

Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department

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INTRODUCTION

The clinical presentation of myocardial ischemia is most often acute chest discomfort. The goal of emergency department evaluation is to determine the cause of such discomfort or other related symptoms (eg, dyspnea, weakness) and to initiate appropriate therapy promptly. It is essential that initial assessment and management be rapid but methodical and evidence based.

Diagnostic evaluation emphasizes distinguishing between the following potential, life-threatening causes of chest pain:

- Acute coronary syndrome (ACS; myocardial infarction or unstable angina)
- Nonischemic chest pain, including potentially life-threatening conditions such as aortic dissection, pulmonary embolism, and esophageal rupture ([table 1](#) and [table 2A-B](#))

The diagnosis of acute coronary-related ischemia depends upon the characteristics of the chest pain (if present), specific associated symptoms, abnormalities on electrocardiogram (ECG), and levels of serum markers of cardiac injury. A patient with a possible ACS should be evaluated and treated rapidly. Thus, initial management steps must be undertaken before or during the time the diagnosis is being established.

The initial management of the patient likely to have an ACS will be reviewed here. Discussions of related subjects are found separately:

- (See "[Evaluation of the adult with chest pain in the emergency department](#)".)
 - (See "[Approach to the adult with dyspnea in the emergency department](#)".)
 - (See "[Overview of the acute management of non-ST-elevation acute coronary syndromes](#)".)
 - (See "[Evaluation of emergency department patients with chest pain at low or intermediate risk for acute coronary syndrome](#)".)
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GUIDANCE AND RESOURCES FOR INSTITUTIONS

The American College of Cardiology (ACC) and the American Heart Association (AHA) recommend 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. The management of diagnosed ACS is discussed in detail separately. (See "[Overview of the acute management of ST-elevation myocardial infarction](#)" and "[Overview of the acute management of non-ST-elevation acute coronary syndromes](#)" and "[Evaluation of emergency department patients with chest pain at low or intermediate risk for acute coronary syndrome](#)".)

Immediate cardiology consultation should be available for cases in which the initial diagnosis and treatment plan are unclear or are not addressed directly by available protocols. A number of life-threatening conditions can cause chest pain and dyspnea. It is important to avoid premature diagnostic closure. (See "[Evaluation of the adult with chest pain in the emergency department](#)" and "[Approach to the adult with dyspnea in the emergency department](#)".)

WHEN TO SUSPECT ACUTE CORONARY SYNDROME

Clinicians should consider the possibility of ACS in any adult who presents to the emergency department complaining of chest discomfort or dyspnea. A personal or family history of ACS or other cardiovascular disease, older age, diabetes, dyslipidemia, cigarette smoking, hypertension, or cocaine use all increase the likelihood. (See "[Overview of established risk factors for cardiovascular disease](#)".)

Characteristic presentations (as described below) heighten concern, but clinicians must keep in mind that many patients with ACS present with symptoms such as dyspnea or malaise, either

alone or in addition to chest pain. Females are more likely to have associated dyspnea than males, and patients who are older or have diabetes are more likely to present with dyspnea without chest pain. (See '[Clinical presentation](#)' below and '[Atypical presentations](#)' below and "[Clinical features and diagnosis of coronary heart disease in women](#)" and "[Silent myocardial ischemia: Epidemiology, diagnosis, treatment, and prognosis](#)".)

Pain from coronary-related ischemia is more often characterized as non-focal chest discomfort or pressure rather than pain, is generally gradual in onset, and is exacerbated by activity. Discomfort that radiates, particularly to either or both arms, should increase suspicion for ACS.

IMMEDIATE EMERGENCY DEPARTMENT INTERVENTIONS

In the emergency department, patients presenting with chest pain or other symptoms possibly due to ACS (eg, dyspnea) should be rapidly evaluated to determine if their symptoms are suggestive of acute ischemia or some other potentially life-threatening illness. (See '[Clinical presentation](#)' below and "[Evaluation of the adult with chest pain in the emergency department](#)" and "[Approach to the adult with dyspnea in the emergency department](#)".)

If ACS is the leading diagnosis, initial assessment and interventions must be performed rapidly. The attached algorithm provides a simple approach to risk stratification while the table provides a concise outline of the immediate interventions needed to manage ST elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI), and unstable angina ([algorithm 1](#) and [table 3](#)).

During the initial assessment phase, the following steps should be accomplished for any patient at significant risk for ACS:

- Airway, breathing, and circulation assessed
- Preliminary history and examination obtained
- 12-lead electrocardiogram (ECG) interpreted
- Resuscitation equipment brought to the bedside
- Cardiac monitor attached to patient
- Oxygen given as necessary
- Intravenous (IV) access and blood work (including high-sensitivity troponin) obtained
- [Aspirin](#) 162 to 325 mg given
- Nitrates given (contraindications to nitrates include severe aortic stenosis, hypertrophic cardiomyopathy, suspected right ventricular infarct, hypotension, marked bradycardia or tachycardia, and recent use of phosphodiesterase 5 inhibitor [eg, Viagra])

Caution should be employed in evaluating possible ACS in older adults and patients with diabetes, who are more likely to present with symptoms such as dyspnea or weakness without chest pain even in the presence of acute coronary-related ischemia [1]. (See '[Atypical presentations](#)' below.)

The institution's specific chest pain protocol should be implemented if the history or symptoms are suggestive of acute ischemia. The time for initial assessment, including ECG, and preliminary management of a patient with possible acute coronary-related ischemia is ideally 10 minutes from presentation. Large observational studies report that ECG acquisition is frequently delayed and that females are significantly less likely to be assessed within the recommended 10 minutes or to receive testing with a cardiac biomarker [2-4].

- **Initial history and exam** – Obtain a brief history and perform a focused physical examination. Important elements of the history include confirmation of the presenting symptoms, characteristics of the pain, exacerbating and alleviating factors, and important associated symptoms, past history of or risk factors for cardiovascular disease, and potential contraindications to thrombolytic therapy ([table 4](#)). (See '[Clinical presentation](#)' below.)

The examination should include assessment of hemodynamic status and a screening neurologic examination, especially if thrombolysis is entertained as a potential therapy. (See '[Physical examination](#)' below.)

The history should address other potentially life-threatening, noncardiac causes of chest pain, such as acute aortic dissection, pulmonary embolism, tension pneumothorax, perforating peptic ulcer, and esophageal rupture ([table 2A-B](#)). Until the diagnosis of ACS is established, the clinician should keep these diagnoses in mind. (See "[Evaluation of the adult with chest pain in the emergency department](#)".)

- **Twelve-lead electrocardiogram** – A 12-lead ECG should be obtained within 10 minutes of arrival in all patients with possible coronary-related ischemia. (See '[Electrocardiogram assessment](#)' below.)

The 12-lead ECG provides an important basis for initial diagnosis and management and should immediately be shown to an emergency physician for interpretation. If the initial ECG reveals an STEMI, immediate intervention is required. STEMI management is discussed in detail separately. (See "[Overview of the acute management of ST-elevation myocardial infarction](#)" and "[Acute ST-elevation myocardial infarction: Selecting a reperfusion strategy](#)" and "[Acute ST-elevation myocardial infarction: Management of anticoagulation](#)" and "[Acute ST-elevation myocardial infarction: Antiplatelet therapy](#)".)

The initial ECG is often **not** diagnostic in patients with ACS. Thus, the ECG should be repeated at 15- to 30-minute intervals if the initial study is not diagnostic but the patient remains symptomatic and high clinical suspicion for ACS persists.

- **Cardiac monitor** – The patient should be placed on a cardiac monitor with emergency resuscitation equipment (including a defibrillator and airway equipment) nearby.
- **Supplemental oxygen** – Supplemental oxygen should be given as necessary to maintain oxygen saturation above 90 percent. In patients with ACS with normal oxygen saturation and no signs of respiratory distress, we suggest **not** routinely treating with supplemental oxygen. (See "[Overview of the acute management of ST-elevation myocardial infarction](#)", section on 'Therapy for specific symptoms and syndromes'.)
- **Intravenous access** – IV access should be established and blood drawn for initial laboratory work, including cardiac biomarkers (contemporary or high-sensitivity troponin), basic electrolyte concentrations, renal function studies, and a complete blood count with platelets. Indices of coagulation should be obtained in patients at increased risk of coagulopathy due to [warfarin](#) or heparin use, a history of liver disease, or a history of excessive or spontaneous bleeding [5]. (See '[Cardiac biomarkers and other laboratory testing](#)' below.)
- **Aspirin** – All patients with suspected ACS should be given aspirin in a dose of 162 to 325 mg to chew and swallow unless there is a compelling contraindication (eg, history of anaphylactic reaction) or it has been taken prior to presentation. Despite its well-demonstrated benefit, aspirin remains underutilized in the setting of ACS. (See "[Aspirin for the secondary prevention of atherosclerotic cardiovascular disease](#)".)
- **Sublingual nitrates** – In most cases of suspected ACS, sublingual [nitroglycerin](#) should be administered at a dose of 0.4 mg every five minutes for a total of three doses, after which an assessment of blood pressure and pain relief should guide the need for IV nitroglycerin.

Before this is done, all patients should be questioned about the use of phosphodiesterase-5 inhibitors, such as [sildenafil](#) (Viagra), [vardenafil](#) (Levitra), or [tadalafil](#) (Cialis); nitrates are contraindicated if these drugs have been used in the last 24 hours (or perhaps as long as 36 hours with tadalafil) because of the propensity to cause potentially severe hypotension. (See "[Nitrates in the management of acute coronary syndrome](#)" and "[Sexual activity in patients with cardiovascular disease](#)".)

Extreme care should also be taken before giving nitrates in the setting of an inferior myocardial infarction with possible involvement of the right ventricle ([waveform 1](#)). In this setting, patients are dependent upon preload to maintain cardiac output, and nitrates can cause severe hypotension. (See "[Right ventricular myocardial infarction](#)".)

Pain that responds to sublingual [nitroglycerin](#) is frequently thought to have a cardiac etiology or to be due to esophageal spasm. However, pain relief with nitroglycerin in an acute care setting is **not** helpful in distinguishing cardiac from noncardiac chest pain [6]. In a study of 459 patients who presented to an emergency department with chest pain and were admitted to the hospital, the percentage of patients who had relief of chest pain with nitroglycerin was similar among the 141 patients with ACS and the 275 patients without active coronary disease (35 versus 41 percent experienced relief) [7]. As such, nitroglycerin administration should not be given as a diagnostic test in patients with chest pain or a presentation that is not consistent with ACS, as the response to treatment may cloud the clinical picture. (See "[Evaluation of the adult with chest pain in the emergency department](#)".)

- **Intravenous morphine sulfate** – IV morphine sulfate (initial dose of 2 to 4 mg, with increments of 2 to 8 mg, repeated at 5- to 15-minute intervals) may be given for the relief of severe, persistent chest pain not relieved by other means but should not be given routinely. Morphine can reduce sympathetic stimulation caused by pain and anxiety, thereby decreasing cardiac workload and risks associated with excess catecholamines. However, morphine may worsen outcomes in patients with acute myocardial infarction. (See "[Overview of the acute management of ST-elevation myocardial infarction](#)", section on '[Therapies of unclear benefit](#)'.)

DETERMINING WHO NEEDS AN ELECTROCARDIOGRAM

In general, a resting electrocardiogram (ECG) should be obtained in all adults with chest discomfort that does not have an obvious noncardiac cause. An ECG is also obtained routinely in older adults or patients with diabetes with symptoms of unclear etiology, such as dyspnea, nausea, or malaise; patients with syncope; and patients with symptoms or signs consistent with an arrhythmia. However, identifying at triage or early in the emergency department evaluation which younger adult patients should be evaluated with an ECG is occasionally difficult. Keep in mind that a substantial number of patients ultimately diagnosed with an ACS, particularly older patients and those with diabetes, present with complaints other than chest discomfort or do not manifest clear signs of ischemia in their initial ECG. Such presentations are discussed in

detail separately. (See '[Atypical presentations](#)' below and '[Importance of serial electrocardiograms](#)' below.)

One research group has proposed the following rule for identifying patients at high risk for ST elevation myocardial infarction (STEMI) who should be evaluated with an ECG [8]:

- Any patient over 30 with chest pain
- Any patient over 50 with any of the following: dyspnea, altered mental status, upper extremity pain, syncope, or weakness
- Any patient over 80 with abdominal pain, nausea, or vomiting

This rule was derived and validated using information from over three million emergency department visits included in a state-wide health surveillance database. Included in the database were 6464 cases of STEMI diagnosed during 2007 to 2008 that provided the dataset for the study. When applied to the validation sample (3294 STEMI cases), the rule had a sensitivity of 91.9 percent (95% CI 90.9-92.8) and a specificity of 76.2 percent (95% CI 76.1-76.2).

This proposed rule should not replace clinical judgement (in fact, the authors encourage clinicians to obtain an ECG based upon such judgement). As an example, a patient in whom a noncardiac diagnosis is likely (eg, younger patient with obvious zoster [shingles] lesions and no other symptoms) may not need an ECG. Nevertheless, this study provides some evidence for clinicians trying to devise appropriate triage protocols for the emergency department or to determine for which patients, including those not presenting with chest pain, an ECG assessment is needed.

Prospective validation of this decision rule in other patient populations is needed before widespread adoption can be recommended. Among the rule's limitations are its relatively low sensitivity and the age cutoffs, which may be too high in some settings. It is worth reiterating the importance of paying close attention to older patients and those with diabetes with symptoms consistent with ACS and using a low threshold for obtaining an ECG in such patients.

ELECTROCARDIOGRAM ASSESSMENT

Initial interpretation and criteria for ischemia — Specific approaches to patients judged to have definite or probable ACS based upon a targeted history and physical examination are initially guided by the accompanying 12-lead electrocardiogram (ECG) [9]. ECG interpretation for this purpose is discussed in detail separately; a focused review is provided below. (See "[Electrocardiogram in the diagnosis of myocardial ischemia and infarction](#)" and "[Diagnosis of acute myocardial infarction](#)", section on 'ECG'.)

Clinicians must be aware that the initial ECG is often **not** diagnostic in patients with ACS. Forty-one percent of patients with non-ST elevation myocardial infarction (NSTEMI) do not have the typical ST-depression or T-wave inversion [10]. In patients at intermediate to high risk for ACS without a clear diagnosis, ECGs should be repeated at frequent intervals until the patient's chest pain resolves or a definitive diagnosis is made. (See '[Importance of serial electrocardiograms](#)' below.)

