are we pushing the envelope too far? *J Am Coll Cardiol*. 2004;43:343–345.
94. Antoniucci D, Santoro GM, Bolognese L, et al. A clinical trial comparing primary stenting of the infarct-related artery with optimal primary angioplasty for acute myocardial infarction: results from the Florence Randomized Elective Stenting in Acute Coronary Occlusions (FRESCO) trial. *J Am Coll Cardiol*. 1998;31:1234–1239.
95. Rodríguez A, Bernardi V, Fernández M, et al. In-hospital and late results of coronary stents versus conventional balloon angioplasty in acute myocardial infarction: Gianturco-Roubin in Acute Myocardial Infarction (GRAMI trial). *Am J Cardiol*. 1998;81:1286–1291.
96. Suryapranata H, Ottervanger JP, Nibbering E, et al. Long term outcome and cost-effectiveness of stenting versus balloon angioplasty for acute myocardial infarction. *Heart*. 2001;85:667–671.
97. Saito S, Hosokawa G, Tanaka S, et al, for the PASTA Trial Investigators. Primary stent implantation is superior to balloon angioplasty in acute myocardial infarction: final results of the primary angioplasty versus stent implantation in acute myocardial infarction (PASTA) trial. *Cathet Cardiovasc Interv*. 1999;48:262–268.
98. Grines CL, Cox DA, Stone GW, et al, for the Stent Primary Angioplasty in Myocardial Infarction Study Group. Coronary angioplasty with or without stent implantation for acute myocardial infarction. *N Engl J Med*. 1999;341:1949–1956.
99. Kawashima A, Ueda K, Nishida Y, et al. Quantitative angiographic analysis of restenosis of primary stenting using Wiktor stent for acute myocardial infarction: results from a multicenter randomized PRISAM study. *Circulation*. 1999;100(suppl I):I-856. Abstract.
100. Maillard L, Hamon M, Khalife K, et al, for the STENTIM-2 Investigators. A comparison of systematic stenting and conventional balloon angioplasty during primary percutaneous transluminal coronary angio-

plasty for acute myocardial infarction. *J Am Coll Cardiol*. 2000;35:1729–1736.
101. Scheller B, Hennen B, Severin-Kneib S, et al. Long-term follow-up of a randomized study of primary stenting versus angioplasty in acute myocardial infarction. *Am J Med*. 2001;110:1–6.
102. Zhu MM, Feit A, Chadow H, et al. Primary stent implantation compared with primary balloon angioplasty for acute myocardial infarction: a meta-analysis of randomized clinical trials. *Am J Cardiol*. 2001;88:297–301.
103. Stone GW, Grines CL, Cox DA, et al, for the Controlled Abciximab and Device Investigation to Lower Late Angioplasty Complications (CADILLAC) Investigators. Comparison of angioplasty with stenting, with or without abciximab, in acute myocardial infarction. *N Engl J Med*. 2002;346:957–966.
104. Lemos PA, Saia F, Hofma SH, et al. Short- and long-term clinical benefit of sirolimus-eluting stents compared to conventional bare stents for patients with acute myocardial infarction. *J Am Coll Cardiol*. 2004;43:704–708.
104a. Ellis SG, Armstrong P, Betriu A, et al. Facilitated percutaneous coronary intervention versus primary percutaneous coronary intervention: design and rationale of the Facilitated Intervention with Enhanced Reperfusion Speed to Stop Events (FINESSE) trial. *Am Heart J*. 2004;147:E16.
105. The TIMI Research Group. Immediate vs delayed catheterization and angioplasty following thrombolytic therapy for acute myocardial infarction: TIMI II A results. *JAMA*. 1988;260:2849–2858.
106. Topol EJ, Califf RM, George BS, et al. A randomized trial of immediate versus delayed elective angioplasty after intravenous tissue plasminogen activator in acute myocardial infarction. *N Engl J Med*. 1987;317:581–588.
107. Sadanandan S, Buller C, Menon V, et al. The late open artery hypothesis: a decade later. *Am Heart J*. 2001;142:411–421.
