Management of suspected acute coronary syndrome in the emergency department

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 the chest discomfort and promptly initiate appropriate therapy. It is essential that initial assessment and management be rapid but methodical and evidence-based [1-5].

Diagnostic evaluation emphasizes distinguishing among the following potential causes of chest pain:

- Acute coronary syndrome (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 ischemia depends upon the characteristics of the chest pain, specific associated symptoms, abnormalities on electrocardiogram (ECG), and levels of serum markers of cardiac injury. A patient with a possible acute coronary syndrome (ACS) should be 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 topics are found separately:

- (See "Evaluation of chest pain in the emergency department").
- (See "Evaluation of the adult with dyspnea in the emergency department").
- (See "Overview of the acute management of acute ST elevation myocardial infarction").
- (See "Overview of the acute management of unstable angina and acute non-ST elevation myocardial infarction").
- (See "Evaluation of patients with chest pain at low or intermediate risk for acute coronary syndrome").

GENERAL PRINCIPLES — A 2004 task force of the American College of Cardiology (ACC) and the American Heart Association (AHA) recommended that all hospitals establish multidisciplinary teams to develop guideline-based, institution-specific written protocols for triaging and managing patients who present with symptoms suggestive of myocardial ischemia [6]. The task force gave a class I recommendation to having the choice of initial treatment made by the emergency medicine clinician based upon the institutional protocol [6]. It was recommended that immediate cardiology consultation should be available for cases in which the initial diagnosis and treatment plan are unclear or are not covered directly by the protocol. Efforts at establishing systems of care for ST elevation myocardial infarction are underway [7].

IMMEDIATE ED INTERVENTIONS — In the emergency department (ED), patients presenting with chest pain 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 chest pain in the emergency department" and "Evaluation of the adult with dyspnea in the emergency department").

If acute coronary syndrome (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).

Caution should be employed in evaluating possible ACS in women, diabetics, and the elderly, who are more likely to present with "atypical" symptoms even in the presence of acute coronary ischemia [8]. (See 'Atypical symptoms' 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 ischemia is ideally 10 minutes from presentation [6]. Data from a national registry have shown that ECG acquisition is frequently delayed, and that women are significantly less likely to have ECGs performed within the recommended 10 minutes [9].

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 ECG interpreted
Resuscitation equipment brought to the bedside
Cardiac monitor attached to patient
Oxygen given
IV access and blood work obtained
Aspirin 162 to 325 mg given
Nitrates and morphine given (unless contraindicated)

- **Initial history and exam** — Obtain a brief history and perform a focused physical examination [6]. Important elements of the history include confirmation of the presenting symptoms, characteristics of the pain 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 thrombolyis is entertained as a potential therapy. (See ‘Physical examination’ below.)

The history should also address potentially emergent noncardiac causes of chest pain, such as acute aortic dissection, pulmonary embolism, tension pneumothorax, perforating peptic ulcer, and esophageal rupture (table 2A-B) [6]. (See “Evaluation of chest pain in the emergency department”.)

- **Twelve-lead ECG** — A 12-lead ECG should be obtained in all patients with possible coronary ischemia. (See ‘ECG Assessment’ below.)

The 12-lead ECG provides the basis for initial diagnosis and management and should immediately be shown to an emergency physician for interpretation.

The initial ECG is often NOT diagnostic in patients with ACS. The ECG should be repeated at 5 to 10 minute intervals if the initial study is not diagnostic but the patient remains symptomatic and high clinical suspicion for ACS persists [6].

- **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 initiated to maintain oxygen saturation above 90 percent.

- **Intravenous access** — Intravenous access should be established, with blood drawn for initial laboratory work, including cardiac biomarkers, electrolytes, indices of coagulation and renal function, and serum lipid profile [6]. (See ‘Cardiac biomarkers’; below.)

- **Aspirin** — All patients with suspected ACS should be given aspirin in a dose of 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 [6]. (See “Benefits and risks of aspirin in secondary and primary prevention of cardiovascular disease”.)

