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Overview of the acute management of ST-elevation myocardial infarction

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INTRODUCTION

Patients with severe and acute myocardial infarction (ie, ST-elevation myocardial infarction [STEMI]) require rapid diagnosis and treatment to reduce the risk of death and permanent myocardial injury [1].

This topic provides an overview of STEMI management from presentation to the period immediately after revascularization.

Aspects of management that typically arise in the hours to days after revascularization are discussed in a separate topic. (See "Overview of the nonacute management of ST-elevation myocardial infarction".)

The initial evaluation of chest pain in the emergency department and the diagnosis and management of non-ST-elevation MI are covered in separate topics:

- (See "Evaluation of the adult with chest pain in the emergency department".)
- (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department".)
- (See "Overview of the acute management of non-ST-elevation acute coronary syndromes".)

GOALS OF THERAPY

The primary goal of STEMI management is to reduce the risk of death and the extent of permanent cardiac injury associated with MI. Because therapy for patients with STEMI becomes less effective with each minute its delivery is delayed (figure 1), another goal of therapy is to rapidly treat patients with STEMI before treatment becomes ineffective.

INITIAL ASSESSMENT

All patients with chest pain (acute coronary syndrome [ACS]) should have an initial assessment (ie, electrocardiogram [ECG], history, physical examination) to rapidly confirm or exclude the diagnosis of STEMI and to identify other conditions that would change management.

Rapid diagnosis of STEMI — The rapid diagnosis of STEMI only requires the presence of symptoms suspicious for an ACS (eg, chest discomfort, dyspnea, sudden death) and a confirmatory ECG; it does not require evidence of elevated cardiac biomarkers such as troponin. Thus, patients with suspected ACS should undergo a focused history, physical, and ECG within ten minutes of hospital arrival to identify the key findings of STEMI (table 1):

- **Characteristic symptoms and signs** – The following signs and symptoms suggest the presence of STEMI:

  - Chest pain or chest discomfort
  - Dyspnea
  - Ventricular arrhythmias, cardiac arrest, or syncope
  - Atypical symptoms such as malaise, weakness, and back pain

  The evaluation of these symptoms is discussed separately. (See "Evaluation of the adult with chest pain in the emergency department", section on 'History' and "Diagnosis of acute myocardial infarction", section on 'History and physical examination'.)

- **ECG findings** – ECGs should be reviewed for signs of severe myocardial ischemia, which include:

  - ST-segment elevation with standard lead placement (waveform 1 and waveform 2)
  - Newly identified left bundle branch block (waveform 3)
  - ST elevation with posterior (waveform 4) or right-sided (waveform 5) lead placement

- Other high-risk ECG findings (eg, de Winter sign, transient ST-segment elevation)

A detailed description of each of these ECG findings can be found elsewhere. (See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction".)

Monitoring and testing — For patients with STEMI, initial monitoring and testing typically include the following (table 1):

- **General measures** – Patients with STEMI require frequent blood pressure measurements, continuous heart rhythm monitoring, and continuous pulse oximetry. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department", section on 'Immediate emergency department interventions'.)

- **Laboratory studies** – All patients with STEMI should have laboratory studies to evaluate for metabolic abnormalities, acute kidney injury, anemia, thrombocytopenia, and coagulopathy. Troponin levels should be obtained, but the acute management of STEMI does not require elevated troponin levels. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department", section on 'Cardiac biomarkers and other laboratory testing'.)

- **Imaging studies** – Patients with STEMI should have a chest radiograph to evaluate for other causes of chest discomfort and to assess for the complications of MI (eg, pulmonary edema). (See "Evaluation of the adult with chest pain in the emergency department".)

  Echocardiography, computed tomographic angiography, and other imaging studies are not routinely obtained unless a specific diagnosis is suspected (eg, aortic dissection, pericardial tamponade). (See 'Evaluation for life-threatening conditions' below and "Role of echocardiography in acute myocardial infarction".)

Evaluation for life-threatening conditions — The initial assessment of patients with STEMI includes a brief evaluation for conditions that require additional treatment or that alter the approach to STEMI therapy. These conditions include:

- **Shock** – Patients with STEMI should be assessed for evidence of shock and, if present, for signs or symptoms (eg, cool extremities, jugular venous distension) that help to characterize the type of shock (ie, cardiogenic, distributive). Patients with shock require specific management of shock as well as appropriate and timely reperfusion. The clinical manifestations and causes of shock, including the mechanical complications of STEMI, are discussed separately. (See "Clinical manifestations and diagnosis of cardiogenic shock in

acute myocardial infarction" and "Evaluation of and initial approach to the adult patient with undifferentiated hypotension and shock".)

- **Heart failure** – All patients with STEMI should be assessed for signs and symptoms of heart failure (HF; eg, orthopnea, jugular venous distension, pulmonary edema). The causes, clinical manifestations, and treatment of acutely decompensated HF are covered separately. (See "Approach to diagnosis and evaluation of acute decompensated heart failure in adults".)

- **Aortic dissection** – Aortic dissection is a rare cause of STEMI but should be considered in all patients with STEMI. The signs and symptoms of aortic dissection include severe pain or tearing located in the chest or back, asymmetric upper extremity pulses or pulse deficits, new aortic valve murmurs, and widening of the mediastinum on chest radiograph. Patients with STEMI caused by aortic dissection require management that is different from the typical management of STEMI. (See "Overview of acute aortic dissection and other acute aortic syndromes".)

- **Coagulopathy and/or thrombocytopenia** – The management of patients with STEMI typically requires treatments that increase the risk of bleeding. Thus, all patients with STEMI should be assessed for chronic use of anticoagulant or antiplatelet medications, history of bleeding or coagulation disorders (eg, uremia, heparin-induced thrombocytopenia), and the presence of abnormal coagulation studies or thrombocytopenia. The approach to assessing the risk of bleeding in patents with ACS can be found elsewhere. (See "High bleeding risk patients undergoing percutaneous coronary intervention", section on 'Assessing individual patient risk' and "Acute ST-elevation myocardial infarction: The use of fibrinolytic therapy", section on 'Contraindications'.)

INITIAL MANAGEMENT

The initial management of patients with STEMI requires rapid selection and administration of reperfusion therapy. Patients with STEMI should also receive treatments that prevent further coronary artery thrombosis, minimize myocardial injury, and treat the symptoms of MI.

Choosing and initiating reperfusion with PCI or fibrinolysis — For patients diagnosed with STEMI, the primary goal of acute management is to rapidly restore blood flow to the acutely occluded coronary artery (ie, culprit artery) with a reperfusion therapy (ie, percutaneous coronary intervention [PCI], fibrinolysis) (algorithm 1). Thus, a reperfusion strategy should be chosen within minutes of arrival. After a reperfusion strategy is chosen, local STEMI protocols

should be initiated without delay; these protocols mobilize the key personnel (eg, interventional cardiologist, pharmacist) and resources (eg, interhospital transfer) required to deliver reperfusion therapy as quickly as possible.

Key factors that influence the choice of reperfusion strategy include:

- Time delay between first medical contact and performance of PCI
- Time from symptom onset to presentation
- Presence of cardiogenic shock
- Diagnostic uncertainty
- Contraindications to PCI or fibrinolysis
- High-risk factors that favor no reperfusion

The details on the approach to selecting a reperfusion strategy can be found elsewhere. (See "Acute ST-elevation myocardial infarction: Selecting a reperfusion strategy".)

Routine medical therapy — Regardless of the chosen reperfusion strategy, the following therapies are routinely given to patients with STEMI to slow the progression of coronary artery thrombus formation, minimize the extent of myocardial injury, and treat symptoms (table 1):

- **Aspirin** – All patients with STEMI should receive aspirin as soon as possible. Further details on the use of aspirin in patients with STEMI, including patients with aspirin allergy, can be found elsewhere. (See "Acute ST-elevation myocardial infarction: Antiplatelet therapy", section on 'Aspirin for all patients'.)

- **Nitrates** – In patients with STEMI, nitrates can reduce the symptoms of chest discomfort and HF as well as treat hypertension. However, nitrates can occasionally produce profound hypotension in patients with right ventricular infarction, aortic stenosis, or who recently used sildenafil. Further information on the use of nitrates in acute coronary syndromes (ACS) can be found elsewhere. (See "Nitrates in the management of acute coronary syndrome".)

- **Beta blockers** – Patients with STEMI who do not have shock, HF, bradycardia, or heart block typically receive an oral beta blocker as part of the initial therapy for STEMI. The type of agent, dosing, and evidence for use of beta blockers in ACS are discussed separately. (See "Acute myocardial infarction: Role of beta blocker therapy", section on 'Choice of drug and route of administration'.)

- **Anticoagulation and additional antiplatelet agents** – Most patients with STEMI receive treatment with an anticoagulant and a $P2Y_{12}$ inhibitor. However, the approach to the use

and selection of these agents is determined by the reperfusion strategy and other patient characteristics. Anticoagulant and $P2Y_{12}$ inhibitor therapy for each reperfusion strategy are reviewed elsewhere in this topic. (See 'Subsequent management by reperfusion strategy' below.)

