2007 Focused Update of the ACC/AHA/SCAI 2005 Guideline Update for Percutaneous Coronary Intervention

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

2007 Writing Group to Review New Evidence and Update the ACC/AHA/SCAI 2005 Guideline Update for Percutaneous Coronary Intervention, Writing on Behalf of the 2005 Writing Committee

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This document is a limited update to the 2005 guideline update and is based on a review of certain evidence, not a full literature review.

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Preamble

A primary challenge in the development of clinical practice guidelines is keeping pace with the stream of new data upon which recommendations are based. In an effort to respond more quickly to new evidence, the American College of Cardiology/American Heart Association (ACC/AHA) Task Force on Practice Guidelines has created a new "focused update" process to revise the existing guideline recommendations that are affected by evolving data or opinion. Before the initiation of this focused approach, periodic updates and revisions of existing guidelines required up to 3 years to complete. Now, however, new evidence will be reviewed in an ongoing fashion to more efficiently respond to important science and treatment trends that could have a major impact on patient outcomes and quality of care. Evidence will be reviewed at least twice a year, and updates will be initiated on an as needed basis as quickly as possible while maintaining the rigorous methodology that the ACC and AHA have developed during their more than 20 years of partnership.

These updated guideline recommendations reflect a consensus of expert opinion following a thorough review primarily of late-breaking clinical trials identified through a broad-based vetting process as important to the relevant patient population and of other new data deemed to have an impact on patient care (see Section 1.1 for details regarding this focused update). It is important to note that this focused update is not intended to represent an update based on a full literature review from the date of the previous guideline publication. Specific criteria/considerations for inclusion of new data include:

- Publication in a peer-reviewed journal
- Large, randomized, placebo-controlled trial(s)
- Nonrandomized data deemed important on the basis of results that impact current safety and efficacy assumptions
- Strengths/weakness of research methodology and findings
- Likelihood of additional studies influencing current findings
- Impact on current performance measure(s) and/or likelihood of the need to develop new performance measure(s)
- Requests and requirements for review and update from the practice community, key stakeholders, regulatory agencies, and other sources free of relationships with industry or other potential bias
- Number of previous trials showing consistent results
- Need for consistency with other new guidelines or guideline revisions

In analyzing the data and developing updated recommendations and supporting text, the focused update writing group used evidence-based methodologies developed by the ACC/AHA Task Force on Practice Guidelines, which are described elsewhere.[1,2]

The schema for class of recommendation and level of evidence is summarized in Table 1, which also illustrates how the grading system provides estimates of the size of the treatment effect and the certainty of the treatment effect. Note that a recommendation with Level of Evidence B or C does not imply that the recommendation is weak. Many important clinical questions addressed in guidelines do not lend themselves to clinical trials. Although randomized trials may not be available, there may be a very clear clinical consensus that a particular test or therapy is useful and effective. Both the class of recommendation and level of evidence listed in the focused updates are based on consideration of the evidence reviewed in previous iterations of the guidelines as well as the focused update. Of note, the implications of older studies that have informed recommendations but have not been repeated in contemporary settings are carefully considered.

The ACC/AHA practice guidelines address patient populations (and health care providers) residing in North America. As such, drugs that are not currently available in North America are discussed in the text without a specific class of recommendation. For studies performed in large numbers of subjects outside of North America, each writing committee reviews the potential impact of different practice patterns and patient populations on the treatment effect and on the relevance to the ACC/AHA target population to determine whether the findings should form the basis of a specific recommendation.

The ACC/AHA practice guidelines are intended to assist health care providers in clinical decision making by describing a range of generally acceptable approaches for 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

Table 1. Applying Classification of Recommendations and Level of Evidence†

SIZE OF TREATMENT EFFECT →

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

*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations, such as gender, age, history of diabetes, history of prior myocardial infarction, history of heart failure, and prior aspirin use. 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. Even though randomized trials are not available, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

†In 2003, the ACC/AHA Task Force on Practice Guidelines developed a list of suggested phrases to use when writing recommendations. All guideline recommendations have been written in full sentences that express a complete thought, such that a recommendation, even if separated and presented apart from the rest of the document (including headings above sets of recommendations), would still convey the full intent of the recommendation. It is hoped that this will increase readers' comprehension of the guidelines and will allow queries at the individual recommendation level.

circumstances. The ultimate judgment regarding care of a particular patient must be made by the health care provider and patient in light of all the circumstances presented by that patient. Thus, there are circumstances in which deviations from these guidelines may be appropriate. Clinical decision making should consider the quality and availability of expertise in the area where care is provided. These guidelines may be used as the basis for regulatory or payer decisions, but the ultimate goal is quality of care and serving the patient's best interests.

Prescribed courses of treatment in accordance with these recommendations are only effective if they are followed by the patient. Because lack of patient adherence may adversely affect treatment outcomes, health care providers should make every effort to engage the patient in active participation with prescribed treatment.

The ACC/AHA Task Force on Practice Guidelines makes every effort to avoid any actual, potential, or perceived conflict of interest arising from industry relationships or personal interests of a writing committee member. All writing committee members and peer reviewers were required to provide disclosure statements of all such relationships pertaining to the trials and other evidence under consideration (see Appendixes 1 and 2). Final recommendations were balloted to all writing committee members. Writing committee members with significant (greater than $10 000) relevant relationships with industry (RWI) were required to recuse themselves from voting on that recommendation. Writing committee members who did not participate are not listed as authors of this focused update.

With the exception of the recommendations presented in this statement, the full guidelines remain current. Only the

recommendations from the affected section(s) of the full guidelines are included in this focused update. For easy reference, all recommendations from any section of guidelines impacted by a change are presented with a notation as to whether they remain current, are new, or have been modified. When evidence impacts recommendations in more than 1 set of guidelines, those guidelines are updated concurrently.

The recommendations in this focused update will be considered current until they are superseded by another focused update or the full-text guidelines are revised. This focused update is published in the January 15, 2008, issue of the *Journal of the American College of Cardiology*, the January 15, 2008, issue of *Circulation*, and e-published in *Catheterization and Cardiovascular Interventions* as an update to the full-text guidelines and is posted on the ACC (www.acc.org), AHA (my.americanheart.org), and Society for Angiography and Interventions (SCAI) (www.scai.org) Web sites. Copies of the focused update are available from all organizations.

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1. Introduction

1.1. Evidence Review

Selected late-breaking clinical trials presented at the 2005 and 2006 annual scientific meetings of the ACC, AHA, and European Society of Cardiology, as well as selected other data, were reviewed by the standing guideline writing committee along with the parent Task Force and other experts to identify those trials and other key data that might impact guideline recommendations. On the basis of the criteria/considerations noted above, recent trial data and other clinical information were considered important enough to prompt a focused update of the ACC/AHA/SCAI 2005 Guideline Update for Percutaneous Coronary Intervention.[3–13]

To provide clinicians with a comprehensive set of data, whenever possible, the exact event rates in various treatment arms of clinical trials are presented to permit calculation of the absolute risk difference (ARD) and number needed to treat (NNT) or harm (NNH); the relative treatment effects are described either as odds ratio (OR), relative risk (RR), or hazard ratio (HR), depending on the format in the original publication.

Consult the full-text version or executive summary of the ACC/AHA/SCAI 2005 Guideline Update for Percutaneous Coronary Intervention for policy on clinical areas not covered by the focused update.[13a] Individual recommendations updated in this focused update will be incorporated into future revisions and/or updates of the full-text guidelines.

1.2. Organization of Committee and Relationships With Industry

For this focused update, all members of the 2005 PCI writing committee were invited to participate; those who agreed (referred to as the 2007 focused update writing group) were required to disclose all RWI relevant to the data under consideration.[2] Focused update writing group members who had no significant relevant RWI wrote the first draft of the focused update; the draft was then reviewed and revised by the full writing group. Each recommendation required a confidential vote by the writing group members before external review of the document. Any writing committee member with a significant (greater than $10 000) RWI relevant to the recommendation was recused from voting on that recommendation.

1.3. Review and Approval

This document was reviewed by 2 outside reviewers nominated by each cosponsoring organization (ACC, AHA, and SCAI) and 24 individual content reviewers. All reviewer RWI information was collected and distributed to the writing committee and is published in this document (see Appendix 2 for details).

This document was approved for publication by the governing bodies of the American College of Cardiology Foundation, AHA, and SCAI.

2. Patients With Unstable Angina/Non–ST-Elevation Myocardial Infarction

This 2007 PCI Focused Update section regarding patients with unstable angina (UA)/non–ST-elevation myocardial infarction (NSTEMI) is based on recommendations from the ACC/AHA 2007 Guidelines for the Management of Patients With Unstable Angina/Non–ST-Elevation Myocardial Infarction,[14] which emphasize the importance of assessing risk of cardiovascular events as a guide to therapeutic decision making and the need for interventional therapy (see Table 2).

Because of the importance of several new changes in the ACC/AHA 2007 UA/NSTEMI Guidelines, selected text from the guidelines is included in the following paragraphs and summarized in Table 2.

A number of risk-assessment tools have been developed to assist in assessing risk of death and ischemic events in patients with UA/NSTEMI, thereby providing a basis for therapeutic decision making. It should be recognized that the predictive ability of these commonly used risk assessment scores for risk of nonfatal coronary heart disease (CHD) is only moderate.

The Thrombolysis in Myocardial Infarction (TIMI) risk score[15] is a simple tool composed of 7 (1-point) risk indicators rated on presentation (Table 4). The composite end points (all-cause mortality, new or recurrent myocardial infarction [MI], or severe recurrent ischemia prompting urgent revascularization within 14 days) increase as the TIMI risk score increases. The TIMI risk score has been validated internally within the TIMI IIB trial and 2 separate cohorts of patients from the ESSENCE (Efficacy and Safety of Subcutaneous Enoxaparin in Unstable Angina and Non–Q-Wave Myocardial Infarction) trial.[16] The model remained a significant predictor of events and appeared relatively insensitive to missing information, such as knowledge of previously documented coronary stenosis of 50% or greater. The model's

predictive ability remained intact, with a cutoff of 65 years of age. The TIMI risk score was recently studied in an unselected emergency department population with chest pain syndrome; its performance was similar to that in the acute coronary syndrome (ACS) population from which it was derived and validated.[17] The TIMI risk calculator is available at www.timi.org. The TIMI risk index, a modification of the TIMI risk score that uses the variables age, systolic blood pressure, and heart rate, has not only been shown to predict short-term mortality in ST-elevation myocardial infarction (STEMI) but also has been useful in prediction of 30-day and 1-year mortality rates across the spectrum of patients with ACS, including UA/NSTEMI.[18]

The PURSUIT (Platelet Glycoprotein IIb-IIIa in Unstable Angina: Receptor Suppression Using Integrilin Therapy) trial risk model,[19] based on patients enrolled in the PURSUIT trial, is another useful tool to guide the clinical decision-making process when the patient is admitted to the hospital. In the PURSUIT risk model, critical clinical features associated with an increased 30-day incidence of death and the composite of death or myocardial (re)infarction were (in order of strength) age, heart rate, systolic blood pressure, ST-segment depression, signs of heart failure (HF), and cardiac enzymes.[19]

The GRACE (Global Registry of Acute Coronary Events) study risk model, which predicts in-hospital mortality (and death or MI), can be useful to clinicians to guide treatment type and intensity.[20,21] The GRACE risk tool was developed on the basis of 11 389 patients in GRACE and validated in subsequent GRACE and GUSTO (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) IIb cohorts and predicts in-hospital death in patients with STEMI, NSTEMI, or UA (C statistic=0.83). The 8 variables used in the GRACE risk model are older age (OR 1.7 per 10 years), Killip class (OR 2.0 per class), systolic blood pressure (OR 1.4 per 20 mm Hg decrease), ST-segment deviation (OR 2.4), cardiac arrest during presentation (OR 4.3), serum creatinine level (OR 1.2 per 1 mg per dL increase), positive initial cardiac markers (OR 1.6), and heart rate (OR 1.3 per 30-bpm increase). The sum of scores is applied to a reference nonogram to determine the corresponding all-cause mortality from hospital discharge to 6 months. The GRACE clinical application tool can be downloaded to a handheld PDA (personal digital

Table 2. Updates to Section 5.3: Initial Conservative Versus Initial Invasive Strategies (Patients With UA/NSTEMI)

2005 PCI Guideline Update Recommendation	2007 PCI Focused Update Recommendation	Comments
	Class I	
An early invasive PCI strategy is indicated for patients with UA/NSTEMI who have no serious comorbidity† and coronary lesions amenable to PCI. Patients must have any of the following high-risk features: a. Recurrent ischemia despite intensive anti-ischemic therapy. *(Level of Evidence: A)* b. Elevated troponin level. *(Level of Evidence: A)* c. New ST-segment depression. *(Level of Evidence: A)* d. HF symptoms or new or worsening MR. *(Level of Evidence: A)* e. Depressed LV systolic function. *(Level of Evidence: A)* f. Hemodynamic instability. *(Level of Evidence: A)* g. Sustained ventricular tachycardia. *(Level of Evidence: A)* h. PCI within 6 months. *(Level of Evidence: A)* i. Prior CABG. *(Level of Evidence: A)* j. High risk score (e.g., TIMI, GRACE). *(Level of Evidence: A)* k. High risk findings from non-invasive testing. *(Level of Evidence: A)*	1. An early invasive PCI strategy is indicated for patients with UA/NSTEMI who have no serious comorbidity† and who have coronary lesions amenable to PCI and who have characteristics for invasive therapy (see Table 3 and Section 3.3 of the ACC/AHA 2007 UA/NSTEMI Guidelines).[14] *(Level of Evidence: A)*	Modified recommendation*
	2. Percutaneous coronary intervention (or CABG) is recommended for UA/NSTEMI patients with 1- or 2-vessel CAD with or without significant proximal left anterior descending CAD but with a large area of viable myocardium and high-risk criteria on noninvasive testing. *(Level of Evidence: B)*	New recommendation*
	3. Percutaneous coronary intervention (or CABG) is recommended for UA/NSTEMI patients with multivessel coronary disease with suitable coronary anatomy, with normal LV function, and without diabetes mellitus. *(Level of Evidence: A)*	New recommendation*
	4. An intravenous platelet GP IIb/IIIa inhibitor is useful in UA/NSTEMI patients undergoing PCI. *(Level of Evidence: A)* See Section 3.2.3 and Table 13 of the 2007 ACC/AHA 2007 UA/NSTEMI Guidelines.[14]	New recommendation*
	5. An early invasive strategy (i.e., diagnostic angiography with intent to perform revascularization) is indicated in UA/NSTEMI patients who have refractory angina or hemodynamic or electrical instability (without serious comorbidities or contraindications to such procedures). *(Level of Evidence: B)*	New recommendation*

Table 2. Continued

2005 PCI Guideline Update Recommendation	2007 PCI Focused Update Recommendation	Comments
	Class IIa	
It is reasonable that PCI be performed in patients with UA/NSTEMI and single-vessel or multivessel CAD who are undergoing medical therapy with focal saphenous vein graft lesions or multiple stenoses who are poor candidates for reoperative surgery. *(Level of Evidence: C)*	1. Percutaneous coronary intervention is reasonable for focal saphenous vein graft lesions or multiple stenoses in UA/NSTEMI patients who are undergoing medical therapy and who are poor candidates for reoperative surgery. *(Level of Evidence: C)*	Modified recommendation*
In the absence of high-risk features associated with UA/NSTEMI, it is reasonable to perform PCI in patients with amenable lesions and no contraindication for PCI with either an early invasive or early conservative strategy. *(Level of Evidence: B)*		Deleted recommendation*
	2. Percutaneous coronary intervention (or CABG) is reasonable for UA/NSTEMI patients with 1- or 2-vessel CAD with or without significant proximal left anterior descending CAD but with a moderate area of viable myocardium and ischemia on noninvasive testing. *(Level of Evidence: B)*	New recommendation*
	3. Percutaneous coronary intervention (or CABG) can be beneficial compared with medical therapy for UA/NSTEMI patients with 1-vessel disease with significant proximal left anterior descending CAD. *(Level of Evidence: B)*	New recommendation*
Use of PCI is reasonable in patients with UA/NSTEMI with significant left main CAD (greater than 50% diameter stenosis) who are candidates for revascularization but are not eligible for CABG. *(Level of Evidence: B)*	4. Use of PCI is reasonable in patients with UA/NSTEMI with significant left main CAD (greater than 50% diameter stenosis) who are candidates for revascularization but are not eligible for CABG or who require emergency intervention at angiography for hemodynamic instability. *(Level of Evidence: B)*	2005 recommendation remains current in 2007 PCI Update but receives additional wording.
	Class IIb	
In the absence of high-risk features associated with UA/NSTEMI, PCI may be considered in patients with single-vessel or multivessel CAD who are undergoing medical therapy and who have 1 or more lesions to be dilated with a less than optimal likelihood of success. *(Level of Evidence: B)*	1. In the absence of high-risk features associated with UA/NSTEMI, PCI may be considered in patients with single-vessel or multivessel CAD who are undergoing medical therapy and who have 1 or more lesions to be dilated with a reduced likelihood of success. *(Level of Evidence: B)*	Modified recommendation*
PCI may be considered in patients with UA/NSTEMI who are undergoing medical therapy who have 2- or 3-vessel disease, significant proximal LAD CAD, and treated diabetes or abnormal LV function. *(Level of Evidence: B)*	2. PCI may be considered in patients with UA/NSTEMI who are undergoing medical therapy who have 2- or 3-vessel disease, significant proximal left anterior descending CAD, and treated diabetes or abnormal LV function, with anatomy suitable for catheter-based therapy. *(Level of Evidence: B)*	2005 recommendation remains current in 2007 PCI Update but receives additional wording.
	3. In initially stabilized patients, an initially conservative (i.e., a selectively invasive) strategy may be considered as a treatment strategy for UA/NSTEMI patients (without serious comorbidities or contraindications to such procedures†) who have an elevated risk for clinical events (see Table 3) including those who are troponin positive. *(Level of Evidence: B).* The decision to implement an initial conservative (versus initial invasive) strategy‡ in these patients may be made by considering physician and patient preference. *(Level of Evidence: C)*	New recommendation*
	4. An invasive strategy may be reasonable in patients with chronic renal insufficiency. *(Level of Evidence: C)*	New recommendation*
	Class III	
	1. Percutaneous coronary intervention (or CABG) is not recommended for patients with 1- or 2-vessel CAD without significant proximal left anterior descending CAD with no current symptoms or symptoms that are unlikely to be due to myocardial ischemia and who have no ischemia on noninvasive testing. *(Level of Evidence: C)*	New recommendation*