When present, ECG abnormalities are an early sign of myocardial ischemia. Criteria for the two major categories of ECG manifestations of acute myocardial ischemia are listed here:

- **Findings consistent with ST elevation myocardial infarction (STEMI):**

- New or presumed new ST elevation at the J point in two anatomically contiguous leads using the following diagnostic thresholds:

≥0.1 mV (1 mm) in all leads other than V2 to V3, where the following diagnostic thresholds apply:

- ≥0.2 mV (2 mm) in males ≥ 40 years
- ≥0.25 mV (2.5 mm) in males <40 years
- ≥0.15 mV (1.5 mm) in females

- **Findings consistent with NSTEMI:**

- New or presumed new horizontal or down-sloping ST depression ≥0.05 mV (0.5 mm) in two anatomically contiguous leads

and/or

- T wave inversion ≥0.1 mV (1 mm) in two anatomically contiguous leads with prominent R wave or R/S ratio >1

Localization of ischemia — The ECG leads are more helpful in localizing regions of transmural than subendocardial ischemia. The anatomic location of a transmural infarct is determined by which ECG leads show ST elevation and/or increased T wave positivity:

- **Anterior wall ischemia** – Two or more of the precordial leads (V1 to V6)

([waveform 2A-B](#))

- **Anteroseptal ischemia** – Leads V1 to V3 ([waveform 3](#) and [waveform 4](#))

- **Apical or lateral ischemia** – Leads aVL and I, and leads V4 to V6 ([waveform 5](#) and [waveform 6](#))
- **Inferior wall ischemia** – Leads II, III, and aVF ([waveform 1](#))
- **Right ventricular ischemia** – Right-sided precordial leads ([waveform 1](#))
- **Posterior wall ischemia** – Septal precordial leads (V1 to V2) ([waveform 7](#)) and posterior precordial leads ([waveform 8](#))

The right-sided leads V4R, V5R, and V6R ([figure 1](#)) should be obtained if there is evidence of inferior wall ischemia demonstrated by ST elevation in leads II, III, and aVF.

The posterior leads V7, V8, and V9 ([figure 2](#)) may also be helpful if there is evidence of posterior wall ischemia as suggested by prominent R waves and ST depressions in leads V1 and V2.

Importance of serial electrocardiograms — If the initial ECG is not diagnostic but the patient remains symptomatic and clinical suspicion for ACS remains high, the ECG should be repeated at least every 15 to 30 minutes. Patients whose repeat ECGs are diagnostic for or strongly suggestive of either STEMI or NSTEMI should be managed for those diagnoses. (See "[Overview of the acute management of ST-elevation myocardial infarction](#)" and "[Overview of the acute management of non-ST-elevation acute coronary syndromes](#)".)

The initial ECG is often **not** diagnostic in patients with ACS. In two series, the initial ECG was nondiagnostic in 45 percent and normal in 20 percent of patients subsequently shown to have an acute myocardial infarction [1,11]. In the early hours of infarction, peaked, hyperacute T waves may be the only abnormality. In addition to evolution of the ECG, an uncommon source of error is pseudonormalization of baseline T wave inversion [12].

Some clinicians assume that an ECG obtained while the patient is experiencing chest pain that fails to show evidence of ischemia rules out the possibility of ACS. This assumption is false, as demonstrated by two prospective observational studies [13,14].

Left bundle branch block or pacemaker — Both left bundle branch block (LBBB) ([waveform 9](#) and [waveform 10](#)), which is present in approximately 7 percent of patients with an acute myocardial infarction, and pacing can interfere with the ECG diagnosis of coronary-related ischemia [15]. Another problem is that approximately one-half of patients with LBBB and an acute myocardial infarction do not have chest pain as a symptom of their ischemia [16]. As a result, patients with LBBB are much less likely to receive **aspirin**, beta blockers, and

reperfusion therapy [17], particularly if they present without chest pain [16]. Similar observations have been made in patients with a paced rhythm [18].

Careful evaluation of the ECG may show some evidence of ACS in patients with these abnormalities (see "[Electrocardiographic diagnosis of myocardial infarction in the presence of bundle branch block or a paced rhythm](#)"). However, the clinical history and cardiac biomarkers are of primary importance in diagnosing an ACS in this setting.

CLINICAL PRESENTATION

Certain characteristics of the patient's chest discomfort and associated symptoms increase the likelihood of ACS, while others make the diagnosis unlikely. Important characteristics are highlighted below. A more detailed discussion is found separately. (See "[Approach to the patient with suspected angina pectoris](#)".)

Of note, patients who are older or have diabetes are more likely to present with symptoms such as dyspnea, weakness, nausea and vomiting, palpitations, and syncope, and may not manifest chest discomfort. (See '[Atypical presentations](#)' below.)

Ischemic chest pain — Ischemic pain has a number of features that tend to distinguish it from noncardiac pain. These characteristics are described below using the OPQRST mnemonic. Symptoms associated with the highest relative risk of myocardial infarction include radiation to an upper extremity, particularly when there is radiation to both arms, and pain associated with diaphoresis or with nausea and vomiting [19-21]. An important question is whether current pain is reminiscent of prior myocardial infarction. Of note, no one pain characteristic has sufficient positive or negative predictive value to definitively diagnose or exclude ACS.

- **Onset** – Ischemic pain is typically gradual in onset, although the intensity of the discomfort may wax and wane.
- **Provocation and palliation** – Ischemic pain is generally provoked by an activity, such as exercise, that increases cardiac oxygen demand [20,22]. Ischemic pain does not change with respiration or position. It may or may not respond to [nitroglycerin](#) and, if there is improvement, this may only be temporary. Relief of pain following the administration of therapeutic interventions (eg, nitroglycerin, "gastrointestinal cocktail" of viscous [lidocaine](#) and antacid) does **not** reliably distinguish nonischemic from ischemic chest pain [23-25].
- **Quality** – Ischemic pain is often characterized more as a discomfort than pain, and it may be difficult for the patient to describe. Terms frequently used by patients include

squeezing, tightness, pressure, constriction, crushing, strangling, burning, heartburn, fullness in the chest, band-like sensation, knot in the center of the chest, lump in throat, ache, heavy weight on chest (elephant sitting on chest), like a bra too tight, and toothache (when there is radiation to the lower jaw). It is generally not described as sharp, fleeting, knife-like, stabbing, or like "pins and needles" [20].

Increased pain severity does not appear to correlate with an increased likelihood of acute myocardial infarction [26].

In some cases, the patient cannot qualify the nature of the discomfort but places his or her clenched fist in the center of the chest, known as the "Levine sign."

- **Radiation** – Ischemic pain often radiates to other parts of the body including the upper abdomen (epigastrium), shoulders, arms (upper and forearm), wrist, fingers, neck and throat, lower jaw and teeth (but not upper jaw), and not infrequently to the back (specifically the interscapular region). The old dictum that "pain above the nose or below the navel is rarely cardiac in origin" still holds. Pain radiating to the upper extremities is highly suggestive of ischemic pain [19,20,22,27].
- **Site** – Ischemic pain is not felt in one specific spot; rather, it is a diffuse discomfort that may be difficult to localize. The patient often indicates the entire chest, rather than localizing it to a specific area by pointing a single finger.
- **Time course** – Angina is usually brief (two to five minutes) and is relieved by rest or with **nitroglycerin**. In comparison, patients with an ACS may have chest pain at rest, and the duration is variable but generally lasts longer than 30 minutes. Classic anginal pain lasting more than 20 minutes suggests ACS. Continuous pain that does not wax and wane and persists for over 24 hours is unlikely to be due to ACS [28].

Historical features increasing likelihood of ACS — Historical features that increase the likelihood of ACS include the following:

- Patients with a prior history of ACS have a significantly increased risk of recurrent ischemic events.
- A prior history of other vascular disease is associated with a risk of cardiac ischemic events comparable to that seen with a prior history of ACS.
- Risk factors for ACS, particularly age, male sex, diabetes, hypertension, dyslipidemia, and cigarette smoking [29]. (See "[Overview of established risk factors for cardiovascular disease](#)".)

- Recent use of cocaine or other sympathomimetic drugs (eg, methamphetamine, cathinones) [30]. (See "[Clinical manifestations, diagnosis, and management of the cardiovascular complications of cocaine abuse](#)" and "[Methamphetamine: Acute intoxication](#)" and "[Acute amphetamine and synthetic cathinone \("bath salt"\) intoxication](#)".)

Associated symptoms — Ischemic pain is often associated with other symptoms. The most common is shortness of breath, which may reflect mild pulmonary congestion resulting from ischemia-mediated diastolic dysfunction. Other symptoms may include belching, nausea, indigestion, vomiting, diaphoresis, dizziness, lightheadedness, clamminess, and fatigue.

Noncardiac chest pain — Specific chest pain characteristics can be used to help differentiate cardiac from noncardiac causes. (See "[Evaluation of the adult with chest pain in the emergency department](#)".)

Systematic reviews have identified the following characteristics as more typical of **nonischemic** chest discomfort [19]:

- Pleuritic pain, sharp or knife-like pain related to respiratory movements or cough
- Primary or sole location in the mid or lower abdominal region
- Any discomfort localized with one finger
- Any discomfort reproduced by movement or palpation
- Constant pain lasting for days
- Fleeting pains lasting for a few seconds or less
- Pain radiating into the lower extremities or above the mandible

However, some patients with ACS present with so-called "atypical" chest pain. This was illustrated in the Multicenter Chest Pain Study in which acute ischemia was diagnosed in 22 percent of patients who presented with sharp or stabbing pain and 13 percent who presented with pleuritic-type pain [31]. (See '[Atypical presentations](#)' below.)

In addition, some patients who appear to have a noncardiac cause of chest pain have other serious conditions including acute aortic dissection, pulmonary embolism, tension pneumothorax, myocarditis, perforating peptic ulcer, and esophageal rupture ([table 5](#)). It is essential to consider these alternate diagnoses to avoid potentially dangerous errors in management, such as the administration of antiplatelet, anticoagulant, or thrombolytic therapy to a patient with an aortic dissection.

ATYPICAL PRESENTATIONS

Some patients with ACS present with symptoms other than chest pain. In a review of over 430,000 patients with confirmed acute myocardial infarction from the National Registry of Myocardial Infarction II, one-third had no chest pain on presentation to the hospital [32]. These patients often present with symptoms such as dyspnea alone, weakness, nausea and/or vomiting, epigastric pain or discomfort, palpitations, syncope, or cardiac arrest. They are more likely to be older or have diabetes [20,32-34]. (See "[Clinical features and diagnosis of coronary heart disease in women](#)", section on '[Clinical presentation](#)' and "[Prevalence of and risk factors for coronary heart disease in patients with diabetes mellitus](#)", section on '[Silent ischemia and infarction](#)'.)

The absence of chest pain has important implications for therapy and prognosis. In the Registry report, patients without chest pain were much less likely to be diagnosed with a confirmed myocardial infarction on admission (22 versus 50 percent in those with chest pain) and were less likely to be treated with appropriate medical therapy and to receive thrombolytic therapy or primary percutaneous coronary intervention (PCI; 25 versus 74 percent) [32]. Not surprisingly, these differences were associated with an increase in in-hospital mortality (23.3 versus 9.3 percent, adjusted odds ratio [aOR] 2.21, 95% CI 2.17-2.26).

PHYSICAL EXAMINATION

The initial physical examination should focus on findings that permit rapid triage and aid in immediate diagnosis and management and should include the following:

- **Responsiveness, airway, breathing, and circulation** – In patients in respiratory or cardiorespiratory arrest, the appropriate resuscitation algorithms should be followed ([algorithm 2](#) and [algorithm 3](#) and [algorithm 4](#)). (See "[Advanced cardiac life support \(ACLS\) in adults](#)" and "[Overview of advanced airway management in adults for emergency medicine and critical care](#)" and "[Basic airway management in adults](#)" and "[Rapid sequence intubation in adults for emergency medicine and critical care](#)".)
- **Evidence of systemic hypoperfusion (hypotension; tachycardia; impaired cognition; cool, clammy, pale, ashen skin)** – Cardiogenic shock complicating acute myocardial infarction requires aggressive evaluation and management ([table 6](#)). (See "[Clinical manifestations and diagnosis of cardiogenic shock in acute myocardial infarction](#)" and "[Prognosis and treatment of cardiogenic shock complicating acute myocardial infarction](#)".)
- **Evidence of heart failure (jugular venous distention, new or worsening pulmonary crackles, hypotension, tachycardia, new S3 gallop, new or worsening mitral**

regurgitation murmur) – Aggressive management is needed for patients with heart failure complicating an acute myocardial infarction, depending upon the severity of the heart failure and the presence of other risk factors ([table 7](#) and [table 6](#)) [35]. (See "Treatment of acute decompensated heart failure in acute coronary syndromes".)

- **Focused neurologic examination** – A screening neurologic examination should be performed to assess for focal lesions or cognitive deficits that might preclude safe use of thrombolytic therapy ([table 4](#)). (See "[Acute ST-elevation myocardial infarction: The use of fibrinolytic therapy](#)".)

The cardiac and general physical examinations of patients with possible ACS are reviewed in greater detail separately. (See "[Auscultation of heart sounds](#)" and "[Auscultation of cardiac murmurs in adults](#)" and "[Evaluation of the adult with chest pain in the emergency department](#)", section on 'Physical examination' and "[Approach to the adult with dyspnea in the emergency department](#)", section on 'Physical examination').

CARDIAC BIOMARKERS AND OTHER LABORATORY TESTING

Serial serum cardiac biomarkers (sometimes referred to as cardiac enzymes) of acute myocardial damage are essential in the evaluation of patients with suspected ACS. They should be obtained in any patient at significant risk of ACS. High-sensitivity troponin is the preferred test. The use and test characteristics of troponin are discussed separately. (See "[Troponin testing: Clinical use](#)" and "[Elevated cardiac troponin concentration in the absence of an acute coronary syndrome](#)".)

Basic electrolyte concentrations, kidney function studies, and a complete blood count with platelets should be obtained in patients with suspected ACS. Anemia can exacerbate myocardial ischemia, and patients with a low hemoglobin may require blood transfusion. Transfusion thresholds and implementation are reviewed separately. (See "[Indications and hemoglobin thresholds for RBC transfusion in adults](#)", section on 'Acute MI').

Indices of coagulation should be obtained in patients at increased risk of coagulopathy due to [warfarin](#) or heparin use, a history of liver disease, or a history of excessive or spontaneous bleeding. Additional laboratory testing is obtained depending on clinical circumstances, including patient comorbidities.

MANAGEMENT

ST elevation — The management of patients who meet the criteria for ST elevation myocardial infarction (STEMI) is discussed separately. The accompanying table provides a concise summary of the immediate treatment interventions needed in these patients ([table 3](#)). (See 'Electrocardiogram assessment' above and "Overview of the acute management of ST-elevation myocardial infarction".)

Selection and implementation of the optimal reperfusion strategy is the most important step in the management of STEMI and is discussed separately. Reperfusion therapy, whether percutaneous coronary intervention (PCI) or thrombolytics, should **not** await the result of cardiac biomarker measurement. (See "Acute ST-elevation myocardial infarction: Selecting a reperfusion strategy" and "Primary percutaneous coronary intervention in acute ST elevation myocardial infarction: Determinants of outcome" and "Acute ST-elevation myocardial infarction: The use of fibrinolytic therapy".)