- **Sublingual nitrates** — In most cases, 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 intravenous nitroglycerin [6].

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 heart disease”, section on “Treatment of sexual dysfunction”.)

**Extreme care should also be taken before giving nitrates in the setting of an inferior myocardial infarction with possible involvement of the right ventricle (figure 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 [10]. 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) [11]. (See "Evaluation of chest pain in the emergency department").

- **Morphine** — Intravenous morphine sulfate at an initial dose of 2 to 4 mg, with increments of 2 to 8 mg, repeated at 5 to 15 minute intervals, should be given for the relief of chest pain and anxiety [6]. Morphine can reduce sympathetic stimulation caused by pain and anxiety, thereby decreasing cardiac workload and risks associated with excess catecholamines. (See "Overview of the acute management of acute ST elevation myocardial infarction").

**ECG ASSESSMENT** — Specific approaches to patients judged to have definite or probable acute coronary syndrome (ACS) based upon a targeted history and physical examination are initially guided by the accompanying 12-lead electrocardiogram (ECG). (See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction").

The initial ECG 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 until the patient’s chest pain resolves or a definitive diagnosis is made.

**Four major ischemic syndromes** — ECG abnormalities are an early sign of myocardial ischemia and can be identified within 90 minutes of symptom onset [10]. There are four major types of cardiac ischemic syndromes that can be identified on ECG [1,12]:

- **ST elevation (Q wave) MI**, manifested in most patients by Q waves that are usually preceded by hyperacute T waves and
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ST elevations, and followed by T wave inversions. Clinically significant ST segment elevation is considered to be present if it is greater than 1 mm (0.1 mV) in at least two anatomically contiguous leads or 2 mm (0.2 mV) in two contiguous precordial leads.

- Non-ST elevation (non-Q wave) MI, manifested by ST depressions or T wave inversions without Q waves. Isolated T wave inversions correlate with increased risk for MI during initial presentation in the ED [13,14], and may represent Wells’ syndrome.

- Noninfarction subendocardial ischemia (classic angina), manifested by transient ST segment depressions.

- Noninfarction transmural ischemia (Prinzmetal’s variant angina), manifested by transient ST segment elevations or paradoxical T wave normalization.

Localization of ischemia — The ECG leads are more helpful in localizing regions of transtumal than subendocardial ischemia [1,12]. 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 — One or more of the precordial leads (V1-V6) (figure 2A-B)
- Anteroseptal ischemia — Leads V1 to V3
- Apical or lateral ischemia — Leads aVL and I, and leads V4 to V6
- Inferior wall ischemia — Leads II, III, and aVF (figure 1)
- Right ventricular ischemia — Right-sided precordial leads
- Posterior wall ischemia — Posterior precordial leads

The right-sided leads V4R, V5R, and V6R should be obtained if there is evidence of inferior wall ischemia, demonstrated by ST elevation in leads II, III, and aVF. The recording of right-sided leads in this setting was given a class I recommendation by the 2004 ACC/AHA task force [6].

The posterior leads V7, V8, and V9 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 ECGs — 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 20 to 30 minutes [6]. 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 acute ST elevation myocardial infarction" and "Overview of the acute management of unstable angina and acute non-ST elevation myocardial infarction".)

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 MI [8,15]. 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 [16].

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 [17,18].

LBBB or pacemaker — Both left bundle branch block (LBBB), which is present in approximately 7 percent of patients with an acute MI, and pacing can interfere with the electrocardiographic diagnosis of coronary ischemia. Another problem is that approximately one-half of patients with LBBB and an acute MI do not have chest pain as a symptom of their ischemia [19]. As a result, patients with LBBB are much less likely to receive aspirin, beta blockers, and reperfusion therapy [20], particularly if they present without chest pain [19]. Similar observations have been made in patients with a paced rhythm [21].

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 enzymes 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 "Pathophysiology and clinical presentation of ischemic chest pain").

Of note, older patients, diabetics, and women 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 symptoms" below.)