Table 2. Continued

2005 PCI Guideline Update Recommendation	2007 PCI Focused Update Recommendation	Comments
In the absence of high-risk features associated with UA/NSTEMI, PCI is not recommended for patients with UA/NSTEMI who have single-vessel or multivessel CAD and no trial of medical therapy, or who have 1 or more of the following: a. Only a small area of myocardium at risk. *(Level of Evidence: C)* b. All lesions or the culprit lesion to be dilated with morphology that conveys a low likelihood of success. *(Level of Evidence: C)* c. A high risk of procedure-related morbidity or mortality. *(Level of Evidence: C)* d. Insignificant disease (less than 50% coronary stenosis). *(Level of Evidence: C)* e. Significant left main CAD and candidacy for CABG. *(Level of Evidence: B)*	2. In the absence of high-risk features associated with UA/NSTEMI, PCI is not recommended for patients with UA/NSTEMI who have single-vessel or multivessel CAD and no trial of medical therapy, or who have 1 or more of the following: a. Only a small area of myocardium at risk. *(Level of Evidence: C)* b. All lesions or the culprit lesion to be dilated with morphology that conveys a low likelihood of success. *(Level of Evidence: C)* c. A high risk of procedure-related morbidity or mortality. *(Level of Evidence: C)* d. Insignificant disease (less than 50% coronary stenosis). *(Level of Evidence: C)* e. Significant left main CAD and candidacy for CABG. *(Level of Evidence: B)*	2005 recommendation remains current in 2007 PCI Update
	3. A PCI strategy in stable patients (see Table 12 Class III No. 1 for specific recommendations) with persistently occluded infarct related coronary arteries after STEMI/NSTEMI is not indicated. *(Level of Evidence: B)*	New recommendation*

*Based on the ACC/AHA 2007 UA/NSTEMI Guidelines.[14]

†For example, severe hepatic, pulmonary, or renal failure, or active/inoperable cancer. Clinical judgment is required in such cases.

‡Diagnostic angiography with intent to perform revascularization.

CABG indicates coronary artery bypass graft; CAD, coronary artery disease; GP, glycoprotein; GRACE, Global Registry of Acute Coronary Events; HF, heart failure; IV, intravenous; LAD, left anterior descending; LV, left ventricular; MR, mitral regurgitation; PCI, percutaneous coronary intervention; SVG, saphenous vein graft; TIMI, Thrombolysis in Myocardial Infarction; and UA/NSTEMI, unstable angina/non–ST-elevation myocardial infarction.

assistant) to be used at the bedside and is available at www.outcomes-umassmed.org/grace (Figure 1).[21] An analysis comparing the 3 risk scores (TIMI, GRACE, and PURSUIT) concluded that all 3 demonstrated good predictive accuracy for death and MI at 1 year, thus identifying patients who might be likely to benefit from aggressive therapy, including early myocardial revascularization.[22]

Table 3. Selection of Initial Treatment Strategy: Invasive Versus Conservative Strategy

Preferred Strategy	Patient Characteristics
Invasive	Recurrent angina or ischemia at rest or with low-level activities despite intensive medical therapy
	Elevated cardiac biomarkers (TnT or TnI)
	New or presumably new ST-segment depression
	Signs or symptoms of HF or new or worsening mitral regurgitation
	High-risk findings from noninvasive testing
	Hemodynamic instability
	Sustained ventricular tachycardia
	PCI within 6 months
	Prior CABG
	High-risk score (e.g., TIMI, GRACE)
	Reduced LV function (LVEF less than 40%)
Conservative	Low-risk score (e.g., TIMI, GRACE)
	Patient or physician preference in absence of high-risk features

Reprinted from the ACC/AHA 2007 UA/NSTEMI Guidelines.[14]

CABG indicates coronary artery bypass graft surgery; GRACE, Global Registry of Acute Coronary Events; HF, heart failure; LV, left ventricular; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention; TIMI, Thrombolysis In Myocardial Infarction; TnI, troponin I; and TnT, troponin T.

The electrocardiogram (ECG) provides unique and important diagnostic and prognostic information (see also Section 2.1 below). Accordingly, ECG changes have been incorporated into quantitative decision aids for the triage of patients who present with chest discomfort.[23] Although ST elevation carries the highest early risk of death, ST depression on the presenting ECG portends the highest risk

Table 4. TIMI Risk Score for Unstable Angina/ Non–ST-Elevation Myocardial Infarction

TIMI Risk Score	All-Cause Mortality, New or Recurrent MI, or Severe Recurrent Ischemia Requiring Urgent Revascularization Through 14 Days After Randomization, %
0–1	4.7
2	8.3
3	13.2
4	19.9
5	26.2
6–7	40.9

The TIMI risk score is determined by the sum of the presence of 7 variables at admission; 1 point is given for each of the following variables: age 65 years or older; at least 3 risk factors for CAD; prior coronary stenosis of 50% or more; ST-segment deviation on ECG presentation; at least 2 anginal events in prior 24 hours; use of aspirin in prior 7 days; and elevated serum cardiac biomarkers. Prior coronary stenosis of 50% or more remained relatively insensitive to missing information and remained a significant predictor of events. Reprinted with permission from Antman EM, Cohen M, Bernink PJ, et al. The TIMI risk score for unstable angina/non-ST elevation MI: a method for prognostication and therapeutic decision making. *JAMA.* 2000;284:835–42.[15] Copyright © 2000 American Medical Association.

CAD indicates coronary artery disease; ECG, electrocardiogram; MI, myocardial infarction; and TIMI, Thrombolysis in Myocardial Infarction.

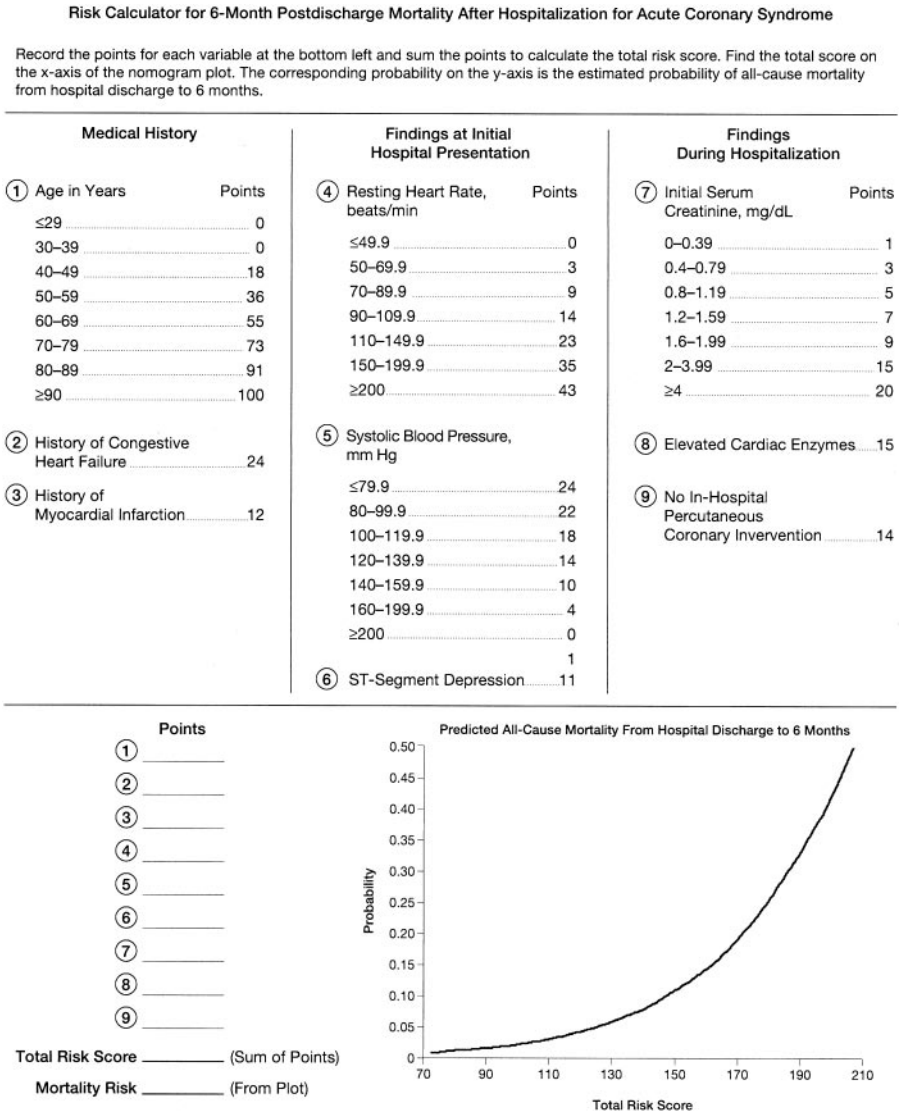

Figure 1. GRACE Prediction Score Card and Nomogram for All-Cause Mortality From Discharge to 6 Months. Reprinted with permission.[20] Copyright © 2004 American Medical Association.

of death at 6 months, with the degree of ST-segment depression showing a strong relationship to outcome.[24]

The recommendations in the ACC/AHA 2007 UA/NSTEMI Guidelines[14] recognize recent data from the ACUITY (Acute Catheterization and Urgent Intervention Triage strategY) trial, which showed that in patients with ACS who were undergoing invasive treatment, bivalirudin alone was associated with rates of ischemia similar to those treated with glycoprotein (GP) IIb/IIIa inhibitors plus heparin and significantly less bleeding.[25]

The ACC/AHA 2007 UA/NSTEMI Guidelines cite a progressively greater benefit from newer, more aggressive therapies such as low-molecular-weight heparin (LMWH),[16,26] platelet GP IIb/IIIa inhibition,[27] and an invasive strategy[28] with increasing risk score.

2.1. Electrocardiogram

The ECG lies at the center of the decision pathway for the evaluation and management of patients with acute ischemic discomfort (Table 5). The diagnosis of MI is confirmed with serial cardiac biomarkers in more than 90% of patients who present with ST-segment elevation greater than or equal to 1 mm (0.1 mV) in at least 2 contiguous leads, and such patients should be considered candidates for acute reperfusion therapy. Patients who present with ST-segment depression are initially considered to have either UA or NSTEMI; the distinction between the 2 diagnoses is ultimately based on the detection of markers of myocardial necrosis in the blood.[29–31]

Up to 25% of patients with NSTEMI and elevated CK-MB go on to develop Q-wave MI during their hospital stay, whereas the remaining 75% have non–Q-wave MI. Acute fibrinolytic therapy is contraindicated for ACS patients without ST-segment elevation, except for those with electrocardiographic true posterior MI manifested as ST-segment depression in 2 contiguous anterior precordial leads and/or isolated ST-segment elevation in posterior chest lead.[32–34] Inverted T waves may also indicate UA/NSTEMI. In patients suspected of having ACS on clinical

Table 5. Likelihood That Signs and Symptoms Represent an Acute Coronary Syndrome Secondary to CAD

Feature	High Likelihood *Any of the following:*	Intermediate Likelihood *Absence of high-likelihood features and presence of any of the following:*	Low Likelihood *Absence of high- or intermediate-likelihood features but may have the following:*
History	Chest or left arm pain or discomfort as chief symptom reproducing prior documented angina Known history of CAD, including MI	Chest or left arm pain or discomfort as chief symptom Age greater than 70 years Male sex Diabetes mellitus	Probable ischemic symptoms in absence of any intermediate-likelihood characteristics Recent cocaine use
Examination	Transient MR murmur, hypotension, diaphoresis, pulmonary edema, or rales	Extracardiac vascular disease	Chest discomfort reproduced by palpation
ECG	New, or presumably new, transient ST-segment deviation (1 mm or greater) or T-wave inversion in multiple precordial leads	Fixed Q waves ST depression 0.5 to 1 mm or T-wave inversion greater than 1 mm	T-wave flattening or inversion less than 1 mm in leads with dominant R waves Normal ECG
Cardiac markers	Elevated cardiac TnI, TnT, or CK-MB	Normal	Normal

Modified from reference 46. In the public domain.

CAD indicates coronary artery disease; CK-MB, MB fraction of creatine kinase; ECG, electrocardiogram; MI, myocardial infarction; MR, mitral regurgitation; TnI, troponin I; and TnT, troponin T.

grounds, marked (greater than or equal to 2 mm [0.2 mV]) symmetrical precordial T-wave inversion strongly suggests acute ischemia, particularly that associated with a critical stenosis of the left anterior descending coronary artery (LAD).[35] Patients with this ECG finding often exhibit hypokinesis of the anterior wall and are at high risk if given medical treatment alone.[36] Revascularization will often reverse both the T-wave inversion and wall-motion disorder.[37] Nonspecific ST-segment and T-wave changes, usually defined as ST-segment deviation less than 0.5 mm (0.05 mV) or T-wave inversion less than or equal to 2 mm (0.2 mV), are less diagnostically helpful than the foregoing findings. Established Q waves greater than or equal to 0.04 second are also less helpful in the diagnosis of UA, although by suggesting prior MI, they do indicate a high likelihood of significant coronary artery disease (CAD). Isolated Q waves in lead III may be a normal finding, especially in the absence of repolarization abnormalities in any of the inferior leads. A completely normal ECG in a patient with chest pain does not exclude the possibility of ACS, because 1% to 6% of such patients eventually are proven to have had an MI (by definition, NSTEMI), and at least 4% will be found to have UA.[38–40]

In addition to the presence or absence of ST-segment deviation or T-wave inversion patterns noted earlier, there is evidence that the magnitude of the ECG abnormality provides important prognostic information. Thus, Lloyd-Jones et al.[41] reported that the diagnosis of acute non–Q-wave MI was 3 to 4 times more likely in patients with ischemic discomfort who had at least 3 ECG leads that showed ST-segment depression and maximal ST depression of greater than or equal to 0.2 mV. Investigators from the TIMI III Registry[42] reported that the 1-year incidence of death or new MI in patients with at least 0.5 mm (0.05 mV) of ST-segment deviation was 16.3% compared with 6.8% for patients with isolated T-wave changes and 8.2% for patients with no ECG changes.

Cardiogenic shock can occur in the setting of both STEMI and NSTEMI, and there is high mortality and morbidity in each. The SHOCK (SHould we emergently revascularize Occluded Coronaries for cardiogenic shocK) study[43] found that approximately 20% of all cardiogenic shock complicating MI was associated with NSTEMI. The GUSTO-II[44] and PURSUIT[45] trials found that cardiogenic shock occurs in up to 5% of patients with NSTEMI and that mortality rates are greater than 60%. Thus, hypotension and evidence of organ hypoperfusion can occur and constitute a medical emergency in NSTEMI.

2.1.1. Comparison of Early Invasive and Initial Conservative Strategies for UA/NSTEMI

Prior meta-analyses concluded that routine invasive therapy (the "invasive" or "early" strategy triages patients to undergo an invasive diagnostic evaluation without first getting a noninvasive stress test or without failing medical treatment [i.e., an initial conservative diagnostic strategy or sometimes now known as the "selective invasive strategy"][14]) is better than an initial conservative or selectively invasive approach (the "initial conservative strategy" [also referred to as "selective invasive management"] calls for proceeding with an invasive evaluation only for those patients who fail medical therapy [refractory angina or angina at rest or with minimal activity despite rigorous medical therapy] or in whom objective evidence of ischemia [dynamic ECG changes, high-risk stress test] is identified[14]). Mehta et al[47] concluded that the routine invasive strategy resulted in an 18% relative reduction in death or MI, including a significant reduction in MI alone. The routine invasive arm was associated with higher in-hospital mortality (1.8% versus 1.1%), but this disadvantage was more than compensated for by a significant reduction in mortality between discharge and the end of follow-up (3.8% versus 4.9%). In those analyses, the invasive strategy was associated with less angina and fewer rehospitalizations than the conservative pathway. Patients undergoing routine invasive treatment also had improved quality of life.

