ICSI Institute for Clinical Systems Improvement

Health Care Guideline

Diagnosis and Treatment of Chest Pain and Acute Coronary Syndrome (ACS)

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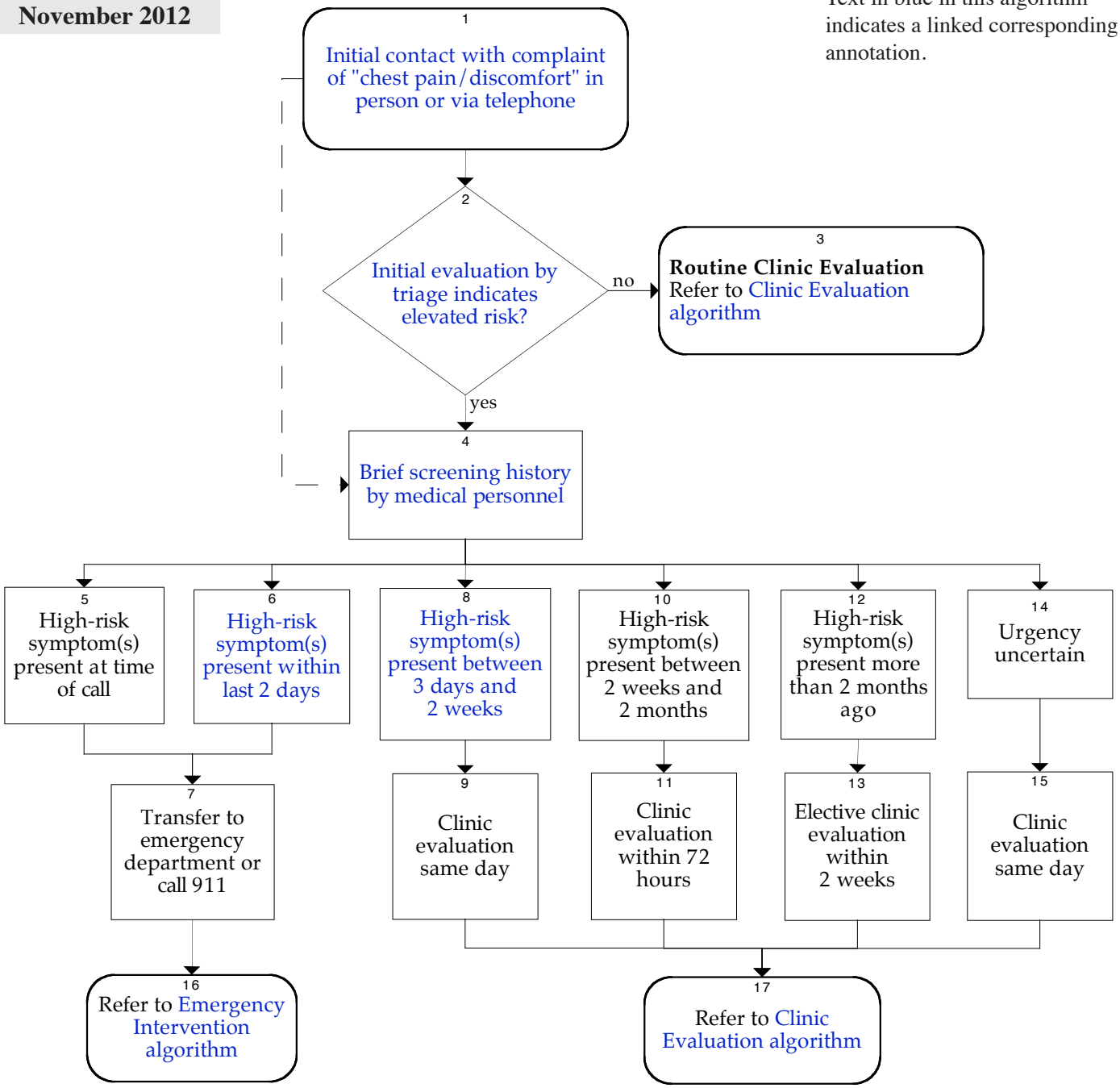
Diagnosis and Treatment of Chest Pain and Acute Coronary Syndrome (ACS)

ICSI Institute for Clinical Systems Improvement

Eighth Edition
November 2012

Chest Pain Screening Algorithm

Text in blue in this algorithm indicates a linked corresponding annotation.



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Emergency Intervention Algorithm

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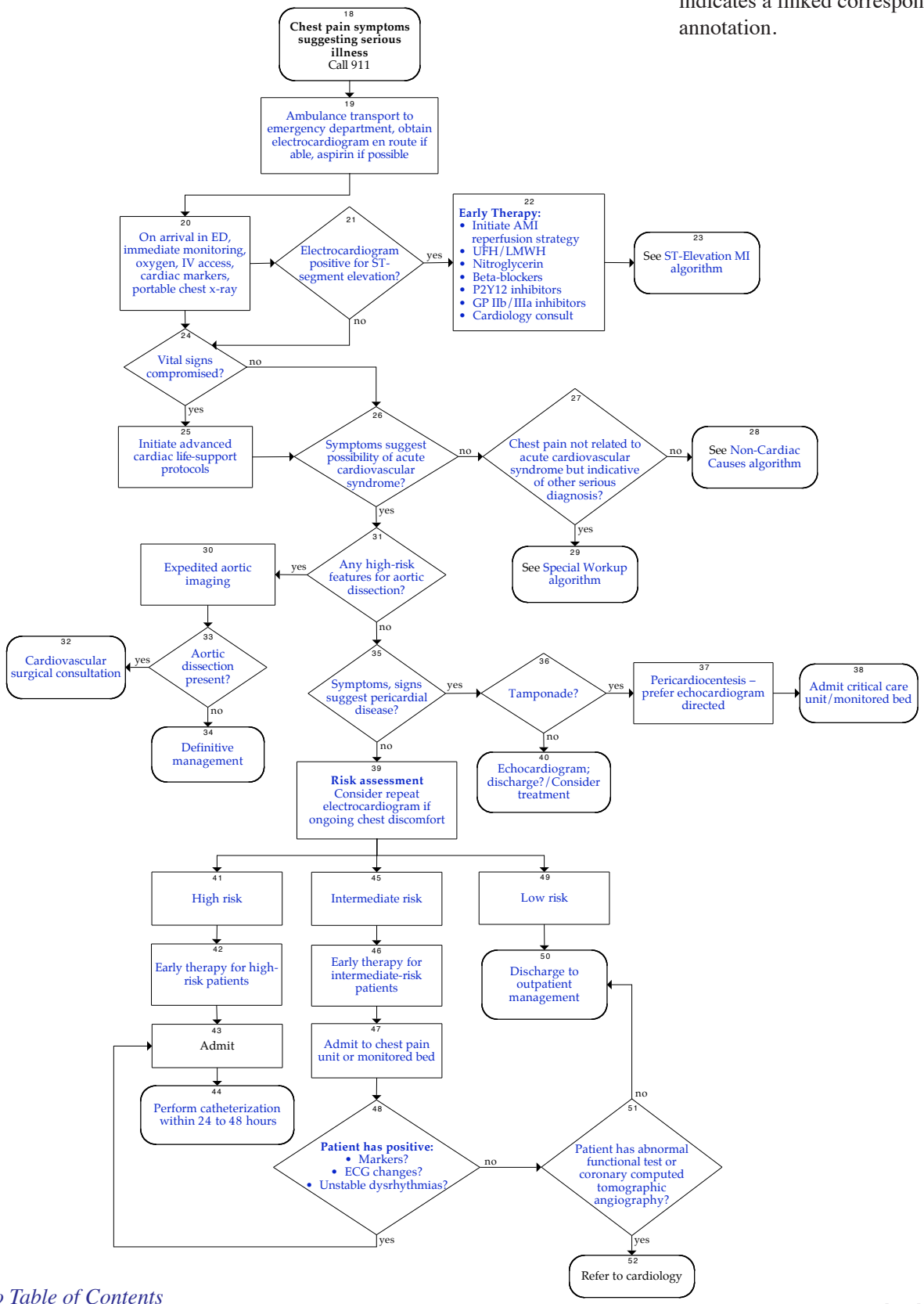

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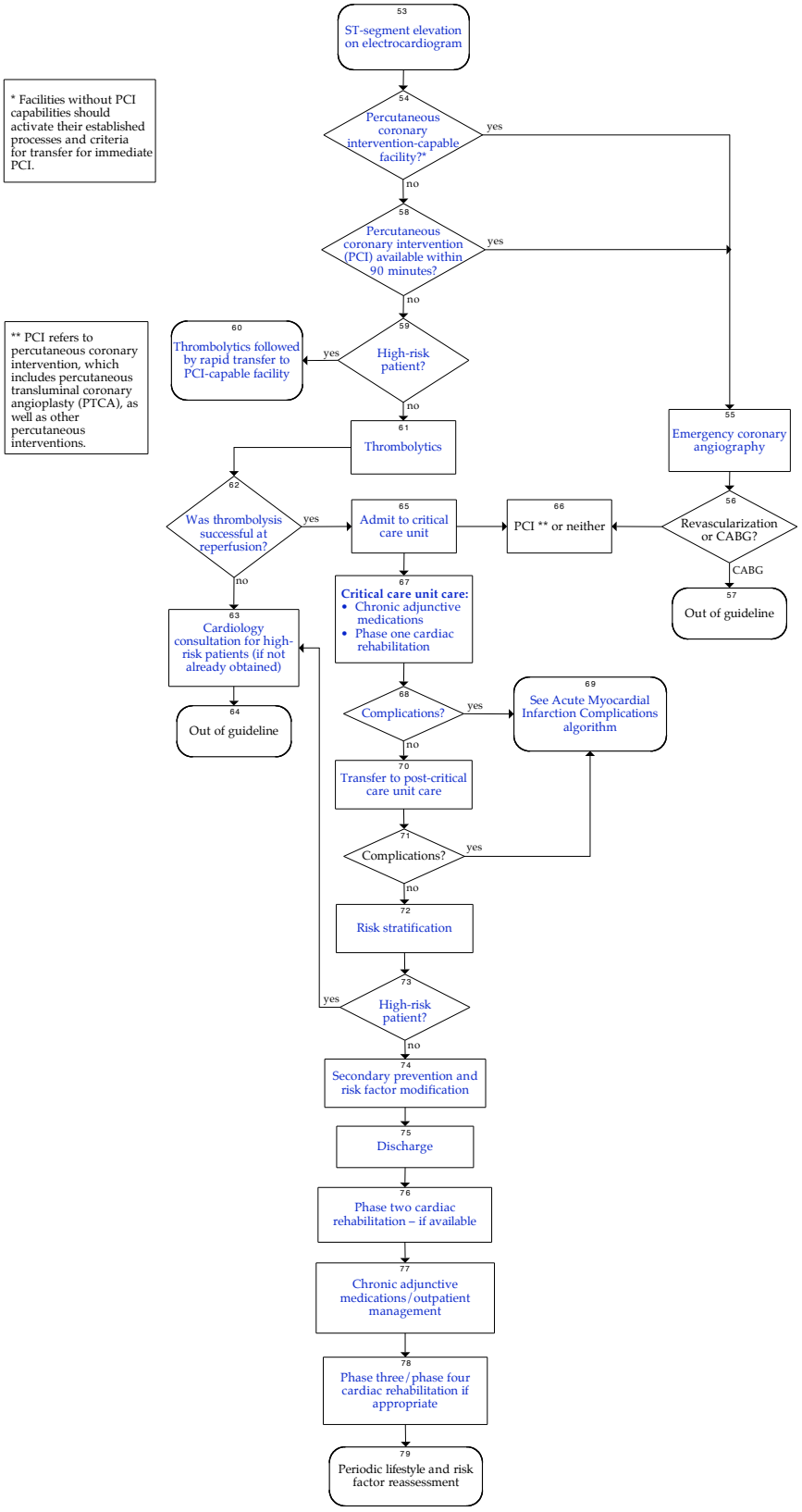
ST-Elevation Myocardial Infarction (STEMI) Algorithm



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Acute Myocardial Infarction Complications Algorithm

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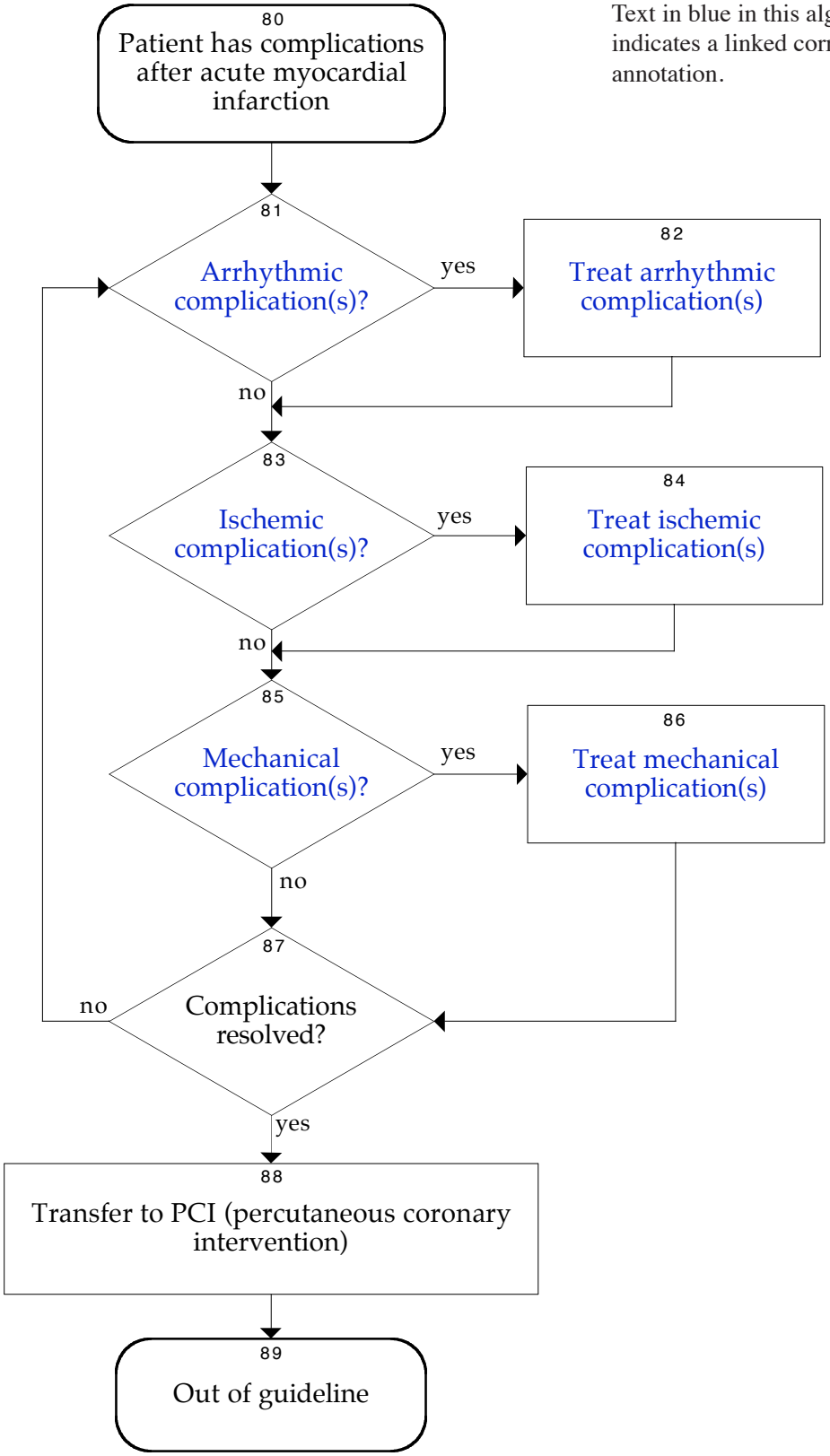

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Special Workup Algorithm

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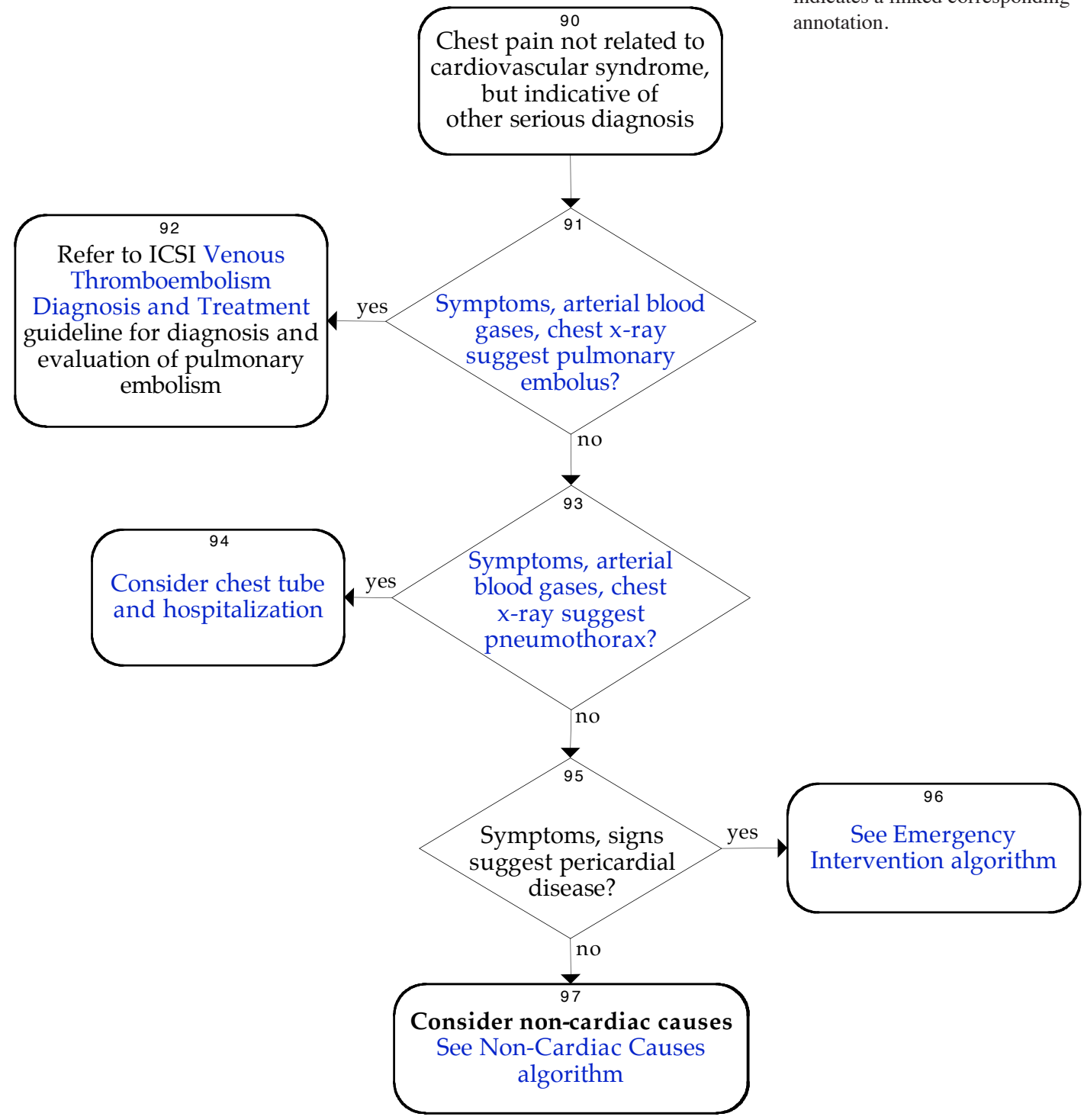

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Non-Cardiac Causes Algorithm

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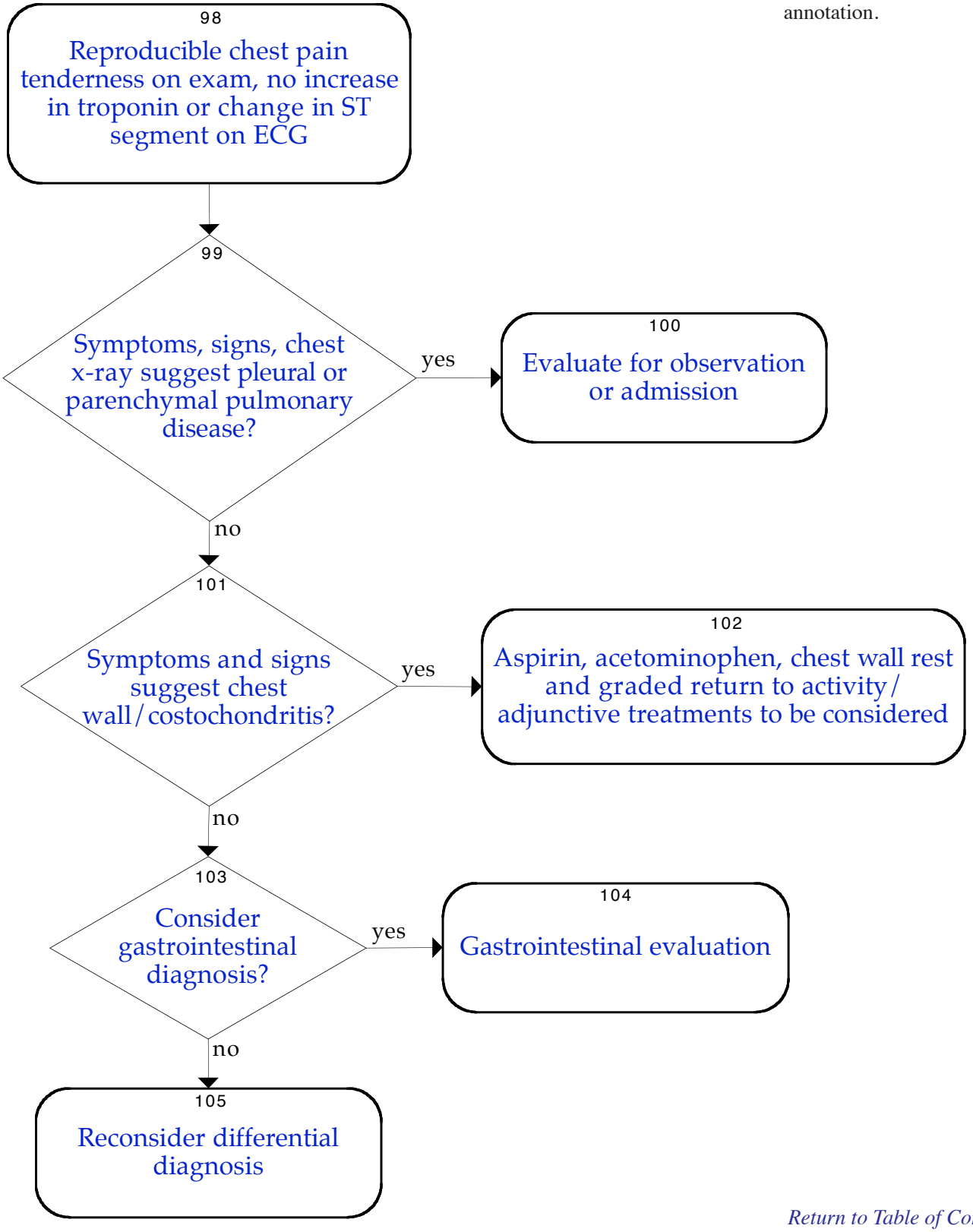

