2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction: Executive Summary

A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines

Developed in Collaboration With the American College of Emergency Physicians and Society for Cardiovascular Angiography and Interventions

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This document was approved by the American College of Cardiology Foundation Board of Trustees and the American Heart Association Science and Advisory Coordinating Committee in June 2012.

The online-only Data Supplement is available with this article at http://circ.ahajournals.org/lookup/suppl/doi:10.1161/CIR.0b013e3182742c84/-/DC1. The online-only Comprehensive Relationships Table is available with this article at http://circ.ahajournals.org/lookup/suppl/doi:10.1161/CIR.0b013e3182742c84/-/DC2.


This article is copublished in the Journal of the American College of Cardiology and Catheterization and Cardiovascular Interventions.

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DOI: 10.1161/CIR.0b013e3182742c84

Circulation is available at http://circ.ahajournals.org

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Preamble

The medical profession should play a central role in evaluating the evidence related to drugs, devices, and procedures for the detection, management, and prevention of disease. When properly applied, expert analysis of available data on the benefits and risks of these therapies and procedures can improve the quality of care, optimize patient outcomes, and favorably affect costs by focusing resources on the most effective strategies. An organized and directed approach to a thorough review of evidence has resulted in the production of clinical practice guidelines that assist physicians in selecting the best management strategy for an individual patient. Moreover, clinical practice guidelines can provide a foundation for other applications, such as performance measures, appropriate use criteria, and both quality improvement and clinical decision support tools.

The American College of Cardiology Foundation (ACCF) and the American Heart Association (AHA) have jointly produced guidelines in the area of cardiovascular disease since 1980. The ACCF/AHA Task Force on Practice Guidelines (Task Force), charged with developing, updating, and revising practice guidelines for cardiovascular diseases and procedures, directs and oversees this effort. Writing committees are charged with regularly reviewing and evaluating all available evidence to develop balanced, patient-centric recommendations for clinical practice.

Experts in the subject under consideration are selected by the ACCF and AHA to examine subject-specific data and write guidelines in partnership with representatives from other medical organizations and specialty groups. Writing committees are asked to perform a literature review; weigh the strength of evidence for or against particular tests, treatments, or procedures; and include estimates of expected outcomes where such data exist. Patient-specific modifiers, comorbidities, and issues of patient preference that may influence the choice of tests or therapies are considered.
When available, information from studies on cost is considered, but data on efficacy and outcomes constitute the primary basis for the recommendations contained herein.

In analyzing the data and developing recommendations and supporting text, the writing committee uses evidence-based methodologies developed by the Task Force. The Class of Recommendation (COR) is an estimate of the size of the treatment effect considering risks versus benefits in addition to evidence and/or agreement that a given treatment or procedure is or is not useful/effective or in some situations may cause harm. The Level of Evidence (LOE) is an estimate of the certainty or precision of the treatment effect. The writing committee reviews and ranks evidence supporting each recommendation with the weight of evidence ranked as LOE A, B, or C according to specific definitions that are included in Table 1. Studies are identified as observational, retrospective, prospective, or randomized where appropriate. For certain conditions for which inadequate data are available, recommendations are based on expert consensus and clinical experience and are ranked as LOE C. When recommendations at LOE C are supported by historical clinical data, appropriate references (including clinical reviews) are cited if available. For issues for which sparse data are available, a survey of current practice among the clinician members of the writing committee is the basis for LOE C recommendations and no references are cited. The schema for

A recommendation with Level of Evidence B or C does not imply that the recommendation is weak. Many important clinical questions addressed in the guidelines do not lend themselves to clinical trials. Although randomized trials are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

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

†For comparative effectiveness recommendations (Class I and IIa; Level of Evidence A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

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COR and LOE is summarized in Table 1, which also provides suggested phrases for writing recommendations within each COR.

A new addition to this methodology is separation of the Class III recommendations to delineate whether the recommendation is determined to be of “no benefit” or is associated with “harm” to the patient. In addition, in view of the increasing number of comparative effectiveness studies, comparator verbs and suggested phrases for writing recommendations for the comparative effectiveness of one treatment or strategy versus another are included for COR I and IIa, LOE A or B only.

In view of the advances in medical therapy across the spectrum of cardiovascular diseases, the Task Force has designated the term guideline-directed medical therapy (GDMT) to represent optimal medical therapy as defined by ACCF/AHA guideline-recommended therapies (primarily Class I). This new term, GDMT, will be used throughout subsequent guidelines.

Because the ACCF/AHA practice guidelines address patient populations (and healthcare providers) residing in North America, drugs that are not currently available in North America are discussed in the text without a specific COR. For studies performed in large numbers of subjects outside North America, each writing committee reviews the potential influence of different practice patterns and patient populations on the treatment effect and relevance to the ACCF/AHA target population to determine whether the findings should inform a specific recommendation.

The ACCF/AHA practice guidelines are intended to assist healthcare providers in clinical decision making by describing a range of generally acceptable approaches to the diagnosis, management, and prevention of specific diseases or conditions. The guidelines attempt to define practices that meet the needs of most patients in most circumstances. The ultimate judgment regarding care of a particular patient must be made by the healthcare provider and patient in light of all the circumstances presented by that patient. As a result, situations may arise for which deviations from these guidelines may be appropriate. Clinical decision making should involve consideration of the quality and availability of expertise in the area where care is provided. When these guidelines are used as the basis for regulatory or payer decisions, the goal should be improvement in quality of care. The Task Force recognizes that situations arise in which additional data are needed to inform patient care more effectively; these areas are identified as the basis for regulatory or payer decisions, the goal should be improvement in quality of care. The Task Force recognizes that situations arise in which additional data are needed to inform patient care more effectively; these areas are identified within each respective guideline when appropriate.

Prescribed courses of treatment in accordance with these recommendations are effective only if followed. Because lack of patient understanding and adherence may adversely affect outcomes, physicians and other healthcare providers should make every effort to engage the patient’s active participation in prescribed medical regimens and lifestyles. In addition, patients should be informed of the risks, benefits, and alternatives to a particular treatment and should be involved in shared decision making whenever feasible, particularly for COR IIa and IIb, for which the benefit-to-risk ratio may be lower.

The Task Force makes every effort to avoid actual, potential, or perceived conflicts of interest that may arise as a result of relationships with industry and other entities (RWI) among the members of the writing committee. All writing committee members and peer reviewers of the guideline are required to disclose all current healthcare related relationships, including those existing 12 months before initiation of the writing effort. In December 2009, the ACCF and AHA implemented a new RWI policy that requires the writing committee chair plus a minimum of 50% of the writing committee to have no relevant RWI. (Appendix 1 includes the ACCF/AHA definition of relevance.) These statements are reviewed by the Task Force and all members during each conference call and/or meeting of the writing committee, and members provide updates as changes occur. All guideline recommendations require a confidential vote by the writing committee and must be approved by a consensus of the voting members. Members may not draft or vote on any text or recommendations pertaining to their RWI. Members who recused themselves from voting are indicated in the list of writing committee members, and specific section recusals are noted in Appendix 1. Authors’ and peer reviewers’ RWI pertinent to this guideline are disclosed in Appendixes 1 and 2, respectively. In addition, to ensure complete transparency, writing committee members’ comprehensive disclosure information—including RWI not pertinent to this document—is available as an online supplement. Comprehensive disclosure information for the Task Force is also available online at http://www.cardiosource.org/ACC/About-ACC/Who-We-Are/Leadership/Guidelines-and-Documents-Task-Forces.aspx. The work of writing committees is supported exclusively by the ACCF and AHA without commercial support. Writing committee members volunteered their time for this activity.

In an effort to maintain relevance at the point of care for practicing physicians, the Task Force continues to oversee an ongoing process improvement initiative. As a result, in response to pilot projects, several changes to these guidelines will be apparent, including limited narrative text, a focus on summary and evidence tables (with references linked to abstracts in PubMed), and more liberal use of summary recommendation tables (with references that support LOE) to serve as a quick reference.

In April 2011, the Institute of Medicine released 2 reports: Finding What Works in Health Care: Standards for Systematic Reviews and Clinical Practice Guidelines We Can Trust. It is noteworthy that the IOM cited ACCF/AHA practice guidelines as being compliant with many of the proposed standards. A thorough review of these reports and of our current methodology is under way, with further enhancements anticipated.

The recommendations in this guideline are considered current until they are superseded by a focused update or the full-text guideline is revised. The reader is encouraged to consult the full-text guideline for additional guidance and details about the care of the patient with ST-elevation myocardial infarction (STEMI), because the Executive Summary contains only the recommendations. Guidelines are official policy of both the ACCF and AHA.

Jeffrey L. Anderson, MD, FACC, FAHA
Chair, ACCF/AHA Task Force on Practice Guidelines
1. Introduction

1.1. Methodology and Evidence Review
The recommendations listed in this document are, whenever possible, evidence based. The current document constitutes a full revision and includes an extensive evidence review which was conducted through November 2010, with additional selected references added through August 2012. Searches were limited to studies conducted in human subjects and reviews and other evidence pertaining to human subjects; all were published in English. Key search words included but were not limited to: acute coronary syndromes, percutaneous coronary intervention, coronary artery bypass graft, myocardial infarction, ST-elevation myocardial infarction, coronary stent, revascularization, anticoagulant therapy, antiplatelet therapy, antithrombotic therapy, glycoprotein IIb/IIIa inhibitor therapy, pharmacotherapy, proton-pump inhibitor, implantable cardioverter-defibrillator therapy, cardiogenic shock, fibrinolytic therapy, thrombyolytic therapy, nitrates, mechanical complications, arrhythmia, angina, chronic stable angina, diabetes, chronic kidney disease, mortality, morbidity, elderly, ethics, and contrast nephropathy. Additional searches cross-referenced these topics with the following subtopics: percutaneous coronary intervention, coronary artery bypass graft, cardiac rehabilitation, and secondary prevention. Additionally, the committee reviewed documents related to the subject matter previously published by the ACCF and AHA. References selected and published in this document are representative and not all inclusive.

The focus of this guideline is the management of patients with STEMI. Updates to the 2004 STEMI guideline were published in 2007 and 2009. Particular emphasis is placed on advances in reperfusion therapy, organization of regional systems of care, transfer algorithms, evidence-based anti-thrombotic and medical therapies, and secondary prevention strategies to optimize patient-centered care. By design, the document is narrower in scope than the 2004 STEMI Guideline, in an attempt to provide a more focused tool for practitioners. References related to management guidelines are provided whenever appropriate, including those pertaining to percutaneous coronary intervention (PCI), coronary artery bypass graft (CABG), heart failure (HF), cardiac devices, and secondary prevention.

1.2. Organization of the Writing Committee
The writing committee was composed of experts representing cardiovascular medicine, interventional cardiology, electrophysiology, HF, cardiac surgery, emergency medicine, internal medicine, cardiac rehabilitation, nursing, and pharmacy. The American College of Physicians, American College of Emergency Physicians, and Society for Cardiovascular Angiography and Interventions assigned official representatives.

1.3. Document Review and Approval
This document was reviewed by 2 outside reviewers each nominated by the ACCF and the AHA, as well as 2 reviewers each from the American College of Emergency Physicians and Society for Cardiovascular Angiography and Interventions and 22 individual content reviewers (including members from the ACCF Interventional Scientific Council and ACCF Surgeons’ Scientific Council). All reviewer RWI information was distributed to the writing committee and is published in this document (Appendix 2).

This document was approved for publication by the governing bodies of the ACCF and the AHA and was endorsed by the American College of Emergency Physicians and Society for Cardiovascular Angiography and Interventions.

