

**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**

Patrick T. O'Gara, Frederick G. Kushner, Deborah D. Ascheim, Donald E. Casey, Jr, Mina K. Chung, James A. de Lemos, Steven M. Ettinger, James C. Fang, Francis M. Fesmire, Barry A. Franklin, Christopher B. Granger, Harlan M. Krumholz, Jane A. Linderbaum, David A. Morrow, L. Kristin Newby, Joseph P. Ornato, Narith Ou, Martha J. Radford, Jacqueline E. Tamis-Holland, Jacqueline E. Tommaso, Cynthia M. Tracy, Y. Joseph Woo and David X. Zhao

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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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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.

Table 1. Applying Classification of Recommendation and Level of Evidence

		SIZE OF TREATMENT EFFECT											
		CLASS I <i>Benefit >>> Risk</i> Procedure/Treatment SHOULD be performed/administered	CLASS IIa <i>Benefit >> Risk</i> <i>Additional studies with focused objectives needed</i> IT IS REASONABLE to perform procedure/administer treatment	CLASS IIb <i>Benefit ≥ Risk</i> <i>Additional studies with broad objectives needed; additional registry data would be helpful</i> Procedure/Treatment MAY BE CONSIDERED	CLASS III <i>No Benefit or CLASS III Harm</i> <table border="1"> <tr> <th colspan="2">Procedure/Treatment</th> </tr> <tr> <th>Test</th> <th>Treatment</th> </tr> <tr> <td>COR III: No benefit</td> <td>Not Helpful No Proven Benefit</td> </tr> <tr> <td>COR III: Harm</td> <td>Excess Cost w/o Benefit or Harmful Harmful to Patients</td> </tr> </table>	Procedure/Treatment		Test	Treatment	COR III: No benefit	Not Helpful No Proven Benefit	COR III: Harm	Excess Cost w/o Benefit or Harmful Harmful to Patients
Procedure/Treatment													
Test	Treatment												
COR III: No benefit	Not Helpful No Proven Benefit												
COR III: Harm	Excess Cost w/o Benefit or Harmful Harmful to Patients												
ESTIMATE OF CERTAINTY (PRECISION) OF TREATMENT EFFECT	LEVEL A Multiple populations evaluated* Data derived from multiple randomized clinical trials or meta-analyses	<ul style="list-style-type: none"> Recommendation that procedure or treatment is useful/effective Sufficient evidence from multiple randomized trials or meta-analyses 	<ul style="list-style-type: none"> Recommendation in favor of treatment or procedure being useful/effective Some conflicting evidence from multiple randomized trials or meta-analyses 	<ul style="list-style-type: none"> Recommendation’s usefulness/efficacy less well established Greater conflicting evidence from multiple randomized trials or meta-analyses 	<ul style="list-style-type: none"> Recommendation that procedure or treatment is not useful/effective and may be harmful Sufficient evidence from multiple randomized trials or meta-analyses 								
	LEVEL B Limited populations evaluated* Data derived from a single randomized trial or nonrandomized studies	<ul style="list-style-type: none"> Recommendation that procedure or treatment is useful/effective Evidence from single randomized trial or nonrandomized studies 	<ul style="list-style-type: none"> Recommendation in favor of treatment or procedure being useful/effective Some conflicting evidence from single randomized trial or nonrandomized studies 	<ul style="list-style-type: none"> Recommendation’s usefulness/efficacy less well established Greater conflicting evidence from single randomized trial or nonrandomized studies 	<ul style="list-style-type: none"> Recommendation that procedure or treatment is not useful/effective and may be harmful Evidence from single randomized trial or nonrandomized studies 								
	LEVEL C Very limited populations evaluated* Only consensus opinion of experts, case studies, or standard of care	<ul style="list-style-type: none"> Recommendation that procedure or treatment is useful/effective Only expert opinion, case studies, or standard of care 	<ul style="list-style-type: none"> Recommendation in favor of treatment or procedure being useful/effective Only diverging expert opinion, case studies, or standard of care 	<ul style="list-style-type: none"> Recommendation’s usefulness/efficacy less well established Only diverging expert opinion, case studies, or standard of care 	<ul style="list-style-type: none"> Recommendation that procedure or treatment is not useful/effective and may be harmful Only expert opinion, case studies, or standard of care 								
Suggested phrases for writing recommendations		should is recommended is indicated is useful/effective/beneficial	is reasonable can be useful/effective/beneficial is probably recommended or indicated	may/might be considered may/might be reasonable usefulness/effectiveness is unknown/unclear/uncertain or not well established	COR III: No Benefit is not recommended is not indicated	COR III: Harm potentially harmful causes harm							
Comparative effectiveness phrases [†]		treatment/strategy A is recommended/indicated in preference to treatment B treatment A should be chosen over treatment B	treatment/strategy A is probably recommended/indicated in preference to treatment B it is reasonable to choose treatment A over treatment B		should not be performed/administered/other is not useful/beneficial/effective	associated with excess morbidity/mortality should not be performed/administered/other							

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.

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.¹ 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

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 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.ccardiosource.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*.^{2,3} 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 guideline⁴ 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, thrombolytic 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 2009.⁵⁻⁷ 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.⁸⁻¹¹ (Level of Evidence: B)
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.¹¹⁻¹⁵ (Level of Evidence: B)
3. Reperfusion therapy should be administered to all eligible patients with STEMI with symptom onset within the prior 12 hours.^{16,17} (Level of Evidence: A)
4. Primary PCI is the recommended method of reperfusion when it can be performed in a timely fashion by experienced operators.¹⁷⁻¹⁹ (Level of Evidence: A)
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.^{*11,14,15} (Level of Evidence: B)
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.^{*18-21} (Level of Evidence: B)
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.^{16,22,23} (Level of Evidence: B)
8. When fibrinolytic therapy is indicated or chosen as the primary reperfusion strategy, it should be administered within 30 minutes of hospital arrival.^{*24-28} (Level of Evidence: B)

Class IIa

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

*The proposed time windows are system goals. For any individual patient, every effort should be made to provide reperfusion therapy as rapidly as possible.

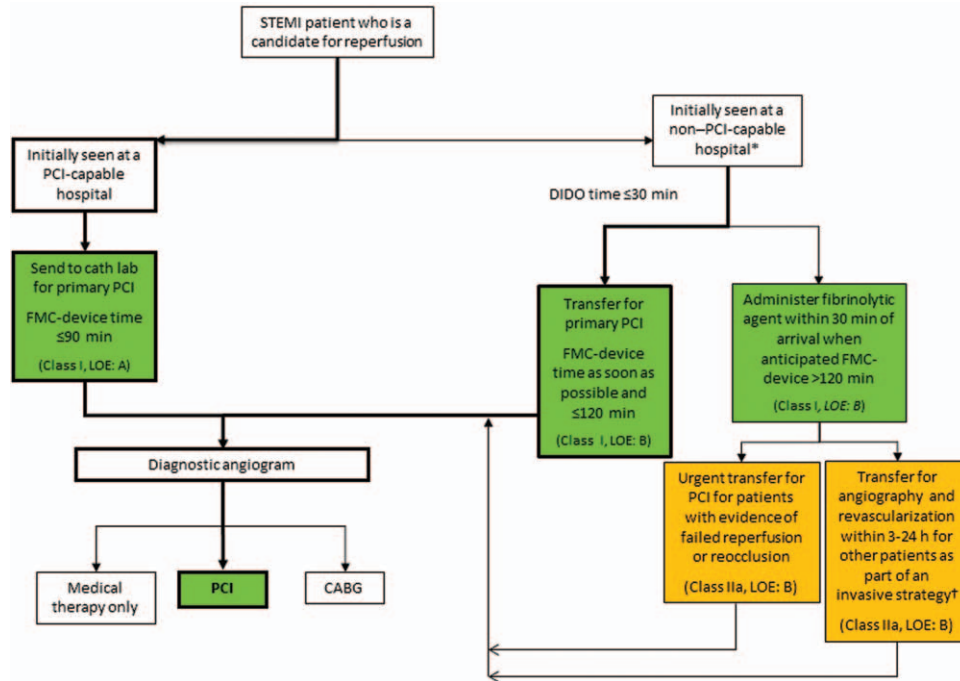


Figure 1. Reperfusion therapy for patients with STEMI. The bold arrows and boxes are the preferred strategies. Performance of PCI is dictated by an anatomically appropriate culprit stenosis. *Patients with cardiogenic shock or severe heart failure initially seen at a non-PCI-capable hospital should be transferred for cardiac catheterization and revascularization as soon as possible, irrespective of time delay from MI onset (Class I, LOE: B). †Angiography and revascularization should not be performed within the first 2 to 3 hours after administration of fibrinolytic therapy. CABG indicates coronary artery bypass graft; DIDO, door-in-door-out; FMC, first medical contact; LOE, Level of Evidence; MI, myocardial infarction; PCI, percutaneous coronary intervention; and STEMI, ST-elevation myocardial infarction.