108. Hochman JS, Sleeper LA, White HD, et al, for the SHOCK Investigators: Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock: one-year survival following early revascularization for cardiogenic shock. *JAMA*. 2001;285:190–192.
109. Dzavik V, Sleeper LA, Cocke TP, et al, for the SHOCK Investigators. Early revascularization is associated with improved survival in elderly patients with acute myocardial infarction complicated by cardiogenic shock: a report from the SHOCK Trial Registry. *Eur Heart J*. 2003;24:828–837.
110. Dauerman HL, Goldberg RJ, Malinski M, et al. Outcomes and early revascularization for patients greater than or equal to 65 years of age with cardiogenic shock. *Am J Cardiol*. 2001;87:844–848.
111. Dauerman HL, Ryan TJ, Piper WD, et al. Outcomes of percutaneous coronary intervention among elderly patients in cardiogenic shock: a multicenter, decade-long experience. *J Invasive Cardiol*. 2003;15:380–384.
112. Simoons ML, Arnold AE, Betriu A, et al. Thrombolysis with tissue plasminogen activator in acute myocardial infarction: no additional benefit from immediate percutaneous coronary angioplasty. *Lancet*. 1988;1:197–203.
113. Ross AM, Coyne KS, Reiner JS, et al, for the PACT (Plasminogen-activator Angioplasty Compatibility Trial). Investigators: A randomized trial comparing primary angioplasty with a strategy of short-acting thrombolysis and immediate planned rescue angioplasty in acute myocardial infarction: the PACT trial. *J Am Coll Cardiol*. 1999;34:1954–1962.
114. Montalescot G, Barragan P, Wittenberg O, et al, for the ADMIRAL (Abciximab before Direct Angioplasty and Stenting in Myocardial Infarction Regarding Acute and Long-Term Follow-up). Investigators. Platelet glycoprotein IIb/IIIa inhibition with coronary stenting for acute myocardial infarction. *N Engl J Med*. 2001;344:1895–1903.
115. Zeymer U, Uebis R, Vogt A, et al, for the ALKK-Study Group. Randomized comparison of percutaneous transluminal coronary angioplasty and medical therapy in stable survivors of acute myocardial infarction with single-vessel disease: a study of the Arbeitsgemeinschaft Leitende Kardiologische Krankenhausärzte. *Circulation*. 2003;108:1324–1328.
116. Scheller B, Hennen B, Hammer B, et al, for the SIAM III Study Group. Beneficial effects of immediate stenting after thrombolysis in acute myocardial infarction. *J Am Coll Cardiol*. 2003;42:634–641.
117. Braunwald E, Antman E, Beasley J, et al. ACC/AHA 2002 guideline update for the management of patients with unstable angina and non–ST-segment elevation myocardial infarction: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients With Unstable Angina). *J Am Coll Cardiol*. 2002;40:1366.
118. Stenestrand U, Wallentin L. Early revascularisation and 1-year survival in 14-day survivors of acute myocardial infarction: a prospective cohort study. *Lancet*. 2002;359:1805–1811.
119. Alter DA, Tu JV, Austin PC, et al. Waiting times, revascularization modality, and outcomes after acute myocardial infarction at hospitals with and without on-site revascularization facilities in Canada. *J Am Coll Cardiol*. 2003;42:410–419.
120. Gupta M, Chang WC, Van de Werf F, et al, for the ASSENT II Investigators. International differences in in-hospital revascularization and outcomes following acute myocardial infarction: a multilevel analysis of patients in ASSENT-2. *Eur Heart J*. 2003;24:1640–1650.
121. Gibson CM, Karha J, Murphy SA, et al, for the TIMI Study Group. Early and long-term clinical outcomes associated with reinfarction following fibrinolytic administration in the thrombolysis in myocardial infarction trials. *J Am Coll Cardiol*. 2003;42:7–16.
122. Lablanche JM, Gueret P, Blanchard D, et al. Beneficial effects of very early PTCA in patients with intravenous thrombolysis for acute myocardial infarction: data from a nation-wide registry in France. *Circulation*. 2002;106(suppl II):II-630. Abstract.