2. Onset of Myocardial Infarction: Recommendations

2.1. Regional Systems of STEMI Care, Reperfusion Therapy, and Time-to-Treatment Goals
See Figure 1.

Class I

1. All communities should create and maintain a regional system of STEMI care that includes assessment and continuous quality improvement of emergency medical services and hospital-based activities. Performance can be facilitated by participating in programs such as Mission: Lifeline and the Door-to-Balloon Alliance.

2. Performance of a 12-lead electrocardiogram (ECG) by emergency medical services personnel at the site of first medical contact (FMC) is recommended in patients with symptoms consistent with STEMI.

3. Reperfusion therapy should be administered to all eligible patients with STEMI with symptom onset within the prior 12 hours.

4. Primary PCI is the recommended method of reperfusion when it can be performed in a timely fashion by experienced operators.

5. Emergency medical services transport directly to a PCI-capable hospital for primary PCI is the recommended triage strategy for patients with STEMI, with an ideal FMC-to-device time system goal of 90 minutes or less.

6. Immediate transfer to a PCI-capable hospital for primary PCI is the recommended triage strategy for patients with STEMI who initially arrive at or are transported to a non–PCI-capable hospital, with an FMC-to-device time system goal of 120 minutes or less.

7. In the absence of contraindications, fibrinolytic therapy should be administered to patients with STEMI at non–PCI-capable hospitals when the anticipated FMC-to-device time at a PCI-capable hospital exceeds 120 minutes because of unavoidable delays.

8. When fibrinolytic therapy is indicated or chosen as the primary reperfusion strategy, it should be administered within 30 minutes of hospital arrival.

Class IIa

1. Reperfusion therapy is reasonable for patients with STEMI and symptom onset within the prior 12 to 24 hours.

*The proposed time windows are system goals. For any individual patient, every effort should be made to provide reperfusion therapy as rapidly as possible.
hours who have clinical and/or ECG evidence of ongoing ischemia. Primary PCI is the preferred strategy in this population.\(^{16,29,30}\) (Level of Evidence: B)

### 2.2. Evaluation and Management of Patients With STEMI and Out-of-Hospital Cardiac Arrest

Class I

1. Therapeutic hypothermia should be started as soon as possible in comatose patients with STEMI and out-of-hospital cardiac arrest caused by ventricular fibrillation or pulseless ventricular tachycardia, including patients who undergo primary PCI.\(^{31–33}\) (Level of Evidence: B)

2. Immediate angiography and PCI when indicated should be performed in resuscitated out-of-hospital cardiac arrest patients whose initial ECG shows STEMI.\(^{34–49}\) (Level of Evidence: B)

### 3. Reperfusion at a PCI-Capable Hospital: Recommendations

#### 3.1. Primary PCI in STEMI

See Table 2 for a summary of recommendations from this section.

Class I

1. Primary PCI should be performed in patients with STEMI and ischemic symptoms of less than 12 hours’ duration.\(^{17,50,51}\) (Level of Evidence: A)

2. Primary PCI should be performed in patients with STEMI and ischemic symptoms of less than 12 hours’ duration who have contraindications to fibrinolytic therapy, irrespective of time delay from MI onset (Class I, LOE: B).

3. Primary PCI should be performed in patients with STEMI and cardiogenic shock or acute severe HF, irrespective of time delay from myocardial infarction (MI) onset (Section 8.1).\(^{54–57}\) (Level of Evidence: B)

Class IIa

1. Primary PCI is reasonable in patients with STEMI if there is clinical and/or ECG evidence of ongoing ischemia between 12 and 24 hours after symptom onset.\(^{29,20}\) (Level of Evidence: B)

<table>
<thead>
<tr>
<th>Reperfusion at a PCI-Capable Hospital: Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Therapeutic hypothermia should be started as soon as possible in comatose patients with STEMI and out-of-hospital cardiac arrest caused by ventricular fibrillation or pulseless ventricular tachycardia, including patients who undergo primary PCI.(^{31–33}) (Level of Evidence: B)</td>
</tr>
<tr>
<td>2. Immediate angiography and PCI when indicated should be performed in resuscitated out-of-hospital cardiac arrest patients whose initial ECG shows STEMI.(^{34–49}) (Level of Evidence: B)</td>
</tr>
</tbody>
</table>

### Table 2. Primary PCI in STEMI

<table>
<thead>
<tr>
<th>Ischemic symptoms &lt;12 h</th>
<th>I</th>
<th>A</th>
<th>17, 50, 51</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic symptoms &lt;12 h and contraindications to fibrinolytic therapy irrespective of time delay from FMC</td>
<td>I</td>
<td>B</td>
<td>52, 53</td>
</tr>
<tr>
<td>Cardiogenic shock or acute severe HF irrespective of time delay from MI onset</td>
<td>I</td>
<td>B</td>
<td>54–57</td>
</tr>
<tr>
<td>Evidence of ongoing ischemia 12 to 24 h after symptom onset</td>
<td>IIa</td>
<td>B</td>
<td>29, 30</td>
</tr>
<tr>
<td>PCI of a noninfarct artery at the time of primary PCI in patients without hemodynamic compromise</td>
<td>III: Harm</td>
<td>B</td>
<td>58–60</td>
</tr>
</tbody>
</table>

COR indicates Class of Recommendation; FMC, first medical contact; HF, heart failure; LOE, Level of Evidence; MI, myocardial infarction; PCI, percutaneous coronary intervention; and STEMI, ST-elevation myocardial infarction.
Class III: Harm

1. PCI should not be performed in a noninfarct artery at the time of primary PCI in patients with STEMI who are hemodynamically stable.58-60 (Level of Evidence: B)

3.2. Aspiration Thrombectomy

Class IIa

1. Manual aspiration thrombectomy is reasonable for patients undergoing primary PCI.41-44 (Level of Evidence: B)

Class IIb

1. It may be reasonable to administer intravenous GP IIb/IIIa receptor antagonist in the precatheterization laboratory setting (eg, ambulance, emergency department) to patients with STEMI for whom primary PCI is intended.91,94-101 (Level of Evidence: B)

Class III: Harm

1. Prasugrel should not be administered to patients with a history of prior stroke or transient ischemic attack.93 (Level of Evidence: B)

3.4. Antiplatelet Therapy to Support Primary PCI for STEMI

See Table 3 for a summary of recommendations from this section.

Class I

1. Aspirin 162 to 325 mg should be given before primary PCI.74-76 (Level of Evidence: B)

2. After PCI, aspirin should be continued indefinitely.77,78,80 (Level of Evidence: A)

3. A loading dose of a P2Y12 receptor inhibitor should be given as early as possible or at time of primary PCI to patients with STEMI. Options include:
   a. Clopidogrel 600 mg79,81,82 (Level of Evidence: B); or
   b. Prasugrel 60 mg83 (Level of Evidence: B); or
   c. Ticagrelor 180 mg84 (Level of Evidence: B)

4. P2Y12 inhibitor therapy should be given for 1 year to patients with STEMI who receive a stent (bare-metal or drug-eluting) during primary PCI using the following maintenance doses:
   a. Clopidogrel 75 mg daily85,86 (Level of Evidence: B); or
   b. Prasugrel 10 mg daily87 (Level of Evidence: B); or
   c. Ticagrelor 90 mg twice a day.88 (Level of Evidence: B)

Class IIa

1. It is reasonable to use 81 mg of aspirin per day in preference to higher maintenance doses after primary PCI.76,77,86,87 (Level of Evidence: B)

2. It is reasonable to start treatment with an intravenous glycoprotein (GP) IIb/IIIa receptor antagonist such as abciximab85-90 (Level of Evidence: A), high-bolus-dose tirofiban91,92 (Level of Evidence: B), or double-bolus eptifibatide93 (Level of Evidence: B) at the time of primary PCI (with or without stenting or clopidogrel pretreatment) in selected patients with STEMI who are receiving unfractionated heparin (UFH).

Class IIb

1. In patients with STEMI undergoing PCI who are at high risk of bleeding, it is reasonable to use bivalirudin monotherapy in preference to the combination of UFH and a GP IIb/IIIa receptor antagonist.109 (Level of Evidence: B)

Class III: Harm

1. Fondaparinux should not be used as the sole anticoagulant to support primary PCI because of the risk of catheter thrombosis.110 (Level of Evidence: B)

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†Balloon angioplasty without stent placement may be used in selected patients.
‡The recommended maintenance dose of aspirin to be used with ticagrelor is 81 mg daily.
### Table 3. Adjunctive Antithrombotic Therapy to Support Reperfusion With Primary PCI

<table>
<thead>
<tr>
<th><strong>Antithrombotic Therapy</strong></th>
<th><strong>COR</strong></th>
<th><strong>LOE</strong></th>
<th><strong>References</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antiplatelet therapy</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Aspirin</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• 162- to 325-mg load before procedure</td>
<td>I</td>
<td>B</td>
<td>74–76</td>
</tr>
<tr>
<td>• 81- to 325-mg daily maintenance dose (indefinite)*</td>
<td>I</td>
<td>A</td>
<td>77, 78, 80</td>
</tr>
<tr>
<td>• 81 mg daily is the preferred maintenance dose*</td>
<td>IIA</td>
<td>B</td>
<td>76, 77, 86, 87</td>
</tr>
<tr>
<td><strong>P2Y&lt;sub&gt;12&lt;/sub&gt; inhibitors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Loading doses</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Clopidogrel: 600 mg as early as possible or at time of PCI</td>
<td>I</td>
<td>B</td>
<td>83, 82</td>
</tr>
<tr>
<td>• Prasugrel: 60 mg as early as possible or at time of PCI</td>
<td>I</td>
<td>B</td>
<td>83</td>
</tr>
<tr>
<td>• Ticagrelor: 180 mg as early as possible or at time of PCI</td>
<td>I</td>
<td>B</td>
<td>84</td>
</tr>
<tr>
<td><strong>Maintenance doses and duration of therapy</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>DES placed: Continue therapy for 1 y with:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Clopidogrel: 75 mg daily</td>
<td>I</td>
<td>B</td>
<td>83, 85</td>
</tr>
<tr>
<td>• Prasugrel: 10 mg daily</td>
<td>I</td>
<td>B</td>
<td>85</td>
</tr>
<tr>
<td>• Ticagrelor: 90 mg twice a day*</td>
<td>I</td>
<td>B</td>
<td>84</td>
</tr>
<tr>
<td><strong>BMS† placed: Continue therapy for 1 y with:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Clopidogrel: 75 mg daily</td>
<td>I</td>
<td>B</td>
<td>83, 85</td>
</tr>
<tr>
<td>• Prasugrel: 10 mg daily</td>
<td>I</td>
<td>B</td>
<td>85</td>
</tr>
<tr>
<td>• Ticagrelor: 90 mg twice a day*</td>
<td>I</td>
<td>B</td>
<td>84</td>
</tr>
<tr>
<td><strong>DES placed:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Clopidogrel, prasugrel, or ticagrelor* continued beyond 1 y</td>
<td>IIb</td>
<td>C</td>
<td>N/A</td>
</tr>
<tr>
<td>• Patients with STEMI with prior stroke or TIA: prasugrel</td>
<td>III: Harm</td>
<td>B</td>
<td>83</td>
</tr>
<tr>
<td><strong>IV GP IIb/IIa receptor antagonists in conjunction with UFH or bivalirudin in selected patients</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Abciximab: 0.25-mg/kg IV bolus, then 0.125 mcg/kg/min (maximum 10 mcg/min)</td>
<td>IIA</td>
<td>A</td>
<td>88–90</td>
</tr>
<tr>
<td>• Tirofiban: (high-bolus dose): 25-mcg/kg IV bolus, then 0.15 mcg/kg/min</td>
<td>IIA</td>
<td>B</td>
<td>91, 92</td>
</tr>
<tr>
<td>• In patients with CrCl &lt;30 mL/min, reduce infusion by 50%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Eptifibatide: (double bolus): 180-mcg/kg IV bolus, then 2 mcg/kg/min; a second 180-mcg/kg bolus is administered 10 min after the first bolus</td>
<td>IIA</td>
<td>B</td>
<td>93</td>
</tr>
<tr>
<td>• In patients with CrCl &lt;50 mL/min, reduce infusion by 50%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Avoid in patients on hemodialysis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Pre–catheterization laboratory administration of intravenous GP IIb/IIa receptor antagonist</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Intracoronary abciximab 0.25-mg/kg bolus</td>
<td>IIB</td>
<td>B</td>
<td>91, 94–101</td>
</tr>
<tr>
<td>• Fondaparinux: Not recommended as sole anticoagulant for primary PCI</td>
<td>III: Harm</td>
<td>B</td>
<td>64, 102–108</td>
</tr>
<tr>
<td><strong>Anticoagulant therapy</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>UFH:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• With GP IIb/IIa receptor antagonist planned: 50- to 70-U/kg IV bolus to achieve therapeutic ACT‡</td>
<td>I</td>
<td>C</td>
<td>N/A</td>
</tr>
<tr>
<td>• With no GP IIb/IIa receptor antagonist planned: 70- to 100-U/kg bolus to achieve therapeutic ACT§</td>
<td>I</td>
<td>C</td>
<td>N/A</td>
</tr>
<tr>
<td>• Bivalirudin: 0.75-mg/kg IV bolus, then 1.75-mg/kg/h infusion with or without prior treatment with UFH. An additional bolus of 0.3 mg/kg can be given if needed.</td>
<td>I</td>
<td>B</td>
<td>109</td>
</tr>
<tr>
<td>• Reduce infusion to 1 mg/kg/h with estimated CrCl &lt;30 mL/min</td>
<td>IIa</td>
<td>B</td>
<td>109</td>
</tr>
<tr>
<td>• Preferred over UFH with GP IIb/IIa receptor antagonist in patients at high risk of bleeding</td>
<td>IIa</td>
<td>B</td>
<td>109</td>
</tr>
<tr>
<td>• Fondaparinux: Not recommended as sole anticoagulant for primary PCI</td>
<td>III: Harm</td>
<td>B</td>
<td>110</td>
</tr>
</tbody>
</table>

