Overview of the acute management of acute ST elevation myocardial infarction

Introduction — The first step in the management of the patient with an acute ST elevation myocardial infarction (STEMI) is prompt recognition, since the beneficial effects of therapy with reperfusion are greatest when performed soon after presentation. For patients presenting to the emergency department with chest pain suspicious for an acute coronary syndrome (ACS), the diagnosis of STEMI can be confirmed by the ECG. Biomarkers may be normal early. (See "Criteria for the diagnosis of acute myocardial infarction" and "Management of suspected acute coronary syndrome in the emergency department").

Once the diagnosis of an acute STEMI is made, the early management of the patient involves the simultaneous achievement of several goals, as outlined by an ACC/AHA task force [1,2]:

- Relief of ischemic pain
- Assessment of the hemodynamic state and correction of abnormalities that are present
- Initiation of reperfusion therapy with primary percutaneous coronary intervention (PCI) or fibrinolysis
- Antithrombotic therapy to prevent rethrombosis or acute stent thrombosis
- Beta blocker therapy to prevent recurrent ischemia and life-threatening ventricular arrhythmias

This is then followed by the in-hospital initiation of different drugs that may improve the long-term prognosis [1]:

- Antiplatelet therapy to reduce the risk of recurrent coronary artery thrombosis or, with PCI, coronary artery stent thrombosis
- Angiotensin converting enzyme (ACE) inhibitor therapy to prevent remodeling of the left ventricle
- Statin therapy
- Anticoagulation in the presence of left ventricular thrombus or chronic atrial fibrillation to prevent embolization

This topic will summarize emergent/early management issues for patients with acute STEMI and then direct the reader to a more detailed discussion in other topics. The management of the patient after a reperfusion strategy has been chosen and carried out is discussed separately. (See "Overview of the non-acute management of acute ST elevation myocardial infarction").
The management of the patient with a non-ST elevation MI (NSTEMI) or with a complication of an acute MI (eg, cardiogenic shock, mitral regurgitation, ventricular septal defect) is discussed separately. (See "Overview of the acute management of unstable angina and acute non-ST elevation myocardial infarction" and "Prognosis and treatment of cardiogenic shock complicating acute myocardial infarction" and "Mechanical complications of acute myocardial infarction".)

**GENERAL PRINCIPLES** — The 2004 American College of Cardiology/American Heart Association (ACC/AHA) guidelines on STEMI recommended that all hospitals establish multidisciplinary teams to develop guideline-based, institution-specific written protocols for triaging and managing patients who present with symptoms suggestive of myocardial ischemia [1,2]. In addition the 2009 focused update recommended that each community develop a STEMI system of care that encourages [3]:

- Ongoing multidisciplinary team (including emergency medical services, non-PCI capable hospitals/STEMI referral centers, and PCI capable hospitals/STEMI receiving centers) meetings to evaluate outcomes and measures of performance.
- A process for prehospital identification and activation
- Transfer protocols for patients who arrive at STEMI referral centers who are candidates for primary PCI.

An increasing number of centers use structured algorithms, checklists, or critical pathways to screen patients with a suspected ACS [4-9]. These strategies combine diagnostic evaluation such as electrocardiography and serum biomarkers with therapeutic interventions such as aspirin, beta blockers, antithrombotic therapy, and primary PCI or fibrinolytic therapy (table 1). (See "Management of suspected acute coronary syndrome in the emergency department".)

**Elderly patients** — Although the majority of myocardial infarctions (MI) in the elderly population present with ECGs that are nondiagnostic or have ST segment depression, STEMI is not uncommon [10]. It is estimated that 60 to 65 percent of STEMI occur in patients ≥65 years of age and 28 to 33 percent occur in patients ≥75 years of age [10-12]. In addition, as many as 80 percent of all deaths related to MI occur in persons ≥65 years of age. (See "Overview of the acute management of unstable angina and acute non-ST elevation myocardial infarction", section on 'Elderly patients'.)

Although patients age 75 and older have been underrepresented in clinical trials of ACS, the following observations concerning acute MI in elderly compared to younger patients are generally accepted [10]:

- **Elderly patients more frequently have an atypical presentation**, including silent or unrecognized MI [10,13]. As an example, chest pain is present in 57 percent of patients ≥85 years of age compared to 90 percent for those under age 65. Left bundle-branch block and Killip class ≥2 acute heart failure are much more common in patients ≥85 years of age (34 and 45 percent, respectively). Delays in diagnosis have been well documented and often lead to delays in therapy.

- Patients ≥75 years of age have a higher in-hospital mortality, which often occurs in those with electrical and mechanical complications [10].

- **Outcomes in elderly patients, as in younger patients, appear to be better with primary PCI than fibrinolysis** [10]. (See 'Percutaneous coronary intervention' below.)

- Elderly patients are more likely to have frequent and severe bleeding as a consequence of antithrombotic therapy [10]. As an example, the risk of stroke as a consequence of fibrinolysis is approximately 2.9 percent in patients ≥85 years of age [10]. Nevertheless, patients ≥85 years of age who have no contraindications to fibrinolysis, including a high
risk for intracranial hemorrhage, can be treated with fibrinolysis. (See 'Fibrinolysis' below and "Fibrinolytic (thrombolytic) agents in acute ST elevation myocardial infarction: Therapeutic use", section on 'Stroke'.)

**Women** — The approach to women and men should be the same, despite the fact that women have more atypical symptoms, are older, have greater delays to presentation, and have higher prevalence of hypertension. In addition they are at higher risk of bleeding.

**Cocaine associated MI** — MI is a well-described complication among patients presenting with cocaine-induced ischemic symptoms. (See "Cardiovascular complications of cocaine abuse", section on 'Myocardial infarction'.)

We agree with the 2008 American Heart Association scientific statement on the management of cocaine-associated chest pain and myocardial infarction, which states that these patients should be managed in a manner similar to other ACS patients [14]. The following two points were also made:

- Benzodiazepines should be administered early
- Beta blockers should not be used in the setting of acute cocaine intoxication with chest pain due to the possibility of exacerbation of coronary artery vasoconstriction

**Possible stent thrombosis** — The in-hospital mortality of STEMI is higher in patients with coronary artery stent thrombosis (ST) as the cause, as opposed to a ruptured plaque. Immediate PCI is the treatment of choice, similar to spontaneous MI. Fibrinolysis has also been used for patients with STEMI due to coronary artery stent thrombosis. (See "Coronary artery stent thrombosis: General issues" and "Coronary artery stent thrombosis: Prevention and management".)