123. Fernandez-Aviles F, Alonso JJ, Castro-Beiras A, et al. Prospective randomized trial comparing a routine invasive strategy within 24 hours to thrombolysis versus an ischemia-guided conservative approach to acute myocardial infarction with ST-segment elevation: the Gracia-1 trial. *Lancet*. 2004. In press.
124. ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. Randomised trial of intravenous streptokinase, oral aspirin, both, or neither among 17,187 cases of suspected acute myocardial infarction: ISIS-2. *Lancet*. 1988;2:349–360.
125. Granger CB, Hirsch J, Califf RM, et al. Activated partial thromboplastin time and outcome after thrombolytic therapy for acute myocardial infarction: results from the GUSTO-I trial. *Circulation*. 1996;93:870–878.
126. White HD, Yusuf S. Issues regarding the use of heparin following streptokinase therapy. *J Thromb Thrombolysis*. 1995;2:5–10.
127. Thrombin-specific anticoagulation with bivalirudin versus heparin in patients receiving fibrinolytic therapy for acute myocardial infarction: the HERO-2 randomised trial. *Lancet*. 2001;358:1855–1863.
128. Bertrand ME, Rupprecht HJ, Urban P, et al. Double-blind study of the safety of clopidogrel with and without a loading dose in combination with aspirin compared with ticlopidine in combination with aspirin after coronary stenting : the Clopidogrel Aspirin Stent International Cooperative Study (CLASSICS). *Circulation*. 2000;102:624–629.
129. Mehta SR, Yusuf S, Peters RJ, et al, for the Clopidogrel in Unstable angina to prevent Recurrent Events trial (CURE) Investigators. Effects of pretreatment with clopidogrel and aspirin followed by long-term therapy in patients undergoing percutaneous coronary intervention: the PCI-CURE study. *Lancet*. 2001;358:527–533.
130. Steinhubl SR, Berger PB, Mann JT III, et al, for the CREDO (Clopidogrel for the Reduction of Events During Observation) Investigators. Early and sustained dual oral antiplatelet therapy following percutaneous coronary intervention: a randomized controlled trial. *JAMA*. 2002;288:2411–2420.
131. CYPHER™ sirolimus-eluting coronary stent on RAPTORTM over-the-wire delivery system and CYPHER™ sirolimus-eluting coronary stent on RAPTORRAIL® rapid exchange delivery system [instructions for use]. Miami, FL; Cordis, a Johnson & Johnson Company; April 2003.
132. TAXUS: paclitaxel-eluting coronary stent system Monorail and over the wire coronary stent delivery system [directions for use]. Natick, MA: Boston Scientific; March 2004.
133. Patrono C, Bachmann F, Baigent C, et al. Expert consensus document on the use of antiplatelet agents: The Task Force on the Use of Antiplatelet Agents in Patients With Atherosclerotic Cardiovascular Disease of the European Society of Cardiology. *Eur Heart J*. 2004;25:166–181.
134. Levine GN, Kern MJ, Berger PB, et al, for the American Heart Association Diagnostic and Interventional Catheterization Committee and Council on Clinical Cardiology. Management of patients undergoing percutaneous coronary revascularization. *Ann Intern Med*. 2003;139:123–136.
135. Lee DP, Herity NA, Hiatt BL, et al, for the TIrofiban Given in the Emergency Room before Primary Angioplasty. Adjunctive platelet glycoprotein IIb/IIIa receptor inhibition with tirofiban before primary angioplasty improves angiographic outcomes: results of the TIrofiban Given in the Emergency Room before Primary Angioplasty (TIGER-PA) pilot trial. *Circulation*. 2003;107:1497–1501.
136. Finney SJ, Zekveld C, Elia A, et al. Glucose control and mortality in critically ill patients. *JAMA*. 2003;290:2041–2047.

136a. Clement S, Braithwaite SS, Magee MF, et al. Management of diabetes and hyperglycemia in hospitals. *Diabetes Care*. 2004;27:553-597.
137. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106:3143–3421.
138. Malan SS. Psychosocial adjustment following MI: current views and nursing implications. *J Cardiovasc Nurs*. 1992;6:57–70.
139. Havik OE, Maeland JG. Patterns of emotional reactions after a myocardial infarction. *J Psychosom Res*. 1990;34:271–285.