Ischemic chest pain — Ischemic pain has a number of features that tend to distinguish it from noncardiac pain [5,6]. These characteristics are described below using the OPQRST mnemonic. Symptoms associated with the highest relative risk of myocardial infarction (MI) include radiation to an upper extremity, particularly when there is radiation to both arms, and pain associated with diaphoresis or with nausea and vomiting [22,23]. An important question is whether current pain is reminiscent of prior MI.

- 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, which increases cardiac oxygen demand [22,24]. 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, "GI cocktail" of viscous lidocaine and antacid) does NOT reliably distinguish nonischemic from ischemic chest pain [25,26].
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- **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, constricting, crushing, strangling, burning, heart burn, 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 pins and needles-like.

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.

- **Site** — Ischemic pain is not felt in one specific spot, but 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 acute coronary syndrome (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.

### Historic features increasing likelihood of ACS

**Historical features increasing likelihood of ACS** — Historical features that increase the likelihood of ACS include the following (table 5):

- 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, hyperlipidemia, and cigarette smoking. (See "Overview of the risk factors for cardiovascular disease").
- Recent cocaine use.

**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 chest pain in the emergency department").

In two systematic reviews, the following characteristics were found to be more typical of nonischemic chest discomfort:

- 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. (See "Atypical symptoms" 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 6). It is essential to consider these alternate diagnoses to avoid potentially dangerous errors in management, such as the administration of thrombolytic therapy to a patient with an aortic dissection.

**ATYPICAL SYMPTOMS** — Some patients with acute coronary syndrome (ACS) present with atypical symptoms rather than chest pain. In a review of over 430,000 patients with confirmed acute myocardial infarction (MI) from the National Registry of Myocardial Infarction II, one-third had no chest pain on presentation to the hospital. These patients often present with symptoms such as dyspnea alone, weakness, nausea and/or vomiting, palpitations, syncope, or cardiac arrest. They are more likely to be older, diabetic, and women. (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 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 MI 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). Not surprisingly, these differences were associated with an increase in inhospital mortality (23.3 versus 9.3 percent, adjusted odds ratio 2.21, 95 percent CI 2.17-2.26).

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

- Inspection: Look for signs of anemia, fever, cyanosis, clubbing, peripheral edema, etc.
- Auscultation: Listen for heart sounds, murmur, pericardial friction rub, etc.
- Palpation: Feel for pulses, check for bruises, etc.
- Neurological examination: Assess for neurological deficits, etc.
- Examination of the skin: Look for rashes, lesions, etc.
- Examination of the abdomen: Look for tenderness, distention, masses, etc.
- Examination of the extremities: Look for pulses, swelling, etc.

I hope this helps!
- **Responsiveness, airway, breathing and circulation** — In patients in respiratory or cardiorespiratory arrest, the appropriate resuscitation algorithms should be followed (algorithm 2). (See "Advanced cardiac life support (ACLS) in adults").

- **Evidence of systemic hypoperfusion** (hypotension; tachycardia; impaired cognition; cool, clammy, pale, ashen skin) — Cardiogenic shock complicating acute MI requires aggressive evaluation and management. (See "Clinical manifestations and diagnosis of cardiogenic shock" 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 MR murmur) — Aggressive management is needed for patients with heart failure complicating an acute MI, depending upon the severity of the heart failure and the presence of other risk factors (table 2) [34].

- 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 "Fibrinolytic (thrombolytic) agents in acute ST elevation myocardial infarction: Therapeutic use").

**CARDIAC BIOMARKERS** — Serial serum biomarkers (sometimes referred to as cardiac enzymes) of acute myocardial damage, such as troponin T and I, are essential for confirming the diagnosis of infarction. They should be obtained in any patient at significant risk of ACS. The use of biomarkers is discussed separately. (See "Troponins and creatine kinase as biomarkers of cardiac injury" and "Elevated cardiac troponin concentration in the absence of an acute coronary syndrome").