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Clinic Evaluation Algorithm

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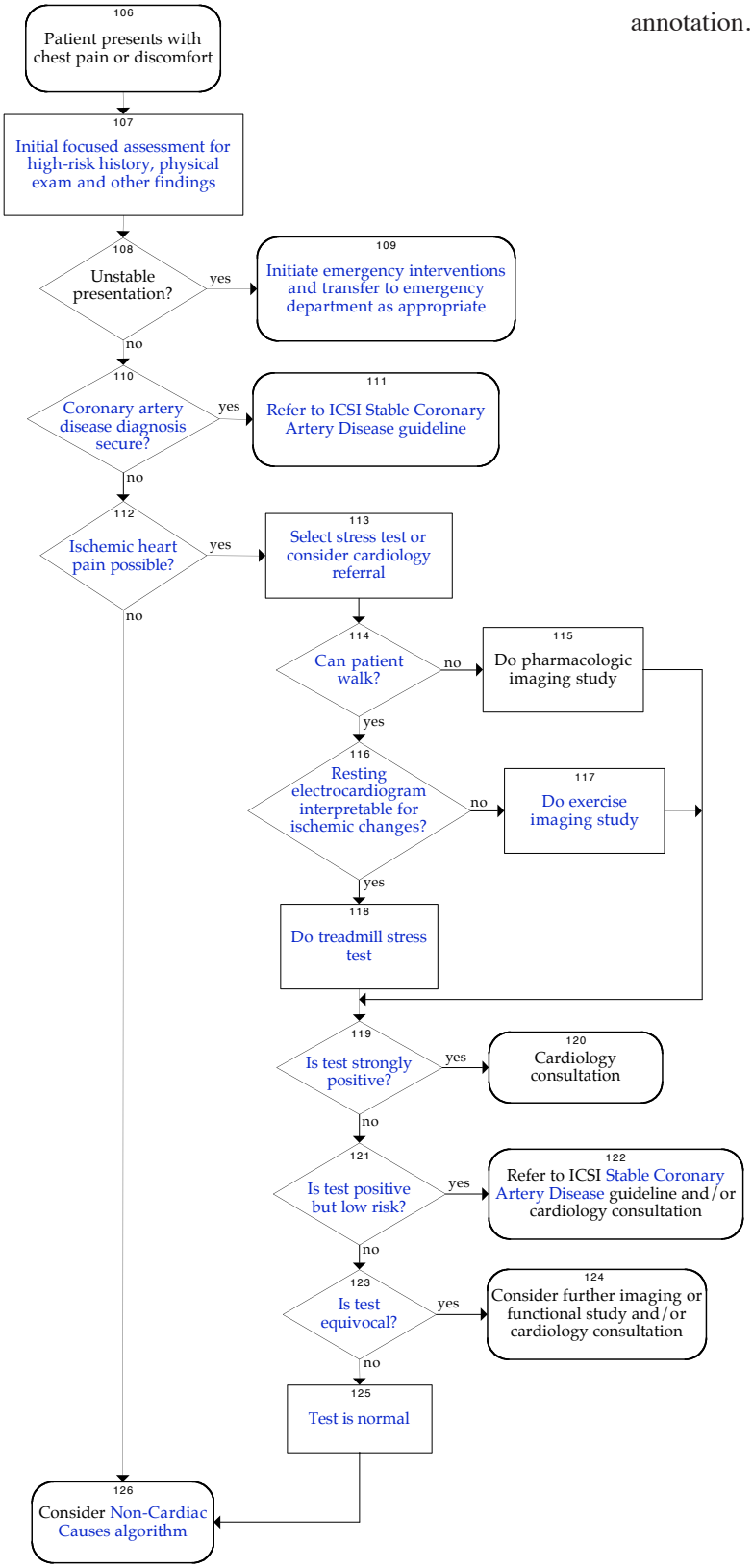

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Evidence Grading

Literature Search

A consistent and defined process is used for literature search and review for the development and revision of ICSI guidelines. The literature search was divided into two stages to identify systematic reviews, (stage I) and randomized controlled trials, meta-analysis and other literature (stage II). Literature search terms used for this revision are from February 2010 to February 2012 and include acute coronary syndrome (ACS) and bivalirudin, ACS and triponen scale, ticagrelor and acute coronary syndrome, magnetic resonance imaging in emergency department for ACS, emergency medical services role (therapeutic interventions) in ACS and ejection fraction measures for ACS.

GRADE Methodology

Following a review of several evidence rating and recommendation writing systems, ICSI has made a decision to transition to the Grading of Recommendations Assessment, Development and Evaluation (GRADE) system.

GRADE has advantages over other systems including the current system used by ICSI. Advantages include:

- developed by a widely representative group of international guideline developers;
- explicit and comprehensive criteria for downgrading and upgrading quality of evidence ratings;
- clear separation between quality of evidence and strength of recommendations that includes a transparent process of moving from evidence evaluation to recommendations;
- clear, pragmatic interpretations of strong versus weak recommendations for clinicians, patients and policy-makers;
- explicit acknowledgement of values and preferences; and
- explicit evaluation of the importance of outcomes of alternative management strategies.

This document is in transition to the GRADE methodology

Transition steps incorporating GRADE methodology for this document include the following:

- Priority placed upon available Systematic Reviews in literature searches.
- All existing Class A (RCTs) studies have been considered as high quality evidence unless specified differently by a work group member.
- All existing Class B, C and D studies have been considered as low quality evidence unless specified differently by a work group member.
- All existing Class M and R studies are identified by study design versus assigning a quality of evidence. Refer to Crosswalk between ICSI Evidence Grading System and GRADE.
- All new literature considered by the work group for this revision has been assessed using GRADE methodology.

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Crosswalk between ICSI Evidence Grading System and GRADE

ICSI GRADE System	Previous ICSI System	
High, if no limitation	**Class A:**	Randomized, controlled trial
Low	**Class B:**	[observational] Cohort study
	Class C:	[observational] Non-randomized trial with concurrent or historical controls
Low		Case-control study
Low		Population-based descriptive study
***Low**		Study of sensitivity and specificity of a diagnostic test
* Following individual study review, may be elevated to Moderate or High depending upon study design		
	Class D:	[observational]
Low		Cross-sectional study Case series Case report
Meta-analysis	**Class M:**	Meta-analysis
Systematic Review		Systematic review
Decision Analysis		Decision analysis
Cost-Effectiveness Analysis		Cost-effectiveness analysis
Low	**Class R:**	Consensus statement
Low		Consensus report
Low		Narrative review
Guideline	**Class R:**	Guideline
Low	**Class X:**	Medical opinion

Evidence Definitions:

High Quality Evidence = Further research is very unlikely to change our confidence in the estimate of effect.

Moderate Quality Evidence = Further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate.

Low Quality Evidence = Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate or any estimate of effect is very uncertain.

In addition to evidence that is graded and used to formulate recommendations, additional pieces of literature will be used to inform the reader of other topics of interest. This literature is not given an evidence grade and is instead identified as a **Reference** throughout the document.

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Foreword

Introduction

Studies by the National Heart, Lung, and Blood Institute estimate that each year approximately 785,000 Americans will have a new coronary attack and approximately 470,000 will have a recurrent attack *(Lloyd-Jones, 2010 [Low Quality Evidence])*.

With recent evidence-based changes in both interventional percutaneous coronary intervention (PCI) and pharmacological interventions in patients with acute coronary syndromes (ACS), the Global Registry of Acute Coronary Events (GRACE) found that in the United States, adhering to these new changes, rates of in-hospital death, cardiogenic shock, and new myocardial infarctions in patients with non-elevation myocardial infarction (non-STEMI) events have significantly decreased. Similarly, in the STEMI population there has been a significant decrease in rates of in-hospital death, cardiogenic shock, heart failure and pulmonary edema *(Lloyd-Jones, 2010 [Low Quality Evidence])*.

The National Quality Improvement Initiative found that the guidelines and treatments recommended by the American College of Cardiology/American Heart Association (ACC/AHA) were only followed 74% of the time in 350 of the U.S. hospitals it studied. Not adhering to the ACC/AHA guidelines for recommended care of patients with ACS/NSTEMI has been associated with increased in-hospital mortality *(Lloyd-Jones, 2010 [Low Quality Evidence])*.

The ICSI guideline for the Diagnosis and Treatment of Chest Pain and Acute Coronary Syndrome (ACS) was developed to aid the clinician and institutions to provide the most recent evidence-based guideline for a patient who presents with ACS. This guideline focuses mainly on the treatment of acute coronary syndromes, but the algorithms also address the possibility of other cardiovascular causes of chest pain that are life threatening and would require different treatment.

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Scope and Target Population

Adults presenting with past or present symptoms of chest pain/discomfort and/or indications of acute cardiovascular syndromes.

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Aims

1. Increase the success of emergency intervention for patients with chest pain symptoms suggestive of serious illness. *(Annotations #1, 2, 4, 5, 6, 20, 22)*
2. Minimize the delay in administering fibrinolysis or angioplasty to patients with acute myocardial infarction (AMI). *(Annotations #55, 58)*
3. Increase the timely initiation of treatment to reduce postinfarction mortality in patients with acute myocardial infarction. *(Annotations #20, 42, 44, 46, 55, 58, 65)*
4. Increase the percentage of patients with acute myocardial infarction using cardiac rehabilitation. *(Annotations #76, 78)*

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Clinical Highlights

- On initial contact with the health care system, high-risk patients need to be identified quickly and referred to an emergency department via the 911 system. *(Annotations #1, 2, 4, 5, 6; Aim #1)*
- Patients whose chest pain symptoms are suggestive of serious illness need immediate assessment in a monitored area and early therapy to include an immediate EKG, intravenous access, oxygen, aspirin and other appropriate medical therapies. *(Annotations #20, 30; Aims #1, 3)*
- Triage and management of patients with chest pain and unstable angina should be based on a validated risk assessment system and clinical findings. *(Annotation #39)*
- Patients with low-risk symptoms could be evaluated as outpatients. *(Annotations #39, 49, 50)*
- Thrombolysis for ST-elevation, MI or left bundle branch block should be instituted within 30 to 60 minutes of arrival, or angiogram/primary percutaneous coronary intervention should be performed within 90 minutes of arrival, with a target of less than 60 minutes. High-risk patients initially treated at non-PCI-capable facilities who cannot be transferred for PCI within 90 minutes should receive thrombolysis followed by as-soon-as-possible transfer to a PCI-capable facility. *(Annotations #54, 55, 58, 59, 60, 61; Aim #2)*
- Recommend use of the following medications: P2Y12 inhibitor and aspirin (or P2Y12 inhibitor alone if aspirin allergic) at admission. Avoid P2Y12 inhibitor if cardiac surgery is anticipated. Use beta-blockers whenever possible and/or ACE inhibitors/angiotensin receptor blockers at 24 hours if stable, nitrates (when indicated), and statins whenever possible. *(Annotations #22, 65, 67; Aim #3)*
- Recommend use of cardiac rehabilitation. *(Annotations #76, 78)*

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Implementation Recommendation Highlights

The following system changes were identified by the guideline work group as key strategies for health care systems to incorporate in support of the implementation of this guideline.

- Clinics should have a process in place for a patient to be referred for emergency intervention via 911, or be seen in the clinic the same day, within 72 hours, or as an elective clinic evaluation based upon the presence of high-risk symptoms and duration.
- Hospitals should develop and implement emergency department critical pathways and consider standard orders to accomplish rapid evaluation and treatment of acute coronary syndrome. Standard discharge orders/instructions should also be considered.
- A process should be in place for the patient and family that will rapidly orient them to the suspected diagnosis, emergency department and coronary care unit process and other treatment measures to be considered. This could include caregiver face-to-face interactions with the patient and family, as well as teaching tools in written form.
- Institutions that cannot meet the recommended treatment times for primary percutaneous coronary intervention should consider preferential use of intravenous thrombolytic therapy, followed by as-soon-as-possible transfer to a PCI-capable facility for high-risk patients. Lower-risk patients may be observed at the initial hospital with later transfer for PCI as indicated. These institutions should have a predetermined plan for treating patients who present with contraindications to thrombolytics. Such plans may employ delayed local primary percutaneous coronary intervention or transfer to another institution.

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Related ICSI Scientific Documents

Guidelines

- Antithrombotic Therapy Supplement
- Assessment and Management of Chronic Pain
- Heart Failure in Adults
- Hypertension Diagnosis and Treatment
- Lipid Management in Adults
- Major Depression in Adults in Primary Care
- Stable Coronary Artery Disease

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Algorithm Annotations

Chest Pain Screening Algorithm Annotations

1. Initial Contact with Complaint of "Chest Pain/Discomfort" in Person or via Telephone

Initial presentation may be in person or on the phone, etc.

Definitions:

Chest: Upper abdomen, chest, upper back, throat, jaw, shoulders, upper arms

Pain: "Discomfort" or other abnormal sensation such as "gas," "indigestion," "fullness," "pressure," "tightness" or "heaviness"

(Hutter, 2000 [Low Quality Evidence])

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2. Initial Evaluation by Triage Indicates Elevated Risk?

Triage should move patients with suspicious symptoms forward (especially diabetic and middle-aged or older) to immediate electrocardiogram and prompt clinician assessment (with expedited cardiac enzymes if appropriate). Triage staff should be systematically trained to recognize chest pain and cardiovascular risk indicators.

Reception and other staff should bring all complaints of chest pain and breathlessness to medical personnel for further evaluation, especially when:

- patient is currently having symptoms,
- interviewer senses distress,
- symptoms have been present for less than eight weeks (or are getting worse),
- patient feels the pain was at least moderate,
- other symptoms of ill health are present (e.g., shortness of breath, weakness, sweating, nausea), and
- patient requests an immediate opportunity to discuss the symptoms with medical personnel.

(Klinkman, 1994 [Low Quality Evidence]; Buntinx, 1991 [Low Quality Evidence])

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4. Brief Screening History by Medical Personnel

Angina, typical angina, atypical angina, atypical chest pain and non-cardiac chest pain are not consistently defined and used in medical practice. Sometimes they are used to describe a symptom complex; at other times they are used to describe an etiology. For the purposes of this guideline, the following definitions will be used to categorize the patient's chest pain or discomfort as a symptom complex and not an etiology:

Typical angina – pain or discomfort that is 1) substernal, 2) provoked by exercise and/or emotion, and 3) relieved by rest and/or nitroglycerin

Atypical angina – pain or discomfort that has two of the three features listed for typical angina

Non-anginal chest pain – pain or discomfort that has one or none of the three features listed for typical angina

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It should be emphasized that patients with non-anginal chest pain may still be at risk for acute myocardial infarction or acute coronary syndrome. Several serious illnesses are included in the differential diagnosis of chest pain. Assessment of these illnesses requires office or emergency department evaluation. The initial phone interview is limited to determining the timing and location of the initial office or emergency department evaluation.

The risk of immediate adverse outcome is a function of the time course of the chest pain. If the symptoms have been stable for more than two weeks, the risk of an immediate adverse outcome is low. The phone history should stress symptoms suggestive of life-threatening illnesses and the time course of the symptoms.

High-Risk Symptoms

Symptoms suggestive of a high risk of immediate adverse outcome include, but are not limited to:

- severe or ongoing pain,
- pain lasting 20 minutes or more,
- new pain at rest or with minimal activity,
- severe dyspnea, and
- loss of consciousness.

(Jneid, 2012 [Guideline])

The interviewer may use his/her discretion with respect to the need to obtain further history for such symptoms or to refer to a physician.

All patients with high-risk chest pain symptoms should be instructed on the proper use of 911.

The interviewer must use his or her judgment. This guideline focuses on serious complaints that the interviewer feels may signify a serious illness. Chest pain that is not high risk in the judgment of the interviewer (e.g., a young person with chest wall pain) may be evaluated in the office.

Teach medical triage personnel to appropriately conduct the brief screening history.

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6. High-Risk Symptom(s) Present within Last Two Days

Patients who have had high-risk symptom(s) within the previous two days are at the highest risk and should enter the 911 system. The interviewer may judge the need for ambulance transport and office or emergency department evaluation for patients who call hours or days after transient symptoms resolve.

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8. High-Risk Symptom(s) Present between Three Days and Two Weeks

Patients who have had high-risk symptom(s) within the previous two weeks but not the previous two days may be safely evaluated in either a properly equipped office or the emergency department.

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Emergency Intervention Algorithm Annotations

19. Ambulance Transport to Emergency Department, Obtain Electrocardiogram en Route If Able, Aspirin If Possible

A patient complaining of chest pain suggestive of serious etiology should be transported via ambulance with advanced cardiac life support capabilities whether he/she is being transported from home or outpatient clinic to the emergency department.

Systems exist that allow 12 lead EKGs to be obtained by ambulance personnel en route to the ED. If available, this should be done and the EKG transmitted to the ED physician. This may allow more rapid identification of patients with ST-elevation myocardial infarction (STEMI), hastening definitive management. Similarly, if not given prior to the arrival of the ambulance personnel, 324 mg chewable aspirin should be given to patients who do not have a serious allergy.

Patients who are critically ill or unstable should be taken to a hospital capable of performing cardiac catheterization and cardiac surgery unless this would lead to excessive transport time. Plans for triage of a critically ill patient to a tertiary care institution should be part of every community hospital plan.

If a patient is seen in a clinic or physician's office complaining of chest pain suggesting a serious condition, the patient must be transported to the emergency department as soon as possible. Attempts should be made to stabilize the patient as well as possible prior to transport. The referring physician must call the receiving physician and send copies of all medical records pertaining to the current illness.

(de Fillipi, 2000 [Low Quality Evidence]; American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures, 1990 [Guideline])

Each community should develop a ST-elevation myocardial infarction (STEMI) protocol for care that includes a process for prehospital identification and activation, destination protocols for STEMI receiving centers, and ongoing multidisciplinary team meetings involving emergency medical services personnel, representatives from non-percutaneous coronary intervention (PCI) capable hospitals and referral centers, and representatives from PCI capable hospitals to evaluate data, discuss outcomes, and work on quality improvement.

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20. On Arrival in Emergency Department, Immediate Monitoring, Oxygen, IV Access, Cardiac Markers, Portable Chest X-Ray

Recommendations:

- Cardiac markers, such as troponin I or T should be evaluated on arrival *(Dierks, 2012 [Low Quality Evidence]; Keller, 2011 [Low Quality Evidence])*.

On arrival in the emergency department, an immediate electrocardiogram should be obtained if not previously done. A loading dose of 324 mg aspirin should be given, preferably chewed, if not received pre-hospital (for palatability, consider using four 81 mg chewable tablets). Oxygen should be started via nasal cannula, cardiac monitoring initiated and the treating physician called for.

The emergency department physician should also be called to the patient's bedside urgently. On arrival, the physician should perform a brief initial assessment based on vitals, brief historical information, and physical examination. Institution of stabilizing therapy (including chewable aspirin, nitroglycerin and morphine for suspect anginal pain) prior to completing history or physical is appropriate and as often necessary at this level.

Troponin I or T have proven to be very sensitive and specific for myocardial injury, as well as predictive of short-term risk for myocardial infarction or death *(Waxman, 2006 [Low Quality Evidence])*. A newer highly

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sensitive Troponin I assay (hsTnl) has been adopted by many hospitals. While both conventional troponin and highly sensitive assays show similar sensitivity and specificity, hsTnI has been shown to have the best performance characteristics *(Keller, 2011 [Low Quality Evidence])*. Creatine kinase-MB should no longer be used as the primary marker for myocardial infarction.

The use of troponin can present diagnostic challenges in subgroups of patients where it may be chronically elevated or when the initial troponin measure and a subsequent measure both reflect tiny elevations of the biomarker in the setting of non-ischemic cardiac conditions. As with any diagnostic test, the interpretation of an abnormal serum troponin is dependent upon the clinical setting in which the myocardial injury occurred. It is appropriate to measure serial troponin values on arrival and after at least three hours of observation. A diagnosis of acute coronary syndrome can be established when the change in troponin value is significant in the appropriate clinical setting. Significant change is generally considered a second troponin greater than the 99th percentile *(Keller, 2011 [Low Quality Evidence])*.

Other diagnostic testing such as brain natriuretic peptide and chest x-ray may add value to the patient evaluation.

(deFillipi, 2000 [Low Quality Evidence]; American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures, 1990 [Guideline])

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21. Electrocardiogram Positive for ST-Segment Elevation?

The recognition of coronary artery disease and evaluation of its severity cannot be adequately carried out without an electrocardiogram. The early performance of an electrocardiogram following arrival at the emergency department is therefore critical. When patients have new or typical chest pain presumably new ST-segment elevation of greater than 1 mm in two or more contiguous limb leads, or equal to 2 mm or more in precordial leads, they should be considered to have acute myocardial infarction. Patients with new or presumably new left bundle branch block should be treated similarly to those with ST-segment elevation. Although some patients with left bundle branch block will prove not to have acute myocardial infarction, thrombolytic therapy of patients with left bundle branch block is nevertheless associated with a reduction in patient mortality.