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 the time delay from FMC.^{52,53} (Level of Evidence: 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.^{29,30} (Level of Evidence: B)

Table 2. Primary PCI in STEMI

	COR	LOE	References
Ischemic symptoms <12 h	I	A	17, 50, 51
Ischemic symptoms <12 h and contraindications to fibrinolytic therapy irrespective of time delay from FMC	I	B	52, 53
Cardiogenic shock or acute severe HF irrespective of time delay from MI onset	I	B	54-57
Evidence of ongoing ischemia 12 to 24 h after symptom onset	IIa	B	29, 30
PCI of a noninfarct artery at the time of primary PCI in patients without hemodynamic compromise	III: Harm	B	58-60

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.⁶¹⁻⁶⁴ (*Level of Evidence: B*)

3.3. Use of Stents in Patients With STEMI**Class I**

1. Placement of a stent (bare-metal stent or drug-eluting stent) is useful in primary PCI for patients with STEMI.^{65,66} (*Level of Evidence: A*)
2. Bare-metal stents[†] should be used in patients with high bleeding risk, inability to comply with 1 year of dual antiplatelet therapy (DAPT), or anticipated invasive or surgical procedures in the next year. (*Level of Evidence: C*)

Class III: Harm

1. Drug-eluting stents should not be used in primary PCI for patients with STEMI who are unable to tolerate or comply with a prolonged course of DAPT because of the increased risk of stent thrombosis with premature discontinuation of one or both agents.⁶⁷⁻⁷³ (*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 mg^{76,81,82} (*Level of Evidence: B*); or
 - b. Prasugrel 60 mg⁸³ (*Level of Evidence: B*); or
 - c. Ticagrelor 180 mg.⁸⁴ (*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 daily.^{83,85} (*Level of Evidence: B*); or
 - b. Prasugrel 10 mg daily⁸⁵ (*Level of Evidence: B*); or
 - c. Ticagrelor 90 mg twice a day.⁸⁴ (*Level of Evidence: B*)

[†]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.

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 abciximab⁸⁸⁻⁹⁰ (*Level of Evidence: A*), high-bolus-dose tirofiban^{91,92} (*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. 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*)
2. It may be reasonable to administer intracoronary abciximab to patients with STEMI undergoing primary PCI.^{64,102-108} (*Level of Evidence: B*)
3. Continuation of a P2Y₁₂ inhibitor beyond 1 year may be considered in patients undergoing drug-eluting stent placement. (*Level of Evidence: C*)

Class III: Harm

1. Prasugrel should not be administered to patients with a history of prior stroke or transient ischemic attack.⁸³ (*Level of Evidence: B*)

3.5. Anticoagulant Therapy to Support Primary PCI**Class I**

1. For patients with STEMI undergoing primary PCI, the following supportive anticoagulant regimens are recommended:
 - a. UFH, with additional boluses administered as needed to maintain therapeutic activated clotting time levels, taking into account whether a GP IIb/IIIa receptor antagonist has been administered (*Level of Evidence: C*); or
 - b. Bivalirudin with or without prior treatment with UFH.¹⁰⁹ (*Level of Evidence: B*)

Class IIa

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*)

Table 3. Adjunctive Antithrombotic Therapy to Support Reperfusion With Primary PCI

	COR	LOE	References
Antiplatelet therapy			
Aspirin			
• 162- to 325-mg load before procedure	I	B	74–76
• 81- to 325-mg daily maintenance dose (indefinite)*	I	A	77, 78, 80
• 81 mg daily is the preferred maintenance dose*	IIa	B	76, 77, 86, 87
P2Y₁₂ inhibitors			
Loading doses			
• Clopidogrel: 600 mg as early as possible or at time of PCI	I	B	76, 81, 82
• Prasugrel: 60 mg as early as possible or at time of PCI	I	B	83
• Ticagrelor: 180 mg as early as possible or at time of PCI	I	B	84
Maintenance doses and duration of therapy			
<i>DES placed: Continue therapy for 1 y with:</i>			
• Clopidogrel: 75 mg daily	I	B	83, 85
• Prasugrel: 10 mg daily	I	B	85
• Ticagrelor: 90 mg twice a day*	I	B	84
<i>BMS† placed: Continue therapy for 1 y with:</i>			
• Clopidogrel: 75 mg daily	I	B	83, 85
• Prasugrel: 10 mg daily	I	B	85
• Ticagrelor: 90 mg twice a day*	I	B	84
<i>DES placed:</i>			
• Clopidogrel, prasugrel, or ticagrelor* continued beyond 1 y	IIb	C	N/A
• Patients with STEMI with prior stroke or TIA: prasugrel	III: Harm	B	83
IV GP IIb/IIIa receptor antagonists in conjunction with UFH or bivalirudin in selected patients			
• Abciximab: 0.25-mg/kg IV bolus, then 0.125 mcg/kg/min (maximum 10 mcg/min)	IIa	A	88–90
• Tirofiban: (high-bolus dose): 25-mcg/kg IV bolus, then 0.15 mcg/kg/min	IIa	B	91, 92
• Eptifibatid: (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	IIa	B	93
• In patients with CrCl <30 mL/min, reduce infusion by 50%			
• In patients with CrCl <50 mL/min, reduce infusion by 50%			
• Avoid in patients on hemodialysis			
• Pre-catheterization laboratory administration of intravenous GP IIb/IIIa receptor antagonist	IIb	B	91, 94–101
• Intracoronary abciximab 0.25-mg/kg bolus	IIb	B	64, 102–108
Anticoagulant therapy			
• UFH:	I	C	N/A
• With GP IIb/IIIa receptor antagonist planned: 50- to 70-U/kg IV bolus to achieve therapeutic ACT‡	I	C	N/A
• With no GP IIb/IIIa receptor antagonist planned: 70- to 100-U/kg bolus to achieve therapeutic ACT§	I	B	109
• 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.	I	B	109
• Reduce infusion to 1 mg/kg/h with estimated CrCl <30 mL/min			
• Preferred over UFH with GP IIb/IIIa receptor antagonist in patients at high risk of bleeding	IIa	B	109
• Fondaparinux: Not recommended as sole anticoagulant for primary PCI	III: Harm	B	110

*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 P2Y₁₂ 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/IIIa receptor antagonist treatment is 200 to 250 s.

§The recommended ACT with no planned GP IIb/IIIa 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.

Table 4. Indications for Fibrinolytic Therapy When There Is a >120-Minute Delay From FMC to Primary PCI (Figure)

	COR	LOE	References
Ischemic symptoms <12 h	I	A	16, 111–116
Evidence of ongoing ischemia 12 to 24 h after symptom onset, and a large area of myocardium at risk or hemodynamic instability	IIa	C	N/A
ST depression except if true posterior (inferobasal) MI suspected or when associated with ST-elevation in lead Avr	III: Harm	B	16, 117–120

COR indicates Class of Recommendation; FMC, first medical contact; LOE, Level of Evidence; MI, myocardial infarction; N/A, not available; and PCI, percutaneous coronary intervention.

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.^{16,111–116} (Level of Evidence: 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. (Level of Evidence: 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.^{16,117–120} (Level of Evidence: 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 ≤75 years

of age, 75-mg dose for patients >75 years of age) should be administered to patients with STEMI who receive fibrinolytic therapy.^{113,121,122} (Level of Evidence: A)

2. Aspirin should be continued indefinitely^{113,121,122} (Level of Evidence: A) and clopidogrel (75 mg daily) should be continued for at least 14 days^{121,122} (Level of Evidence: A) and up to 1 year (Level of Evidence: 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.^{77,80,86,87} (Level of Evidence: 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.^{123,124} (Level of Evidence: 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 (Level of Evidence: 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^{124–127} (Level of Evidence: 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.¹¹⁰ (Level of Evidence: B)

4.3. Transfer to a PCI-Capable Hospital After Fibrinolytic Therapy

4.3.1. Transfer of Patients With STEMI to a PCI-Capable Hospital for Coronary Angiography After Fibrinolytic Therapy

See Table 6 for a summary of recommendations from this section; *Online Data Supplement 4* for additional data on early catheterization and rescue PCI for fibrinolytic failure in the stent era; and *Online Data Supplement 5* for additional data on early catheterization and PCI after fibrinolysis in the stent era.

Class I

1. Immediate transfer to a PCI-capable hospital for coronary angiography is recommended for suitable patients with STEMI who develop cardio-

Table 5. Adjunctive Antithrombotic Therapy to Support Reperfusion With Fibrinolytic Therapy

	COR	LOE	References
Antiplatelet therapy			
Aspirin			
• 162- to 325-mg loading dose	I	A	113, 121, 122
• 81- to 325-mg daily maintenance dose (indefinite)	I	A	113, 121, 122
• 81 mg daily is the preferred maintenance dose	IIa	B	77, 80, 86, 87
P2Y₁₂ receptor inhibitors			
• Clopidogrel:			
• Age ≤75 y: 300-mg loading dose	I	A	121, 122
• Followed by 75 mg daily for at least 14 d and up to 1 y in absence of bleeding	I	A (14 d) C (up to 1 y)	121, 122 N/A
• Age >75 y: no loading dose, give 75 mg	I	A	121, 122
• Followed by 75 mg daily for at least 14 d and up to 1 y in absence of bleeding	I	A (14 d) C (up to 1 y)	121, 122 N/A
Anticoagulant therapy			
• UFH:			
• Weight-based IV bolus and infusion adjusted to obtain aPTT of 1.5 to 2.0 times control for 48 h or until revascularization. IV bolus of 60 U/kg (maximum 4000 U) followed by an infusion of 12 U/kg/h (maximum 1000 U) initially, adjusted to maintain aPTT at 1.5 to 2.0 times control (approximately 50 to 70 s) for 48 h or until revascularization.	I	C	N/A
• Enoxaparin:			
• If age <75 y: 30-mg IV bolus, followed in 15 min by 1 mg/kg subcutaneously every 12 h (maximum 100 mg for the first 2 doses)	I	A	124-127
• If age ≥75 y: no bolus, 0.75 mg/kg subcutaneously every 12 h (maximum 75 mg for the first 2 doses)			
• Regardless of age, if CrCl <30 mL/min: 1 mg/kg subcutaneously every 24 h			
• Duration: For the index hospitalization, up to 8 d or until revascularization			
• Fondaparinux:			
• Initial dose 2.5 mg IV, then 2.5 mg subcutaneously daily starting the following day, for the index hospitalization up to 8 d or until revascularization	I	B	110
• Contraindicated if CrCl <30 mL/min			

aPTT indicates activated partial thromboplastin time; COR, Class of Recommendation; CrCl, creatinine clearance; IV, intravenous; LOE, Level of Evidence; N/A, not available; and UFH, unfractionated heparin.