**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 'ECG Assessment' above and "Overview of the acute management of acute 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 "Selecting a reperfusion strategy for acute STE elevation myocardial infarction" and "Primary percutaneous coronary intervention in acute ST elevation myocardial infarction: Determinants of outcome" and "Fibrinolytic (thrombolytic) agents in acute ST elevation myocardial infarction: Therapeutic use").

Rapid implementation of primary PCI is more likely to be achieved if the hospital protocol involves immediate activation of the PCI team by the emergency clinician and immediate transfer of the patient to the catheterization laboratory [35].

**Non-ST elevation**

**Diagnosis and treatment** — Patients whose presentation raises concern for coronary ischemia but who do not manifest ST elevations on ECG are considered to have unstable angina (UA) or a non-ST elevation myocardial infarction (NSTEMI). The management of patients with UA or NSTEMI is discussed separately. The accompanying table provides a concise summary of the immediate treatment interventions needed in these patients (table 3). (See 'ECG Assessment' above and "Overview of the acute management of unstable angina and acute non-ST elevation myocardial infarction").

UA 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 UA by the presence of elevated serum biomarkers. ST segment elevations and Q waves are absent in both UA and NSTEMI. As a result, UA and NSTEMI are frequently indistinguishable at initial evaluation since an elevation in serum biomarkers is usually not detectable for four to six hours after an MI, and at least 12 hours are required to detect elevations in all patients. (See 'Cardiac biomarkers' above and "Classification of unstable angina and non-ST elevation myocardial infarction").

**Thrombolytic therapy should NOT be administered to patients with UA or NSTEMI unless subsequent ECG monitoring documents ST segment elevations that persist** [36]. (See "Overview of the acute management of unstable angina and acute non-ST elevation myocardial infarction").

An aggressive approach to reperfusion using PCI is best suited for patients with an elevated troponin level or a TIMI risk score ≥5 or possibly other high-risk features. (See 'High-risk patient' below.) For patients at lower risk, approaches vary based upon hospital protocol.

**Risk stratification** — Both patient outcomes and the effectiveness of specific treatments for patients with UA and NSTEMI vary according to the presence or absence of high-risk features in their presentation. This fact underscores the importance of accurate risk stratification in the emergency department. Risk stratification is discussed in detail separately. (See "Risk stratification after unstable angina or non-ST elevation myocardial infarction", section on 'Immediate high-risk patients' and "Risk stratification after unstable angina or non-ST elevation myocardial infarction", section on 'Early risk stratification").

**Cardiac arrhythmias during ACS** — Disturbances of cardiac rhythm during an MI are usually detected by cardiac monitor rather than by physical examination or 12-lead ECG (algorithm 2):

- 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 "Clinical features and treatment of ventricular arrhythmias during acute myocardial infarction").
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 3). (See "Supraventricular arrhythmias after myocardial infarction").

Bradyarrhythmias occurring early in the setting of an inferior wall MI (within the first 24 hours) may respond to treatment with atropine (algorithm 4). Later bradyarrhythmias, wide QRS-complex bradyarrhythmias, and those occurring in the setting of an anterior wall MI 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. The utility of epidemiologic risk factors in determining acute, individual risk has been questioned [37].

High-risk patient — The patient has a high-risk ACS if ST segment depression (≥0.05 mV [0.5 mm]) is present in two or more contiguous leads, elevated serum biomarkers, and/or the TIMI risk score is ≥2. 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 referred for early elective angiography and revascularization if appropriate.

If there is no ST segment elevation or depression or new LBBB, 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 for further evaluation. Those patients manifesting high-risk features either on presentation or during their emergency department course should be admitted and considered for early PCI. (See "Risk stratification after unstable angina or non-ST elevation myocardial infarction", section on 'Immediate high-risk patients' and "Risk stratification after unstable angina or non-ST elevation myocardial infarction", section on 'Early risk stratification'.)