Large studies establish the high positive predictive value of new ST-segment elevation, which has been subsequently used for entry in a number of very large clinical trials *(GUSTO Investigators, The, 1993 [High Quality Evidence])*.

The mortality benefit of acute thrombolytic reperfusion therapy has been firmly established in such patients. Pooled data from the available large trials have also demonstrated that patients with left bundle branch block have a significant reduction in mortality with thrombolytic therapy *(ISIS-2 Collaborative Group, 1988 [High Quality Evidence])*.

It should be recognized that not all patients with left bundle branch block will in fact have myocardial infarction. Their apparent mortality advantage with thrombolytic therapy reflects the very high risk of those patients with left bundle branch block who do have acute infarction *(Wang, 2003 [Low Quality Evidence]; Rude, 1983 [Low Quality Evidence])*.

Regardless of ST-segment elevation, consider cardiology consultation early.

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22. Early Therapy

Recommendations:

- Immediately treat with aspirin and P2Y12 inhibitor loading dose and oxygen to keep saturations > 90% and keep patient in a monitored area of the emergency department where critical care interventions can be completed.

Aspirin reduces mortality, reinfarction and stroke, and the addition of UFH, LMWH or Xa inhibitors to aspirin improves outcomes (decrease risk of death and MI). In eligible patients, beta-blockers reduce mortality, reinfarction and stroke. Although no mortality benefit has been shown with the use of nitroglycerin, it is still appropriate for relief of ischemic pain *(Jneid, 2012 [Guideline])*.

Therapy for acute myocardial infarction has been the subject of multiple large randomized trials, many with a primary endpoint of patient mortality. Clinicians caring for patients with acute myocardial infarction should be familiar with the available definitive evidence.

(Antman, 2004 [Guideline]; ISIS-4 Collaborative Group, 1995 [High Quality Evidence]; Lau, 1992 [Meta-analysis]; Saketkhou, 1997 [Low Quality Evidence])

P2Y12 inhibitors

For STEMI and primary PCI, a loading dose of a P2Y12 inhibitor should be given as soon as possible or before the PCI. **

For STEMI patients undergoing non-primary PCI, the following regimens are recommended:

- If the patient has received fibrinolytic therapy and has been given clopidogrel, clopidogrel should be continued as the P2Y12 inhibitor of choice.
- If the patient received fibrinolytic therapy without a P2Y12 inhibitor, a loading dose of clopidogrel should be given as the P2Y12 inhibitor of choice.
- If the patient did not receive fibrinolytic therapy, either a loading dose of clopidogrel or ticagrelor should be given, or once the coronary anatomy is known and PCI is planned, a loading dose of prasugrel should be given promptly and no later than one hour after PCI.

** Prasugrel is not recommended to be used in patients with a prior history of stroke or transient ischemic attack (TIA), or who are > 75 years of age due to increased risk of bleeding except in high-risk situations (diabetes mellitus or prior MI history).

If the patient requires revascularization and a CABG is planned, it is recommended to withhold clopidogrel and ticagrelor for at least five days and prasugrel for at least seven days if possible to decrease the risk of excess bleeding.

In March 2010, the Food and Drug Administration issued a new boxed warning to the product label of clopidogrel bisulfate to warn about patients who do not effectively metabolize the drug and therefore may not receive the full benefits of the drug. Specifically, the purpose is to:

- warn about reduced effectiveness in patients who are poor metabolizers of clopidogrel – poor metabolizers do not effectively convert clopidogrel to its active form in the body;
- inform health care professionals that tests are available to identify genetic differences in CYP2C19 function; and
- advise health care professionals to consider use of other anti-platelet medications or alternative dosing strategies for clopidogrel bisulfate in patients identified as poor metabolizers.

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(http://www.fda.gov/Safety/MedWatch/SafetyInformation/SafetyAlertsforHumanMedicalProducts/ucm204256.htm last accessed October 10, 2012)

In response to both Food and Drug Administration warnings (2009 and 2010), the American College of Cardiology issued statements pointing to the lack of definitive data to guide endorsement of a specific treatment strategy, noting that clinical trials are currently under way to help address the matter.

Anticoagulants

In STEMI patients undergoing primary PCI, intravenous UFH should be started before and continued throughout to maintain therapeutic Activated Clotting Time (ACT) levels. Bivalrudin may be an alternative anticoagulant during the PCI, especially if the patient is at high risk for bleeding *(Kumar, 2010 [Low Quality Evidence])*.

Data supports the use of LMWH, or the Xa inhibitor fondaparinux as alternatives to intravenous UFH.

In high-risk patients, early administration of subcutaneous low-molecular-weight heparin or intravenous unfractionated heparin with aspirin and/or P2Y12 inhibitor is associated with a decrease in the incidence of acute myocardial infarction and ischemia.

Enoxaparin has been shown to have a moderate benefit over intravenous UFH in decreasing the rate of death, myocardial infarction and recurrent ischemia. A meta-analysis of two trials showed a statistically significant reduction by 20% in the rate of death and myocardial infarction *(Cohen, 1997 [High Quality Evidence])*.

Enoxaparin should be used with caution in patients with renal insufficiency.

Switching patients from unfractionated heparin to enoxaparin or vice versa at the time of referral to tertiary care institutions has been shown to increase adverse events. Hence, start and maintain the patient on a single drug continuously during transfer and treatment at referring and referral institutions *(SYNERGY Trial Investigators, The, 2004 [High Quality Evidence])*.

Fondaparinux, a selective Xa inhibitor, may also be used. Maintenance dosing with fondaparinux should be continued the duration of the hospitalization, up to eight days *(Fifth Organization to Assess Strategies in Acute Ischemic Syndromes Investigators, The, 2006 [High Quality Evidence])*. Fondaparinux appears to reduce all-cause mortality at 30 days with the effect becoming more significant at 180 days when compared to UFH and LMWH in ACS. It has been associated with a decreased risk of major and minor bleeding when compared to enoxaparin *(Brito, 2011 [Systematic Review])*.

Due to risk of catheter thrombosis, do not use fondaparinux as the sole anticoagulant to support PCI. Administer an additional anticoagulant with anti-IIa activity (UFH, bivalirudin, argatroban).

Fondaparinux has a long elimination half-life, and there is no antidote for reversal; therefore, patients who may require rapid reversal are not candidates for this therapy. Fondaparinux is contraindicated in patients with a CrCl < 30 mL/min.

For additional information about LMWH/fondaparinux or heparin-induced thrombocytopenia, please refer to the ICSI Antithrombotic Therapy Supplement.

Beta-blockers

Beta-blockers have been a cornerstone of acute coronary syndrome therapy. The Chinese Cardiac Study 2 (CCS2), also called the Clopidogrel and Metoprolol for Myocardial Infarction Trial (COMMIT), demonstrated no overall benefit from early administration of intravenous metoprolol in ST-elevation myocardial infarction patients receiving medical therapy +/- thrombolysis *(COMMIT, 2005a [High Quality Evidence])*. In this population with no primary or delayed angioplasty, post-hoc analysis revealed a survival advantage with reduced ventricular tachycardia/ventricular fibrillation if the presenting systolic blood pressure was over 120 mmHg, no benefit if the blood pressure was 100-120 mmHg, and significant mortality attributed to the

development of cardiogenic shock if the blood pressure was under 100 mmHg. Exercise caution in administering intravenous beta-blocker until after revascularization and stabilization of the patient's blood pressure. Avoid intravenous beta-blockers in Killip III/IV patients. Hypertensive and tachycardic patients may benefit from early ancillary intravenous beta-blocker therapy *(Agency for Healthcare Research and Quality, 2005 [Low Quality Evidence])*. Beta-blocker therapy remains indicated for non ST-segment elevation myocardial infarction and unstable angina unless hypotension, shock, heart block or other contraindication is present.

Initiate beta-blockers early, in the absence of any contraindications. In high-risk patients, they should be given initially intravenously, followed by the oral route with a goal target resting heart rate of 50-60 beats per minute. Patients with low to intermediate risk may start out with oral therapy. The duration of benefit is uncertain. A meta-analysis of double-blinded randomized trials in patients with evolving myocardial infarction showed a 13% reduction in risk progression to acute myocardial infarction. Other multiple randomized trials in coronary artery disease patients have shown a decrease in mortality and/or morbidity rates.

Beta-blockers should be used in most patients with ST-segment elevation myocardial infarction. They remain underutilized in patients with chronic obstructive pulmonary disease and diabetes mellitus where definite benefit has been demonstrated. Patients with low blood pressure or heart failure, or who were recently hemodynamically unstable, should be started on the lowest dose. Caution should be used in patients with reactive airway disease. A cardioselective beta-blocker such as metoprolol may be preferred in patients with reactive airway disease.

Indications for not starting a beta-blocker are:

- history of intolerance or adverse drug reaction to beta-blockers,
- symptomatic bradycardia or advanced heart block (excluding treatment by pacemaker),
- evidence of fluid overload or volume depletion,
- recent treatment with an intravenous positive inotropic agent (e.g., digoxin, nesiritide and others),
- suspected cocaine ingestion, and

  (Completely avoid beta-blockers in cocaine-induced ST-segment elevation myocardial infarction because there is a risk of exacerbating coronary spasm.)

- cardiogenic shock *(COMMIT, 2005b [High Quality Evidence])*

Consider intravenous esmolol if concerned about potential adverse effects of beta-blockers.

Nitroglycerin

ISIS-4 and GISSI-3 failed to show a benefit of nitroglycerin on reduction of mortality in acute myocardial infarction.

Nitroglycerin should be given sublingually to relieve ischemic symptoms. If symptoms are ongoing or recurrent despite the administration of intravenous beta-blockers, intravenous nitroglycerin can be initiated.

Nitroglycerin is contraindicated in patients who are hypotensive, have documented severe aortic stenosis, have hypertrophic cardiomyopathy, or who have received sildenafil or vardenafil within the previous 24 hours or tadalafil in the previous 48 hours *(Jneid, 2007 [Guideline])*.

GP IIb/IIIa Inhibitors

GP IIb/IIIa inhibitors may be started at the time of primary PCI but the usefulness in starting prior to the catheterization laboratory is uncertain.

Contraindications to GP IIb/IIIa inhibitors include active or recent bleeding in the last 30 days, history of intracranial hemorrhage, recent stroke in previous 30 days, uncontrolled hypertension (greater than 200/100

mmHg), major surgery within the previous six weeks, aortic dissection, acute pericarditis, or platelet count less than 100,000 mm^3 (eptifibatide is contraindicated in patients who are dialysis dependent).

(Antman, 2004 [Guideline]; GUSTO IV-ACS Investigators, The, 2001 [High Quality Evidence]; Bhatt, 2000 [Moderate Quality Evidence])

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24. Vital Signs Compromised?

In the critically ill patient whose vitals are compromised (i.e., cardiac arrest, tachyarrhythmias, severe bradycardia, shock or hypotension), the Advanced Cardiac Life Support guideline developed by the American Heart Association should be followed *(deFillipi, 2000 [Low Quality Evidence]; American College of Cardiology/ American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures, 1990 [Guideline])*.

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25. Initiate Advanced Cardiac Life-Support Protocols

The American Heart Association Advanced Cardiac Life Support guideline provides the most recent protocols for initial management of patients whose vital signs are compromised.

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26. Symptoms Suggest Possibility of Acute Cardiovascular Syndrome?

The symptoms that suggest acute coronary syndrome are, in order of importance:

1. chest pain description (See Annotation #4, "Brief Screening History by Medical Personnel");
2. history or evidence of ischemic heart disease;
3. age, gender and comorbidities (atypical presentation in female, elderly and diabetic); and
4. presence of cardiac risk factors.

The description of the patient's chest pain or discomfort is the most critical part of the history. Although multiple other features of the chest pain may be incorporated into an experienced clinician's judgment, the clinician should ultimately attempt to classify the patient as having typical angina, atypical angina or non-anginal chest pain as described in Annotation #4, "Brief Screening History by Medical Personnel," of the Chest Pain Screening algorithm.

Additionally, clinicians should be alert for signs, symptoms and medical history that suggest other serious acute cardiovascular syndromes where prompt intervention is necessary such as aortic dissection and cardiac tamponade.

These may include:

- Abrupt or instantaneous onset
- Pain that is severe in intensity
- Pain that has a ripping, tearing, stabbing or sharp quality
- Chest pain accompanied by back or abdominal pain
- Pain worsened with inspiration, coughing, position changes or swallowing
- Tachypnea or severe shortness of breath

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- Focal neurologic deficit
- New murmur consistent with aortic regurgitation
- Pericardial friction rub
- Connective tissue disease such as Marfan syndrome
- Known aortic valve disease
- Known thoracic or aortic aneurysm
- Recent myocardial infarction
- Recent cardiovascular procedure

(Hiratzka, 2010 [Guideline]; Schwartz, 1992a [Low Quality Evidence])

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27. Chest Pain Not Related to Acute Cardiovascular Syndrome but Indicative of Other Serious Diagnosis?

Pulmonary embolus, expanding pneumothorax or serious gastrointestinal pathology are all potentially life threatening and may closely mimic presentations of an acute coronary syndrome. Further, the presence or absence of reproductible chest wall pain does not preclude the possibility of a more serious underlying cause.

In evaluating a patient with chest pain, it is important to keep in mind the entire differential diagnosis, including non-cardiac causes. Missed or misdiagnosis may have serious implications, both in regards to medico-legal issues and resource utilization.

(Schwartz, 1992a [Low Quality Evidence])

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30. Expedited Aortic Imaging

- Computerized tomography angiogram is generally the quickest and most readily available diagnostic test.
- Trans-esophageal echocardiogram with a biplane probe is equally diagnostic and preferable in patients with renal insufficiency or allergy to contrast dye.
- Magnetic resonance imaging remains the most accurate test but requires a stable patient. Magnetic resonance imaging should be avoided if a type A dissection is suspected.

A careful comparison of magnetic resonance imaging and transesophageal echocardiography has been published elsewhere *(Cigarroa, 1993 [Low Quality Evidence]; Nienaber, 1993 [Low Quality Evidence])*.

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31. Any High-Risk Features for Aortic Dissection?

- Clinical findings of ischemia involving several organ systems
- Pain typically "tearing" or "ripping"
- Pain radiation from chest to back, hips and lower extremities
- Common findings: hypertension, cardiac murmurs, systolic bruits, diminished or absent pulses
- Chest x-ray – abnormalities around aortic knob, increased diameter of ascending aorta
- Blood pressure discrepancy between right and left arm

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32. Cardiovascular Surgical Consultation

- Surgical intervention for symptomatic thoracic aneurysms and proximal (type A; ascending aorta) dissections *(DeSanctis, 1987 [Low Quality Evidence])*
- Control blood pressure with esmolol or labetolol drips +/- nitroprusside

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33. Aortic Dissection Present?

The imaging procedure should establish the presence or absence of an aneurysm and the presence or absence, and location, of a dissection.

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34. Definitive Management

- Distal (type B; distal to left subclavian artery) aortic dissections generally appropriate for pharmacological therapy
  - IV beta-blocker (esmolol or labetolol) +/- nitroprusside to control heart rate and blood pressure along with appropriate pain management
  - Consider surgery if therapy not effective

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35. Symptoms, Signs Suggest Pericardial Disease?

- Chest pain worsened with inspiration, coughing, position changes or swallowing
- Pericardial friction rub
- Electrocardiogram – ST-T changes
- Etiology – infectious, neoplastic, metabolic, inflammatory autoimmune disorders, post-myocardial infarction (Dressler's syndrome)
- Drug related – hydralazine, procainamide, isoniazid, phenytoin, doxorubicin
- Consider blunt trauma, postop

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36. Tamponade?

- Chest pressure and shortness of breath
- Exam – elevated jugular venous pressure, hypotension, tachnypnea, narrow pulse pressure, pulsus paradoxus greater than 20 mmHg
- Electrocardiogram may reveal electrical alternans
- Chest x-ray – normal or enlarged cardiac silhouette
- Echocardiogram diagnostic test of choice
- Pericardial space typically contains 50 cc of fluid, with chronic accumulation may contain up to 2,000 cc
- With acute, rapid accumulation, overt tamponade may develop with as little as 150 cc

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37. Pericardiocentesis – Prefer Echocardiogram Directed

- Echocardiogram-directed apical pericardiocentesis procedure of choice
- Subxyphoid approach if echocardiogram not available and patient unstable

(Kopecky, 1986 [Low Quality Evidence]; Callahan, 1985 [Low Quality Evidence]; Callahan, 1983 [Low Quality Evidence])

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38. Admit Critical Care Unit/Monitored Bed

The patient should be observed in a critical care unit/monitored bed setting.

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39. Risk Assessment/Consider Repeat Electrocardiogram If Ongoing Chest Discomfort

Recommendations:

- The patients with suspected ACS should undergo early and follow-up risk assessment based on history, exam, EKGs, biomarkers (quality of evidence high, recommendations strong), *(Eagle, 2004 [Low Quality Evidence]; Antman, 2000 [Low Quality Evidence]; Jneid, 2012 [Guideline])*.
- Serial electrocardiograms are indicated at 15-30 minute intervals if suspicion for ACS is high, the patient has ongoing chest discomfort and original EKG is non-diagnostic.

There are a variety of risk assessment criteria for patients presenting with chest pain and suspected acute coronary syndrome. This section will focus on risk assessment for chest pain symptoms and subsequent risk assessment for those with suspected or documented acute coronary syndrome.

For patients with continuing chest discomfort highly suggestive of ACS, serial EKGs, initially at 15-30 minute intervals, should be strongly considered to evaluate for development of ST shift or other evolution. There is a wealth of evidence that any dynamic EKG changes portend both diagnostic and prognostic value in evaluation and treatment of patients with suspected ACS. In addition, dynamic EKGs call for more aggressive medical therapy and timing of invasive coronary evaluation in the emergency department for possible evolving STEMI not seen on initial EKG.

Patients who are deemed low risk by ACCF, AHA guideline criteria may be safely evaluated as outpatients. These will include some patients with mild symptoms, which may reflect non-compliance with medications, increasing activity, emotional stress or other exacerbating factors. Patients with a low likelihood of coronary artery disease on the basis of chest pain description, age, gender and risk factor assessment, and patients at intermediate likelihood may be initially medically treated and risk stratified with non-invasive testing.

Patients who are intermediate risk by the guideline criteria need definitive emergency department assessment and may be most suitable for admission to an observation or chest pain unit. Patients with intermediate risk symptoms should undergo risk stratification with assessment of cardiac biomarkers, repetitive electrocardiograph assessment, and ultimately cardiac imaging and stress testing.

Patients who fulfill high-risk criteria should be admitted to the hospital, and treated with aggressive medical therapy and early invasive coronary evaluation. A large number of studies have confirmed this risk, and support a strategy of hospitalization and subsequent risk assessment for acute coronary syndrome *(Jneid, 2012 [Guideline]; Antman, 2008 [Guideline]; Eagle, 2004 [Low Quality Evidence]; Farkouh, 1998 [High Quality Evidence])*.

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Complete certainty of the etiology of a patient's chest pain is difficult to achieve by an evaluation and commonly cannot be attained in the emergency department. It is therefore vitally important to assess risk in order to safely and yet cost effectively triage chest pain patients.

Diagnosis and risk assessment of patients with suspected ACS is a continuous process that includes clinical observation for recurrent symptoms, hemodynamic features, dynamic EKG and biomarkers data.

For patients with unstable coronary artery disease and/or an acute coronary syndrome, it is important to use objective risk assessment criteria for purposes of triage (critical care unit, monitored bed or immediate catheterization lab referral). There are a number of ways to risk stratify patients with unstable coronary disease. The initial examination in the emergency department often provides insight into the patient's risk. An astute clinician can often assess risk from the physical examination and laboratory assessment.

A variety of risk factors for short-term risk of death or non-fatal MI have been determined. They include accelerated tempo of ischemic symptoms in the preceding 48 hours, prolonged (greater than 20 minutes) ongoing rest angina, pulmonary congestion, mitral insufficiency, S3 heart sound, hypotension, excessive brady or tachycardia, age over 75, dynamic, even 0.5 mm ST deviation, new LBBB, paroxysmal sustained ventricular tachycardia, and elevated markers of myonecrosis *(Jneid, 2012 [Guideline])*. Many risk calculators are available for health care providers for rapid and easy risk calculation. One of the most popular risk assessment tools – the TIMI (thrombolysis in myocardial infarction) risk score – displayed a potent predictive gradient of short-term risk of about 10 times magnitude between the lowest and the highest risk patients *(Antman, 2000 [Low Quality Evidence])*. Health care providers are encouraged to use this calculator to obtain an objective risk assessment of short-term patient's risk.

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40. Echocardiogram; Discharge?/Consider Treatment

- Pericarditis without tamponade – obtain echocardiogram
- Non-steroidal anti-inflammatory drugs, aspirin or colchicine and close follow-up *(Imazio, 2005 [High Quality Evidence])*

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41. High Risk

High-risk ACS patients, as determined by TIMI risk score of equal or higher than three, require a high level of care with close monitoring and intravenous access for the initiation of intravenous medications including UFH/LMWH, beta-blockers and nitroglycerin. These therapies should be started in the emergency department setting. Hospitalization usually requires an intensive care unit setting or competent nursing in a monitored bed setting *(Jneid, 2012 [Guideline])*.

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42. Early Therapy for High-Risk Patients

See Annotation #22, "Early Therapy," for general recommendations for aspirin, P2Y12 inhibitors, nitrates, oxygen, beta-blockers, anticoagulation and GP IIb/IIIa inhibitors.

An early invasive strategy involves diagnostic catheterization within 24 to 48 hours, followed by percutaneous coronary intervention (PCI) or coronary artery bypass graft if warranted.

For patients where an **initial invasive strategy** is planned, it is recommended to give a loading dose of a clopidogrel or prasugrel as soon as possible prior to PCI, in addition to aspirin. If clopidogrel is not given before PCI, prasugrel or ticagrelor may be added at the time of PCI. Cardiology may elect to add a GP IIb/IIIa inhibitor in some patients. Treatment with a P2Y12 inhibitor should be continued for 12 months unless contraindicated. If the risk of bleeding outweighs the benefit, early discontinuation of P2Y12 inhibitor therapy may be considered specifically in patients not treated with an implantation of a drug-eluting

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coronary stent. A GP IIb/IIIa inhibitor may be omitted if the patient has been on bivalirudin as the anticoagulant and P2Y12 inhibitor has been given at least six hours prior to the PCI *(Vandvik, 2012 [Guideline]; Wright, 2011 [Guideline])*.