140. Moser DK, Dracup K. Is anxiety early after myocardial infarction associated with subsequent ischemic and arrhythmic events? *Psychosom Med*. 1996;58:395–401.
141. Frasure-Smith N, Lespérance F, Talajic M. The impact of negative emotions on prognosis following myocardial infarction: is it more than depression? *Health Psychol*. 1995;14:388–398.
142. Chae Cu, Hennekens CH. Beta blockers. In: Hennekens CH, ed. *Clinical Trials in Cardiovascular Disease: A Companion to Braunwald's Heart Disease.* Philadelphia, Pa: WB Saunders 1999;79–94.
143. Latini R, Maggioni AP, Flather M, et al. ACE inhibitor use in patients with myocardial infarction: summary of evidence from clinical trials. *Circulation*. 1995;92:3132–3137.
144. ACE Inhibitor Myocardial Infarction Collaborative Group. Indications for ACE inhibitors in the early treatment of acute myocardial infarction: systematic overview of individual data from 100,000 patients in randomized trials. *Circulation*. 1998;97:2202–2212.
145. Flather MD, Yusuf S, Køber L, et al, for the ACE-Inhibitor Myocardial Infarction Collaborative Group. Long-term ACE-inhibitor therapy in patients with heart failure or left-ventricular dysfunction: a systematic overview of data from individual patients. *Lancet*. 2000;355:1575–1581.
146. Pitt B, Zannad F, Remme WJ, et al, for the Randomized Aldactone Evaluation Study Investigators. The effect of spironolactone on morbidity and mortality in patients with severe heart failure. *N Engl J Med*. 1999;341:709–717.
147. Pitt B, Remme W, Zannad F, et al, for the Eplerenone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study Investigators. Eplerenone, a selective aldosterone blocker, in patients with left ventricular dysfunction after myocardial infarction. *N Engl J Med*. 2003;348:1309–1321.
148. Dickstein K, Kjekshus J, for the OPTIMAAL Steering Committee of the Optimal Trial in Myocardial Infarction with Angiotensin II Antagonist Losartan (OPTIMAAL) Study Group. Effects of losartan and captopril on mortality and morbidity in high-risk patients after acute myocardial infarction: the OPTIMAAL randomised trial: Optimal Trial in Myocardial Infarction with Angiotensin II Antagonist Losartan. *Lancet*. 2002;360:752–760.
149. Pfeffer MA, McMurray JJ, Velazquez EJ, et al, for the Valsartan in Acute Myocardial Infarction Trial Investigators. Valsartan, captopril, or both in myocardial infarction complicated by heart failure, left ventricular dysfunction, or both. *N Engl J Med*. 2003;349:1893–1906.
150. Mann DL, Deswal A. Angiotensin-receptor blockade in acute myocardial infarction: a matter of dose. *N Engl J Med*. 2003;349:1963–1965.
151. Gibbons RJ, Miller TD, Christian TF. Infarct size measured by single photon emission computed tomographic imaging with (99m) Tc-sestamibi: a measure of the efficacy of therapy in acute myocardial infarction. *Circulation*. 2000;101:101–108.
152. Klocke FJ, Baird MG, Bateman TM, et al. ACC/AHA/ASNC guidelines for the clinical use of cardiac radionuclide imaging: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/ASNC Committee to Revise the 1995 Guidelines for the Clinical Use of Radionuclide Imaging). Available at: http://www.acc.org/clinical/guidelines/radio/rni_fulltext.pdf, 2003. Accessed January 15, 2004.
153. Cheitlin MD, Armstrong WF, Aurigemma GP, et al. ACC/AHA/ASE 2003 guideline update for the clinical application of echocardiography: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/ASE Committee to Update the 1997 Guidelines for the Clinical Application of Echocardiography). Available at: www.acc.org/clinical/guidelines/echo/index.pdf, 2003. Accessed January 15, 2004.
154. Mahrholdt H, Wagner A, Holly TA, et al. Reproducibility of chronic infarct size measurement by contrast-enhanced magnetic resonance imaging. *Circulation*. 2002;106:2322–2327.