*The recommended maintenance dose of aspirin to be used with ticagrelor is 81 mg daily.
†Balloon angioplasty without stent placement may be used in selected patients. It might be reasonable to provide P2Y<sub>12</sub> inhibitor therapy to patients with STEMI undergoing balloon angioplasty alone according to the recommendations listed for BMS. (LOE: C)
‡The recommended ACT with planned GP IIb/IIa receptor antagonist treatment is 200 to 250 s.
§The recommended ACT with no planned GP IIb/IIa receptor antagonist treatment is 250 to 300 s (HemoTec device) or 300 to 350 s (Hemochron device). ACT indicates activated clotting time; BMS, bare-metal stent; CrCl, creatinine clearance; COR, Class of Recommendation; DES, drug-eluting stent; GP, glycoprotein; IV, intravenous; LOE, Level of Evidence; N/A, not available; PCI, percutaneous coronary intervention; STEMI, ST-elevation myocardial infarction; TIA, transient ischemic attack; and UFH, unfractionated heparin.
4. Reperfusion at a Non–PCI-Capable Hospital: Recommendations

4.1. Fibrinolytic Therapy When There Is an Anticipated Delay to Performing Primary PCI Within 120 Minutes of FMC

See Table 4 for a summary of recommendations from this section.

Class I

1. In the absence of contraindications, fibrinolytic therapy should be given to patients with STEMI and onset of ischemic symptoms within the previous 12 hours when it is anticipated that primary PCI cannot be performed within 120 minutes of FMC.\(^1\)\(^6\),\(^1\)\(^1\)\(^1\)–\(^1\)\(^1\)\(^6\) \((\text{Level of Evidence}: \text{A})\)

Class IIa

1. In the absence of contraindications and when PCI is not available, fibrinolytic therapy is reasonable for patients with STEMI if there is clinical and/or electrocardiographic evidence of ongoing ischemia within 12 to 24 hours of symptom onset and a large area of myocardium at risk or hemodynamic instability. \((\text{Level of Evidence}: \text{C})\)

Class III: Harm

1. Fibrinolytic therapy should not be administered to patients with ST depression except when a true posterior (inferobasal) MI is suspected or when associated with ST elevation in lead aVR.\(^1\)\(^6\),\(^1\)\(^1\)\(^7\)–\(^1\)\(^1\)\(^2\) \((\text{Level of Evidence}: \text{B})\)

4.2. Adjunctive Antithrombotic Therapy With Fibrinolysis

See Table 5 for a summary of recommendations from this section.

4.2.1. Adjunctive Antiplatelet Therapy With Fibrinolysis

Class I

1. Aspirin (162- to 325-mg loading dose) and clopidogrel (300-mg loading dose for \(\leq 75\) years of age, 75-mg dose for patients \(>75\) years of age) should be administered to patients with STEMI who receive fibrinolytic therapy.\(^1\)\(^1\)\(^3\),\(^1\)\(^2\)\(^1\)\(^2\),\(^1\)\(^2\) \((\text{Level of Evidence}: \text{A})\)

2. Aspirin should be continued indefinitely\(^1\)\(^1\)\(^3\),\(^1\)\(^2\)\(^1\)\(^2\),\(^1\)\(^2\) \((\text{Level of Evidence}: \text{A})\) and clopidogrel (75 mg daily) should be continued for at least 14 days\(^1\)\(^2\)\(^1\)\(^2\),\(^1\)\(^2\) \((\text{Level of Evidence}: \text{A})\) and up to 1 year \((\text{Level of Evidence}: \text{C})\) in patients with STEMI who receive fibrinolytic therapy.

Class IIa

1. It is reasonable to use aspirin 81 mg per day in preference to higher maintenance doses after fibrinolytic therapy.\(^7\)\(^7\),\(^8\)\(^0\),\(^8\)\(^6\),\(^8\)\(^7\) \((\text{Level of Evidence}: \text{B})\)

4.2.2. Adjunctive Anticoagulant Therapy With Fibrinolysis

Class I

1. Patients with STEMI undergoing reperfusion with fibrinolytic therapy should receive anticoagulant therapy for a minimum of 48 hours, and preferably for the duration of the index hospitalization, up to 8 days or until revascularization if performed.\(^1\)\(^2\)\(^3\),\(^1\)\(^2\)\(^4\) \((\text{Level of Evidence}: \text{A})\) Recommended regimens include

a. UFH administered as a weight-adjusted intravenous bolus and infusion to obtain an activated partial thromboplastin time of 1.5 to 2.0 times control, for 48 hours or until revascularization \((\text{Level of Evidence}: \text{C})\);

b. Enoxaparin administered according to age, weight, and creatinine clearance, given as an intravenous bolus, followed in 15 minutes by subcutaneous injection for the duration of the index hospitalization, up to 8 days or until revascularization\(^1\)\(^2\)\(^4\)–\(^1\)\(^2\)\(^7\) \((\text{Level of Evidence}: \text{A})\); or

c. Fondaparinux administered with initial intravenous dose, followed in 24 hours by daily subcutaneous injections if the estimated creatinine clearance is greater than 30 mL/min, for the duration of the index hospitalization, up to 8 days or until revascularization.\(^1\)\(^1\)\(^0\) \((\text{Level of Evidence}: \text{B})\)
genic shock or acute severe HF, irrespective of the time delay from MI onset.128 (Level of Evidence: B)

Class IIa

1. Urgent transfer to a PCI-capable hospital for coronary angiography is reasonable for patients with STEMI who demonstrate evidence of failed reperfusion or reocclusion after fibrinolytic therapy.129–132 (Level of Evidence: B)

2. Transfer to a PCI-capable hospital for coronary angiography is reasonable for patients with STEMI who have received fibrinolytic therapy even when hemodynamically stable1 and with clinical evidence of successful reperfusion. Angiography can be performed as soon as logistically feasible at the receiving hospital, and ideally within 24 hours, but should not be performed within the first 2 to 3 hours after administration of fibrinolytic therapy.133–138 (Level of Evidence: B)

Although individual circumstances will vary, clinical stability is defined by the absence of low output, hypotension, persistent tachycardia, apparent shock, high-grade ventricular or symptomatic supraventricular tachyarrhythmias, and spontaneous recurrent ischemia.

5. Delayed Invasive Management: Recommendations

5.1. Coronary Angiography in Patients Who Initially Were Managed With Fibrinolytic Therapy or Who Did Not Receive Reperfusion

See Table 7 for a summary of recommendations from this section.

Class I

1. Cardiac catheterization and coronary angiography with intent to perform revascularization should be performed after STEMI in patients with any of the following:
   a. Cardiogenic shock or acute severe HF that develops after initial presentation127,128,139,140 (Level of Evidence: B);
   b. Intermediate- or high-risk findings on pre-discharge noninvasive ischemia testing141,142 (Level of Evidence: B); or
   c. Myocardial ischemia that is spontaneous or provoked by minimal exertion during hospitalization. (Level of Evidence: C)
Table 6. Indications for Transfer for Angiography After Fibrinolytic Therapy

<table>
<thead>
<tr>
<th>COR</th>
<th>LOE</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immediate transfer for cardiogenic shock or severe acute HF irrespective of time delay from MI onset</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Urgent transfer for failed reperfusion or reocclusion</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td>As part of an invasive strategy in stable* patients with PCI between 3 and 24 h after successful fibrinolysis</td>
<td>IIa</td>
<td>B</td>
</tr>
</tbody>
</table>

*Although individual circumstances will vary, clinical stability is defined by the absence of low output, hypotension, persistent tachycardia, apparent shock, high-grade ventricular or symptomatic supraventricular tachyarrhythmias, and spontaneous recurrent ischemia.

COR indicates Class of Recommendation; HF, heart failure; LOE, Level of Evidence; MI, myocardial infarction; N/A, not available; and PCI, percutaneous coronary intervention.

Class IIa

1. Coronary angiography with intent to perform revascularization is reasonable for patients with evidence of failed reperfusion or reocclusion after fibrinolytic therapy. Angiography can be performed as soon as logistically feasible.133–138,143 (Level of Evidence: B)

2. Coronary angiography is reasonable before hospital discharge in stable§ patients with STEMI after successful fibrinolytic therapy. Angiography can be performed as soon as logistically feasible, and ideally within 24 hours, but should not be performed within the first 2 to 3 hours after administration of fibrinolytic therapy.133-138,143 (Level of Evidence: B)

5.2. PCI of an Infarct Artery in Patients Who Initially Were Managed With Fibrinolysis or Who Did Not Receive Reperfusion Therapy

See Table 8 for a summary of recommendations from this section.

Table 7. Indications for Coronary Angiography in Patients Who Were Managed With Fibrinolytic Therapy or Who Did Not Receive Reperfusion Therapy

<table>
<thead>
<tr>
<th>COR</th>
<th>LOE</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiogenic shock or acute severe HF that develops after initial presentation</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Intermediate- or high-risk findings on predischarge noninvasive ischemia testing</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Spontaneous or easily provoked myocardial ischemia</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Failed reperfusion or reocclusion after fibrinolytic therapy</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td>Stable* patients after successful fibrinolysis, before discharge and ideally between 3 and 24 h</td>
<td>IIa</td>
<td>B</td>
</tr>
</tbody>
</table>

*Although individual circumstances will vary, clinical stability is defined by the absence of low output, hypotension, persistent tachycardia, apparent shock, high-grade ventricular or symptomatic supraventricular tachyarrhythmias, and spontaneous recurrent ischemia.

COR indicates Class of Recommendation; HF, heart failure; LOE, Level of Evidence; N/A, not available.