**INITIAL ASSESSMENT** — Clinical assessment of the patient with a possible acute coronary syndrome (ACS) begins as soon as the patient arrives in the emergency department and continues in the coronary care unit. Initial assessment consists of acute triage and early risk stratification. An ECG should be obtained within 10 minutes of arrival, if it has not been obtained already by EMS providers in the prehospital arena. A detailed approach to the evaluation and management of patients with an ACS in the emergency department is found separately. (See "Management of suspected acute coronary syndrome in the emergency department".)

**Acute triage** — A focused evaluation on presentation should address, in order of importance, those findings that permit rapid triage and initial diagnosis and management [1]:

- **Responsiveness, airway, breathing and circulation** — In patients who present with respiratory or cardiorespiratory arrest, the appropriate resuscitation algorithms should be followed. (See "Advanced cardiac life support (ACLS) in adults" and "Supportive data for advanced cardiac life support in adults with sudden cardiac arrest" and "Basic life support (BLS) in adults".)

- **Evidence of systemic hypoperfusion** (hypotension; tachycardia; impaired cognition; cool, clammy, pale, ashen skin) — Cardiogenic shock complicating acute MI requires aggressive evaluation and management. This issue is discussed in detail separately. (See "Clinical manifestations and diagnosis of cardiogenic shock" and "Prognosis and treatment of cardiogenic shock complicating acute myocardial infarction".)

- **Left heart failure with hypoxia** — Patients who present with dyspnea, hypoxia, pulmonary edema, and/or impending respiratory compromise require aggressive oxygenation, airway stabilization, diuretic therapy, and afterload reduction in addition to the standard treatments. (See "Treatment of acute decompensated heart failure: General considerations".)
- Ventricular arrhythmias — Sustained ventricular tachyarrhythmias in the perinfarction period must be treated immediately because of their deleterious effect on cardiac output, possible exacerbation of myocardial ischemia, and the risk of deterioration into VF. (See "Clinical features and treatment of ventricular arrhythmias during acute myocardial infarction" and 'Arrhythmia management' below.)

Early risk stratification — Analyses from several large clinical trials and registries have established a number of clinical predictors of adverse outcomes among patients with STEMI. There are many clinical prognostic factors that are immediately available to the physician based upon the initial history, physical examination, electrocardiogram (ECG), and chest X-ray. Given the speed with which reperfusion therapy is administered in patients with STEMI, their clinical utility in early medical decision making in the ED is often limited. They do provide good prognostic information that has utility in the post-reperfusion period, however, and may provide guidance regarding the optimum method of reperfusion.

High-risk features include advanced age, low blood pressure, tachycardia, heart failure (HF), and an anterior MI. Specific scoring systems, such as the TIMI risk score, permit a fairly precise determination of the risk of in-hospital mortality (calculator 1) [15,16].

Patients at high risk require an aggressive management strategy in addition to standard medical management. Direct prehospital transport or, less optimally, prompt interhospital transfer to a facility with revascularization capabilities is recommended for such patients [1].

INITIAL THERAPY — The patient with STEMI should have continuous cardiac monitoring, oxygen, and intravenous access. Therapy should be started to relieve ischemic pain, stabilize hemodynamic status, and reduce ischemia while the patient is being assessed as a candidate for fibrinolysis or primary PCI. Other routine hospital measures include anxiolytics, serial ECGs, and blood pressure monitoring. The 2004 ACC/AHA guidelines recommended that all initial therapy be carried out in the emergency department based upon a predetermined, institution-specific, written protocol [1,2].

The following sections summarize acute therapy. A detailed description of the initial therapy in STEMI is found separately. (See "Management of suspected acute coronary syndrome in the emergency department", section on 'Immediate ED interventions'.)

Oxygen — We recommend supplemental oxygen to patients with an arterial saturation less than 90 percent, patients in respiratory distress, or those with other high-risk features for hypoxia [17].

The role of supplemental oxygen in patients without hypoxia has not been well studied. A 2010 Cochrane review evaluated three trials of 387 patients with presumed myocardial infarction (MI) who were randomly assigned to supplemental oxygen or room air. Enrolled patients were either hypoxic and normoxic. The study found no significant difference in mortality (pooled relative risk 2.88, [95% CI 0.88-9.39] in an intention-to-treat analysis and 3.03, [95% CI 0.93-9.83] among those with confirmed MI). No subgroup analysis was performed on those with normoxia [18].

The suggestion of harm with supplemental oxygen found in this Cochrane review is of concern, particularly in patients with normoxia, as a pathophysiologic basis for such harm has been articulated [19]. Hyperoxia, which might occur with the administration of oxygen to normoxic individuals, has been shown to have a direct vasoconstrictor effect on the coronary arteries [19].

Until better evidence to support the use of supplemental oxygen in normoxic patients with acute MI is available, we suggest (a weak recommendation) its use.

Reperfusion — Prompt restoration of myocardial blood flow is essential to optimize myocardial salvage and to reduce mortality (figure 1) [20]. A decision must be made as soon as possible as to whether reperfusion will be achieved with fibrinolytic agents or primary (direct) percutaneous
coronary intervention (PCI). (See "Selecting a reperfusion strategy for acute ST elevation myocardial infarction").

**Percutaneous coronary intervention** — If high-quality PCI is available, multiple randomized trials have shown enhanced survival compared to fibrinolysis with a lower rate of intracranial hemorrhage and recurrent MI [21]. As a result, 2007 focused update of the ACC/AHA 2004 Guidelines for the Management of Patients With STEMI recommended the use of primary PCI for any patient with an acute STEMI who can undergo the procedure within 90 minutes of first medical contact by persons skilled in the procedure (table 2) [2]. This was not changed in the 2009 update [3].

Patients with typical and persistent symptoms in the presence of a new or presumably new left bundle branch block are also considered eligible. (See "Primary percutaneous coronary intervention in acute ST elevation myocardial infarction: Determinants of outcome").

For patients presenting 12 to 24 hours after symptom onset, the performance of primary PCI is reasonable if the patient has severe HF, hemodynamic or electrical instability, or persistent ischemic symptoms [1]. Randomized trials of routine late PCI have shown an improvement in left ventricular function but not in hard clinical end points. This approach is not recommended (table 2). (See "Coronary artery patency and outcome after myocardial infarction", section on 'Late PCI to open an occluded artery'.)