In contrast to these findings, other studies, most recently ICTUS (Invasive versus Conservative Treatment in Unstable

coronary Syndromes), have favorably highlighted a strategy of selective invasive therapy.[48] In ICTUS, 1200 high-risk ACS patients without ST-segment elevation were randomized to receive routine invasive versus selective invasive management and followed up for 1 year with respect to the combined incidence of death, MI, and ischemic rehospitalization. All patients were treated with optimal medical therapy that included aspirin, clopidogrel, LMWH, and lipid-lowering therapy; abciximab was given to those undergoing revascularization. At the end of 1 year, there was no significant difference in the composite end point between groups. This study suggests that a selective invasive strategy could be reasonable for ACS patients. A possible explanation for the lack of benefit of the invasive approach in this trial (and other trials)[49] could be related to the relatively high rate of revascularization actually performed in patients treated in the selective invasive arm (47%), thereby reducing observed differences between treatment strategies,[22] and to the lower event rate (lower-risk population) than in other studies. Results were unchanged during longer-term follow-up.[50,51] Nevertheless, ICTUS required troponin positivity for entry. Thus, troponin alone might no longer be an adequate criterion for strategy selection, especially with increasingly sensitive troponin assays. The degree of troponin elevation and other high-risk clinical factors taken together should be considered in selecting a treatment strategy. The ICTUS trial was relatively underpowered for hard end points, and it used a controversial definition for post procedural MI (i.e., even minimal asymptomatic CK-MB elevation).[48,50,51]

Additionally, 1-year follow-up may be inadequate to fully realize the long-term impact and benefit of the routine invasive strategy. In the RITA-3 trial (Third Randomized Intervention Trial of Angina), 5-year but not 1-year event rates favored the early invasive arm (see Figure 2 and text below).[52] In ICTUS, however, results were maintained during a 3-year follow-up.[53]

Thus, the 2007 UA/NSTEMI Guidelines[14] recommend that in initially stabilized UA/NSTEMI patients, an initial conservative (selective invasive) strategy may be considered as an alternative treatment option. The writing committee also believes that additional comparative trials of the selective invasive with the routine initial invasive strategies are indicated, using aggressive contemporary medical therapies in both arms, including routine dual antiplatelet therapy (DAT) in medically treated patients as well as aggressive lipid lowering and other updated secondary prevention measures.

Nevertheless, a meta-analysis of contemporary randomized trials in NSTEMI, including ICTUS, currently support long-term mortality and morbidity benefits of an early invasive compared with an initial conservative strategy.[54] Nonfatal MI at 2 years (7.6% vs. 9.1%, respectively; RR 0.83 [95% CI 0.72 to 0.96]; p = 0.012) and hospitalization (at 13 months; RR = 0.69 [95% CI 0.65 to 0.74]; p less than 0.0001) also were reduced by an early invasive strategy (Figure 3). A separate review of contemporary randomized trials in the stent era using the Cochrane Database arrived at similar conclusions.[55] Details of selected contemporary trials of invasive versus conservative strategies may be found in the ACC/AHA 2007 UA/NSTEMI Guidelines.[14]

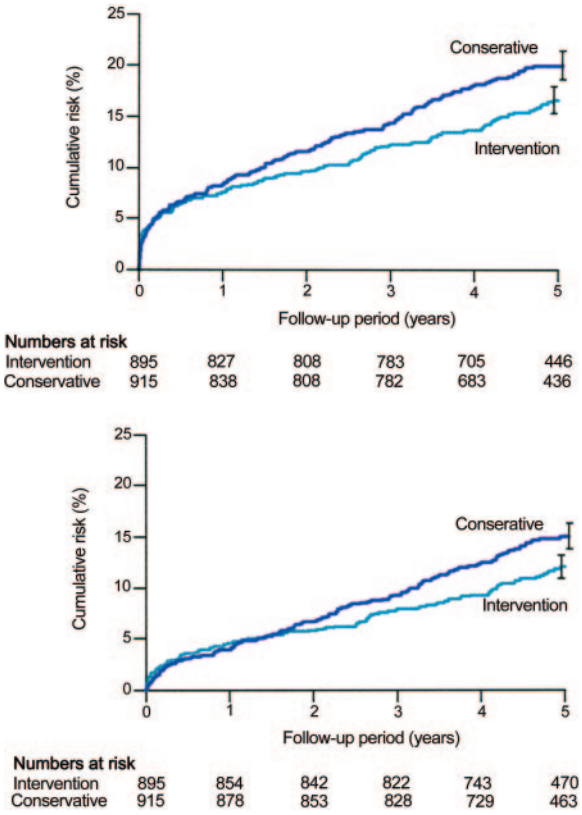

Figure 2. Cumulative Risk of Death or Myocardial Infarction in RITA-3. Top: Cumulative risk of death or myocardial infarction in the RITA-3 trial of patients with non-ST acute coronary syndrome. Bottom: Cumulative risk of death in the RITA-3 trial of patients with non-ST acute coronary syndromes. Reprinted with permission.[52]

Thus, the FRISC-II (Fragmin and Fast Revascularisation during InStability in Coronary artery disease)[56] and TACTICS (Treat Angina with Aggrastat and Determine Cost of Therapy with an Invasive or Conservative Strategy)-TIMI 18[28] trials showed a benefit in patients assigned to invasive strategy. In contrast to earlier trials, a large majority of patients undergoing percutaneous coronary intervention (PCI) in these 2 trials received coronary stenting as opposed to balloon angioplasty alone. Also, there was a differential rate of thienopyridine use between the 2 arms; only stented patients were treated. In FRISC-II, the invasive strategy involved treatment with LMWH, aspirin, nitrates, and beta blockers for an average of 6 days in the hospital before coronary angiography, an approach that would be difficult to adopt in US hospitals. In TACTICS-TIMI 18, treatment included the GP IIb/IIIa antagonist tirofiban, which was administered for an average of 22 hours before coronary angiography. The routine use of the GP IIb/IIIa inhibitor in this trial may have eliminated the excess risk of early (within 7 days) MI in the invasive arm, a risk that was observed in FRISC-II and other trials in which there was no routine "upstream" use of a GP IIb/IIIa blocker. Therefore, an invasive strategy is associated with a better outcome in UA/NSTEMI patients at high risk as defined in Table 3 and as demonstrated in TACTICS-TIMI 18 when a GP IIb/IIIa inhibitor is used.[28] Although the benefit of intravenous GP

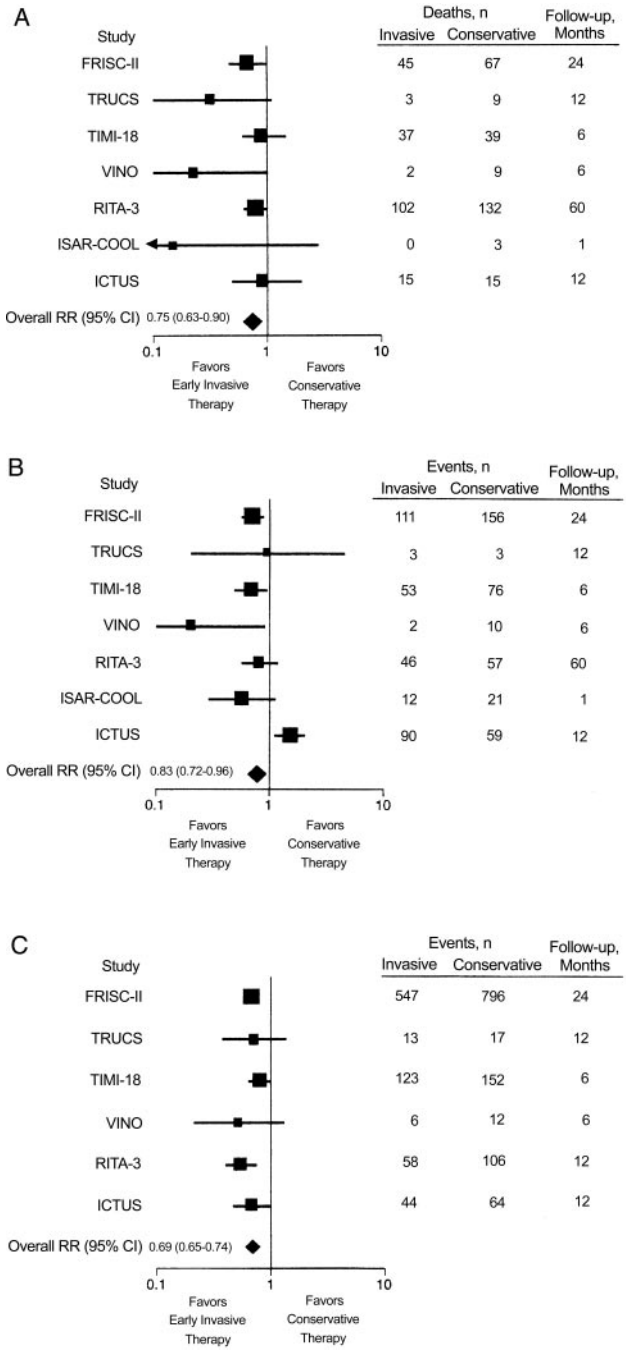

Figure 3. Relative Risk of Outcomes With Early Invasive Versus Conservative Therapy in UA/NSTEMI. A: Relative risk of all-cause mortality for early invasive therapy compared with conservative therapy at a mean follow-up of 2 years. B: Relative risk of recurrent nonfatal myocardial infarction for early invasive therapy compared with conservative therapy at a mean follow-up of 2 years. C: Relative risk of recurrent unstable angina resulting in rehospitalization for early invasive therapy compared with conservative therapy at a mean follow-up of 13 months.[54] CI indicates confidence interval; FRISC-II, FRagmin and fast Revascularization during InStability in Coronary artery disease; ICTUS, Invasive versus Conservative Treatment in Unstable coronary Syndromes; ISAR-COOL, Intracoronary Stenting with Antithrombotic Regimen COOLing-off; RITA-3, Third Randomized Intervention Trial of Angina; RR, relative risk; TIMI-18, Thrombolysis In Myocardial Infarction-18; TRUCS, Treatment of Refractory Unstable angina in geographically isolated areas without

IIb/IIIa inhibitors is established for UA/NSTEMI patients undergoing PCI, the optimal time to start these drugs before the procedure has not been established. In the PURSUIT trial,[45] in patients with UA/NSTEMI who were admitted to community hospitals, the administration of eptifibatide was associated with a reduced need for transfer to tertiary referral centers and improved outcomes.[57]

The RITA-3 trial[52] compared early and conservative therapy in 1810 moderate-risk patients with ACS. Patients with positive cardiac biomarkers (CK-MB greater than 2 times the upper limit of normal at randomization) were excluded from randomization, as were those with new Q waves, MI within 1 month, PCI within 1 year, and any prior coronary artery bypass graft (CABG). The combined end point of death, nonfatal MI, and refractory angina was reduced from 14.5% to 9.6% by early invasive treatment. The benefit was driven primarily by a reduction in refractory angina. There was a late divergence of the curves, with reduced 5-year death and MI in the early invasive arm (Figure 2).

In the VINO trial (Value of first day angiography/ angioplasty In evolving Non-ST segment elevation myocardial infarction: Open multicenter randomized trial),[58] 131 patients with NSTEMI were randomized to cardiac catheterization on the day of admission versus conservative therapy. Despite the fact that 40% of the conservatively treated patients crossed over to revascularization by the 6-month follow-up, there was a significant reduction in death or reinfarction for patients assigned to early angiography and revascularization (6% versus 22%).

The ISAR-COOL (Intracoronary Stenting with Antithrombotic Regimen Cooling-off) trial[59] randomized 410 intermediate- to high-risk patients to very early angiography and revascularization versus a delayed invasive strategy. All patients were treated with intensive medical therapy that included aspirin, heparin, clopidogrel (600-mg loading dose), and the intravenous GP IIb/IIIa receptor inhibitor tirofiban. In the very early arm, patients underwent cardiac catheterization at a mean time of 2.4 hours versus 86 hours in the delayed invasive arm. The very early invasive strategy was associated with significantly better outcome at 30 days, as measured by reduction in death and large MI (5.9% versus 11.6%). More importantly, the benefit seen was attributable to a reduction in events before cardiac catheterization, which raises the possibility that there is a hazard associated with a "cooling-down" period.

2.1.2. Selection for Coronary Angiography

In contrast to the noninvasive tests, coronary angiography provides detailed structural information to allow assessment of prognosis and provide direction for appropriate management. When combined with left ventricular (LV) angiography, it also allows an assessment of global and regional LV function. Indications for coronary angiography are interwoven with indications for possible therapeutic plans, such as PCI or CABG.

Cardiac Surgery; UA/NSTEMI, unstable angina/non-ST-segment elevation myocardial infarction: Open multicenter randomized trial. Modified with permission.[54]

Table 6. Noninvasive Risk Stratification

High risk (greater than 3% annual mortality rate)
Severe resting LV dysfunction (LVEF less than 0.35)
High-risk treadmill score (score –11 or less)
Severe exercise LV dysfunction (exercise LVEF less than 0.35)
Stress-induced large perfusion defect (particularly if anterior)
Stress-induced multiple perfusion defects of moderate size
Large, fixed perfusion defect with LV dilation or increased lung uptake (thallium-201)
Stress-induced moderate perfusion defect with LV dilation or increased lung uptake (thallium-201)
Echocardiographic wall-motion abnormality (involving more than 2 segments) developing with low dose of dobutamine (10 mg/kg per min or less) or at a low heart rate (less than 120 bpm)
Stress echocardiographic evidence of extensive ischemia
Intermediate risk (1% to 3% annual mortality rate)
Mild/moderate resting LV dysfunction (LVEF 0.35 to 0.49)
Intermediate-risk treadmill score (−11 to 5)
Stress-induced moderate perfusion defect without LV dilation or increased lung intake (thallium-201)
Limited stress echocardiographic ischemia with a wall-motion abnormality only at higher doses of dobutamine involving less than or equal to 2 segments
Low risk (less than 1% annual mortality rate)
Low-risk treadmill score (score 5 or greater)
Normal or small myocardial perfusion defect at rest or with stress*
Normal stress echocardiographic wall motion or no change of limited resting wall-motion abnormalities during stress*

*Although published data are limited, patients with these findings will probably not be at low risk in the presence of either a high-risk treadmill score or severe resting LV dysfunction (LVEF less than 0.35). Reprinted from reference 60.

LV indicates left ventricular, and LVEF, left ventricular ejection fraction.

Coronary angiography is usually indicated in patients with UA/NSTEMI who either have recurrent symptoms or ischemia despite adequate medical therapy or are at high risk as categorized by clinical findings (HF, serious ventricular arrhythmias) or noninvasive test findings (significant LV dysfunction: ejection fraction less than 0.35, large anterior or multiple perfusion defects) (Tables 6, 7, and 8). Patients with UA/NSTEMI who have had previous PCI or CABG also should generally be considered for early coronary angiography unless prior coronary angiography data indicate that further revascularization is not likely to be possible. The placement of an intra-aortic balloon pump (IABP) may allow coronary angiography and revascularization in those with hemodynamic instability. Patients with suspected Prinzmetal's variant angina also are candidates for coronary angiography.

In all cases, the general indications for coronary angiography and revascularization are tempered by individual patient characteristics and preferences. Patient and physician judgments regarding risks and benefits are particularly important for patients who might not be candidates for coronary revascularization, such as very frail older adults and those with serious comorbid conditions (i.e., severe hepatic, pulmonary, or renal failure or active or inoperable cancer).

Table 7. Noninvasive Test Results That Predict High Risk for Adverse Outcome (Left Ventricular Imaging)

Stress Radionuclide Ventriculography	Stress Echocardiography
Exercise EF 0.50 or less	Rest EF 0.35 or less
Rest EF 0.35 or less	Wall-motion score index greater than 1
Fall in EF 0.10 or greater	

Modified from references 61 and 62.

EF indicates ejection fraction.

2.1.3. Chronic Kidney Disease

The following recommendations have been added to the PCI Focused Update in accordance with new recommendations appearing in the 2007 UA/NSTEMI Guidelines[14] (Table 9). Supporting text from that guidelines statement is presented in the following paragraphs.