Table 8. Indications for PCI of an Infarct Artery in Patients Who Were Managed With Fibrinolytic Therapy or Who Did Not Receive Reperfusion Therapy

<table>
<thead>
<tr>
<th>COR</th>
<th>LOE</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiogenic shock or acute severe HF</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Intermediate- or high-risk findings on predischarge noninvasive ischemia testing</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Spontaneous or easily provoked myocardial ischemia</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Patients with evidence of failed reperfusion or reocclusion after fibrinolytic therapy (as soon as possible)</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td>Stable* patients after successful fibrinolysis, ideally between 3 and 24 h</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td>Stable* patients &gt;24 h after successful fibrinolysis</td>
<td>IIb</td>
<td>B</td>
</tr>
<tr>
<td>Delayed PCI of a totally occluded infarct artery &gt;24 h after STEMI in stable patients</td>
<td>III: No Benefit</td>
<td>B</td>
</tr>
</tbody>
</table>

*Although individual circumstances will vary, clinical stability is defined by the absence of low output, hypotension, persistent tachycardia, apparent shock, high-grade ventricular or symptomatic supraventricular tachyarrhythmias, and spontaneous recurrent ischemia.

§Although individual circumstances will vary, clinical stability is defined by the absence of low output, hypotension, persistent tachycardia, apparent shock, high-grade ventricular or symptomatic supraventricular tachyarrhythmias, and spontaneous recurrent ischemia.

Class IIa

1. Delayed PCI is reasonable in patients with STEMI and evidence of failed reperfusion or reocclusion after fibrinolytic therapy. PCI can be performed as soon as logistically feasible at the receiving hospital130,130a–130c (Level of Evidence: B)

2. Delayed PCI of a significant stenosis in a patent infarct artery is reasonable in stable§ patients with STEMI after fibrinolytic therapy. PCI can be performed as soon as logistically feasible at the receiving hospital, and ideally within 24 hours, but should not be performed within the first 2 to 3 hours after administration of fibrinolytic therapy.133–138 (Level of Evidence: B)

Class IIb

1. Delayed PCI of a significant stenosis in a patent infarct artery greater than 24 hours after STEMI
may be considered as part of an invasive strategy in stable§ patients.\textsuperscript{55,141-148} (Level of Evidence: B)

Class III: No Benefit

1. Delayed PCI of a totally occluded infarct artery greater than 24 hours after STEMI should not be performed in asymptomatic patients with 1- or 2-vessel disease if they are hemodynamically and electrically stable and do not have evidence of severe ischemia.\textsuperscript{55,146} (Level of Evidence: B)

5.3. PCI of a Noninfarct Artery Before Hospital Discharge

Class I

1. PCI is indicated in a noninfarct artery at a time separate from primary PCI in patients who have spontaneous symptoms of myocardial ischemia. (Level of Evidence: C)

Class IIa

1. PCI is reasonable in a noninfarct artery at a time separate from primary PCI in patients with intermediate- or high-risk findings on noninvasive testing.\textsuperscript{58,141,142} (Level of Evidence: B)

5.4. Adjunctive Antithrombotic Therapy to Support Delayed PCI After Fibrinolytic Therapy

See Table 9 for a summary of recommendations from this section.

5.4.1. Antiplatelet Therapy to Support PCI After Fibrinolytic Therapy

Class I

1. After PCI, aspirin should be continued indefinitely.\textsuperscript{76,77,80,82,121,122} (Level of Evidence: A)

2. Clopidogrel should be provided as follows:
   a. A 300-mg loading dose should be given before or at the time of PCI to patients who did not receive a previous loading dose and who are undergoing PCI within 24 hours of receiving fibrinolytic therapy (Level of Evidence: C);
   b. A 600-mg loading dose should be given before or at the time of PCI to patients who did not receive a previous loading dose and who are undergoing PCI more than 24 hours after receiving fibrinolytic therapy (Level of Evidence: C); and
   c. A dose of 75 mg daily should be given after PCI.\textsuperscript{80,121,122} (Level of Evidence: C)

Class IIa

1. After PCI, it is reasonable to use 81 mg of aspirin per day in preference to higher maintenance doses.\textsuperscript{76,82,86,87} (Level of Evidence: B)

2. Prasugrel, in a 60-mg loading dose, is reasonable once the coronary anatomy is known in patients who did not receive a previous loading dose of clopidogrel at the time of administration of a fibrinolytic agent, but prasugrel should not be given sooner than 24 hours after administration of a fibrin-specific agent or 48 hours after administration of a non–fibrin-specific agent.\textsuperscript{83,85} (Level of Evidence: B)

3. Prasugrel, in a 10-mg daily maintenance dose, is reasonable after PCI.\textsuperscript{83,85} (Level of Evidence: B)

Class III: Harm

1. Prasugrel should not be administered to patients with a history of prior stroke or transient ischemic attack.\textsuperscript{59} (Level of Evidence: B)

5.4.2. Anticoagulant Therapy to Support PCI After Fibrinolytic Therapy

Class I

1. For patients with STEMI undergoing PCI after receiving fibrinolytic therapy with intravenous UFH, additional boluses of intravenous UFH should be administered as needed to support the procedure, taking into account whether GP IIb/IIIa receptor antagonists have been administered. (Level of Evidence: C)

2. For patients with STEMI undergoing PCI after receiving fibrinolytic therapy with enoxaparin, if the last subcutaneous dose was administered within the prior 8 hours, no additional enoxaparin should be given; if the last subcutaneous dose was administered between 8 and 12 hours earlier, enoxaparin 0.3 mg/kg IV should be given.\textsuperscript{127,149} (Level of Evidence: B)

Class III: Harm

1. Fondaparinux should not be used as the sole anticoagulant to support PCI. An additional anticoagulant with anti-IIa activity should be administered because of the risk of catheter thrombosis.\textsuperscript{119} (Level of Evidence: C)

6. Coronary Artery Bypass Graft Surgery: Recommendations

6.1. CABG in Patients With STEMI

Class I

1. Urgent CABG is indicated in patients with STEMI and coronary anatomy not amenable to PCI who have ongoing or recurrent ischemia, cardiogenic shock, severe HF, or other high-risk features.\textsuperscript{151-152} (Level of Evidence: B)

2. CABG is recommended in patients with STEMI at time of operative repair of mechanical defects.\textsuperscript{153-157} (Level of Evidence: B)

Class IIa

1. The use of mechanical circulatory support is reasonable in patients with STEMI who are hemodynamically unstable and require urgent CABG. (Level of Evidence: C)

Class IIb

1. Emergency CABG within 6 hours of symptom onset may be considered in patients with STEMI who do not
have cardiogenic shock and are not candidates for 
PCI or fibrinolytic therapy. (Level of Evidence: C)

6.2. Timing of Urgent CABG in Patients With 
STEMI in Relation to Use of Antiplatet Agents

Class I

1. Aspirin should not be withheld before urgent 
CABG.158 (Level of Evidence: C)

2. Clopidogrel or ticagrelor should be discontinued at 
least 24 hours before urgent on-pump CABG, if possible.159–163 (Level of Evidence: B)

3. Short-acting intravenous GP IIb/IIIa receptor antago-
nists (eprotifibatide, tirofiban) should be discontinued 
at least 2 to 4 hours before urgent CABG.164,165 (Level of Evidence: B)

4. Abciximab should be discontinued at least 12 hours 
before urgent CABG.137 (Level of Evidence: B)

Class IIb

1. Urgent off-pump CABG within 24 hours of clopidog-
rel or ticagrelor administration might be consid-
ered, especially if the benefits of prompt revascular-
ization outweigh the risks of bleeding.166–168 (Level of Evidence: B)

2. Urgent CABG within 5 days of clopidogrel or ti-
cagrelor administration or within 7 days of prasu-
grel administration might be considered, especially 
if the benefits of prompt revascularization outweigh 
the risks of bleeding. (Level of Evidence: C)
7. Routine Medical Therapies: Recommendations

7.1. Beta Blockers

Class I

1. Oral beta blockers should be initiated in the first 24 hours in patients with STEMI who do not have any of the following: signs of HF, evidence of a low-output state, increased risk for cardiogenic shock, or other contraindications to use of oral beta blockers (PR interval more than 0.24 seconds, second- or third-degree heart block, active asthma, or reactive airways disease).168-171 (Level of Evidence: B)

2. Beta blockers should be continued during and after hospitalization for all patients with STEMI and with no contraindications to their use.172,173 (Level of Evidence: B)

3. Patients with initial contraindications to the use of beta blockers in the first 24 hours after STEMI should be reevaluated to determine their subsequent eligibility. (Level of Evidence: C)

Class IIa

1. It is reasonable to administer intravenous beta blockers at the time of presentation to patients with STEMI and no contraindications to their use who are hypertensive or have ongoing ischemia.169-171 (Level of Evidence: B)

7.2. Renin-Angiotensin-Aldosterone System Inhibitors

Class I

1. An angiotensin-converting enzyme inhibitor should be administered within the first 24 hours to all patients with STEMI with anterior location, HF, or ejection fraction less than or equal to 0.40, unless contraindicated.174-177 (Level of Evidence: A)

2. An angiotensin receptor blocker should be given to patients with STEMI who have indications for but are intolerant of angiotensin-converting enzyme inhibitors.178,179 (Level of Evidence: B)

3. An aldosterone antagonist should be given to patients with STEMI and no contraindications who are already receiving an angiotensin-converting enzyme inhibitor and beta blocker and who have an ejection fraction less than or equal to 0.40 and either symptomatic HF or diabetes mellitus.180 (Level of Evidence: B)

Class IIa

1. Angiotensin-converting enzyme inhibitors are reasonable for all patients with STEMI and no contraindications to their use.181-183 (Level of Evidence: A)

7.3. Lipid Management

Class I

1. High-intensity statin therapy should be initiated or continued in all patients with STEMI and no contraindications to its use.184,188,189 (Level of Evidence: B)

Class IIa

1. It is reasonable to obtain a fasting lipid profile in patients with STEMI, preferably within 24 hours of presentation. (Level of Evidence: C)

8. Complications After STEMI: Recommendations

8.1. Treatment of Cardiogenic Shock

Class I

1. Emergency revascularization with either PCI or CABG is recommended in suitable patients with cardiogenic shock due to pump failure after STEMI irrespective of the time delay from MI onset.54,190,191 (Level of Evidence: B)

2. In the absence of contraindications, fibrinolytic therapy should be administered to patients with STEMI and cardiogenic shock who are unsuitable candidates for either PCI or CABG.16,192,193 (Level of Evidence: B)

Class IIa

1. The use of intra-aortic balloon pump counterpulsation can be useful for patients with cardiogenic shock after STEMI who do not quickly stabilize with pharmacological therapy.194-197,197a (Level of Evidence: B)

Class IIb

1. Alternative left ventricular (LV) assist devices for circulatory support may be considered in patients with refractory cardiogenic shock. (Level of Evidence: C)

8.2. Implantable Cardioverter-Defibrillator Therapy Before Discharge

Class I

1. Implantable cardioverter-defibrillator therapy is indicated before discharge in patients who develop sustained ventricular tachycardia/ventricular fibrillation more than 48 hours after STEMI, provided the arrhythmia is not due to transient or reversible ischemia, reinfarction, or metabolic abnormalities.198-200 (Level of Evidence: B)

8.3. Pacing in STEMI

Class I

1. Temporary pacing is indicated for symptomatic bradycardias unresponsive to medical treatment. (Level of Evidence: C)
8.4. Management of Pericarditis After STEMI

Class I

1. Aspirin is recommended for treatment of pericarditis after STEMI.\(^\text{201}\) (Level of Evidence: B)

Class IIb

1. Administration of acetaminophen, colchicine, or narcotic analgesics may be reasonable if aspirin, even in higher doses, is not effective. (Level of Evidence: C)