If primary PCI is not available on site, rapid transfer to a PCI center can produce better outcomes than fibrinolysis, as long as the door-to-balloon time, including interhospital transport time, is less than 90 minutes. This door-to-balloon time is difficult to obtain unless rapid transport protocols and relatively short transport distances are in place. (See "Primary percutaneous coronary intervention in acute ST elevation myocardial infarction: Determinants of outcome").

We suggest the following approach for patients with STEMI at hospitals without on-site PCI capability:

- For patients who present within two hours of the onset of symptoms we suggest full dose lytic therapy and transfer to a PCI center. This assumes that primary PCI cannot be performed in less than 90 minutes at a local PCI center.

- For patients who present with symptoms greater than two to three hours we suggest transfer for primary PCI (and give a glycoprotein IIb/IIIa inhibitor before transfer). However, there are times when the patient presents after two hours, PCI cannot be accomplished in less than 120 minutes. In this setting clinical judgement needs to be exercised; **fibrinolytic therapy may be appropriate in patients with up to 12 hours of symptoms.**

As noted above, all patients who undergo primary PCI should be pretreated at diagnosis with anticoagulant and antiplatelet therapy. (See 'Antiplatelet therapy' below and 'Anticoagulant therapy' below.)

**Fibrinolysis** — The 2007 ACC/AHA focused update (not changed in the 2009 focused update) recommended the use of fibrinolytic therapy in the following patients [2,3,22]:

- Any patient with STEMI who presents within 12 hours of symptom onset has no contraindications for fibrinolysis (table 3A-B), and presents to a facility without the capability for expert, prompt intervention with primary PCI within 90 minutes of first medical contact (table 4A-B) [1].

- Patients who present to a facility in which the relative delay necessary to perform primary PCI (the expected door-to-balloon time minus the expected door-to-needle time) is greater than one hour.
The time interval from first patient contact to initiation of fibrinolytic drug infusion should be less than 30 minutes [1,22,23]. (See "Fibrinolytic (thrombolytic) agents in acute ST elevation myocardial infarction: Therapeutic use", section on 'Time to therapy (door-to-needle time)' and "Selecting a reperfusion strategy for acute ST elevation myocardial infarction".)

Fibrinolytic therapy has generally not improved outcomes in patients presenting at 12 hours or later and is therefore not indicated in those who are stable and asymptomatic. However, fibrinolysis can be considered up to 24 hours after symptom onset if the patient has ongoing or stuttering chest pain and PCI is not available. (See "Fibrinolytic (thrombolytic) agents in acute ST elevation myocardial infarction: Therapeutic use", section on 'Time to presentation'.)

A number of different fibrinolytic regimens have been evaluated, and each agent has its own preferred dosing regimen (table 5). (See "Characteristics of fibrinolytic (thrombolytic) agents and clinical trials in acute ST elevation myocardial infarction".)

Patients receiving fibrinolytic therapy benefit from pretreatment with clopidogrel but not a GP IIb/IIIa inhibitor [1,23]. (See "Antiplatelet agents in acute ST elevation myocardial infarction".)

**Angiography after fibrinolysis** — Fibrinolysis immediately before primary PCI, previously called facilitated PCI, is not recommended. (See 'Percutaneous coronary intervention after fibrinolysis for acute ST elevation myocardial infarction', section on 'Facilitated PCI'.)

The data evaluating the role of elective coronary angiography, which might include adjunctive and early elective percutaneous coronary intervention (PCI), are discussed in detail elsewhere. (See "Percutaneous coronary intervention after fibrinolysis for acute ST elevation myocardial infarction", section on 'Adjunctive or early elective PCI'.)

The use of "rescue PCI" for patients with recurrent ischemia or infarction is better established. (See "Management of failed fibrinolysis (thrombolysis) or threatened reocclusion in acute ST elevation myocardial infarction", section on 'Primary failure'.)

**Bypass surgery** — Coronary artery bypass graft surgery (CABG) is infrequently performed in patients with STEMI. The main indications are for emergent or urgent CABG related to failure of fibrinolysis or PCI, cardiogenic shock, or life threatening ventricular arrhythmias associated with left main or three-vessel disease (table 6) [1].

The benefit of revascularization must be weighed against the increase in mortality associated with CABG in the first three to seven days after STEMI. Thus, if the patient has stabilized, surgery should be delayed to allow myocardial recovery. Patients with critical anatomy should undergo CABG during the initial hospitalization (table 6). (See "Coronary artery bypass graft surgery after acute ST elevation myocardial infarction".)

**Medications** — A summary of the specific agents listed below and their usual dosing regimens is found elsewhere. (See "Management of suspected acute coronary syndrome in the emergency department", section on 'ST elevation'.)

**Antiplatelet therapy** — Antiplatelet therapy including aspirin, thienopyridine, and, in patients undergoing primary PCI, a GP IIb/IIIa inhibitor improve outcomes. Initial thienopyridine dosing varies with the reperfusion strategy. (See "Antiplatelet agents in acute ST elevation myocardial infarction", section on 'Alternative to aspirin'.)

**Anticoagulant therapy** — The evidence to support parenteral anticoagulant therapy in most cases of STEMI is strong. However, the evidence to recommend one agent over another is less robust, in part because it is derived from many studies that were performed before the current era of aggressive antiplatelet therapy or studies which conflict with each other. The choice of agent depends upon the overall treatment strategy designed for each patient: fibrinolytic therapy with either fibrin specific or non-fibrin specific agents, primary percutaneous coronary intervention, or no reperfusion. (See "Anticoagulant therapy in acute ST elevation myocardial infarction".)
Nitrates — Intravenous nitroglycerin is useful in patients with persistent chest pain after three sublingual nitroglycerin tablets, as well as in patients with hypertension or heart failure. (See "Nitrates in the management of acute coronary syndrome").

However, nitrates must be used with caution or avoided in settings in which hypotension is likely or could result in serious hemodynamic decompensation, such as right ventricular infarction or severe aortic stenosis. In addition, nitrates are contraindicated in patients who have taken a phosphodiesterase inhibitor for erectile dysfunction (or pulmonary hypertension) within the previous 24 hours. (See "Right ventricular myocardial infarction", section on 'Avoid drugs that decrease preload' and "Sexual activity in patients with heart disease").