Low and moderate risk patient — The management, including methods for risk assessment, of patients who have no ECG changes and normal serum biomarkers, but are still considered to be at low or moderate risk for ACS is discussed separately. (See "Evaluation of patients with chest pain at low or intermediate risk for acute coronary syndrome").

IMPACT OF MISSED DIAGNOSIS — In different studies, 1.9 to 4 percent of patients with an ACS are mistakenly discharged from the emergency department [38-40]; these patients have an increase in short-term mortality [38,39]. This issue was best evaluated in a review of 10,689 patients who presented to the emergency department with symptoms suggesting acute coronary ischemia: 8 percent had an acute MI and 9 percent had unstable angina [38]. 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 MI had the following characteristics:

- Women less than 55 years of age
- Nonwhite
- 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 MI and unstable angina, respectively).

A similar frequency of missed diagnosis was noted in a second report in which 1.9 percent of 1050 patients with an acute MI were mistakenly discharged from the emergency department [40]. Among these 20 patients, five had missed ST elevation and seven were discharged with a diagnosis of ACS.

OBSERVATION — Some patients without clear evidence of acute coronary syndrome (ACS) by clinical history, ECG, or biomarker measurement ultimately sustain a myocardial infarction (MI) 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 patients with chest pain at low or intermediate risk for acute coronary syndrome").

REST AND STRESS IMAGING TESTS — 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 acute coronary syndrome (ACS), a nondiagnostic 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 "Noninvasive imaging and stress testing in patients with suspected acute coronary syndrome").

In addition to detecting myocardial dysfunction or ischemia consistent with the diagnosis of an MI, imaging studies such as a contrast-enhanced chest CT scan, or cardiovascular magnetic resonance imaging can be used to differentiate an MI from an aortic dissection in patients for whom this distinction is initially unclear. (See "Clinical manifestations and diagnosis of aortic dissection", section on 'Imaging'.)

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REFERENCES


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

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# Characteristics of major noncardiac causes of chest pain

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

Excludes pain above the neck or below the umbilicus.
Algorithm for management of patients with suspected acute myocardial infarction in the emergency department

**Initial laboratory work should include:** serum cardiac biomarkers (cTnI or cTnT preferred), CBC with platelet count, PT and INR, aPTT, electrolytes, magnesium, BUN, creatinine, blood glucose, and serum lipid profile.
Initial assessment:

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

Initial interventions:

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

Acute management STEMI:

- Select reperfusion strategy: Primary percutaneous coronary intervention (PCI) strongly preferred, especially for patients with cardiogenic shock, heart failure, late presentation, or contraindications to fibrinolysis. Activate cardiac catheterization team as indicated. For patients with symptoms of >12 hours, fibrinolytic therapy is not indicated, but emergent PCI may be considered, particularly for patients with evidence of ongoing ischemia or those at high risk.
- Treat with fibrinolysis if PCI unavailable within 90-120 minutes, symptoms <12 hours, and no contraindications.
- Give antiplatelet therapy (in addition to aspirin) to all patients:
  
  1. Prasugrel: For patients undergoing primary PCI and NOT at high risk of bleeding (age <75 years, weight >60 kilograms, no stroke or TIA), give prasugrel 60 mg.
  
  2. Clopidogrel: For patients undergoing primary PCI who are not candidates for prasugrel due to a high risk of bleeding, give clopidogrel 600 mg. (Some patients, such as those with recent GI bleed or head trauma, may not be candidates for either). For patients undergoing fibrinolysis or no reperfusion therapy, give clopidogrel loading dose of 300 mg if age less than 75 years; if age 75 years or older, give 75 mg daily dose only.
- Give glycoprotein IIb/IIIa inhibitor (GPIIb/IIIa): IF PCI is planned give GPIIb/IIIa in consultation with cardiology. Patients who receive bivalirudin should not receive GPIIb/IIIa.
- Give anticoagulant therapy to all patients:
  
  1. Unfractionated heparin (UFH): For patients undergoing primary PCI who are receiving a GP IIb/IIIa inhibitor, we suggest an IV bolus of 50 to 70 units/kg (target activated clotting time >200 seconds) up to a maximum of 4000 units. For those patients not receiving a GP IIb/IIIa inhibitor, we suggest an intravenous bolus of 60 to 100 units/kg up to a maximum of 4000 units (give after fibrinolytic therapy).
  