Class III: Harm

1. Glucocorticoids and nonsteroidal anti-inflammatory drugs are potentially harmful for treatment of pericarditis after STEMI.\(^\text{202,203}\) (Level of Evidence: B)

8.5. Anticoagulation¶

Class I

1. Anticoagulant therapy with a vitamin K antagonist should be provided to patients with STEMI and atrial fibrillation with CHADS2 score\(^\text{#} \geq 2\), mechanical heart valves, venous thromboembolism, or hypercoagulable disorder. (Level of Evidence: C)

2. The duration of triple-antithrombotic therapy with a vitamin K antagonist, aspirin, and a P2Y\(_12\) receptor inhibitor should be minimized to the extent possible to limit the risk of bleeding.** (Level of Evidence: C)

Class IIa

1. Anticoagulant therapy with a vitamin K antagonist is reasonable for patients with STEMI and asymptomatic LV mural thrombi. (Level of Evidence: C)

Class IIb

1. Anticoagulant therapy may be considered for patients with STEMI and anteriorapical akinesis or dyskinesis. (Level of Evidence: C)

2. Targeting vitamin K antagonist therapy to a lower international normalized ratio (eg, 2.0 to 2.5) might be considered in patients with STEMI who are receiving DAPT. (Level of Evidence: C)

9. Risk Assessment After STEMI: Recommendations

9.1. Use of Noninvasive Testing for Ischemia Before Discharge

Class I

1. Noninvasive testing for ischemia should be performed before discharge to assess the presence and extent of inducible ischemia in patients with STEMI who have not had coronary angiography and do not have high-risk clinical features for which coronary angiography would be warranted.\(^\text{209-211}\) (Level of Evidence: B)

Class IIb

1. Noninvasive testing for ischemia might be considered before discharge to guide the postdischarge exercise prescription. (Level of Evidence: C)

9.2. Assessment of LV Function

Class I

1. LV ejection fraction should be measured in all patients with STEMI. (Level of Evidence: C)

9.3. Assessment of Risk for Sudden Cardiac Death

Class I

1. Patients with an initially reduced LV ejection fraction who are possible candidates for implantable cardioverter-defibrillator therapy should undergo reevaluation of LV ejection fraction 40 or more days after discharge.\(^\text{212-215}\) (Level of Evidence: B)

10. Posthospitalization Plan of Care: Recommendations

Class I

1. Posthospital systems of care designed to prevent hospital readmissions should be used to facilitate the transition to effective, coordinated outpatient care for all patients with STEMI.\(^\text{216-220}\) (Level of Evidence: B)

2. Exercise-based cardiac rehabilitation/secondary prevention programs are recommended for patients with STEMI.\(^\text{221-224}\) (Level of Evidence: B)

3. A clear, detailed, and evidence-based plan of care that promotes medication adherence, timely follow-up with the healthcare team, appropriate dietary and physical activities, and compliance with interventions for secondary prevention should be provided to patients with STEMI. (Level of Evidence: C)

4. Encouragement and advice to stop smoking and to avoid secondhand smoke should be provided to patients with STEMI.\(^\text{225-228}\) (Level of Evidence: A)
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**Key Words:** AHA Scientific Statements ▪ anticoagulants ▪ antiplatelets ▪ door-to-balloon ▪ fibrinolysis ▪ percutaneous coronary intervention ▪ reperfusion ▪ ST-elevation myocardial infarction
Appendix 1. Author Relationships With Industry and Other Entities (Relevant)—2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction

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†No financial benefit.
‡Significant relationship.
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ACS indicates acute coronary syndromes; DSMB, data safety monitoring board; NHLBI, National Heart, Lung, and Blood Institute; NIH, National Institutes of Health; and PI, principal investigator.
## Appendix 2. Reviewer Relationships With Industry and Other Entities (Relevant)—2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction

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Circulation. 2013;127:529-555; originally published online December 17, 2012;
doi: 10.1161/CIR.0b013e3182742c84

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

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| Jane A. Linderbaum    | Mayo Clinic—Assistant Professor of Medicine                                            | None                                                                                             |
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*Indicates significant ($\geq$ $10,000$) relationship.
†Indicates no financial benefit.

ACS indicates acute coronary syndromes; DSMB, data safety monitoring board; NHLBI, National Heart, Lung, and Blood Institute; NIH, National Institutes of Health; and PI, principal investigator.
Data Supplement 1. ECG Criteria for Diagnosis of STEMI in the Setting of LBBB

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CI indicates confidence interval.

Reprinted from Sgarbossa et al. (2). 8559200

In the NRMI-2 registry, 6.7% of MI patients had left bundle branch block (LBBB) and 6.2% had right bundle branch block (RBBB) on initial ECG (1). ECG diagnosis of STEMI in the setting of RBBB and left anterior and posterior fascicular blocks does not require special diagnostic criteria. However, interpreting the ST-segments is more difficult in patients with LBBB. Criteria for the ECG diagnosis of STEMI in the setting of LBBB have been developed and may help identify patients presenting with chest pain and LBBB who are more likely to be experiencing an MI. Sgarbossa identified 3 criteria used in a 10-point scale that improved the specificity of the diagnosis of STEMI in patients with LBBB: ST-elevation of at least 1 mm that was concordant with the QRS complex (5 points), ST-segment depression of at least 1 mm in lead V1, V2, or V3 (3 points), and ST-elevation of at least 5 mm that was discordant with the QRS complex (2 points) (2). A meta-analysis of studies exploring the utility of the Sgarbossa criteria demonstrated that a score of 3 had a specificity of 98% for acute myocardial infarction, but a score of 0 did not rule out STEMI (3). 18342992.
# Data Supplement 2. PCI for Cardiac Arrest Evidence

<table>
<thead>
<tr>
<th>Study Name</th>
<th>Aim of study</th>
<th>Study Type</th>
<th>Study Size</th>
<th>Patient Population/Inclusion &amp; Exclusion Criteria</th>
<th>Endpoint</th>
<th>Statistical Analysis Reported</th>
<th>P-Values &amp; 95% CI</th>
<th>OR: HR: RR:</th>
<th>Study Summary</th>
<th>Study Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary coronary angioplasty for AMI complicated by OOH-CA. Kahn at al., 1995 (4)</td>
<td>First report of PPCI in OOH-CA pts</td>
<td>Case series</td>
<td>11</td>
<td>Clinical judgment of cardiologist. No prespecified criteria used.</td>
<td>Survival to hospital discharge</td>
<td>Neurological outcome</td>
<td>None</td>
<td></td>
<td>Single institution, Selection bias</td>
<td></td>
</tr>
<tr>
<td>Immediate coronary angiography in survivors of OOH-CA. Spaulding at al., 1997 (5) 9171064</td>
<td>Determine impact of PPCI on OOH-CA survivors</td>
<td>Consecutive case series</td>
<td>84</td>
<td>OOH-CA, 30-75 y, &lt;6 h onset of symptoms in pts previously leading a normal life, no obvious noncardiac etiology.</td>
<td>Survival to hospital discharge</td>
<td>Prevalence of CAD on angiography</td>
<td>Multivariate logistic regression showed successful PPCI was an independent predictor of survival.</td>
<td>p&lt;0.04; 95% CI: 1.1 - 24.5</td>
<td>OR: 5.2</td>
<td>84 pt OOH-CA consecutive pts brought to cath/PPCI. 48% had acute coronary occlusion. Presence of chest pain, ECG ST-elevation poor predictors. Successful PCI independent predictor of survival.</td>
</tr>
<tr>
<td>Early direct coronary angioplasty in survivors of OOH-CA. Keelan et al., (6) 12804734</td>
<td>Determine impact of PPCI on OOH-CA VF survivors</td>
<td>Case series</td>
<td>15</td>
<td>OOH-CA, VF initial rhythm not VF</td>
<td>Survival to hospital discharge</td>
<td>None</td>
<td></td>
<td></td>
<td>15 pts with OOH-CA due to VF treated with PPCI, 11/14 survived.</td>
<td>Selection bias</td>
</tr>
<tr>
<td>Impact of PCI or CABG on outcome after nonfatal CA outside the hospital. Borger van der Burg et al., 2003 (7) 12667561</td>
<td>Determine impact of revascularization on outcome from OOH-CA</td>
<td>Case series</td>
<td>142</td>
<td>OOH-CA, VFpVT as initial rhythm</td>
<td>2 y recurrence-free survival</td>
<td>Survival to hospital discharge</td>
<td>Kaplan-Meier</td>
<td>p&lt;0.001</td>
<td>142 non-AMI, OOH-CA pts. Revascularized pts had a better recurrence-free survival.</td>
<td>Nonrandomized case series, selection bias</td>
</tr>
</tbody>
</table>
### Long-term prognosis after OOH-CA and PPCI

<table>
<thead>
<tr>
<th>Study</th>
<th>Authors</th>
<th>Year</th>
<th>Patients</th>
<th>Follow-up</th>
<th>Methodology</th>
<th>Survival to hospital discharge</th>
<th>p=NS between groups after discharge from hospital</th>
<th>Outcome after 36 mo</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bendz et al., 2004</td>
<td>8</td>
<td>40</td>
<td>OOH-CA, STEMI</td>
<td>Kaplan-Meier comparison of 36 mo survival in OOH-CA STEMI pts receiving PPCI (n=40) vs nonarrest STEMI pts receiving PPCI (n=325)</td>
<td>p=NS between groups after discharge from hospital</td>
<td>Found no significant difference in 36 mo survival in OOH-CA STEMI pts receiving PPCI (n=40) vs nonarrest STEMI pts receiving PPCI (n=325).</td>
<td>Nonrandomized case series, selection bias</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Treatment and outcome in post-resuscitation care after OOH-CA when a modern therapeutic approach was introduced.

<table>
<thead>
<tr>
<th>Study</th>
<th>Authors</th>
<th>Year</th>
<th>Patients</th>
<th>Methodology</th>
<th>Survival to hospital discharge</th>
<th>Factors associated with survival:</th>
<th>36 mo survival in OOH-CA STEMI pts receiving PPCI (n=40) vs nonarrest STEMI pts receiving PPCI (n=325).</th>
</tr>
</thead>
<tbody>
<tr>
<td>Werling et al., 2007</td>
<td>9</td>
<td>85</td>
<td>OOH-CA</td>
<td>Fisher's exact test</td>
<td>Initial VF p=0.002; coronary angiography p=0.003; PCI p=0.01; CABG p=0.001</td>
<td>85 pt case series, factors associated with increased survival: initial VF; coronary angiography; PCI; CABG, PCI or CABG.</td>
<td>Selection bias</td>
</tr>
</tbody>
</table>

### Six-month outcome of emergency PCI in resuscitated pts after CA complicating STEMI

<table>
<thead>
<tr>
<th>Study</th>
<th>Authors</th>
<th>Year</th>
<th>Patients</th>
<th>Methodology</th>
<th>Survival to 6 mo after hospital discharge</th>
<th>Factors associated with 6 mo survival in pts receiving PPCI: absence of shock 12.7%; absence of diabetes 7.3%; absence of prior PCI 11.0%;</th>
<th>186 pts resuscitated from OOH-CA complicating acute MI; factors associated with 6 mo survival in pts receiving PPCI: absence of shock; absence of diabetes; absence of prior PCI.</th>
<th>Selection bias</th>
</tr>
</thead>
<tbody>
<tr>
<td>Garot al., 2007</td>
<td>10</td>
<td>186</td>
<td>OOH-CA, STEMI referred for PCI</td>
<td>Multiple stepwise regression</td>
<td>Factors associated with 6 mo survival in pts receiving PPCI: absence of shock 12.7%; absence of diabetes 7.3%; absence of prior PCI 11.0%;</td>
<td>186 pts resuscitated from OOH-CA complicating acute MI; factors associated with 6 mo survival in pts receiving PPCI: absence of shock; absence of diabetes; absence of prior PCI.</td>
<td>Selection bias</td>
<td></td>
</tr>
</tbody>
</table>