Rapid implementation of primary PCI is more likely to be achieved if the hospital protocol involves immediate activation of the PCI team by prehospital emergency medical services (EMS) [36] or emergency department clinician and immediate transfer of the patient to the catheterization laboratory [37].

Anemic patients may require blood transfusion. Transfusion thresholds and implementation are reviewed separately. (See "Indications and hemoglobin thresholds for RBC transfusion in adults", section on 'Acute MI').

Non-ST elevation

A patient whose presentation raises concern for coronary-related ischemia but who does not manifest ST elevations on electrocardiogram (ECG) is suspected of having a non-ST elevation myocardial infarction (NSTEMI) or possibly unstable angina. The management of patients with NSTEMI is discussed separately. The accompanying table provides a concise summary of the immediate treatment interventions needed in these patients ([table 3](#)). (See 'Electrocardiogram assessment' above and "Overview of the acute management of non-ST-elevation acute coronary syndromes".)

Unstable angina and NSTEMI comprise part of the spectrum of ACS. Angina is considered unstable if it presents in any of the following three ways:

- Rest angina, generally lasting longer than 20 minutes
- New-onset angina that markedly limits physical activity
- Increasing angina that is more frequent, lasts longer, or occurs with less exertion than previous angina

NSTEMI is distinguished from unstable angina by the presence of elevated serum biomarkers. ST segment elevations and Q waves are absent in both unstable angina and NSTEMI. As a result, unstable angina and NSTEMI can be indistinguishable at initial evaluation since an elevation in serum biomarkers may not be detectable for two to four hours after a myocardial infarction with contemporary troponin assays and one to three hours with high-sensitivity assays, and at least 12 hours are required to detect elevations in all patients. (See '[Cardiac biomarkers and other laboratory testing](#)' above and '[Acute coronary syndrome: Terminology and classification](#)').

Thrombolytic therapy should **not** be administered to patients with unstable angina or NSTEMI unless subsequent ECG monitoring documents ST segment elevations that persist. Repeat ECGs are an essential part of management in these patients. (See '[Overview of the acute management of non-ST-elevation acute coronary syndromes](#)' and '[Importance of serial electrocardiograms](#)' above.)

Anemic patients may require blood transfusion. Transfusion thresholds and implementation are reviewed separately. (See '[Indications and hemoglobin thresholds for RBC transfusion in adults](#)', section on '[Acute MI](#)').

Cardiac arrhythmias during ACS — Disturbances of cardiac rhythm during a myocardial infarction are usually detected by cardiac monitor rather than by physical examination or 12-lead ECG:

- Sustained ventricular tachyarrhythmias in the peri-infarction period must be treated immediately because of their deleterious effect on cardiac output, possible exacerbation of myocardial ischemia, and the risk of deterioration into ventricular fibrillation ([algorithm 2](#)). (See '[Ventricular arrhythmias during acute myocardial infarction: Incidence, mechanisms, and clinical features](#)').
- While supraventricular tachyarrhythmias in the peri-infarction period may pose less immediate risk of cardiac arrest, the management of such arrhythmias is important because any tachycardia can increase myocardial oxygen demand, thereby exacerbating ischemia and possibly decreasing cardiac output ([algorithm 4](#)). (See '[Supraventricular arrhythmias after myocardial infarction](#)').
- Bradyarrhythmias occurring early in the setting of an inferior wall myocardial infarction (within the first 24 hours) may respond to treatment with **atropine** ([algorithm 3](#)). Later bradyarrhythmias, wide QRS-complex bradyarrhythmias, and those occurring in the setting of an anterior wall myocardial infarction may require temporary pacemaker placement. (See '[Conduction abnormalities after myocardial infarction](#)').

Disposition of patient without STEMI — For patients without STEMI, the ECG remains a critical component in determining risk for adverse outcomes in ACS. The patient's hemodynamic status, serum biomarkers, and historical risk factors, as well as available hospital resources, should also be used to determine appropriate disposition. Cardiology consultation is useful generally and for any case in which the diagnosis or treatment plan are unclear. The utility of epidemiologic risk factors in determining acute, individual risk has been questioned [38].

The diagnosis of myocardial infarction is secured when there is a rise and/or fall of troponin (high sensitivity assays are preferred) along with supportive evidence in the form of typical symptoms, suggestive ECG changes (eg, ST segment depression ≥ 0.05 mV [0.5 mm] in two or more contiguous leads), or imaging evidence of new loss of viable myocardium or new regional wall motion abnormality. (See "[Diagnosis of acute myocardial infarction](#)".)

This patient is typically admitted to an intensive care unit, coronary care unit, or monitored cardiac unit depending upon the persistence of symptoms and evidence of hemodynamic compromise. Those with persistent pain or hemodynamic compromise generally undergo urgent angiography and revascularization. Others with resolution of symptoms and stable hemodynamics are typically admitted for early elective angiography and revascularization if appropriate.

A high score on a validated ACS risk stratification tool suggests the patient is at high risk. Such scores include the HEART Score ([table 8](#)) and EDACS Score [39]. If there is no ST segment elevation or depression, regardless of the presence or absence of Q waves, the patient with definite or probable ACS should still be admitted to a monitored care unit or ED observation unit for further evaluation. (See "[Evaluation of emergency department patients with chest pain at low or intermediate risk for acute coronary syndrome](#)", section on 'Risk scores'.)

IMPACT OF MISSED DIAGNOSIS

With careful evaluation using effective risk scores, serial electrocardiograms (ECGs), and appropriate diagnostic testing, only a very small percentage of patients with an ACS are mistakenly discharged from the emergency department [40,41]. However, patients whose diagnosis is missed initially have an increase in short-term mortality [33,40,42,43]. This issue was evaluated in a review of 10,689 Black, White, and Hispanic American patients who presented to the emergency department with symptoms suggesting acute coronary-related ischemia: 8 percent had an acute myocardial infarction, and 9 percent had unstable angina [33]. Among the patients with an ACS, 2.2 percent were mistakenly discharged from the emergency

department. Atypical presentation most frequently led to missed diagnosis. The patients with missed myocardial infarction had the following characteristics:

- Females less than 55 years of age
- Not White Americans
- Shortness of breath as the major presenting symptom
- Normal or nondiagnostic ECG

Misreading of the ECG was an infrequent problem. There was a nonsignificant trend toward an increased risk-adjusted 30-day mortality ratio for patients who were not hospitalized (1.9 and 1.7 in the patients with myocardial infarction and unstable angina, respectively). While improvements in care since the publication of this study have reduced the rate of missed ACS in the emergency department, lessons about which patient groups are most at risk bear remembering.

OBSERVATION

Some patients without clear evidence of ACS by clinical history, electrocardiogram (ECG), or biomarker measurement ultimately sustain a myocardial infarction or develop unstable angina. Therefore, patients with an uncertain diagnosis after initial assessment require further observation and evaluation. The management of such patients, including the use of chest pain observation units, is discussed separately. (See "[Evaluation of emergency department patients with chest pain at low or intermediate risk for acute coronary syndrome](#)".)

REST AND STRESS IMAGING STUDIES

Rest imaging tests, including radionuclide myocardial perfusion imaging (rMPI) and echocardiography, may be of value in the evaluation of patients who have persistent chest pain suggestive of an ACS, a nondiagnostic electrocardiogram (ECG), and initially or serially negative cardiac biomarkers. Some patients in whom the chest pain has resolved may undergo stress testing with or without imaging. Use of these diagnostic tests is discussed separately. (See "[Evaluation of emergency department patients with chest pain at low or intermediate risk for acute coronary syndrome](#)", section on 'Noninvasive evaluation' and "[Noninvasive testing and imaging for diagnosis in patients at low to intermediate risk for acute coronary syndrome](#)".)

In addition to detecting myocardial dysfunction or ischemia consistent with the diagnosis of a myocardial infarction, imaging studies such as a contrast-enhanced chest computed tomography (CT) or cardiovascular magnetic resonance imaging (MRI) can be used to

differentiate a myocardial infarction from an aortic dissection in patients for whom this distinction is initially unclear. (See "[Clinical features and diagnosis of acute aortic dissection](#)", [section on 'Diagnosis'](#).)

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: Non-ST-elevation acute coronary syndromes \(non-ST-elevation myocardial infarction\)](#)" and "[Society guideline links: ST-elevation myocardial infarction \(STEMI\)](#)" and "[Society guideline links: Adult with chest pain in the emergency department](#)".)

SUMMARY AND RECOMMENDATIONS

- **Differential diagnosis of chest pain** – Chest discomfort can be caused by a number of life-threatening conditions, including pulmonary embolism, aortic dissection, and pneumothorax. Emergency clinicians must avoid premature diagnosis of acute coronary syndrome (ACS). The evaluation of chest pain in the emergency department is discussed separately. (See "[Evaluation of the adult with chest pain in the emergency department](#)".)
- **Immediate interventions** – If ACS is the leading diagnosis, initial assessment and interventions must be performed rapidly to minimize potential injury to the myocardium. During the initial assessment phase, the following steps should be accomplished for any patient at significant risk for ACS:
 - Airway, breathing, and circulation assessed
 - Preliminary history and examination obtained (See '[Clinical presentation](#)' above.)
 - 12-lead electrocardiogram (ECG) interpreted
 - Resuscitation equipment brought to the bedside
 - Cardiac monitor attached to patient
 - Supplemental oxygen given to maintain oxygen saturation ≥90 percent
 - Intravenous (IV) access and blood work obtained (including contemporary or high-sensitivity troponin; hemoglobin to assess for anemia) (See '[Cardiac biomarkers and other laboratory testing](#)' above.)
 - **Aspirin** 162 to 325 mg given
 - Nitrates given (unless contraindicated) (see '[Immediate emergency department interventions](#)' above)

- **Electrocardiogram** – The initial ECG should be obtained within 10 minutes of presentation but is often **not** diagnostic in patients with ACS. In patients without a clear diagnosis but at risk for ACS, ECGs should be repeated at frequent intervals (every 15 to 30 minutes) until the patient's chest pain resolves or a definitive diagnosis is made. ECG assessment is described in the text. (See '[Electrocardiogram assessment](#)' above.)
- **Risk factors** – Important risk factors for acute myocardial infarction include a personal or family history of ACS or other cardiovascular disease, older age, diabetes, dyslipidemia, cigarette smoking, hypertension, and abuse of cocaine or other sympathomimetic drugs. (See "[Overview of established risk factors for cardiovascular disease](#)" and '[When to suspect acute coronary syndrome](#)' above.)
- **Clinical presentation** – Characteristics of the patient's chest discomfort and associated symptoms that increase or decrease the likelihood of ACS are described in the text. Many patients with ACS present with symptoms such as dyspnea or malaise, either alone or in addition to chest pain. Females are more likely to have associated dyspnea than males, and patients who are older or have diabetes are more likely to present with dyspnea without chest pain. Relief of symptoms following the administration of therapeutic interventions (eg, [nitroglycerin](#), "gastrointestinal cocktail" of viscous [lidocaine](#) and antacid) does **not** reliably distinguish nonischemic from ischemic chest pain. (See '[Clinical presentation](#)' above and '[Atypical presentations](#)' above.)
- **Physical examination** – The initial physical examination should focus on findings that permit rapid triage and aid in immediate diagnosis and management. (See '[Physical examination](#)' above.)
- **Algorithm and rapid overview table for management** – The attached algorithm provides a simple approach to risk stratification while the table provides a concise outline of the immediate interventions needed to manage ST elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI), and unstable angina ([algorithm 1](#) and [table 3](#)). (See '[Management](#)' above.)
- **Missed diagnosis** – With careful evaluation using appropriate risk scores and diagnostic testing, only a very small percentage of patients with an ACS are mistakenly discharged from the emergency department. However, these patients have an increase in short-term mortality. American patients whose ACS was missed are more likely to be females less than 55 years of age, not White Americans, patients with shortness of breath as the major presenting symptom, and patients with a normal or nondiagnostic initial ECG. (See '[Impact of missed diagnosis](#)' above.)

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Topic 184 Version 56.0

GRAPHICS

Abbreviated list of causes of chest pain in patients presenting to the emergency department

Cardiac

Myocardial ischemia or infarction

Aortic dissection

Pericarditis

Pulmonary

Pulmonary embolus and other causes of pulmonary hypertension

Chest wall trauma

Myocardial or pericardial contusion

Traumatic valve injury

Aortic injury

Noncardiac chest pain

Gastrointestinal

Musculoskeletal

Graphic 54763 Version 2.0

Diagnoses other than cardiac ischemia for patients with chest pain

Cardiovascular not caused by epicardial CAD	Pulmonary	Gastrointestinal
Aortic dissection*	Pleuritis	Biliary
Aortic aneurysm*	Pneumonia	Cholangitis
Myocarditis*	Pulmonary embolus*	Cholecystitis
Pericarditis	Tension pneumothorax*	Choledocholithiasis
Stress (takotsubo) cardiomyopathy*		Colic
Chest wall	Psychiatric	Esophageal
Cervical disc disease	Affective disorders (eg, depression)	Esophagitis
Costochondritis	Anxiety disorders	Spasm
Fibrositis	Hyperventilation	Reflux
Herpes zoster (before the rash)	Panic disorder	Rupture*
Neuropathic pain	Primary anxiety	Pancreatitis
Rib fracture	Somatoform disorders	Peptic ulcer disease
Sternoclavicular arthritis	Thought disorders (eg, fixed delusions)	Nonperforating
		Perforating*

* Potentially life-threatening conditions.

Adapted with permission from: ACC/AHA/ACP Guidelines for the Management of Patients with Chronic Stable Angina. J Am Coll Cardiol 1999; 33:2092. Copyright ©1999 American College of Cardiology.