Morphine — Intravenous morphine sulfate at an initial dose of 2 to 4 mg, with increments of 2 to 8 mg repeated at 5 to 15 minute intervals, should be given for the relief of chest pain or anxiety.

Beta blockers — Oral beta blockers are administered universally to all patients without contraindications who experience an acute STEMI [1]. Contraindications include heart failure, evidence of a low output state, high risk for cardiogenic shock, bradycardia, heart block, or reactive airway disease. (See "Beta blockers in the management of acute coronary syndrome").

The 2007 focused update of the ACC/AHA 2004 guidelines on the management of STEMI suggest that an intravenous beta blocker may be considered in hypertensive patients who are not at high risk of cardiogenic shock on the basis of clinical risk factors [2]. This was not changed in the 2009 focused update [3].

Other

Arrhythmia management — Both atrial and ventricular arrhythmias can be seen during and after the acute phase of STEMI. These include atrial fibrillation or flutter, which can cause symptomatic hypoperfusion due to a rapid rate, and life-threatening ventricular tachycardia or ventricular fibrillation. (See "Supraventricular arrhythmias after myocardial infarction" and "Clinical features and treatment of ventricular arrhythmias during acute myocardial infarction").

Prophylactic intravenous or intramuscular lidocaine to prevent VT/VF in the acute MI patient is NOT recommended. Recommended prophylactic measures include early administration of an intravenous beta blocker and treatment of hypokalemia and hypomagnesemia. Treatment of ventricular tachyarrhythmias in the setting of acute MI is discussed separately. (See "Clinical features and treatment of ventricular arrhythmias during acute myocardial infarction").

Sinus bradycardia can occur in patients with STEMI, especially when the inferior wall is involved. If the patient is symptomatic, therapy with atropine is indicated (table 7A-B). Persistent sinus bradycardia may require temporary pacing. (See "Supraventricular arrhythmias after myocardial infarction", section on 'Sinus bradycardia'.)

Atrioventricular nodal and intraventricular conduction abnormalities also may be seen in STEMI, particularly of the anterior wall. If the patient is symptomatic, temporary pacing is indicated. Asymptomatic patients with certain types of conduction abnormalities may also require prophylactic temporary pacemaker therapy, and some may require permanent pacemaker implantation (table 8A-C). (See "Conduction abnormalities after myocardial infarction").

Non-steroidal antiinflammatory drugs — Nonsteroidal antiinflammatory drugs (except aspirin) should be discontinued immediately due to an increased risk of cardiovascular events associated with their use. (See "Nonselective NSAIDs: Cardiovascular effects").

Potassium and magnesium — Although there are no clinical trials documenting the benefits of electrolyte replacement in acute MI, the ACC/AHA guidelines recommend maintaining the serum potassium concentration above 4.0 meq/L and a serum magnesium concentration above...
2.0 meq/L (2.4 mg/dL or 1 mmol/L) [1,2]. (See "Clinical features and treatment of ventricular arrhythmias during acute myocardial infarction", section on 'Ventricular fibrillation'.)

**MI WITH NORMAL CORONARY ARTERIES** — At the time of coronary angiography, as many as seven percent of patients with STEMI do not have a critical coronary artery lesion [24], including approximately three percent who have normal epicardial coronary arteries [25-27]. The prevalence is greater in younger patients and in women [24]. Potential mechanisms that can be identified in some of these patients include coronary spasm, acquired or inherited coagulation disorders, toxins such as cocaine, collagen vascular disease, embolism, myocarditis, and microvascular disease [26]. The prevalence of lack of a critical lesion or normal epicardial coronary arteries may also be higher in referral populations, due in part to misinterpretation of the presenting ECG (respective values 14 and 9.5 percent, respectively, in a review of 1335 referred patients) [28]. (See "Coronary heart disease and myocardial infarction in young men and women" and "Variant angina" and "Cardiovascular complications of cocaine abuse" and "Cardiac syndrome X: Angina pectoris with normal coronary arteries", section on 'Acute coronary syndrome' and "Clinical manifestations and diagnosis of myocarditis in adults", section on 'Electrocardiogram'.)

Stress-induced cardiomyopathy (takotsubo cardiomyopathy), is an increasingly reported syndrome generally characterized by transient systolic dysfunction of the apical and/or mid segments of the left ventricle that mimics myocardial infarction (MI), but in the absence of significant coronary artery disease. This issue is discussed elsewhere. (See "Stress-induced (takotsubo) cardiomyopathy".)

**SUMMARY** — Acute ST elevation myocardial infarction (STEMI) is a medical emergency requiring the simultaneous application of multiple therapies. After the emergent period, other therapies may need to be started. (See "Overview of the non-acute management of acute ST elevation myocardial infarction".)

Women should be managed similarly to men. Other patients with acute STEMI, such as the elderly and those with either cocaine associated MI or possible stent thrombosis, are managed somewhat differently. (See 'General principles' above.)

The initial assessment and therapy of STEMI is summarized in table form (table 1).

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**REFERENCES**


Rapid overview: Management of acute coronary syndrome (ACS)

**Initial assessment:**

- Consider the diagnosis: women, the elderly, and patients with diabetes may have atypical presentations
- Obtain 12 lead ECG within 10 minutes of arrival; repeat every 10 minutes if initial ECG nondiagnostic but clinical suspicion remains high (initial ECG often **NOT** diagnostic)
  1. STEMI: ST segment elevations of 1 mm (0.1 mV) in 2 anatomically contiguous leads or 2 mm (0.2 mV) in 2 contiguous precordial leads, **OR** new left bundle branch block and presentation consistent with ACS. If ECG suspicious but not diagnostic, consult cardiology early.
  2. Non-STEMI or unstable angina: ST segment depressions or deep T wave inversions without Q waves or possibly no ECG changes
- Obtain emergent cardiology consultation for ACS patients with cardiogenic shock, left heart failure, or sustained ventricular tachyarrhythmia