Chronic kidney disease (CKD) is not only a coronary risk equivalent for ascertainment of coronary risk but also a risk factor for the development and progression of cardiovascular disease (CVD).[63] CKD constitutes a risk factor for adverse outcomes after MI,[64] including NSTEMI and other coronary patient subsets. In the highly validated GRACE risk score, serum creatinine is 1 of 8 independent predictors of death.[20,65] In 1 recent study, even early CKD constituted a significant risk factor for cardiovascular events and death.[64,66] CKD also predicts an increase in recurrent cardiovascular events.[67] Cardiovascular

Table 8. Noninvasive Test Results That Predict High Risk for Adverse Outcome on Stress Radionuclide Myocardial Perfusion Imaging

Abnormal myocardial tracer distribution in more than 1 coronary artery region at rest or with stress or a large anterior defect that reperfuses
Abnormal myocardial distribution with increased lung uptake
Cardiac enlargement

Modified from reference 61.

Table 9. Indications for Chronic Kidney Disease

2005 PCI Guideline Update Recommendation	2007 PCI Focused Update Recommendation	Comments
	Class I	
	1. Creatinine clearance should be estimated in UA/NSTEMI patients, and the doses of renally cleared drugs should be adjusted appropriately. *(Level of Evidence: B)*	New recommendation*
	2. In chronic kidney disease patients undergoing angiography, isosmolar contrast agents are indicated and are preferred. *(Level of Evidence: A)*	New recommendation*

*Based on the ACC/AHA 2007 UA/NSTEMI Guidelines.[14]
CKD indicates chronic kidney disease; PCI, percutaneous coronary intervention; and UA/NSTEMI, unstable angina/non–ST-elevation myocardial infarction.

death is 10 to 30 times higher in dialysis patients than in the general population. The underrepresentation of patients with renal disease in randomized controlled trials of CVD is a concern.[68] Current opinion and most of the limited evidence available suggest that when appropriately monitored, cardiovascular medications and interventional strategies can be applied safely in those with renal impairment and provide therapeutic benefit.[64] However, not all recent evidence is consistent with this premise: atorvastatin did not significantly reduce the primary end point of cardiovascular death, nonfatal MI, or stroke in a prospective randomized trial of patients with diabetes and end-stage CKD who were undergoing hemodialysis.[69] The preference for primary PCI has also been questioned.[70]

Particularly in the setting of ACS, bleeding complications are higher in this patient subgroup because of platelet dysfunction and dosing errors; benefits of fibrinolytic therapy, antiplatelet agents, and anticoagulants can be negated or outweighed by bleeding complications; and renin-angiotensin-aldosterone inhibitors can impose a greater risk because of the complications of hyperkalemia and worsening renal function in the patient with CKD. Angiography carries an increased risk of contrast-induced nephropathy; the usual benefits of PCI can be lessened or abolished; and PCI in patients with CKD is associated with a higher rate of early and late complications of bleeding, restenosis, and death.[68] Thus, identification of CKD is important in that it represents an ACS subgroup with a far more adverse prognosis but for whom interventions have less certain benefit.

Coronary arteriography is a frequent component of the care of ACS patients. As such, contrast-induced nephropathy can constitute a serious complication of diagnostic and interventional procedures. In patients with CKD or CKD and diabetes, isosmolar contrast material lessens the rise in creatinine and is associated with lower rates of contrast-induced nephropathy than low-osmolar contrast media. This has been documented in a randomized clinical trial (RECOVER [Renal Toxicity Evaluation and Comparison Between Visipaque (Iodixanol) and Hexabrix (Ioxaglate) in Patients With Renal Insufficiency Undergoing Coronary Angiography]) comparing iodixanol with ioxaglate[71] and in a meta-analysis of 2727 patients from 16 randomized clinical trials.[72]

Identification of patients with CKD as recommended in the AHA Science Advisory on Detection of CKD in patients with or at increased risk of CVD should guide the use of isosmolar contrast agents.[63] The advisory, which was developed in collaboration with the National Kidney Foundation, recommends that all patients with CVD be screened for evidence of kidney disease by estimating glomerular filtration rate, testing for microalbuminuria, and measuring the albumin-to-creatinine ratio. A glomerular filtration rate of less than 60 ml per min per 1.73 square meters of body surface should be regarded as abnormal. Furthermore, the albumin-to-creatinine ratio should be used to screen for the presence of kidney damage in adult patients with CVD, with values greater than 30 mg of albumin per 1 g of creatinine considered abnormal.

A diagnosis of renal dysfunction is critical to proper medical therapy for UA/NSTEMI. Many cardiovascular drugs used in patients with UA/NSTEMI are renally cleared; their doses should be adjusted for estimated creatinine clearance [see also Section 3 of the 2007 UA/NSTEMI Guidelines[14]]. In a large community-based registry study, 42% of patients with UA/NSTEMI received excessive initial dosing of at least 1 antiplatelet or antithrombin agent (unfractionated heparin [UFH], LMWH, or GP IIb/IIIa inhibitor).[73] Renal insufficiency was an independent predictor of excessive dosing. Dosing errors predicted an increased risk of major bleeding. Clinical studies and labeling that defines adjustments for several of these drugs have been based on the Cockcroft-Gault formula for estimating creatinine clearance, which is not identical to the Modification of Diet and Renal Disease (MDRD) formula. Use of the Cockcroft-Gault formula to generate dose adjustments is recommended. The impact of renal dysfunction on biomarkers of necrosis (i.e., troponin) is discussed in Section 2.2.8.2.1 of the 2007 UA/NSTEMI Guidelines.[14]

To increase the meager evidence base and to optimize care for this growing high-risk population, the recognition of CKD patients with or at risk of CVD and the inclusion and reporting of renal disease in large CVD trials must be increased in the future.

3. Facilitated PCI

Facilitated PCI refers to a strategy of planned immediate PCI after administration of an initial pharmacological regimen intended to improve coronary patency before the procedure. These regimens have included high-dose heparin, platelet GP IIb/IIIa inhibitors, full-dose or reduced-dose fibrinolytic therapy, and the combination of a GP IIb/IIIa inhibitor with a reduced-dose fibrinolytic agent (e.g., fibrinolytic dose typically reduced 50%). Facilitated PCI should be differentiated from primary PCI without fibrinolytic therapy, from primary PCI with a GP IIb/IIIa inhibitor started at the time of PCI,

from early or delayed PCI after successful fibrinolytic therapy, and from rescue PCI after unsuccessful fibrinolytic therapy. Potential advantages of facilitated PCI include earlier time to reperfusion, smaller infarct size, improved patient stability, lower infarct artery thrombus burden, greater procedural success rates, higher TIMI flow rates, and improved survival rates. Potential risks include increased bleeding complications, especially in older patients; potential limitations include added cost.

Despite the potential advantages, clinical trials of facilitated PCI have not demonstrated any benefit in reducing infarct size or improving outcomes. The largest of these was the ASSENT-4 (Assessment of the Safety and Efficacy of a New Treatment Strategy with Percutaneous Coronary Intervention) PCI trial,[5] in which 1667 patients were randomized to full-dose tenecteplase and PCI versus primary PCI. The trial was terminated prematurely because of a higher in-hospital mortality rate in the facilitated PCI group (6% vs. 3%, $p = 0.01$). The primary end point, a composite of death, shock, and congestive heart failure within 90 days, was significantly higher with facilitated PCI than with primary PCI (18.6% vs. 13.4%; $p = 0.0045$), and there was a trend toward higher 90-day mortality (6.7% vs. 4.9%; $p = 0.14$). Defenders of the facilitated PCI strategy point out that the absence of an infusion of heparin after bolus administration and of a loading dose of clopidogrel, plus prohibition of GP IIb/IIIa inhibitors except in bail-out situations, made adjunctive antithrombotic therapy suboptimal for the facilitated PCI group. Moreover, the median treatment delay between tenecteplase and PCI was only 104 minutes, and mortality rates with facilitated PCI were higher in PCI centers. Whether earlier (pre-hospital) administration of fibrinolytic therapy, better antithrombotic therapy, longer delays to PCI, or selective use of PCI as a rescue strategy would make the facilitated PCI strategy beneficial is unclear and requires further study. On the basis of these data, however, facilitated PCI offered no clinical benefit.

Keeley and coworkers performed a quantitative review of 17 trials that compared facilitated PCI and primary PCI[74] (Figure 4). Included were 9 trials with GP IIb/IIIa inhibitors alone ($n = 1148$), 6 trials with fibrinolytic therapy (including ASSENT-4 PCI) ($n = 2953$), and 2 trials with a fibrinolytic agent plus a GP IIb/IIIa inhibitor ($n = 399$). Facilitated PCI with fibrinolytic therapy had significantly higher rates of mortality, nonfatal reinfarction, urgent target vessel revascularization, total and hemorrhagic stroke, and major bleeding compared with primary PCI. There were no differences in efficacy or safety when facilitated PCI with a GP IIb/IIIa inhibitor was compared with primary PCI.

A planned reperfusion strategy using full-dose fibrinolytic therapy followed by immediate PCI may be harmful (Table 10). Nevertheless, selective use of the facilitated strategy with regimens other than full-dose fibrinolytic therapy in high-risk subgroups of patients (large MI or hemodynamic or electrical instability) with low bleeding risk who present to hospitals without PCI capability might be performed when transfer delays for primary PCI are anticipated. Although the quantitative analysis showed no advantage for pretreatment with a GP IIb/IIIa inhibitor, neither did it document any major disadvantage. The use of GP IIb/IIIa inhibitors, particularly abciximab, during primary PCI is well established. Further trials of reduced-dose fibrinolytic therapy, with or without GP IIb/IIIa inhibitors, are in progress and may yield different efficacy and/or safety results. For further clarification, please see Section 6.3.1.6.2.1 of the 2004 ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction.[75]

Pharmacological reperfusion with full-dose fibrinolysis is not uniformly successful in restoring antegrade flow in the infarct artery. In such situations, a strategy of prompt coronary angiography with intent to perform PCI is frequently contemplated. In certain patients, such as those with cardiogenic shock (especially in those less than 75 years of age), severe congestive heart failure/pulmonary edema, or hemodynamically compromising ventricular arrhythmias (regardless of age), a strategy of coronary angiography with intent to perform PCI is a useful approach regardless of the time since initiation of fibrinolytic therapy, provided further invasive management is not considered futile or unsuitable given the clinical circumstances (Table 11). Further discussion of the

Table 10. Updates to Section 5.4.3: PCI for STEMI in Conjunction With Concomitant Fibrinolytic Therapy

2005 PCI Guideline Update Recommendation	2007 PCI Focused Update Recommendation	Comments
	Class IIb	
Facilitated PCI might be performed as a reperfusion strategy in higher-risk patients when PCI is not immediately available and bleeding risk is low. *(Level of Evidence: B)*	1. Facilitated PCI using regimens other than full-dose fibrinolytic therapy might be considered as a reperfusion strategy when all of the following are present: a. Patients are at high risk, b. PCI is not immediately available within 90 minutes, and c. Bleeding risk is low (younger age, absence of poorly controlled hypertension, normal body weight). *(Level of Evidence: C)*	Modified recommendation (changed LOE and text)
	Class III	
	1. A planned reperfusion strategy using full-dose fibrinolytic therapy followed by immediate PCI may be harmful. *(Level of Evidence: B)*	New recommendation

LOE indicates level of evidence; PCI, percutaneous coronary intervention; and STEMI, ST-elevation myocardial infarction.

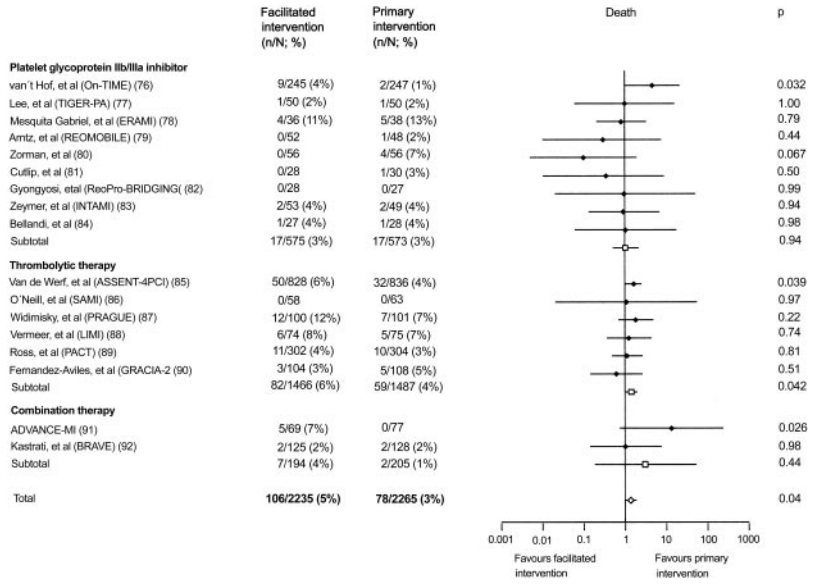

Figure 4. Short-Term Death in Patients Treated With Facilitated Or Primary PCI. Trials were classified by facilitated regimen. Diamonds and squares indicate odds ratios. Lines indicate 95% confidence intervals. Reprinted with permission.[74]

management of such patients may be found in Section 5.4.4 (which has been updated in this document) of the 2005 PCI Guideline Update.[13a]

4. Rescue PCI

In other patients who do not exhibit the clinical instability noted above, PCI may also be reasonable if there is clinical suspicion of failure of fibrinolysis. This is referred to as rescue PCI. Critical to the success of rescue PCI is the initial clinical identification of patients who are suspected of having failed reperfusion with full-dose fibrinolysis. Because the presence or absence of ischemic discomfort may be unreliable for identifying failed reperfusion, clinicians should search for evidence of inadequate ST-segment resolution on the 12-lead ECG. Operationally, the 12-lead ECG should be scrutinized after adequate time has elapsed before making the judgment that fibrinolytic therapy has not been effective. Although earlier periods have been used in some studies, the writing committee felt that 90 minutes after initiation of fibrinolysis provided the best time for evaluating the need for rescue PCI: hence, if there is less than 50% ST resolution in the lead showing the greatest degree of ST-segment elevation at presentation, fibrinolytic therapy has likely failed to produce reperfusion.

The 2005 PCI Guideline Update[13a] recommendations for rescue PCI were based on observational data and 2 small randomized clinical trials (n = 179) from the early 1990s.[94,95] More recently, MERLIN (Middlesbrough Early Revascularization to Limit Infarction) (n = 307) and REACT (Rescue Angioplasty versus Conservative Treatment or Repeat Thrombolysis) (n = 427) and 3 meta-analyses have refocused attention on rescue PCI.[96–100] This subject has been studied with fewer than 1000 patients enrolled in randomized trials.

In the period between trials studying rescue PCI, there was a transition between angiographic and electrocardiographic diagnosis to detect failed reperfusion. Importantly, in the earlier studies, rescue PCI was performed in infarct arteries with TIMI 0/1 flow, often after a protocol-mandated 90-minute angiogram. In MERLIN and REACT, however, patients were randomized if they had less than 50% ST-segment elevation resolution at 60 or 90 minutes, respectively. Many patients had patent infarct arteries at angiography; only 54% of patients in MERLIN and 74% of patients in REACT (which required less than TIMI grade 3 flow for PCI) actually underwent PCI. From a procedural standpoint, stents have replaced balloon angioplasty, antiplatelet therapy has improved with the addition of a thienopyridine agent and often a GP IIb/IIIa receptor antagonist, and procedural success rates are higher.

Despite these historical differences, recent data support the initial observation that rescue PCI decreases adverse clinical events compared with medical therapy. In the Wijeysundera meta-analysis[100]) (Figure 5, there was a trend toward reduced mortality rates with rescue PCI from 10.4% to 7.3% (RR 0.69 [95% CI 0.46 to 1.05]; p = 0.09), reduced reinfarction rates from 10.7% to 6.1% (RR 0.58 [95% CI 0.35 to 0.97]; p = 0.04), and reduced HF rates from 17.8% to 12.7% (RR 0.73 [95% CI 0.54 to 1.00]; p = 0.05). These event rates suggest that high-risk patients were selected for enrollment, so these data do not define the role of rescue PCI in lower-risk patients. Also, the benefits of rescue PCI need to be balanced against the risk. There was an excess occurrence of stroke in 2 trials (10 events versus 2 events), but the majority were thromboembolic rather than hemorrhagic, and the sample size was small, so more data are required to define this risk. There was also an increase of 13% in absolute risk of bleeding, suggesting that adjustments in antithrombotic medication dosing are needed to improve safety. It should be noted that the majority of patients who underwent rescue PCI received streptokinase as fibrinolytic therapy.