Graphic 53227 Version 4.0

Characteristics of major noncardiac causes of chest pain

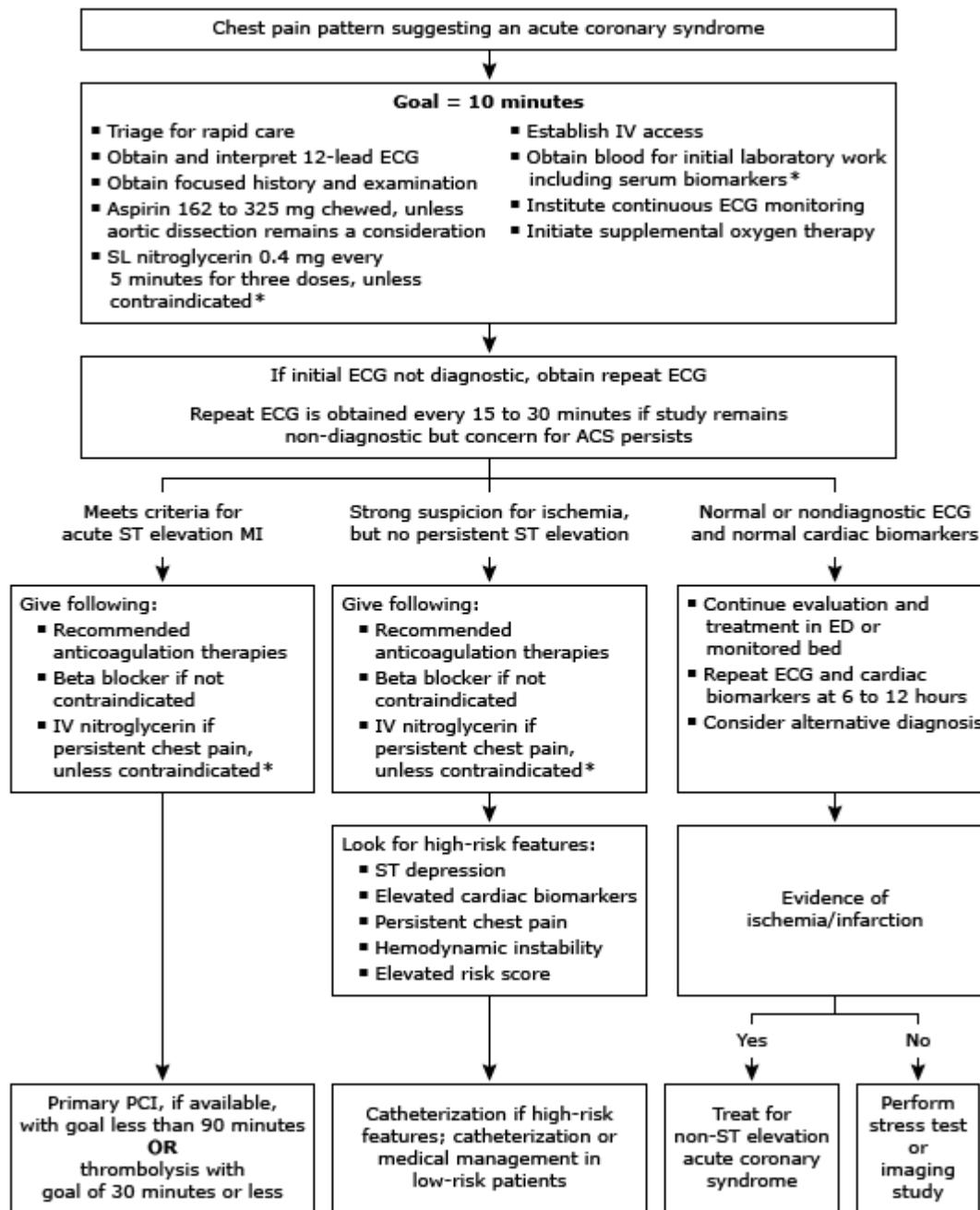
Condition	Duration of pain	Character of pain
Gastroesophageal reflux	5 to 60 minutes	Visceral, substernal, worse with recumbency, no radiation, relief with food, antacids
Esophageal spasm	5 to 60 minutes	Visceral, spontaneous, substernal, associated with cold liquids, relief with nitroglycerin
Peptic ulcer	Hours	Visceral, burning, epigastric, relief with food, antacids, normal ECG
Biliary disease	Hours	Visceral, epigastric, interscapular colic, occurs after meals
Cervical disc	Variable	Superficial, positional, arm, neck
Musculoskeletal	Variable	Superficial, positional, worse with movement, local tenderness
Hyperventilation	2 to 3 minutes	Visceral, substernal, tachypneic, anxious
Thyroiditis	Persistent	Aggravated by swallowing, neck, throat tenderness

Excludes pain above the neck or below the umbilicus.

ECG: electrocardiogram.

Graphic 77753 Version 3.0

Overview of approach to patients with suspected acute myocardial infarction in the emergency department



ED: emergency department; ECG: electrocardiogram; SL: sublingual; IV: intravenous; ACS: acute coronary syndrome; MI: myocardial infarction; PCI: percutaneous coronary intervention; CBC: complete blood count; PT: prothrombin time; aPTT: activated partial thromboplastin time; INR: international normalized ratio; BUN: blood urea nitrogen.

* Initial laboratory work may vary by institution, but often includes: serum cardiac biomarkers, CBC with platelet count, PT and INR, aPTT, basic electrolytes, magnesium, BUN, creatinine, blood glucose, and serum lipid profile. Contraindications to nitrates include: severe aortic stenosis, hypertrophic

cardiomyopathy, suspected right ventricular infarct, hypotension, marked bradycardia or tachycardia, and recent use of phosphodiesterase 5 inhibitor (eg, Viagra).

Graphic 56813 Version 10.0

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 O₂ 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 <1 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 ($\text{CrCl} < 30 \text{ mL/minute}$)^{*}: 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 ($\text{CrCl} < 30 \text{ mL/minute}$)^{*}: 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 therapeutic 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^o 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 $\text{CrCl} < 30 \text{ mL/minute}$.

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 ($\text{CrCl} < 30 \text{ mL/minute}$)^{*}: 1 mg/kg subcutaneously every 24 hours.
- **Fondaparinux:** 2.5 mg subcutaneously every 24 hours. This drug should be avoided in patients with a $\text{CrCl} < 30 \text{ mL/minute}$.

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

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

Absolute contraindications

History of any intracranial hemorrhage

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

Presence of a cerebral vascular malformation or a primary or metastatic intracranial malignancy

Symptoms or signs suggestive of an aortic dissection

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

Significant closed-head or facial trauma within the preceding three months

Relative contraindications

History of chronic, severe, poorly controlled hypertension or uncontrolled hypertension at presentation (eg, blood pressure >180 mmHg systolic and/or >110 mmHg diastolic; severe hypertension at presentation can be an absolute contraindication in patients at low risk)

History of ischemic stroke more than three months previously

Dementia

Any known intracranial disease that is not an absolute contraindication

Traumatic or prolonged (>10 min) cardiopulmonary resuscitation

Major surgery within the preceding three weeks

Internal bleeding within the preceding two to four weeks or an active peptic ulcer

Noncompressible vascular punctures

Pregnancy

Current warfarin therapy; the risk of bleeding increases as the INR increases

For streptokinase or anistreplase, a prior exposure (more than five days previously) or allergic reaction to these drugs

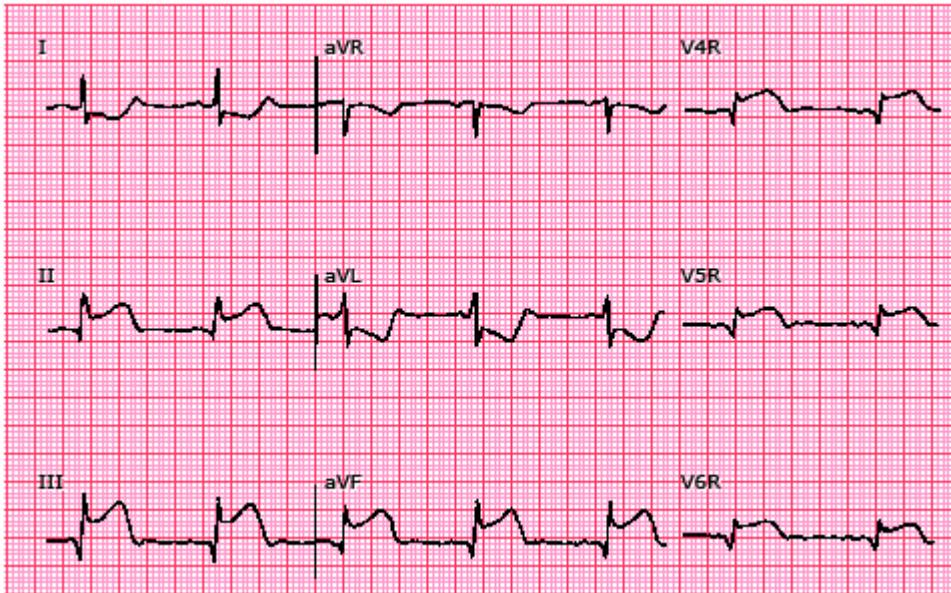
INR: international normalized ratio.

* May not be all-inclusive or definitive.

Data from: Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction--executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infarction). Circulation 2004; 110:588.

Graphic 68784 Version 11.0

ECG of acute inferior and right ventricular myocardial infarction



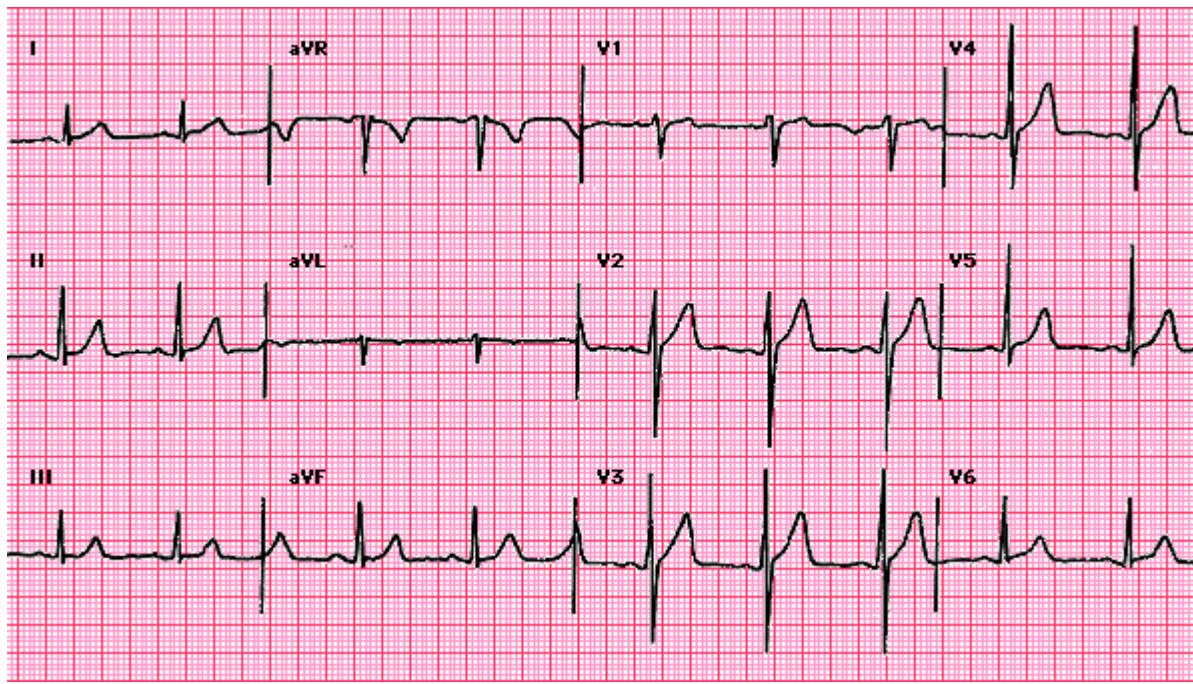
ECG shows Q waves and prominent doming ST-segment elevation in II, III, and aVF, findings which are characteristic of an acute inferior myocardial infarction. ST elevation in the right precordial leads - V4R, V5R, and V6R - indicates right ventricular involvement as well. The ST depressions in leads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

ECG: electrocardiogram.

Courtesy of Ary Goldberger, MD.

Graphic 82739 Version 4.0

Normal ECG

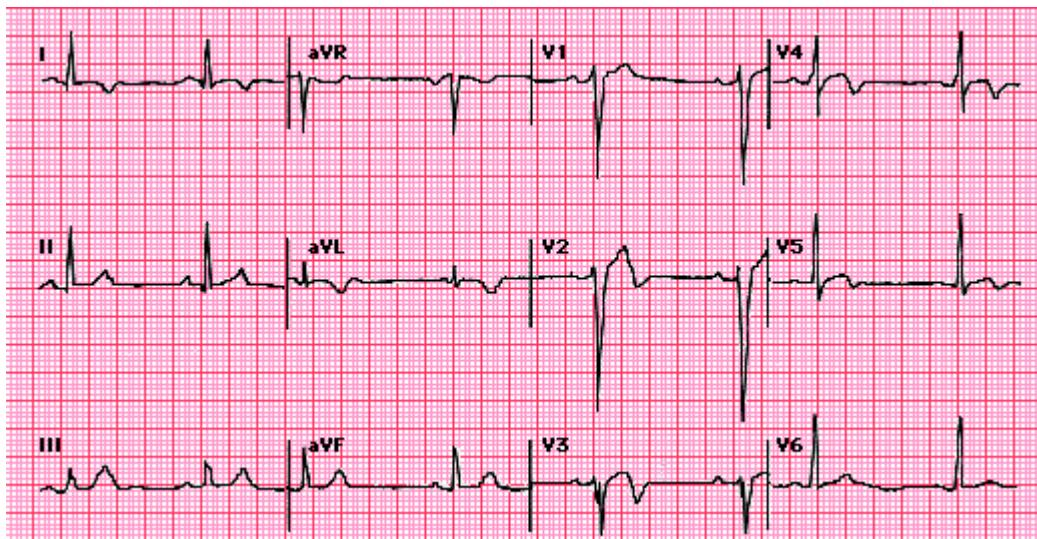


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

Electrocardiogram (ECG) in an evolving anterior myocardial infarction

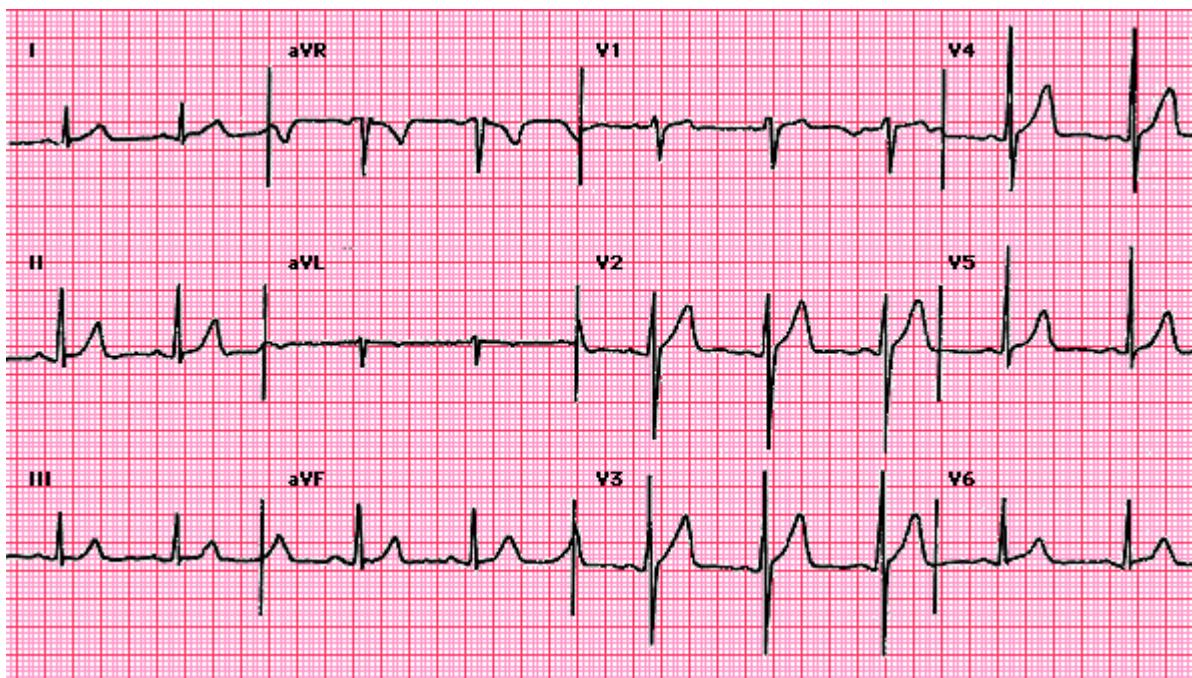


Electrocardiogram shows findings typical of an evolving Q-wave anterior MI: loss of R waves in leads V1 to V3, ST segment elevations in V2 to V4, and T wave inversions in leads I, aVL, and V2 to V5. Sinus bradycardia (55 beats/min) is present due to concurrent therapy with a beta blocker.