If an **initial non-invasive (conservative) strategy** is planned, a loading dose of P2Y12 inhibitor (clopidogrel or ticagrelor) should be given in addition to aspirin as soon as possible. The P2Y12 inhibitor should be continued for at least one month and ideally up to 12 months. A GP IIb/IIIa inhibitor may be added to the regimen of aspirin, P2Y12 inhibitor and anticoagulant therapy before diagnostic angiography in patients who fail initial conservative management (recurrent ischemia/ heart failure/arrhythmias). If coronary disease is found on angiography, patient should be continued on aspirin indefinitely and a P2Y12 inhibitor for up to one year if possible. UFH should be continued for 48 hours or LMWH or fondaparinux should be continued for duration of hospitalization up to a maximum of eight days.

When an **initial conservative management** is planned, GP IIb/IIIa inhibitors may be considered in high-risk UA/NSTEMI patients in addition to aspirin and a P2Y12 inhibitor prior to PCI if the risk of bleeding is low. GP IIb/IIIa inhibitors are not recommended to be added to aspirin and P2Y12 inhibitor if the risk of bleeding is high *(Wright, 2011 [Guideline])*.

If diagnostic angiography is not required in patients initially managed conservatively, the patient should undergo stress testing. If after stress testing the patient is found to be of low risk, the patient should be continued on aspirin indefinitely, and P2Y12 inhibitor should be continued for a minimum of one month and preferably up to one year. UFH should be continued for 48 hours, or LMWH or fondaparinux should be continued for duration of hospitalization up to a maximum of eight days.

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44. Perform Catheterization within 24 to 48 Hours

Recommendation:

- Perform angiography within the first 24 hours for patients diagnosed with high-risk ACS and any patients who are unstable or having recurrent symptoms *(Bavry, 2006 [Systematic Review])*.

An early invasive strategy is beneficial in many patients with non-ST-segment elevation myocardial infarction, or high-risk unstable angina, especially when coupled with aggressive adjunctive medical therapy. Certainly the aggressive anticoagulation and antiplatelet agents should be utilized when there are recurrent symptoms and no ability to proceed to early angiography, such as a weather-related delay or the catheterization lab is not available. However, in patients who become unstable or have recurrent symptoms, the delay of coronary angiography and coronary revascularization should be minimized.

Contraindications to GP IIb/IIIa inhibitors include active or recent bleeding in the last 30 days, history of intracranial hemorrhage, stroke in previous 30 days, uncontrolled hypertension (greater than 200/100 mmHg), major surgery within the previous six weeks, aortic dissection, acute pericarditis or platelet count less than 100,000 mm^3 (eptifibatide is contraindicated in patients who are dialysis dependent) *(Jneid, 2012 [Guideline]; Antman, 2008 [Guideline]; GUSTO IV-ACS Investigators, 2001 [High Quality Evidence])*.

In patients with unstable angina/non-STEMI in whom an initial invasive procedure is selected, it is reasonable to omit upstream GPIIb/IIIa before angiography if bivalirudin is selected as the anticoagulant and a loading dose of P2Y12 inhibitor was administered at least six hours prior to PCI. Bivalirudin alone as compared with heparin plus GPIIb/IIIa has similar rates of major adverse cardiac events, lower minor bleeding complications and similar net adverse cardiac events *(Kumar, 2010 [Moderate Quality Evidence])*.

An analysis of invasive therapy in high-risk, predominantly biomarker positive patients has shown 25% reduction of all-cause mortality and 17% reduction of recurrent non-fatal MI for early invasive therapy compared with conservative therapy at a mean follow-up of two years *(Bavry, 2006 [Systematic Review])*.

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45. Intermediate Risk

It is difficult to predict which patients truly have an acute coronary syndrome after the initial evaluation in the emergency department. As the short-term risk of a significant cardiac event may be 10% *(Jneid, 2012 [Guideline])*, it is imperative to treat each patient according to protocol during the evaluation process. The work group recommends a standardized approach or critical pathway approach to these patients that strives to fully diagnose and risk stratify. These patients should be observed in the emergency department for at least three hours *(Keller, 2011 [Low Quality Evidence])* or admitted to a chest pain unit or observation unit where serial troponin biomarkers and electrocardiographic assessment can be obtained. It is crucial to perform serial clinical reassessments during the observation period to determine if the symptoms have worsened or the initial baseline risk category assessment remains accurate. Many of these patients should undergo cardiac imaging and stress testing assessment, and some may require outpatient referral to a cardiologist for subsequent evaluation and management. It may be appropriate to consider diagnostic catheterization in certain subgroups of these patients *(Jneid, 2012 [Guideline]; Antman, 2004 [Guideline])*.

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46. Early Therapy for Intermediate-Risk Patients

See Annotations #22, "Early Therapy" and Annotation #42, "Early Therapy for High-Risk Patients."

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47. Admit to Chest Pain Unit or Monitored Bed

If the patient's risk assessment is not clearly in a high- or low-risk category, and the institution has an emergency department-based chest pain observation unit, admit to the chest pain unit or a monitored bed. Otherwise, management using a critical pathway for unstable angina with a similar protocol on a monitored bed unit is recommended.

A chest pain unit critical pathway provides monitoring capabilities, a dedicated nurse, serial cardiac markers (markers should be negative for at least three hours from admission to the emergency department and a post-observation stress test prior to final triage decision. Generally, after successful completion of the evaluation, patients can be classified as low risk and safely followed up as outpatients in the next few days. In the case of a positive or indeterminate lab test, electrocardiogram or stress/imaging test, or if there is recurrent chest pain during the observation period, a patient should be considered high risk and managed accordingly.

If a patient requires repeated doses of nitroglycerin and/or intravenous nitroglycerin or paste, or requires beta-blockade for pain control, assess the patient as high risk *(Gibbons, 1997 [Guideline])*.

Refer to Annotation #39, "Risk Assessment/Consider Repeat Electrocardiogram If Ongoing Chest Discomfort," for more information on risk stratification.

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48. Patient Has Positive: Markers? Electrocardiogram Changes? Unstable Dysrhythmias?

If a patient develops recurrent chest discomfort during the observation period, the patient should be considered having failed the observation unit intervention and should be considered high risk. Admit to monitored bed or an intensive care unit setting. If the serial cardiac markers, troponin T or I on the second blood draw are positive, or the patient develops new or dynamic ST-T wave changes, the patient should also be considered high risk. If a patient develops an unstable dysrhythmia (e.g., ventricular tachycardia or multifocal premature ventricular complexes, etc.), he/she should also be considered high risk and admitted.

Most patients in this category will have an uneventful observation period and should undergo an endpoint stress test. This can be done after two negative troponins prior to patient discharge in a patient in whom

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symptoms have resolved, or it can be arranged for within 72 hours after discharge. The choice of a treadmill exercise test utilizing the Bruce treadmill score should be preferred in all patients who can walk and have an interpretable electrocardiogram. In some instances additional imaging may be beneficial. If the patient is unable to walk, a pharmacologic stress test should be considered. Patients needing continued beta-blockade may be candidates for nuclear imaging instead of standard treadmill stress testing. Patients can also be considered for coronary computed tomographic angiography (CCTA).

(Farkouh, 1998 [High Quality Evidence]; Gibler, 1995 [Low Quality Evidence]; Gibler, 1992 [Low Quality Evidence])

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49. Low Risk

Patients with a history of brief episodes of chest pain (less than 20 minutes) but suggestive of accelerating and/or class three or four angina should be considered low risk if indeed an electrocardiogram can be obtained during the chest pain episodes. If, however, an electrocardiogram cannot be obtained during a chest pain episode or other atypical features are present, the patient may be managed as intermediate risk and evaluated in a cardiac observation unit.

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50. Discharge to Outpatient Management

If the diagnosis is low-risk unstable angina, a follow-up appointment, preferably with a cardiologist, should be done. Otherwise, a follow-up with a primary care physician may also be appropriate. These appointments should occur within one to three days. If the chest pain is considered stable angina and non-anginal chest pain, an arrangement for follow-up with a primary care physician should be arranged in the near future. The primary care physician may want to follow the clinical evaluation algorithm provided within this guideline.

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51. Patient has Abnormal Functional Test or Coronary Computed Tomographic Angiography (CCTA)?

If patient has an abnormal stress test without recurrence of signs of acute coronary syndrome, refer him/her as an outpatient for cardiology consultation. If a patient has recurrence of more persistent symptoms at rest or signs of escalating angina, consider him/her to be high risk within this acute coronary syndrome guideline.

Some patients may be anxious for more immediate evaluation by cardiology if they have an abnormal functional stress test or CCTA. Patients can be reassured that outpatient consultation with cardiology is appropriate. Patients should all be counseled to seek immediate evaluation with any recurrence of anginal symptoms *(Antman, 2008 [Guideline])*.

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ST-Elevation Myocardial Infarction (STEMI) Algorithm Annotations

53. ST-Segment Elevation on Electrocardiogram

About 40% of patients with acute myocardial infarction present with ST-segment elevation. They can be treated with thrombolytics or with emergency coronary angiography and percutaneous coronary intervention. Patients presenting with chest pain but no ST-segment elevation may be triaged to the telemetry unit if they are hemodynamically stable and pain-free.

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Acute myocardial infarctions are divided into two categories: those causing ST-segment elevation (transmural) and those not causing ST-segment elevation (non-transmural or subendocardial). Infarctions associated with ST-segment elevations will be positively effected by early thrombolytic therapy. There is no question that if unable to get to a PCI-capable facility < 90 minutes, patients with anterior myocardial infarctions and those who present very early (less than four to six hours after onset of symptoms) benefit tremendously from any thrombolytic agent, and both in-hospital and late mortality are significantly reduced *(Hochman, 1995 [Low Quality Evidence])*.

Facilities without percutaneous coronary intervention capabilities should activate their established processes and criteria for transfer for immediate percutaneous coronary intervention.

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54. Percutaneous Coronary Intervention-Capable Facility?

The major distinction among hospitals in regard to STEMI management is between PCI-capable hospitals, which are STEMI receiving centers, and non-PCI-capable hospitals, which are STEMI-referring centers. Both types of facilities need to develop multidisciplinary STEMI systems of care that include prehospital identification and activation, emergency medical services, transfer and destination protocols *(Kushner, 2009 [Guideline])*.

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55. Emergency Coronary Angiography

Recommendation:

- If experienced interventional cardiologists and rapid deployment of primary percutaneous coronary intervention (PCI) are available, prioritize PCI over thrombolysis, as PCI has been demonstrated to be more effective in opening acutely occluded arteries *(Antman, 2008 [Guideline])*.

Time to open artery is critical to effective primary percutaneous coronary intervention. Current American College of Cardiology/American Heart Association guidelines suggest that institutions wishing to apply primary percutaneous coronary intervention for ST-segment elevated myocardial infarction should achieve a median door-to-balloon time of 90 minutes or less. The ACC/AHA Consensus Panels have set a 60-minute median door-to-balloon time as the benchmark for top-performing institutions *(Jneid, 2007 [Guideline]; Antman, 2008 [Guideline])*.

Institutions that cannot meet the recommended treatment times should consider preferential use of intravenous thrombolytic therapy. *These institutions should have a predetermined plan for treating patients who present with contraindication to thrombolytics*.

Aspirin, anti-platelet/clopidogrel or prasugel UFH/LMWH/fondaparinux, nitrates and beta-blockers should be administered early to these patients, unless contraindicated *(Wiviott, 2007 [High Quality Evidence])*.

Primary percutaneous coronary intervention may also play a role in the treatment of non-ST-segment elevation myocardial infarction/refractory angina pectoris if angina symptoms fail to resolve within an hour of instituting aggressive anti-anginal therapy with aspirin, prasugrel, UFH, LMWH, beta-blockers and GP IIb/IIIa inhibitors, or serial electrocardiogram or echocardiogram suggest a large amount of myocardium at risk.

For centers that have demonstrated high success rates and low complication rates, this strategy is at least equal in efficacy to that of initial thrombolytic therapy, especially for those patients at high risk of mortality, and may be considered in thrombolytic candidates, as well as in patients with thrombolytic contraindications. It is the preferred therapy for cardiogenic shock. Immediate transfer of patients to an institution capable of treating this condition is indicated for the presentation or development of cardiogenic shock *(Berger, 1999 [Low Quality Evidence])*.

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Current American College of Cardiology/American Heart Association guidelines recommend treating the culprit vessel when feasible and deferring surgical- or percutaneous coronary intervention-based revascularization of other vessels until the patient has stabilized and the clinically most appropriate strategy determined.

(GUSTO IIb Investigators, 1997 [High Quality Evidence])

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58. Percutaneous Coronary Intervention (PCI) Available within 90 Minutes?

Recommendations:

- Systems should be in place to transfer patient with STEMI or high-risk features to angiography within 90 minutes wherever possible *(Rathore, 2009 [High Quality Evidence])*.
- When PCI is not available within 90 minutes, employ a strategy of early thrombolytics prior to transfer *(Jneid, 2012 [Guideline])*.

The medical evidence is clear the more rapidly reperfusion is obtained in ACS, the better the outcomes that are obtained. ACC/AHA standards call for door-to-needle time for initiation of fibrinolytic therapy within 30 minutes or that door-to-balloon time for PCI is kept under 90 minutes *(Antman, 2004 [Guideline])*. They state further "because there is not considered to be a threshold effect for the benefit of shorter times to reperfusion, these goals should not be understood as 'ideal' times but the longest times that should be considered acceptable." In the 2009 focused updates, this standard was left in place, although "the writing groups continue to believe the focus should be on developing systems of care to increase the number of patients with timely access to primary PCI rather than extending the acceptable window for door to balloon time." A recent study of 43,801 patients reinforced this, showing a continuous non-linear increase in in-hospital mortality with any delay in reperfusion time *(Rathore, 2009 [High Quality Evidence])*. The practical standard in care should be an "as-soon-as-possible" one.

Whichever strategy – fibrinolytic or PCI – is employed will be affected by both facility and patient factors. However "for facilities that can offer PCI, the literature suggests that this approach is superior to pharmacologic reperfusion." This is primarily due to reduction in the rate of non-fatal recurrent MI. The 22 randomized clinical trials that were reviewed in the ACC/AHA guidelines demonstrated that, compared to fibrinolytics, patients treated with PCI experienced "lower short term mortality rates, less nonfatal reinfarction and less hemorrhagic stroke." However there was an increase in major bleeding risk. The difference was even more pronounced among high-risk patients such as those with cardiogenic shock, severe heart failure or electrical instability.

Patients who are best suited for fibrinolysis alone include "those who present early after symptom with a low bleeding risk." Patients who may benefit the most from immediate transfer for PCI include high-risk patients, those with high bleeding risk and patients presenting more than four hours after symptom onset.

In facilities that do not offer PCI but must transfer the patient to another center, generally fibrinolysis can be offered more quickly. If the transfer cannot be accomplished so that the door-to-balloon time is less than 90 minutes, the differences in outcomes are reduced and fibrinolysis may be preferred *(Antman, 2004 [Guideline])*. Consultation with cardiology at the tertiary care center may be indicated to determine the best strategy in these situations.

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59. High-Risk Patient?

Patients at high-risk of acute MI complications include those with extensive ST-segment elevation; new-onset left bundle branch block; previous MI, Killip class II or III; left ventricular ejection fraction 35% or less; systolic blood pressure less than 100, heart rate > 100 bpm and patients with diabetes.

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60. Thrombolytics Followed by Rapid Transfer to PCI-Capable Facility

Studies have addressed the best management of high-risk patients with acute MI initially treated at non-PCI capable facilities. The CARESS-in-AMI trial *(Di Mario, 2008 [High Quality Evidence])* studied 600 STEMI patients < 75 years with high-risk features. Patients treated with thrombolytics followed by immediate transfer to a PCI facility had a significantly lower primary outcome measure (all-cause mortality, reinfarction and refractory myocardial ischemia) with no increase in major bleeding compared to delayed transfer for rescue PCI as indicated (4.4% versus 10.7%, P=0.004). Similarly, the TRANSFER-AMI trial *(Kushner, 2009 [Guideline])* followed 1,059 patients randomized to either standard rescue PCI strategy or "pharmacointensive" care (thrombolysis followed by immediate transfer for PCI). The primary endpoints of the trial were a 30-day composite of the first occurrence death, reinfarction, recurrent ischemia, new or worsening heart failure, and cardiogenic shock. The pharmacointensive group reached primary endpoints 11.0% of the time, compared to 17.2% of the rescue PCI group (P=0.004).

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61. Thrombolytics

Recommendation:

- Thrombolytics should be administered early in the course of acute myocardial infarction if a cath lab with experienced personnel is not readily available *(Jneid, 2012 [Guideline])*.

Indications for Thrombolytics

- ST-segment elevation of 1 mm or more in two or more contiguous limb leads **or**
  - ST-segment elevation of 2 mm or more in precordial leads, **or**
  - new or presumably new left bundle branch block; ST-segment depression of 2 mm or more in V_1V_2 (true posterior infarction), **and** anginal chest pain between 30 minutes and 12 hours in duration that is unrelieved with sublingual nitroglycerin

(GUSTO IIb Investigators, 1997 [High Quality Evidence]; Weaver, 1997 [Low Quality Evidence]; Gibbons, 1993 [Low Quality Evidence]; Grines, 1993 [High Quality Evidence]; Zijlstra, 1993 [High Quality Evidence]).

Low patient weight has been identified as an ongoing risk factor for significant intracranial hemorrhage (ICH) when thrombolytics are administered. It is imperative to accurately estimate the weight of patients with acute myocardial infarction to determine the proper dose of thrombolytic to minimize the risk of intracranial hemorrhage.

Single-bolus agents, such as tenecteplase, simplify administration; however, patient weight remains important in calculating dose.

The use of any particular thrombolytic agent is very controversial and continuously being reassessed *(Antman, 2004 [Guideline])*. It is recommended that each facility/institution that is non-PCI-capable develop a protocol on a specific thrombolytic agent and dose, and administration prior to transferring the patient to a PCI-capable facility.

A. The earlier thrombolytic therapy is initiated in the course of acute myocardial infarction, the greater the reduction in mortality. Thrombolytics started within one hour of symptoms has been demonstrated to lead to a 47% reduction in mortality *(Simoons, 1993 [Low Quality Evidence])*. Some hospital systems utilize pre-hospital acquisition of 12-lead electrocardiographs and activation of an acute coronary reperfusion team. Such an approach may involve direct notification of the coronary reperfusion team without stopping in an emergency department, or the administration of intravenous lytic therapy in the field pre-hospital, but with the supervision of an emergency department physician. The goals of such

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a system must be to most rapidly initiate and deliver acute reperfusion therapy and establish full coronary patency.

B. Common causes of delay in initiation of thrombolytics include *(Sharkey, 1989 [Low Quality Evidence])*:

1. Patient is not accessing the emergency medical system promptly
2. Failure to obtain an electrocardiogram promptly on the patient's arrival in the emergency department
3. Delay in diagnosis after electrocardiogram has been obtained
4. Delay in delivery of drug once decision is made to initiate therapy

C. Patients who may have a mortality benefit with tPA:

1. Patients with larger myocardial infarctions such as an anterior myocardial infarction or complicated inferior myocardial infarction have slightly lower mortality with tPA *(GUSTO Investigators, The, 1993 [High Quality Evidence])*.
2. Patients with prior coronary artery bypass graft usually have a thrombus in the bypass graft, a larger thrombus burden and a significantly decreased mortality when treated with tPA versus streptokinase *(GUSTO Angiographic Investigators, The, 1993 [High Quality Evidence])*.

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62. Was Thrombolysis Successful at Reperfusion?

Recurrent or ongoing symptoms following treatment with thrombolytics is an indication for PCI. Rescue PCI refers to PCI within 12 hours after failed fibrinolysis for patients with continuing or recurrent myocardial ischemia. Thrombolytic failure may be evident by failure of ST-elevation to resolve within 30 to 60 minutes of thrombolytic therapy and usually includes persistent symptoms. Guidelines for time from arrival to balloon inflation are not established for this complex subset of patients, but rescue percutaneous coronary intervention should be accomplished within 90 to 120 minutes of thrombolytic failure if possible.

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

According to the ACC/ AHA guidelines for patients with ST-elevation myocardial infarction *(Antman, 2004 [Guideline])*:

> Rescue PCI should be performed in patients less than 75 years old with ST-elevation or left bundle branch block (LBBB) who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock, unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care.
>
> Perform rescue PCI in patients with symptom onset within 12 hours and severe CHF and/or pulmonary edema (Killip class III).

Consider rescue PCI for patients with one or more of the following:

a. Hemodynamic or electrical instability

b. Persistent ischemic symptoms

Given the uncertainty surrounding this issue, the work group suggests contemplation of cardiology consultation to aid in properly treating these patients.

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63. Cardiology Consultation for High-Risk Patients (If Not Already Obtained)

Angiography should be performed for patients at increased risk as defined in Emergency Intervention Algorithm, Annotation #39, "Risk Assessment/Consider Repeat Electrocardiogram If Ongoing Chest Discomfort."

Trials (collectively FRISC II and TACTICS-TIMI 18) suggest an early invasive approach.

- Early diagnostic coronary angiography and appropriate percutaneous coronary intervention or coronary artery bypass graft within 48 hours of presentation is recommended if STEMI is present.

Cannon, et al. present data from a clinical trial comparing early invasive and conservative strategies in treatment of acute coronary syndrome. Boden and McKay summarize current approaches in treating acute coronary syndrome, including coronary artery bypass graft (*Boden, 2001 [Low Quality Evidence]; Cannon, 2001 [High Quality Evidence]).*

Consider coronary artery bypass graft for patients with left main, three-vessel or two-vessel disease with left anterior descending coronary artery involvement and demonstration of ischemia or for patients who would not receive the ideal benefit from percutaneous coronary intervention. Pharmacologic or stress test imaging may be helpful if myocardial viability is uncertain and revascularization is considered.

Consider percutaneous coronary intervention for patients with acceptable anatomy in whom its prognostic effect has been most clearly demonstrated: significant residual ischemia, coronary artery bypass graft candidacy and failure of maximal medical therapy (two of three medications) to control angina or contraindications to medications.