Given the association between bleeding events and subsequent ischemic events,[103] it might be reasonable to select moderate- and high-risk patients for PCI after fibrinolysis and to treat low-risk patients with medical therapy. As noted above, patients with cardiogenic shock,

Table 11. Updates to Section 5.4.4: PCI After Failed Fibrinolysis (All 2005 Recommendations Provided for Clarity)

2005 PCI Guideline Update Recommendation	2007 PCI Focused Update Recommendation	Comments
	Class I	
Rescue PCI should be performed in patients less than 75 years old with ST elevation or left bundle-branch block who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care. *(Level of Evidence: B)*	1. A strategy of coronary angiography with intent to perform PCI (or emergency CABG) is recommended for patients who have received fibrinolytic therapy and have any of the following: a. Cardiogenic shock in patients less than 75 years who are suitable candidates for revascularization. *(Level of Evidence: B)* b. Severe congestive heart failure and/or pulmonary edema (Killip class III). *(Level of Evidence: B)* c. Hemodynamically compromising ventricular arrhythmias. *(Level of Evidence: C)*	Modified recommendation (changed LOE and text)
Rescue PCI should be performed in patients with severe congestive heart failure and/or pulmonary edema (Killip class 3) and onset of symptoms within 12 hours. *(Level of Evidence: B)*		Deleted recommendation
	Class IIa	
Rescue PCI is reasonable for selected patients 75 years or older with ST elevation or left bundle-branch block or who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock. Patients with good prior functional status who are suitable for revascularization and agree to invasive care may be selected for such an invasive strategy. *(Level of Evidence: B)*	1. A strategy of coronary angiography with intent to perform PCI (or emergency CABG) is reasonable in patients 75 years of age or older who have received fibrinolytic therapy and are in cardiogenic shock, provided that they are suitable candidates for revascularization. *(Level of Evidence: B)*	Modified recommendation (changed text)
It is reasonable to perform rescue PCI for patients with 1 or more of the following: a. Hemodynamic or electrical instability. *(Level of Evidence: C)* b. Evidence of persistent ischemia. *(Level of Evidence: C)*	2. It is reasonable to perform rescue PCI for patients with 1 or more of the following: a. Hemodynamic or electrical instability. *(Level of Evidence: C)* b. Persistent ischemic symptoms. *(Level of Evidence: C)*	2005 recommendation remains current in 2007 PCI Update
	3. A strategy of coronary angiography with intent to perform rescue PCI is reasonable for patients in whom fibrinolytic therapy has failed (ST-segment elevation less than 50% resolved after 90 minutes following initiation of fibrinolytic therapy in the lead showing the worst initial elevation) and a moderate or large area of myocardium at risk (anterior MI, inferior MI with right ventricular involvement or precordial ST-segment depression). *(Level of Evidence: B)*	New recommendation
	Class IIb	
Rescue PCI in the absence of 1 or more of the above Class I or IIa indications is not recommended. *(Level of Evidence: C)*	1. A strategy of coronary angiography with intent to perform PCI in the absence of 1 or more of the above Class I or IIa indications might be reasonable in moderate- and high-risk patients, but its benefits and risks are not well established. The benefits of rescue PCI are greater the earlier it is initiated after the onset of ischemic discomfort. *(Level of Evidence: C)*	Modified recommendation (changed COR from III to IIb and changed text)
	Class III	
	1. A strategy of coronary angiography with intent to perform PCI (or emergency CABG) is not recommended in patients who have received fibrinolytic therapy if further invasive management is contraindicated or the patient or designee does not wish further invasive care. *(Level of Evidence: C)*	New recommendation

CABG indicates coronary artery bypass graft; COR, class of recommendation; HF, heart failure; LOE, level of evidence; MI, myocardial infarction; PCI, percutaneous coronary intervention; and STEMI, ST-elevation myocardial infarction.

severe HF, or hemodynamically compromising ventricular arrhythmias are excellent candidates. An electrocardiographic estimate of potential infarct size in patients with persistent ST-segment elevation (less than 50% resolution at 90 minutes after initiation of fibrinolytic therapy in the lead showing the worst initial elevation) and ongoing ischemic pain is useful in selecting other patients for rescue PCI. Anterior MI or inferior MI with right ventricular involvement or precordial ST-segment depression usually predicts increased risk.[104] Conversely, patients

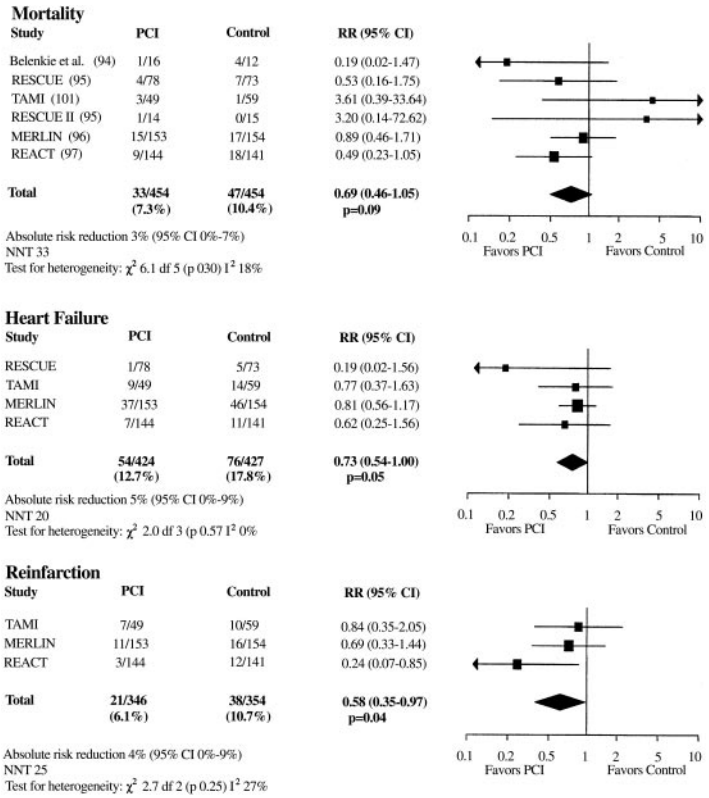

Figure 5. Efficacy End Points for Rescue PCI Versus Conservative Therapy. CI indicates confidence interval; MERLIN, Middlesbrough Early Revascularization to Limit Infarction trial; NNT, number needed to treat; PCI, percutaneous coronary intervention; REACT, Rescue Angioplasty versus Conservative Treatment or Repeat Thrombolysis trial; RESCUE, Randomized Comparison of Rescue Angioplasty with Conservative Management of Patients with Early Failure of Thrombolysis for Acute Anterior Myocardial Infarction trial; RR, relative risk; and TAMI, Thrombolysis and Angioplasty in Myocardial Infarction study. Reprinted with permission.[100]

with symptom resolution, improving ST-segment elevation (less than 50% resolution), or inferior MI localized to 3 ECG leads probably should not be referred for angiography. Likewise, it is doubtful that PCI of a branch artery (diagonal or obtuse marginal branch) will change prognosis in the absence of the high-risk criteria noted above.

5. PCI After Fibrinolysis or for Patients Not Undergoing Primary Reperfusion

The open artery hypothesis suggests that late patency of an infarct artery is associated with improved LV function, increased electrical stability, and provision of collateral vessels to other coronary beds for protection against future events (see Table 12). The OAT (Occluded Artery Trial)[12] tested the hypothesis that routine PCI for total occlusion 3 to 28 days after MI would reduce the composite of death, reinfarction, or Class IV heart failure. Stable patients (n = 2166) with an occluded infarct artery after MI (about 20% of whom received fibrinolytic therapy for the index event) were randomized to optimal medical therapy and PCI with stenting or optimal medical therapy alone. The qualifying period of 3 to 28 days was based on calendar days; thus, the minimal time from symptom onset to angiography was just over 24 hours. Inclusion criteria included total occlusion of the infarct-related artery with TIMI grade 0 or 1 antegrade flow and LV ejection fraction (LVEF) less than 50% or proximal occlusion of a major epicardial artery with a large risk region. Exclusion criteria included NYHA Class III or IV heart failure, serum creatinine greater than 2.5 mg per dL, left main or 3-vessel disease, clinical instability, or severe inducible ischemia on stress testing if the infarct zone was not akinetic or dyskinetic. The 4-year cumulative end point was 17.2% in the PCI group and 15.6% in the medical therapy group (HR 1.16 [95% CI 0.92 to 1.45] p = 0.2). Reinfarction rates tended to be higher in the PCI group, which may have attenuated any benefit in LV remodeling. There was no interaction between treatment effect and any subgroup variable.

Preclinical studies have suggested that late opening of an occluded infarct artery may reduce adverse LV remodeling and preserve LV volumes. However, 5 previous clinical studies in 363 patients have demonstrated inconsistent improvement in LVEF or LV end-systolic and end-diastolic volumes after PCI. The largest of these, the DECOPI (DEsobstruction COronaire en Post-Infarctus) trial, found a higher LVEF at 6 months with PCI.[105] TOSCA-2 (Total Occlusion Study of Canada)[13] enrolled 381 stable patients in a mechanistic ancillary study of OAT and had the same eligibility criteria.[12] The PCI procedure success rate was 92% and the complication rate was 3%, although 9% had periprocedural MI as measured by biomarkers. At 1 year, patency rates (n = 332) were higher with PCI (83% vs. 25%; p less than 0.0001), but each group (n = 286) had equivalent improvement in LVEF (4.2% vs. 3.5%; p = 0.47). There was modest benefit of PCI on preventing LV dilation over 1 year in a multivariate model, but only 42% had paired volume determinations, so it is unclear whether this finding extends to the whole cohort. The potential benefit of PCI in attenuating remodeling may have been decreased by periprocedural MI and the high rate of use of beta blockers and ACE inhibitors. There was no significant interaction between treatment effect and time, infarct artery, or infarct size.

Table 12. Updates to Section 5.4.5: PCI After Successful Fibrinolysis or for Patients Not Undergoing Primary Reperfusion

2005 PCI Guideline Update Recommendation	2007 PCI Focused Update Recommendation	Comments
	Class I	
In patients whose anatomy is suitable, PCI should be performed when there is objective evidence of recurrent MI. *(Level of Evidence: C)*	1. In patients whose anatomy is suitable, PCI should be performed when there is objective evidence of recurrent MI. *(Level of Evidence: C)*	2005 recommendation remains current in 2007 PCI Update
In patients whose anatomy is suitable, PCI should be performed for moderate or severe spontaneous or provocable myocardial ischemia during recovery from STEMI. *(Level of Evidence: B)*	2. In patients whose anatomy is suitable, PCI should be performed for moderate or severe spontaneous or provocable myocardial ischemia during recovery from STEMI. *(Level of Evidence: B)*	2005 recommendation remains current in 2007 PCI Update
In patients whose anatomy is suitable, PCI should be performed for cardiogenic shock or hemodynamic instability. *(Level of Evidence: B)*	3. In patients whose anatomy is suitable, PCI should be performed for cardiogenic shock or hemodynamic instability. *(Level of Evidence: B)*	2005 recommendation remains current in 2007 PCI Update
	Class IIa	
It is reasonable to perform routine PCI in patients with LV ejection fraction less than or equal to 0.40, HF, or serious ventricular arrhythmias. *(Level of Evidence: C)*	1. It is reasonable to perform routine PCI in patients with LV ejection fraction less than or equal to 0.40, HF, or serious ventricular arrhythmias. *(Level of Evidence: C)*	2005 recommendation remains current in 2007 PCI Update
It is reasonable to perform PCI when there is documented clinical heart failure during the acute episode, even though subsequent evaluation shows preserved LV function (LV ejection fraction greater than 0.40). *(Level of Evidence: C)*	2. It is reasonable to perform PCI when there is documented clinical heart failure during the acute episode, even though subsequent evaluation shows preserved LV function (LV ejection fraction greater than 0.40). *(Level of Evidence: C)*	2005 recommendation remains current in 2007 PCI Update
	Class IIb	
PCI might be considered as part of an invasive strategy after fibrinolytic therapy. *(Level of Evidence: C)*	1. PCI of a hemodynamically significant stenosis in a patent infarct artery greater than 24 hours after STEMI may be considered as part of an invasive strategy. *(Level of Evidence: B)*	Modified recommendation (changed COR/LOE and text)
	Class III	
	1. PCI of a totally occluded infarct artery greater than 24 hours after STEMI is not recommended in asymptomatic patients with 1- or 2-vessel disease if they are hemodynamically and electrically stable and do not have evidence of severe ischemia. *(Level of Evidence: B)*	New recommendation

COR/LOE indicates class of recommendation/level of evidence; HF, heart failure; LV, left ventricular; PCI, percutaneous coronary intervention; and STEMI, ST-elevation myocardial infarction.

6. Ancillary Therapy for Patients Undergoing PCI for STEMI

The 2007 STEMI Guidelines Focused Update[106] includes a new section on the use of anticoagulant therapy for patients undergoing PCI to establish reperfusion for STEMI. The recommendations associated with PCI are summarized in Table 13.

Full discussion of the background and basis of these recommendations may be found in the 2007 STEMI Guidelines Focused Update. When moving to PCI after fibrinolytic therapy, those patients who received upstream UFH or enoxaparin can continue to receive those anticoagulants in a seamless fashion (i.e., without crossover to another agent) under the dosing regimens listed in the recommendations.[106,107] On the basis of reports of catheter thrombosis with fondaparinux alone during primary PCI in OASIS-6 (Organization for Assessment of Strategies for Ischemic Syndromes)[7] and the experience with fondaparinux in the OASIS-5 trial,[108] the STEMI focused update writing group recommended that fondaparinux should not be used as the sole anticoagulant during PCI but should be coupled with an additional agent that has anti-IIa activity to ameliorate the risk of catheter complications. Although bivalirudin or UFH are potential options for supplemental anticoagulation with fondaparinux, the available experience, albeit limited, is largely with UFH. The only available data from the CREATE (Clinical Trial of Reviparin and Metabolic Modulation in Acute Myocardial Infarction Treatment) trial that bear on this point are with UFH.[109]

Given the complexities of the characteristics of the individual agents and their actions on the coagulation cascade, clinicians are cautioned about extrapolating any of the observations with agents discussed in this update to other anticoagulant regimens. In particular, as noted by the Food and Drug Administration (FDA), the LMWHs are sufficiently distinct that they should be evaluated individually rather than considered as a class of interchangeable agents.[110]

7. Antiplatelet Therapy

The 2005 PCI Guideline Update[13a] recommended aspirin antiplatelet therapy of 325 mg, which was based primarily on results from the TAXUS IV and SIRIUS trials.[111–128] Since that time, experience has been gained with doses of aspirin ranging from 75 mg to 325 mg (see Table 14 for further information and Table 15 for a list of the trials). No significant trials have been reported comparing lower-dose

Table 13. Ancillary Therapy

2005 PCI Guideline Update Recommendation	2007 PCI Focused Update Recommendation	Comments
	Class I	
	1. For patients undergoing PCI after having received an anticoagulant regimen, the following dosing recommendations should be followed:	
	a. For prior treatment with UFH, administer additional boluses of UFH as needed to support the procedure, taking into account whether GP IIb/IIIa receptor antagonists have been administered. *(Level of Evidence: C)* Bivalirudin may also be used in patients treated previously with UFH. *(Level of Evidence: C)*	New recommendation*
	b. For prior treatment with enoxaparin, if the last subcutaneous dose was administered at least 8 to 12 hours earlier, an IV dose of 0.3 mg/kg of enoxaparin should be given; if the last subcutaneous dose was administered within the prior 8 hours, no additional enoxaparin should be given. (*Level of Evidence: B*)	New recommendation*
	c. For prior treatment with fondaparinux, administer additional intravenous treatment with an anticoagulant possessing anti-IIa activity, taking into account whether GP IIb/IIIa receptor antagonists have been administered. (*Level of Evidence: C*)	New recommendation*
	Class III	
	1. Because of the risk of catheter thrombosis, fondaparinux should not be used as the sole anticoagulant to support PCI. An additional anticoagulant with anti-IIa activity should be administered. (*Level of Evidence: C*)	New recommendation*

*Based on 2007 STEMI Focused Update.[106]

GP indicates glycoprotein; IV, intravenous; PCI, percutaneous coronary intervention; STEMI, ST-elevation myocardial infarction; and UFH, unfractionated heparin.

aspirin (75 mg to 100 mg) with higher-dose aspirin (162 mg to 325 mg) in subacute or late stent thrombosis with the incidence of bleeding as the initial course of therapy after placement of drug-eluting stents (DES). Two major trials[129,130] involving patients not undergoing placement of DES report an increase in risk of bleeding on higher-dose aspirin. No conclusive data are available regarding higher-dose aspirin and subacute stent thrombosis among patients who are considered aspirin resistant.

Continued treatment with the combination of aspirin and clopidogrel after PCI appears to reduce rates of cardiovascular ischemic events.[130,131] On the basis of randomized clinical trial protocols, aspirin 162 mg to 325 mg daily should be given for at least 1 month after implantation of a bare-metal stent (BMS), 3 months after implantation of a sirolimus-eluting stent (SES), and 6 months after implantation of a paclitaxel-eluting stent (PES), after which daily long-term use of aspirin should be continued indefinitely at a dose of 75 mg to 162 mg. In patients for whom there is concern about bleeding, the opinion of the writing group is that lower doses of aspirin—75 mg to 162 mg—can be used.