Courtesy of Ary Goldberger, MD.

Graphic 81914 Version 3.0

Normal ECG

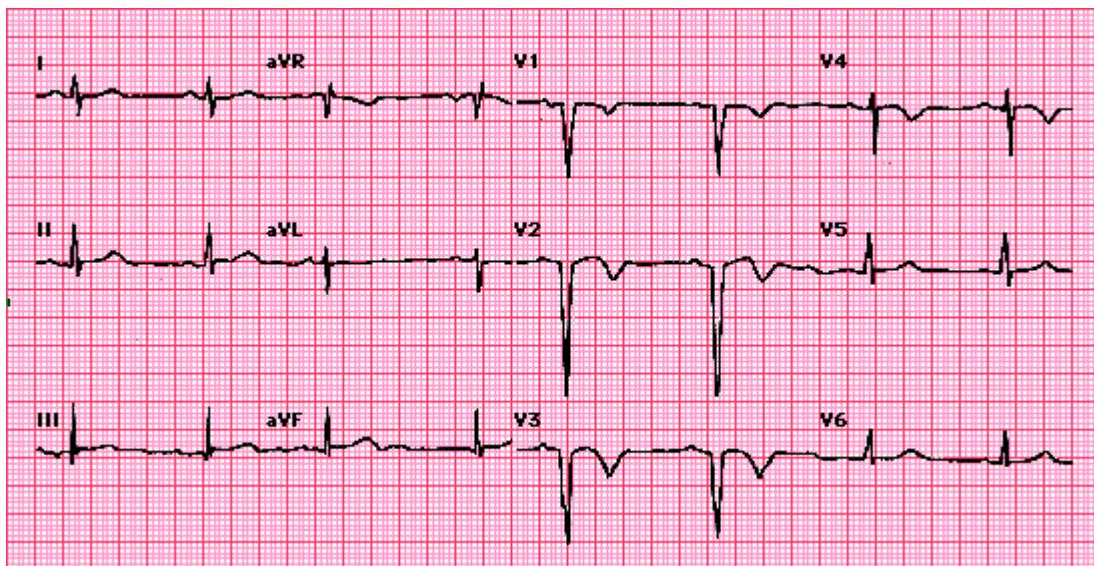


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

Electrocardiogram (ECG) late in the evolution of an anterior myocardial infarction

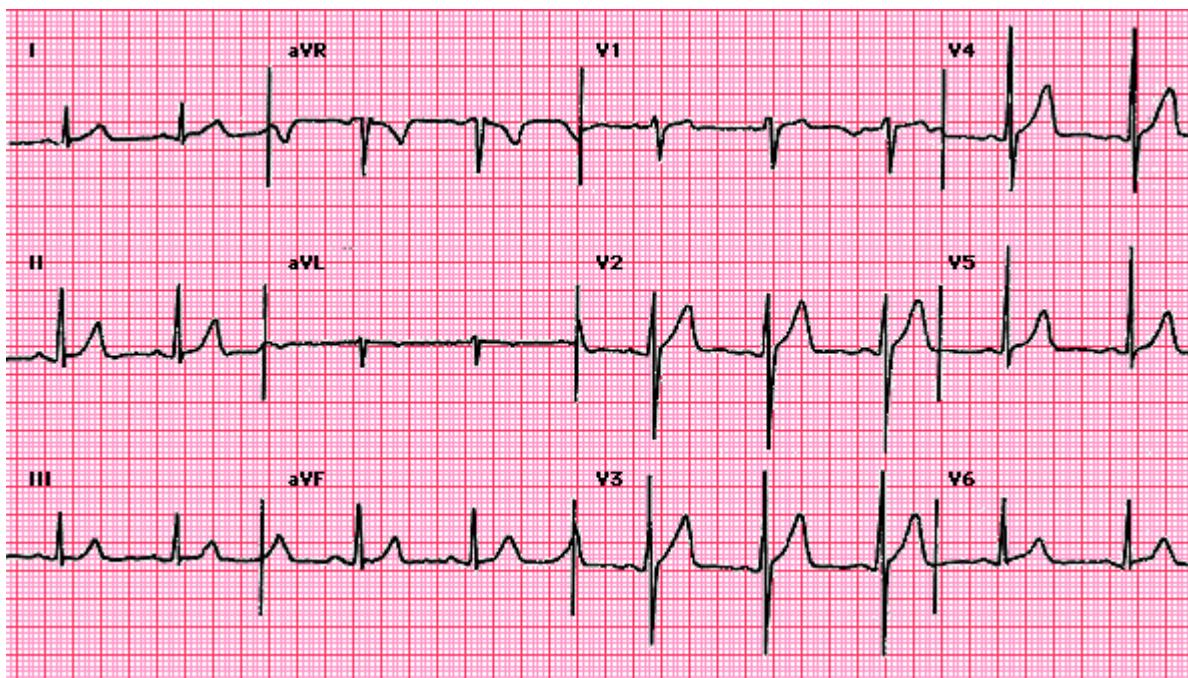


Later stage in the evolution of an acute anterior myocardial infarction. There is a QS pattern in leads V1 to V3 and T wave inversion in leads V2 to V4. The ST segment elevations in these leads have almost disappeared.

Courtesy of Ary Goldberger, MD.

Graphic 62059 Version 3.0

Normal ECG

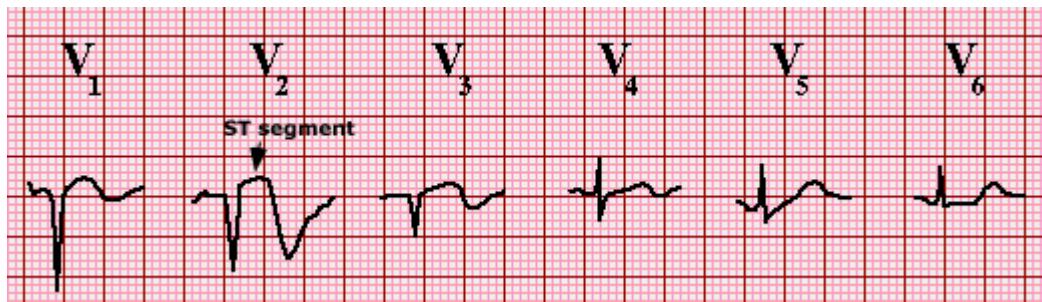


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

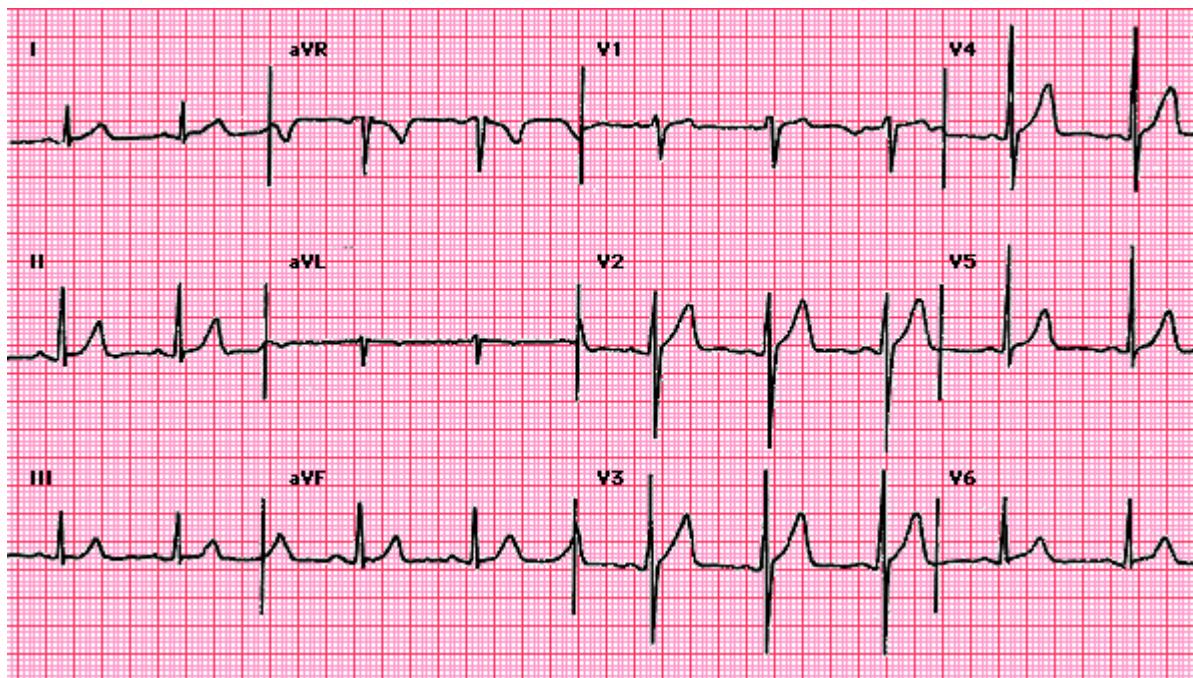
Acute anteroseptal myocardial infarction



ST segment elevation in V₁ and V₂ is characteristic of an acute anteroseptal infarct. There is also reciprocal ST segment depression in V₅ and V₆.

Graphic 75683 Version 2.0

Normal ECG

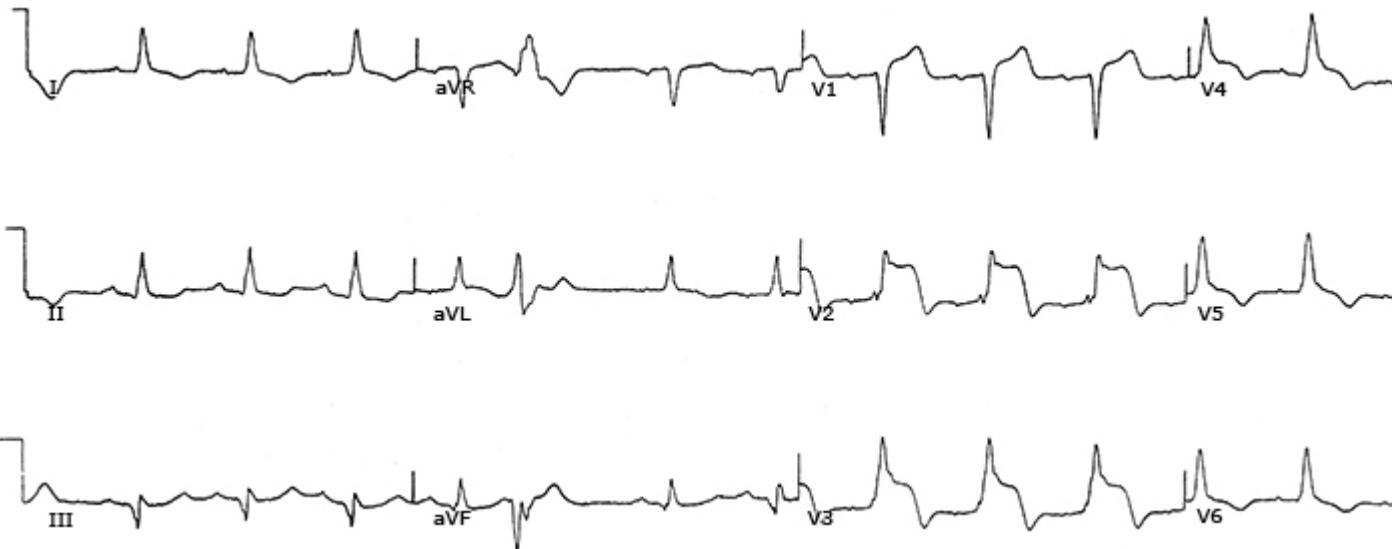


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

Anteroseptal acute MI ECG



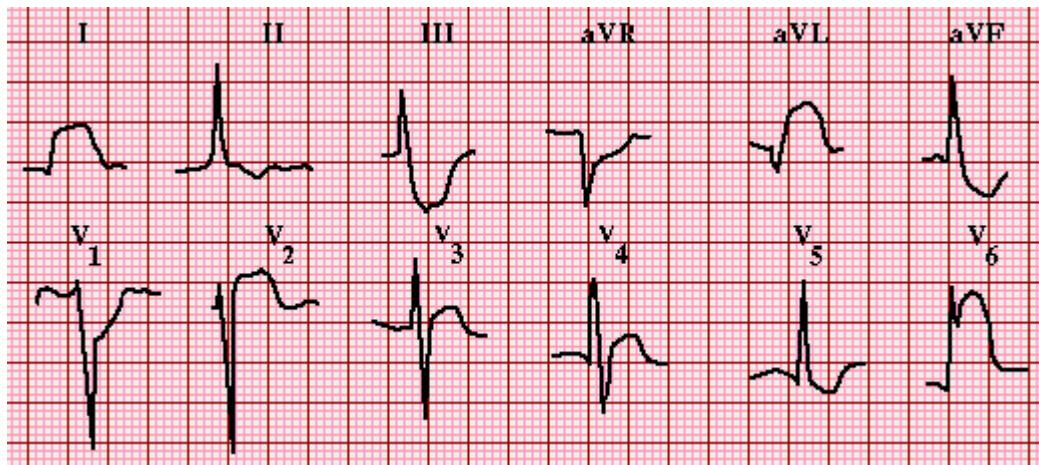
ECG shows a deep Q wave in lead V1 and ST elevations in leads V1 through V4, findings consistent with an acute anteroseptal myocardial infarction.

MI: myocardial infarction; ECG: electrocardiogram.

From: Textbook of Pediatric Emergency Medicine, 5th ed, Fleisher GR (Ed), Wolters Kluwer 2005. Copyright © 2005. Reproduced with permission from Wolters Kluwer Health.