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 stable[§] 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 presentation^{57,128,139,140} (Level of Evidence: B);
 - b. Intermediate- or high-risk findings on pre-discharge noninvasive ischemia testing^{141,142} (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

	COR	LOE	References
Immediate transfer for cardiogenic shock or severe acute HF irrespective of time delay from MI onset	I	B	128
Urgent transfer for failed reperfusion or reocclusion	IIa	B	129–132
As part of an invasive strategy in stable* patients with PCI between 3 and 24 h after successful fibrinolysis	IIa	B	133–138

*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.^{129–132} (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

	COR	LOE	References
Cardiogenic shock or acute severe HF that develops after initial presentation	I	B	57, 128, 139, 140
Intermediate- or high-risk findings on pre-discharge noninvasive ischemia testing	I	B	141, 142
Spontaneous or easily provoked myocardial ischemia	I	C	N/A
Failed reperfusion or reocclusion after fibrinolytic therapy	IIa	B	129–132
Stable* patients after successful fibrinolysis, before discharge and ideally between 3 and 24 h	IIa	B	133–138, 143

*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

	COR	LOE	References
Cardiogenic shock or acute severe HF	I	B	128
Intermediate- or high-risk findings on pre-discharge noninvasive ischemia testing	I	C	141, 142
Spontaneous or easily provoked myocardial ischemia	I	C	N/A
Patients with evidence of failed reperfusion or reocclusion after fibrinolytic therapy (as soon as possible)	IIa	B	130,130a–130c
Stable* patients after successful fibrinolysis, ideally between 3 and 24 h	IIa	B	133–138
Stable* patients >24 h after successful fibrinolysis	IIb	B	55, 141–148
Delayed PCI of a totally occluded infarct artery >24 h after STEMI in stable patients	III: No Benefit	B	55, 146

*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; PCI, percutaneous coronary intervention; and STEMI, ST-elevation myocardial infarction.

Class I

- 1. PCI of an anatomically significant stenosis in the infarct artery should be performed in patients with suitable anatomy and any of the following:**
 - a. Cardiogenic shock or acute severe HF¹²⁸ (Level of Evidence: B);**
 - b. Intermediate- or high-risk findings on pre-discharge noninvasive ischemia testing^{141,142} (Level of Evidence: C); or**
 - c. Myocardial ischemia that is spontaneous or provoked by minimal exertion during hospitalization. (Level of Evidence: C)**

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 hospital^{130,130a–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**

§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.

may be considered as part of an invasive strategy in stable§ patients.^{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.^{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.^{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.^{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.^{83,85,121,122} (*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.^{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.^{83,85} (*Level of Evidence: B*)

3. Prasugrel, in a 10-mg daily maintenance dose, is reasonable after PCI.^{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.⁸³ (*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.^{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.¹¹⁰ (*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.^{150–152} (*Level of Evidence: B*)
2. CABG is recommended in patients with STEMI at time of operative repair of mechanical defects.^{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

Table 9. Adjunctive Antithrombotic Therapy to Support PCI After Fibrinolytic Therapy

	COR	LOE	References
Antiplatelet therapy			
Aspirin			
• 162- to 325-mg loading dose given with fibrinolytic agent (before PCI). See Section 4.2.1 and Table 5.	I	A	113, 121, 122
• 81- to 325-mg daily maintenance dose after PCI (indefinite)	I	A	76, 77, 80, 82, 121, 122
• 81 mg daily is the preferred daily maintenance dose	IIa	B	76, 82, 86, 87
P2Y₁₂ receptor inhibitors			
Loading doses			
<i>For patients who received a loading dose of clopidogrel with fibrinolytic therapy:</i>			
• Continue clopidogrel 75 mg daily without an additional loading dose	I	C	83, 85, 121, 122
<i>For patients who have not received a loading dose of clopidogrel:</i>			
• If PCI is performed ≤24 h after fibrinolytic therapy: clopidogrel 300-mg loading dose before or at the time of PCI	I	C	N/A
• If PCI is performed >24 h after fibrinolytic therapy: clopidogrel 600-mg loading dose before or at the time of PCI	I	C	N/A
• If PCI is performed >24 h after treatment with a fibrin-specific agent or >48 h after a non-fibrin-specific agent: prasugrel 60 mg at the time of PCI	IIa	B	83, 85
<i>For patients with prior stroke/TIA: prasugrel</i>	III: Harm	B	83
Maintenance doses and duration of therapy			
<i>DES placed: Continue therapy for at least 1 y with:</i>			
• Clopidogrel: 75 mg daily	I	C	83, 85, 121, 122
• Prasugrel: 10 mg daily	IIa	B	83, 85
<i>BMS* placed: Continue therapy for at least 30 d and up to 1 y with:</i>			
• Clopidogrel: 75 mg daily	I	C	121, 122
• Prasugrel: 10 mg daily	IIa	B	83, 85
Anticoagulant therapy			
• Continue UFH through PCI, administering additional IV boluses as needed to maintain therapeutic ACT depending on use of GP IIb/IIIa receptor antagonist†	I	C	N/A
• Continue enoxaparin through PCI: <ul style="list-style-type: none"> • No additional drug if last dose was within previous 8 h • 0.3-mg/kg IV bolus if last dose was 8 to 12 h earlier 	I	B	127, 149
• Fondaparinux: <ul style="list-style-type: none"> • As sole anticoagulant for PCI 	III: Harm	C	110

*Balloon angioplasty without stent placement may be used in selected patients. It might be reasonable to provide P2Y₁₂ inhibitor therapy to patients with STEMI undergoing balloon angioplasty after fibrinolysis alone according to the recommendations listed for BMS. (Level of Evidence: C)

†The recommended ACT with no planned GP IIb/IIIa receptor antagonist treatment is 250–300 s (HemoTec device) or 300–350 s (Hemochron device). ACT indicates activated clotting time; BMS, bare-metal stent; COR, Class of Recommendation; DES, drug-eluting stent; GP, glycoprotein; IV, intravenous; LOE, Level of Evidence; N/A, not available; PCI, percutaneous coronary intervention; TIA, transient ischemic attack; and UFH, unfractionated heparin.

have cardiogenic shock and are not candidates for PCI or fibrinolytic therapy. (Level of Evidence: C)

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

6.2. Timing of Urgent CABG in Patients With STEMI in Relation to Use of Antiplatelet 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 antagonists (eptifibatide, tirofiban) should be discontinued at least 2 to 4 hours before urgent CABG.^{164,165} (Level of Evidence: B)

Class IIb

1. Urgent off-pump CABG within 24 hours of clopidogrel or ticagrelor administration might be considered, especially if the benefits of prompt revascularization outweigh the risks of bleeding.^{160,166–168} (Level of Evidence: B)
2. Urgent CABG within 5 days of clopidogrel or ticagrelor administration or within 7 days of prasugrel 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,^l 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).^{169–171} (*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*)

^lRisk factors for cardiogenic shock (the greater the number of risk factors present, the higher the risk of developing cardiogenic shock) are age >70 years, systolic blood pressure <120 mm Hg, sinus tachycardia >110 bpm or heart rate <60 bpm, and increased time since onset of symptoms of STEMI.

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 bradyarrhythmias 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.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 antiinflammatory drugs are potentially harmful for treatment of pericarditis after STEMI.^{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# greater than or equal to 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 P2Y₁₂ 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*)

¶These recommendations apply to patients who receive intracoronary stents during PCI for STEMI. Among individuals with STEMI who do not receive an intracoronary stent, the duration of DAPT beyond 14 days has not been studied adequately for patients who undergo balloon angioplasty alone, are treated with fibrinolysis alone, or do not receive reperfusion therapy. In this subset of patients with STEMI who do not receive an intracoronary stent, the threshold for initiation of oral anticoagulation for secondary prevention, either alone or in combination with aspirin, may be lower, especially if a shorter duration (ie, 14 days) of DAPT is planned.²⁰⁴

#CHADS2 (Congestive heart failure, Hypertension, Age \geq 75 years, Diabetes mellitus, previous Stroke/transient ischemic attack [doubled risk weight]) score.