  2. Bivalirudin is an acceptable alternative to heparin plus GP IIb/IIIa in patients undergoing primary PCI. Initial bolus of 0.75 mg/kg IV followed by IV infusion of 1.75 mg/kg per hour; can be discontinued after PCI.
  
  3. Enoxaparin: For patients not managed with PCI and <75 years and whose serum creatinine is <2.5 mg/dL [220 micromol/L] in men and <2.0 mg/dL [175 micromol/L] in women, give a loading dose of 1.5 mg IV bolus and 1 mg/kg subcutaneously every 12 hours. The clearance requirement should be used to determine the timing of subsequent doses. UFH is preferred in patients with dialysis dependent renal failure.

Acute management of unstable angina or non-STEMI:

- Give antiplatelet therapy (in addition to aspirin):
  
  1. Thienopyridine: If the patient is undergoing same day PCI, give clopidogrel 600 mg; for patients undergoing early PCI in whom thienopyridine is withheld before PCI AND who are not at high risk of bleeding (age <75 years, weight >60 kilograms, no history of stroke or TIA) give prasugrel 60 mg instead of clopidogrel.
  
  2. GP IIb/IIIa inhibitor (either eptifibatide or tirofiban) may be added to or in place of clopidogrel in some circumstances (eg, high risk patient). Abciximab is contraindicated in patients not referred for cardiac catheterization.
- Give anticoagulant therapy in all patients:
  
  1. UFH is preferred, particularly for emergent PCI; give an IV bolus of 50 to 60 units/kg followed by 12 units/kg per hour IV (goal aPTT time of 1.5 to 2 times control or approximately 50 to 75 seconds).
2. Enoxaparin is an alternative to UFH for patients not undergoing early PCI: Give as described in the STEMI section above, using the regimen for patients with STEMI who receive fibrinolytics.

3. Fondaparinux is an alternative to enoxaparin in patients with NSTEMI at increased risk of bleeding being managed without PCI or fibrinolysis. The dose is 2.5 mg subcutaneously.

4. Bivalirudin is an acceptable alternative to UFH in patients going for PCI. It is given as an IV bolus of 0.1 mg/kg and an infusion of 0.25 mg/kg per hour before angiography. If used, bivalirudin is combined with oral clopidogrel 600 mg.

**Other considerations:**

- Cocaine-related ACS: Give benzodiazepines (eg, Lorazepam 2 to 4 mg IV every 15 minutes or so) as needed to alleviate symptoms; do **NOT** give beta blockers.
- Stop NSAID therapy if possible.
- Correct any electrolyte abnormalities, especially hypokalemia and hypomagnesemia.
### 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 (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

Electrocardiogram 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 (arrows). The ST depressions in leads I and aVL represent reciprocal changes.

*Courtesy of Ary Goldberger, MD.*
Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/min, a PR interval of 0.14 sec, a QRS interval of 0.10 sec, and a QRS axis of approximately 75°. Courtesy of Ary Goldberger, MD.
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.
Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/min, a PR interval of 0.14 sec, a QRS interval of 0.10 sec, and a QRS axis of approximately 75°. *Courtesy of Ary Goldberger, MD.*
Late evolution of anterior MI

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.
Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/min, a PR interval of 0.14 sec, a QRS interval of 0.10 sec, and a QRS axis of approximately 75°. Courtesy of Ary Goldberger, MD.
Clinical features that increase the probability of myocardial infarction in patients presenting with new chest pain