155. Menon V, Slater JN, White HD, et al. Acute myocardial infarction complicated by systemic hypoperfusion without hypotension: report of the SHOCK trial registry. *Am J Med*. 2000;108:374–380.
156. Hochman JS, Boland J, Sleeper LA, et al, for the SHOCK Registry Investigators. Current spectrum of cardiogenic shock and effect of early revascularization on mortality: results of an International Registry. *Circulation*. 1995;91:873–881.
157. Hunt SA, Baker DW, Chin MH, et al. ACC/AHA guidelines for the evaluation and management of chronic heart failure in the adult: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1995 Guidelines for the Evaluation and Management of Heart Failure). Available at: http://www.acc.org/clinical/guidelines/failure/hf_index.htm, 2001. Accessed June 5, 2004.
158. Kinch JW, Ryan TJ. Right ventricular infarction. *N Engl J Med*. 1994;330:1211–1217.
159. Núñez L, de la Llana R, López Sendón J, et al. Diagnosis and treatment of subacute free wall ventricular rupture after infarction. *Ann Thorac Surg*. 1983;35:525–529.
160. Guidelines for cardiopulmonary resuscitation and emergency cardiac care: Emergency Cardiac Care Committee and Subcommittees, American Heart Association: Part III: adult advanced cardiac life support. *JAMA*. 1992;268:2199–2241.
161. Campbell RWF. Arrhythmias. In: Julian DG. Braunwald E, eds. *Management of Acute Myocardial Infarction*. London, England: WB Saunders 1994;223–240.
162. Kontoyannis DA, Anastasiou-Nana MI, Kontoyannis SA, et al. Intravenous amiodarone decreases the duration of atrial fibrillation associated with acute myocardial infarction. *Cardiovasc Drugs Ther*. 2001;15:155–160.
163. Ryan TJ, Antman EM, Brooks NH, et al. ACC/AHA guidelines for the management of patients with acute myocardial infarction: 1999 guideline update: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). *J Am Coll Cardiol*. 1999;34:890–911.
164. Gregoratos G, Abrams J, Epstein AE, et al. ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/NASPE Committee to Update the 1998 Pacemaker Guidelines). *J Am Coll Cardiol*. 2002;40:1703–1719.
165. Lamas GA, Ellenbogen KA. Evidence base for pacemaker mode selection: from physiology to randomized trials. *Circulation*. 2004;109:443–451.
166. Eagle KA, Guyton RA, Davidoff R, et al. ACC/AHA guidelines for coronary artery bypass graft surgery: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1991 Guidelines for Coronary Artery Bypass Graft Surgery). American College of Cardiology/American Heart Association. *J Am Coll Cardiol*. 1999;34:1262–1347.
167. Dyke C, Bhatia D. Inhibitors of the platelet receptor glycoprotein IIb-IIIa and complications during percutaneous coronary revascularization: management strategies for the cardiac surgeon. *J Cardiovasc Surg (Torino)*. 1999;40:505–516.
168. Yusuf S, Zhao F, Mehta SR, et al, for the Clopidogrel in Unstable Angina to Prevent Recurrent Events Trial Investigators. Effects of clopidogrel in addition to aspirin in patients with acute coronary syndromes without ST-segment elevation. *N Engl J Med*. 2001;345:494–502.
169. *Physician's Desk Reference*. Clopidogrel package insert. Montvale, NJ: Medical Economics Co; 2756.
170. Gibbons RJ, Balady GJ, Bricker JT, et al. ACC/AHA 2002 guideline update for exercise testing: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Exercise Testing). Available at: http://www.acc.org/clinical/guidelines/exercise/exercise_clean.pdf, 2002. Accessed February 12, 2004.
171. Théroux P, Waters DD, Halphen C, et al. Prognostic value of exercise testing soon after myocardial infarction. *N Engl J Med*. 1979;301:341–345.
172. DeBusk RF, Kraemer HC, Nash E, et al. Stepwise risk stratification soon after acute myocardial infarction. *Am J Cardiol*. 1983;52:1161–1166.
173. Krone RJ, Gillespie JA, Weld FM, et al. Low-level exercise testing after myocardial infarction: usefulness in enhancing clinical risk stratification. *Circulation*. 1985;71:80–89.