- **Statins** – In patients with STEMI who do not already take a statin, a statin is typically started soon after presentation to the hospital. The type and dose of statin are discussed separately. (See "Low-density lipoprotein-cholesterol (LDL-C) lowering after an acute coronary syndrome", section on 'Inpatient management'.)

- **Morphine and oxygen** – The routine use of morphine and oxygen are discussed elsewhere. (See 'Therapies of unclear benefit' below.)

Therapy for specific symptoms and syndromes — In patients with STEMI, common symptoms and syndromes that require acute management include the following:

- **Chest discomfort and/or pain** – Patients with chest discomfort and/or pain typically receive initial treatment with nitrates (table 1). Patients with resistant chest symptoms who are hemodynamically stable may be sequentially treated with higher doses of nitrates and opioids. The approach to the use of nitrates and opioids in patients with ACS is discussed elsewhere. (See "Nitrates in the management of acute coronary syndrome" and 'Therapies of unclear benefit' below.)

- **Heart failure** – In addition to appropriate reperfusion, patients with STEMI and HF may require therapy for volume overload (eg, diuretics) and respiratory distress (eg, supplemental oxygen, positive pressure ventilation). The acute management of patients with HF is discussed elsewhere. (See "Treatment of acute decompensated heart failure: Specific therapies", section on 'Initial therapy'.)

- **Arrhythmias** – In patients with STEMI, the management of arrhythmias is focused on advanced cardiac life support (ACLS) for unstable patients, rapid reperfusion, and additional therapies that reduce the risk of arrhythmias.

  - Patients with life-threatening arrhythmias require emergency management (algorithm 2). (See "Advanced cardiac life support (ACLS) in adults".)

  - The treatment and prevention of arrhythmias in hemodynamically stable patients with acute MI are discussed elsewhere. (See "Ventricular arrhythmias during acute myocardial infarction: Prevention and treatment" and "Supraventricular arrhythmias after myocardial infarction".)

- **Right ventricular infarction** – Patients with right ventricular infarction may require additional management of complications such as bradyarrhythmias, hypotension, and shock. The complications associated with right ventricular infarction and their management are discussed elsewhere. (See "Right ventricular myocardial infarction".)

- **Cardiogenic shock** – The management of patients with STEMI and cardiogenic shock depends on the cause of cardiogenic shock (eg, HF, myocardial wall rupture). In addition to specific therapies for shock (eg, intraaortic balloon pump), patients with shock typically benefit from rapid reperfusion. The treatment of cardiogenic shock in patients with STEMI is discussed elsewhere. (See 'Choosing and initiating reperfusion with PCI or fibrinolysis' above and "Prognosis and treatment of cardiogenic shock complicating acute myocardial infarction".)

Therapies of unclear benefit — In patients with STEMI, the following therapies have no clear clinical benefit or may cause harm.

- **Routine morphine use** – In patients with STEMI, we only use morphine to treat chest symptoms refractory to nitrates and other medical therapy; we suggest not to routinely administer morphine to all patients with STEMI. (See 'Routine medical therapy' above.)

  The available observational data suggest that routine use of morphine does not reduce mortality, and limited trial data suggest that morphine may diminish the effect of $P2Y_{12}$ inhibitors [2-5]. Examples of these studies include:

  - In a study of 57,039 patients with STEMI, patients treated with morphine had a higher risk of mortality (5.5 versus 4.7 percent; propensity-matched odds ratio 1.4, 95% CI 1.3-1.6) [2]. The higher risk of mortality may be partly explained by selection bias (ie, patients with more severe ischemia were more likely to receive morphine).

  - In a trial of 70 patients with acute MI who were treated with ticagrelor, patients who were randomly assigned to receive intravenous morphine (5 mg) had lower levels of plasma ticagrelor and higher levels of platelet activity when compared with patients assigned to placebo [3].

- **Oxygen treatment despite normal oxygen saturation** – In patients with STEMI who have an oxygen saturation ≥94 percent and no signs of respiratory distress, we suggest **not** routinely treating with supplemental oxygen. Patients with lower oxygen saturation or respiratory distress should be treated with oxygen as needed. This approach is based on trials that did not demonstrate a benefit of empiric oxygen use:

- In the DETO2X-AMI trial, 6629 patients with suspected MI and an oxygen saturation of 90 percent or higher (median 97 percent, lower interquartile range 95 percent) were randomly assigned to receive either supplemental oxygen (6 L/min via facemask for 6 to 12 hours) or ambient air [6]. After 30 days of observation, the two groups had a similar risk of all-cause death (2.2 versus 2.0 percent in the ambient air group; hazard ratio [HR] 1.1, 95% CI 0.77-1.5) and rehospitalization for MI (1.4 versus 0.9 percent; HR 1.5, 95% CI 0.92-2.3). After one year of observation, the groups had a similar risk of the combined endpoint of all-cause death, rehospitalization for MI, or hospitalization for HF.

- In the 2015 AVOID trial, 441 patients with oxygen saturation ≥94 percent and a confirmed diagnosis of STEMI were randomly assigned to treatment with oxygen (8 L/min) or to no treatment with supplemental oxygen [7]. Peak troponin levels were nonsignificantly higher in the oxygen group (57 versus 48 mcg/L; means ratio 1.20, 95% CI 0.92-1.56), and the extent of myocardial scar measured by magnetic resonance imaging was greater in the oxygen group (20 versus 13 g of myocardial scar).

- **Prophylactic use of antiarrhythmics** – During the early phases of acute MI, ventricular arrhythmias (eg, premature depolarizations, nonsustained ventricular tachycardia) are common. However, antiarrhythmic agents (other than beta blockers) are not typically used to prevent ventricular arrhythmias in acute MI. Further details on antiarrhythmic drug use in the setting of acute STEMI are discussed elsewhere. (See "Ventricular arrhythmias during acute myocardial infarction: Prevention and treatment", section on 'Antiarrhythmic drugs'.)

- **Blood transfusion to treat mild anemia** – Red blood cell transfusion may be beneficial in select patients with STEMI. The approach to blood transfusion in patients with ACS is discussed separately. (See "Indications and hemoglobin thresholds for RBC transfusion in adults", section on 'Acute MI'.)

- **NSAIDs** – Nonsteroidal antiinflammatory drugs (NSAIDs; except aspirin) should not be used in the acute phases of STEMI management. The details on NSAID use in patients with known cardiovascular disease are discussed elsewhere. (See "NSAIDs: Adverse cardiovascular effects", section on 'Patients with known coronary heart disease'.)

SUBSEQUENT MANAGEMENT BY REPERFUSION STRATEGY

Patients undergoing PCI — Patients with STEMI who will undergo percutaneous coronary intervention (PCI) require assessment and treatment related to the PCI procedure. These aspects of care are discussed below in chronological order:

- **Prior to PCI** – Prior to PCI, patients with STEMI may require transfer to a PCI-capable center and/or appropriate treatment with additional antiplatelet and anticoagulant therapies (table 1).

  - **Patients who present at non-PCI capable centers** – Patients who initially present to a center that is not capable of PCI may require rapid transfer to a PCI-capable center for rapid reperfusion (algorithm 1). Additional information on issues related to transfer at the time of STEMI are discussed separately. (See "Primary percutaneous coronary intervention in acute ST elevation myocardial infarction: Determinants of outcome", section on 'Transfer from a non-PCI center'.)

  - **Choice of additional platelet inhibitors** – In patients undergoing PCI for treatment of STEMI, treatment with a $P2Y_{12}$ inhibitor (eg, clopidogrel, prasugrel, ticagrelor) may be indicated. The timing of therapy, choice of agent, and dosing of $P2Y_{12}$ inhibitors are discussed elsewhere. (See "Acute ST-elevation myocardial infarction: Antiplatelet therapy", section on 'Patients receiving primary PCI'.)

  - **Choice of anticoagulant** – Patients with STEMI who will undergo PCI typically receive an anticoagulant (eg, heparin, bivalirudin) prior to PCI. The approach to anticoagulation in patients undergoing reperfusion with PCI is discussed elsewhere. (See "Acute ST-elevation myocardial infarction: Management of anticoagulation", section on 'Primary percutaneous coronary intervention'.)

- **During PCI** – The PCI procedure consists of arterial access via the radial or femoral artery, diagnostic angiography, and interventional procedures (eg, stenting, thrombectomy) that open the acutely obstructed coronary artery. Additional treatments or management may be necessary depending on the patient's condition and angiography findings. The following are common scenarios encountered during the cardiac catheterization procedure:

  - **Acutely or chronically obstructed coronary arteries** – Diagnostic angiography may reveal acute lesions causative of MI ("culprit lesions") but may also reveal lesions that appear more chronic in nature ("nonculprit lesions"). The management of culprit and nonculprit lesions is discussed elsewhere. (See "Primary percutaneous coronary intervention in acute ST-elevation myocardial infarction: Periprocedural management",

section on 'Technical issues' and "Acute coronary syndromes: Approach to nonculprit lesions".)