<table>
<thead>
<tr>
<th>Clinical feature</th>
<th>Likelihood ratio (95 percent CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain in chest or left arm</td>
<td>2.7*</td>
</tr>
<tr>
<td>Chest pain radiation</td>
<td></td>
</tr>
<tr>
<td>Right shoulder</td>
<td>2.9 (1.4-6.0)</td>
</tr>
<tr>
<td>Left arm</td>
<td>2.3 (1.7-3.1)</td>
</tr>
<tr>
<td>Both left and right arm</td>
<td>7.1 (3.6-14.2)</td>
</tr>
<tr>
<td>Chest pain most important symptom</td>
<td>2.0*</td>
</tr>
<tr>
<td>History of myocardial infarction</td>
<td>1.5-3.0•</td>
</tr>
<tr>
<td>Nausea or vomiting</td>
<td>1.9 (1.7-2.3)</td>
</tr>
<tr>
<td>Diaphoresis</td>
<td>2.0 (1.9-2.2)</td>
</tr>
<tr>
<td>Third heart sound on auscultation</td>
<td>3.2 (1.6-6.5)</td>
</tr>
<tr>
<td>Hypotension (systolic BP ≤80 mmHg)</td>
<td>3.1 (1.8-5.2)</td>
</tr>
<tr>
<td>Pulmonary crackles on auscultation</td>
<td>2.1 (1.4-3.1)</td>
</tr>
</tbody>
</table>

* Data not available to calculate confidence interval (CI).
• In heterogeneous studies the likelihood ratios are reported as ranges. Adapted from Panju, AA, Hemmelgarn, BR, Guyatt, GH, Simel, DL, JAMA 1998; 280:1256.
<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Historical features</th>
<th>Examination findings</th>
<th>Electrocardiogram</th>
<th>Chest X-ray</th>
<th>Additional tests</th>
<th>Additional important information</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acute coronary syndrome</strong></td>
<td>• Substernal/left sided chest pressure or tightness is common</td>
<td>• Nonspecific</td>
<td>• ST segment elevations, Q waves, new left bundle</td>
<td>• Nonspecific</td>
<td>• Troponin and/or CK-MB elevations diagnose AMI</td>
<td>• Asssume symptoms of ACS within days or a few weeks of PCI or CABG is from an occluded artery or graft</td>
</tr>
<tr>
<td></td>
<td>• Onset is gradual</td>
<td>• May detect signs of HF</td>
<td>branch block are evidence of AMI</td>
<td></td>
<td>• Single set of biomarkers is not sufficiently sensitive to rule out AMI</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Pain radiating to shoulders or pain with exertion increases relative risk</td>
<td></td>
<td>• Single ECG is not sensitive for ACS</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>• &quot;Atypical&quot; symptoms (eg, dyspnea, weakness) more common in elderly, women,</td>
<td></td>
<td>• Prominent R waves with ST segment</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>depressions in V1 and V2 strongly suggests</td>
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<td></td>
<td></td>
<td></td>
<td>posterior AMI</td>
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<tr>
<td></td>
<td>• Elderly can present with dyspnea, weakness, syncope, or ¾MS alone</td>
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<tr>
<td><strong>Aortic dissection</strong></td>
<td>• Sudden onset of sharp, tearing, or ripping pain</td>
<td>• Absent upper extremity or carotid pulse is suggestive</td>
<td>• Ischemic changes in 15 percent</td>
<td>• Wide mediastinum or loss of normal aortic knob contour is common (up to 76 percent)</td>
<td>• 10 percent have normal CXR</td>
<td>• Can mimic many diseases depending on branch arteries involved (eg, AMI, stroke)</td>
</tr>
<tr>
<td></td>
<td>• Maximal severity at onset</td>
<td>• Discrepancy in systolic BP &gt;20 mmHg between right and left upper extremity is suggestive</td>
<td>• Nonspecific ST and T changes in 30 percent</td>
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<tr>
<td></td>
<td>• Most often begins in chest, can begin in back</td>
<td>• Up to 30 percent with neurologic findings</td>
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</tr>
<tr>
<td></td>
<td>• Can mimic: stroke, ACS, mesenteric ischemia, kidney stone</td>
<td>• Findings vary with arteries affected</td>
<td></td>
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</tr>
<tr>
<td><strong>Pulmonary embolism</strong></td>
<td>• Many possible presentations, including pleuritic pain and painless dyspnea</td>
<td>• No finding is sensitive or specific</td>
<td>• Usually abnormal but nonspecific</td>
<td>• Great majority are normal</td>
<td>• A high-sensitivity D-dimer is useful to rule out PE only when negative in low-risk patients</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Often sudden onset</td>
<td>• Extremity exam generally normal</td>
<td>• Signs of right heart strain suggestive (eg, RAD, RBBB, RAE)</td>
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<tr>
<td></td>
<td>• Dyspnea often dominant feature</td>
<td>• Lung exam generally nonspecific; focal wheezing may be present; tachypnea is common</td>
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</tr>
<tr>
<td><strong>Tension pneumothorax</strong></td>
<td>• Often sudden onset</td>
<td>• Ipsilateral diminished or absent breath sounds</td>
<td>• Demonstrates air in pleural space</td>
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</tr>
<tr>
<td></td>
<td>• Initial pain often sharp and pleuritic</td>
<td>• Subcutaneous emphysema is uncommon</td>
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</tr>
<tr>
<td></td>
<td>• Dyspnea often dominant feature</td>
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</table>
### Pericardial tamponade