174. Valentine PA, Frew JL, Mashford ML, et al. Lidocaine in the prevention of sudden death in the pre-hospital phase of acute infarction: a double-blind study. *N Engl J Med*. 1974;291:1327–1331.
175. Ross J, Gilpin EA, Madsen EB, et al. A decision scheme for coronary angiography after acute myocardial infarction. *Circulation*. 1989;79: 292–303.
176. Risk stratification and survival after myocardial infarction. *N Engl J Med*. 1983;309:331–336.
177. Zaret BL, Wackers TH, Terrin M, et al. Does left ventricular ejection fraction following thrombolytic therapy have the same prognostic impact described in the prethrombolytic era? Results of the TIMI II Trial. *J Am Coll Cardiol*. 1991;17:214A. Abstract.
178. Roig E, Magriñá J, Garcia A, et al. Prognostic value of exercise radionuclide angiography in low-risk acute myocardial infarction survivors. *Eur Heart J*. 1993;14:213–218.
179. Gibbons RJ, Abrams J, Chatterjee K, et al. ACC/AHA 2002 guideline update for the management of patients with chronic stable angina: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1999 Guidelines for the Management of Patients with Chronic Stable Angina). Available at: www.acc.org/clinical/guidelines/stable/stable.pdf 2002. Accessed March 18, 2004.
180. O'Neill WW. "Watchful waiting" after thrombolysis: it's time for a re-evaluation. *J Am Coll Cardiol*. 2003;42:17–19.
181. Smith SC, Blair SN, Bonow RO, et al. AHA/ACC scientific statement: AHA/ACC guidelines for preventing heart attack and death in patients with atherosclerotic cardiovascular disease: 2001 update: a statement for healthcare professionals from the American Heart Association and the American College of Cardiology. *Circulation*. 2001;104:1577–1579.
182. Mosca L, Appel LJ, Benjamin EJ, et al. Evidence-based guidelines for cardiovascular disease prevention in women. *Circulation*. 2004;109: 672–693.
183. Dalal H, Evans PH, Campbell JL. Recent developments in secondary prevention and cardiac rehabilitation after acute myocardial infarction. *BMJ*. 2004;328:693–697.
184. Gutstein DE, Fuster V. Pathophysiologic bases for adjunctive therapies in the treatment and secondary prevention of acute myocardial infarction. *Clin Cardiol*. 1998;21:161–168.
185. CAPRIE Steering Committee. A randomised, blinded, trial of clopidogrel versus aspirin in patients at risk of ischaemic events (CAPRIE). *Lancet*. 1996;348:1329–1339.
186. Hurlen M, Smith P, Arnesen H. Effects of warfarin, aspirin and the two combined, on mortality and thromboembolic morbidity after myocardial infarction: the WARIS-II (Warfarin-Aspirin Reinfarction Study) design. *Scand Cardiovasc J*. 2000;34:168–171.
187. van Es RF, Jonker JJ, Verheugt FW, et al, for the Antithrombotics in the Secondary Prevention of Events in Coronary Thrombosis-2 (ASPECT-2) Research Group. Aspirin and coumadin after acute coronary syndromes (the ASPECT-2 study): a randomised controlled trial. *Lancet*. 2002;360:109–113.
188. Brouwer MA, van den Bergh PJ, Aengevaeren WR, et al. Aspirin plus coumarin versus aspirin alone in the prevention of reocclusion after fibrinolysis for acute myocardial infarction: results of the Antithrombotics in the Prevention of Reocclusion In Coronary Thrombolysis (APRICOT)-2 Trial. *Circulation*. 2002;106:659–665.
189. Yusuf S, Sleight P, Pogue J, et al, for the Heart Outcomes Prevention Evaluation Study Investigators. Effects of an angiotensin-converting-enzyme inhibitor, ramipril, on cardiovascular events in high-risk patients. *N Engl J Med*. 2000;342:145–153.
190. Fox KM, for the EURopean trial On reduction of cardiac events with Perindopril in stable coronary Artery disease Investigators. Efficacy of perindopril in reduction of cardiovascular events among patients with stable coronary artery disease: randomised, double-blind, placebo-controlled, multicentre trial (the EUROPA study). *Lancet*. 2003;362: 782–788.