Likewise, clopidogrel 75 mg daily should be given for a minimum of 1 month after implantation of a BMS [minimum 2 weeks for patients at significant increased risk of bleeding[132]] and for 12 months after implantation of a SES or PES and ideally in all patients post PCI who are not at high risk of bleeding. Under urgent circumstances that prevent the use of clopidogrel for 1 year, the duration studied for FDA approvals was 3 months for an SES and 6 months for a PES. The optimal duration of clopidogrel therapy after 1 year has not been established and should depend on the judgment of the risk–benefit ratio for the individual patient. Predictors of late stent thrombosis have included stenting of small vessels, multiple lesions, long stents, overlapping stents, ostial or bifurcation lesions, prior brachytherapy, suboptimal stent result, low ejection fraction, advanced age, diabetes mellitus, renal failure, ACS, and premature discontinuation of antiplatelet agents.[133,134] Patients should be counseled on the need for and risks of DAT before placement of intracoronary stents, especially a DES, and alternative therapies to pursue if they are unwilling or unable to comply with the recommended duration of DAT. To reduce the incidence of bleeding complications associated with DAT, lower-dose aspirin (75 mg to 162 mg daily) is reasonable for long-term therapy.[135,136] Given the importance of a 1-year course of DAT, it is recommended that elective surgery be postponed for 1 year, and among those patients for whom surgery cannot be deferred, aspirin therapy should be considered during the perioperative period in high-risk patients with DES.[133]

Several investigations have explored various loading doses of clopidogrel before or during PCI. Consistent findings are that compared with a 300-mg loading dose, doses of either 600 or 900 mg achieve greater degrees of platelet inhibition with less variability among patients.[137] Fewer patients may demonstrate "resistance" or nonresponsiveness to clopidogrel following the 600-mg dose. There appears to be no significant additive value of the 900-mg dose over the 600-mg dose.[137]

The 600-mg dose appears to achieve maximum inhibition more rapidly than the 300-mg dose.[138] Superior clinical outcomes at 30 days, primarily reduction in evidence of MI, have been reported after the 600-mg dose given 2 hours before the procedure, although this salutary

Table 14. Updates to Section 6.2.1: Oral Antiplatelet Therapy

2005 PCI Guideline Update Recommendation	2007 PCI Focused Update Recommendation	Comments
	Class I	
Patients already taking daily chronic aspirin therapy should take 75 to 325 mg of aspirin before the PCI procedure is performed. *(Level of Evidence: A)*	1. Patients already taking daily long-term aspirin therapy should take 75 mg to 325 mg of aspirin before PCI is performed. *(Level of Evidence: A)*	2005 recommendation remains current in 2007 PCI Update
Patients not already taking daily chronic aspirin therapy should be given 300 to 325 mg of aspirin at least 2 hours and preferably 24 hours before the PCI procedure is performed. *(Level of Evidence: C)*	2. Patients not already taking daily long-term aspirin therapy should be given 300 mg to 325 mg of aspirin at least 2 hours and preferably 24 hours before PCI is performed. *(Level of Evidence: C)*	2005 recommendation remains current in 2007 PCI Update
After the PCI procedure, in patients with neither aspirin resistance, allergy, nor increased risk of bleeding, aspirin 325 mg daily should be given for at least 1 month after bare-metal stent implantation, 3 months after sirolimus-eluting stent implantation, and 6 months after paclitaxel-eluting stent implantation, after which daily chronic aspirin use should be continued indefinitely at a dose of 75 to 162 mg. *(Level of Evidence: B)*	3. After PCI, in patients without allergy or increased risk of bleeding, aspirin 162 mg to 325 mg daily should be given for at least 1 month after BMS implantation, 3 months after sirolimus-eluting stent implantation, and 6 months after paclitaxel-eluting stent implantation, after which daily long-term aspirin use should be continued indefinitely at a dose of 75 mg to 162 mg. *(Level of Evidence: B)*	Modified recommendation (changed text)
A loading dose of clopidogrel should be administered before PCI is performed. *(Level of Evidence: A)* An oral loading dose of 300 mg, administered at least 6 hours before the procedure, has the best established evidence of efficacy. *(Level of Evidence: B)*	4. A loading dose of clopidogrel,* generally 600 mg, should be administered before or when PCI is performed. *(Level of Evidence: C)* In patients undergoing PCI within 12 to 24 hours of receiving fibrinolytic therapy, a clopidogrel oral loading dose of 300 mg may be considered. *(Level of Evidence: C)*	Modified recommendation (changed LOE and text)
In patients who have undergone PCI, clopidogrel 75 mg daily should be given for at least 1 month after bare-metal stent implantation (unless the patient is at increased risk of bleeding; then it should be given for a minimum of 2 weeks), 3 months after sirolimus stent implantation, and 6 months after paclitaxel stent implantation, and **ideally up to 12 months** in patients who are not at high risk of bleeding. *(Level of Evidence: B)*	5. For all post-PCI stented patients receiving a DES, clopidogrel 75 mg daily should be given for **at least 12 months** if patients are not at high risk of bleeding. For post-PCI patients receiving a BMS, clopidogrel should be given for a minimum of 1 month and **ideally up to 12 months** (unless the patient is at increased risk of bleeding; then it should be given for a minimum of 2 weeks). *(Level of Evidence: B)*	Modified recommendation (changed text)
	Class IIa	
If clopidogrel is given at the time of procedure, supplementation with GP IIb/IIIa receptor antagonists can be beneficial to facilitate earlier platelet inhibition than with clopidogrel alone. *(Level of Evidence: B)*	1. If clopidogrel is given at the time of procedure, supplementation with GP IIb/IIIa receptor antagonists can be beneficial. *(Level of Evidence: B)*	Modified recommendation (changed text)
For patients with an absolute contraindication to aspirin, it is reasonable to give a 300-mg loading dose of clopidogrel, administered at least 6 hours before PCI, and/or GP IIb/IIIa antagonists, administered at the time of PCI. *(Level of Evidence: C)*	2. For patients with an absolute contraindication to aspirin, it is reasonable to give a 300-mg to 600-mg loading dose of clopidogrel, administered at least 6 hours before PCI, and/or GP IIb/IIIa antagonists, administered at the time of PCI. *(Level of Evidence: C)*	2005 recommendation remains current in 2007 PCI Update
	3. In patients for whom the physician is concerned about risk of bleeding, a lower dose of 75 mg to 162 mg of aspirin is reasonable during the initial period after stent implantation. *(Level of Evidence: C)*	New recommendation
	Class IIb	
	1. Continuation of clopidogrel therapy beyond 1 year may be considered in patients undergoing DES placement. *(Level of Evidence: C)*	New recommendation

*Some uncertainty exists about optimal loading dose of clopidogrel. Randomized trials establishing its efficacy and providing data on bleeding risks used a loading dose of 300 mg orally followed by a daily oral dose of 75 mg. Higher oral loading doses such as 600 mg or 900 mg of clopidogrel more rapidly inhibit platelet aggregation and achieve a higher absolute level of inhibition of platelet aggregation, but the additive clinical efficacy and safety of higher oral loading doses have not been rigorously established.

BMS indicates bare-metal stent; DES, drug-eluting stent; GP, glycoprotein; IV, intravenous; LOE, level of evidence; PCI, percutaneous coronary intervention; and STEMI, ST-elevation myocardial infarction.

Table 15. Aspirin Dosages of Major Clinical Trials Involving PCI

Trial Name	Stents Compared	Total Patients	Duration of Treatment	Aspirin Dose
RAVEL[111]	SES versus BMS	238	Indefinite	100 mg once a day
E-SIRIUS[112]	SES versus BMS	352	Indefinite	100 mg once a day
TAXUS I[113]	PES versus BMS	61	Greater than or equal to 12 months	Greater than 80 mg once a day
TAXUS II[114]	PES versus BMS	536	Indefinite	75 mg once a day
TAXUS III[115]	PES for ISR only	28	Not stated	Greater than or equal to 75 mg
C-SIRIUS[116]	SES versus BMS	100	Indefinite	81 to 325 mg once a day
DELIVER[117]	ACHIEVE versus ML PENTA	1043	1 year	325 mg once a day
ELUTES[118]	PES versus BMS	190	3 months	Not stated
SIRIUS[119]	SES versus BMS	1058	Indefinite	325 mg once a day
TAXUS IV[120]	PES versus BMS EXPRESS	1314	Indefinite	325 mg once a day
ISAR-DESIRE[121]	SES versus PES versus balloon angioplasty	300	Indefinite	500 mg IV during; 100 mg bid after
ISAR-DIABETES[122]	SES versus PES	250	Indefinite	100 mg twice a day
SIRTAX[123]	SES versus PES	1012	Indefinite	100 mg once a day
TAXi[124]	SES versus PES	202	"Long term"	100 mg once a day
TAXUS V[125]	PES versus BMS	1172	Indefinite	325 mg once a day
TAXUS VI[126]	PES versus BMS	448	Greater than or equal to 6 months	75 mg at least 2 hours prior; greater than or equal to 75 mg after
REALITY[127]	SES versus PES	1353	Indefinite	100 mg once a day
TAXUS V ISR[128]	PES versus VBT for ISR	396	Indefinite (9-month minimum, indefinite recommended)	325 mg once a day

ACHIEVE indicates a brand-name paclitaxel-coated stent; BMS, bare-metal stent; C-SIRIUS, Canadian Sirolimus-Eluting Stent in Coronary Lesions; ELUTES, European evaLUation of pacliTaxel Eluting Stent; E-SIRIUS, European Sirolimus-Eluting Stent in Coronary Lesions; h, hour; ISAR-DESIRE, Drug-Eluting Stents for in-stent REstenosis; ISAR-DIABETES, Paclitaxel-Eluting Stent Versus Sirolimus-Eluting Stent for the Prevention of Restenosis in Diabetic Patients With Coronary Artery Disease; ISR, in-stent restenosis; IV, intravenous; ML PENTA, multilink stainless steel bare metal stent; PCI, percutaneous coronary intervention; PES, paclitaxel-eluting stent; RAVEL, A Randomized Comparison of a Sirolimus-Eluting Stent With a Standard Stent for Coronary Revascularization; REALITY, Prospective Randomized Multi-Center Head-to-Head Comparison of the Sirolimus-Eluting Stent (Cypher) and the Paclitaxel-Eluting Stent (TAXUS); SES, sirolimus-eluting stent; SIRIUS, Sirolimus-Eluting Stent in Coronary Lesions; SIRTAX, Sirolimus-Eluting Stent Compared With Paclitaxel-Eluting Stent for Coronary Revascularization; TAXi, Paclitaxel and sirolimus stents in the real world of interventional cardiology; TAXUS V ISR, Paclitaxel-Eluting Stents versus Brachytherapy for In-Stent Restenosis; and VBT, vascular brachytherapy.

effect was not confirmed in 1 investigation.[139] No excess hazard has been reported with the 600-mg compared with the 300-mg dose for patients treated with fibrinolytic therapy; however, loading doses greater than 300 mg have not been studied.[140] Larger trials will more fully evaluate higher doses of clopidogrel on clinical events, as well as further evaluate safety (e.g., bleeding). The OASIS-7 trial is comparing 600-mg with 300-mg loading doses of clopidogrel and will provide further evidence about the optimal treatment strategy.

There is agreement that the loading dose should be administered before PCI. What is unclear is the precise time when the loading dose must be given to achieve a desirable therapeutic effect. Evidence from the CREDO (Clopidogrel for the Reduction of Events During Observation) trial suggests that with a 300-mg dose, 6 hours is the minimum time.[131] With the 600-mg dose, 2 hours may be sufficient (141), although maximal platelet inhibition may not be achieved until 3 to 4 hours.[142]

Long-term clopidogrel therapy alone may not achieve adequate inhibition for PCI. Patients on long-term therapy with clopidogrel experience significant additional incremental inhibition of platelet aggregation when given a loading dose.[143] In patients treated with fibrinolytic therapy, however, loading doses of greater than 300 mg have not been studied.[144]

8. Bare-Metal and Drug-Eluting Stents

8.1. Selection of a Bare-Metal or Drug-Eluting Stent

Observational studies indicate that physicians routinely implant stents when performing coronary interventions. Two types of stents are available: BMS and DES. Drug-eluting stents have become increasingly popular as standard therapy. In 2005, a sampling of 140 US hospitals indicated that 94% of patients treated with a stent received at least 1 DES.[145] More recently, however, because of concerns about stent thrombosis and the mandate that each DES-treated patient take prolonged DAT, the proportion of DES use has declined to 60% to 70%.

The results of the clinical trials that led to FDA approval of the DES provide support for its use in suitable patients. Extended follow-up of the initial investigated patient cohorts to 4 years confirms the sustained benefit of DES in decreasing the need for repeat revascularization but without differences in death or MI.[146–148] Randomized clinical

Table 16. Updates to Section 7.3.5: Drug-Eluting and Bare-Metal Stents

2005 PCI Guideline Update Recommendation	2007 PCI Focused Update Recommendation	Comments
	Class I	
A drug-eluting stent (DES) should be considered as an alternative to the bare-metal stent in subsets of patients in whom trial data suggest efficacy. *(Level of Evidence: A)*	1. A DES should be considered as an alternative to a BMS in those patients for whom clinical trials indicate a favorable effectiveness/safety profile. *(Level of Evidence: A)*	Modified recommendation (changed text)
	2. Before implanting a DES, the interventional cardiologist should discuss with the patient the need for and duration of DAT and confirm the patient's ability to comply with the recommended therapy for DES. *(Level of Evidence: B)*	New recommendation
	3. In patients who are undergoing preparation for PCI and are likely to require invasive or surgical procedures for which DAT must be interrupted during the next 12 months, consideration should be given to implantation of a BMS or performance of balloon angioplasty with a provisional stent implantation instead of the routine use of a DES. *(Level of Evidence: C)*	New recommendation
	Class IIa	
	1. In patients for whom the physician is concerned about risk of bleeding, a lower dose of 75 mg to 162 mg of aspirin is reasonable. *(Level of Evidence: C)*	New recommendation
	Class IIb	
A DES may be considered for use in anatomic settings in which the usefulness, effectiveness, and safety have not been fully documented in published trials. *(Level of Evidence: C)*	1. A DES may be considered for clinical and anatomic settings in which the effectiveness/safety profile appears favorable but has not been fully confirmed by clinical trials. *(Level of Evidence: C)*	Modified recommendation (changed text)

BMS indicates bare-metal stent; DAT, dual antiplatelet therapy; DES, drug-eluting stent; and PCI, percutaneous coronary intervention.

trials in selected clinical subsets such as BMS in-stent restenosis, total occlusions, diabetes mellitus, and small-diameter arteries have also demonstrated the value of DES and have prompted physicians to extend the application of DES beyond the narrow patient populations included in the initial approval trials.[122,126,149–154] The duration of follow-up of these "off-label" studies and the small number of patients enrolled, however, limit the detection of subtle differences in important end points such as stent thrombosis, death, or MI.

It is important to recognize certain differences between the BMS and DES when selecting a stent for an individual patient or lesion. First, in general, a DES may be more difficult to implant than a BMS. The DES has a polymer coating that stiffens the stent and makes it less conformable. Accordingly, one reason for using a BMS is that it can be used in patients in whom a DES cannot be implanted successfully. Second, the DES is substantially more expensive than the BMS. When financial resources are limited, use of the DES may be rationed, with implantation only in those patients at greatest risk for restenosis.

A third but very important difference relates to the inhibition of endothelial coverage of the DES and the need for extended DAT (Table 16). After introduction of the BMS, it was associated with a disturbingly high incidence of stent thrombosis.[141] Stent thrombosis often presented as MI or even death and usually occurred in the first 30 days after implantation. Changes in technique such as high inflation pressure and intravascular ultrasound (IVUS)-guided deployment and use of concomitant combined aspirin and thienopyridine therapy substantially reduced the incidence of stent thrombosis to a clinically acceptable level.[155] Importantly, the requisite duration of DAT was only 4 weeks, and some advocated only 2 weeks. The importance of DAT in preventing stent thrombosis was further strengthened by the outcome of patients for whom DAT was discontinued prematurely because of the need for those patients to undergo surgical procedures. These patients experienced a disturbingly high incidence of stent thrombosis.[156] The critical role of DAT in preventing stent thrombosis was also noted among patients with BMS who had received brachytherapy for in-stent restenosis. Presumably these patients were less likely to develop subsequent neointimal coverage of the endoluminal stent surface and were accordingly then more susceptible to stent thrombosis.

In the initial randomized trials that compared the DES with BMS, DAT was administered for 30 days to 6 months. The most recent guidelines update describes a minimum duration of 3 months of DAT for an SES and 6 months for a PES. On the basis of results from other trials that suggest a sustained benefit of DAT, these guidelines further state that ideally DAT should be extended to 12 months. Although these recommendations were to some extent arbitrary, subsequent studies have confirmed that premature discontinuation of DAT, that is, at a time less than "minimal duration" (3 months for the SES and 6 months for the PES) was highly associated with stent thrombosis.[157]

The tight relationship between DAT and stent thrombosis for patients treated with DES warrants emphasis and has

implications for selecting the type of stent deployed at the time of PCI. For example, the clinician should not select a DES for a patient who does not have access to DAT for financial reasons or who is unlikely to be compliant in taking DAT. One study revealed that 14% of patients had stopped DAT 1 month after implantation of the DES.[158] Also, implantation of a BMS may be more appropriate in a patient with a known increased risk of bleeding. In situations such as these, the consequences of developing restenosis are considered less untoward than those of stent thrombosis or significant bleeding.