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65. Admit to Critical Care Unit

Patients who present with acute ST-segment elevation, hemodynamic instability or both should be considered for admit to the critical care unit. Reconsider the early use of adjunctive medications. Once the issue of surgery is clarified, consider the early use of P2Y12 inhibitor for those in whom percutaneous coronary intervention is planned. (See Emergency Intervention Algorithm.) A critical care unit admission order set is included in this guideline.

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67. Critical Care Unit Care: Chronic Adjunctive Medications/Phase One Cardiac Rehabilitation

A protocol should be in place to guide routine orders for continuous monitoring, oxygen delivery, intravenous access therapy, activity, laboratory and diagnostic tests, diet and medications.

The following medications are recommended:

- **Aspirin** should be continued. Aspirin has been shown to reduce reinfarction and mortality long term and should be continued whenever possible. Use of non-steroidal anti-inflammatory drugs and COX-2 inhibitors may reduce the cardioprotective benefits of aspirin *(U.S. Food and Drug Administration, 2006 [Reference]).*
- **P2Y12 inhibitors (clopidogrel/prasugrel/ticagrelor)** For patients with allergies to aspirin, P2Y12 inhibitors may be used alone and continued indefinitely. For patients undergoing a bare metal stent or drug-eluting stent, a P2Y12 inhibitor should be continued for 12 months.

  For patients who present with unstable angina, non-ST-segment elevation myocardial infarction or ST-segment elevation myocardial infarction without revascularization who are not at high risk for bleeding, clopidogrel/ticagrelor should be continued for at least one year.

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If the morbidity risk due to bleeding outweighs the benefit of P2Y12 inhibitor therapy, early discontinuation should be considered. If a P2Y12 inhibitor is given and a coronary artery bypass surgery is planned, clopidogrel and ticagrelor should be held for at least five days and prasugrel for at least seven days prior to surgery due to increased risk of perioperative bleeding. Prasugrel is not recommended as part of dual antiplatelet therapy in patients with a history of stroke or TIA due to the increased risk of bleeding.

Proton pump inhibitors (PPIs), which are often used in conjunction to reduce the chance of gastrointestinal blood loss, result in reduced plasma concentrations of active metabolite of clopidogrel, thus lowering the antiplatelet effect of clopidogrel in vitro *(Gilard, 2008 [High Quality Evidence])*. This interaction is due to competitive inhibition of the metabolism of clopidogrel by CYP2C19, which generates its active metabolite.

In November 2009 the FDA issued a statement advising prescribers that in patients taking clopidogrel to avoid selected PPIs and other drugs (e.g., cimetidine, esomeprazole, fluoxetine, fluconazole, ketoconazole) that inhibit CYP2C19.

(http://www.fda.gov/Drugs/DrugSafety/PostmarketDrugSafetyInformationforPatientsandProviders/DrugSafetyInformationforHeathcareProfessionals/ucm190787.htm last accessed February 11, 2011)

Though the FDA issued a warning, post-hoc analysis of two recent studies *(Ray, 2010 [Low Quality Evidence]; O'Donoghue, 2009 [Low Quality Evidence])* did not confirm these adverse cardiovascular outcomes. The ACC/AHA issued a statement that suggested that additional clinical studies are needed before a formal recommendation could be made *(Kushner, 2009 [Guideline])*. Additional management guidelines on this topic are being prepared by the ACC/AHA.

The gastroprotective effects of PPIs were demonstrated in results from the COGENT trial *(Bhatt, 2010 [High Quality Evidence])*. In patients requiring dual antiplatelet therapy (clopidogrel and aspirin), the incidence of gastrointestinal (GI) hemorrhage in patients on omeprazole (1.1%) was reduced compared to patients on placebo (2.9%) HR 0.34, 95% CI, 0.18 to 0.16; p<0.001. Although the rate of cardiovascular events in patients on omeprazole was not increased, this study was not powered to detect such a difference.

After a consensus-building discussion, the ICSI Antithrombotic work group recommends:

- Risks and benefits of concomitant clopidogrel and PPI use must be carefully evaluated and documented on an individual patient basis.
- Discontinue PPI if there is no strong indication for one.
- Consider H2 blockers (famotidine, nizatidine and ranitidine).
- Pantoprazole does not inhibit CYP2C19 and is a reasonable option. However, this has not been shown to be significant in clinical trials *(O'Donoghue, 2009 [Low Quality Evidence])*.

- **Beta-blockers*.** Beta-blockers reduce mortality, readmission and reinfarction for both coronary artery disease and congestive heart failure. They should be instituted and/or continued whenever possible. Patients who have clinical contraindications for beta-blockers in the hospital should be reconsidered for beta-blocker therapy after discharge *(Hjalmarson, 2000 [High Quality Evidence])*. Prescribing beta-blockers for acute myocardial infarction patients is a CMS/The Joint Commission quality measure.

  * Shown in large clinical trials to reduce infarction mortality in all myocardial infarctions.

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- **Statins:** Patients diagnosed with ACS should be treated with statins. Statins may reduce recurrent ischemic event after ACS, all-cause mortality and revascularization *(Mills, 2011 [Systematic Review]; Cannon, 2004 [Low Quality Evidence]; Schwartz, 2001 [Moderate Quality Evidence])*.

  Patients should be started on statins regardless of baseline LDL. Higher baseline LDL level at the time of ACS will draw more benefits from statin therapy than lower LDL levels *(Giraldez, 2008 [Low Quality Evidence])*.

  For more information on statins, see the ICSI Lipid Management guideline.

- **ACE inhibitors*.** ACE inhibitors are indicated (angiotensin receptor blockers if ACE inhibitors aren't tolerated – in addition to beta-blockers, when possible) for most patients following acute myocardial infarction to reduce mortality and morbidity associated with large infarcts with significant left ventricular dysfunction, to reduce adverse ventricular remodeling that may result in further reduction in ejection fraction, and for potential reduction of future myocardial infarction and stroke. Consider hydralazine/isosorbide dinitrate if intolerant to ACE inhibitors or angiotensin receptor blockers or either drug is contraindicated.

  - The SOLVD trial and ISIS-4 have confirmed a mortality reduction for patients with left ventricular dysfunction treated as early as three days postinfarction *(ISIS-4, 1995 [High Quality Evidence]; SOLVD Investigators, The, 1991 [High Quality Evidence])*.
  - Randomized studies have shown short-term and long-term outcomes were significantly improved in anterior myocardial infarction patients who were treated with ACE inhibitors *(ISIS-4, 1995 [High Quality Evidence]; SOLVD Investigators, The, 1991 [High Quality Evidence])*.
  - Low-dose ACE inhibitor use in hemodynamically stable patients has been shown to reduce mortality *(Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico, 1994 [Moderate Quality Evidence])*.

  * Shown in large clinical trials to reduce infarction mortality in all myocardial infarctions.

- **Calcium channel blockers** may be useful for control of blood pressure and ischemic pain when beta-blockers are contraindicated but should be avoided in patients with decreased left ventricular dysfunction or heart failure. The short-acting dihydropyridine calcium channel blockers (e.g., nifedipine) may be associated with increased risk and should be avoided in acute ischemic syndromes.

- **Oral nitrates** may benefit selected patients with postinfarction angina or congestive heart failure.

- **Low-molecular-weight heparin** has been shown to be superior to unfractionated heparin in patients without ST-segment elevation and can preferentially be used in subcutaneous dosing (e.g., enoxaparin sodium, 1 mg/kg every 12 hours). Unfractionated heparin/low-molecular-weight heparin may be continued for two-four days or maintained until conversion to warfarin is completed. If unfractionated heparin is used, the dose should be regulated to maintain an activated partial thromboplastin time of 50-75 seconds.

- **Warfarin** therapy may be initiated in certain clinical situations (e.g., postinfarction congestive heart failure or anterior myocardial infarction with high risk of left ventricular thrombus) as soon as clinical stability is achieved and invasive diagnostic studies are completed. The usual target international normalized ratio is 2.0-3.0.

- **Oral antiarrhythmics** are not recommended, especially when left ventricular function is reduced. Flecainide acetate and sotalol hydrochloride should be avoided in patients with significant structural heart disease unless clearly indicated on the basis of electrophysiologic study for the suppression of life-threatening ventricular arrhythmias. Beta-blockers are the current drug of choice when

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tolerated. Routine use of amiodarone hydrochloride in post-myocardial infarction patients with non-sustained ventricular ectopy has not been shown to reduce mortality.

- CAST demonstrated significantly reduced survival when encainide and flecainide were used to treat premature ventricular complexes and non-sustained ventricular tachycardia post-myocardial infarction with reduced left ventricular function *(Amiodarone Trials Meta-Analysis Investigators, 1997 [Meta-analysis] ; Akiyama, 1991 [Low Quality Evidence])*.

- **Tobacco cessation** should be addressed as soon as possible for patients who smoke or use tobacco products.

- **Glycemic control.** Tight control of blood glucose in patients with diabetes is recommended. Patients with diabetes mellitus have greater short-term and long-term mortality after acute myocardial infarction than patients without diabetes *(Haffner, 1998 [Low Quality Evidence])*. Diabetes is also an independent predictor of mortality following other acute coronary syndromes *(Malmberg, 2000 [Low Quality Evidence])*. Even in patients without a previous diagnosis of diabetes, hyperglycemia on admission for an acute myocardial infarction is associated with higher mortality than those without elevations of glucose. DIGAMI, DIGAMI-2 *(Malmberg, 2005 [High Quality Evidence])*, HI-5 *(Cheung, 2006 [High Quality Evidence])* and CREATE-ELCA *(CREATE-ECLA Trial Group Investiators, The, 2005 [High Quality Evidence])* have important limitations in terms of the efficacy of glycemic control in patients with an acute myocardial infarction. Whether control of glycemia is sufficient to reduce morbidity and mortality is not proven at this time. Given the lack of convincing evidence, the glucose targets during an acute myocardial infarction are not clearly defined. Previously, the ACC/AHA guidelines for ST-elevation myocardial infarction recommended an insulin infusion to "normalize" blood glucose in patients with both uncomplicated or complicated courses. The 2009 Focused Update recommends that it is reasonable to use an insulin-based regimen to achieve and maintain glucose levels less than 180 mg/dL while avoiding hypoglycemia for patients with STEMI with either a complicated or uncomplicated course *(Kushner, 2009 [Guideline])*.

Phase One Cardiac Rehabilitation

With shortened length of stay, teachable moments may be limited. As a result, timely initiation of education on lifestyle modification is crucial. Phase one cardiac rehabilitation should begin as soon as the patient is stable and pain-free. Goals are to minimize harmful effects of immobilization, assess the hemodynamic response to exercise, manage the psychosocial issues of cardiac disease, and educate the patient and family about lifestyle modification including:

- tobacco cessation;
- dietary instruction, including a heart healthy diet; and
- manageable exercise regimen.

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68. Complications?

Arrhythmic complications include sinus bradycardia, Möbitz I block (Wenckebach), Möbitz II block, complete heart block or asystole, premature ventricular contractions, ventricular tachycardia, ventricular fibrillation, accelerated idioventricular rhythm, and supraventricular arrhythmias (atrial flutter, atrial fibrillation, and supraventricular tachycardia). Ischemic complications include postinfarction angina. Mechanical complications include papillary muscle dysfunction, rupture with significant mitral regurgitation, ventricular septal rupture, myocardial rupture, right ventricular infarction, pericarditis with or without tamponade, left ventricular dysfunction, and aneurysm formation *(Latini, 2000 [Low Quality Evidence]; Menon, 2000 [Low Quality Evidence])*.

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69. See Acute Myocardial Infarction Complications Algorithm

Transfer to a PCI-capable facility any patients with post-MI complications such as those outlined above.

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70. Transfer to Post-Critical Care Unit Care

Patients should be transferred from the critical care unit to the telemetry or step-down unit when they are pain-free, hemodynamically stable, and meet the institution's protocol for admission to the telemetry unit (usually 12-24 hours after myocardial infarction). Discontinuation of cardiac monitoring should be considered for patients who attain electrical stability (usually within three days of infarction).

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72. Risk Stratification

Assessment of ejection fraction is important in predicting prognosis. Most patients should undergo echocardiography or other assessment of left ventricular ejection fraction. As a treadmill test is not useful for predicting recurrence of acute myocardial infarction, consider angiography when ST-segment depression or angina appear early in treatment. Consider pharmacologic stress testing if the patient is unable to exercise. If the electrocardiogram is not interpretable, consider stress imaging (nuclear or echocardiographic).

Patients with no high-risk indications following thrombolytics therapy may be stratified non-invasively into low, medium and high risk.

Some clinicians may elect to measure multiple cardiac biomarkers in patients with myocardial infarction. This may especially be helpful when risk stratification is not available by other clinical evidence. The work by Sabatine, Morrow, et al. demonstrated the utility of cardiac troponins, C-reactive protein and B-type natriuretic peptide measurements. This work demonstrated that patients with elevations of all three cardiac biomarkers had significantly higher risks of recurrent myocardial infarction and death than those with only two or one elevated. There was a progressive stepwise increase in risk going from one abnormality to two abnormalities to elevations of all three biomarkers.

(Morrow, 2003 [Low Quality Evidence]); Deedwania, 1997 [Cost-Effective Analysis]; Peterson, 1997 [Guideline]; American College of Cardiology/American Heart Association Task Force on Practice Guidelines, 1996 [Guideline]; Reeder, 1995 [Systematic Review])

B-type natriuretic peptide is a cardiac neurohormone released upon ventricular myocyte stretch as proBNP, which is enzymatically cleaved to the N-terminal proBNP (NT-proBNP) and, subsequently, to BNP. A number of prospective studies and large data sets have documented its prognostic value, independent of conventional risk factors for mortality in patients with stable and unstable coronary syndromes *(Galvani, 2004 [Low Quality Evidence]; James, 2003 [Low Quality Evidence]; Morrow, 2003 [Low Quality Evidence]; de Lemos, 2002 [Low Quality Evidence]; Jernberg, 2002 [Low Quality Evidence]; Omland, 2002 [Low Quality Evidence])*.

When measured at first patient contact or during the hospital stay or follow-up, the natriuretic peptides are strong, independent predictors of both short- and long-term mortality in patients with STEMI and UA/NSTEMI *(Eggers, 2009 [Low Quality Evidence]; Galvani, 2004 [Low Quality Evidence])*.

Increasing levels of NT-proBNP are associated with proportionally higher short- and long-term mortality rates at one year. Prognostic value was independent of a previous history of heart failure and of clinical or laboratory signs of left ventricular dysfunction on admission or during hospital stay *(Eggers, 2009 [Low Quality Evidence]; Galvani, 2004 [Low Quality Evidence])*. Moreover, the prognostic significance of an elevated BNP appears to be independent of echocardiographic findings *(Ang, 2008 [Low Quality Evidence])*.

Measurement of BNP or NT-proBNP may be considered to supplement assessment of global risk in patients with suspected ACS. However, further studies are needed to determine optimal treatment of elevated levels of natriuretic peptides *(Jneid, 2007 [Guideline])*.

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73. High-Risk Patient?

Patients who are at increased risk for adverse prognosis after acute myocardial infarction and who are also candidates for short-term intervention include those with a large amount of myocardial necrosis (ejection fraction less than 40%), residual ischemia (angina during hospitalization or exercise testing), electrical instability (greater than 10 PVC/hr), left main or three-vessel coronary artery disease, limited exercise tolerance, or rales/crackles in more than one-third of lung fields.

The following factors increase long-term risk:

- Age 70 years or greater
- Previous infarction
- Anterior-wall myocardial infarction
- Hypotension and sinus tachycardia
- Diabetes
- Female gender
- Continued smoking
- Atrial fibrillation
- Heart failure

Patients able to exercise more than four metabolic equivalents (METs) had less than a 2% subsequent incidence of death or myocardial infarction within one year, compared with 18% for those in the high-risk group *(Madsen, 1985 [Moderate Quality Evidence])*.

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74. Secondary Prevention and Risk Factor Modification

Modification of risk factors, such as high lipid levels, hypertension and smoking, significantly reduces subsequent cardiovascular mortality. Document risk factor counseling in the medical record in a consistent manner. Many health systems invoke a "care plan" or "critical pathway" approach with flow sheets. Ongoing patient monitoring and feedback are important. Continue the patient's adjunctive therapy (aspirin, plus a P2Y12 inhibitor [if patient managed by PCI] or a P2Y12 inhibitor alone if aspirin allergic, beta-blockers, warfarin for large anterior infarctions, ACE inhibitors/angiotensin receptor blockers and statins).

Efforts targeted at exercise (as an adjunct, in the management of other risk factors), lipid management, hypertension control and smoking cessation can reduce cardiovascular mortality, improve functional capacity, attenuate myocardial ischemia, retard the progression and foster the reversal of coronary atherosclerosis, and reduce the risk of further coronary events *(Sacks, 1996 [High Quality Evidence]; Scandinavian Simvastatin Survival Study Group, 1994 [High Quality Evidence])*.

The Cooperative Cardiovascular Project has documented a discrepancy between risk factor counseling documentation and actual practice during hospital stays of patients with myocardial infarction. Therefore, documentation of smoking cessation counseling has become one of 13 indicators judged to be representative of quality care by the Cooperative Cardiovascular Project steering committee *(Ellerbeck, 1995 [Low Quality Evidence])*.

1. Smoking cessation is clearly linked to mortality and morbidity after myocardial infarction.
2. Aggressive treatment of dyslipidemia can reduce subsequent myocardial ischemia.
3. Hypertension control will reduce recurrent cardiac events.

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4. Exercise alone is only modestly effective for secondary prevention.
5. A case management system may be more effective than usual care in long-lasting risk factor modification.
6. Initiate depression screening and medical management when appropriate.

Teaching must be done when the patient is ready, and ideally is based on patient-derived learning priorities. Teaching moments may be best taken advantage of by a team approach involving physician and nursing staff during the hospital stay. Ongoing outpatient follow-up and progress feedback are important for patient adherence *(Leon, 1990 [Low Quality Evidence]; Oldridge, 1988 [Low Quality Evidence])*.

Depression affects one in four acute myocardial infarction patients, and delay in treatment of depression is associated with poorer outcomes *(Rumsfeld, 2005 [Low Quality Evidence]; Writing Committee for the ENRICHD Investigators, 2003 [High Quality Evidence])*. The SADHART trial *(Glassman, 2002 [Low Quality Evidence])* suggested a benefit from the early diagnosis and treatment of depression in acute myocardial infarction patients. Depression associated with myocardial infarction is underdiagnosed, and referral for treatment initiation is inefficient. The Primary Health Questionnaire 9 (PHQ-9) is a validated tool for the rapid diagnosis of moderate and severe depression. A score of 15 or higher indicates moderate depression, and a score of 20 or higher indicates severe depression. Refer to the ICSI guideline Major Depression in Adults in Primary Care for additional information.

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75. Discharge

Complete and document the following before discharge:

- Patient education that includes discharge diagnosis, medical regimen, lifestyle modification issues and functional limitation (including resumption of sexual activity and driving)
- Scheduling of a follow-up appointment with the primary care physician
- Targeting a return-to-work date. Patients with sedentary jobs often return to work in two-three weeks. More physically demanding jobs often can be resumed in four-six weeks unless significant ischemia is present.

Patients are commonly discharged in less than three days following successful primary percutaneous coronary intervention with evidence of complete or near-complete salvage of threatened myocardium. Though patients should avoid strenuous exertion for several weeks during the stent healing phase, many such patients may return to sedentary or only moderately active work activities within days of discharge.

Most patients with uncomplicated myocardial infarctions should be discharged within five days. Patients undergoing primary percutaneous coronary intervention who are at low risk with an uncomplicated course may be discharged on the third day. Early reperfusion and definitive angiography revealing little or no residual injury or disease has increasingly demonstrated improved myocardial salvage and enhanced patient stability. Discharge may be individualized according to the degree of salvage and stability. In many centers some patients are safely discharged within 24 hours when salvage is nearly complete *(Grines, 1998 [Low Quality Evidence])*.

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76. Phase Two Cardiac Rehabilitation – If Available

Outpatient cardiac rehabilitation/secondary prevention programs are recommended for patients diagnosed with ST-elevation or non-ST-elevation myocardial infarction. Of particular concern are those patients who carry a moderate or high risk or have multiple modifiable risk factors for coronary artery disease and for whom supervised exercise training is deemed appropriate.

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There are exceptions to this recommendation, which include patient-oriented barriers, provider-oriented criteria (such as a patient who is deemed to have a high-risk condition or contraindication to exercise), or health care system barriers (such as patient who resides a significant distance from a program) (*Thomas, 2010 [Guideline]*). However, age, gender, race or socioeconomic status should not limit participation in a cardiac rehabilitation or secondary prevention program *(Lavie, 2009 [Low Quality Evidence])*.

Home exercise training programs have been shown to be beneficial in certain low-risk patient groups but lack the valuable elements of education and group interaction *(DeBusk, 1994 [High Quality Evidence])*.

Certain patients felt to be at higher risk of complications post-discharge are more likely to require monitoring during exercise in the immediate post-discharge period. There is strong evidence to suggest that cardiac rehabiliation programs have been shown to decrease mortality rates in all populations, including younger, more selective populations, as well as the socioeconimically and clinically diverse, older population (age 65 and older) *(Suaya, 2009 [Moderate Quality Evidence])*.

The U.S. Public Health Service described Phase Two cardiac rehabilitation as a "comprehensive, long-term program including medical evaluation, prescribed exercise, cardiac risk factor modification, education and counseling. Phase Two refers to outpatient, medically supervised programs that are typically initiated one to three weeks after hospital discharge and provide appropriate electrocardiographic monitoring." In the past, the main emphasis was exercise based, but today the focus includes risk factor modification, education and counseling.

Research shows that a cardiac rehabilitation program based on regular exercise and education focused on risk factor reduction is both efficient and effective in altering the course of coronary heart disease *(Ades, 2001 [Low Quality Evidence])*. The initial outpatient phase includes a comprehensive evaluation, education and treatment for outpatients who have experienced a cardiac-related event. Phase Two patients are monitored with continuous electrocardiogram, blood pressure, heart rate and subjective Rating of Perceived Exertion. For certain patients, referral to a Phase Two program may facilitate earlier hospital discharge by providing emotional support in the outpatient hospital setting.