- **Multivessel coronary artery disease** – In patients whose angiography demonstrates multivessel coronary artery disease (CAD) or left main CAD, the culprit lesions are typically treated with immediate PCI. In some patients with residual three-vessel disease or left main CAD, coronary artery bypass graft surgery (CABG) may be performed later. The timing and indications for CABG in patients with STEMI are discussed elsewhere. (See "Coronary artery bypass graft surgery in patients with acute ST-elevation myocardial infarction".)

- **Patients with shock or severe heart failure** – Patients with STEMI who have severe HF or cardiogenic shock may benefit from the placement of a pulmonary artery pressure monitor or a temporary mechanical circulatory support (tMCS) device during the PCI procedure. The indications for hemodynamic monitors and tMCS are discussed separately. (See "Pulmonary artery catheterization: Indications, contraindications, and complications in adults", section on 'Severe cardiogenic shock'.)

- **Patients without obstructive, atherosclerotic coronary artery disease** – Some patients with a presentation consistent with STEMI do not have obstructive atherosclerotic CAD at the time of coronary angiography. The diagnosis and management of conditions that can mimic the clinical presentation of STEMI are discussed elsewhere (table 2). (See "Myocardial infarction or ischemia with no obstructive coronary atherosclerosis".)

- **After PCI** – After performance of any interventional procedures, the next steps in management include:

  - **Optimal medical therapy** – Immediately after PCI, patients with STEMI may require changes (eg, duration, dose) to their anticoagulation or antiplatelet regimen. In addition, stable patients can begin long-term pharmacologic therapy for STEMI. These aspects of management are covered elsewhere:

    - Anticoagulant therapy. (See "Acute ST-elevation myocardial infarction: Management of anticoagulation", section on 'Primary percutaneous coronary intervention'.)

    - Antiplatelet therapy. (See "Acute ST-elevation myocardial infarction: Antiplatelet therapy", section on 'Duration of dual antiplatelet therapy'.)

- Long-term therapy. (See "Overview of the nonacute management of unstable angina and non-ST-elevation myocardial infarction", section on 'Inpatient medical therapy'.)

- **Monitoring for complications of PCI** – Patients who undergo coronary artery angiography with or without stenting should be monitored and treated for complications related to the procedure. Patients who undergo specialized coronary interventions (eg, rotational atherectomy) may be at higher risk for complications than patients who undergo standard PCI procedures. The clinical manifestations and management of PCI complications are discussed separately. (See "Periprocedural complications of percutaneous coronary intervention" and "Specialized revascularization devices in the management of coronary heart disease".)

- **Management of mechanical support devices** – Patients who undergo placement of a tMCS device require additional management and treatment that is different from patients who do not undergo tMCS device placement. The management of these devices and of their associated medical therapies (eg, anticoagulation) are discussed separately. (See "Short-term left ventricular mechanical circulatory support: Use of echocardiography during initiation and management" and "Short-term mechanical circulatory assist devices".)

Patients undergoing fibrinolysis — For patients who will undergo fibrinolysis, the goals of management are to safely administer fibrinolytics as soon as possible, monitor the response to fibrinolysis, and prepare for subsequent management (eg, transfer for PCI) (table 1).

- **Administration of fibrinolytic agents and related treatments** – The administration of fibrinolysis typically requires appropriate selection of an anticoagulant agent, fibrinolytic agent, and additional antiplatelet agent. The approach to choosing the fibrinolytic regimen is discussed separately. (See "Acute ST-elevation myocardial infarction: The use of fibrinolytic therapy", section on 'Our approach'.)

- **Monitoring and management after fibrinolysis** – After the fibrinolytic agent and its associated treatments have been given, the patient should be monitored for their response to fibrinolysis. (See "Diagnosis and management of failed fibrinolysis or threatened reocclusion in acute ST-elevation myocardial infarction".)

  The next steps in management are determined by the response to fibrinolysis and the patient's clinical presentation:

- **Patients with failed fibrinolysis or hemodynamic instability** – Patients with evidence of failed fibrinolysis or hemodynamic instability may benefit from urgent PCI. This issue is discussed elsewhere. (See "Percutaneous coronary intervention after fibrinolysis for acute ST-elevation myocardial infarction", section on 'Failed fibrinolysis'.)

- **Patients with successful fibrinolysis** – Patients with STEMI who were initially treated with fibrinolysis may benefit from routine PCI (ie, pharmacoinvasive strategy) in the hours or days following successful fibrinolysis. The approach to PCI after successful fibrinolysis is discussed elsewhere. (See "Acute ST-elevation myocardial infarction: Selecting a reperfusion strategy".)

Patients who do not undergo reperfusion — In patients with STEMI who will not undergo reperfusion due to contraindications or lack of access to reperfusion therapy, the acute management consists of continued monitoring and continued medical therapy for STEMI. The management of these patients typically includes:

- **Monitoring for complications** – Patients who do not undergo reperfusion should undergo standard post-MI evaluation. Notably, patients who are not reperfused in a timely manner are at higher risk of mechanical complications of MI (eg, myocardial wall rupture). These complications are discussed elsewhere. (See "Acute myocardial infarction: Mechanical complications".)

- **Medical therapy** – In patients who do not undergo reperfusion, the approach to treatment with anticoagulation, antiplatelet agents may differ from patients who undergo reperfusion. The approach to medical therapy in this group of patients and the overall approach to nonacute management are discussed separately. (See "Acute ST-elevation myocardial infarction: Management of anticoagulation", section on 'No reperfusion therapy' and "Acute ST-elevation myocardial infarction: Antiplatelet therapy", section on 'Patients not reperfused' and "Overview of the nonacute management of unstable angina and non-ST-elevation myocardial infarction".)

SPECIAL POPULATIONS

Patients with COVID-19 — In patients with STEMI and confirmed or suspected coronavirus disease 2019 (COVID-19), management requires particular attention to delays in reperfusion related to the pandemic and to the presence of other life-threatening illnesses (eg, acute respiratory distress syndrome). These issues are discussed elsewhere. (See "COVID-19:

Myocardial infarction and other coronary artery disease issues", section on 'Acute coronary syndrome patients'.)

Perioperative myocardial infarction — The management of patients with perioperative MI requires an assessment of the risks and benefits of STEMI therapies, which may increase the risk of bleeding at the surgical site. The management of these patients is discussed elsewhere. (See "Perioperative myocardial infarction or injury after noncardiac surgery", section on 'ST-elevation myocardial infarction'.)

Women and pregnant patients — The management of female patients and pregnant patients with STEMI is discussed separately. (See "Management of coronary heart disease in women", section on 'STEMI' and "Acute myocardial infarction and pregnancy".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: ST-elevation myocardial infarction (STEMI)" and "Society guideline links: Secondary prevention of cardiovascular disease".)

SUMMARY AND RECOMMENDATIONS

- **Goals of therapy** – The goal of STEMI management is to reduce the risk of death and permanent cardiac injury associated with myocardial infarction. (See 'Goals of therapy' above.)
- **Initial assessment** – All patients with suspected acute coronary syndrome (ACS) should have an initial assessment to rapidly confirm or exclude the presence of STEMI and to identify other conditions that would change management (table 1). (See 'Initial assessment' above.)
  - **Rapid diagnosis** – The rapid diagnosis of STEMI only requires the presence of symptoms suspicious for an ACS and a confirmatory ECG; it does not require evidence of elevated cardiac biomarkers such as troponin. (See 'Rapid diagnosis of STEMI' above.)
  - **Monitoring and testing** – Monitoring and testing should be used to identify disorders that are life-threatening or that significantly alter the usual management of STEMI. (See

'Monitoring and testing' above.)

- **Evaluation of life-threatening conditions** – The initial assessment of patients with STEMI includes a brief evaluation for conditions that require additional treatment or that alter the approach to STEMI therapy. (See 'Evaluation for life-threatening conditions' above.)

- **Initial management** – The initial management of patients with STEMI consists of the following steps which are summarized in the table (table 1) and discussed in detail separately (see 'Initial management' above):

  - Choose and initiate reperfusion with either percutaneous coronary intervention (PCI) or fibrinolysis. (See 'Choosing and initiating reperfusion with PCI or fibrinolysis' above.)

  - Administer routine medical therapies such as aspirin and nitrates. (See 'Routine medical therapy' above.)

  - Treat for specific symptoms and syndromes. (See 'Therapy for specific symptoms and syndromes' above.)

  - Avoid routine use of therapies of unclear benefit (eg, morphine, red blood cell transfusion). For example (see 'Therapies of unclear benefit' above):

    - **Oxygen** – In patients with STEMI who have an oxygen saturation ≥94 percent and no signs of respiratory distress, we suggest **not** routinely treating with supplemental oxygen (**Grade 2C**).

    - **Morphine** – In patients with STEMI, we suggest **not** routinely treating with morphine (**Grade 2C**).