**Initial interventions:**

- Assess and stabilize airway, breathing, and circulation
- Provide oxygen; attach cardiac and oxygen saturation monitors; establish IV access
- Treat sustained ventricular arrhythmia rapidly according to ACLS protocols
- Give aspirin 325 mg (non-enteric coated), to be chewed and swallowed (unless aortic dissection is being considered). If oral administration not feasible, give as rectal suppository.
- Perform focused history and examination: Look for signs of hemodynamic compromise and left heart failure; determine baseline neurologic function, particularly if fibrinolytic therapy is to be given
- Obtain blood for cardiac biomarkers (troponin preferred), electrolytes, coagulation studies, hematocrit/hemoglobin
- Treat left heart failure: Give afterload reducing agent (eg, nitroglycerin sublingual tablet and/or IV drip at 40 mcg/minute provided no phosphodiesterase inhibitors [eg, for erectile dysfunction]); titrate drip up quickly based on response; give loop diuretic (eg, furosemide 80 mg IV)
- Give three sublingual nitroglycerin tablets (0.4 mg) one at a time, spaced five minutes apart, or one aerosol spray under tongue every 5 minutes for three doses **IF** patient has persistent chest discomfort, hypertension, or signs of heart failure **AND** there is no sign of hemodynamic compromise (eg, right ventricular infarction) and no use of phosphodiesterase inhibitors (eg, for erectile dysfunction); add IV nitroglycerin for persistent symptoms
- Give beta blocker (eg, metoprolol 25 mg orally) **IF** no signs of heart failure (or high risk for heart failure), hemodynamic compromise, bradycardia, or severe reactive airway disease. If hypertensive, may initiate beta blocker IV instead (eg, metoprolol 5 mg intravenous every 5 minutes times three dose as tolerated).
- Give morphine sulfate (2 to 4 mg slow IV push every 5 to 15 minutes) for persistent discomfort or anxiety
- Start 80 mg of atorvastatin as early as possible, and preferably before PCI, in patients not on statin

**Acute management STEMI:**

- Select reperfusion strategy: Primary percutaneous coronary intervention (PCI) strongly preferred, especially for patients with cardiogenic shock, heart failure, late presentation, or contraindications to fibrinolysis. Activate cardiac catheterization team as indicated. For patients with symptoms of >12 hours, fibrinolytic therapy is not indicated, but emergent PCI may be considered, particularly for patients with evidence of ongoing ischemia or
PCI may be considered, particularly for patients with evidence of ongoing ischemia or those at high risk.

- Treat with fibrinolysis if PCI unavailable within 90-120 minutes, symptoms <12 hours, and no contraindications

- Give antiplatelet therapy (in addition to aspirin) to all patients:
  1. Prasugrel: For patients undergoing primary PCI and NOT at high risk of bleeding (age <75 years, weight >60 kilograms, no stroke or TIA), give prasugrel 60 mg
  2. Clopidogrel: For patients undergoing primary PCI who are not candidates for prasugrel due to a high risk of bleeding, give clopidogrel 600 mg. (Some patients, such as those with recent GI bleed or head trauma, may not be candidates for either). For patients undergoing fibrinolysis or no reperfusion therapy, give clopidogrel loading dose of 300 mg if age less than 75 years; if age 75 years or older, give 75 mg daily dose only.
  3. Glycoprotein IIb/IIIa inhibitor (GPIIb/IIIa): If PCI is planned give GPIIb/IIIa in consultation with cardiology. Patients who receive bivalirudin should not receive GPIIb/IIIa.

- Give anticoagulant therapy to all patients:
  1. Unfractionated heparin (UFH): For patients undergoing primary PCI who are receiving a GP IIb/IIIa inhibitor, we suggest an IV bolus of 50 to 70 units/kg (target activated clotting time >200 seconds) up to a maximum of 4000 units. For those patients not receiving a GP IIb/IIIa inhibitor, we suggest an intravenous bolus of 60 to 100 units/kg up to a maximum of 4000 units (give after fibrinolytic therapy).
  2. Bivalirudin is an acceptable alternative to heparin plus GP IIb/IIIa in patients undergoing primary PCI. Initial bolus of 0.75 mg/kg IV followed by IV infusion of 1.75 mg/kg per hour; can be discontinued after PCI.
  3. Enoxaparin: For patients not managed with PCI and <75 years and whose serum creatinine is <2.5 mg/dL [220 micromol/L] in men and <2.0 mg/dL [175 micromol/L] in women, give a loading dose of 30 mg IV bolus and 1 mg/kg subcutaneously every 12 hours. The creatinine clearance should be used to determine the timing of subsequent doses. UFH is preferred in patients with dialysis dependent renal failure.

**Acute management of unstable angina or non-STEMI:**

- Give antiplatelet therapy (in addition to aspirin):
  1. Thienopyridine: If the patient is undergoing same day PCI, give clopidogrel 600 mg; for patients undergoing early PCI in whom thienopyridine is withheld before PCI AND who are not at high risk of bleeding (age <75 years, weight ≥60 kilograms, no history of stroke or TIA) give prasugrel 60 mg instead of clopidogrel
  2. GP IIb/IIIa inhibitor (either eptifibatide or tirofiban) may be added to or in place of clopidogrel in some circumstances (eg, high risk patient). Abciximab is contraindicated in patients not referred for cardiac catheterization.

- Give anticoagulant therapy in all patients:
  1. UFH is preferred, particularly for emergent PCI; give an IV bolus of 50 to 60 units/kg followed by 12 units/kg per hour IV (goal aPTT time of 1.5 to 2 times control or approximately 50 to 75 seconds)
  2. Enoxaparin is an alternative to UFH for patients not undergoing early PCI: Give as described in the STEMI section above, using the regimen for patients with STEMI who receive fibrinolytics
  3. Fondaparinux is an alternative to enoxaparin in patients with NSTEMI at increased risk of bleeding being managed without PCI or fibrinolysis. The dose is 2.5 mg subcutaneously.
  4. Bivalirudin is an acceptable alternative to UFH in patients going for PCI. It is given as an IV bolus of 0.1 mg/kg and an infusion of 0.25 mg/kg per hour before angiography. If used, bivalirudin is combined with oral clopidogrel 600 mg.