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# 2013 STEMI Guideline Data Supplements

<table>
<thead>
<tr>
<th>PPCI after OOH-CA: pts and outcomes. Markusohn et al., 2007 (11) 17491217</th>
<th>To define the demographic, clinical and angiographic characteristics, and the prognosis of STEMI pts undergoing primary PCI after OOH-CA</th>
<th>Case series</th>
<th>25</th>
<th>OOH-CA, STEMI</th>
<th>1 y survival</th>
<th>1 y survival without severe disability</th>
<th>25 OOH-CA, STEMI pts receiving PPCI. 1 y survival 72%; 1 y survival without severe disability 64%.</th>
<th>Selection bias</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute STEMI after successful CPR. Gorjup et al., 2007 (12) 17161902</td>
<td>To define the demographic, clinical and angiographic characteristics, and the prognosis of STEMI pts undergoing primary PCI after OOH-CA</td>
<td>Case series</td>
<td>135</td>
<td>CA, STEMI</td>
<td>Survival to hospital discharge with CPC 1 or 2</td>
<td>Ordinal logistic regression</td>
<td>Predictors of hospital survival with CPC 1 or 2: smoking OR: 0.57; 95% CI: 0.36-0.89; inhospital arrest OR: 0.31; 95% CI: 0.18-0.54; shockable rhythm OR: 0.66; 95% CI: 0.53-0.81; motor response to pain OR: 0.32; 95% CI: 0.19-0.57; pupil light reflexes OR: 0.10; 95% CI: 0.01-0.64; breathing OR: 0.29; 95% CI: 0.16-0.52; seizures OR: 1.39; 95% CI: 1.08-1.77; PPCI OR: 0.69, 95% CI: 0.56-0.84</td>
<td>135 pts with STEMI, CA; predictors of survival included smoking, inhospital CA, shockable rhythm, neurological status on admission, PPCI</td>
</tr>
</tbody>
</table>
### 2013 STEMI Guideline Data Supplements

<table>
<thead>
<tr>
<th>Study Description</th>
<th>Case Series/Study Design</th>
<th>Case Number</th>
<th>Main Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Thrombolytic therapy vs PPCI after VF CA due to STEMI and its effect on outcome</strong></td>
<td>Case series</td>
<td>147</td>
<td>Witnessed OOH-CA, STEMI, VF initial rhythm, ROSC, treated with either thrombolysis or PPCI.</td>
</tr>
<tr>
<td><strong>Survival and neurologic recovery in pts with STEMI resuscitated from CA</strong></td>
<td>Case series</td>
<td>98</td>
<td>OOH-CA, STEMI</td>
</tr>
<tr>
<td><strong>Coronary angiography predicts improved outcome following CA: propensity-adjusted analysis</strong></td>
<td>Case series</td>
<td>241</td>
<td>CA</td>
</tr>
</tbody>
</table>

*Selection bias* for non-randomized studies.
## 2013 STEMI Guideline Data Supplements

<table>
<thead>
<tr>
<th>Study Description</th>
<th>Study Design</th>
<th>Case Series</th>
<th>Finding Description</th>
<th>Analysis Method</th>
<th>Multivariable Analysis</th>
<th>Selection Bias</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute coronary angiographic findings in survivors of OOH-CA</td>
<td>Case series</td>
<td>72 OOH-CA</td>
<td>Coronary angiographic findings</td>
<td>Survival to hospital discharge</td>
<td>64% had angiographic CAD, 38% had an acute lesion; PCI attempted in 33% ROSC; OR: 0.0004; need for inotropic support during angiography p=0.0009</td>
<td>Independent predictors of hospital death: prolonged interval from CA onset to ROSC OR: 14.6; 95% CI: 3.3-63.5; need for inotropic support during angiography OR: 11.2; 95% CI: 2.7-46.9</td>
</tr>
<tr>
<td>Anyfantakis et al., 2009 (16)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Assess the prevalence of coronary lesions in OOH-CA survivors</td>
<td></td>
<td></td>
<td>Coronary angiographic findings</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emergent PCI for resuscitated victims of OOH-CA</td>
<td>Case series</td>
<td>5 OOH-CA</td>
<td>Coronary angiographic and ECG findings</td>
<td>Combining these therapies resulted in long-term survival rates of 70% with &gt;80% of all such survivors neurologically functional</td>
<td></td>
<td>Selection bias</td>
</tr>
<tr>
<td>Kern et al., 2010 (17)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Assess the value of early angiography/PCI and hypothermia in OOH-CA</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>AMI indicates acute myocardial infarction; CA, cardiac arrest; CABG, coronary artery bypass graft surgery; CAD, coronary artery disease; cath, catheterization; CI, confidence interval; CPC, circulating progenitor cell; CPR, cardio pulmonary resuscitation; CPT, current procedural terminology; ECG, electrocardiogram; EP, electrophysiology; GCS, Glasgow coma scale; n, number; NS, nonsignificant; OOH-CA, out-of-hospital cardiac arrest; PCI, percutaneous coronary intervention; PPCI, primary percutaneous coronary intervention; pt, patient; pVT, paroxysmal ventricular tachycardia; ROSC, return of spontaneous circulation; STEMI, ST-elevation myocardial infarction; VF, ventricular fibrillation; and VT, ventricular tachycardia.</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial Name</td>
<td>Study Type</td>
<td>N</td>
<td>n</td>
<td>Study Population (experimental and comparator/control)</td>
<td>Primary Efficacy Endpoint</td>
<td>Primary Safety Endpoint</td>
</tr>
<tr>
<td>------------------</td>
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<td>--------------------------------------------------------</td>
<td>----------------------------</td>
<td>------------------------</td>
</tr>
<tr>
<td>CURRENT-OASIS 7</td>
<td>RCT</td>
<td>25,087</td>
<td>7327</td>
<td>Cardiovascular death, MI, and stroke at 30 d: double-dose clopidogrel 4.2% vs standard-dose clopidogrel 4.4%, HR: 0.94; 95% CI: 0.83-1.06; p=0.30; higher-dose ASA 4.2% vs lower-dose ASA 4.4%, HR 0.97; 95% CI: 0.86-1.09; p=0.61. Major bleeding: double-dose clopidogrel 2.5% vs standard-dose clopidogrel 2.0%, HR: 1.24; 95% CI: 1.05-1.46; p=0.01; higher-dose ASA 2.3% vs lower-dose ASA 2.3%, HR: 0.99; 95% CI 0.84-1.17; p=0.90. Prespecified subgroup analyses (both clopidogrel and ASA dose comparisons included) qualifying condition (STEMI vs non-STEMI, age &gt;65 or &gt;75 y, body weight &lt;60 kg, prior stroke/TIA). Additional prespecified subgroup analyses for the clopidogrel dose comparison included: ACS (STEMI) subjects undergoing PCI vs those not undergoing PCI. In the subgroup of pts who underwent PCI after randomization (69%, n=17263), double-dose clopidogrel was associated with a significant reduction in the rate of the prespecified secondary outcome of stent thrombosis (1.6% vs 2.3%; HR: 0.68; 95% CI: 0.55-0.85; p&lt;0.001 and 0.7% vs 1.3% for definite stent thrombosis, HR: 0.54; 95% CI: 0.39-0.74; p=0.0001). There was also reduction of the prespecified outcome of probable or definite (by ARC criteria) stent thrombosis consistent across DES and non-DES subtypes. In addition, double-dose clopidogrel reduced the rate of the primary composite outcome in this subgroup (3.9% vs 4.5%, HR: 0.88; 95% CI: 0.74-0.99; p=0.039). Higher and lower dose ASA did not differ with respect to the primary composite outcome. Major bleeding occurred more frequently with double-dose clopidogrel (1.6% vs 1.1%; HR: 1.41; 95% CI: 1.09-1.83; p=0.009.)</td>
<td>In pts with ACS including STEMI referred for an invasive strategy, there was no significant difference between a 7 d double-dose clopidogrel regimen and the standard dose regimen, or between higher dose ASA and lower dose ASA, with respect to the primary outcome of cardiovascular death, MI or stroke.</td>
<td></td>
</tr>
</tbody>
</table>
TRITON-TIMI 38 trial (19) 19249633

RCT

13,608 pts with moderate to high risk ACS

3534 (26%)

Pts with moderate to high risk ACS undergoing planned invasive strategy randomized to prasugrel (60 mg LD and a 10 mg daily maintenance dose) or clopidogrel (300 mg LD and a 75 mg daily maintenance dose), for 6 to 15 mo.

Cardiovascular death, nonfatal MI, or nonfatal stroke at 15 mo: prasugrel 9.9% vs clopidogrel 12.1%; HR: 0.81; 95% CI 0.73-0.90; p< 0.001. The HR for prasugrel, as compared with clopidogrel, for the primary efficacy endpoint at 30 d was HR: 0.77; 95% CI 0.67-0.88; P<0.001 and at 90 d HR: 0.80; 95% CI 0.71-0.90; p<0.001. The difference between the treatment groups with regard to the rate of the primary endpoint was largely related to a significant reduction in MI in the prasugrel group (9.7% in the clopidogrel group vs 7.4% in the prasugrel group; HR: 0.76; 95% CI 0.67-0.85; p<0.001).

Major bleeding was observed in 2.4% of pts receiving prasugrel and in 1.8% of pts receiving clopidogrel (HR: 1.32; 95% CI 1.03-1.68; p=0.03). Also greater in the prasugrel group was the rate of life-threatening bleeding (1.4% vs 0.9%; p=0.01), including nonfatal bleeding (1.1% vs 0.9%; HR: 1.25; p=0.23) and fatal bleeding (0.4% vs 0.1%; p=0.002) and CABG-related TIMI major bleeding (13.4% vs 3.2%; HR: 4.73; 95%CI 1.9 - 11.2; p<.001).

Major benefit of prasugrel was observed in the STEMI cohort alone (HR: 0.79; 95% CI, 0.65 - 0.97; P = 0.02). The benefit with prasugrel tended to be greater among the 3146 pts with diabetes (17.0% of whom had the primary end point in the clopidogrel group, vs 12.2% in the prasugrel group; HR: 0.70; 95% CI 0.58-0.85; p=0.001) than among 10,462 pts without diabetes (10.6% of whom had the primary endpoint in the clopidogrel group, vs 9.2% in the prasugrel group; HR: 0.86; 95% CI: 0.76-0.98; p= 0.02). The rate of definite or probable stent thrombosis, as defined by the Academic Research Consortium, was significantly reduced in the prasugrel group as compared with the clopidogrel group, with 68 pts (1.1%) and 142 pts (2.4%), respectively, having at least 1 occurrence (HR: 0.48; 95% CI 0.36 - 0.64; p<0.001). Pts who had a previous stroke or TIA had net harm from prasugrel (HR:1.54; 95% CI: 1.02-2.32; p=0.04), pts age ≥75 y had no net benefit from prasugrel (HR: 0.99; 95% CI: 0.81-1.21; P = 0.92), and pts weighing <60 kg had no net benefit from prasugrel (HR: 1.03; 95% CI: 0.69 -1.53; p=0.89)

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PLATO (20) 21060072

RCT 18,624

ACS pts 7026 (38%)

Pts with ACS with or without ST-elevation randomized to ticagrelor (180-mg LD, 90 mg twice daily thereafter) vs clopidogrel (300- or 600-mg LD, 75 mg daily thereafter)

Primary composite endpoint: death from vascular causes, MI, or stroke at 12 mo: 9.8% ticagrelor group vs 11.7% clopidogrel group, HR: 0.84; 95% CI: 0.77-0.92; p<0.001.