- **Subsequent management by reperfusion strategy** – After initial assessment and management, the next steps in management are determined by the type of reperfusion strategy. (See 'Subsequent management by reperfusion strategy' above.)

  - **Patients undergoing PCI** – Patients with STEMI who will undergo PCI require assessment and treatment related to the PCI procedure. (See 'Patients undergoing PCI' above.)

  - **Patients undergoing fibrinolysis** – For patients who will undergo fibrinolysis, the goals of management are to safely administer fibrinolytics as soon as possible, monitor

the effect of fibrinolysis, and prepare for subsequent management (eg, transfer for PCI) (table 1).

- **Patients who do not undergo reperfusion** – In patients with STEMI who will not undergo reperfusion due to contraindications or lack of access to reperfusion therapy, the acute management consists of continued monitoring and continued medical therapy for STEMI. (See 'Patients who do not undergo reperfusion' above.)

- **Special populations** – Patients with STEMI who may require a different approach to acute management include:
  - Patients with suspected or confirmed COVID-19. (See 'Patients with COVID-19' above.)
  - Patients presenting with STEMI in the perioperative period. (See 'Perioperative myocardial infarction' above.)
  - Females may have atypical presentations of MI, and pregnant patients require specialized management. (See 'Women and pregnant patients' above.)

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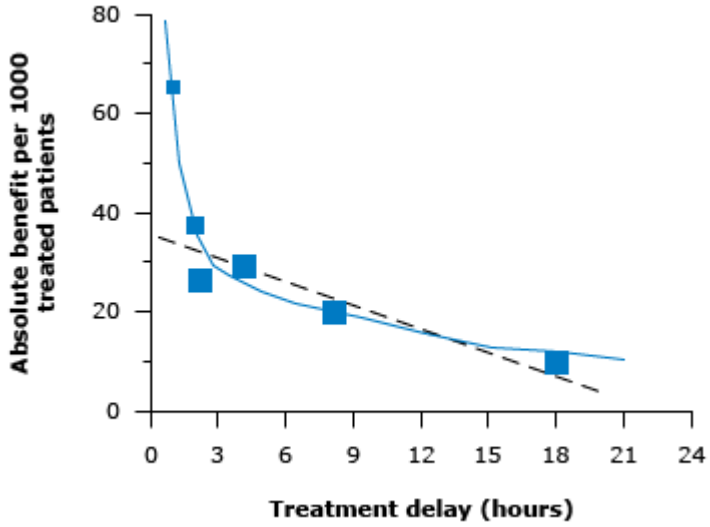
GRAPHICS

Time to thrombolysis and 35-day mortality



The importance of time to thrombolysis in acute myocardial infarction and the absolute reduction in 35-day mortality in a meta-analysis of over 50,000 patients. The benefit from thrombolytic therapy is greatest when it is administered within 2 hours of symptom onset. The survival benefit is progressively reduced as the delay in therapy increases; after 2 hours, the benefit from thrombolytic therapy fits a linear function (black dashed line) in which the benefit falls by approximately 1.6 lives per 1000 patients per hour of treatment delay.

Data from Boersma E, Maas ACP, Simoon ML. Lancet 1996; 348:771.

Graphic 53374 Version 6.0

ST-elevation myocardial infarction (STEMI) or non-ST-elevation acute coronary syndrome (NSTEACS): Rapid overview of emergency management

Initial assessment:

- Consider the diagnosis in patients with chest discomfort, shortness of breath, or other suggestive symptoms. Women, older adults, and patients with diabetes may have "atypical" presentations.
- Obtain 12-lead ECG within 10 minutes of arrival; repeat every 10 to 15 minutes if initial ECG is nondiagnostic but clinical suspicion remains high (initial ECG often **not** diagnostic).
    1. STEMI: ST-segment elevations ≥1 mm (0.1 mV) in 2 anatomically contiguous leads or ≥2 mm (0.2 mV) in leads V2 and V3 **or** new left bundle branch block and presentation consistent with ACS. If ECG suspicious but not diagnostic, consult cardiologist early.
    2. Non-STEMI or unstable angina: ST-segment depressions or deep T-wave inversions without Q waves or possibly no ECG changes.
- Obtain emergency cardiology consultation for ACS patients with cardiogenic shock, left heart failure or sustained ventricular tachyarrhythmia.

Initial interventions:

- Assess and stabilize airway, breathing, and circulation.
- Attach cardiac and oxygen saturation monitors; provide supplemental oxygen as needed to maintain O_2 saturation >90%. Establish IV access.
- Treat sustained ventricular arrhythmia rapidly according to ACLS protocols.
- Give aspirin 325 mg (nonenteric coated) to be chewed and swallowed (unless aortic dissection is being considered). If oral administration is 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, hematocrit/hemoglobin. Perform coagulation studies for patients taking anticoagulants or as otherwise indicated (eg, known coagulopathy).
- Give 3 sublingual nitroglycerin tablets (0.4 mg) 1 at a time, spaced 5 minutes apart, or 1 aerosol spray under tongue every 5 minutes for 3 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.
- Treat left heart failure if present: Give afterload-reducing agent (eg, nitroglycerin sublingual tablet and/or IV drip at 40 mcg/minute provided no hypotension and no phosphodiesterase inhibitors [eg,

for erectile dysfunction]; titrate drip up quickly based on response); give loop diuretic (eg, intravenous furosemide); administer noninvasive positive pressure ventilation (eg, BLPAP) to appropriate patients.

- Give beta blocker (eg, metoprolol tartrate 25 mg orally) **if** no signs of heart failure and not at high risk for heart failure and no signs of hemodynamic compromise, bradycardia, or severe reactive airway disease. If hypertensive, may initiate beta blocker IV instead (eg, metoprolol tartrate 5 mg intravenous every 5 minutes for 3 doses as tolerated).

- Morphine sulfate is indicated for chest discomfort refractory to nitrates and other antiischemic therapies. Give 2 to 4 mg slow IV push every 5 to 15 minutes.

- Start 80 mg of atorvastatin as early as possible and preferably before PCI in patients not on statin. If patient is taking a low- to moderate-intensity statin, switch to atorvastatin 80 mg.

Acute management STEMI:

- Select reperfusion strategy: Primary 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 those at high risk of death.

- Treat with fibrinolysis if PCI unavailable within **120 minutes of first medical contact,** symptoms <12 hours, and no contraindications.*

- **Give oral antiplatelet therapy (in addition to aspirin) to all patients:**

1. **Patients treated with fibrinolytic therapy:** Give clopidogrel loading dose 300 mg if age 75 years or less; if age over 75 years, give loading dose of 75 mg.

2. **Patients treated with no reperfusion therapy:** Give ticagrelor loading dose 180 mg.

3. **Patients treated with primary PCI:** Give ticagrelor loading dose of 180 mg or prasugrel loading dose of 60 mg (if no contraindications: prior stroke or TIA, or relative contraindications for prasugrel such as those age 75 years or older, weight less than 60 kg). For patients at high risk of bleeding or those for whom prasugrel or ticagrelor cannot be used, we give clopidogrel 600 mg.

- **Give anticoagulant therapy to all patients:**

1. **For patients treated with primary PCI,** we prefer UFH to bivalirudin. This recommendation assumes that patients will receive a potent oral antiplatelet agent (ticagrelor or prasugrel), which we prefer to clopidogrel. For those patients who receive clopidogrel, we prefer bivalirudin.

    - **Dosing of UFH:** An initial IV bolus of 50 to 70 units/kg up to a maximum of 5000 units. Additional heparin may be given in the catheterization laboratory based on the results of ACT monitoring.
    - **Dosing of bivalirudin:** Initial bolus of 0.75 mg/kg IV followed by IV infusion of 1.75 mg/kg per hour; can be discontinued after PCI.

2. **For patients treated with fibrinolysis,** we prefer enoxaparin for patients not at high bleeding risk or fondaparinux for those at high bleeding risk. For those patients in whom PCI is possible or likely after fibrinolytic therapy, UFH is reasonable.

- **Dosing of enoxaparin**
  - Patients <75 years: Loading dose of 30 mg IV bolus followed by 1 mg/kg subcutaneously every 12 hours; maximum of 100 mg for the first 2 subcutaneous doses. The first subcutaneous dose should be administered with the IV bolus.
    - Dose adjustment for renal impairment (CrCl <30 mL/minute)*: Loading dose of 30 mg IV followed by 1 mg/kg subcutaneously every 24 hours. The first subcutaneous dose should be administered with the IV bolus.
  - Patients ≥75 years: No IV loading dose. Administer 0.75 mg/kg subcutaneously every 12 hours; maximum of 75 mg for the first 2 doses.
    - Dose adjustment for renal impairment (CrCl <30 mL/minute)*: No IV loading dose. Administer 1 mg/kg subcutaneously every 24 hours.
  - Supplemental IV bolus dose for patients who will receive PCI after >1 dose of therapeuti enoxaparin: 0.3 mg/kg if last enoxaparin dose was given 8 to 12 hours earlier; no supplemental IV dose if last enoxaparin dose was within 8 hours; use UFH if last enoxaparin dose was more than 12 hours ago.
- **Dosing of UFH:** IV bolus of 60 to 100 units/kg to a maximum of 4000 units, followed by an I infusion of 12 units/kg per hour (maximum 1000 units per hour) adjusted to achieve a goal aPTT of approximately 50 to 70 seconds (1.5 to 2 times control).
- **Dosing of fondaparinux:** 2.5 mg intravenously, followed by 2.5 mg subcutaneously every 24 hours. This drug should be avoided in CrCl <30 mL/minute.