**Individual circumstances will vary and depend on the indications for triple therapy and the type of stent placed during PCI. After this initial treatment period, consider therapy with a vitamin K antagonist plus a single antiplatelet agent. For patients treated with fibrinolysis, consider triple therapy for 14 days, followed by a vitamin K antagonist plus a single antiplatelet agent.²⁰⁵⁻²⁰⁸

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.²⁰⁹⁻²¹¹ (*Level of Evidence: B*)

Class IIb

1. Noninvasive testing for ischemia might be considered before discharge to evaluate the functional significance of a noninfarct artery stenosis previously identified at angiography. (*Level of Evidence: C*)
2. 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.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.²¹⁶⁻²²⁰ (*Level of Evidence: B*)
2. Exercise-based cardiac rehabilitation/secondary prevention programs are recommended for patients with STEMI.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.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

Committee Member	Employment	Consultant	Speaker’s Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness	Voting Recusals by Section*
Patrick T. O’Gara, Chair	Harvard Medical School—Professor of Medicine	None	None	None	None	None	None	None
Frederick G. Kushner, Vice Chair	Tulane University School of Medicine—Clinical Professor of Medicine; Heart Clinic of Louisiana—Medical Director	None	None	None	None	• Novartis†	None	8.1 8.2
Deborah D. Ascheim	Mount Sinai School of Medicine—Associate Professor; InCHOIR—Clinical Director of Research	None	None	None	None	None	None	None
Donald E. Casey, Jr	Atlantic Health—Chief Medical Officer and Vice President of Quality	None	None	None	None	None	None	None
Mina K. Chung	Cleveland Clinic Foundation—Associate Professor of Medicine	• Biotronik† • Boston Scientific† • Nexcura † • PGx† • Sanofi-aventis† • St. Jude Medical†	None	None	• Biotronik† • Boston Scientific† • GlaxoSmithKline† • Medtronic† • Siemens Medical Solutions† • St. Jude Medical† • ZOLL†	• Medtronic† • Boston Scientific† • St. Jude Medical†	None	4.4.1 5.1.4 7.2 9.5.2
James A. de Lemos	UT Southwestern Medical School—Professor of Medicine	• Johnson & Johnson • Tethys • AstraZeneca • Daiichi-Sankyo	• BMS/ Sanofi-aventis	None	• Bristol-Myers Squibb (DSMB) • Roche • Merck/Schering-Plough • Daiichi-Sankyo	None	None	4.4.1 4.4.2 5.1.4.1 5.1.4.2 6.4.1 6.4.2 7.2 9.6
Steven M. Ettinger	Penn State Heart & Vascular Institute—Professor of Medicine and Radiology	None	None	None	• Medtronic§	None	None	4.3.1
James C. Fang	University Hospitals Case Medical Center—Director, Heart Transplantation	• Accordia • Novartis • Thoratec	None	None	None	• Medtronic	None	9.5.4.1
Francis M. Fesmire	Heart Stroke Center—Director	• Abbott	None	None	None	None	• Plaintiff, Missed ACS, 2010	8.3
Barry A. Franklin	William Beaumont Hospital—Director, Cardiac Rehabilitation and Exercise Laboratories	None	None	None	None	None	None	None
Christopher B. Granger	Duke Clinical Research Institute—Director, Cardiac Care Unit; Assistant Professor of Medicine	• AstraZeneca • Boehringer Ingelheim‡ • Bristol-Myers Squibb • GlaxoSmithKline • Hoffman La Roche • Novartis • Sanofi-aventis‡ • The Medicines Company	None	None	• Astellas • AstraZeneca • Boehringer Ingelheim‡ • Bristol-Myers Squibb • Eli Lilly • GlaxoSmithKline • Medtronic • Merck • Sanofi-aventis‡ • The Medicines Company	None	None	4.4.1 6.4.2 9.7.1

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Appendix 1. Continued

Committee Member	Employment	Consultant	Speaker's Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness	Voting Recusals by Section*
Harlan M. Krumholz	Yale University School of Medicine—Professor of Medicine	• United HealthCare (Science Advisory Group)	None	None	None	None	None	None
Jane A. Linderbaum	Mayo Clinic—Assistant Professor of Medicine	None	None	None	None	None	None	None
David A. Morrow	Harvard Medical School—Associate Professor of Medicine	<ul style="list-style-type: none"> • Beckman-Coulter • Boehringer Ingelheim • Daiichi-Sankyo • Eli Lilly • Genentech • Merck • Novartis • OrthoClinical Diagnostics/ Johnson & Johnson • Roche Diagnostics • Sanofi-aventis • Schering-Plough Research Institute • Siemens Medical Solutions 	None	None	<ul style="list-style-type: none"> • AstraZeneca‡ • Beckman-Coulter‡ • Daiichi-Sankyo‡ • Eli Lilly‡ • GlaxoSmithKline‡ • Merck‡ • Nanosphere‡ • Novartis‡ • Roche Diagnostics‡ • Sanofi-aventis‡ • Schering-Plough Research Institute‡ • Siemens Medical Solutions‡ • Singulex‡ 	• AstraZeneca‡	None	3.2 4.4.1 4.4.2 5.1 6.4.1 6.4.2 7.2 8.2 8.3 9.6
L. Kristin Newby	Duke University Medical Center, Division of Cardiology—Professor of Medicine	<ul style="list-style-type: none"> • Amgen‡ • AstraZeneca • BioVascular • Johnson & Johnson • Novartis 	None	None	<ul style="list-style-type: none"> • BG Medicine • Bristol-Myers Squibb • diaDexus‡ • Eli Lilly • GlaxoSmithKline‡ • Johnson & Johnson • Merck‡ • Regado • Schering-Plough‡ 	None	None	4.4.1 7.2
Joseph P. Ornato	Department of Emergency Medicine Virginia Commonwealth University—Professor and Chairman	<ul style="list-style-type: none"> • European Resuscitation Council‡ • ZOLL Circulation 	None	None	<ul style="list-style-type: none"> • NIH/NINDS Neurological Emergency Treatment Trials Consortium—PI‡ 	None	None	None
Narith Ou	Mayo Clinic—Pharmacotherapy Coordinator, Cardiology	None	None	None	None	None	None	None
Martha J. Radford	NYU Langone Medical Center—Chief Quality Officer; NYU School of Medicine—Professor of Medicine (Cardiology)	None	None	None	None	None	None	None
Jacqueline E. Tamis-Holland	St Luke's-Roosevelt Hospital Center—Director, Interventional Cardiology Fellowship Program; Columbia University, College of Physicians and Surgeons—Assistant Professor of Clinical Medicine	None	None	None	None	None	None	None

(Continued)

Appendix 1. Continued

Committee Member	Employment	Consultant	Speaker’s Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness	Voting Recusals by Section*
Carl L. Tommaso	Skokie Hospital—Director of Catheterization Laboratory; North Shore University Health Systems	None	None	None	None	None	None	None
Cynthia M. Tracy	George Washington University Medical Center—Associate Director, Division of Cardiology	None	None	None	None	None	None	None
Y. Joseph Woo	Hospital of the University of Pennsylvania—Associate Professor of Surgery	None	None	None	None	None	None	None
David X. Zhao	Vanderbilt University Medical Center—Director, Cardiac Catheterization and Interventional Cardiology	None	None	None	<ul style="list-style-type: none"> • Abbot Vascular • Accumetrics • AGA Medical • Osiris • Volcano 	None	None	4.3.1

This table represents the relationships of committee members with industry and other entities that were determined to be relevant to this document. These relationships were reviewed and updated in conjunction with all meetings and/or conference calls of the writing committee during the document development process. The table does not necessarily reflect relationships with industry at the time of publication. A person is deemed to have a significant interest in a business if the interest represents ownership of ≥5% of the voting stock or share of the business entity, or ownership of ≥\$10 000 of the fair market value of the business entity; or if funds received by the person from the business entity exceed 5% of the person’s gross income for the previous year. Relationships that exist with no financial benefit are also included for the purpose of transparency. Relationships in this table are modest unless otherwise noted.

According to the ACCF/AHA, a person has a *relevant* relationship IF: a) The *relationship or interest* relates to the same or similar subject matter, intellectual property or asset, topic, or issue addressed in the *document*; or b) The *company/entity* (with whom the relationship exists) makes a drug, drug class, or device addressed in the *document*, or makes a competing drug or device addressed in the *document*; or c) The *person or a member of the person’s household* has a reasonable potential for financial, professional, or other personal gain or loss as a result of the issues/content addressed in the *document*.

*Writing committee members are required to recuse themselves from voting on sections to which their specific relationships with industry and other entities could apply. Section numbers apply to the full-text guideline.

†No financial benefit.

‡Significant relationship.

§Dr. Ettinger’s relationship with Medtronic was added just before balloting of the recommendations, so it was not relevant during the writing stage; however, the addition of this relationship makes the writing committee out of compliance with the minimum 50% no relevant RWI requirement.