Services delivered by a cardiac rehabilitation program may be considered "reasonable and necessary" for up to 36 sessions, and patients typically participate two to three times per week for 12-18 weeks *(CMS Decision Memo for Cardiac Rehabilitation Programs [CAG-00089R], 2006 [Reference])*.

Program Requirements

A cardiac rehabilitation program should include evaluation and assessment of modifiable cardiovascular risk factors, development of individualized interventions, and communication with other health care providers. Submeasures should include the following individualized assessments:

1. Tobacco cessation
2. Blood pressure control
3. Lipid control
4. Physical activity habits
5. Weight management
6. Diabetes management
7. Presence or absence of depression
8. Exercise capacity
9. Adherence to preventive medications

(Thomas, 2010 [Guideline])

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Additional goals of Phase Two rehabilitation:

- Increase exercise tolerance and endurance to enable patient to perform activities of daily living, at a level that resumes or exceeds his or her previous level of function
- Improve quality of life
- Improve psychological well-being and provide emotional support
- Provide educational support and resources

Education topics:

- Anatomy and physiology of the heart
- Nutrition
- Heart disease risk factors and modification
- Stress reduction
- Emotional aspects of heart disease
- Cardiac medications
- Aerobic exercise and exercise progression
- Cardiac signs and symptoms

Exercise prescription

An exercise prescription will be developed, taking into consideration the following factors:

- Patient's past medical history
- Recent cardiac or pulmonary event with symptomatology, interventions, estimated ejection fraction, complications in recovery process
- Risk factor identification
- Current medications, oxygen use
- Past exercise history
- Exercise history since cardiac event
- Orthopedic impairments
- Barriers to learning
- Vocational and leisure time activities

An exercise prescription consists of:

Mode – The emphasis is aerobic exercise – continuous activity for 30-40 minutes, using large muscle groups. Options include treadmill, stationary bike, recumbent bike, Airdyne® bike, NuStep®, elliptical machine, upper body ergometer, hall walking and chair aerobics. Pure isometric exercise should be minimized because it may result in left ventricular decompensation in patients with poor left ventricular function.

Frequency – Two to three times per week supervised in rehab and additional home exercise program daily.

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Duration – A goal of 30-40 minutes total including five-minute warm-up and five-minute cool-down.

Intensity – Initial exercise intensity will be based on diagnosis and previous exercise history. If the patient is just beginning an exercise program, initial training will usually range from two-three METs, e.g., two-three miles per hour, 0% grade on treadmill, or 25-50 watts on bicycle. In patients with an angina threshold of two-three METs, exercise training may not be appropriate.

Progression – A gradual increase of 0.5-1.0 METs will be prescribed as tolerated with a MET goal established individually at initial evaluation session.

Exercise Tolerance and Assessment Tools

Exercise tolerance will be assessed by monitoring heart rate response, blood pressure response and Borg Rating of Perceived Exertion, with desired level being 11 to 13.

Exercise heart rate – Taking into consideration the above information, an exercise heart rate guideline will be calculated. This applies to patients who are not taking a beta-blocker and who have been shown to tolerate the exercise heart rate without ischemia.

- Age-adjusted maximum heart rate multiplied by 60-75%
- Age-adjusted multiplied by 60-80% if approved by physician
- 20-30 above resting heart rate
- Graded stress test

Monitoring rate of perceived exertion is very useful. This is advantageous for many reasons: it is unaffected by negative chronotropic medications, unlike heart rate monitoring; it is quite reproducible across age, gender and cultural origin; and lastly, it only requires patient attunement to symptoms *(Squires, 1990 [Low Quality Evidence])*.

Monitoring METs – Monitoring is determined by the patient's post-myocardial infarction exercise tolerance test and/or in rehabilitation and is highly individual.

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77. Chronic Adjunctive Medications/Outpatient Management

Recommendation:

- The following medications should be considered for therapy:
  1. Aspirin should be administered indefinitely unless contraindicated or not tolerated.
  2. P2Y12 inhibitor for 12 months should be given to patients following placement of drug-eluting stents.
  3. Beta-blockers should be administered indefinitely in all patients who have had a myocardial infarction unless contraindicated.
  4. Patients who do not receive a beta-blocker during the first 24 hours because of early contraindications should be reevaluated for use of beta-blocker.
  5. Angiotensin-converting enzyme inhibitors should be administered to all patients with a history of acute myocardial infarction. The benefit is greater if the left ventricular ejection fraction is less than 40%.

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6. Statins should be administered to all patients with acute coronary syndrome unless contraindicated. For all patients with acute coronary syndrome, the LDL goal should be < 100 mg/dL with the option of < 70 mg/dL for very high risk patients.

   (See the ICSI Lipid Management guideline.)

7. Consider oral nitrates for patients with ongoing angina.

Continue the use of enteric-coated aspirin or aspirin plus a P2Y12 inhibitor when indicated. Beta-blocker use following myocardial infarction has been shown to reduce ischemia, prevent arrhythmias and reinfarction, and improve survival. Patients with large anterior infarctions may benefit from therapeutic warfarin therapy (international normalized ratio 2-3), usually for three months to reduce risk of systemic emboli.

Most patients should be receiving a statin (or alternative lipid-lowering medication if intolerant to statins) at discharge from the hospital. Lipid-lowering therapy should be considered for patients who have undergone percutaneous coronary intervention or coronary artery byprass graft and patients whose low-density lipoprotein cholesterol level is 70 mg/dL or greater. Calcium channel blockers should be considered only for patients with non-ST-elevation myocardial infarction who cannot take beta-blockers, and patients without congestive heart failure or decreased left ventricular ejection fraction. Oral nitrates should be considered for patients with ongoing ischemia *(Nichols-English, 2000 [Low Quality Evidence])*.

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78. Phase Three/Phase Four Cardiac Rehabilitation If Appropriate

Phase Three Cardiac Rehabilitation

Phase Three is a maintenance program based on the continuation of a heart healthy lifestyle. The program is designed for patients who have completed a Phase Two cardiac rehabilitation program or for individuals with a cardiac history or significant cardiac risk factors. Patients are not continually monitored by electrocardiogram, but spot-check electrocardiograms and daily blood pressures and heart rates are often recorded. Trained staff, when available, continues to provide support and education for risk factor modification and exercise progression. Warm-up, aerobic exercise, stretching and strength training (when appropriate) are included in Phase Three.

Phase Four Cardiac Rehabilitation

Phase Four cardiac rehabilitation begins after the desired functional capacity has been attained (usually greater than or equal to eight METs) and/or VO_2 max has reached a plateau. Maintenance is the principal goal. The exercise prescription should continue as at the end of Phase Three unless angina or exercise intolerance develops, either of which requires cessation of exercise and urgent medical attention.

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Acute Myocardial Infarction Complications Algorithm Annotations

81. Arrhythmic Complication(s)?

Arrhythmic complications including sinus bradycardia, Möbitz I (Wenckebach), premature ventricular complexes, accelerated idioventricular rhythm, and supraventricular arrhythmias (transient atrial flutter, atrial fibrillation, supraventricular tachycardia and hemodynamic instability) are generally benign and may require symptomatic therapy. Transient Mobitz II block with myocardial infarction may be treated symptomatically. Permanent pacing is indicated for persistent and symptomatic second- and third-degree atrioventricular block *(Col, 1972 [Low Quality Evidence])*.

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Centers for Medicare and Medicaid (CMS) Services – covered indications for defibrillators

1. Documented episode of cardiac arrest due to ventricular fibrillation (VF), not due to transient or reversible cause
2. Documented sustained ventricular tachyarrythmia, either spontaneous or induced by an electrophysiology study, not associated with an acute myocardial infarction and not due to transient or reversible cause
3. Documented familial or inherited conditions with a high risk of life-threatening ventricular tachyarrhythmia, such as long QT syndrome or hypertrophic cardiomyopathy
4. Coronary artery disease with documented prior myocardial infarction, ejection fraction less than 35%, and inducible sustained ventricular tachyarrythmia or ventricular fibrillation at electrophysiology study
5. Documented prior myocardial infarction, ejection fraction less than or equal to 30%, and QRS duration of greater than 120 msec (the patient must not have NYHA Class IV heart failure, shock, coronary artery bypass graft, percutaneous coronary intervention, myocardial infarction within three months or a need for coronary revascularization or predicted survival less than one year)
6. Patients with dilated cardiomyopathy, documented prior myocardial infarction, NYHA Class II and III heart failure and left ventricular ejection fraction less than or equal to 35% for longer than nine months

Additional indications may be found at https://www.cms.gov/medicare-coverage-database/details/ncd-details.aspx?NCDId=110&ncdver=3&bc=AAAAQAAAAAAA& (last accessed September 19, 2012).

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82. Treat Arrhythmic Complication(s)

A. Atrioventricular/bundle branch blocks:

Atrioventricular blocks are more common in acute inferior infarction and usually occur within 72 hours after the onset of infarction. Bundle branch blocks are more common in anterior infarctions and occur below the atrioventricular node. Blocks occurring with anterior infarctions have a poor prognosis and high mortality rate because of the extensive amount of myocardial necrosis present and the higher incidence of mechanical complications *(Nicod, 1988 [Low Quality Evidence]; Hindman, 1978 [Low Quality Evidence])*.

B. Ventricular arrhythmias:

Premature ventricular contractions are detected in more than 75% of patients.

Ventricular tachycardia occurs in about 20% of patients with acute myocardial infarction and is more often seen in patients with transmural infarction and in those with a large infarction that causes severe left ventricular dysfunction *(Campbell, 1981 [Low Quality Evidence])*.

C. Accelerated idioventricular rhythm:

This rhythm is seen in 12-25% of patients with acute myocardial infarction, usually by the second or third day after onset. It is seen with equal frequency in inferior and anterior myocardial infarctions. It should not be treated and usually has no adverse prognosis *(Eldar, 1992 [Low Quality Evidence])*.

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D. Supraventricular arrhythmias:

Atrial fibrillation occurs rarely. These arrhythmias occur most commonly within 24 hours after infarction and are associated with increased morbidity and mortality, particularly in patients with anterior infarctions *(Behar, 1992 [Low Quality Evidence])*.

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83. Ischemic Complication(s)?

Ischemic complications include postinfarction angina.

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84. Treat Ischemic Complication(s)

Treatment of postinfarction angina should be correlated with electrocardiogram changes, if possible. Optimal therapy consists of beta-blockers and long-acting nitrates. If beta-blockers are not tolerated or are ineffective and left ventricular function is not significantly depressed, a calcium channel blocker may be used. Early coronary angiography should be considered. Angina after myocardial infarction may be confused with pericarditis. Aneurysm formation should be a consideration.

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85. Mechanical Complication(s)?

Monitor patients for mechanical complications during hospital care and counsel patients to seek immediate emergency room care for symptoms of complications after discharge.

Mechanical complications may include papillary muscle dysfunction or rupture with significant mitral regurgitation, ventricular septal rupture, myocardial rupture, right ventricular infarction, pericarditis with or without tamponade, left ventricular dysfunction and aneurysm formation.

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86. Treat Mechanical Complication(s)

Papillary muscle dysfunction is evidenced by the murmur of mitral regurgitation, typically within five days of infarction.

Papillary muscle rupture may occur within 10 days of the event. Findings include development of sudden congestive heart failure or pulmonary edema, often but not always accompanied by a new holosystolic apical murmur. Diagnosis is verified by echocardiography. Stabilization is achieved by one or more of the following: aggressive use of diuretics and vasodilators, insertion of a Swan-Ganz catheter and insertion of an intra-aortic balloon pump. Because of the high mortality rate with this complication, urgent surgical repair is indicated.

- Some papillary muscle dysfunction occurs in more than half of acute myocardial infarctions. Papillary muscle rupture is a rare but catastrophic complication. It is seen three to five times more often in inferior infarctions than anterior and results in severe mitral regurgitation *(Kishon, 1992 [Low Quality Evidence])*.
- The mortality rate without surgical intervention is less than 50% within 24 hours and 94% within eight weeks. The mortality rate in mitral valve surgery done early after infarction is 38% *(Subramaniam, 1994 [Low Quality Evidence])*.

Ventricular septal rupture occurs within one week of infarction and results in left-to-right shunting and subsequent hemodynamic deterioration. Ventricular septal rupture is suggested by the presence of a new, harsh, holosystolic murmur that is loudest along the lower left sternal border; this may be accompanied by

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a thrill. Patients may also have symptoms of right-sided heart failure with right ventricular PO_2 step-up and may have less pulmonary congestion than patients with papillary muscle rupture. The diagnosis is confirmed by two-dimensional echocardiography. Patients are best stabilized by vasodilator therapy, insertion of a Swan-Ganz catheter, or an intra-aortic balloon pump, or all of these. Because of the high mortality rate, urgent surgical repair is indicated.

- Ventricular septal rupture occurs within one week of infarction in 1-3% of patients with almost equal frequency in anterior and inferior infarctions *(Radford, 1981 [Low Quality Evidence])*.
- The mortality rate without surgical intervention is 24% within 24 hours, 50% within a week and 90% within two months. Although surgical mortality rates are also high, it should be considered in all patients as the mortality with medical therapy alone is even higher *(Menon, 2000 [Low Quality Evidence])*.

Myocardial rupture is a common cause of sudden death after acute myocardial infarction. Symptoms or findings include emesis, persistent restlessness, anxiety and persistent ST-wave elevation on electrocardiogram. Rupture usually occurs within five to seven days of myocardial infarction. Left ventricular free-wall rupture leads to hemopericardium and subsequent death from tamponade. Contained rupture may result in formation of a pseudoaneurysm. Surgical resection is recommended.

- Free-wall rupture accounts for about 10% of fatal infarctions.
- Free-wall rupture occurs more frequently in transmural rather than subendocardial infarctions and is 8-10 times more common than papillary muscle or septal rupture.
- Women are four times more often at risk than men for myocardial rupture.
- Pseudoaneurysm occurs in one-third of all cases, and surgical resection of myocardial rupture is recommended.

(Subramaniam, 1994 [Low Quality Evidence]; Oliva, 1993 [Low Quality Evidence]; Pohjola-Sintonen, 1989 [Moderate Quality Evidence])

Right ventricular infarction is suspected in patients with inferior infarction complicated by low cardiac output, hypotension, oliguria, jugular venous distention, and clear lung fields without radiographic evidence of pulmonary venous congestion. Infarction can be confirmed by electrocardiogram findings (ST-segment elevation in right precordial leads V_4R through V_6R in the presence of inferior ST-elevation), two-dimensional echocardiography or pulmonary artery catheter demonstrating a disproportionate elevation of right atrial pressure compared with pulmonary capillary wedge pressure. Treatment consists of intravascular volume expansion and use of inotropic agents; if the patient loses sinus rhythm, temporary pacing to reestablish atrioventricular synchrony should be considered. Agents that reduce right ventricular preload, such as nitroglycerin, diuretics and large doses of morphine, should be avoided. ACE inhibitors and beta-blockers may require dose reduction or discontinuation with milder presentation of right ventricular dysfunction post-myocardial infarction *(Lavie, 2009 [Systematic Review]; Cintron, 1981 [Low Quality Evidence])*.

- About one-third of patients with inferior infarctions have some impairment of the right ventricle.
- The prognosis of patients with right ventricular infarction depends primarily on the presence of shock and hypotension, as well as systolic function of the left ventricular. The prognosis for patients with corrected hemodynamic compromise is excellent.

Post-myocardial infarction pericarditis can be early (occurring within 72-96 hours after acute myocardial infarction) or occasionally delayed (typically occurring weeks after myocardial infarction); the latter is called Dressler's syndrome. Early pericarditis is suspected in patients with pericardial friction rub, usually heard on the second or third day after acute myocardial infarction, and chest pain that may extend to the back, neck or shoulders that is intensified by movement and respiration and relieved by sitting up or leaning forward. Treatment consists of anti-inflammatory agents and reassurance. Echocardiography to assess for possible incomplete myocardial rupture should be considered. It is important to emphasize to the patient

that the recurrent pain is not the result of recurrent infarction. Risk of hemopericardium is increased in patients receiving anticoagulants; development of a pericardial effusion can be detected by close clinical observation and echocardiography *(Widimsky, 1995 [Low Quality Evidence])*.

Dressler's syndrome is characterized by an increase in erythrocyte sedimentation rate, leukocytosis and more frequent pleural and pericardial effusions than in early pericarditis. The incidence of Dressler's syndrome is roughly 1-3% of acute myocardial infarction patients. Because of the increased incidence of pericardial effusion, anticoagulation should be used with caution. Treatment for pericardial effusion with impending tamponade is pericardiocentesis, preferably guided by echocardiography *(Widimsky, 1995 [Low Quality Evidence])*.

Risk of developing left ventricular dysfunction and subsequent heart failure is greatly increased in patients with more extensive myocardial infarction. Restricted diastolic filling patterns on echocardiography may predict subsequent clinical heart failure.

- Pulmonary congestion in the early phases of acute myocardial infarction is a serious finding that requires prompt evaluation.
- Treatment of heart failure in the setting of acute myocardial infarction may include vasodilators, diuretics and in certain situations, beta-blockers and/or positive inotropic agents. Long-term management of patients with varying degrees of left ventricular dysfunction should include ACE inhibitors. ACE inhibitors have been shown to alter the process of ventricular enlargement and, subsequently, decrease the incidence and severity of heart failure in patients with left ventricular dysfunction and improve survival.

(Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico, 1994 [Moderate Quality Evidence]; SOLVD Investigators, The, 1991 [High Quality Evidence])

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Special Workup Algorithm Annotations

91. Symptoms, Arterial Blood Gases, Chest X-Ray Suggest Pulmonary Embolus?

- Symptoms may include dyspnea, tachypnea, pleuritic chest pain
- Physical findings extremely variable, may include fever, wheezing
- Electrocardiogram – non-specific ST-T changes
- Chest x-ray – normal, pleural effusion, wedge-shaped infiltrate
- Arterial blood gases – abnormal A-a gradient

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93. Symptoms, Arterial Blood Gases, Chest X-Ray Suggest Pneumothorax?

- Idiopathic or spontaneous pneumothorax – sudden onset of pleuritic chest pain and dyspnea (pleuritic pain more prominent with small pneumothorax, dyspnea with large)
- Arterial blood gases may be abnormal

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94. Consider Chest Tube and Hospitalization

- Pneumothorax greater than 10-20% usually requires chest tube
  - Primary pneumothorax – occurs in otherwise healthy people (idiopathic most frequently in tall young males, catamenial associated with endometriosis and menses) *(May, 1992 [Low Quality Evidence]; Schwartz, 1992b [Low Quality Evidence])*
  - Secondary pneumothorax – chronic obstructive pulmonary disease, asthma, pneumonia, cystic fibrosis *(May, 1992 [Low Quality Evidence]; Schwartz, 1992 [Low Quality Evidence])*
- Outpatient treatment possible if progression unlikely and patient reliable
  - Catheter aspiration followed by several hours of observation
  - Indwelling catheter attached to Heimlich valve
- Inpatient treatment if pneumothorax is secondary or significant symptoms
- Reabsorption slow – 1.25% per day

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Non-Cardiac Causes Algorithm Annotations

98. Reproducible Chest Pain Tenderness on Exam, No Increase in Troponin or Change in ST Segment on ECG

The examination should also target potential non-cardiac causes for the patient's symptoms such as prominent murmurs (endocarditis), pericardial friction rub (pericarditis), fever and abnormal lung sounds, pneumonia, reproducible chest pain after palpation (musculoskeletal) *(Kontos, 2010 [Low Quality Evidence])*.

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99. Symptoms, Signs, Chest X-Ray Suggest Pleural or Parenchymal Pulmonary Disease?

Patients with pulmonary or pleural disease frequently have a presenting complaint of chest pain with or without shortness of breath. A detailed history, physical examination, electrocardiogram, chest x-ray and laboratory evaluation typically will often suggest the diagnosis. Differential diagnoses include chronic obstructive pulmonary disease (COPD), asthma, infectious processes, and malignancies. Specific management of these diagnoses is beyond the scope of this guideline.

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100. Evaluate for Observation or Admission

Disposition decisions are largely dependent on the patient's stability. The initial treatment must be directed toward treating any instability and searching for the etiology of the symptoms. Pulse, blood pressure, respirations and level of consciousness must be assessed. Other factors that need to be considered are age, general state of health and immuno-competency and reliability. If a patient is labile or unstable, or at risk of becoming unstable, admit the patient *(Schwartz, 1992 [Low Quality Evidence])*.

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101. Symptoms and Signs Suggest Chest Wall/Costochondritis?

Costochrondritis (Tietze's Syndrome, intercostal muscular strain) can present with chest pain. If not adequately addressed, the patient will present repeatedly for chest pain evaluation. It is essential that this opportunity for teaching be maximized *(Parkash, 2009 [Low Quality Evidence])*.

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102. Aspirin, Acetominophen, Chest Wall Rest and Graded Return to Activity/Adjunctive Treatments to Be Considered

It should not be assumed that because this particular episode of chest pain has been attributed to a non-cardiac cause that the patient should therefore be treated with agents that increase cardiovascular risk. Several authors have raised concerns about the safety and efficacy of non-steroidal drugs even in a healthy population *(Conaghan, 2012 [Low Quality Evidence]; Musu, 2011 [Low Quality Evidence]; Fosbøl, 2010 [Low Quality Evidence]; Gooch, 2007 [Low Quality Evidence])*. Thus, we recommend aspirin, acetaminophen, topical strategies including cold and heat, gentle stretching and avoidance of lifting more than 10 lbs. for 48 hours as initial interventions. If the pain is localized over a joint, topical non-steroidal anti-inflammatory drugs such as diclofenac (Volteran gel, Flector patch) can be effective. These agents have limited systemic absorption estimated at 6%, so provide a greatly reduced renal risk as compared to systemically used NSAIDS. If the region is over a muscular area, then topical Lidoderm patch can offer partial day relief.

If the pain is of a chronic nature, in addition to being referred back to their primary care physician, the patient can be offered gabapentin or other medications used to treat neuropathic pain such as tricyclic antidepressants, SSRIs, or SNRIs. Muscle relaxation agents such as cyclobenzaprine, tizanidine, baclofen or benzodiazepines are not only ineffective but may predispose patient towards increased medication usage. (See the ICSI Assessment and Management of Chronic Pain guideline.)