191. Granger CB, McMurray JJ, Yusuf S, et al, for the CHARM Investigators and Committees. Effects of candesartan in patients with chronic heart failure and reduced left-ventricular systolic function intolerant to angiotensin-converting-enzyme inhibitors: the CHARM-Alternative trial. *Lancet*. 2003;362:772–776.
192. McMurray JJ, Ostergren J, Swedberg K, et al, for the CHARM Investigators and Committees. Effects of candesartan in patients with chronic heart failure and reduced left-ventricular systolic function taking angiotensin-converting-enzyme inhibitors: the CHARM-Added trial. *Lancet*. 2003;362:767–771.
193. Yusuf S, Pfeffer MA, Swedberg K, et al, for the CHARM Investigators and Committees. Effects of candesartan in patients with chronic heart failure and preserved left-ventricular ejection fraction: the CHARM-Preserved trial. *Lancet*. 2003;362:777–781.
194. Executive Council of the Heart Failure Society of America. Implications of recent clinical trials for heart failure performance measures. *J Card Fail*. 2004;10:4–5.
195. Deleted in press.
196. Antman E, Braunwald E. Acute myocardial infarction. In: Braunwald E, Zipes DP, Libby P, eds. *Heart Disease: A Textbook of Cardiovascular Medicine*. 6th ed. Philadelphia, Pa: WB Saunders;2001:1114–1251. Chapter 5.
197. Gheorghiade M, Colucci WS, Swedberg K. Beta-blockers in chronic heart failure. *Circulation*. 2003;107:1570–1575.
198. Chen J, Radford MJ, Wang Y, et al. Are beta-blockers effective in elderly patients who undergo coronary revascularization after acute myocardial infarction? *Arch Intern Med*. 2000;160:947–952.
199. Seventh report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7): resetting the hypertension sails. *Hypertension*. 2003;41: 1178–1179.
200. Hulley S, Grady D, Bush T, et al, for the Heart and Estrogen/progestin Replacement Study (HERS) Research Group. Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women. *JAMA*. 1998;280:605–613.
201. Grady D, Herrington D, Bittner V, et al, for the HERS Research Group. Cardiovascular disease outcomes during 6.8 years of hormone therapy: Heart and Estrogen/progestin Replacement Study follow-up (HERS II). *JAMA*. 2002;288:49–57.
202. Rossouw JE, Anderson GL, Prentice RL, et al, for the Writing Group for the Women's Health Initiative Investigators. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women's Health Initiative randomized controlled trial. *JAMA*. 2002;288:321–333.
203. Cairns JA, Markham BA. Economics and efficacy in choosing oral anticoagulants or aspirin after myocardial infarction. *JAMA*. 1995;273:965–967.
203a. Kris-Etherton PM, Lichtenstein AH, Howard BV, Steinberg D, Witzman JL. Antioxidant vitamin supplements and cardiovascular disease. *Circulation*. In press.
204. Glassman AH, O'Connor CM, Califf RM, et al, for the Sertraline Antidepressant Heart Attack Randomized Trial (SADHEART) Group. Sertraline treatment of major depression in patients with acute MI or unstable angina. *JAMA*. 2002;288:701–709.
205. Swenson JR, O'Connor CM, Barton D, et al, for the Sertraline Antidepressant Heart Attack Randomized Trial (SADHART) Group. Influence of depression and effect of treatment with sertraline on quality of life after hospitalization for acute coronary syndrome. *Am J Cardiol*. 2003; 92:1271–1276.
206. Berkman LF, Blumenthal J, Burg M, et al, for the Enhancing Recovery in Coronary Heart Disease Patients Investigators (ENRICHD). Effects of treating depression and low perceived social support on clinical events after myocardial infarction: the Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD) Randomized Trial. *JAMA*. 2003; 289:3106–3116.
207. Alonzo AA. Acute myocardial infarction and posttraumatic stress disorder: the consequences of cumulative adversity. *J Cardiovasc Nurs*. 1999;13:33–45.