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

Reviewer	Representation	Consultant	Speaker's Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness
Elliott M. Antman	Official Reviewer—ACCF Board of Trustees	None	None	None	<ul style="list-style-type: none"> • Accumetrics • AstraZeneca • Beckman Coulter • Bristol-Myers Squibb Pharmaceutical Research Institute • Daiichi-Sankyo* • Eli Lilly* • GlaxoSmithKline • Merck • Millennium Pharmaceuticals • Novartis Pharmaceuticals • Ortho-Clinical Diagnostics • Sanofi-Synthelabo Recherche • Schering-Plough Research Institute 	None	None
Gary J. Balady	Official Reviewer—AHA	None	None	None	None	None	None
Christopher P. Cannon	Official Reviewer—AHA	• Novartis†	None	None	<ul style="list-style-type: none"> • Accumetrics* • AstraZeneca* • Bristol-Myers Squibb† • GlaxoSmithKline • Merck* 	<ul style="list-style-type: none"> • GlaxoSmithKline • Merck (DSMB) 	None
Judith S. Hochman	Official Reviewer—ACCF/AHA Task Force on Practice Guidelines	<ul style="list-style-type: none"> • BMS/Sanofi • Eli Lilly • GlaxoSmithKline 	None	None	None	<ul style="list-style-type: none"> • Johnson & Johnson Pharmaceutical Research & Development (DSMB) • Merck/Schering Plough (DSMB) 	None
Austin H. Kutscher	Official Reviewer—ACCF Board of Governors	None	None	None	None	None	None
Charles J. Davidson	Organizational Reviewer—SCAI	<ul style="list-style-type: none"> • Abbott* • Abbott Vascular 	None	None	<ul style="list-style-type: none"> • Edwards Lifesciences* 	None	None
Deborah B. Diercks	Organizational Reviewer—ACEP	<ul style="list-style-type: none"> • Abbott Cardiovascular • Daiichi-Sankyo 	None	None	<ul style="list-style-type: none"> • Beckman Coulter† • Nanosphere† 	None	None
Jonathan M. Tobis	Organizational Reviewer—SCAI	None	<ul style="list-style-type: none"> • AGA Medical • Boston Scientific 	None	<ul style="list-style-type: none"> • AGA Medical* 	None	None
Jeffrey L. Anderson	Content Reviewer—ACCF/AHA Task Force on Practice Guidelines	None	None	None	<ul style="list-style-type: none"> • Toshiba† 	<ul style="list-style-type: none"> • AstraZeneca (DSMB) 	Defendant, Postoperative Ablation Case, 2010
James C. Blankenship	Content Reviewer	None	None	None	<ul style="list-style-type: none"> • AstraZeneca† • Boston Scientific† • Novartis† • Schering-Plough† 	None	None
Jeffrey J. Cavendish	Content Reviewer—ACCF Prevention of Cardiovascular Disease Committee	None	None	None	None	None	None
Harold L. Dauerman	Content Reviewer	None	None	None	None	None	None
John S. Douglas, Jr.	Content Reviewer	None	None	None	<ul style="list-style-type: none"> • Abbott† • Medtronic† • The Medicines Company† 	None	None
Stephen G. Ellis	Content Reviewer	<ul style="list-style-type: none"> • Abbott Vascular • Boston Scientific† 	None	None	None	None	None
Joseph Fredi	Content Reviewer—ACCF Surgeons' Scientific Council	<ul style="list-style-type: none"> • AGA Medical† 	None	None	None	None	None

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Appendix 2. Continued

Reviewer	Representation	Consultant	Speaker's Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness
Anthony Gershlick	Content Reviewer	<ul style="list-style-type: none"> • Abbott • AstraZeneca • Boehringer Ingelheim • Boston Scientific • Cordis • Eli Lilly • Medtronic 	None	None	• Boehringer Ingelheim	None	None
Howard C. Herrmann	Content Reviewer	<ul style="list-style-type: none"> • AstraZeneca • Merck Sharpe and Dohme 	None	None	<ul style="list-style-type: none"> • Accumetrics • Boston Scientific* • Edwards Lifesciences* • eValve • Medtronic* • St. Jude Medical • The Medicines Company* 	None	None
James Bernard Hermiller	Content Reviewer—ACCF Interventional Scientific Council	<ul style="list-style-type: none"> • Abbott • Boston Scientific • St. Jude Medical 	• Eli Lilly	None	None	None	None
Fred M. Kosumoto	Content Reviewer	None	None	None	None	None	None
Glenn Levine	Content Reviewer	None	None	None	None	None	None
Roxana Mehran	Content Reviewer	<ul style="list-style-type: none"> • Abbott Vascular • AstraZeneca • Ortho-McNeill 	None	None	<ul style="list-style-type: none"> • BMS/Sanofi-aventis* • The Medicines Company* 	None	None
M. Eugene Sherman	Content Reviewer—ACCF Board of Governors	None	Eli Lilly*	None	None	None	None
Daniel I. Simon	Content Reviewer	<ul style="list-style-type: none"> • Cordis/Johnson & Johnson • Daiichi-Sankyo • Eli Lilly • Medtronic • Sanofi-aventis • The Medicines Company 	None	None	None	None	Defendant, DES Intellectual Property Case, 2010
Richard W. Smalling	Content Reviewer—ACCF Interventional Scientific Council	• AGA Medical	None	None	<ul style="list-style-type: none"> • AGA Medical* • Cordis* • eValve* 	<ul style="list-style-type: none"> • AGA Medical • Cordis • eValve 	None
William G. Stevenson	Content Reviewer—ACCF/AHA Task Force on Practice Guidelines	None	None	None	None	None	None
William A. Tansey III	Content Reviewer	None	None	None	None	None	None
David D. Waters	Content Reviewer	<ul style="list-style-type: none"> • Bristol-Myers Squibb • Pfizer 	None	None	None	<ul style="list-style-type: none"> • Merck/Schering-Plough • Sanofi-aventis (DSMB) 	None
Christopher J. White	Content Reviewer	None	None	None	<ul style="list-style-type: none"> • Boston Scientific† • St. Jude Medical 	None	None
Clyde W. Yancy	Content Reviewer—ACCF/AHA Task Force on Practice Guidelines	None	None	None	None	None	None
Yerem Yeghiazarians	Content Reviewer	None	None	None	None	None	None

This table represents the relationships of reviewers with industry and other entities that were disclosed at the time of peer review and determined to be relevant. It does not necessarily reflect relationships with industry at the time of publication. A person is deemed to have a significant interest in a business if the interest represents ownership of $\geq 5\%$ of the voting stock or share of the business entity, or ownership of $\geq \$10,000$ of the fair market value of the business entity; or if funds received by the person from the business entity exceed 5% of the person's gross income for the previous year. A relationship is considered to be modest if it is less than significant under the preceding definition. Relationships that exist with no financial benefit are also included for the purpose of transparency. Relationships in this table are modest unless otherwise noted. Names are listed in alphabetical order within each category of review.

According to the ACCF/AHA, a person has a *relevant* relationship IF: a) The *relationship or interest* relates to the same or similar subject matter, intellectual property or asset, topic, or issue addressed in the *document*; or b) The *company/entity* (with whom the relationship exists) makes a drug, drug class, or device addressed in the *document*, or makes a competing drug or device addressed in the *document*; or c) The *person or a member of the person's household* has a reasonable potential for financial, professional, or other personal gain or loss as a result of the issues/content addressed in the *document*.

*Significant relationship.

†No financial benefit.

ACCF indicates American College of Cardiology Foundation; ACEP, American College of Emergency Physicians; AHA, American Heart Association; DES, drug-eluting stent; DSMB, data safety monitoring board; and SCAI, Society for Cardiovascular Angiography and Interventions.

AUTHOR RELATIONSHIPS WITH INDUSTRY AND OTHER ENTITIES (Comprehensive)—ACCF/AHA 2013 GUIDELINE FOR THE MANAGEMENT OF ST-ELEVATION MYOCARDIAL INFARCTION (October, 2012)

Committee Member	Employment	Consultant	Speaker's Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational or Other Financial Benefit	Expert Witness
Patrick T. O'Gara, <i>Chair</i>	Harvard Medical School— Professor of Medicine	None	None	None	<ul style="list-style-type: none"> • Lantheus Medical Imaging (DSMB) • NIH (DSMB)* 	<ul style="list-style-type: none"> • American Heart Association 	None
Frederick G. Kushner, <i>Vice Chair</i>	Tulane University School of Medicine— Clinical Professor of Medicine; Heart Clinic of Louisiana— Medical Director	None	None	None	None	<ul style="list-style-type: none"> • Novartis† 	None
Deborah D. Ascheim	Mount Sinai School of Medicine— Associate Professor; InCHOIR— Clinical Director of Research	None	None	None	None	None	None
Donald E. Casey, Jr.	Atlantic Health— Chief Medical Officer and Vice President of Quality	None	None	None	None	None	None
Mina K. Chung	Cleveland Clinic Foundation — Associate Professor of Medicine	<ul style="list-style-type: none"> • Biotronik† • Boston Scientific† • Nexcura† • PGx† • Sanofi-aventis† • St. Jude 	None	None	<ul style="list-style-type: none"> • Biosense Webster† • Biotronik† • Boston Scientific† • CardioDx† • CardioInsight† • GlaxoSmithKline† • Medtronic† 	<ul style="list-style-type: none"> • Boston Scientific† • Medtronic † • St. Jude Medical† 	None

		Medical†				<ul style="list-style-type: none"> • Siemens Medical Solutions† • St. Jude Medical† • Zin Medical† • ZOLL† 	
James A. de Lemos	UT Southwestern Medical School—Professor of Medicine	<ul style="list-style-type: none"> • Johnson & Johnson • Tethys 	<ul style="list-style-type: none"> • BMS/Sanofi -aventis† 	None	<ul style="list-style-type: none"> • Bristol-Myers Squibb (DSMB) • Roche† 	<ul style="list-style-type: none"> • Astra-Zeneca† • Daiichi-Sankyo 	None
Steven M. Ettinger	Penn State Heart and Vascular Institute—Professor of Medicine and Radiology	None	None	None	None	None	None
James C. Fang	University Hospitals Case Medical Center—Director, Heart Transplantation	<ul style="list-style-type: none"> • Thoratec 	None	None	<ul style="list-style-type: none"> • Accordia • Amgen • NIH • Novartis • Paracor 	<ul style="list-style-type: none"> • Medtronic 	None
Francis B. Fesmire	Heart Stroke Center—Director	<ul style="list-style-type: none"> • Abbott 	None	None	None	<ul style="list-style-type: none"> • Society of Chest Pain Centers (Board of Directors) 	<ul style="list-style-type: none"> • Plaintiff, Missed ACS, 2010
Barry A. Franklin	William Beaumont Hospital—Director, Cardiac Rehabilitation and Exercise Laboratories	None	None	None	None	None	None
Christopher B. Granger	Duke Clinical Research Institute—Director, Cardiac Care Unit; Assistant Professor of	<ul style="list-style-type: none"> • AstraZeneca • Boehringer Ingelheim* • Bristol-Myers Squibb • GlaxoSmithKline • Hoffman La 	None	None	<ul style="list-style-type: none"> • Astellas • AstraZeneca • Boehringer Ingelheim* • Bristol-Myers Squibb • Eli Lilly 	None	None