Essential non-pharmacologic pain management techniques should not be overlooked. The impact of sleep hygiene, adequate daily aerobic exercise, physical therapy and desensitization techniques are essential to regaining full function. (See the ICSI Assessment and Management of Chronic Pain guideline.)

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103. Consider Gastrointestinal Diagnosis?

Gastrointestinal disorders are sometimes perceived by the patient as chest pain. Once the clinician is confident that no intra-thoracic processes are the cause of the discomfort, a gastrointestinal diagnosis should be considered.

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104. Gastrointestinal Evaluation

Commonly history, physical examination and a laboratory evaluation will suggest a gastrointestinal diagnosis. Further evaluation of this is beyond the scope of this guideline.

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105. Reconsider Differential Diagnosis

If the clinician, after initial evaluation and workup, does not arrive at a likely working diagnosis, he/she may have to go back and reconsider the entire differential diagnosis a second time in order to make certain that no serious condition has been missed. The clinician may then have to redirect his/her search for a diagnosis to conditions of the thoracic spine and thoracic nerves. Other considerations are somatization and anxiety disorders. These may be more or less obvious after careful consideration.

Differential diagnoses of thoracic spine and thoracic neuralgias include metastatic malignancy, multiple myeloma, arthritic processes, anklyosing spondylitis, osteomyelitis, kyphoscoliosis and herpes zoster.

Atypical chest pain associated with mitral valve prolapse is a poorly understood symptom *(Schwartz, 1992a [Low Quality Evidence])*.

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Clinic Evaluation Algorithm Annotations

107. Initial Focused Assessment for High-Risk History, Physical Examination and Other Findings

History should include characterization of pain, exacerbating or relieving factors, associated symptoms and risk factors for coronary disease. Physical examination should include careful cardiovascular and pulmonary examination, peripheral vascular examination, and evaluation for hypertension and hypercholesterolemia. Lab studies may include resting electrocardiogram, chest x-ray, hemoglobin and others if clinically indicated *(Pryor, 1983 [Low Quality Evidence])*.

The patient's description of pain and the history of previous coronary disease are by far the most important parts of the history.

Carotid bruits, peripheral vascular disease, and xanthomas on physical examination suggest a higher likelihood of coronary disease. The resting electrocardiogram may show evidence of previous infarction.

Direct provider education toward completing the history evaluation.

High-risk symptoms on initial presentation include:

History

- Severe or ongoing pain
- Pain lasting 20 minutes or more
- New pain at rest or with minimal activity
- Severe dyspnea
- Loss of consciousness

Physical findings

- Hypotension or other signs of underperfusion
- Tachycardia or bradycardia
- Pulmonary edema, cyanosis

Electrocardiogram findings

- ST-segment elevation greater than 1 mm on two contiguous leads suggesting acute myocardial infarction
- New ST or T wave changes
- ST depression greater than 1 mm at rest
- New left bundle branch block

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109. Initiate Emergency Interventions and Transfer to Emergency Department as Appropriate

Initiate emergency intervention as appropriate and transfer the patient as soon as possible for further emergency intervention.

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A patient complaining of chest pain should immediately be placed on a cardiac monitor. Vital signs should be taken, intravenous access started, oxygen administered and immediate electrocardiogram taken. Institution of stabilizing therapy (including nitroglycerin and chewable aspirin for suspect anginal pain) prior to the completion of the history or physical is appropriate and often necessary at this level.

(Jneid, 2007 [Guideline]; American Heart Association, 1992 [Guideline])

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110. Coronary Artery Disease Diagnosis Secure?

When the clinical setting and history suggest typical angina pectoris (substernal pain provoked by exertion and relieved by nitroglycerin or rest), the physician is very likely correct in assuming an ischemic coronary syndrome. Treatment and prognostic evaluation may proceed as outlined under the ICSI Stable Coronary Artery Disease guideline.

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111. Refer to ICSI Stable Coronary Artery Disease Guideline

Typical angina pectoris, if stable for 60 days and without evidence of recent myocardial infarction, may be treated under the ICSI Stable Coronary Artery Disease guideline.

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112. Ischemic Heart Pain Possible?

When coronary disease is of intermediate probability, a stress test may contribute supplemental information. When coronary disease is unlikely based on highly atypical symptoms and low prevalence of coronary disease among the population to which the patient belongs, stress testing may be misleading.

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113. Select Stress Test or Consider Cardiology Referral

Choose the best type of cardiac stress test based on:

- the resting cardiogram,
- the patient's ability to walk, and
- local expertise.

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114. Can Patient Walk?

For patients who cannot exercise, consider pharmacologic stress and imaging test (with adenosine, dipyridamole or dobutamine). Physical exercise is the most physiologic form of cardiovascular stress. If one doubts how far a patient will be able to walk, it might still be worthwhile to attempt treadmill exercise. The occasional patient with orthopedic restriction may be able to perform bicycle ergometry *(Braunwald, 1992 [Low Quality Evidence])*.

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116. Resting Electrocardiogram Interpretable for Ischemic Changes?

Marked resting electrocardiogram abnormalities such as left bundle branch block, left ventricular hypertrophy with repolarization abnormality, ventricular pre-excitation, or ventricular paced rhythm render the exercise electrocardiogram uninterpretable for ischemic changes. Patients with less than 1 mm resting ST depression may undergo standard electrocardiogram stress testing, provided the clinician realizes

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that further ST depression with exercise has minimal diagnostic significance. It is recommended that patients who are on digoxin undergo imaging studies since digoxin can produce abnormal ST-segment depression. A stable abnormality with exercise is reassuring *(American College of Cardiology/American Heart Association Task Force on Assessment of Cardiovascular Procedures, 1986 [Guideline]; Goldman, 1982 [Low Quality Evidence]; Weiner, 1979 [Low Quality Evidence])*.

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117. Do Exercise Imaging Study

When the resting electrocardiogram is markedly abnormal, use an exercise imaging test (stress echocardiogram, stress radionuclear perfusion or stress radionuclear ventriculogram) *(Braunwald, 1992 [Low Quality Evidence])*.

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118. Do Treadmill Stress Test

Use the Bruce protocol, modified if need be for debilitated patients. Adequacy of exercise and myocardial challenge is generally accepted as achieving greater than or equal to 85% of age-predicted maximum heart rate. The Bruce protocol, because of extensive use and long-term follow-up, provides the most reliable prognostic information *(Gibbons, 1997 [Guideline]; Fletcher, 1990 [Low Quality Evidence])*.

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119. Is Test Strongly Positive?

Stress testing may be strongly positive and suggest a moderate to high risk of cardiovascular events as indicated by the Duke treadmill score, which is based upon the Bruce protocol.

A stress test predicts the patient's prognosis and provides evidence of the presence or absence of coronary artery disease. Of these two types of information, the first, establishing the patient's prognosis, is the more reliable.

Treadmill findings that signify a poor prognosis are:

- poor exercise tolerance,
- hypotension, and
- marked ST abnormality at a low workload.

Conversely, good exercise tolerance to a high heart rate and blood pressure signifies a good prognosis, even if the exercise electrocardiogram is somewhat abnormal (for example, a patient who walks nine minutes and has 1 mm of asymptomatic ST depression).

Mark, et al. (Duke treadmill score) validated an easy-to-use treadmill score that stratifies high-, intermediate-, and low-risk patients. The Duke treadmill score was developed from a retrospective study of 2,842 inpatients. It was prospectively tested on an outpatient population of 613 patients with an endpoint of patient mortality. Consequently, it is well validated and the best measurement for the prognostic interpretation of treadmill tests.

A Duke score of greater than or equal to five is generally accepted as a passing score, and such patients may be discharged to home with follow-up within 72 hours.

(Farkouh, 1998 [High Quality Evidence]; Braunwald, 1992 [Low Quality Evidence]; Mark, 1991 [Low Quality Evidence]; Dubach, 1988 [Low Quality Evidence]; Mark, 1987 [Low Quality Evidence]; Chaitman, 1986 [Low Quality Evidence])

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Unless advanced age, comorbidity or patient preference suggests medical treatment, high-risk patients should be considered for revascularization. Patients identified as high risk by treadmill testing often have left ventricular dysfunction, left main coronary stenosis, or other serious coronary disease. Revascularization may offer a better prognosis *(Weiner, 1986 [Low Quality Evidence]; European Coronary Surgery Study Group, 1982 [High Quality Evidence])*.

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121. Is Test Positive but Low Risk?

A stress cardiogram may be positive but without features that signify a poor prognosis as noted above. For example, a 65-year-old man with atypical angina and 1 mm ST depression at 10 minutes has a good prognosis even though he has coronary disease.

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123. Is Test Equivocal?

Because of resting abnormality, limited exercise performance, limited heart rate or minor exercise abnormalities, the test may not be clearly normal or abnormal, yet high-risk treadmill findings are absent *(Kotler, 1990 [Low Quality Evidence]; Cohn, 1979 [Low Quality Evidence])*.

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125. Test Is Normal

A normal test may confirm the clinical impression of non-cardiac symptoms. Refer to cardiology if symptoms are worrisome despite a normal stress test.

Compared with the prognostic information contained in a stress test, the diagnostic information is more variable. The physician must consider:

1. How to estimate the pretest likelihood of coronary disease based upon the patient's age, sex and description of chest pain. If pretest likelihood is very high or very low, a test of intermediate predictive value, such as treadmill stress testing, may be misleading *(Diamond, 1979 [Low Quality Evidence])*.

Percent Prevalance of Angiographic Coronary Disease

Age	Nonanginal Chest Pain		Atypical Angina		Typical Angina	
	Men	*Women*	*Men*	*Women*	*Men*	*Women*
30-39	5.2 ± 0.8	0.8 ± 0.3	21.8 ± 2.4	4.2 ± 1.3	67.9 ± 3.2	25.8 ± 6.6
40-49	14.1 ± 1.3	2.8 ± 0.7	46.1 ± 1.8	13.3 ± 2.9	87.3 ± 1.0	55.2 ± 6.5
50-59	21.5 ± 1.7	8.4 ± 1.2	58.9 ± 1.5	32.4 ± 3.0	92.0 ± 0.6	79.4 ± 2.4
60-69	28.1 ± 1.9	18.6 ± 1.9	67.1 ± 1.3	54.4 ± 2.4	94.3 ± 0.4	90.6 ± 1.0

2. How abnormal are the exercise findings?

   Greater than 1 mm flat or 1.5 mm upsloping ST depression measured 80 msec after the J point occurring with a normal resting electrocardiogram is considered a positive test. However, "positive" is not all or nothing. Downsloping ST depression, greater degrees of ST depression, persistent ST depression, and ST depression at a low workload are "more positive." Conversely, upsloping ST depression, ST depression at a high workload, and rapidly resolving ST depression are "less positive." Refer to the table published in Diamond and Forrester, which describes the relationship between symptoms, demographics, ST findings and angiographic coronary disease.

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3. How good is the test itself? Is exercise challenge adequate, heart rate high enough? Resting abnormality present?

4. The natural history of a coronary plaque. A non-obstructive plaque may become active, provoking unstable symptoms by platelet emboli or vasoconstriction, yet not impair exercise coronary flow. **A normal test isn't reassuring if the symptoms are worrisome.**

5. What is the diagnostic goal? Absolute certainty for airline pilots? Reasonable reassurance?

Despite the complexities of interpretation, stress testing is a valuable tool in the evaluation of a patient with chest pain. Clinical judgment is paramount.

(Giroud, 1992 [Low Quality Evidence]; Kotler, 1990 [Low Quality Evidence]; Patterson, 1989 [Low Quality Evidence]; Cohn, 1979 [Low Quality Evidence])

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The Aims and Measures section is intended to provide guideline users with a menu of measures for multiple purposes, which may include the following:

- Population health improvement measures
- Quality improvement measures for delivery systems
- Measures from regulatory organizations such as The Joint Commission
- Measures that are currently required for public reporting
- Measures that are part of Center for Medicare Services Physician Quality Reporting initiative
- Other measures from local and national organizations aimed at measuring population health and improvement of care delivery

This section provides resources, strategies and measurement for use in closing the gap between current clinical practice and the recommendations set forth in the guideline.

The subdivisions of this section are:

- Aims and Measures
- Implementation Recommendations
- Implementation Tools and Resources
- Implementation Tools and Resources Table

Aims and Measures

1. Increase the success of emergency intervention for patients with chest pain symptoms suggestive of serious illness. *(Annotations #1, 2, 4, 5, 6, 20, 22)*

   Measures for accomplishing this aim:

   a. Percentage of acute myocardial infarction (AMI) patients who received aspirin within 24 hours before or after hospital arrival. *(Joint Commission Acute Myocardial Infarction Measure Set, NQF endorsed)*

   b. Median time from arrival to EKG (performed in the ED prior to transfer) for acute myocardial infraction (AMI) or chest pain patients (with probable cardiac chest pain). *(CMS Hospital Outpatient Measure 2012, NQF endorsed)*

2. Minimize the delay in administering fibrinolysis or angioplasty to patients with acute myocardial infarction (AMI). *(Annotations #55, 58)*

   Measures for accomplishing this aim:

   a. Percentage of AMI patients with ST-segment elevation or LBBB on the ECG closest to the arrival time receiving fibrinolytic therapy during the hospital stay and having a time from hospital arrival to fibrinolysis of 30 minutes or less. *(Joint Commission Acute Myocardial Infarction Measure Set, NQF endorsed)*

   b. Percentage of AMI patients with ST-segment elevation or LBBB on the ECG closest to the arrival time receiving primary percutaneous coronary intervention (PCI) during the hospital stay with a time from hospital arrival to PCI of 90 minutes or less. *(Joint Commission Acute Myocardial Infarction Measure Set, NQF endorsed)*

3. Increase the timely initiation of treatment to reduce post-infarction mortality in patients with AMI. *(Annotations #20, 31, 42, 44, 46, 55, 58, 65)*

   Measures for accomplishing this aim:

   a. Percentage of AMI patients who are prescribed aspirin at hospital discharge. *(Joint Commission Acute Myocardial Infarction Measure Set, NQF endorsed)*

   b. Percentage of AMI patients who are prescribed a beta-blocker at hospital discharge. *(Joint Commission Acute Myocardial Infarction Measure Set, NQF endorsed)*

   c. Percentage of AMI patients with left ventricular systolic dysfunction (LVSD) who are prescribed an ACEI or ARB at hospital discharge. *(Joint Commission Acute Myocardial Infarction Measure Set, NQF endorsed)*

   d. Percentage of AMI patients who receive a statin agent within 24 hours of arrival and at discharge for whom this treatment is appropriate.

4. Increase the percentage of patients with AMI using cardiac rehabilitation. *(Annotations #76, 78)*

   Measure for accomplishing this aim:

   a. Percentage of patients with AMI who are referred to an appropriate cardiac rehabilitation program:

      Phase Two Programs: electrocardiogram-monitored, outpatient

      and

      Phase Three Programs: non-monitored, outpatient.

   b. Percentage of patients with AMI with referral to an appropriate cardiac rehabilitation program (Phase 2 or Phase 3) post-discharge who enroll in the program.

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Measurement Specifications

Measurement #1a

Percentage of acute myocardial infarction (AMI) patients who received aspirin within 24 hours before or after hospital arrival.

Notes

This is a measure from Joint Commission Acute Myocardial Infarction Measure Set and is NQF-endorsed consensus standard for hospital care.

Full specifications for this measure can be found at the Joint Commission Web site at http://www.jointcommission.org/performance_measurement.aspx.

Web site link up to date as of October 11, 2010.

Measurement #1b

Median time from emergency department arrival to ECG (performed in the ED prior to transfer) for acute myocardial infraction (AMI) or chest pain patients (with probable cardiac chest pain).

Notes

This is a Center for Medicare and Medicaid Services (CMS) hospital outpatient measure and is NQF-endorsed consensus standard for hospital care.

Full specifications for this measure can be found via QualityNet Web site at http://www.qualitynet.org; Hospital Outpatient Quality Data Reporting Program (HOP QDRP).

Web site link up to date as of June 5, 2012.

Measurement #2a

Percentage of AMI patients with ST-segment elevation or LBBB on the ECG closest to the arrival time receiving fibrinolytic therapy during the hospital stay and having a time from hospital arrival to fibrinolysis of 30 minutes or less.

Notes

This is a measure from Joint Commission Acute Myocardial Infarction Measure Set and is NQF-endorsed consensus standard for hospital care.

Full specifications for this measure can be found at the Joint Commission Web site at http://www.jointcommission.org/performance_measurement.aspx.

Web site link up to date as of June 5, 2012.

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Measurement #2b

Percentage of AMI patients with ST-segment elevation or LBBB on the ECG closest to the arrival time receiving primary percutaneous coronary intervention (PCI) during the hospital stay with a time from hospital arrival to PCI of 90 minutes or less.

Notes

This is a measure from Joint Commission Acute Myocardial Infarction Measure Set and is NQF-endorsed consensus standard for hospital care.

Full specifications for this measure can be found at the Joint Commission Web site at http://www.jointcommission.org/performance_measurement.aspx.

Web site link up to date as of June 5, 2012.

Measurement #3a

Percentage of AMI patients who are prescribed aspirin at hospital discharge.

Notes

This is a measure from Joint Commission Acute Myocardial Infarction Measure Set and is NQF-endorsed consensus standard for hospital care.

Full specifications for this measure can be found at the Joint Commission Web site at http://www.jointcommission.org/performance_measurement.aspx.

Web site link up to date as of June 5, 2012.

Measurement #3b

Percentage of AMI patients who are prescribed a beta-blocker at hospital discharge.

Notes

This is a measure from Joint Commission Acute Myocardial Infarction Measure Set and is NQF-endorsed consensus standard for hospital care.

Full specifications for this measure can be found at the Joint Commission Web site at http://www.jointcommission.org/performance_measurement.aspx.

Web site link up to date as of June 5, 2012.

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Measurement #3c

Percentage of AMI patients with left ventricular systolic dysfunction (LVSD) who are prescribed an ACEI or ARB at hospital discharge.

Notes

This is a measure from Joint Commission Acute Myocardial Infarction Measure Set and is NQF-endorsed consensus standard for hospital care.

Full specifications for this measure can be found at the Joint Commission Web site at http://www.jointcommission.org/performance_measurement.aspx.

Web site link up to date as of June 5, 2012.

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Measurement #3d

Percentage of AMI patients who receive a statin agent within 24 hours of arrival and at discharge from hospital for whom this treatment is appropriate.

Population Definition

Patients 18 years and older diagnosed with acute myocardial infarction (AMI) and for whom statin treatment is appropriate.

Data of Interest

$$\frac{\text{\# of patients with AMI who receive a statin agent within 24 hours of arrival and at discharge from hospital}}{\text{\# of patients with AMI for whom statin treatment is appropriate}}$$

Numerator/Denominator Definitions

Numerator: Number of patients with AMI receiving statin agent within 24 hours of arrival and on discharge from hospital.

Denominator: Number of patients with AMI for whom statin treatment is appropriate.

Method/Source of Data Collection

Review the records of patients from EMR who had AMI. Review whether patients received a statin agent within 24 hours of arrival and on discharge from hospital, when appropriate.

Time Frame Pertaining to Data Collection

Data can be collected weekly or monthly.

Notes

This measure is a process measure, and improvement is noted as an increase in the rate.

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Measurement #4a

Percentage of patients with AMI who are referred to an appropriate cardiac rehabilitation post-discharge – Phase Two Programs: electrocardiogram-monitored, outpatient, and Phase Three Programs: non-monitored, outpatient.

Population Definition

Patients 18 years and older diagnosed with acute myocardial infarction (AMI) and discharged alive.

Data of Interest

$$\frac{\text{\# of patients with AMI who are referred to an appropriate cardiac rehabilitation program}}{\text{\# of patients with AMI who are discharged alive}}$$

Numerator/Denominator Definitions

Numerator: Number of patients with AMI who are referred to an appropriate cardiac rehabilitation program post-discharge – Phase Two Programs: electrocardiogram-monitored, outpatient, and Phase Three Programs: non-monitored, outpatient.

Denominator: Number of patients with AMI discharged alive.

Method/Source of Data Collection

Review the records of patients from EMR who had AMI and were discharged alive. Review whether patients are using appropriate cardiac rehabilitation post-discharge – Phase Two Programs: electrocardiogram-monitored, outpatient, and Phase Three Programs: non-monitored, outpatient.

Time Frame Pertaining to Data Collection

Data can be collected monthly. This measure will involve chart review for appropriateness of referral.

Notes

This measure is a process measure, and improvement is noted as an increase in the rate.

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Measurement #4b

Percentage of patients with AMI with referral to an appropriate cardiac rehabilitation program (Phase 2 or Phase 3) post-discharge who enroll in the program. Phase Two Programs: electrocardiogram-monitored, outpatient, and Phase Three Programs: non-monitored, outpatient.

Population Definition

Patients 18 years and older diagnosed with acute myocardial infarction (AMI).

Data of Interest

$$\frac{\text{\# of patients with AMI who enroll in an appropriate cardiac rehabilitation}}{\text{\# of patients with AMI and referred in cardiac rehab post-discharge}}$$

Numerator/Denominator Definitions

Numerator: Number of patients with AMI who enroll in an appropriate cardiac rehabilitation program post-discharge: Phase Two Programs: electrocardiogram-monitored, outpatient, and Phase Three Programs: non-monitored, outpatient who enroll in an appropriate cardiac rehab program.

Denominator: Number of patients with AMI who are referred in an appropriate cardiac rehab program post-discharge: Phase Two Programs: electrocardiogram-monitored, outpatient, and Phase Three Programs: non-monitored, outpatient.

Method/Source of Data Collection

Review the records of patients from EMR who had AMI and were referred to an appropriate rehab program post-discharge. Review whether patients were enrolled in an appropriate cardiac rehabilitation post-discharge: Phase Two Programs: electrocardiogram-monitored, outpatient, and Phase Three Programs: non-monitored, outpatient.

Time Frame Pertaining to Data Collection

Data can be collected monthly. This measure will involve chart review for appropriateness of referral and enrollment.

Notes

This measure is a process measure, and improvement is noted as an increase in the rate.