3. **For patients not receiving reperfusion therapy,** we use enoxaparin or UFH.

- **Dosing of enoxaparin:** Dose same as for patients treated with fibrinolysis (refer to section above).
- **Dosing of UFH:** IV bolus of 50 to 70 units/kg to a maximum of 5000 units, followed by an IV infusion of 12 units/kg per hour adjusted to achieve a goal aPTT of approximately 50 to 70 seconds (1.5 to 2 times control).

Acute management of unstable angina or non-STEMI:

- **Give antiplatelet therapy (in addition to aspirin) to all patients:**

1. **Patients not treated with an invasive approach:** Give ticagrelor loading dose 180 mg. For these patients who are at very high risk (eg, recurrent ischemic discomfort, dynamic ECG changes, or hemodynamic instability), consider adding a GP IIb/IIIa inhibitor (either eptifibatide or tirofiban).

2. **For patients managed with an invasive approach:** Give ticagrelor loading dose of 180 mg at presentation. Prasugrel loading dose of 60 mg may be used as an alternative if given after diagnostic coronary angiography.

For patients age 75 years or older, who weigh less than 60 kg, or with past stroke or TIA, ticagrelor or clopidogrel are preferred to prasugrel. Clopidogrel may be given in a dose of 300 to 600 mg, but we prefer 600 mg. For patients otherwise at high risk for bleeding due to prior hemorrhagic stroke, ongoing bleeding, bleeding diathesis, or clinically relevant anemia or thrombocytopenia, clopidogrel 300 to 600 mg is an option.

For patients treated with an invasive approach and who receive bivalirudin, we do not recommend routinely giving a GP IIb/IIIa inhibitor; for those patients treated with heparin and who are troponin-positive, we suggest adding a GP IIb/IIIa inhibitor (either abciximab or eptifibatide) given after diagnostic angiography. For those undergoing an invasive approach who are at very high risk (eg, recurrent ischemic discomfort, dynamic ECG changes, or hemodynamic instability), we consider adding a GP IIb/IIIa inhibitor prior to diagnostic angiography (either eptifibatide or tirofiban) or after diagnostic angiography (abciximab or eptifibatide). Refer to text for dosing.

- **Give anticoagulant therapy in all patients:**

1. **For patients undergoing urgent catheterization (within 4 hours) or those managed with an early invasive strategy (angiography within 4 to 48 hours),** we use either heparin or bivalirudin. We prefer initiation of heparin in the emergency department and a switch to bivalirudin in the catheterization laboratory.

    - **Dosing of UFH:** IV bolus of 60 to 70 units/kg to a maximum of 5000 units, followed by an IV infusion of 12 units/kg per hour adjusted to achieve a goal aPTT of approximately 50 to 70 seconds (1.5 to 2 times control).
    - **Dose of bivalirudin:** If bivalirudin is given in the emergency department, IV bolus of 0.1 mg/kg and an infusion of 0.25 mg/kg per hour before angiography. If PCI is performed, an additional 0.5 mg/kg bolus is given and the infusion rate is increased to 1.75 mg/kg per hour.

2. **For patients receiving a noninvasive approach,** we recommend either fondaparinux or enoxaparin.

    - **Enoxaparin** is an alternative to UFH for patients not undergoing an early invasive approach. No loading dose is necessary. Dosing is 1 mg/kg subcutaneously every 12 hours. Dose adjustment for renal impairment (CrCl <30 mL/minute)*: 1 mg/kg subcutaneously every 24 hours.
    - **Fondaparinux:** 2.5 mg subcutaneously every 24 hours. This drug should be avoided in patients with a CrCl <30 mL/minute.

Other important considerations:

- Cocaine-related ACS: Give benzodiazepines (eg, lorazepam 2 to 4 mg IV every 15 minutes or so) as needed to alleviate symptoms; Give standard therapies (eg, aspirin, nitroglycerin) but do **not** give beta blockers.

- Stop NSAID therapy if possible.

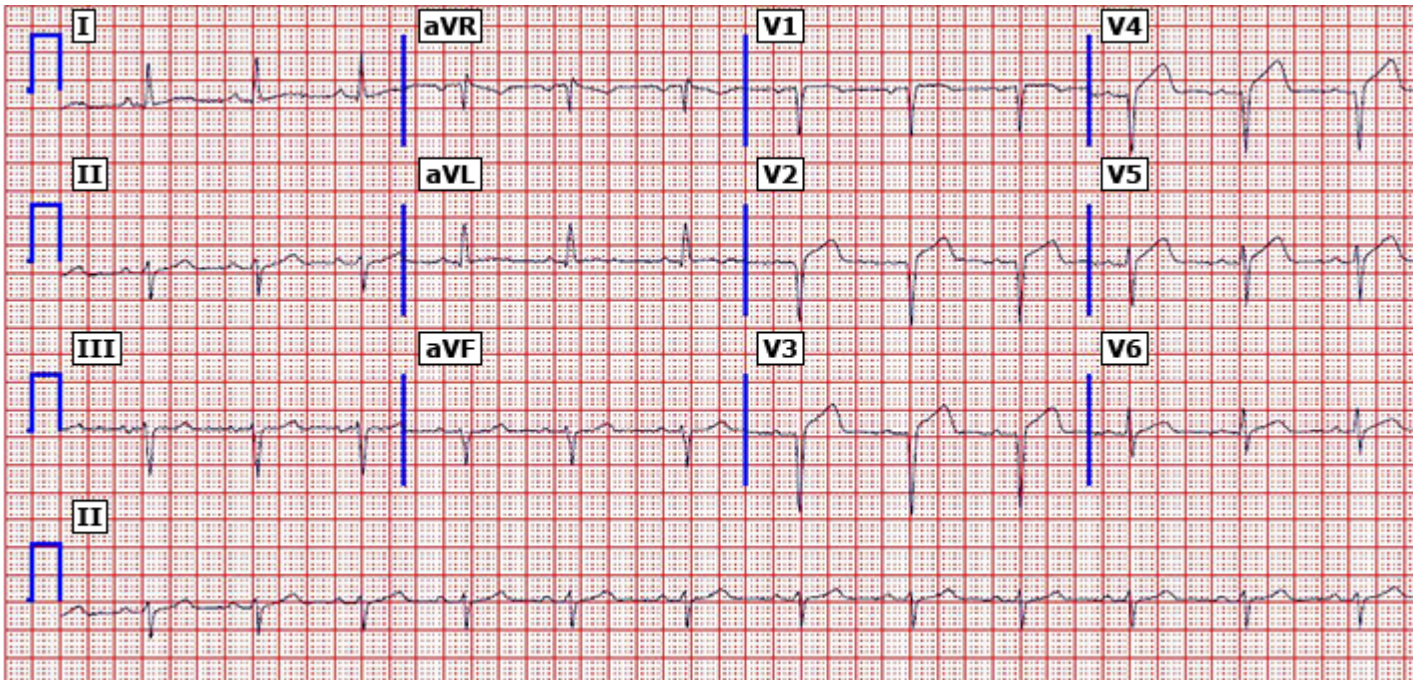
- Correct any electrolyte abnormalities, especially hypokalemia and hypomagnesemia, which often occur together.

ACLS: advanced cardiac life support; ACS: acute coronary syndrome; ACT: activated clotting time; aPTT: activated partial thromboplastin time; BLPAP: bilevel positive airway pressure; CrCl: creatinine clearance estimated using Cockcroft-Gault equation (a calculator is available in UpToDate); ECG: electrocardiogram; GP: glycoprotein; IV: intravenous; NSAID: nonsteroidal antiinflammatory drug; PCI: percutaneous coronary intervention; STEMI: ST-elevation myocardial infarction; TIA: transient ischemic attack; UFH: unfractionated heparin.

* Repeated doses of low molecular weight heparin in patients with renal insufficiency may lead to accumulation and increased risk of bleeding to varying degrees. By contrast, UFH is not dependent primarily upon renal function for clearance and may be a preferred option for patients with CrCl <20 mL/minute, kidney failure, or receiving dialysis.

Graphic 75032 Version 37.0

Anterior ST-elevation/Q wave myocardial infarction



Sinus rhythm is present at a rate of about 75/min. Note the loss of R waves, with frank Q waves in leads V1 to V4 in concert with ST elevations in V2 to V5/V6, as well as more subtly in leads I and aVL. Marked left-axis deviation is present with a normal QRS duration consistent with left anterior fascicular block (LAFB). The patient had sustained a very recent ST-elevation/Q wave anterior myocardial infarction (MI). Cardiac catheterization revealed three-vessel disease with a 90% mid-left anterior descending coronary artery "culprit" lesion that was successfully treated with a percutaneous coronary intervention procedure.