**Other considerations:**

- Cocaine-related ACS: Give benzodiazepines (eg, Lorazepam 2 to 4 mg IV every 15 minutes or so) as needed to alleviate symptoms; do NOT give beta blockers
- Stop NSAID therapy if possible
- Correct any electrolyte abnormalities, especially hypokalemia and hypomagnesemia
TIMI grade 3 coronary flow is associated with improved survival after thrombolysis

In the GUSTO-I trial, the 30 day mortality rate after thrombolysis for acute ST elevation myocardial infarction varied with the degree of vessel patency achieved. The mortality was lowest (4.3 percent) in patients with TIMI grade 3 (normal) flow in the infarct-related artery at 90 minutes. Partial restoration of flow (TIMI grade 2) did not improve outcomes compared to no or faint flow (TIMI grade 0 or 1). Data from The GUSTO Investigators, N Engl J Med 1993; 329:673.
**ACC/AHA and ACC/AHA/SCAI guideline summary: Considerations for selecting primary percutaneous coronary intervention (PCI) for reperfusion therapy in patients with ST elevation myocardial infarction (STEMI)**

### Class I - There is evidence and/or general agreement that primary PCI for reperfusion therapy should be performed in patients with STEMI (including true posterior MI or MI with new or presumably new left bundle branch block) in the following settings:

- **General considerations:**
  1. If immediately available, primary PCI of the infarct-related artery should be performed when the procedure can be initiated within 12 hours of symptom onset and in a timely fashion (balloon inflation within 90 minutes of presentation) by skilled operators (individuals who perform more than 75 PCI procedures per year).
  2. The procedure should be supported by experienced personnel in an appropriate laboratory environment, defined as a laboratory that performs more than 200 PCI procedures per year, of which at least 36 are primary PCI for STEMI, and has cardiac surgery capability.

- **Specific considerations:**
  1. With each of the following indications, primary PCI should be performed as quickly as possible with a goal of a medical contact-to-balloon or door-to-balloon interval of 90 minutes or less.
  2. If the symptom duration is within three hours and the expected door-to-balloon time minus the expected door-to-needle time for fibrinolytic therapy is:
     - a. Within one hour, primary PCI is generally preferred.
     - b. Greater than one hour, fibrinolytic therapy with a fibrin-specific agent is generally preferred.
  3. If the symptom duration is greater than three hours, primary PCI is generally preferred.
  4. Primary PCI should be performed in patients less than 75 years of age 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 or unsuitability for further invasive care.
  5. Primary PCI should be performed in patients with severe HF and/or pulmonary edema (Killip class 3) and symptom onset within 12 hours.

### Class IIa - The evidence/opinion is in favor of the efficacy of primary PCI for reperfusion therapy in patients with STEMI (including true posterior MI or MI with new or presumably new left bundle branch block) in the following settings:

- Selected patients ≥75 years of age who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock. Criteria for selection include good prior functional status, suitability for revascularization, and patient agreement to invasive therapy.
- Patients seen within 12 to 24 hours of symptom onset who have one or more of the following features: severe heart failure; hemodynamic or electrical instability; and/or evidence of persistent ischemia.

### Class IIb - The evidence/opinion is less well established for the efficacy of primary PCI for reperfusion therapy in patients with STEMI (including true posterior MI or MI with new or presumably new left bundle branch block) in the following setting:

- Among patients who are eligible for fibrinolysis, performance of primary PCI by an operator who does less than 75 PCI procedures per year.

### Class III - There is evidence and/or general agreement that primary PCI for reperfusion therapy in patients with STEMI (including true posterior MI or MI with new or presumably new left bundle branch block) may not be useful and, in some cases, may be harmful in the following settings:
**Following settings:**

- Primary PCI should **NOT** be performed in asymptomatic patients who present more than 12 hours after symptom onset who are hemodynamically and electrically stable.

- Among patients undergoing primary PCI who do not have hemodynamic compromise, concurrent elective PCI in a non-infarct-related artery should **NOT** be performed.

# Absolute and relative contraindications to the use of thrombolytic therapy in patients with acute ST elevation myocardial infarction*

## Absolute contraindications

<table>
<thead>
<tr>
<th>Contraindication</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of any intracranial hemorrhage</td>
</tr>
<tr>
<td>History of ischemic stroke within the preceding three months, with the important exception of acute ischemic stroke seen within three hours which may be treated with thrombolytic therapy</td>
</tr>
<tr>
<td>Presence of a cerebral vascular malformation or a primary or metastatic intracranial malignancy</td>
</tr>
<tr>
<td>Symptoms or signs suggestive of an aortic dissection</td>
</tr>
<tr>
<td>A bleeding diathesis or active bleeding, with the exception of menses; thrombolytic therapy may increase the risk of moderate bleeding, which is offset by the benefits of thrombolysis</td>
</tr>
<tr>
<td>Significant closed-head or facial trauma within the preceding three months</td>
</tr>
</tbody>
</table>

## Relative contraindications

<table>
<thead>
<tr>
<th>Contraindication</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of chronic, severe, poorly controlled hypertension or uncontrolled hypertension at presentation (blood pressure &gt;180 mmHg systolic and/or &gt;110 mmHg diastolic; severe hypertension at presentation can be an absolute contraindication in patients at low risk)</td>
</tr>
<tr>
<td>History of ischemic stroke more than three months previously</td>
</tr>
<tr>
<td>Dementia</td>
</tr>
<tr>
<td>Any known intracranial disease that is not an absolute contraindication</td>
</tr>
<tr>
<td>Traumatic or prolonged (&gt;10 min) cardiopulmonary resuscitation</td>
</tr>
<tr>
<td>Major surgery within the preceding three weeks</td>
</tr>
<tr>
<td>Internal bleeding within the preceding two to four weeks or an active peptic ulcer</td>
</tr>
<tr>
<td>Noncompressible vascular punctures</td>
</tr>
<tr>
<td>Pregnancy</td>
</tr>
<tr>
<td>Current warfarin therapy - the risk of bleeding increases as the INR increases</td>
</tr>
<tr>
<td>For streptokinase or anistreplase - a prior exposure (more than five days previously) or allergic reaction to these drugs</td>
</tr>
</tbody>
</table>

Cooperative cardiovascular project risk model for intracranial hemorrhage with thrombolytic therapy

<table>
<thead>
<tr>
<th>Risk Factors*</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Age ≥75 years</td>
<td></td>
</tr>
<tr>
<td>Black race</td>
<td></td>
</tr>
<tr>
<td>Female sex</td>
<td></td>
</tr>
<tr>
<td>Prior history of stroke</td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure ≥160 mmHg</td>
<td></td>
</tr>
<tr>
<td>Weight ≤65 kg for women or ≤80 kg for men</td>
<td></td>
</tr>
<tr>
<td>INR &gt;4 or PT &gt;24•</td>
<td></td>
</tr>
<tr>
<td>Use of alteplase (versus other thrombolytic agent)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Risk score</th>
<th>Rate of intracranial hemorrhage, percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 or 1</td>
<td>0.69</td>
</tr>
<tr>
<td>2</td>
<td>1.02</td>
</tr>
<tr>
<td>3</td>
<td>1.63</td>
</tr>
<tr>
<td>4</td>
<td>2.49</td>
</tr>
<tr>
<td>≥5</td>
<td>4.11</td>
</tr>
</tbody>
</table>

* Each risk factor is worth 1 point if present, 0 points if absent. Points are added to determine the risk score.