Graphic 129526 Version 1.0

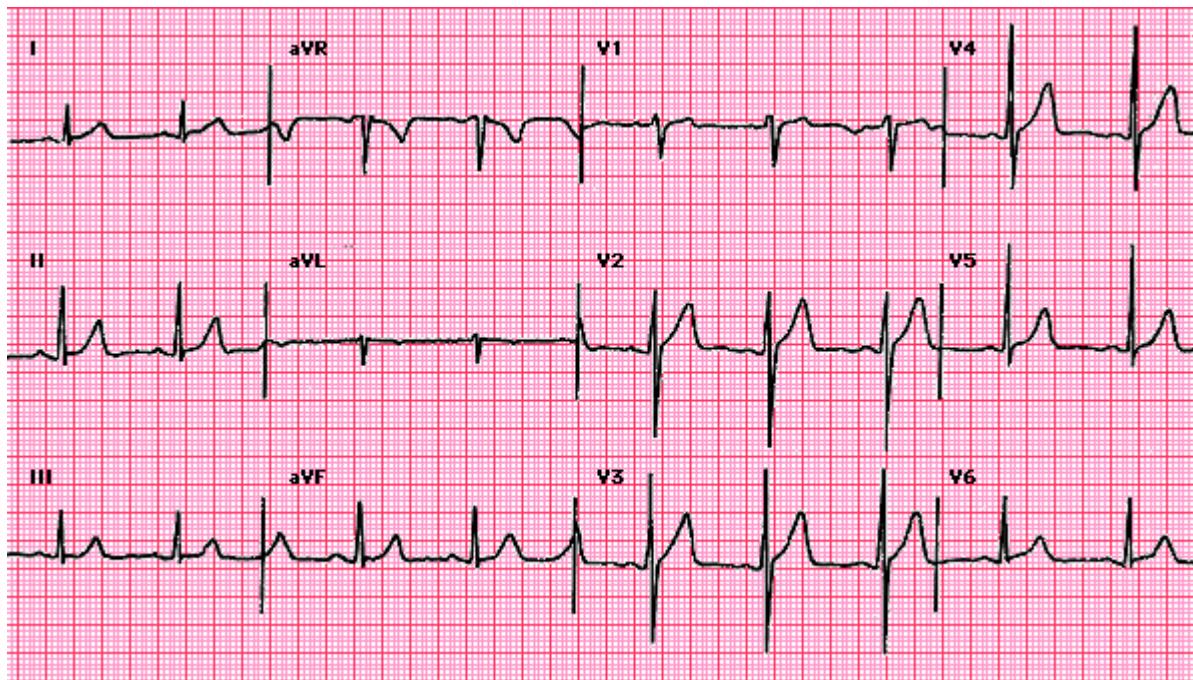
Acute lateral transmural myocardial infarction



ST elevation in leads I and aVL is characteristic of an acute lateral wall infarct. Reciprocal ST depression is evident in this case in the inferior leads (II, III, and aVF) and in V₁.

Graphic 82413 Version 2.0

Normal ECG

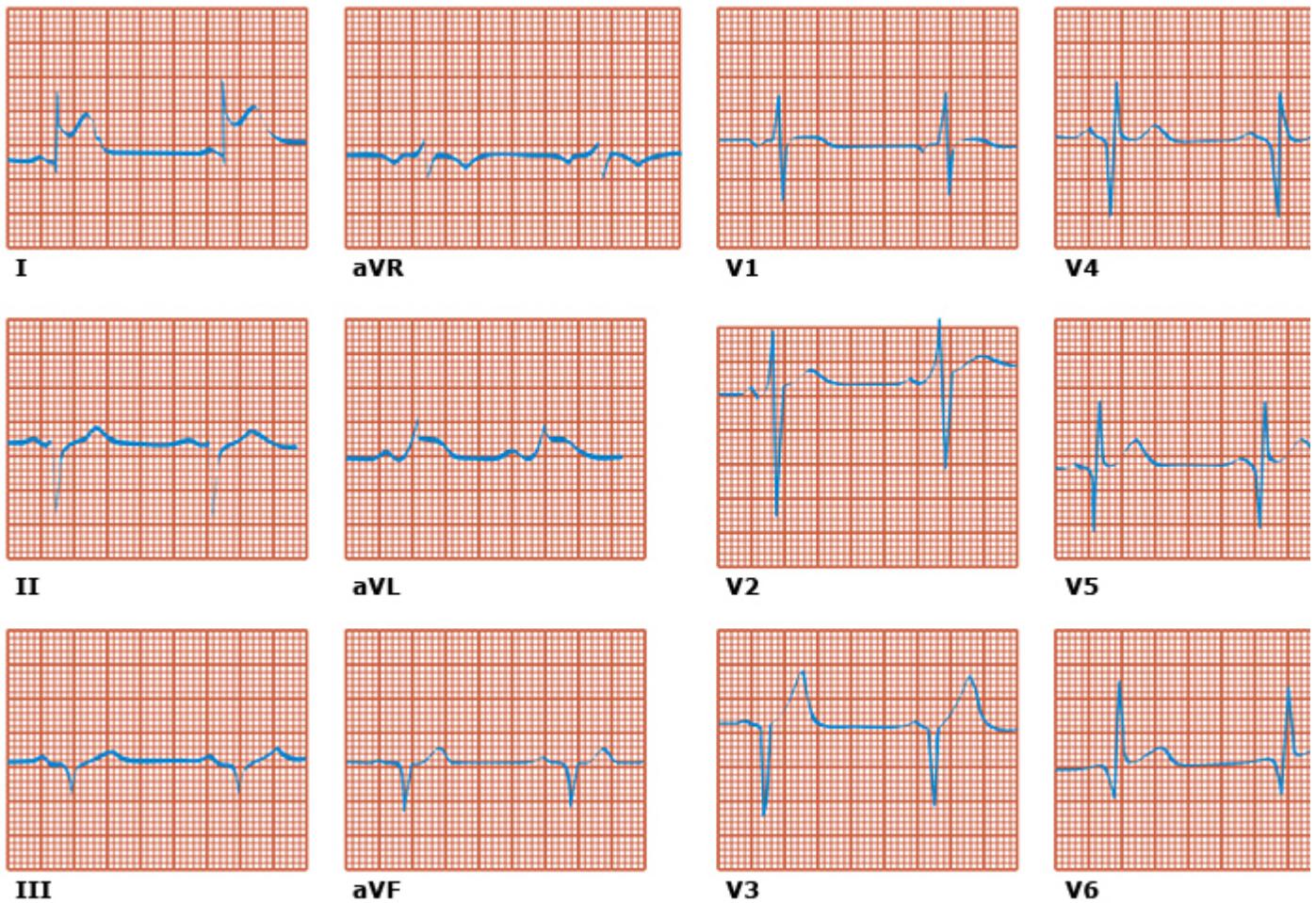


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

Lateral acute MI ECG



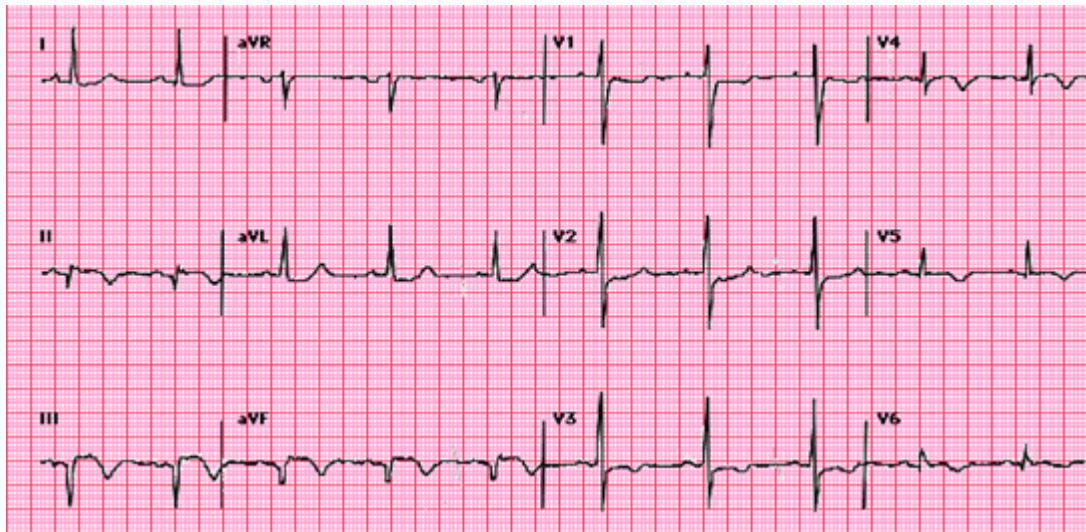
The electrocardiogram above contains findings consistent with a lateral myocardial infarction. Note the ST segment elevations in leads I, aVL, V5, and V6. Q waves are present in multiple leads.

ECG: electrocardiogram; MI: myocardial infarction.

Reproduced with permission from: Knuths LA, Keate JT, Morton PG. Acute coronary syndrome. In: Critical Care Nursing: A Holistic Approach, 12th ed, Morton PG, Thurman P (Eds), Lippincott Williams & Wilkins 2023. Copyright © 2023 Wolters Kluwer Health, Inc.

Graphic 129527 Version 2.0

ECG acute infero-postero-lateral myocardial infarction

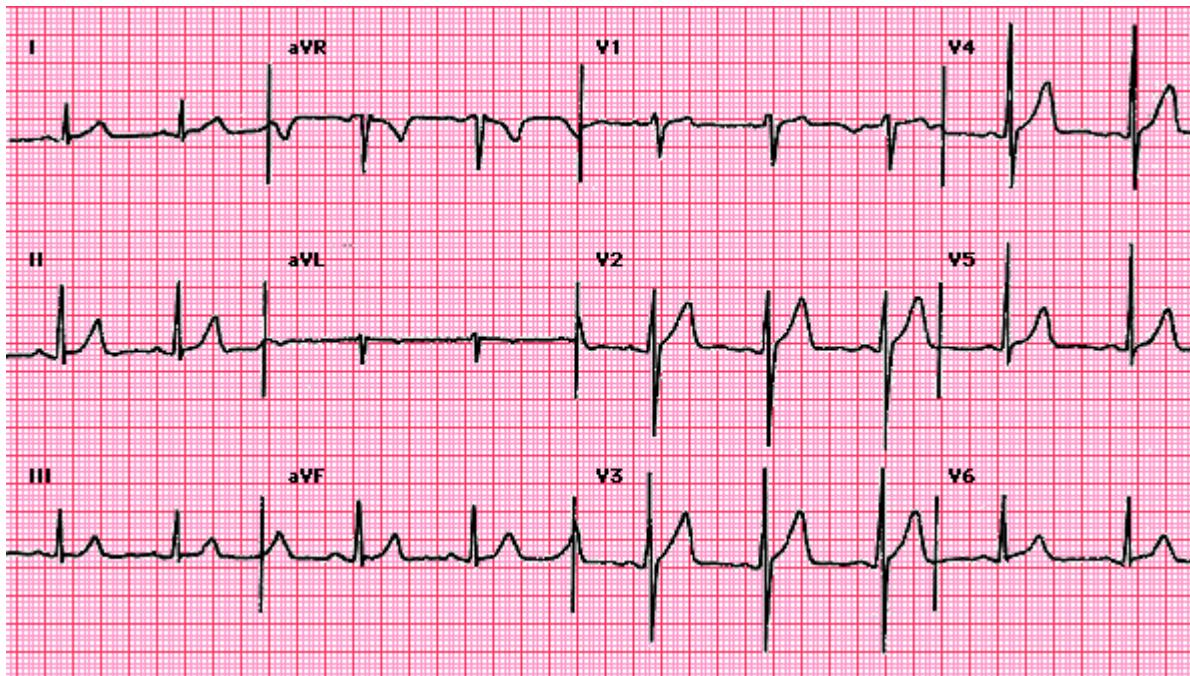


Electrocardiogram showing the major features of an inferior (Q waves, ST elevations, and T wave inversions in II, III, and aVF), posterior (tall R waves in V1 and V2), and lateral (T wave inversions in V4 to V6) myocardial infarction.

Courtesy of Ary Goldberger, MD.