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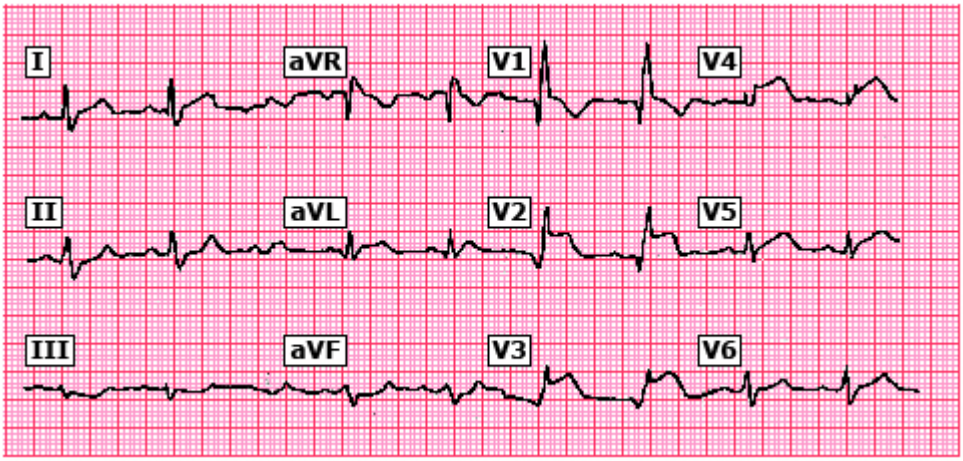
Graphic 118832 Version 3.0

Acute anterior ST-elevation MI (STEMI) with right bundle branch block

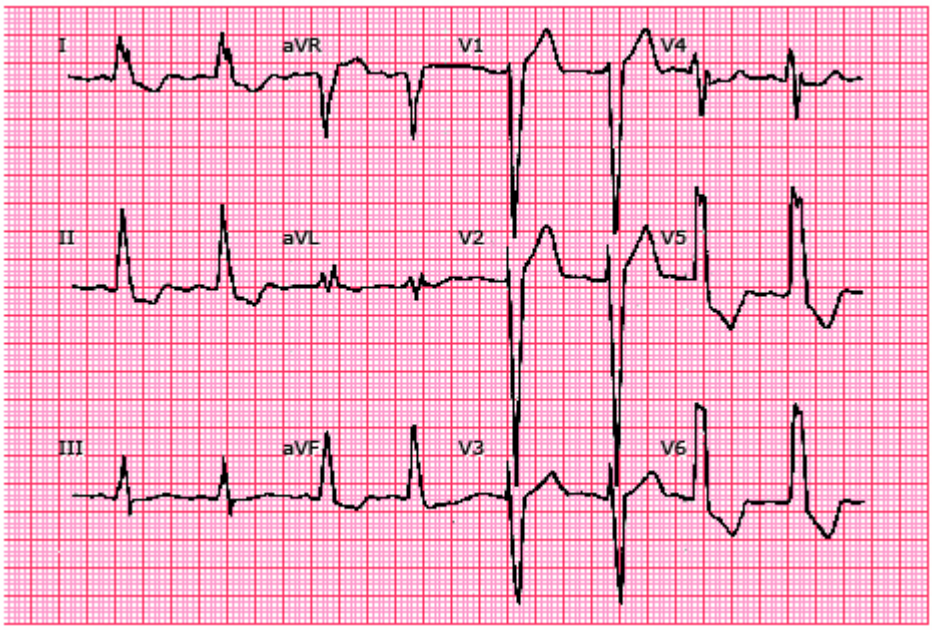


Electrocardiogram showing an acute anterior myocardial infarction (Q waves and ST elevations in leads V2 to V4, and ST elevations alone in leads I and aVL) and right bundle branch block (terminal R wave in lead V1 and terminal S wave in leads I and V6).

Courtesy of Ary Goldberger, MD.

Graphic 134874 Version 1.0

12-lead electrocardiogram (ECG) showing typical left bundle branch block

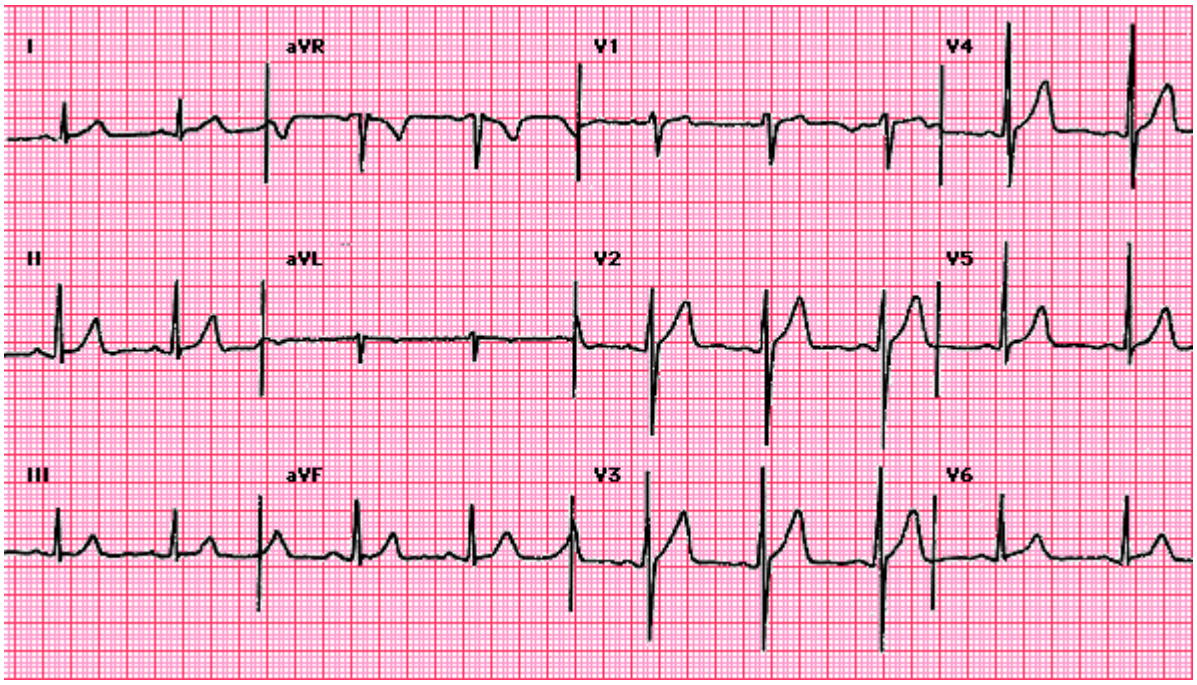


Electrocardiogram in typical complete left bundle branch block. The asynchronous activation of the 2 ventricles increases the QRS duration (0.16 seconds in this example). The abnormal initial vector results in loss of "normal" septal forces as manifested by absence of q waves in leads I, aVL, and V6. The late activation of the left ventricle prolongs the dominant leftward progression of the middle and terminal forces, leading to a positive and widened R wave in the lateral leads. Both the ST segment and T wave vectors are opposite in direction from the QRS, a "secondary" repolarization abnormality.

Courtesy of Ary Goldberger, MD.