Furthermore, prescribed premature discontinuation of DAT in patients treated with a DES should not be done casually. For example, routine dental procedures should not justify cessation of DAT even though it is anticipated DAT will be subsequently resumed.[133] Consideration should be given to delay scheduling of elective procedures that normally warrant discontinuation of antiplatelet agents. The benefit of DES in reducing the need for target vessel revascularization (TVR) also should be taken into account. Some registries have shown 1-digit TVR rates with the BMS, and the absolute reduction in these events using the DES depends on patient and lesion characteristics.

There are also concerns related to the appropriate duration of DAT. More recently, the occurrence of late (up to 1 year) or very late (beyond 1 year) stent thrombosis among DES-treated patients has been described.[159] One database analysis suggests that extended use of DAT may have value in preventing late stent thrombosis, whereas others disagree.[160]

Outcomes of patients in the initial FDA-approval trials to 4 years provides reassurance that, at least for those types of patients, despite a small excess of stent thrombosis, there appears to be no increase in death or MI when comparing DES-treated groups with BMS-treated groups. As noted, protocol-recommended DAT in these patients was not more than 6 months, although extended DAT was not prohibited. (These results are observed despite a significant excess occurrence of stent thrombosis among patients who received a paclitaxel stent.) Some have postulated that the substantial additional revascularization procedures experienced by BMS patients were associated with a small but significant excess rate of death and MI that offset any deaths or MIs that may have occurred in the DES group related to stent thrombosis.

Less data are available regarding the outcomes of patients who receive a DES for an "off-label" indication. Such patients have characteristics of their coronary disease, for example, a lesion in an artery less than 2.5 mm in diameter, very long lesions, bifurcation lesions, or a clinical syndrome such as acute MI, that were excluded in the FDA-approval trials. Reports from large observational studies indicate that "off-label" patients may experience higher rates of repeat revascularization and death and MI at 1 year than DES patients with "on-label" features. Importantly, a similar relationship is observed for patients treated with a BMS. In addition, there appears to be a significant association between "off-label" use and stent thrombosis. Accordingly, the appropriate selection for DAT among "off-label" DES patients may be different than for "on-label" patients.

At this point in time, 12 months of DAT is recommended for all patients who receive a DES[120] (see Section 6.2.1) unless there is a high risk of bleeding. The benefits and indications for treatment with DAT beyond 1 year in patients with DES are the subject of ongoing studies. Low-dose aspirin should be continued indefinitely. For patients with clinical features associated with stent thrombosis, such as renal insufficiency, diabetes, or procedural characteristics such as multiple stents or treatment of a bifurcation lesion, extended DAT beyond 1 year may be reasonable. The risk of stent thrombosis needs to be balanced with other medical conditions and nonmedical factors that might affect the risk-benefit ratio of DAT versus other therapies. Finally, certain DES-treated patients have already discontinued DAT 1 year after stent implantation. No information yet supports restarting DAT in these patients.

9. Secondary Prevention

Table 17 presents revised recommendations based on the 2006 AHA/ACC Secondary Prevention Guidelines for Patients with Coronary and Other Atherosclerotic Vascular Diseases.[11] This table replaces Table 26 from the 2005 PCI Guideline Update.[13a] Classes of recommendation and a corresponding level of evidence have been added for all recommendations. There is a new recommendation for annual influenza vaccination, and the section on antiplatelet agents/anticoagulants has been modified slightly to reflect the recent evidence on aspirin dosage in patients who have undergone PCI with stent placement. Other changes since publication of the 2006 ACC/AHA Secondary Prevention Guidelines include the addition of recommended daily physical activity and a Class IIa recommendation for lowered low-density lipoprotein cholesterol.

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Table 17. Comprehensive Risk Reduction for Patients With Coronary and Other Vascular Disease After PCI

2005 PCI Recommendations	2007 PCI Recommendations	2007 COR and LOE	Comments
	Smoking **Goal:** Complete cessation, no exposure to environmental tobacco smoke		
Ask about tobacco status at every visit.	1. Status of tobacco use should be asked about at every visit.	*I (B)*	Modified recommendation (changed text)
Strongly encourage patient and family to stop smoking and avoid secondhand smoke.	2. Every tobacco user and family members who smoke should be advised to quit at every visit.	*I (B)*	No content change
Assess the tobacco user's willingness to quit.	3. The tobacco user's willingness to quit should be assessed.	*I (B)*	No content change
Assist by counseling and developing a plan for quitting.	4. The tobacco user should be assisted by counseling and developing a plan for quitting.	*I (B)*	No content change
Arrange follow-up, referral to special programs, or pharmacological therapy (including nicotine replacement and bupropion).	5. Follow-up, referral to special programs, or pharmacotherapy (including nicotine replacement and pharmacological treatment) should be arranged.	*I (B)*	No content change
Urge avoidance of exposure to environmental tobacco smoke at work and home.	6. Exposure to environmental tobacco smoke at work and home should be avoided.	*I (B)*	No content change
	Blood Pressure Control **Goal:** Less than 140/90 mm Hg or less than 130/80 mm Hg if patient has diabetes or chronic kidney disease		
Initiate or maintain lifestyle modification (weight control, increased physical activity, alcohol moderation, moderate sodium restriction, and emphasis on fruits, vegetables, and low-fat dairy products) in all patients.	1. For patients with blood pressure greater than or equal to 140/90 mm Hg (or greater than or equal to 130/80 mm Hg for patients with diabetes or chronic kidney disease), it is recommended to initiate or maintain lifestyle modification—weight control; increased physical activity; alcohol moderation; sodium reduction; and emphasis on increased consumption of fresh fruits, vegetables, and low-fat dairy products.	*I (B)*	No content change
Add blood pressure medication,* emphasizing the use of beta blockers and inhibitors of the renin-angiotensin-aldosterone system.	2. For patients with blood pressure greater than or equal to 140/90 mm Hg (or greater than or equal to 130/80 mm Hg for patients with diabetes or chronic kidney disease), it is useful as tolerated, to add blood pressure medication, treating initially with beta blockers and/or ACE inhibitors, with the addition of other drugs such as thiazides as needed to achieve goal blood pressure.	*I (A)*	Modified recommendation (changed text)
	Lipid Management **Goal:** LDL-C substantially less than 100 mg per dL (If triglycerides are greater than or equal to 200 mg per dL, non–HDL-C should be less than 130 mg per dL†.)		
Start dietary therapy in all patients (less than 7% of total calories as saturated fat and less than 200 mg/d cholesterol).	1. Starting dietary therapy is recommended. Reduce intake of saturated fats (to less than 7% of total calories), *trans* fatty acids, and cholesterol (to less than 200 mg per day).	*I (B)*	Modified recommendation (changed text)
	2. Adding plant stanol/sterols (2 g per day) and/or viscous fiber (greater than 10 g per day) is reasonable to further lower LDL-C.	*IIa (A)*	New recommendation
Promote physical activity and weight management.	3. Promotion of daily physical activity and weight management is recommended.	*I (B)*	Modified recommendation (changed text)
Encourage increased consumption of omega-3 fatty acids in fish‡ or 1 g/d omega-3 fatty acids from supplements for risk reduction (for treatment of elevated triglycerides, higher doses are usually necessary for risk reduction).	4. It may be reasonable to encourage increased consumption of omega-3 fatty acids in the form of fish‡ or in capsules (1 g per day) for risk reduction. For treatment of elevated triglycerides, higher doses are usually necessary for risk reduction.	*IIb (B)*	No content change
Assess fasting lipid profile in all patients, preferably within 24 hours of an acute event. For hospitalized patients, initiate lipid-lowering medication as recommended below before discharge according to the following guide:	5. A fasting lipid profile should be assessed in all patients and within 24 hours of hospitalization for those with an acute cardiovascular or coronary event. For hospitalized patients, initiation of lipid-lowering medication is indicated as recommended below before discharge according to the following schedule:	*I (A)*	Modified recommendation (changed text)

Table 17. Continued

2005 PCI Recommendations	2007 PCI Recommendations	2007 COR and LOE	Comments
LDL-C less than 100 mg/dL (baseline or on treatment): Statins preferred to lower LDL-C.	• LDL-C should be less than 100 mg per dL.	*I (A)*	Modified recommendation (changed text)
	• Further reduction of LDL-C to less than 70 mg per dL is reasonable.	*IIa (A)*	New recommendation
If LDL-C is greater than or equal to 100 mg/dL (baseline or on treatment), initiate or intensify LDL-C–lowering therapy with drug treatment. May require combination therapy with standard-dose ezetimide, bile acid sequestrant, or niacin.‖	• If baseline LDL-C is greater than or equal to 100 mg per dL, LDL-lowering drug therapy§ should be initiated.	*I (A)*	Modified recommendation (changed text)
	• If on-treatment LDL-C is greater than or equal to 100 mg per dL, intensify LDL-lowering drug therapy (may require LDL-lowering drug combination¶) is recommended.	*I (A)*	New recommendation
	• If baseline LDL-C is 70 to 100 mg per dL, it is reasonable to treat to LDL-C less than 70 mg per dL.	*IIa (B)*	New recommendation
If triglycerides are greater than or equal to 150 mg/dL or HDL-C is less than 40 mg/dL, emphasize weight management and physical activity. Advise smoking cessation.	• If triglycerides are greater than or equal to 150 mg per dL or HDL-C is less than 40 mg per dL, weight management, physical activity, and smoking cessation should be emphasized.	*I (B)*	Modified recommendation (changed text)
If triglycerides are 200 to 499 mg/dL:	• If triglycerides are 200 to 499 mg per dL††, non–HDL-C target should be less than 130 mg per dL.	*I (B)*	Modified recommendation (changed text)
	• If triglycerides are 200 to 499 mg per dL††, further reduction of non–HDL-C to less than 100 mg per dL is reasonable.	*IIa (B)*	New recommendation
	6. Therapeutic options to reduce non–HDL-C include:		
	• More intense LDL-C–lowering therapy is indicated.	*I (B)*	New recommendation
After LDL-C–lowering therapy,**†† consider adding fibrate or niacin¶	• Niacin‖ (after LDL-C–lowering therapy) can be beneficial.	*IIa (B)*	Modified recommendation (changed text)
	• Fibrate therapy‡‡ (after LDL-C–lowering therapy) can be beneficial.	*IIa (B)*	Modified recommendation (changed text)
If triglycerides are greater than or equal to 500 mg/dL: • Consider fibrate or niacin§ before LDL-C–lowering therapy.¶†† • Consider omega-3 fatty acids as an adjunct for high triglycerides.	7. If triglycerides are greater than or equal to 500 mg per dL,††§§ therapeutic options indicated and useful to prevent pancreatitis are fibrate§‡‡ or niacin§‖ before LDL-lowering therapy, and treat LDL-C to goal after triglyceride-lowering therapy. Achieving a non–HDL-C of less than 130 mg per dL is recommended.	*I (C)*	Modified recommendation (changed text)
	Physical Activity **Goal:** 30 minutes 5 days per week; optimal daily		
Cardiac rehabilitation programs are recommended, particularly for patients with multiple modifiable risk factors and/or moderate- to high-risk patients for whom supervised exercise training is warranted.	1. Advising medically supervised programs (cardiac rehabilitation) for high-risk patients (e.g., recent acute coronary syndrome or revascularization, heart failure) is recommended.	*I (B)*	Modified recommendation (changed text)
Assess risk, preferably with exercise testing, to guide prescription.	2. For all patients, it is recommended that risk be assessed with a physical activity history and/or an exercise test to guide prescription.	*I (B)*	Modified recommendation (changed text)
Encourage a minimum of 30 to 60 minutes of activity, preferably daily or at least 5 days per week (brisk walking, jogging, cycling, or other aerobic activity) supplemented by an increase in daily lifestyle activities (e.g., walking breaks at work, gardening, and household work).	3. For all patients, encouraging 30 to 60 minutes of moderate-intensity aerobic activity is recommended, such as brisk walking on most—preferably all—days of the week, supplemented by an increase in daily lifestyle activities (e.g., walking breaks at work, gardening, and household work).	*I (B)*	Modified recommendation (changed text)
Encourage resistance training 2 days per week.	4. Encouraging resistance training 2 days per week may be reasonable.	*IIb (C)*	No content change

Table 17. Continued

2005 PCI Recommendations	2007 PCI Recommendations	2007 COR and LOE	Comments
	Weight Management **Goal:** BMI: 18.5 to 24.9 kg/m^2 Waist circumference: men less than 40 inches (102 cm), women less than 35 inches (89 cm)		
Calculate BMI and measure waist circumference as part of evaluation. Monitor response of BMI and waist circumference to therapy.	1. It is useful to assess BMI and/or waist circumference on each visit and consistently encourage weight maintenance/reduction through an appropriate balance of physical activity, caloric intake, and formal behavioral programs when indicated to maintain/achieve a BMI between 18.5 and 24.9 kg/m^2.	*I (B)*	Modified recommendation (changed text)
Start weight management and physical activity as appropriate. Desirable BMI range is 18.5 to 24.9 kg/m^2.	2. The initial goal of weight-loss therapy should be to reduce body weight by approximately 10% from baseline. With success, further weight loss can be attempted if indicated through further assessment.	*I (B)*	Modified recommendation (changed text)
If waist circumference is 35 inches or greater in women or 40 inches or greater in men, initiate lifestyle changes and treatment strategies for metabolic syndrome.	3. If waist circumference (measured horizontally at the iliac crest) is 35 inches (89 cm) or greater in women and 40 inches (102 cm) or greater in men, it is useful to initiate lifestyle changes and consider treatment strategies for metabolic syndrome as indicated.	*I (B)*	Modified recommendation (changed text)
	Diabetes Management **Goal:** HbA_{1c} less than 7%		
Appropriate glucose-lowering therapy to achieve near-normal fasting plasma glucose, as indicated by HbA_{1c}.	1. It is recommended to initiate lifestyle and pharmacotherapy to achieve near-normal HbA_{1c}.	*I (B)*	Modified recommendation (changed text)
Treatment of other risk factors (e.g., physical activity, weight management, blood pressure, and cholesterol management).	2. Beginning vigorous modification of other risk factors (e.g., physical activity, weight management, blood pressure control, and cholesterol management as recommended above) is beneficial.	*I (B)*	Modified recommendation (changed text)
	3. Coordination of diabetic care with the patient's primary care physician or endocrinologist is beneficial.	*I (C)*	New recommendation
	Antiplatelet Agents/Anticoagulants: Aspirin		
For all post-PCI stented patients, aspirin 325 mg daily should be given for at least 1 month after BMS implantation, 3 months after sirolimus stent implantation, and 6 months after paclitaxel stent implantation, after which daily long-term aspirin‖ ‖ (75 mg to 162 mg per day) should be continued indefinitely in all patients if not contraindicated.	**1. For all post-PCI stented patients without allergy or increased risk of bleeding, aspirin 162 mg to 325 mg daily should be given for at least 1 month after BMS implantation, 3 months after sirolimus-eluting stent implantation, and 6 months after paclitaxel-eluting stent implantation, after which long-term aspirin use should be continued indefinitely at a dose of 75 mg to 162 mg daily.**	***I (B)***	Modified recommendation (changed text)
	2. In patients for whom the physician is concerned about risk of bleeding, lower-dose 75 mg to 162 mg of aspirin is reasonable during the initial period after stent implantation.	***IIa (C)***	New recommendation
	Antiplatelet Agents/Anticoagulants: Clopidogrel		
For post-PCI stented patients, clopidogrel 75 mg per day should be given for at least 1 month after BMS implantation, 3 months after sirolimus stent implantation, and 6 months after paclitaxel stent implantation, after which clopidogrel should ideally be continued for up to 12 months in all stented patients who are not at high risk of bleeding.	**1. For all post-PCI patients who receive a DES, clopidogrel 75 mg daily should be given for *at least 12 months* if patients are not at high risk of bleeding. For post-PCI patients receiving a BMS, clopidogrel should be given for a minimum of 1 month and *ideally up to 12 months* (unless the patient is at increased risk of bleeding; then it should be given for a minimum of 2 weeks).**	***I (B)***	Modified recommendation (changed text)
	2. For all post-PCI non-stented STEMI patients, treatment with clopidogrel should continue for at least 14 days.	***I (B)***	New recommendation
	3. Long-term maintenance therapy (e.g., 1 year) with clopidogrel (75 mg per day orally) is reasonable in STEMI and non-STEMI patients who undergo PCI without reperfusion therapy.	***IIa (C)***	New recommendation