Age, sex, weight, final diagnosis, time from index event to treatment, troponin I, diabetes mellitus, previous MI, previous CABG, ASA during first hospital admission, GP IIb/IIIa during first hospital admission, geographical region, OL clopidogrel before randomization, total clopidogrel (OL+IP) before randomization to 24 h after first dose IP

Primary PCI subgroup. Definite Stent thrombosis HR: 0.66; p=0.03; MI HR: 0.80; p=0.03

The rate of death from any cause was also reduced with ticagrelor (4.5%, vs 5.9% with clopidogrel; p=0.001). In the ticagrelor group, there was a higher rate of non–CABG-related major bleeding (4.5% vs 3.8%, p=0.03). Episodes of intracranial bleeding (26 [0.3%] vs 14 [0.2%]; p=0.06), including fatal intracranial bleeding were more frequent with ticagrelor (11 [0.1%] vs 1 [0.01%]; p=0.02). There were fewer episodes of other types of fatal bleeding in the ticagrelor group (9 [0.1%], vs 21 [0.3%]; p=0.03).

ARMYDA-6 MI (21) 21958886

RCT 201

201 (100%)

Pts undergoing primary PCI for STEMI randomized to a 600 mg (n=103) or 300 mg (n=98) clopidogrel LD before the procedure

Primary Endpoint: Infarct size determined as the AUC of cardiac biomarkers: 600 mg LD median CK-MB 2,070 ng/mL (IQR: 815 to 2,847 ng/mL) vs 300 mg LD 3,049 ng/mL (IQR: 1,050 to 7,031 ng/mL) in the 300-mg group, p=0.0001; 600 mg LD troponin-I 255 ng/mL (IQR: 130 to 461 ng/mL) vs 300 mg LD 380 ng/mL (IQR: 134 to 1,406 ng/mL), p<0.0001.

30 d bleeding and entry site complications.

Major bleeding: 1.9% in 600 mg group vs 2.0% in 300 mg group.

Entry site complications 2.9% vs 2.1%.

N/A

TIMI flow grade <3 after PCI 600 mg LD 5.8% vs 300 mg LD 16.3%, p=0.031; LVEF at discharge 600 mg LD 52.1 ± 9.5% vs 300 mg LD 48.9 ± 11.3%, p=0.026; 30-d MACE 600 mg LD 5.8% vs 300 mg LD 15%, p=0.049. No difference in bleeding or access site complications.

Surrogate endpoint trial underpowered for clinical events. Measurement of AUC less accurate than cardiac MRI for assessment of infarct size.

ARC indicates Academic Research Consortium; ASA, aspirin; AUC, area under the curve; ARMYDA-6 MI, Antiplatelet therapy for Reduction of Myocardial Damage during Angioplasty-Myocardial Infarction study; CABG, coronary artery bypass surgery; CURRENT-OASIS 7: Clopidogrel and ASA Optimal Dose Usage to Reduce Recurrent Events–Seventh Organization to Assess Strategies in Ischemic Syndromes; DES, drug-eluting stents; GRACE, Global Registry of Acute Coronary Events risk score; GUSTO, Global Use of Strategies To Open Occluded Coronary Arteries; IQR, interquartile range; IP, investigational product; LBBB, left bundle branch block; LD, loading dose; LVEF, left ventricular ejection fraction; MACE, major adverse cardiovascular events; MI, myocardial infarction; MRI, magnetic resonance imaging; PCI, percutaneous coronary intervention; PLATO, Platelet Inhibition and Patient Outcomes trial; pts, patients; OL, open label; STEMI, ST-elevation myocardial infarction; TIA, transient ischemic attack, and TIMI, Thrombolysis In Myocardial Infarction trial.
## 2013 STEMI Guideline Data Supplements

### Data Supplement 4. Early Catheterization and Rescue PCI for Fibrinolytic Failure in the Stent Era

<table>
<thead>
<tr>
<th>Study Name</th>
<th>Study Type</th>
<th>Study Size</th>
<th>Inclusion Criteria</th>
<th>Endpoints</th>
<th>Findings</th>
<th>Limitations</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>MERLIN, 2004</td>
<td>Randomized multicenter study of rescue angioplasty compared with continued medical therapy for pts with acute STEMI and failed thrombolysis.</td>
<td>15261920</td>
<td>STEMI ≤10 h of onset of symptoms, CP &gt;30 min ST-elevation ≥2 mm in ≥2 chest leads or 1 mm in ≥2 limb leads. Failure to respond to FT at 60 min.</td>
<td>All-cause mortality at 30 d.</td>
<td>Death: Conservative vs rescue = 11% vs 9.8%; p=0.7 RD: 1.2; 95% CI: -5.6-8.3</td>
<td>Composite Secondary EP: 50% vs 37.3%; p=0.02; RD: 12.7%; 95% CI: 1.6-23.5</td>
<td>Strokes: 4.6% vs 0.6%; p=0.03</td>
</tr>
<tr>
<td>REACT, 2005</td>
<td>Randomized multicenter study to determine the best treatment for failed fibrinolysis by comparing rescue PCI to repeat fibrinolysis to conservative therapy.</td>
<td>16382062</td>
<td>Age 21 to 85 y, with evidence of failure of fibrinolysis; Rescue PCI could be performed within 12 h of onset of CP.</td>
<td>Composite of death, re-MI, CVA or severe CHF at 6 mo.</td>
<td>Rescue PCI vs repeat FT vs Conservative: 15.3% vs 31% vs 29.8%; p=0.003</td>
<td>PCI vs conservative: HR: 0.47; 95% CI: 0.28-0.79; p=0.004</td>
<td>PCI vs Re-FT: HR: 0.43; 95% CI: 0.26-0.72; p=0.001</td>
</tr>
<tr>
<td>Collet et al., 2006</td>
<td>Meta-analysis of clinical trials of cath following fibrinolysis in various settings. This included Rescue PCI, Immediate PCI (within 24 h) and Facilitated PCI. Focus of this table is on data from rescue PCI.</td>
<td>17250887, 17010790</td>
<td>Trials of pts with failed fibrinolysis randomized to rescue PCI or conservative care.</td>
<td>Mortality and Re-MI</td>
<td>Short term mortality: OR: 0.63; 95% CI: 0.39-0.99; p=0.055</td>
<td>Long term mortality: OR: 0.69; 95% CI: 0.41-1.57; p=0.16</td>
<td>Short term mortality or Re-MI: OR: 0.60; 95% CI: 0.41-0.89; p=0.012</td>
</tr>
</tbody>
</table>
### 2013 STEMI Guideline Data Supplements

<table>
<thead>
<tr>
<th>Wijeysundera et al., 2007 (24) <strong>17259087</strong></th>
<th>Meta-analysis of the benefits of rescue PCI compared with either repeat fibrinolysis or conservative care.</th>
<th>1,177</th>
<th>Trials of pts with clinical or angiographic evidence of failed fibrinolysis randomized to rescue PCI, repeat fibrinolysis or conservative care.</th>
<th>Mortality and Re-MI, CHF, CVA, and bleeding</th>
<th>Higher rate of major bleeding with rescue PCI</th>
<th>Differences in study protocol, study endpoints and duration of follow-up.</th>
<th>Meta-analysis supported rescue PCI compared with conservative care in pts with clinical or angiographic evidence of failure of FT at the expense of a higher incidence of CVA and bleeding complications.</th>
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<td>Rescue PCI vs Conservative:</td>
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<td>Mortality: RR: 0.69; 95% CI: 0.46-1.05; p=0.09</td>
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<td>CHF: RR:0.73; 95% CI: 0.54-1.0; p=0.05</td>
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<td>Re-MI: RR:0.58; 95% CI: 0.35-0.97; p=0.04</td>
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<td>Composite of Death: re-MI and CHF RR: 0.72; 95% CI: 0.59-0.88; p=0.001</td>
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<td>CVA: RR: 4.98, 95% CI: 1.1- 22.5; p=0.04</td>
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<td>Minor bleeding: RR: 4.58, 95% CI: 2.46-8.55; p&lt;0.001</td>
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<td>Rescue PCI vs repeat FT: Mortality RR: 0.68; 95% CI: 0.41-1.14; p=0.14</td>
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<td>Re-MI: RR:1.79; 95% CI: 0.92-3.48; p=0.09</td>
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<td>Minor bleeding: RR: 1.84; 95% CI: 1.06-3.18; p=0.03</td>
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<td>Major bleeding: RR: 1.54; 95% CI: 0.54-4.4; p=0.42</td>
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</table>

Cath indicates catheterization; CHF, congestive heart failure; CI, confidence interval; CP, chest pain; CVA, cerebrovascular accident; FT, fibrinolytic therapy; MI, myocardial infarction; PCI, percutaneous coronary intervention; pts, patients; RD, risk difference; RWMI, regional wall-motion index; and STEMI, ST-elevation myocardial infarction.
### 2013 STEMI Guideline Data Supplements