**ACC/AHA guidelines for management of ST elevation myocardial infarction: Recommendations for thrombolysis**

### Class I

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

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

### Class IIa

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

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

### Class III

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

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

### ACC/AHA classification

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**Class III:** Conditions for which there is evidence and/or general agreement that the procedure/treatment is not useful and in some cases may be harmful.

### ACC/AHA guidelines for management of ST elevation myocardial infarction: Contraindications, cautions, and complications of thrombolysis

**Class I**

1. Healthcare providers should ascertain whether the patient has neurological contraindications to fibrinolytic therapy, including any history of intracranial hemorrhage (ICH), significant closed head or facial trauma within the past 3 months, uncontrolled hypertension, or ischemic stroke within the past 3 months. (Level of Evidence: A)

2. STEMI patients at substantial (greater than or equal to 4 percent) risk of ICH should be treated with PCI rather than with fibrinolytic therapy. (Level of Evidence: A)

3. The occurrence of a change in neurological status during or after reperfusion therapy, particularly within the first 24 hours after initiation of treatment, is considered to be due to ICH until proven otherwise. Fibrinolytic, antiplatelet, and anticoagulant therapies should be discontinued until brain imaging scan shows no evidence of ICH. (Level of Evidence: A)

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

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

**Class IIa**

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

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# Preferred thrombolytic regimens for acute ST elevation myocardial infarction

<table>
<thead>
<tr>
<th>Drug</th>
<th>Recommended IV regimen*</th>
<th>Advantages and limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Streptokinase</td>
<td>1.5 million units over 30 to 60 minutes</td>
<td>Generally much less costly but outcomes inferior. Used extensively in many countries due to lower cost.</td>
</tr>
<tr>
<td>Alteplase (accelerated)</td>
<td>15 mg bolus then 0.75 mg/kg (maximum 50 mg) over 30 minutes then 0.5 mg/kg (maximum 35 mg) over the next 60 minutes</td>
<td>Better outcomes than streptokinase (SK) in GUSTO-1 (30 day mortality 6.3 versus 7.3 percent); more expensive than SK; more difficult to administer because of short half-life.</td>
</tr>
<tr>
<td>Tenecteplase</td>
<td>Single bolus over five to ten seconds based upon body weight:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&lt;60 kg = 30 mg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>60 to 69 kg = 35 mg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>70 to 79 kg = 40 mg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>80 to 89 kg = 45 mg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>≥90 kg = 50 mg</td>
<td>As effective as alteplase in ASSENT-2 with less noncerebral bleeding and need for transfusion; easier to administer (single bolus due to longer half-life) both in and out of hospital; these advantages make tenecteplase the drug of choice in many hospitals in the United States.</td>
</tr>
<tr>
<td>Reteplase</td>
<td>10 U over two minutes then repeat 10 U bolus at 30 minutes</td>
<td>Similar outcomes as alteplase but easier to administer.</td>
</tr>
</tbody>
</table>

* All patients are also given aspirin and, with alteplase, reteplase, and tenecteplase, unfractionated heparin as a 60 U/kg bolus (maximum 4000 U) followed by an intravenous infusion of 12 U/kg per hour (maximum 1000 U/hour). Heparin has not been definitively shown to improve outcomes with non-fibrin-specific agents such as streptokinase. However, heparin is recommended with streptokinase in patients who are at high risk for systemic thromboembolism (large or anterior myocardial infarction, atrial fibrillation, previous embolus, or known left ventricular thrombus).
# ACC/AHA Guideline Summary: Coronary artery bypass graft surgery (CABG) in patients with ST elevation myocardial infarction (STEMI)

## Class I - There is evidence and/or general agreement that CABG should be performed in patients with STEMI in the following settings

- The following are indications for emergent or urgent CABG:
  1. Failed percutaneous coronary intervention (PCI) with persistent pain or hemodynamic instability if coronary anatomy is suitable for surgery.
  2. Persistent or recurrent ischemia refractory to medical therapy if coronary anatomy is suitable for surgery, a significant area of myocardium is at risk, and the patient is not a candidate for PCI.
  3. At the time of surgical repair of postinfarction ventricular septal rupture or mitral regurgitation.
  4. Cardiogenic shock in patients less than 75 years of age who develop shock within 36 hours of MI and are suitable and appropriate candidates for revascularization that can be performed within 18 hours of shock.
  5. Life-threatening ventricular arrhythmias in the presence of at least 50 percent left main stenosis and/or triple-vessel disease.

- If possible, an internal mammary artery graft should be used to bypass a significantly stenosed left anterior descending artery.

## Class IIa - The weight of evidence or opinion is in favor of benefit from CABG in patients with STEMI in the following settings

- For primary reperfusion in patients who have suitable anatomy, are not candidates for or have failed fibrinolysis/PCI, and are in the first 6 to 12 hours of an evolving STEMI.

- Since CABG mortality is elevated for the first 3 to 7 days after infarction, the benefit of revascularization must be balanced against this risk. Patients who are stable (no ongoing ischemia, hemodynamic compromise, or life-threatening arrhythmia) and who have incurred a significant fall in left ventricular function should have their surgery delayed to allow myocardial recovery to occur. If critical anatomy exists, revascularization should be performed during the index hospitalization.

## Class III - There is evidence and/or general agreement that emergency CABG should not be performed in patients with STEMI in the following settings

- Persistent angina and a small area of myocardium at risk in hemodynamically stable patients.

- Successful epicardial reperfusion in the absence of successful microvascular reperfusion.