	Medicine	<ul style="list-style-type: none"> • Roche • Novartis • Otsuka • Sanofi-aventis* • The Medicines Company 			<ul style="list-style-type: none"> • GlaxoSmithKline • Medtronic • Merck • Sanofi-aventis* • The Medicines Company 		
Harlan M. Krumholz	Yale University School of Medicine— Professor of Epidemiology and Public Health	<ul style="list-style-type: none"> • Centegen/Life Tech • United HealthCare (Science Advisory Group) 	None	None	<ul style="list-style-type: none"> • American College of Cardiology 	<ul style="list-style-type: none"> • American Heart Association • Massachusetts Medical Society • UnitedHealth • VHA 	None
Jane A. Linderbaum	Mayo Clinic— Assistant Professor of Medicine	None	None	None	None	None	None
David A. Morrow	Harvard Medical School— Associate Professor of Medicine	<ul style="list-style-type: none"> • Beckman-Coulter • Boehringer Ingelheim • CardioKinetix • CV Therapeutics/ Gilead Pharmaceuticals • Critical Diagnostics • Daiichi-Sankyo • Genentech • Ikaria • Menarini • Novartis • OrthoClinical Diagnostics • Roche Diagnostics • Sanofi-aventis • Schering-Plough Research Institute • Siemens Medical 	None	None	<ul style="list-style-type: none"> • AstraZeneca† • Beckman-Coulter† • CV Therapeutics† • Daiichi-Sankyo† • Eli Lilly† • GlaxoSmithKline† • Merck • Novartis† • Roche Diagnostics† • Sanofi-aventis† • Schering-Plough Research Institute† • Siemens Medical Solutions† • Singulex† 	<ul style="list-style-type: none"> • AstraZeneca† 	None

	Director, Interventional Cardiology Fellowship Program; Columbia University, College of Physicians and Surgeons—Assistant Professor of Clinical Medicine						
Carl L. Tommaso	Skokie Hospital—Director of Catheterization Laboratory; NorthShore University HealthSystems—Partner	None	None	None	None	None	None
Cynthia M. Tracy	George Washington University Medical Center—Associate Director, Division of Cardiology	None	None	None	None	None	None
Y. Joseph Woo	Hospital of the University of Pennsylvania—Associate Professor of Surgery	None	None	None	None	None	None
David X. Zhao	Vanderbilt University Medical Center—Director, Cardiac	None	None	None	<ul style="list-style-type: none"> • Abbot Vascular • Accumetrics • AGA Medical • Osiris • Volcano 	None	None

	Catheterization and Interventional Cardiology						
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*Indicates significant ($> \$10,000$) 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.

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Data Supplement 1. ECG Criteria for Diagnosis of STEMI in the Setting of LBBB

Odds Ratios and Scores for Independent Electrocardiographic Criteria		
Criterion	Odds Ratio (95% CI)	Score
ST-elevation ≥ 1 mm and concordant with QRS complex	25.2 (11.6 - 54.7)	5
ST-segment depression ≥ 1 mm in lead V ₁ , V ₂ , or V ₃	6.0 (1.9 - 19.3)	3
ST-elevation ≥ 5 mm and discordant with QRS complex	4.3 (1.8 - 10.6)	2

CI indicates confidence interval.

Reprinted from Sgarbossa et al. (2). [8559200](#)

In the NRM-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](#).

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Data Supplement 2. PCI for Cardiac Arrest Evidence												
Study Name	Aim of study	Study Type	Study Size	Patient Population/ Inclusion & Exclusion Criteria		Endpoint		Statistical Analysis Reported	P-Values & 95% CI	OR: HR: RR:	Study Summary	Study Limitations
				Inclusion Criteria	Exclusion Criteria	Primary Endpoint	Secondary Endpoint					
Primary coronary angioplasty for AMI complicated by OOH-CA. Kahn et al., 1995 (4) 7747692	First report of PPCI in OOH-CA pts	Case series	11	Clinical judgment of cardiologist. No prespecified criteria used.	Clinical judgment of cardiologist. No prespecified criteria used.	Survival to hospital discharge	Neurological outcome	None			11 pt OOH-CA pts brought to PPCI. 6/11 survived, 4/11 with full neurologic recovery.	Single institution, Selection bias
Immediate coronary angiography in survivors of OOH-CA. Spaulding et al., 1997 (5) 9171064	Determine impact of PPCI on OOH-CA survivors	Consecutive case series	84	OOH-CA, 30-75 y, <6 h onset of symptoms in pts previously leading a normal life, no obvious noncardiac etiology.	None	Survival to hospital discharge	Prevalence of CAD on angiography	Multivariate logistic regression showed successful PPCI was an independent predictor of survival.	p=0.04; 95% CI: 1.1- 24.5	OR: 5.2	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.	Selection bias
Early direct coronary angioplasty in survivors of OOH-CA. Keelan et al., (6) 12804734	Determine impact of PPCI on OOH-CA VF survivors	Case series	15	OOH-CA, VF initial rhythm	Initial rhythm not VF	Survival to hospital discharge		None			15 pts with OOH-CA due to VF treated with PPCI, 11/14 survived.	Selection bias
Impact of PCI or CABG on outcome after nonfatal CA outside the hospital. Borger van der Burg et al., 2003 (7) 12667561	Determine impact of revascularization on outcome from OOH-CA	Case series	142	OOH-CA, VF/pVT as initial rhythm	VF/pVT in the setting of an AMI	2 y recurrence-free survival	Survival to hospital discharge	Kaplan-Meier	p<0.001		142 non-AMI, OOH-CA pts. Revascularized pts had a better recurrence-free survival.	Nonrandomized case series, selection bias

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Long-term prognosis after OOH-CA and PPCI. Bendz et al., 2004 (8) 15451586	Assess outcome in OOH-CA STEMI pts treated with PPCI	Case series	40	OOH-CA, STEMI	Interval from CA onset to start of CPR >10 min	Survival to hospital discharge		Kaplan-Meier comparison of 36 mo survival in OOH-CA STEMI pts receiving PPCI (n=40) vs nonarrest STEMI pts receiving PPCI n=325	p=NS between groups after discharge from hospital		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).	Nonrandomized case series, selection bias
Treatment and outcome in post-resuscitation care after OOH-CA when a modern therapeutic approach was introduced. Werling et al., 2007 (9) 17241730	Assess factors associated with outcome in OOH-CA pts undergoing early coronary angiography	Case series	85	OOH-CA		Survival to hospital discharge		Fisher's exact test	Factors associated with survival: initial VF p=0.002; coronary angiography p<0.0001; PCI p=0.003; CABG p=0.03; PCI or CABG p<0.0001	Factors associated with survival OR: 1. Initial VF OR: 5.7; 95% CI: 2.0-16.5 Coronary angiography OR: 9.1; 95% CI: 3.6-21.5 PCI OR: 6.8; 95% CI: 1.9-24.6; CABG OR 9.9; 95% CI: 1.1-93.5; PCI or CABG OR: 9.8; 95% CI: 3.0-32.3	85 pt case series, factors associated with increased survival: initial VF; coronary angiography; PCI; CABG, PCI or CABG.	Selection bias
Six-month outcome of emergency PCI in resuscitated pts after CA complicating STEMI. Garot et al., 2007 (10) 17353440	Determine impact of revascularization on outcome from OOH-CA	Case series	186	OOH-CA, STEMI, referred for PCI		Survival to 6 mo after hospital discharge		Multiple stepwise regression		Factors associated with 6 mo survival in pts receiving PPCI: absence of shock 12.7%; 95% CI: 3.4-47.6; absence of diabetes 7.3%; 95% CI: 1.6-29.4; absence of prior PCI 11.0%; 95% CI: 1.7-71.4	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.	Selection bias

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<p>PPCI after OOH-CA: pts and outcomes. Markusohn et al., 2007 (11) 17491217</p>	<p>To define the demographic, clinical and angiographic characteristics, and the prognosis of STEMI pts undergoing primary PCI after OOH-CA</p>	<p>Case series</p>	<p>25</p>	<p>OOH-CA, STEMI</p>		<p>1 y survival</p>	<p>1 y survival without severe disability</p>				<p>25 OOH-CA, STEMI pts receiving PPCI. 1 y survival 72%; 1 y survival without severe disability 64%.</p>	<p>Selection bias</p>
<p>Acute STEMI after successful CPR. Gorjup et al., 2007 (12) 17161902</p>	<p>To define the demographic, clinical and angiographic characteristics, and the prognosis of STEMI pts undergoing primary PCI after OOH-CA</p>	<p>Case series</p>	<p>135</p>	<p>CA, STEMI</p>		<p>Survival to hospital discharge with CPC 1 or 2</p>		<p>Ordinal logistic regression</p>	<p>Smoking p<0.001; inhospital arrest p=0.002; shockable rhythm p=0.005; motor response to pain p=0.007; corneal reflexes p<0.001; pupil light response p<0.001; breathing p<0.001; seizures p=0.02; PPCI p=0.02</p>	<p>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; corneal reflexes OR: 0.10; 95% CI: 0.01-0.64; pupil light response. OR: 0.06; 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</p>	<p>135 pts with STEMI, CA; predictors of survival included smoking, inhospital CA, shockable rhythm, neurological status on admission, PPCI</p>	<p>Selection bias</p>