Graphic 70036 Version 3.0

Normal ECG

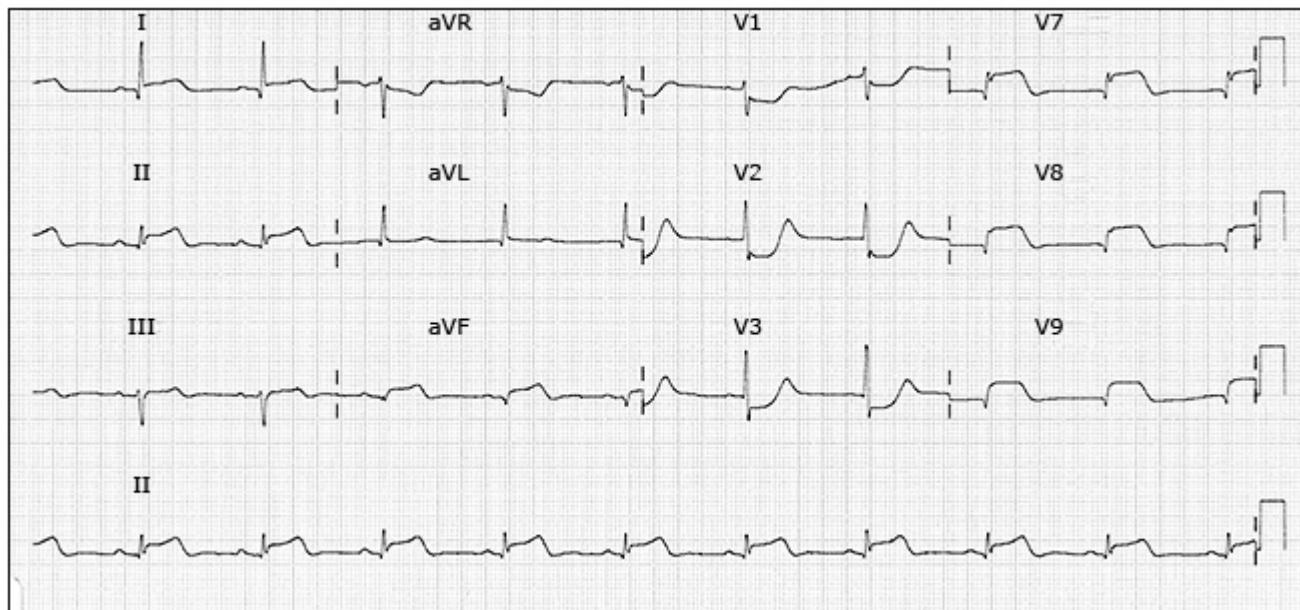


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

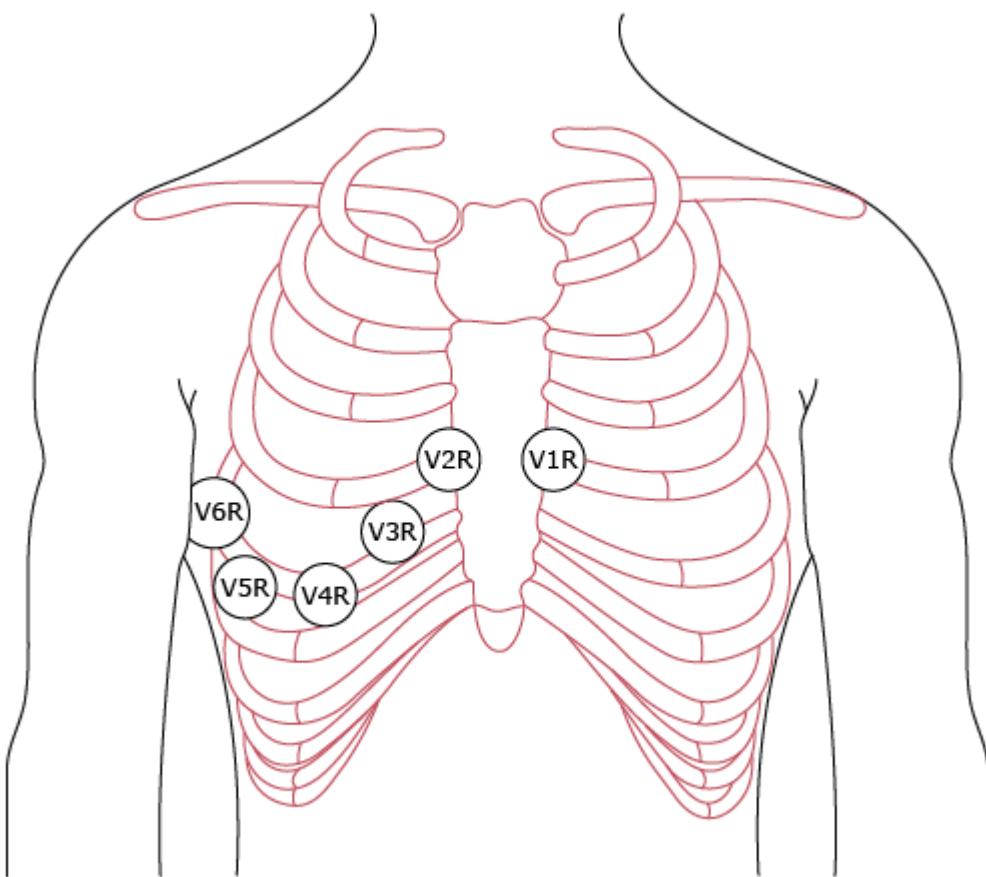
Posterior myocardial infarction leads V7 to V9



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

Right-sided ECG chest leads



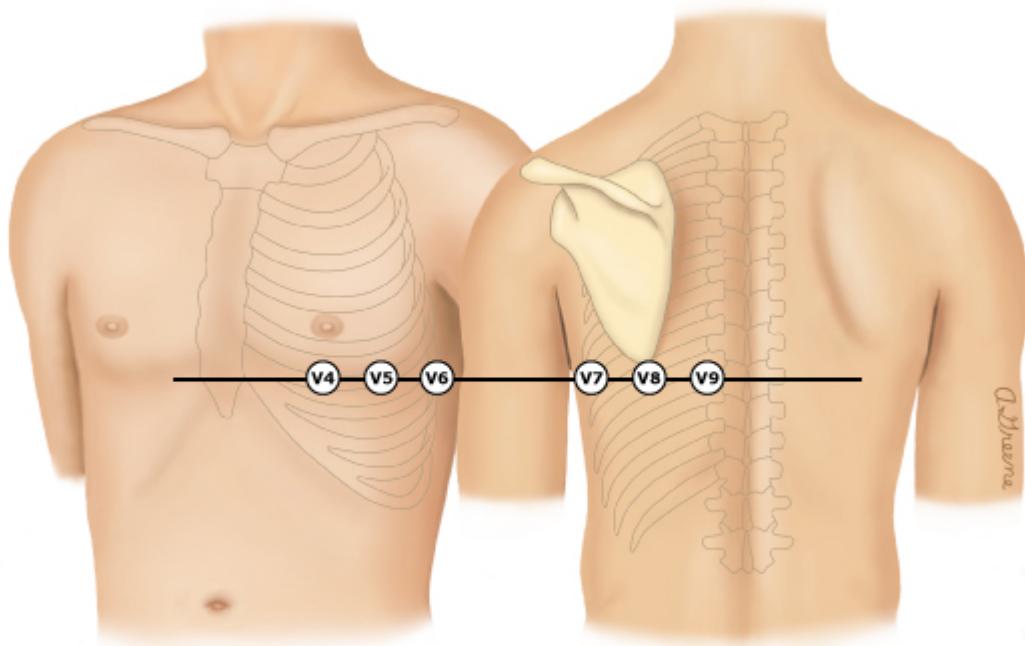
The image above shows placement of right-sided chest leads. These are used to improve detection of coronary ischemia of the right ventricle.

ECG: electrocardiogram.

Reproduced with permission from: Steyers CM III, Smith TW. Advanced electrocardiography: ECG 201. In: The Washington Manual Cardiology Subspecialty Consult, 4th ed, Sadhu J, Husaini M, Williams D (Eds), Lippincott Williams & Wilkins 2022. Copyright © 2022 Wolters Kluwer Health, Inc.

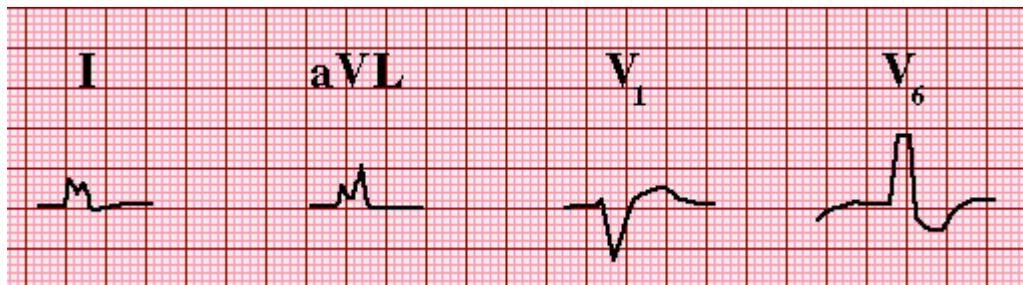
Graphic 129502 Version 3.0

Posterior leads V7 to V9



Graphic 94900 Version 1.0

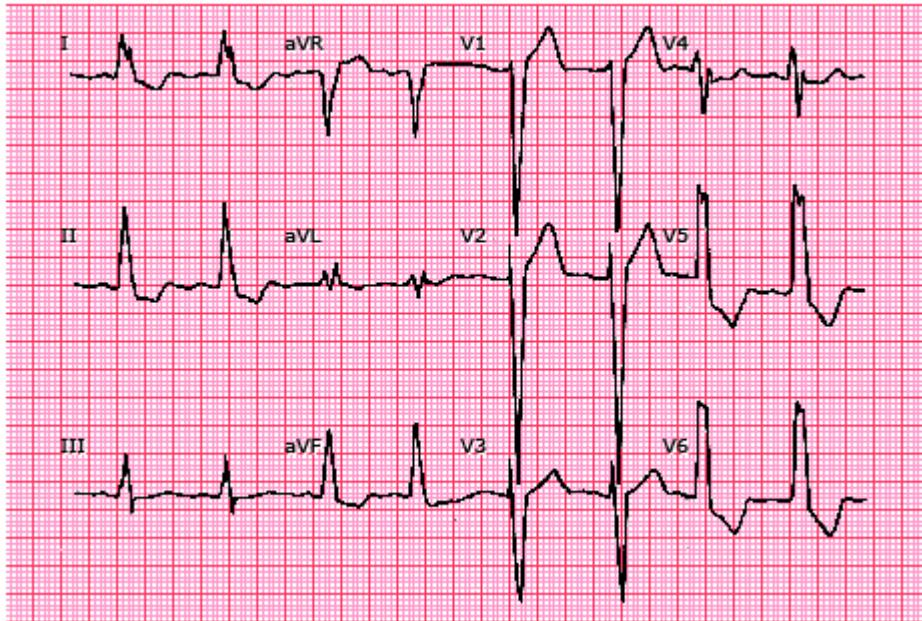
Complete left bundle branch block



Delayed and abnormal activation of the left ventricular myocardium, and a diffuse slowing of conduction throughout the left ventricle leads to the following changes on the ECG: there is a tall monophasic and broadened R wave in leads I, aVL, and V₆ instead of a septal Q wave; there is a QS complex which is abnormal and widened in V₁ instead of a small initial R wave due to septal activation; the QRS interval is prolonged >0.12 seconds; myocardial repolarization changes including T wave inversion and ST segment depression are evident.

Graphic 52836 Version 3.0

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

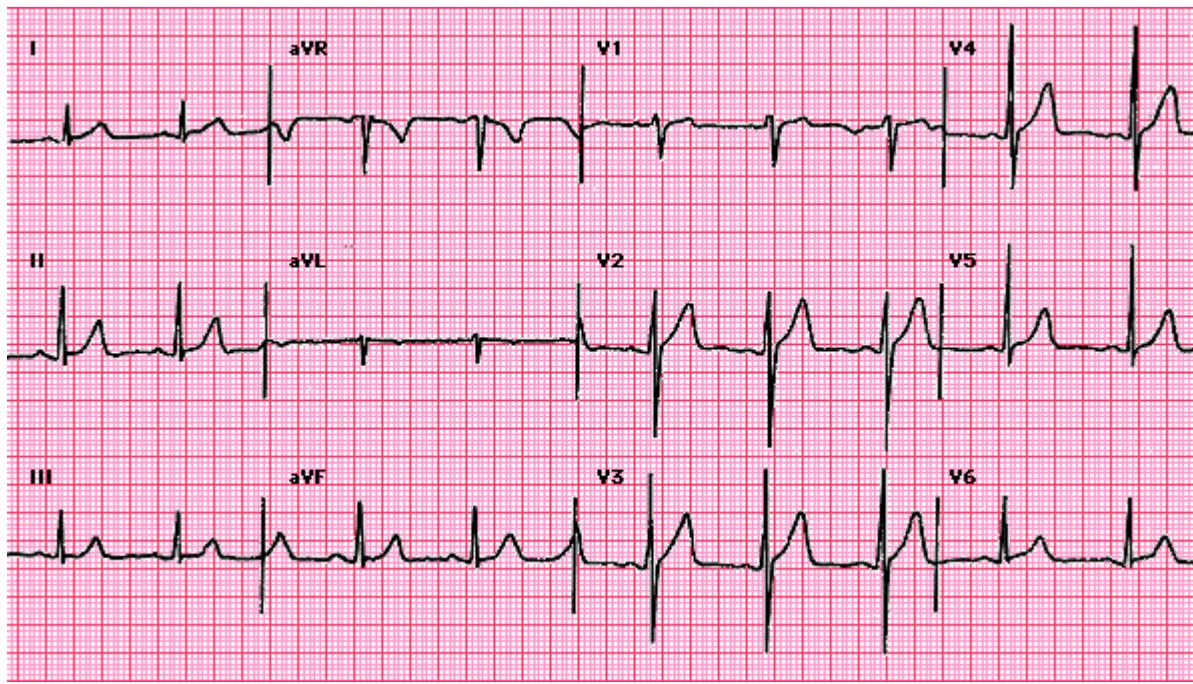


Electrocardiogram in typical complete left bundle branch block. The asynchronous activation of the 2 ventricles increases the QRS duration (0.16 seconds in this example). The abnormal initial vector results in loss of "normal" septal forces as manifested by absence of q waves in leads I, aVL, and V6. 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

Distinguishing among life-threatening causes of chest pain: History, examination, and diagnostic testing

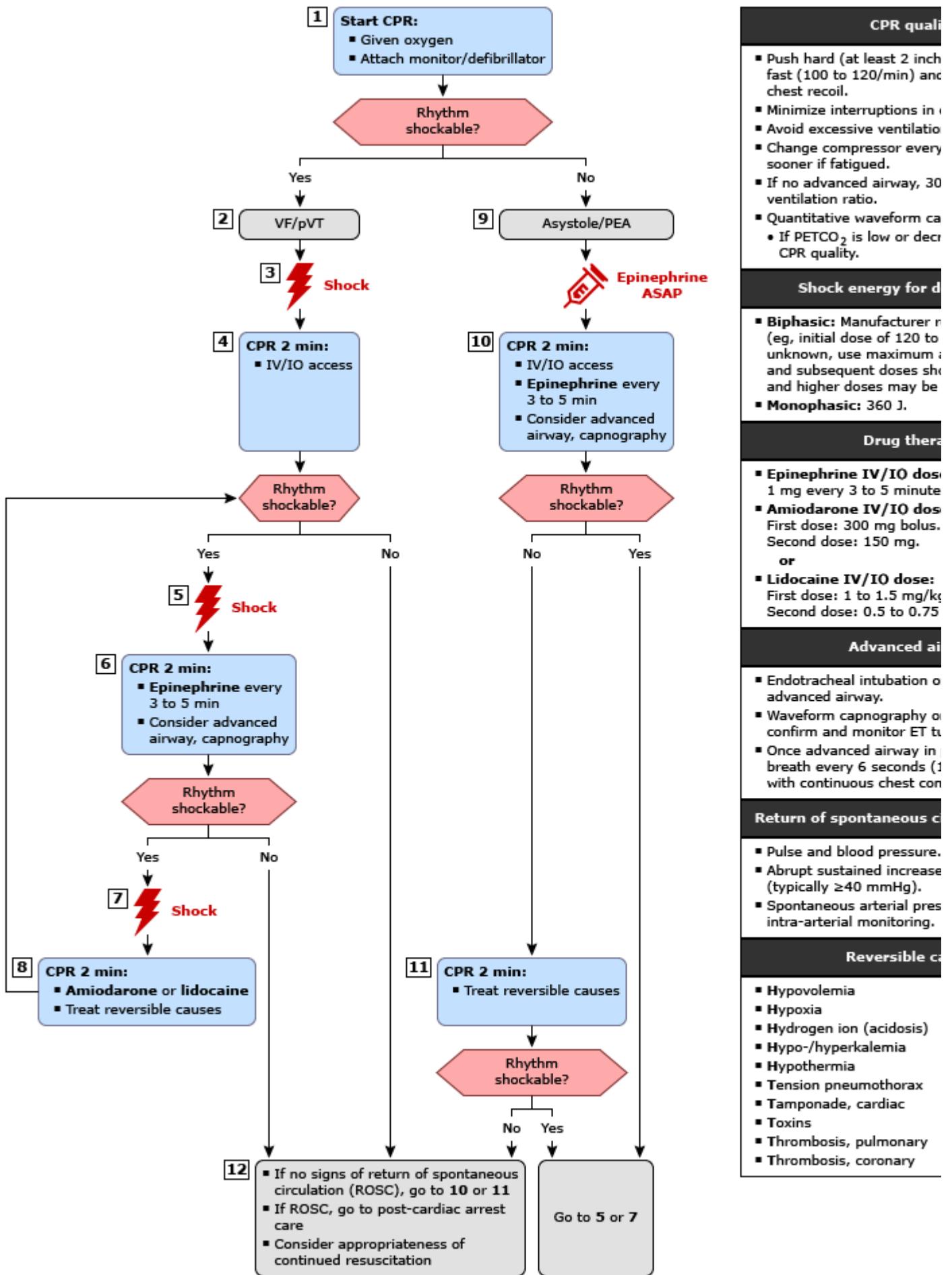
Diagnosis	Historical features	Examination findings	Electrocardiogram	Chest radiograph
Acute coronary syndrome	<ul style="list-style-type: none"> ▪ Substernal/left-sided chest pressure or tightness is common ▪ Onset is gradual ▪ Pain radiating to shoulders or pain with exertion increases relative risk ▪ "Atypical" symptoms (eg, dyspnea, weakness) more common in older adults, women, diabetics ▪ Older adults can present with dyspnea, weakness, syncope, or AMI alone 	<ul style="list-style-type: none"> ▪ Nonspecific ▪ May detect signs of HF 	<ul style="list-style-type: none"> ▪ ST segment elevations, Q waves, new left bundle branch block are evidence of AMI ▪ Single ECG is not sensitive for ACS ▪ Prominent R waves with ST segment depressions in V₁ and V₂ strongly suggests posterior AMI 	<ul style="list-style-type: none"> ▪ Nonspecific ▪ May show evidence of HF
Aortic dissection	<ul style="list-style-type: none"> ▪ Sudden onset of sharp, tearing, or ripping pain ▪ Maximal severity at onset ▪ Most often begins in chest, 	<ul style="list-style-type: none"> ▪ Absent upper extremity or carotid pulse is suggestive ▪ Discrepancy in systolic BP >20 mmHg between right and left upper 	<ul style="list-style-type: none"> ▪ Ischemic changes in 15% ▪ Nonspecific ST and T changes in 30% 	<ul style="list-style-type: none"> ▪ Wide mediastinum loss of normal aortic knob contour is common (up to 75%) ▪ 10% have normal findings