Graphic 61594 Version 9.0

Normal ECG

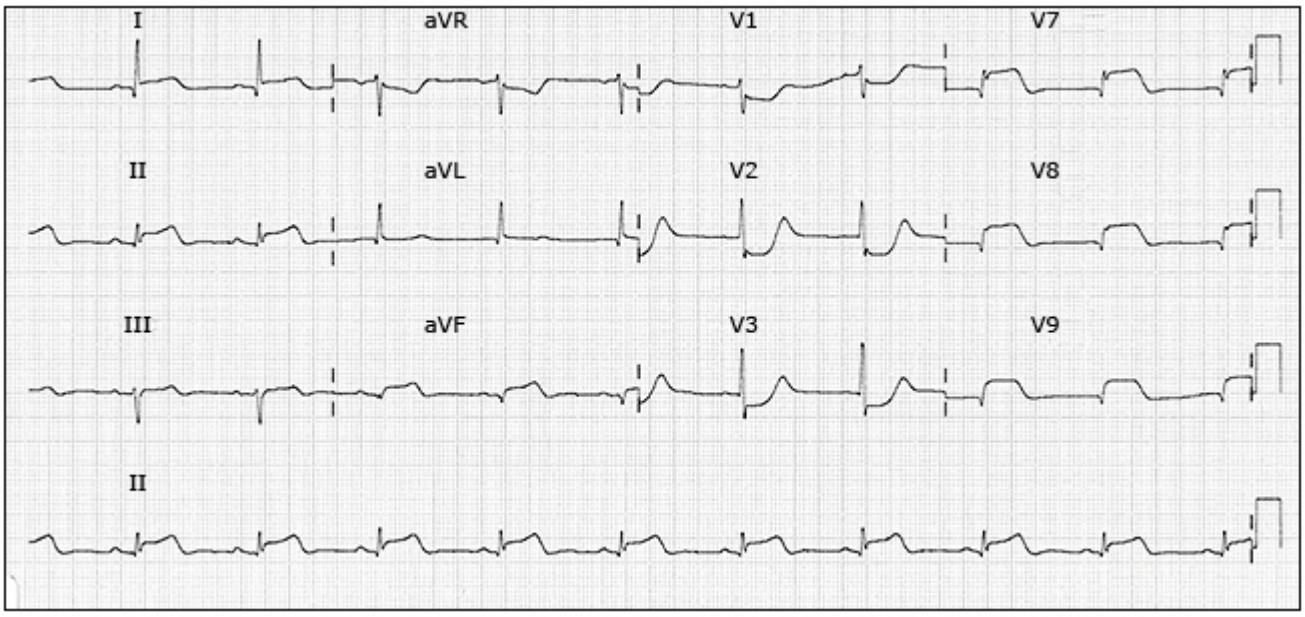


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

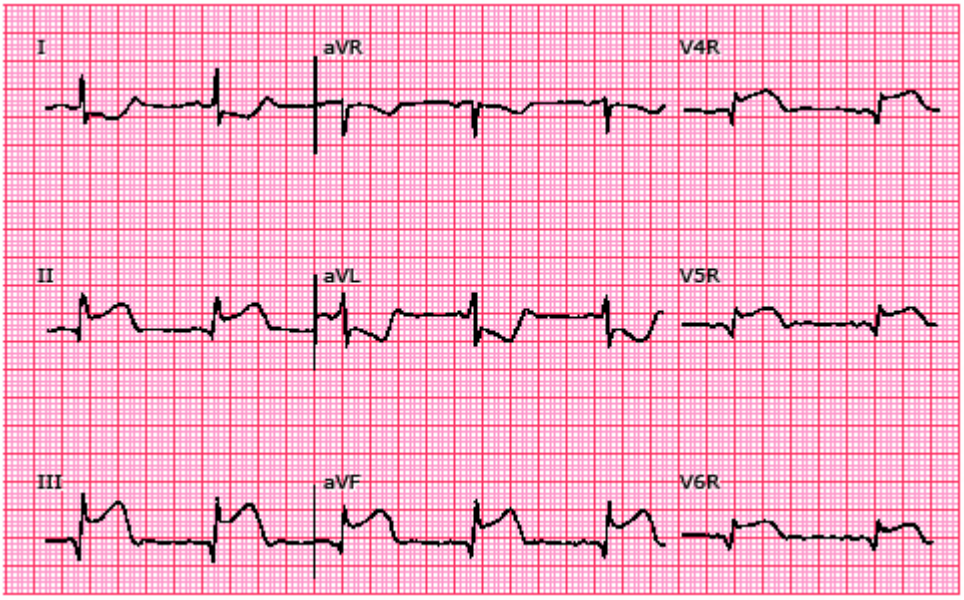
Posterior myocardial infarction leads V7 to V9



Reproduced from: Burns E. ECG Library: Posterior Myocardial Infarction. Life in the Fastlane. Available at: http://lifeinthefastlane.com/ecg-library/pmi/ (Accessed on April 22, 2014).

Graphic 94928 Version 2.0

ECG of acute inferior and right ventricular myocardial infarction



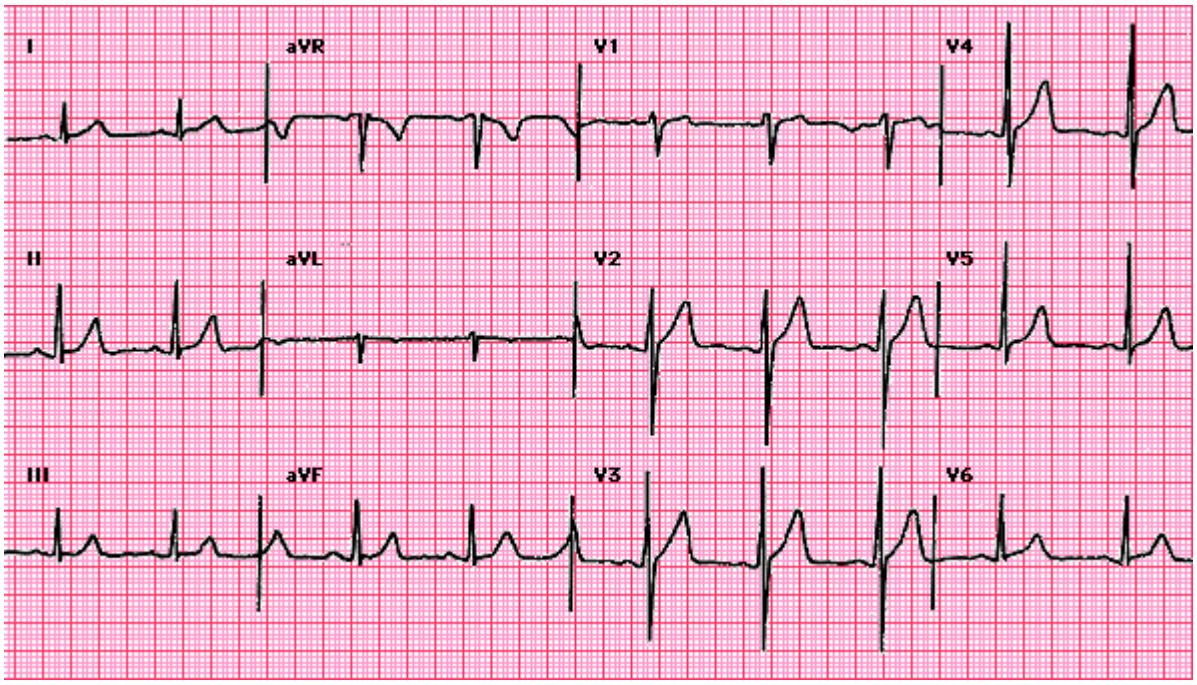
ECG shows Q waves and prominent doming ST-segment elevation in II, III, and aVF, findings which are characteristic of an acute inferior myocardial infarction. ST elevation in the right precordial leads - V4R, V5R, and V6R - indicates right ventricular involvement as well. The ST depressions in leads I and aVL represent reciprocal changes.

ECG: electrocardiogram.

Courtesy of Ary Goldberger, MD.