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Thrombolytic therapy vs PPCI after VF CA due to STEMI and its effect on outcome. Richling et al., 2007 (13) 17543659	Assess outcome in OOH-CA STEMI pts treated with thrombolysis vs PPCI.	Case series	147 (thrombolysis, n=101; PPCI, n=46)	Witnessed OOH-CA, STEMI, VF initial rhythm, ROSC, treated with either thrombolysis or PPCI.		Best neurological outcome at 6 mo	6 mo mortality	Kaplan-Meier	CPC 1 or 2 at 6 mo comparing thrombolysis with PPCI p=0.58; survival at 6 mo p=0.17	CPC 1 or 2 at 6 mo comparing thrombolysis with PPCI aOR:1.24 95% CI: 0.48-2.62; survival at 6 mo aOR: 1.74 95% CI: 0.80-3.80	147 pt nonrandomized case series found no difference in 6 mo neurologically intact survival in OOH-CA, VF, STEMI pts treated with thrombolysis vs PPCI	Selection bias
Survival and neurologic recovery in pts with STEMI resuscitated from CA. Hosmane et al., 2009 (14) 19179198	Assess outcome in CA STEMI pts and predictors of survival	Case series	98	OOH-CA, STEMI	Refused permission for cath, died prior to cath, received thrombolytic therapy.	Survival to hospital discharge, neurological outcome		Multivariable logistic regression	Inhospital mortality lower in revascularized compared to nonrevascularized pts 25% vs 76%; p<0.0001		98 STEMI, OOH-CA pt case series showing inhospital mortality lower in revascularized compared to nonrevascularized pts.	Selection bias
Coronary angiography predicts improved outcome following CA: propensity-adjusted analysis. Reynolds et al., 2009 (15) 19321536	Use propensity-adjusted analysis to assess importance of coronary angiography in predicting outcome from OOH-CA	Case series	241	CA	Early withdrawal of care, first GCS obscured by a sedative or paralytic agent, planned emergent surgical intervention or immediate rearrest.	Discharge to home or acute rehabilitation facility "good outcome".		Propensity-adjusted analysis	Propensity-adjusted analysis showed that cath vs no cath associated with a good outcome independently 54.2% vs 24.8%; p<0.0001; Association between cath and good outcome p<0.02	Propensity adjusted logistic regression demonstrated an association between cath and good outcome OR: 2.16; 95% CI: 1.12-4.19	241 pt case series using propensity-adjusted analysis showing that cath vs no cath associated with a good outcome independently.	Not randomized

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Acute coronary angiographic findings in survivors of OOH-CA. Anyfantakis et al., 2009 (16) 19185639	Assess the prevalence of coronary lesions in OOH-CA survivors	Case series	72	OOH-CA		Coronary angiographic findings	Survival to hospital discharge	Multivariable analysis	64% had angiographic CAD, 38% had an acute lesion; PCI attempted in 33% ROSC p=0.0004; need for inotropic support during angiography p=0.0009	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	72 pt case series showing that 64% had angiographic CAD, 38% had an acute lesion; PCI attempted in 33%	Selection bias
Emergent PCI for resuscitated victims of OOH-CA. Kern et al., 2010 (17) 20049976	Assess the value of early angiography/ PCI and hypothermia in OOH-CA	Case series	5	OOH-CA		Coronary angiographic and ECG findings			Combining these therapies resulted in long-term survival rates of 70% with >80% of all such survivors neurologically functional		5 OOH-CA cases showing little correlation between ST-elevation on ECG and presence of an acute coronary lesion	Selection bias

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.

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Data Supplement 3. Antithrombotic Therapy for Primary PCI									
Trial Name	Study Type	N	n [# of pts who had STEMI (%=n/N)]	Study Population (experimental and comparator/control)	Primary Efficacy Endpoint	Primary Safety Endpoint	Selected Prespecified Subgroups	Subgroup/Other Analyses	Comments
CURRENT-OASIS 7 (18) 20817281	RCT	25,087 pts with ACS	7327 (29%)	2 X 2 factorial design. Pts with ACS randomized to either double dose clopidogrel (600 mg LD, followed by 150 mg/d for 6 d, then 75 mg/d) or standard dose clopidogrel (300 mg LD followed by 75 mg/d) and to either higher dose ASA (300-325 mg/d) or lower dose ASA (75-100 mg/d)	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 >65 or >75 y, body weight <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<0.001 and 0.7% vs 1.3% for <i>definite</i> 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: 0.86; 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.)	Subgroup analyses of the pts who underwent PCI after randomization are hypothesis generating. 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.

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<p>TRITON-TIMI 38 trial (19) 19249633</p>	<p>RCT</p>	<p>13,608 pts with moderate to high risk ACS</p>	<p>3534 (26%)</p>	<p>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.</p>	<p>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).</p>	<p>Major bleeding was observed in 2.4% of pts receiving prasugrel and in 1.8% of ptsreceiving 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).</p>	<p>UA or non-STEMI, STEMI, sex, age, diabetes mellitus, stent placement during index procedure, GP IIb/IIa,</p>	<p>A significant 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 greateramong the 3146 pts with diabetes (17.0% of whom had the primary end point in the clopidogrelgroup, 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 ofdefinite or probable stent thrombosis, as defined by the Academic Research Consortium, was significantlyreduced 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)</p>	<p>In subgroup analyses those with prior stroke/TIA fared worse with prasugrel and no advantage was seen in those >75 y or <60 kg. Pts who presented with STEMI for primary PCI were allowed to receive prasugrel or clopidogrel before angiography or PCI. Pts who presented with STEMI after 12 h to 14 d were randomized to study drug only after the coronary anatomy was defined.</p>
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<p>PLATO (20) 21060072</p>	<p>RCT</p>	<p>18,624 ACS pts</p>	<p>7026 (38%)</p>	<p>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)</p>	<p>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.</p>	<p>Major bleeding: There was no significant difference between ticagrelor and clopidogrel groups in the rates of major bleeding (691 [11.6%] vs 689 [11.2%], p=0.43).</p>	<p>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</p>	<p>Composite primary endpoint in 7,544 pts with ST-elevation or LBBB undergoing primary PCI was reduced from 10.8% in the clopidogrel arm to 9.4% in the ticagrelor arm; HR 0.87; 95% CI: 0.75-1.10; p=0.07. 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).</p>	<p>An interaction between the treatment effect and geographic region (North America) raises the possibility that higher doses of ASA used in that region beyond 100 mg daily may have an adverse effect. This observation, however, may be due to the play of chance.</p>
<p>ARMYDA-6 MI (21) 21958886</p>	<p>RCT</p>	<p>201</p>	<p>201 (100%)</p>	<p>Pts undergoing primary PCI for STEMI randomized to a 600 mg (n=103) or 300 mg (n=98) clopidogrel LD before the procedure</p>	<p>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.</p>	<p>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 3.1%.</p>	<p>N/A</p>	<p>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 48.8 + 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.</p>	<p>Surrogate endpoint trial underpowered for clinical events. Measurement of AUC less accurate than cardiac MRI for assessment of infarct size.</p>

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.

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Data Supplement 4. Early Catheterization and Rescue PCI for Fibrinolytic Failure in the Stent Era							
Study Name	Study Type	Study Size	Inclusion Criteria	Endpoints	Findings	Limitations	Comments
MERLIN, 2004 (22) 15261920	Randomized multicenter study of rescue angioplasty compared with continued medical therapy for pts with acute STEMI and failed thrombolysis.	307	STEMI \leq 10 h of onset of symptoms. CP >30 min ST-elevation \geq 2 mm in \geq 2 chest leads or 1 mm in \geq 2 limb leads. Failure to respond to FT at 60 min.	All-cause mortality at 30 d. Secondary EP: Composite of death, re-MI, CVA, CHF and clinically driven subsequent revascularization within 30 d RWMI	Death: Conservative vs rescue = 11% vs 9.8%; p=0.7 RD: 1.2; 95% CI: -5.8- 8.3 Composite Secondary EP: 50% vs 37.3%; p=0.02; RD: 12.7%; 95% CI: 1.6-23.5 Strokes: 4.6% vs 0.6%; p=0.03 RWMI was not different.		Rescue PCI had no significant effect on total mortality, although the secondary composite clinical endpoint was lower with rescue PCI compared with conservative care. Stroke rates were significantly higher in the rescue PCI group.
REACT, 2005 (23) 16382062	Randomized multicenter study to determine the best treatment for failed fibrinolysis by comparing rescue PCI to repeat fibrinolysis to conservative therapy.	427	Age 21 to 85 y, with evidence of failure of fibrinolysis; Rescue PCI could be performed within 12 h of onset of CP.	Composite of death, re-MI, CVA or severe CHF at 6 mo.	Rescue PCI vs repeat FT vs Conservative: 15.3% vs 31% vs 29.8%; p=0.003 PCI vs conservative: HR: 0.47; 95% CI: 0.28-0.79; p=0.004 PCI vs Re-FT: HR: 0.43; 95% CI: 0.26-0.72; p=0.001 Re-FT vs conservative therapy: HR: 1.09; 95% CI: 0.71-1.67; p=0.69 Minor bleeding more frequent with PCI No significant difference in major bleeding		Rescue PCI demonstrated a benefit when compared with conservative care or repeat fibrinolysis, although minor bleeding was significantly higher. Repeat FT did not offer any clinical benefit to conservative care.
Collet et al., 2006 (24, 25) 17258087 , 17010790	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.	920	Trials of pts with failed fibrinolysis randomized to rescue PCI or conservative care.	Mortality and Re-MI	Short term mortality: OR: 0.63; 95% CI: 0.39- 0.99; p=0.055 Long term mortality: OR: 0.69; 95% CI: 0.41-1.57; p=0.16 Short term mortality or Re-MI: OR: 0.60; 95% CI: 0.41-0.89; p=0.012 Long term mortality or Re-MI: OR: 0.60; 95% CI: 0.39- 0.92; p=0.019	Differences in study protocol, study endpoints and duration of follow-up.	Meta-analysis supported a strategy of rescue PCI for pts with clinical evidence of failure to reperfuse following fibrinolysis.