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Implementation Recommendations

Prior to implementation, it is important to consider current organizational infrastructure that address the following:

- System and process design
- Training and education
- Culture and the need to shift values, beliefs and behaviors of the organization.

The following system changes were identified by the guideline work group as key strategies for health care systems to incorporate in support of the implementation of this guideline:

- Clinics should have a process in place for a patient to be referred for emergency intervention via 911, or be seen in the clinic the same day, within 72 hours, or as an elective clinic evaluation based upon the presence of high-risk symptoms and duration.
- Hospitals should develop and implement emergency department critical pathways and consider standard orders to accomplish rapid evaluation and treatment of acute coronary syndrome. Standard discharge orders/instructions should also be considered.
- A process should be in place for the patient and family that will rapidly orient them to the suspected diagnosis, emergency department and coronary care unit process and other treatment measures to be considered. This could include caregiver face-to-face interactions with the patient and family, as well as teaching tools in written form.
- Institutions that cannot meet the recommended treatment times for primary percutaneous coronary intervention should consider preferential use of intravenous thrombolytic therapy, followed by as-soon-as-possible tranfer to a PCI capable facility for high risk patients. Lower risk patients may be observed at the initial hospital with later transfer for PCI as indicated. These institutions should have a predetermined plan for treating patients who present with contraindications to thrombolytics. Such plans may employ delayed local primary percutaneous coronary intervention or transfer to another institution.

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Implementation Tools and Resources

Criteria for Selecting Resources

The following tools and resources specific to the topic of the guideline were selected by the work group. Each item was reviewed thoroughly by at least one work group member. It is expected that users of these tools will establish the proper copyright prior to their use. The types of criteria the work group used are:

- The content supports the clinical and the implementation recommendations.
- Where possible, the content is supported by evidence-based research.
- The author, source and revision dates for the content are included where possible.
- The content is clear about potential biases and when appropriate conflicts of interests and/or disclaimers are noted where appropriate.

Resources Available to ICSI Members Only

ICSI has knowledge resources that are ***only*** available to ICSI members (these are indicated with an asterisk in far left-hand column of the Resources Table). In addition to the resources listed in the table, ICSI members have access to a broad range of materials including tool kits on Continuous Quality Improvement processes and Rapid Cycling that can be helpful. To obtain copies of these or other Resources, go to http://www.icsi.org/improvement_resources. To access these materials on the Web site, you must be logged in as an ICSI member.

The resources in the table on the next page that are not reserved for ICSI members are available to the public free-of-charge unless otherwise indicated.

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Implementation Tools and Resources Table

Author/Organization	Title/Description	Audience	Web sites/Order Information
American College of Cardiology	Information for ACC members and non-members. Includes clinical statements, advocacy and practice management information.	Health Care Providers	http://www.cardiosource.org
American Heart Association	Information and education on various aspects of heart disease. Includes information on lifestyle change. Provides links to local information.	Health Care Providers; Patients and Families	http://www.heart.org
Krames	Patient education resources based on current practice guidelines and standards of care.	Patients and Families	http://www.krames.com
Mayo Clinic	Health information on various cardiovascular diseases and conditions.	Patients and Families	http://www.mayoclinic.com

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ICSI Order Sets utilize two types of boxes for orders. One is the open box that clinicians will need to check for the order to be carried out. The second box is a pre-checked box; orders that have strong evidence and/or are standard of care and require documentation if the clinician decides to "uncheck" the order.

Organizations are recognizing the benefit of using pre-checked boxes for other orders to promote efficiency. Organizations are encouraged, through a consensus process, to identify those orders to utilize pre-checked boxes to increase efficiency, reduce calls to clinicians, and to reduce barriers for nursing and other professionals to provide care that is within their scope.

Throughout the order set you will note annotation numbers. These annotation numbers correspond with the guideline itself and provide associated discussion and evidence when available.

It is assumed that clinicians will supplement this information from standard pharmaceutical sources to inform their decisions for individual patients.

Order sets are available in MS Word format at http://www.icsi.org.

Order Set

This order set pertains to those orders from ED or direct admit to the CCU and does not include orders that pertain to telemetry admission, step-down or discharge.

Legend:
☐ Open boxes are orders that a clinician will need to order by checking the box.
☑ Pre-checked boxes are those orders with strong supporting evidence and/or regulatory requirements that require documentation if not done. (*See Annotation #1)*

Patient Information *(Two are required.)*

Last Name: _____
First Name: _____
Date of Birth: ___/___/_____
Patient's age: _____
ID #: _____

Admitting data

Date: _____
Admit unit: _____
Attending physician: _____
How to contact: _____

Diagnosis

Admitting diagnosis: _____

Secondary diagnosis: _____
_____

Condition

☐ Stable ☐ Unstable ☐ Other _____

Code status

☐ Full code ☐ DNR/DNI ☐ Comfort care ☐ Not discussed

Vitals

Vital signs and assessments: ☐ Routine per unit protocol ☐ Every _____ hours
☑ **Notify MD** if temperature exceeds 38.3°C (101°F), Systolic BP < 90, HR < 45, significant arrythmia
☐ Continuous monitoring telemetry
☑ **Notify MD** for new ST elevation or depression

☐ Daily weights
Patient weight: _____kg
Patient height: _____cm

Activity *Early ambulation is important for reducing the risk of VTE*

☐ Advance activity as tolerated
☐ Other _____

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Allergies/adverse drug reactions

☐ None
☐ Yes, name: _____ Type of reaction: _____
_____ Type of reaction: _____
_____ Type of reaction: _____

Nursing orders

☐ Oxygen as needed to maintain O_2 saturation greater than or equal 90% by pulse oximetry via:
☐ Nursing discretion – use nasal cannula or mask ☐ Nasal cannula ☐ Mask
☑ **Notify MD** if mask O_2 required
☐ O_2 saturation pulse: ☐ Continuous ☐ Monitor with vital signs ☐ Nursing discretion
Discontinue after: ☐ O_2 saturation greater then 90% on room air
☐ Other_____
☑ **Notify MD** if saturations less than 90%

☑ Glucose by finger stick screening ☐ 4 times daily *(before meals and at bedtime)* for 24 hours
☐ Discontinue after 24 hours for stable blood glucoses less than 140 mg/dL
☐ Subcutaneous Insulin Management Protocol *(consider IV insulin protocol for persistently elevated blood sugars)*

☐ Intake and Output every shift
☑ **Notify MD** if urine output is less than 25 mL/hour
☐ Foley catheter at nursing discretion; insertion and removal ☐ Indwelling

☐ Depression Screen on Day 2

VTE Mechanical Prophylaxis

(If long-term risk for VTE is moderate to high, consider anticoagulation with warfarin.)
☐ Graded compression stockings (*remove twice a day for 30 minutes)*
☐ Knee high ☐ Thigh high
☐ Pneumatic compression
☐ Foot boots ☐ Knee high ☐ Thigh high
☐ Instruct patient in foot pumps

Diet

☐ NPO ☐ Other _____
☐ Low sodium, low cholesterol, low saturated fat ☐ Consistent carbohydrate (CHO) diet
☐ No added salt ☐ Fluid restriction to _____ mL/24 hours
☐ No caffeine if adenosine stress test planned

IVs

☐ Establish IV saline lock *(flush per protocol)*
Indicate IV fluid:
☐ _____ at _____ mL/hour

Sedative/symptom medication

☐ Sleep aid _____ _____ mg by mouth as needed for insomnia.
☐ Antiemetic _____ _____ mg by IV every _____ hours as needed for nausea.
☐ Acetaminophen 650 mg by mouth every 4 hours as needed for discomfort and/or fever.
☐ Antianxiety _____ _____ mg IV every _____ hours as needed for anxiety.
☐ GI stimulant _____ _____mg by mouth once daily as needed for constipation.

Medications, specific:

Antithrombotic medication

*Choose one: (*Aspirin or platelet inhibitors are not recommended as monotherapy.)

☐ **Low-molecular-weight heparin** *(Recommend each institution choose 1 agent, then establish patient selection and dosing criteria guidelines.)*
- ☑ Platelet count every other day beginning day 2 and discontinued on day 14
  - ☑ **Discontinue** LMWH if platelet count drops 50% or more from baseline value
  - ☑ **Notify MD**
- ☑ Hemoglobin every other day beginning day 2
- ☑ Initiate patient education
- ☑ **Notify MD** if bleeding occurs

☐ **Fondaparinux (Xa inhibitor)**
- ☑ Platelet count every other day beginning day 2 and discontinued on day 14
  - ☑ **Discontinue** LMWH if platelet count drops 50% or more from baseline value
  - ☑ **Notify MD**
- ☑ Hemoglobin every other day beginning day 2
- ☑ Initiate patient education
- ☑ **Notify MD** if bleeding occurs

☐ **Unfractionated heparin**
- ☐ 60 units/kg IV (*max. bolus of 4,000 units*)
- ☐ Maintenance infusion 12 units/kg/hr (*max. initial infusion rate of 1,000 units/hr*)
- ☑ Platelet count every other day beginning day 2 and discontinued on day 14
  - ☑ **Discontinue** heparin if platelet count drops 50% or more from baseline value
  - ☑ **Notify MD**
- ☑ PTT every 6 hours after start of drip, then 6 hours after every drip rate change
- ☑ Hemoglobin every other day beginning day 2
- ☑ Initiate patient education
- ☑ **Notify MD** if bleeding occurs

Anti-Platelet medication

☑ Aspirin
- ☐ 325 mg initial dose by mouth **AND**
- ☐ 81 mg ☐ 162 mg ☐ 325 mg daily by mouth (recommended for recent stent placement)

☐ Not indicated due to:
- ☐ Aspirin allergy

☐ P2Y12 Inhibitors
- ☐ Clopidogrel
  - ☐ 600 mg once ☐ 300 mg once ☐ No loading dose
  - ☐ 75 mg once daily maintenance
- ☐ Prasugrel (FDA indication only for those patients undergoing PCI)
  - ☐ 60 mg once
  - ☐ 10 mg once daily maintenance

Note: Prasugrel is contraindicated in patients with a history of TIA or stroke. Prasugrel is generally not recommended in patients ≥ 75 years old or in patients weighing < 60 kg

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☐ Ticagrelor
180 mg loading dose by mouth once
90 mg by mouth twice daily
**Note: maintenance dose of aspirin 81 mg daily when on Ticagrelor

☐ GPIIB/IIIA Inhibitor (in consultation with cardiology)
- ☐ Abciximab 0.25 mg/kg IV bolus followed by 0.125 mcg/kg/min. (max. of 10 mcg/min.)
- ☐ Eptifibatide for CrCl > 50 mL/min: 180 mcg/kg IV bolus (max. of 22.6 mg) followed 10 min. later by a second IV bolus dose of 180 mcg/kg. Maintenance infusion 2 mcg/kg/min (max of 15 mg/hr), started after first bolus;
- ☐ Eptifibatide for CrCl < 50 mL/min: 180 mcg/kg IV bolus (max of 22.6 mg) followed 10 minutes later by a second IV bolus dose of 180 mcg/kg. Maintenance infusion of 1 mcg/kg/min (max of 7.5 mg/hr), started after first bolus. **Eptifibatide is contraindicated in patient on chronic hemodialysis.**
- ☐ Tirofiban for CrCl > 30 mL/min: 25 mcg/kg IV bolus, followed by a maintenance infusion of 0.15 mcg/kg/min
- ☐ Tirofiban for CrCl < 30 mL/min: 25 mcg/kg IV bolus, followed by a maintenance infusion of 0.075 mcg/kg/min

- ☑ **Notify MD** if bleeding occurs or signs of allergic reaction appear.
- ☑ Stop infusion if symptoms of allergic reaction or anaphylaxis appear, and follow treatment protocol.

☑ Beta-blocker: Start on day 1
- ☐ Metoprolol succinate ☐ 6.25 mg ☐ 12.5 mg ☐ 25 mg ☐ 50 mg ☐ 100 mg by mouth once daily
  (*Target dose is 100 mg once daily. Maximum dose is 400 mg once daily.)*
- ☐ Other: _____ mg by mouth every _____ hours
- ☐ Not indicated due to:
  - ☐ History of intolerance or adverse drug reaction
  - ☐ Symptomatic bradycardia or advanced heart block (*excluding treatment by pacemaker)*
  - ☐ Evidence of fluid overload or volume depletion
  - ☐ Has had recent treatment with an intravenous positive inotropic agent (*e.g., digoxin, nesiritide)*
  - ☐ Hypotension or shock
  - ☐ Suspected cocaine ingestion
- ☑ Give 1/2 dose beta-blocker if heart rate less than 60 or blood pressure less than 100 mmHg
- ☑ **HOLD** beta-blocker **and notify MD** if heart rate less than 55 or blood pressure less than 90 mmHg

☑ ACE Inhibitor *(Refer to your institution's formulary.)*
- ☐ Lisinopril _____ mg *(5-80 mg)* by mouth every day (*target dose 20 mg daily*)
- ☐ _____ mg by mouth every _____ hours
- ☐ Not indicated due to:
  - ☐ History of intolerance or adverse reactions
  - ☐ Serum potassium greater than 5.5 mEq/L
  - ☐ Symptomatic hypotension (*excluding excessive diuresis*)
  - ☐ Severe renal artery stenosis
  - ☐ Pregnancy
  - ☐ Recent rise in serum creatinine
- ☑ Give 1/2 dose ACE inhibitor if blood pressure less than 95 mmHg
- ☑ **HOLD** ACE inhibitor **and notify MD** if blood pressure less than 90 mmHg

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If intolerant to ACE Inhibitor:

☐ Angiotensin Receptor Blocker (ARB) *(Refer to your institution's formulary.)*
- ☐ Valsartan 80 mg by mouth twice daily
- ☐ Candasartan 8 mg by mouth once daily
- ☐ Losartan 25 mg by mouth once daily

☐ For special populations consider
- ☐ Hydralazine 25 mg every six hours
- ☐ Isosorbide dinitrate 10 mg three times daily

☑ Statin:
- ☐ Atorvastatin ☐ 80 mg by mouth once daily at bedtime
- ☐ Atorvastatin ☐ 40 mg ☐ 20 mg ☐ 10 mg by mouth once daily at bedtime
- ☐ Simvastatin ☐ 40 mg ☐ 20 mg ☐ 10 mg by mouth once daily at bedtime
- ☐ Simvastatin ☐ 80 mg by mouth once daily at bedtime
  (for patients currently on simvastatin for ≥ 12 months and no signs/symptoms of myopathy)

- ☐ _____ _____mg by mouth daily
- ☐ Not indicated due to:
  - ☐ History of intolerance or adverse reaction

☐ Potassium replacement protocol per institution to maintain K > 4.0 mEq/L
☐ Magnesium replacement protocol per institution to maintain Mg > 2.0 mg/dL

☐ Nitroglycerin 0.4 mg tablet sublingual every 5 minutes as needed for chest pain *(max. 3 doses)*
☐ Nitroglycerin _____ mcg/min IV continuous infusion *(Start at 10-20 mcg/min.)*
- ☑ *Titrate to keep pain-free and BP 90-140 mmHg.*
- ☑ **HOLD** if sildenafil citrate (Viagra®) has been taken within 24 hours
- ☑ **HOLD** if vardenafil (Levitra®) has been taken within 24 hours
- ☑ **HOLD** if tadalafil (Cialis®) has been taken within 48 hours
- ☑ **HOLD and notify MD** if SBP less than 90 mmHg
- ☑ **Notify MD** and obtain EKG if no relief after 3 doses or angina recurs

☐ Narcotic analgesic as needed to relieve angina symptoms
- ☐ Morphine sulfate ☐ 2 ☐ 4 mg one to two hours as needed _____ **OR**
- ☐ _____ _____
- ☑ **Notify MD** and obtain EKG if no relief or angina recurs

☐ Add insulin sliding scale protocol

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Labs/diagnostic tests:
(First day – those not performed in ED)

Indication: _____

	Done in ER
☐ CBC/Plts	☐
☐ AST	☐
☐ Sodium/potassium	☐
☐ BUN	☐
☐ Creatinine	☐
☐ Glucose	☐
☐ Calcium	☐
☐ Magnesium (*if on diuretic or ventricular arrhythmia*)	☐
☐ Hgb A1C (*if diabetic or glucose high and unknown if diabetic)*	☐
☐ Urinalysis	☐
☐ CPK	☐
☐ Troponin repeat at 90 min. and at 3 hours	☐
☐ TSH	☐
☐ Lipid panel (on admission; if necessary, non-fasting)	☐
☐ PT/INR	☐

☐ PTT ☐ PTT every AM while on heparin

☐ Electrocardiogram ☐ With recurrent chest pain
☑ **Notify MD** of ECG results
☐ Repeat every A.M. on day 2 and day 3

☐ Chest x-ray: ☐ PA & lateral ☐ Portable Indication: _____

☐ Echocardiogram ☐ Now ☐ On day _____
Indication: _____

☐ _____

☐ _____

Other orders:

☐ _____

☐ _____

☐ _____

Consults

☐ Cardiologist consult: reason _____

☑ Phase 1 Cardiac Rehabilitation consult

☑ Tobacco Cessation Education consult (*for current users or use within last year)*

☐ Nutrition Consult

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Discharge planning

☐ Social service consult for assistance in discharge planning
☐ Case management
☐ Financial counselor consult
☑ Cardiac rehabilitation program with transition to Phase III if appropriate

☑ Pneumococcal vaccine 0.5 mg IM per protocol on discharge if:
- Never received vaccine or vaccination status unknown
- First vaccination more than 5 years ago and patient was less than 65 at time of first vaccination

☐ Pneumococcal vaccination indicated but not given:
- ☐ Patient states they have received one
- ☐ History of allergy or adverse reaction
- ☐ Patient refuses

☑ Influenza vaccine 0.5 mg IM per protocol on discharge (*September-March only*) if patient has not received

☐ Influenza vaccination indicated but not given:
- ☐ Patient states they have received one
- ☐ History of allergy or adverse reaction
- ☐ Patient refuses

☐ Vaccination record sent to primary care

Authorized Prescriber Signature_____

Printed Name_____

Date & Time of Orders: ____/____/____ _____:_____

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The subdivision of this section is:

- References

References

Links are provided for those new references added to this edition (author name is highlighted in blue).

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ICSI has long had a policy of transparency in declaring potential conflicting and competing interests of all individuals who participate in the development, revision and approval of ICSI guidelines and protocols.

In 2010, the ICSI Conflict of Interest Review Committee was established by the Board of Directors to review all disclosures and make recommendations to the board when steps should be taken to mitigate potential conflicts of interest, including recommendations regarding removal of work group members. This committee has adopted the Institute of Medicine Conflict of Interest standards as outlined in the report, Clinical Practice Guidelines We Can Trust (2011).

Where there are work group members with identified potential conflicts, these are disclosed and discussed at the initial work group meeting. These members are expected to recuse themselves from related discussions or authorship of related recommendations, as directed by the Conflict of Interest committee or requested by the work group.

The complete ICSI policy regarding Conflicts of Interest is available at http://bit.ly/ICSICOI.

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ICSI facilitates and coordinates the guideline development and revision process. ICSI, member medical groups and sponsoring health plans review and provide feedback but do not have editorial control over the work group. All recommendations are based on the work group's independent evaluation of the evidence.

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All ICSI documents are available for review during the revision process by member medical groups and sponsors. In addition, all members commit to reviewing specific documents each year. This comprehensive review provides information to the work group for such issues as content update, improving clarity of recommendations, implementation suggestions and more. The specific reviewer comments and the work group responses are available to ICSI members at http://bit.ly/ACS1112.

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Institute for Clinical Systems Improvement

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ICSI Document Development and Revision Process

Overview

Since 1993, the Institute for Clinical Systems Improvement (ICSI) has developed more than 60 evidence-based health care documents that support best practices for the prevention, diagnosis, treatment or management of a given symptom, disease or condition for patients.

Audience and Intended Use

The information contained in this ICSI Health Care Guideline is intended primarily for health professionals and other expert audiences.

This ICSI Health Care Guideline should not be construed as medical advice or medical opinion related to any specific facts or circumstances. Patients and families are urged to consult a health care professional regarding their own situation and any specific medical questions they may have. In addition, they should seek assistance from a health care professional in interpreting this ICSI Health Care Guideline and applying it in their individual case.

This ICSI Health Care Guideline is designed to assist clinicians by providing an analytical framework for the evaluation and treatment of patients, and is not intended either to replace a clinician's judgment or to establish a protocol for all patients with a particular condition.

Document Development and Revision Process

The development process is based on a number of long-proven approaches and is continually being revised based on changing community standards. The ICSI staff, in consultation with the work group and a medical librarian, conduct a literature search to identify systematic reviews, randomized clinical trials, meta-analysis, other guidelines, regulatory statements and other pertinent literature. This literature is evaluated based on the GRADE methodology by work group members. When needed, an outside methodologist is consulted.

The work group uses this information to develop or revise clinical flows and algorithms, write recommendations, and identify gaps in the literature. The work group gives consideration to the importance of many issues as they develop the guideline. These considerations include the systems of care in our community and how resources vary, the balance between benefits and harms of interventions, patient and community values, the autonomy of clinicians and patients and more. All decisions made by the work group are done using a consensus process.

ICSI's medical group members and sponsors review each guideline as part of the revision process. They provide comment on the scientific content, recommendations, implementation strategies and barriers to implementation. This feedback is used by and responded to by the work group as part of their revision work. Final review and approval of the guideline is done by ICSI's Committee on Evidence-Based Practice. This committee is made up of practicing clinicians and nurses, drawn from ICSI member medical groups.

Implementation Recommendations and Measures

These are provided to assist medical groups and others to implement the recommendations in the guidelines. Where possible, implementation strategies are included that have been formally evaluated and tested. Measures are included that may be used for quality improvement as well as for outcome reporting. When available, regulatory or publicly reported measures are included.

Document Revision Cycle

Scientific documents are revised every 12-24 months as indicated by changes in clinical practice and literature. ICSI staff monitors major peer-reviewed journals every month for the guidelines for which they are responsible. Work group members are also asked to provide any pertinent literature through check-ins with the work group midcycle and annually to determine if there have been changes in the evidence significant enough to warrant document revision earlier than scheduled. This process complements the exhaustive literature search that is done on the subject prior to development of the first version of a guideline.

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