**Table: Early Catheterization and PCI Following Fibrinolysis in the Stent Era**

<table>
<thead>
<tr>
<th>Study Name</th>
<th>Study Type</th>
<th>Study Size</th>
<th>Inclusion Criteria</th>
<th>Endpoints</th>
<th>Findings</th>
<th>Limitations</th>
<th>Comments</th>
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</thead>
<tbody>
<tr>
<td>SIAM III, 2003 (26) 12932593</td>
<td>Randomized multicenter trial of immediate stenting within 6 h of fibrinolysis vs delayed stenting at 2 wk.</td>
<td>195</td>
<td>Age &gt;18 y, symptoms of AMI &lt;12 h, ST-elevation of &gt;1 mm in ≥2 limb leads and ST-elevation &gt;2 mm in precordial leads, or new LBBB; no contraindication to lytics.</td>
<td>Composite of death, re-MI, ischemic events and TLR at 6 mo.</td>
<td>Early stent vs delayed stent MACE: 25.6% vs 50.6%; p=0.001 No differences in bleeding complications.</td>
<td>Study demonstrated a benefit of immediate stenting performed within 6 h of FT as compared with a strategy of delayed stenting. This was primarily driven by reduction in ischemic events (by definition, a pt. in delayed stent arm who required cath before 2 wk was considered to have reached an ischemic endpoint.)</td>
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<td>GRACIA, 2004 (27) 15380963</td>
<td>Randomized multicenter study of routine early cardiac cath (6 to 24 h) following fibrinolysis vs ischemia guided approach.</td>
<td>500</td>
<td>Pts ≥18 y with ST-elevation ≥1 mm in ≥2 contiguous leads, or a nondiagnostic ECG due to LBBB or paced rhythm; symptoms ≥30 min and ≤12 h unresponsive to NTG treated with a fibrin specific agent and consented 6 h after FT.</td>
<td>Composite of death, re-MI and ischemia induced revascularization at 1 y.</td>
<td>Early Cath vs Ischemia Guided RR: 0.44; 95% CI: 0.28 - 0.70; p=0.0008 Endpoint of death or re-MI: HR: 0.58; 95% CI: 0.33-1.05; p=0.07 No difference in major bleeding</td>
<td>Pts randomized 6 h after FT Study demonstrated a benefit of early routine cath compared with an ischemia driven approach. This was largely seen by a 70% reduction in ischemia driven revascularization in the invasive group compared with conservative group at 1 y.</td>
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<tr>
<td>Leipzig Prehospital Fibrinolysis Study, 2005 (28) 16061501</td>
<td>Randomized multicenter study of prehospital fibrinolysis with PCI vs prehospital fibrinolysis alone and standard care.</td>
<td>164</td>
<td>Symptoms for at least 30 min and &lt;6 h, and ST-elevation &gt;0.1 mV in ≥2 limb leads or &gt;0.2 mV in ≥2 precordial leads.</td>
<td>Final infarct size by MRI.</td>
<td>Early Cath vs Standard Care Final infarct size on MRI: 5.2% (IQR: 1.3 to 11.2) vs 10.4% (3.4 to 16.3) p=0.001 Trend towards fewer clinical events.</td>
<td>Small study and surrogate endpoints Immediate cath and PCI following fibrinolysis resulted in smaller infarct size on MRI compared with standard care.</td>
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<tr>
<td>CAPITAL AMI, 2005 (29) 16053952</td>
<td>Randomized multicenter study of fibrinolysis with immediate transfer for cath vs fibrinolysis alone and transfer for unstable symptoms.</td>
<td>170</td>
<td>Symptoms ≤8 h and ≥30 min; ST-elevation ≥1 mm in ≥2 leads or LBBB and 1 of the following: AMI; Extensive nonanterior MI; Killip class 3, SBP (22) &lt;100 mmHg</td>
<td>Composite of death, re-MI, re-UA or CVA at 6 mo.</td>
<td>Early Cath vs Ischemia Guided Approach MACE: 11.6% vs 24.4%; p=0.04 RR: 0.48; 95% CI: 0.24-0.96 Minor bleeding higher in the early cath group. No differences in major bleeding.</td>
<td>Small study, with mix of transfer pts or pts at centers with PCI capabilities. “Standard” care group was managed very conservatively. Demonstrated a benefit to immediate cath compared with standard care (which was stress test at 30 d). This was primarily driven by less recurrent MI or UA in the PCI group within the 1st wk of care.</td>
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<tr>
<td>Di Pasquale et al., 2006 (30) 16022610</td>
<td>Randomized single-center study of immediate cath &lt;2 h and PCI vs delayed PCI 12 to 24 h after fibrinolysis.</td>
<td>451</td>
<td>First STEMI ≤12 h from symptom onset, with ST-elevation &gt;1 mm in peripheral leads, and or 2 mm in ischemic events (MI, abnormal stress test, restenosis, and death) at 6 mo.</td>
<td>Immediate Cath vs Delayed Cath Ischemic events 18.2% vs 9.7%; p=0.005 More minor bleeding in immediate PCI</td>
<td>Pts only included following successful reperfusion.</td>
<td>Study failed to show a benefit to immediate cath and PCI within 2 h, compared with early cath and PCI at 12 to 24 h among pts who have demonstrated evidence of successful</td>
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<td>Study</td>
<td>Design</td>
<td>Participants</td>
<td>Methods</td>
<td>Results</td>
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<td><strong>WEST, 2006</strong> (31) [16757491]</td>
<td>Randomized multicenter feasibility study of PCI vs fibrinolysis with early cath (within 24 h) vs fibrinolysis with standard care.</td>
<td>304 Nonpregnant, ≥18 y, symptoms at least 20 min and ECG with high-risk MI (ST-elevation ≥2 mm in 2 precordial leads or 2 limb leads, or ≥1 mm ST-elevation in limb leads with ≥1 mm ST depression in precordial leads, or presumed new LBBB).</td>
<td>Efficacy: 30 d composite of death, re-MI, reischemia, CHF, shock or major ventricular arrhythmias. Safety endpoints: ICH, CVA, major bleeding.</td>
<td>No difference in the primary efficacy or safety endpoints in the 3 groups. Very small study Feasibility study failed to show a difference in efficacy or safety endpoints for the 3 approaches. A subsequent analysis compared a strategy of primary PCI with fibrinolysis (with or without early cath) and showed a lower rate of 30-d death and MI in the primary PCI group (HR: 0.29; 95% CI: 0.11-0.74; P-log rank=0.021)</td>
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<td><strong>Collet et al., 2006</strong> (25) [17010790]</td>
<td>Meta-analysis of clinical trial of cath following fibrinolysis in various settings. This included rescue PCI, immediate PCI (within 24 h) and facilitated PCI. Focus in this table on results from immediate cath.</td>
<td>1,508 Clinical trials of STEMI pts receiving fibrinolysis and randomized to immediate or early cath compared with ischemia driven cath (excluded trials that looked at early vs delayed cath).</td>
<td>Mortality and Death/MI Early Cath vs Ischemia Driven Cath Death: All studies: OR: 0.83; 95% CI: 0.52-1.35; p=0.47 Stent era: OR: 0.56; 95% CI: 0.29-1.05; p=0.07 POBA: OR: 1.44; 95% CI: 0.69-3.06; p=0.33) Death and MI All studies: OR: 0.85; 95% CI: 0.47-1.55; p=0.42 Stent era: OR: 0.53; 95% CI: 0.33-0.83; p=0.0067 POBA: OR: 1.76; 95% CI: 0.97-3.21; p=0.064</td>
<td>Different regimens of medications and timing to cath and different time periods in which trials were performed. Investigators reviewed overall results of all studies, and then examined the results from studies performed in the stent era. Study showed a benefit to systematic early cath compared with an ischemia driven approach from studies performed in the “stent era” but not for studies performed in the “balloon angioplasty era”.</td>
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<td><strong>Wijeysundera, 2008</strong> (24) [17258087]</td>
<td>A meta-analysis of trials examining fibrinolysis with immediate transfer for cath with</td>
<td>1,235 Clinical trials of STEMI pts receiving fibrinolysis and randomized to routine early</td>
<td>All-cause mortality, Recurrent MI Immediate Cath vs Ischemia Driven Cath Mortality: OR: 0.55; 95% CI: 0.34-0.90; p=0.02</td>
<td>There was a variable definition of early cath for Study showed a benefit to a routine invasive strategy of cath following fibrinolysis compared with an ischemia driven approach in the “stent era”.</td>
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### 2013 STEMI Guideline Data Supplements

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<tr>
<th>Study</th>
<th>Type</th>
<th>Key Characteristics</th>
<th>Outcomes</th>
<th>Interpretation</th>
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<tbody>
<tr>
<td>CARESS-AMI, 2008 (32) 19280326</td>
<td>Randomized multicenter trial of immediate transfer for PCI following FT in high risk patient compared with standard care and rescue PCI.</td>
<td>Symptoms ≤12 h, and ≥1 high-risk features: Cumulative ST-elevation of &gt;15 mm, new onset LBBB, prior MI, Killip class ≥2, or LVEF ≤35%.</td>
<td>Early Cath vs Standard Care MACE: HR: 0.4; 95% CI: 0.21-0.76; log rank p=0.004 Minor or minimal bleeding was higher in the immediate cath group. There was a 47.8% higher major bleeding in immediate cath group (not statistically significant).</td>
<td>Study demonstrated a benefit to immediate transfer of high-risk pts with STEMI following fibrinolysis compared with rescue PCI or standard care. The primary endpoint was driven largely by recurrent ischemia.</td>
</tr>
<tr>
<td>TRANSFER AMI, 2009 (33) 19553646</td>
<td>Randomized multicenter trial of FT followed by immediate transfer for cath compared with fibrinolysis and standard care (rescue cath for cath 24 h to 2 wk).</td>
<td>Symptoms ≤12 h and ST-elevation ≥2 mm in anterior leads, or ST ≥1 mm in the inferior leads with: SBP &lt;100, Killip class 2 or 3, ST-depression of ≥2 mm in the anterior leads, or ST-elevation of ≥1 mm in the right-sided leads. Combined incidence of death, re-MI, recurrent ischemia, new or worsening CHF or shock at 30 d. Early Cath vs Delayed Cath MACE: 11.0% vs 17.2%; RR: 0.64; 0.47-0.87; p=0.004 Significantly more mild GUSTO bleeding in the immediate cath group.</td>
<td></td>
<td>Study demonstrated a benefit to immediate transfer of high-risk pts with STEMI following fibrinolysis compared with transfer for rescue PCI or early cath (24 h-2 wk).</td>
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<tr>
<td>NORDSTEMI, 2010 (34) 19747792</td>
<td>Multicenter randomized study of FT and immediate transfer for PCI compared with FT and standard care.</td>
<td>Age 18 to 75 y, symptoms &lt;6 h; ST-elevation of ≥2 mm ST in 2 precordial leads, or ≥1 in 2 inferior leads or new LBBB; expected time delay for PCI over 90 min. Death, Re-MI, CVA or new ischemia at 12 mo.</td>
<td>Early Cath vs Routine Care Primary Endpoint: 21% vs 27% HR: 0.72; 95% CI: 0.44-1.18; p=0.19 Death, CVA or re-MI: 6% vs 16% HR: 0.36; 95% CI: 0.16-0.81; p=0.01 No differences in bleeding complications.</td>
<td>Study failed to demonstrate a benefit of immediate cath following fibrinolytic therapy in achieving the primary endpoint of death, re-MI, CVA or ischemia at 12 mo. However, immediate cath resulted in a significant reduction in the 2nd endpoint when compared with standard care (rescue PCI ischemia guided PCI or routine cath done 2 to 4 wk) following fibrinolysis.</td>
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<td>Borgia et al., 2010 (35) 20601393</td>
<td>A meta-analysis of trials examining fibrinolysis with immediate transfer for cath with fibrinolysis alone and standard care.</td>
<td>Included all trials of STEMI pts treated with fibrin-specific agents and randomized to immediate PCI or standard care. Death, re-MI or combined endpoint of death, re-MI and re-ischemia and revascularization at 30 d or longer. Safety endpoint was major bleeding a Early Cath vs Delayed Cath or Ischemia Driven Cath 30 d Death: 3.3% vs 3.8%; OR: 0.87; 95% CI: 0.59-1.39; p=0.51 30 d Re-MI 2.6 vs 4.7%; OR: 0.55; 95% CI: 0.36-0.82; Different endpoint definitions which the investigators attempted to resolve by reevaluating some of the endpoints of the individual trials.</td>
<td>Meta-analysis demonstrated a benefit to a routine strategy of early cath following lytic therapy compared with standard care by reducing the combined endpoint of death and re-MI at 30 d, without a significant increase in adverse events including bleeding or stroke. A meta-regression analysis looking at baseline risk of the pts for each study demonstrated a</td>
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<td>30 d Death/Re-MI</td>
<td>5.6 vs 8.3%; OR: 0.65; 95% CI: 0.49-0.88; p=0.004</td>
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<td>30 d Recurrent ischemia</td>
<td>1.9 vs 7.1%; OR: 0.25; 95% CI: 0.13-0.49; p&lt;0.001</td>
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<td>6 to 12 Mo Death</td>
<td>4.8 vs 5.4%; OR: 0.88; 95% CI: 0.62-1.25; p=0.48</td>
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<td>6 to 12 Mo Re-MI</td>
<td>3.9 vs 6%; OR: 0.64; 95% CI: 0.40-0.98; p=0.01</td>
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<td>6 to 12 Mo Death/Re-MI</td>
<td>8.6 vs 11.2%; OR: 0.71; 95% CI: 0.52-0.97; p=0.03</td>
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No difference in Major bleeding.
No difference in stroke.

Time from FT to PCI varied from 84 min to 16.7 h.

Greater benefit to this approach among the higher risk group of pts.

AMI indicates acute myocardial infarction; AWMI, anterior wall myocardial infarction; cath, catheterization; CHF, congestive heart failure; CI, confidence interval; CPK, creatine phosphokinase; CVA, cerebrovascular accident; EP, electrophysiology; FT, fibrinolytic therapy; GPI, glycoprotein inhibitor; GUSTO, Global Utilization of Streptokinase and t-PA for Occluded Coronary Arteries; ICH, intracranial hemorrhage; LBBB, left bundle-branch block; LVEF, left ventricular ejection fraction; MACE, major adverse cardiac events; MI, myocardial infarction; PCI, percutaneous coronary intervention; POBA, plain old balloon angioplasty; pts, patients; RD, risk difference; RPA, reteplase; RWMI, regional wall motion index; SBP, systolic blood pressure; STEMI, ST-elevation myocardial infarction; TLR, transmyocardial laser revascularization; TRP, thrombosis risk panel; and UA, unstable angina.
References