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					Higher rate of major bleeding with rescue PCI		
Wijeyesundera et al., 2007 (24) 17258087	Meta-analysis of the benefits of rescue PCI compared with either repeat fibrinolysis or conservative care.	1,177	Trials of pts with clinical or angiographic evidence of failed fibrinolysis randomized to rescue PCI, repeat fibrinolysis or conservative care.	Mortality and Re-MI, CHF, CVA, and bleeding	<p>Rescue PCI vs Conservative: Mortality: RR: 0.69; 95% CI: 0.46-1.05; p=0.09</p> <p>CHF: RR:0.73; 95% CI: 0.54-1.0; p=0.05</p> <p>Re-MI: RR=0.58; 95% CI: 0.35-0.97; p=0.04</p> <p>Composite of Death: re-MI and CHF RR: 0.72; 95% CI: 0.59-0.88; p=0.001</p> <p>CVA: RR: 4.98, 95% CI: 1.1- 22.5; p=0.04</p> <p>Minor bleeding: RR: 4.58; 95% CI: 2.46-8.55; p<0.001</p> <p>Rescue PCI vs repeat FT: Mortality RR: 0.68; 95% CI: 0.41-1.14; p=0.14</p> <p>Re-MI: RR:1.79; 95% CI: 0.92-3.48; p=0.09</p> <p>Minor bleeding: RR: 1.84; 95% CI: 1.06-3.18; p=0.03</p> <p>Major bleeding: RR: 1.54; 95% CI: 0.54-4.4; p=0.42</p>	Differences in study protocol, study endpoints and duration of follow-up.	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.

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.

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Data Supplement 5. Early Catheterization and PCI Following Fibrinolysis in the Stent Era							
Study Name	Study Type	Study Size	Inclusion Criteria	Endpoints	Findings	Limitations	Comments
SIAM III, 2003 (26) 12932593	Randomized multicenter trial of immediate stenting within 6 h of fibrinolysis vs delayed stenting at 2 wk.	195	Age >18 y, symptoms of AMI <12 h, ST-elevation of >1 mm in ≥2 limb leads and ST-elevation >2 mm in precordial leads, or new LBBB; no contraindication to lytics.	Composite of death, re-MI, ischemic events and TLR at 6 mo.	<u>Early stent vs delayed stent</u> MACE: 25.6% vs 50.6%; p=0.001 No differences in bleeding complications.	Analysis limited to only those pts who had stents	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.)
GRACIA, 2004 (27) 15380963	Randomized multicenter study of routine early cardiac cath (6 to 24 h) following fibrinolysis vs ischemia guided approach.	500	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.	Composite of death, re-MI and ischemia induced revascularization at 1 y. Note: In-hospital ischemia induced revascularization not considered part of primary endpoint.	<u>Early Cath vs Ischemia Guided</u> 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	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.
Lepzig Prehospital Fibrinolysis Study, 2005 (28) 16061501	Randomized multicenter study of prehospital fibrinolysis with PCI vs prehospital fibrinolysis alone and standard care.	164	Symptoms for at least 30 min and <6 h, and ST-elevation >0.1 mV in ≥2 limb leads or >0.2 mV in ≥2 precordial leads.	Final infarct size by MRI.	<u>Early Cath vs Standard Care</u> 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.	Small study and surrogate endpoints	Immediate cath and PCI following fibrinolysis resulted in smaller infarct size on MRI compared with standard care.
CAPITAL AMI, 2005 (29) 16053952	Randomized multicenter study of fibrinolysis with immediate transfer for cath vs fibrinolysis alone and transfer for unstable symptoms.	170	Symptoms ≤6 h and ≥30 min; ST-elevation ≥1 mm in ≥2 leads or LBBB and 1 of the following: AWMi; Extensive nonanterior MI; Killip class 3; SBP (22) <100 mmHg	Composite of death, re-MI, re-UA or CVA at 6 mo.	<u>Early Cath vs Ischemia-Guided Approach</u> 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.	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 1 st wk of care.
Di Pasquale et al., 2006 (30) 16622610	Randomized single-center study of immediate cath <2 h and PCI vs delayed PCI 12 to 24 h after fibrinolysis.	451	First STEMI ≤12 h from symptom onset, with ST-elevation >1 mm in peripheral leads, and or 2 mm in	Ischemic events (MI, abnormal stress test, restenosis, and death) at 6 mo.	<u>Immediate Cath vs Delayed Cath</u> Ischemic events 18.2% vs 9.7%; p=0.005 More minor bleeding in immediate PCI	Pts only included following successful reperfusion.	Study failed to show a benefit to immediate cath and PCI within 2 h, compared with early cath and PCI at 12 to 72 h among pts who have demonstrated evidence of successful

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			precordial leads involving >1 lead, Killip class 1-2, acceptable echo window, and abnormal wall motion on echo. Baseline CPK and TRP normal. Successful reperfusion following lytic therapy. Age of >18 or <75 y.		group. No difference in major bleeding.	Pts treated with unapproved regimen of half dose lytic and GPI.	reperfusion following cath.
WEST, 2006 (31) 16757491	Randomized multicenter feasibility study of PCI vs fibrinolysis with early cath (within 24 h) vs fibrinolysis with standard care.	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.	Efficacy: 30 d composite of death, re-MI, reischemia, CHF, shock or major ventricular arrhythmias. Safety endpoints: ICH, CVA, major bleeding.	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; 90% CI: 0.11- 0.74); P-log rank=0.021)
Collet et al., 2006 (25) 17010790	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.	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).	Mortality and Death/MI	<u>Early Cath vs Ischemia Driven Cath</u> <u>Death:</u> 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) <u>Death and MI</u> 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	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”.
Wijeyesundera, 2008 (24) 17258087	A meta-analysis of trials examining fibrinolysis with immediate transfer for cath with	1,235	Clinical trials of STEMI pts receiving fibrinolysis and randomized to routine early	All-cause mortality, Recurrent MI	<u>Immediate Cath vs Ischemia Driven Cath</u> Mortality: OR: 0.55; 95% CI: 0.34- 0.90; p=0.02;	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

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	fibrinolysis and an ischemia-guided approach.		invasive management compared with ischemia driven cath in the "stent era".		Re-MI: OR: 0.53; 95% CI: 0.33- 0.86; p=0.01 No difference in stroke or major bleeding	each trial, and different durations of follow-up.	era".
CARESS-AMI, 2008 (32) 18280326	Randomized multicenter trial of immediate transfer for PCI following FT in high risk patient compared with standard care and rescue PCI.	600	STEMI with symptoms ≤12 h, and ≥1 high-risk features: Cumulative ST-elevation of >15 mm, new onset LBBB, prior MI, Killip class ≥2, or LVEF ≤35%.	Composite of all-cause death, re-MI and refractory ischemia at 30 d.	<u>Early Cath vs Standard Care</u> 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).	Used an unapproved regimen of half dose RPA.	Study demonstrated a benefit to immediate transfer of high-risk pts with STEMI following fibrinolysis compared with transfer for rescue PCI or standard care. The primary endpoint was driven largely by recurrent ischemia.
TRANSFER AMI, 2009 (33) 19553646	Randomized multicenter trial of FT followed by immediate transfer for cath compared with fibrinolysis and standard care (rescue cath/or cath 24 h to 2 wk).	1,059	Symptoms ≤12 h and ST-elevation ≥2 mm in anterior leads, or ST ≥1 mm in the inferior leads with: SBP <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.	<u>Early Cath vs Delayed Cath</u> 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.		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).
NORDSTEMI, 2010 (34) 19747792	Multicenter randomized study of FT and immediate transfer for PCI compared with FT and standard care.	276	Age 18 to 75 y, symptoms <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.	<u>Early Cath vs Routine Care</u> <u>Primary Endpoint:</u> 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.		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 2 nd endpoint when compared with standard care (rescue PCI/ ischemia guided PCI or routine cath done 2 to 4 wk) following fibrinolysis.
Borgia et al., 2010 (35) 20601393	A meta-analysis of trials examining fibrinolysis with immediate transfer for cath with fibrinolysis alone and standard care.	2,961	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	<u>Early Cath vs Delayed Cath or Ischemia Driven Cath</u> <u>30 d Death</u> 3.3% vs 3.8%; OR: 0.87; 95% CI: 0.59-1.30; p=0.51 <u>30 d Re-MI</u> 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.	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

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				stroke.	<p>p=0.003</p> <p><u>30 d Death/Re-MI</u> 5.6 vs 8.3%; OR: 0.65; 95% CI: 0.49-0.88; p=0.004</p> <p><u>30 d Recurrent ischemia</u> 1.9 vs 7.1%; OR: 0.25; 95% CI: 0.13- 0.49; p<0.001</p> <p><u>6 to 12 Mo Death</u> 4.8 vs 5.4%; OR: 0.88; 95% CI: 0.62-1.25; p=0.48</p> <p><u>6 to 12 Mo Re-MI</u> 3.9 vs 6%; OR: 0.64; 95% CI: 0.40-0.98; p=0.01</p> <p><u>6 to 12 Mo Death/Re-MI</u> 8.6 vs 11.2%; OR: 0.71; 95% CI: 0.52-0.97; p=0.03</p> <p>No difference in Major bleeding. No difference in stroke.</p>	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.
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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.

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