Graphic 82739 Version 4.0

Normal ECG

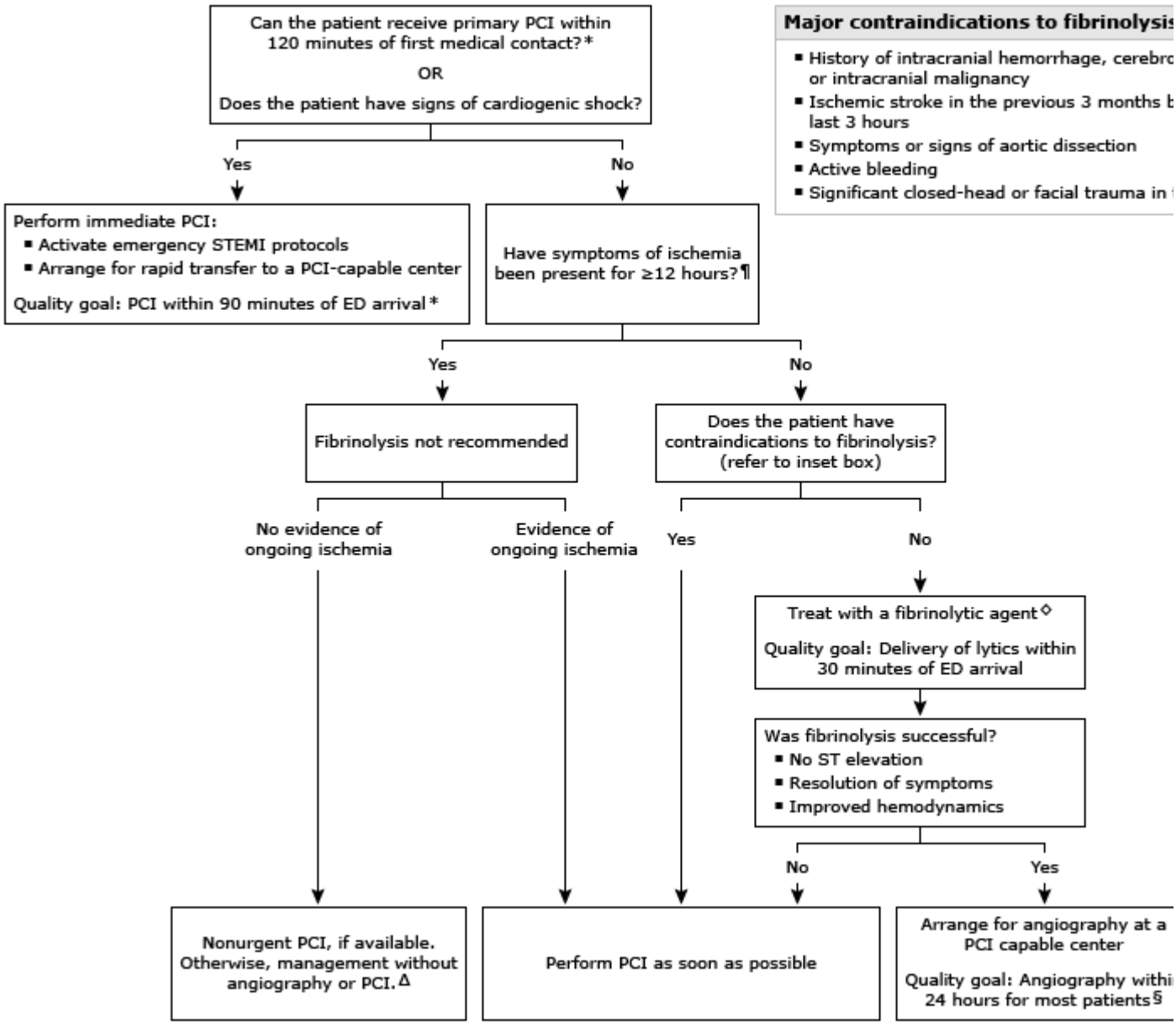


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

Selecting a reperfusion strategy in patients with acute STEMI



STEMI: ST-elevation myocardial infarction; PCI: percutaneous coronary intervention; ED: emergency department; AMI: acute myocardial infarction.

* This time includes all time required for transfer and other aspects of care until balloon dilation, thrombectomy, or other definitive percutaneous intervention is performed.

¶ Refer to UpToDate content on selecting a reperfusion strategy in STEMI for more information on patients with late presentation.

Δ For patients who do not undergo either PCI or fibrinolysis, refer to UpToDate content for a discussion of optimal medical management.

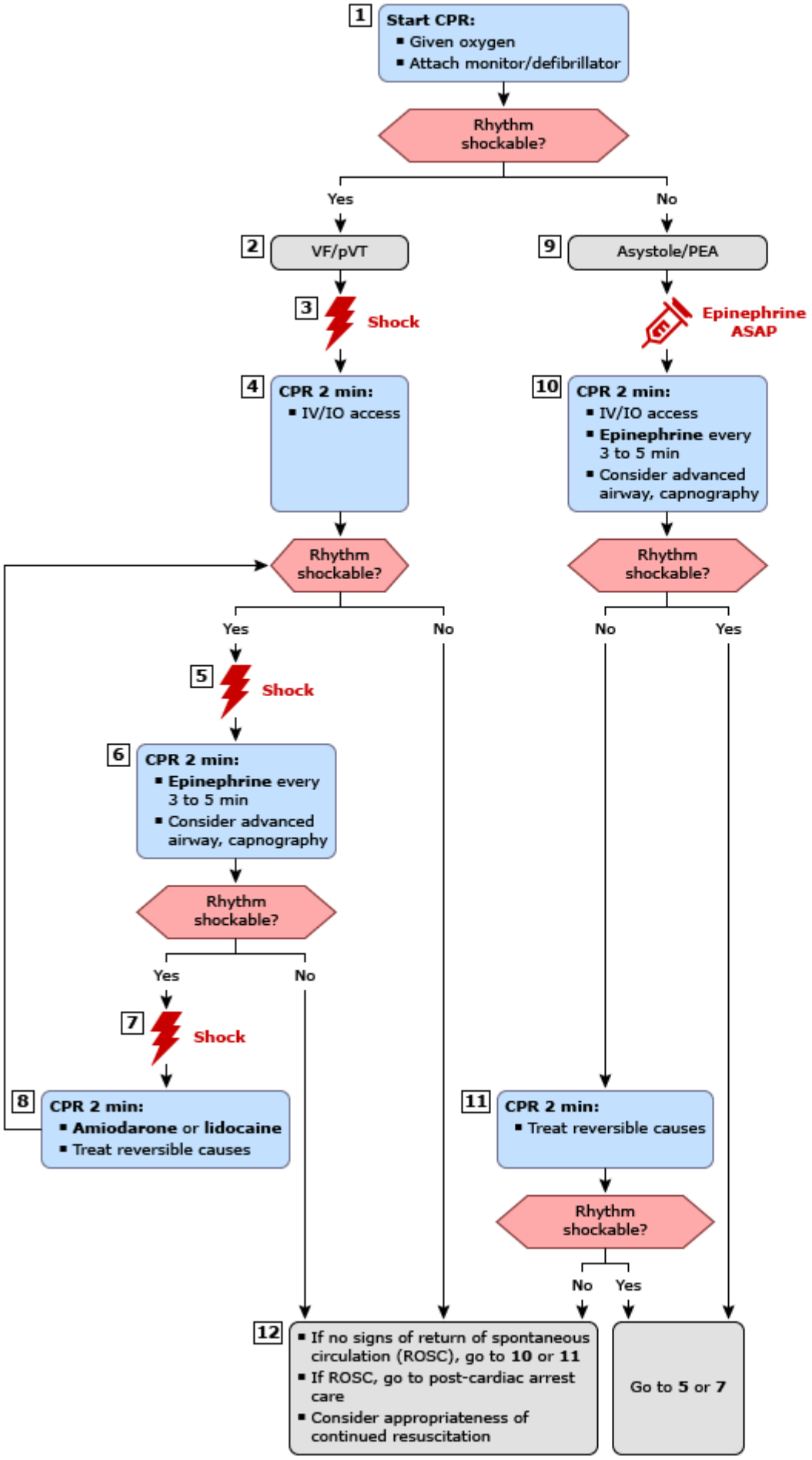
◇ The selection and use of a specific fibrinolytic agent and the associated anticoagulant and antiplatelet therapies are discussed in UpToDate content on fibrinolysis in AMI.

§ Patients at high risk of reocclusion should undergo PCI sooner, while low-risk patients can undergo PCI later. More information on the use of angiography and PCI after fibrionlysis for the treatment of STEMI can be found in UpToDate.

¥ Refer to UpToDate content for the details on the absolute and relative contraindications to fibrinolysis in patients with acute STEMI.

Graphic 138821 Version 1.0

Adult cardiac arrest algorithm



CPR quali

- Push hard (at least 2 inch
  fast (100 to 120/min) and
  chest recoil.
- Minimize interruptions in
- Avoid excessive ventilatio
- Change compressor every
  sooner if fatigued.
- If no advanced airway, 30
  ventilation ratio.
- Quantitative waveform ca
  - If $PETCO_2$ is low or decr
    CPR quality.

Shock energy for d

- **Biphasic:** Manufacturer r
  (eg, initial dose of 120 to
  unknown, use maximum
  and subsequent doses sh
  and higher doses may be
- **Monophasic:** 360 J.

Drug thera

- **Epinephrine IV/IO dos**
  1 mg every 3 to 5 minute
- **Amiodarone IV/IO dos**
  First dose: 300 mg bolus.
  Second dose: 150 mg.
  or
- **Lidocaine IV/IO dose:**
  First dose: 1 to 1.5 mg/k
  Second dose: 0.5 to 0.75

Advanced ai

- Endotracheal intubation o
  advanced airway.
- Waveform capnography o
  confirm and monitor ET t
- Once advanced airway in
  breath every 6 seconds (1
  with continuous chest com

Return of spontaneous c

- Pulse and blood pressure.
- Abrupt sustained increase
  (typically ≥40 mmHg).
- Spontaneous arterial pres
  intra-arterial monitoring.

Reversible ca

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

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Graphic 129983 Version 10.0

Causes of ST-segment elevation

Myocardial ischemia/infarction
Noninfarction, transmural ischemia (eg vasospastic angina and takotsubo cardiomyopathy)
Acute MI especially due to acute epicardial coronary occlusion; may be due to takotsubo cardiomyopathy and other nonatherosclerotic coronary occlusion or severe hypoperfusion syndromes
Post-MI (ventricular aneurysm pattern)
Acute pericarditis
Abnormal early repolarization syndromes
Normal variants (including benign early repolarization)
Left ventricular hypertrophy or left bundle branch block (V1 to V2 or V3)
Other
Myocarditis (including COVID-19 infections)
Massive pulmonary embolism (leads V1 to V2 in occasional cases)
Brugada-type patterns (V1 to V3 with right bundle branch block-appearing morphology)
Myocardial tumor
Myocardial trauma
Hyperkalemia (only leads V1 and V2)
Hypothermia (J wave/Osborn wave)
Hypercalcemia (rarely)
Post-DC cardioversion (rarely)
Neurogenic stress cardiomyopathy

MI: myocardial infarction; COVID-19: coronavirus disease 2019; DC: direct current.

Graphic 81757 Version 8.0