Recommendations for atropine (from early after onset of acute myocardial infarction to 6 to 8 hours afterward)

<table>
<thead>
<tr>
<th>Class I</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Sinus bradycardia with evidence of low cardiac output and peripheral hypoperfusion or frequent premature ventricular complexes at onset of symptoms of acute myocardial infarction (AMI).</td>
</tr>
<tr>
<td>2. Acute inferior infarction with type I second- or third-degree atrioventricular (AV) block associated with symptoms of hypotension, ischemic discomfort, or ventricular arrhythmias.</td>
</tr>
<tr>
<td>3. Sustained bradycardia and hypotension after administration of nitroglycerin.</td>
</tr>
<tr>
<td>4. For nausea and vomiting associated with administration of morphine.</td>
</tr>
<tr>
<td>5. Ventricular asystole.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Class IIa</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Symptomatic patients with inferior infarction and type I second- or third-degree heart block at the level of the AV node (ie, with narrow QRS complex or with known existing bundle branch block).</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Class IIb</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Administration concomitant with (before or after) administration of morphine in the presence of sinus bradycardia.</td>
</tr>
<tr>
<td>2. Asymptomatic patients with inferior infarction and type I second-degree heart block or third-degree heart block at the level of the AV node.</td>
</tr>
<tr>
<td>3. Second- or third-degree AV block of uncertain mechanism when pacing is not available.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Class III</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Sinus bradycardia greater than 40 bpm without signs or symptoms of hypoperfusion or frequent premature ventricular contractions.</td>
</tr>
<tr>
<td>2. Type II AV block and third-degree AV block with new wide QRS complex presumed due to AMI.</td>
</tr>
</tbody>
</table>

**ACC/AHA classification**

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Recommendations for atropine after a myocardial infarction

The following recommendations are applicable > 8 hours after presentation:

**Class I**

1. Symptomatic sinus bradycardia (generally, heart rate less than 50 bpm associated with hypotension, ischemia, or escape ventricular arrhythmia).

2. Ventricular asystole.

3. Symptomatic atrioventricular (AV) block occurring at the AV nodal level (second-degree type I or third-degree with a narrow-complex escape rhythm).

**Class IIa**

None.

**Class III**

1. AV block occurring at an infranodal level (usually associated with anterior myocardial infarction with a wide-complex escape rhythm).

2. Asymptomatic sinus bradycardia.

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Recommendations for placement of transcutaneous patches* and active (demand) transcutaneous pacing in myocardial infarction•

Class I
1. Sinus bradycardia (rate <50 bpm) with symptoms of hypotension (systolic blood pressure <80 mmHg) unresponsive to drug therapy.•
2. Mobitz type II second-degree atrioventricular (AV) block.•
3. Third-degree heart block.•
4. Bilateral bundle branch block (BBB) (alternating BBB, or RBBB and alternating left anterior fascicular block [LAFB], left posterior fascicular block [LPFB]) (irrespective of time of onset).*
5. Newly acquired or age indeterminate LBBB, LBBB and LAFB, RBBB and LPFB.*
6. RBBB or LBBB and first-degree AV block.*

Class IIa
1. Stable bradycardia (systolic blood pressure >90 mmHg, no hemodynamic compromise, or compromise responsive to initial drug therapy).*
2. Newly acquired or age-indeterminate RBBB.*

Class IIb
1. Newly acquired or age-indeterminate first-degree AV block.*

Class III
1. Uncomplicated AMI without evidence of conduction system disease.

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* Apply patches and attach system; system is in either active or standby mode to allow immediate use on demand as required. In facilities in which transvenous pacing or expertise are not available to place an IV system, consideration should be given to transporting the patient to one equipped and competent in placing transvenous systems.
• Transcutaneous patches applied; system may be attached and activated within a brief time if needed. Transcutaneous pacing may be very helpful as an urgent expedient. Because it is associated with significant pain, high-risk patients likely to require pacing should receive a temporary pacemaker. Adapted from Ryan, TJ, Anderson, JL, Antman, EM, et al. J Am Coll Cardiol 1996; 28:1328.
# Recommendations for temporary transvenous pacing in acute myocardial infarction

<table>
<thead>
<tr>
<th>Class I</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Asystole.</td>
<td></td>
</tr>
<tr>
<td>2. Symptomatic bradycardia (includes sinus bradycardia with hypotension and type I second-degree atrioventricular [AV] block with hypotension not responsive to atropine).</td>
<td></td>
</tr>
<tr>
<td>3. Bilateral bundle branch block (BBB) (alternating BBB or RBBB with alternating LAFB/LPFB) (any age).</td>
<td></td>
</tr>
<tr>
<td>4. New or indeterminate age bifascicular block (RBBB with LAFB or LPFB, or LBBB) with first-degree AV block.</td>
<td></td>
</tr>
<tr>
<td>5. Mobitz type II second-degree AV block.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Class IIa</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. RBBB and LAFB or LPFB (new or indeterminate).</td>
<td></td>
</tr>
<tr>
<td>2. RBBB with first-degree AV block.</td>
<td></td>
</tr>
<tr>
<td>3. LBBB, new or indeterminate.</td>
<td></td>
</tr>
<tr>
<td>4. Incessant ventricular tachycardia, for atrial or ventricular overdrive pacing.</td>
<td></td>
</tr>
<tr>
<td>5. Recurrent sinus pauses (&gt;3 seconds) not responsive to atropine</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Class IIb</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Bifascicular block of indeterminate age.</td>
<td></td>
</tr>
<tr>
<td>2. New or age-indeterminate isolated RBBB.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Class III</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. First-degree heart block.</td>
<td></td>
</tr>
<tr>
<td>2. Type I second-degree AV block with normal hemodynamics.</td>
<td></td>
</tr>
<tr>
<td>3. Accelerated idioventricular rhythm.</td>
<td></td>
</tr>
<tr>
<td>4. BBB or fascicular block known to exist before AMI.</td>
<td></td>
</tr>
</tbody>
</table>

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It should be noted that in choosing an intravenous pacemaker system, patients with substantially depressed ventricular performance, including right ventricular infarction, may respond better to atrial/AV sequential pacing than ventricular pacing. *Adapted from Ryans, TJ, Anderson, JL, Antman, EM, et al. J Am Coll Cardiol 1996; 28:1328.*
## Recommendations for permanent pacing after acute myocardial infarction

**Class I**

1. Persistent second-degree atrioventricular (AV) block in the His-Purkinje system with bilateral bundle branch block (BBB) or complete heart block after acute myocardial infarction.
2. Transient advanced (second- or third-degree) AV block and associated BBB.*
3. Symptomatic AV block at any level.

**Class IIb**

1. Persistent advanced (second- or third-degree) block at the AV node level.

**Class III**

1. Transient AV conduction disturbances in the absence of intraventricular conduction defects.
2. Transient AV block in the presence of isolated left anterior fascicular block (LAFB).
3. Acquired LAFB in the absence of AV block.
4. Persistent first-degree AV block in the presence of BBB that is old or age-indeterminate.

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