Table 17. Continued

2005 PCI Recommendations	2007 PCI Recommendations	2007 COR and LOE	Comments
	Antiplatelet Agents/Anticoagulants: Warfarin		
Manage warfarin to an INR of 2.5 to 3.5 for post-MI patients when clinically indicated or for those not able to take aspirin or clopidogrel.	1. Managing warfarin to an INR equal to 2.0 to 3.0 for paroxysmal or chronic atrial fibrillation or flutter is recommended, and in post-MI patients when clinically indicated (e.g., atrial fibrillation, left ventricular thrombus).	*I (A)*	Modified recommendation (changed text)
	2. Use of warfarin in conjunction with aspirin and/or clopidogrel is associated with an increased risk of bleeding and should be monitored closely.	*I (B)*	New Recommendation
	3. In patients requiring warfarin, clopidogrel, and aspirin therapy after PCI, an INR of 2.0 to 2.5 is recommended with low dose aspirin (75 mg to 81 mg) and a 75-mg dose of clopidogrel.	***I (C)***	New recommendation
	Renin-Angiotensin-Aldosterone System Blockers: ACE Inhibitors		
Consider use of ACE inhibitors for all CHD patients indefinitely; start early after MI in stable high-risk patients (anterior MI, previous MI, Killip class greater than or equal to II [S_3 gallop, rales, radiographic HF]).	1. ACE inhibitors should be started and continued indefinitely in all patients with LVEF less than or equal to 40% and for those with hypertension, diabetes, or chronic kidney disease, unless contraindicated.	*I (A)*	Modified recommendation (changed text)
Use as needed to manage blood pressure or consider for long-term therapy in all other patients.	2. ACE inhibitors should be started and continued indefinitely in patients who are not lower risk (lower risk defined as those with normal LVEF in whom cardiovascular risk factors are well controlled and revascularization has been performed), unless contraindicated.	*I (B)*	Modified recommendation (changed text)
Continue indefinitely for all patients with LV dysfunction (ejection fraction less than or equal to 0.40) or symptoms of heart failure.	3. Among lower risk patients (i.e., those with normal LVEF in whom cardiovascular risk factors are well controlled and revascularization has been performed) use of ACE inhibitors is reasonable.	*IIa (B)*	Modified recommendation (changed text)
	Renin-Angiotensin-Aldosterone System Blockers: Angiotensin Receptor Blockers		
Use angiotensin receptor blockers in post-STEMI patients who are intolerant of ACE inhibitors and who have either clinical or radiological signs of heart failure or LVEF less than 0.40.	1. Use of angiotensin receptor blockers is recommended in patients who are intolerant of ACE inhibitors and have HF or have had an MI with LVEF less than or equal to 40%.	*I (A)*	Modified recommendation (changed text)
	2. Angiotensin receptor blockers are useful in other patients who are ACE-inhibitor intolerant and have hypertension.	***I (B)***	New recommendation
	3. Considering use in combination with ACE inhibitors in systolic dysfunction HF may be reasonable.	*IIb (B)*	New recommendation
	Renin-Angiotensin-Aldosterone System Blockers: Aldosterone Blockade		
Aldosterone blockade in post-STEMI patients without significant renal dysfunction¶¶ or hyperkalemia*** who are already receiving therapeutic doses of an ACE inhibitor, have an LVEF less than or equal to 0.40, and have either diabetes or heart failure.	1. Use of aldosterone blockade in post-MI patients without significant renal dysfunction¶¶ or hyperkalemia*** is recommended in patients who are already receiving therapeutic doses of an ACE inhibitor and beta blocker, have an LVEF of less than or equal to 40%, and have either diabetes or HF.	*I (A)*	Modified recommendation (changed text)

Table 17. Continued

2005 PCI Recommendations	2007 PCI Recommendations	2007 COR and LOE	Comments
	Beta Blockers		
Start in all post-MI and acute patients (arrhythmia, LV dysfunction, inducible ischemia). Continue for a minimum of 6 months; continue indefinitely in patients with STEMI. Observe usual contraindications.	1. It is beneficial to start and continue beta-blocker therapy indefinitely in all patients who have had MI, acute coronary syndrome, or LV dysfunction with or without HF symptoms, unless contraindicated.	*I (A)*	Modified recommendation (changed text)
Use as needed to manage angina, rhythm, or blood pressure in all other patients.	2. It is reasonable to consider long-term therapy for all other patients with coronary or other vascular disease or diabetes unless contraindicated.	*IIa (C)*	Modified recommendation (changed text)
	Influenza Vaccination		
	1. Patients with cardiovascular disease should have an annual influenza vaccination.	*I (B)*	New recommendation

Recommendations in bold type are those the writing committee felt deserved extra emphasis. The 2007 PCI recommendations are written in complete sentences, in accordance with ACC/AHA Guidelines methodology.

"No content change" indicates the updated recommendation now includes a LOE and COR and a verb consistent with that LOE and COR as outlined in the ACC/AHA LOE/COR table (Table 1).

*For compelling indications for individual drug classes in specific vascular diseases, see the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7) (161).

†Non-HDL-C indicates total cholesterol minus HDL-C.

‡Pregnant and lactating women should limit their intake of fish to minimize exposure to methylmercury.

§When LDL-lowering medications are used, obtain at least a 30% to 40% reduction in LDL-C levels. If LDL-C less than 70 mg per dL is the chosen target, consider drug titration to achieve this level to minimize side effects and cost. When LDL-C less than 70 mg per dL is not achievable because of high baseline LDL-C levels, it generally is possible to achieve reductions of greater than 50% in LDL-C levels by either statins or LDL-C–lowering drug combinations.

‖Dietary supplement niacin must not be used as a substitute for prescription niacin.

¶Standard dose of statin with ezetimibe, bile acid sequestrant, or niacin.

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

††The use of resin is relatively contraindicated when triglycerides are greater than 200 mg per dL.

‡‡The combination of high-dose statin plus fibrate can increase risk for severe myopathy. Statin doses should be kept relatively low with this combination.

§§Patients with very high triglycerides should not consume alcohol. The use of bile acid sequestrant is relatively contraindicated when triglycerides are greater than 200 mg/dL.

‖‖Some recommend avoiding regular use of ibuprofen, which may limit the cardioprotective effects of aspirin. Use of cyclo-oxygenase-2 inhibitors may be associated with an increased incidence of cardiovascular events.

¶¶Creatinine should be less than 2.5 mg per dL in men and less than 2.0 mg per dL in women.

***Potassium should be less than 5.0 mEq per L.

ACE indicates angiotensin-converting enzyme; BMI, body mass index; COR, class of recommendation; CHF, congestive heart failure; HDL-C, high-density lipoprotein cholesterol; HF, heart failure; INR, international normalized ratio; LDL-C, low-density lipoprotein cholesterol; LOE, level of evidence; LVEF, left ventricular ejection fraction; MI, myocardial infarction; PCI, percutaneous coronary intervention; and STEMI, ST-elevation myocardial infarction.

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Appendix 1. Author Relationships With Industry—Writing Group to Develop the 2007 Percutaneous Coronary Intervention Focused Update of the ACC/AHA/SCAI 2005 Guidelines for Percutaneous Coronary Intervention

Committee Member	Research Grant	Speakers' Bureau/Honoraria	Stock Ownership	Board of Directors	Consultant/ Advisory Member
Dr. John W. Hirshfeld, Jr.	None	None	None	None	None
Dr. Alice K. Jacobs	None	None	None	None	None
Dr. Spencer B. King III*	None	None	None	None	• Medtronic† • Sanofi-Aventis†
Dr. Douglass A. Morrison	None	None	None	None	None
Dr. Sidney C. Smith, Jr.*	None	• Bayer (Speaking Honorarium) • Sanofi-Aventis (Honorarium)	None	None	• Bristol-Myers Squibb • Eli Lilly • GlaxoSmithKline • Pfizer • Sanofi-Aventis
Dr. David O. Williams‡	• Abbott • Boston Scientific • Cordis	None	None	None	• Abbott† • Cordis†

This table represents the actual or potential relationships with industry that were reported as of September 24, 2007. This table was updated in conjunction with all conference calls of this writing committee. A person is deemed to have a significant interest in a business if the interest represents ownership of 5% or more of the voting stock or share of the business entity, or ownership of $10 000 or more 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 in this table are modest unless otherwise noted.

*Recused from voting on Section 7: Antiplatelet Therapy.

†Significant (greater than $10 000) relationship.

‡Recused from voting on Section 8: Bare-Metal and Drug-Eluting Stents.

Appendix 2. Peer-Reviewer Relationships With Industry—2007 Percutaneous Coronary Intervention Focused Update of the ACC/AHA/SCAI 2005 Guidelines for Percutaneous Coronary Intervention

Committee Member*	Representation	Research Grant	Speakers' Bureau	Stock Ownership	Board of Directors	Consultant/Advisory Member
Dr. Vincent F. Carr	• Official Reviewer—ACCF Board of Governors	None	None	None	None	None
Dr. Robert A. Harrington	• Official Reviewer—AHA	• AstraZeneca • Bristol-Myers Squibb • Conor Med System • Cordis • Eli Lilly • GlaxoSmithKline • Merck • Sanofi-Aventis • Schering-Plough • The Medicines Co	None	None	None	None
Dr. David R. Holmes	• Official Reviewer—ACCF Board of Trustees	None	None	None	None	None
Dr. Glenn N. Levine	• Official Reviewer—AHA	• AstraZeneca • Bristol-Myers Squibb • Guidant • Medtronic • Pfizer	• Bristol-Myers Squibb • Sanofi-Aventis • The Medicines Co	None	None	• Bristol-Myers Squibb • Sanofi-Aventis • The Medicines Co
Dr. Roxana Mehran	• Official Reviewer—SCAI	• Boston Scientific • Cordis • Orbus Neiche • The Medicines Co†	• Boston Scientific • Cordis • Johnson & Johnson	None	None	• Guerbert

Committee Member*	Representation	Research Grant	Speakers' Bureau	Stock Ownership	Board of Directors	Consultant/Advisory Member
Dr. Thomas F. Koinis	• Organizational Reviewer—American Association of Family Practice	None	None	None	None	Merck
Dr. Robert C. Marshall	• Organizational Reviewer—American Association of Family Practice	None	None	None	None	None
Dr. Katherine Sherif	• Organizational Reviewer—American College of Physicians	• Novartis	None	None	None	• Novartis • Reliant
Dr. Eric R. Bates	• Content Reviewer—STEMI Writing Committee	None	• Eli Lilly • Hoffman-LaRoche • PDL Biopharma • Sanofi-Aventis • Schering-Plough	None	None	• AstraZeneca • Datascope • Eli Lilly • GlaxoSmithKline • Sanofi-Aventis • The Medicines Co
Dr. Christopher P. Cannon	• Content Reviewer—ACC/AHA Acute Coronary Syndromes Data Standards Committee	• Accumetrics† • AstraZeneca • Bristol-Myers Squibb† • GlaxoSmithKline† • Merck† • Sanofi-Aventis† • Schering-Plough†	• Accumetrics • AstraZeneca • Bristol-Myers Squibb • Merck • Pfizer • Sanofi-Aventis • Schering-Plough	None	None	• AstraZeneca • Bristol-Myers Squibb • GlaxoSmithKline • Merck • Pfizer • Sanofi-Aventis • Schering-Plough
Dr. John G. Canto	• Content Reviewer—Individual Review	• Pfizer • Schering-Plough†	• Bristol-Myers Squibb† • CV Therapeutics • GlaxoSmithKline† • Pfizer† • Sanofi-Aventis†	None	None	• NRMI/Genentech • Pfizer • Sanofi-Aventis
Dr. Bernard R. Chaitman	• Content Reviewer—ACC/AHA Acute Coronary Syndromes Data Standards Committee	• CV Therapeutics • Pfizer	• AstraZeneca • CV Therapeutics • Pfizer	None	None	• Bayer Pharmaceuticals • CV Therapeutics • Eli Lilly • F. Hoffman-La Roche • Merck • Sanofi-Aventis
Dr. James J. Ferguson	• Content Reviewer—ACCF Cardiac Catheterization and Intervention Committee	• Eisai • The Medicines Co • Vitatron/Medtronic	• Bristol-Myers Squibb • Sanofi-Aventis • Schering-Plough	None	None	• Bristol-Myers Squibb • Eisai • GlaxoSmithKline • Prism • Sanofi-Aventis • Schering-Plough • Takeda • The Medicines Co • Therox
Dr. Michael A. Fifer	• Content Reviewer—AHA Acute Cardiac Care Committee	• Merck†	None	None	None	None
Dr. Judith S. Hochman	• Content Reviewer—STEMI Guideline Committee	• Arginox Pharmaceuticals • Eli Lilly	None	None	None	• Bristol-Myers Squibb • CV Therapeutics • Datascope • Eli Lilly • GlaxoSmithKline • Merck • Procter & Gamble • Sanofi-Aventis
Dr. Sharon A. Hunt	• Content Reviewer—ACC/AHA Heart Failure Guideline Committee	None	None	None	None	• Guidant

Committee Member*	Representation	Research Grant	Speakers' Bureau	Stock Ownership	Board of Directors	Consultant/Advisory Member
Dr. Ik-Kyung Jang	• Content Reviewer—AHA Acute Cardiac Care Committee	• Mitsubishi-Tokyo Pharma	• GlaxoSmithKline	None	None	None
Dr. Hani Jneid	Content Reviewer—AHA Diagnostic and Interventional Cardiac Catheterization Committee	None	None	None	None	None
Dr. Morton J. Kern	• Content Reviewer—ACC/AHA/SCAI PCI Guideline Committee	None	None	None	None	None
Dr. Robert C. Marshall	• Content Reviewer—American Association of Family Practice	None	None	None	None	None
Dr. Robert A. O'Rourke	• Content Reviewer—ACC/AHA Chronic Stable Angina Guideline Committee	• Multiple drug companies funding BARI 2D and COURAGE trials	• Pfizer	None	None	• Aventis • Merck • Pfizer
Dr. Martha J. Radford	• Content Reviewer—ACS Data Standards	None	None	None	None	None
Dr. Rita F. Redberg	• Content Reviewer—ACC Prevention Committee	None	None	None	None	None
Dr. Charanjit S. Rihal	• Content Reviewer—AHA Diagnostic and Interventional Cardiac Catheterization Committee	None	None	None	None	None
Dr. Allan M. Ross	• Content Reviewer—Individual Review	• Boehringer Ingelheim • Genentech • Roche	None	None	None	• Boehringer Ingelheim • Roche
Dr. Samuel J. Shubrooks	• Content Reviewer—ACCF Board of Governors	None	None	None	None	None
Dr. Chittur A. Sivaram	• Content Reviewer—ACCF Board of Governors	None	None	None	None	None
Dr. Yerem Yeghiazarians	• Content Reviewer—AHA Diagnostic and Interventional Cardiac Catheterization Committee	None	• Pfizer • Sanofi-Aventis	None	None	None

This table represents the relationships of peer reviewers with industry that were disclosed at the time of peer review of this guideline. It does not necessarily reflect relationships with industry at the time of publication. Participation in the peer review process does not imply endorsement of this document. A person is deemed to have a significant interest in a business if the interest represents ownership of 5% or more of the voting stock or share of the business entity, or ownership of $10 000 or more 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 in this table are modest unless otherwise noted.

*Names are listed in alphabetical order with each category of review.

†Significant (greater than $10,000) relationship.

ACC indicates American College of Cardiology; ACCF, American College of Cardiology Foundation; ACS, American College of Surgeons; AHA, American Heart Association; and SCAI, Society for Cardiovascular Angiography and Interventions.

2007 Focused Update of the ACC/AHA/SCAI 2005 Guideline Update for Percutaneous Coronary Intervention: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines: 2007 Writing Group to Review New Evidence and Update the ACC/AHA/SCAI 2005 Guideline Update for Percutaneous Coronary Intervention, Writing on Behalf of the 2005 Writing Committee

Spencer B. King III, Sidney C. Smith, Jr, John W. Hirshfeld, Jr, Alice K. Jacobs, Douglass A. Morrison, David O. Williams, 2005 WRITING COMMITTEE MEMBERS, Sidney C. Smith, Jr, Ted E. Feldman, John W. Hirshfeld, Jr, Alice K. Jacobs, Morton J. Kern, Spencer B. King III, Douglass A. Morrison, William W. O'Neill, Hartzell V. Schaff, Patrick L. Whitlow, David O. Williams, Sidney C. Smith, Jr, Alice K. Jacobs, Cynthia D. Adams, Jeffrey L. Anderson, Christopher E. Buller, Mark A. Creager, Steven M. Ettinger, Jonathan L. Halperin, Sharon A. Hunt, Harlan M. Krumholz, Frederick G. Kushner, Bruce W. Lytle, Rick Nishimura, Richard L. Page, Barbara Riegel, Lynn G. Tarkington and Clyde W. Yancy

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Correction

In the article by King et al, "2007 Focused Update of the ACC/AHA/SCAI 2005 Guideline Update for Percutaneous Coronary Intervention: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines" that appeared in the January 15, 2008, issue of the journal, folio information for *Circulation* was incomplete in the footnote. The complete citation is *Circulation*. 2008;117:261–295. The current online version of the article has been corrected.

The publisher regrets this error.

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