	<ul style="list-style-type: none"> ■ can begin in back ■ Can mimic stroke, ACS, mesenteric ischemia, kidney stone 	<ul style="list-style-type: none"> ■ extremity is suggestive ■ Up to 30% with neurologic findings ■ Findings vary with arteries affected 		
Pulmonary embolism	<ul style="list-style-type: none"> ■ Many possible presentations, including pleuritic pain and painless dyspnea ■ Often sudden onset ■ Dyspnea often dominant feature 	<ul style="list-style-type: none"> ■ No finding is sensitive or specific ■ Extremity exam generally normal ■ Lung exam generally nonspecific; focal wheezing may be present; tachypnea is common 	<ul style="list-style-type: none"> ■ Usually abnormal but nonspecific ■ Signs of right heart strain suggestive (eg, RAD, RBBB, RAE) 	<ul style="list-style-type: none"> ■ Great majority a normal ■ May show atelec elevated hemidiaphragm, pleural effusion
Tension pneumothorax	<ul style="list-style-type: none"> ■ Often sudden onset ■ Initial pain often sharp and pleuritic ■ Dyspnea often dominant feature 	<ul style="list-style-type: none"> ■ Ipsilateral diminished or absent breath sounds ■ Subcutaneous emphysema is uncommon 		<ul style="list-style-type: none"> ■ Demonstrates air in pleural space

Pericardial tamponade	<ul style="list-style-type: none"> ■ Pain from pericarditis is most often sharp anterior chest pain made worse by inspiration or lying supine and relieved by sitting forward ■ Dyspnea is common 	<ul style="list-style-type: none"> ■ Severe tamponade creates obstructive shock and causes jugular venous distension, pulsus paradoxus ■ Pericardial effusion can cause friction rub 	<ul style="list-style-type: none"> ■ Decreased voltage and electrical alternans can appear with significant effusions ■ Diffuse PR segment depressions and/or ST segment elevations can appear with acute pericarditis 	<ul style="list-style-type: none"> ■ May reveal enlarged heart
Mediastinitis (esophageal rupture)	<ul style="list-style-type: none"> ■ Forceful vomiting often precedes esophageal rupture ■ Recent upper endoscopy or instrumentation increases risk of perforation ■ Odontogenic infection is possible cause ■ Coexistent respiratory and gastrointestinal complaints may occur 	<ul style="list-style-type: none"> ■ Ill-appearing; shock; fever ■ May hear (Hamman's) crunch over mediastinum 		<ul style="list-style-type: none"> ■ Large majority have some abnormalities pneumomediastinum, pleural effusion, pneumothorax

ΔMS: altered mental status; ACS: acute coronary syndrome; AMI: acute myocardial infarction; BP: blood pressure; CABG: coronary artery bypass graft; CK-MB: creatine kinase-MB; CXR: chest radiograph; ECG: electrocardiogram; HF: heart failure; PCI: percutaneous coronary intervention; PE: pulmonary embolism; RAD: right axis deviation; RAE: right atrial enlargement; RBBB: right bundle branch block.

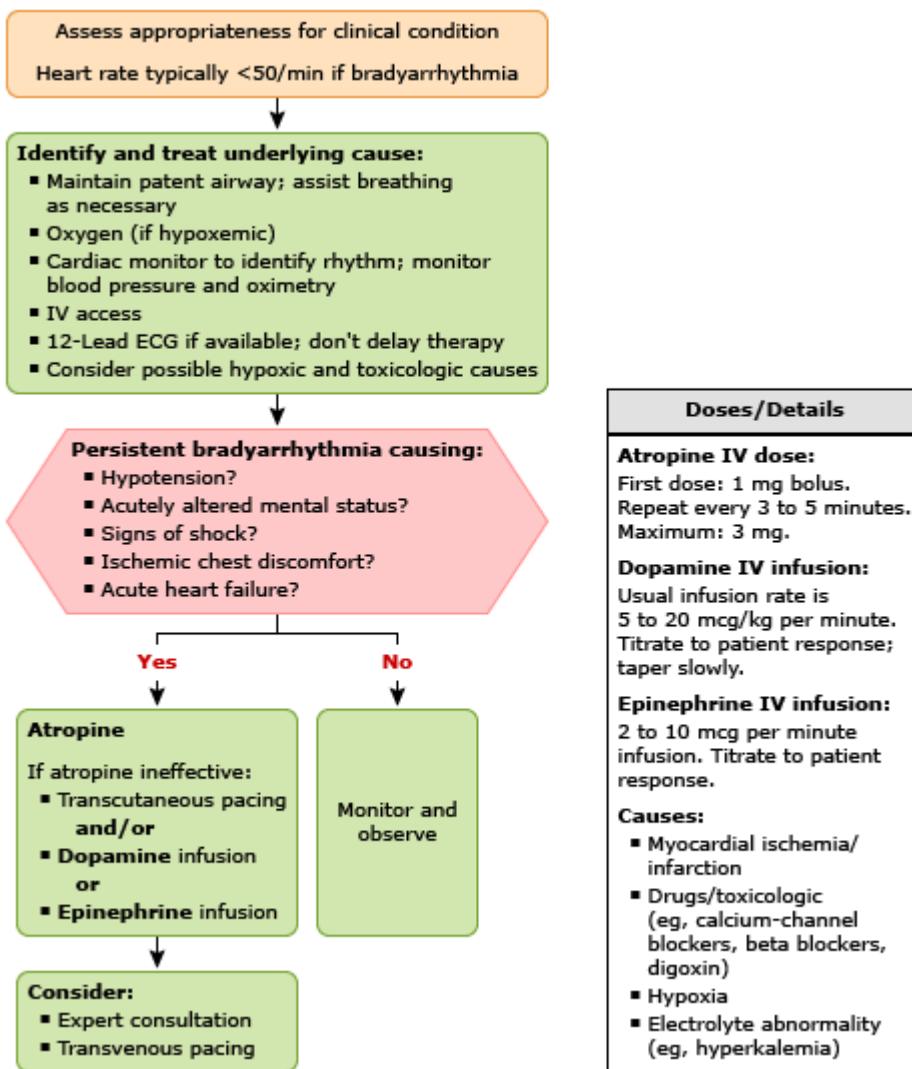
Adult cardiac arrest algorithm



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Graphic 129983 Version 10.0

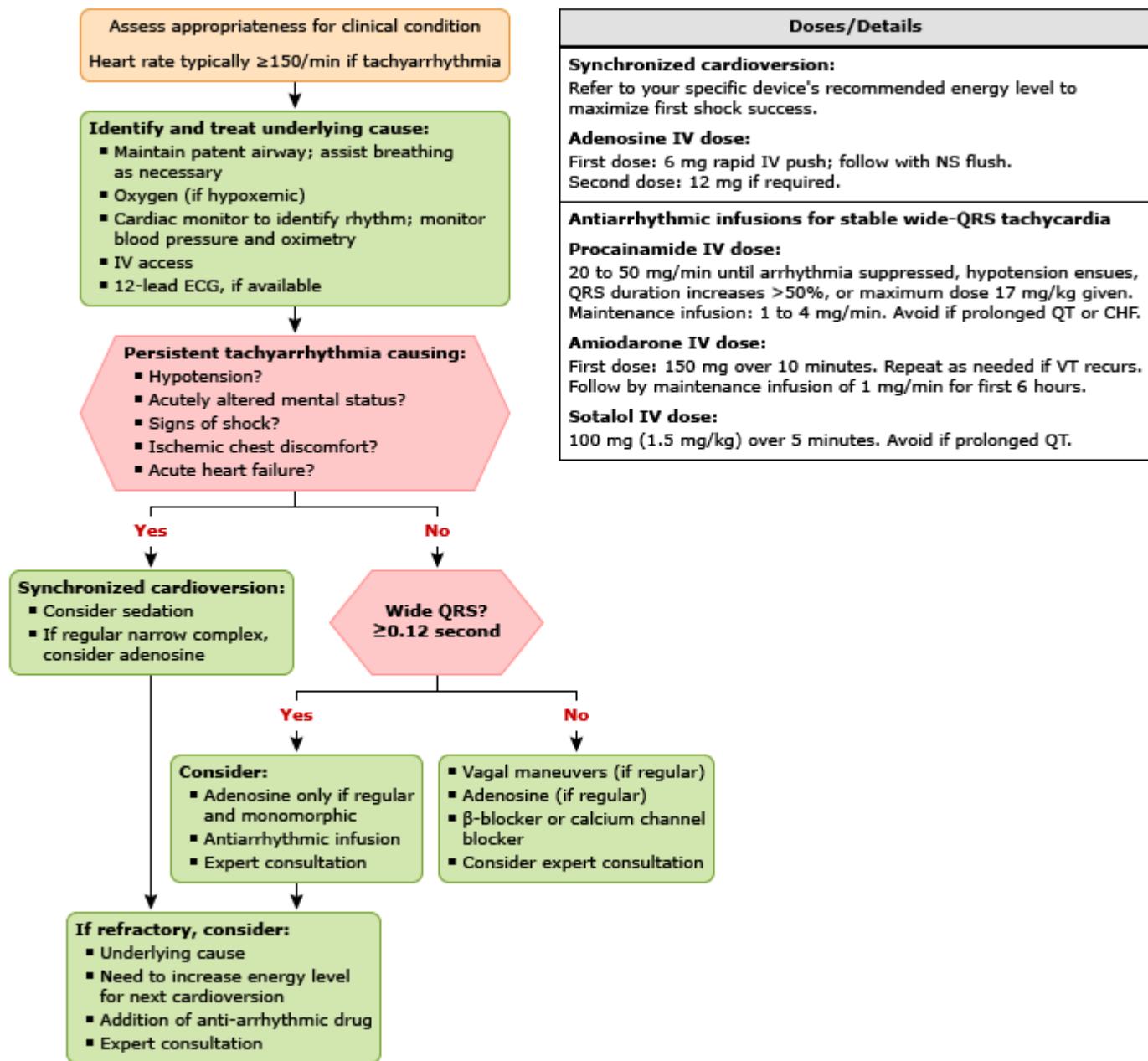
Adult bradycardia algorithm 2020 update



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Graphic 130748 Version 11.0

Adult tachycardia with a pulse algorithm 2020 update



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Graphic 130747 Version 11.0

Causes of cardiogenic shock

Acute myocardial infarction

Pump failure

Large infarction, generally involving ≥40 percent of the left ventricle

Smaller infarction with preexisting left ventricular dysfunction or a prior infarction

Infarction extension or expansion

Reinfarction

Mechanical complications

Acute mitral regurgitation caused by rupture of a papillary muscle or chordae tendinae or severe papillary muscle dysfunction

Ventricular septal defect caused by rupture of the interventricular septum

Left ventricular free wall rupture

Pericardial tamponade due to rupture of the left ventricular free wall or hemorrhagic pericardial effusion

Right ventricular infarction

Other conditions

End-stage cardiomyopathy

Myocarditis

Septic shock with severe myocardial depression

Left ventricular outflow tract obstruction

Aortic stenosis

Hypertrophic obstructive cardiomyopathy

Obstruction to left ventricular filling

Mitral stenosis

Left atrial myxoma

Toxicity causing severe myocardial depression (eg, beta blocker, calcium channel blocker, or clonidine overdose)

Dysrhythmia

Acute mitral regurgitation (chordal rupture)

Acute aortic insufficiency (eg, aortic dissection)

Myocardial contusion

Prolonged cardiopulmonary bypass

Adapted from Hollenberg SM, Kavinsky CJ, Parrillo JE. Ann Intern Med 1999; 131:47.

Graphic 69673 Version 3.0

Killip classification of acute myocardial infarction

Class I	No evidence of heart failure
Class II	Findings consistent with mild to moderate heart failure (eg, S3 gallop, lung rales less than one-half way up the posterior lung fields, or jugular venous distension)
Class III	Overt pulmonary edema
Class IV	Cardiogenic shock

Graphic 65592 Version 7.0

Composition of the HEART score for chest pain patients in the emergency room

HEART score for chest pain patients		Score
History	Highly suspicious	2
	Moderately suspicious	1
	Slightly suspicious	0
ECG	Significant ST depression	2
	Nonspecific repolarisation disturbance	1
	Normal	0
Age	≥65 years	2
	45-65 years	1
	<45 years	0
Risk factors	≥3 risk factors or history of atherosclerotic disease	2
	1 or 2 risk factors	1
	No risk factors known	0
Troponin	>2x normal limit	2
	1-2x normal limit	1
	≤normal limit	0
		Total _____

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Graphic 105869 Version 2.0