- 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.
- Severe tamponade creates obstructive shock, and causes jugular venous distension, pulsus paradoxus.
- Pericardial effusion can cause friction rub.
- Decreased voltage and electrical alternans can appear with significant effusions.
- Diffuse PR segment depressions and/or ST segment elevations can appear with acute pericarditis.
- May reveal enlarged heart.
- Ultrasound reveals pericardial effusion with tamponade.

### Mediastinitis (esophageal rupture)

- 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.
- Ill-appearing; shock, fever.
- May hear (Hamman’s) crunch over mediastinum.
- Large majority have some abnormality; pneumomediastinum, pleural effusion, pneumothorax.

Adult cardiac arrest algorithm: 2010 ACLS guidelines

### Killip classification of acute myocardial infarction

<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No evidence of heart failure</td>
</tr>
<tr>
<td>II</td>
<td>Findings consistent with mild to moderate heart failure (S3 gallop, lung rales less than one-half way up the posterior lung fields, or jugular venous distension)</td>
</tr>
<tr>
<td>III</td>
<td>Overt pulmonary edema</td>
</tr>
<tr>
<td>IV</td>
<td>Cardiogenic shock</td>
</tr>
</tbody>
</table>
Adult tachycardia algorithm (with pulse): 2010 ACLS guidelines

1. Assess appropriateness for clinical condition. Heart rate typically >150/min if tachyarrhythmia.

2. Identify and treat underlying cause
- Maintain patient airway; assist breathing as necessary
- Oxygen (if hypoxic)
- Cardiac monitor to identify rhythm; monitor blood pressure and oximetry

3. Persistent tachyarrhythmia causing:
- Hypotension?
- Acutely altered mental status?
- Signs of shock?
- Ischemic chest discomfort?
- Acute heart failure?

4. Synchronized cardioversion
- Consider sedation
- If regular narrow complex, consider adenosine

5. Wide QRS?
- 20/12 second

6. Yes
- IV access and 12-lead ECG if available
- Consider adenosine only if regular and monomorphic
- Consider antiarrhythmic infusion
- Consider expert consultation

7. No
- IV access and 12-lead ECG if available
- Vagal maneuvers
- Adenosine (if regular)
- β-blocker or calcium channel blocker
- Consider expert consultation

Doses/Details

Synchronized cardioversion

Initial recommended doses:
- Narrow regular: 50-100 J
- Narrow irregular: 120-200 J biphasic or 200 J monophasic
- Wide regular: 100 J
- Wide irregular: defibrillation dose (NOT synchronized)

Adenosine IV dose:
First dose: 6 mg rapid IV push; follow with NS flush.
Second dose: 12 mg if required.

Antiarrhythmic infusions for stable wide-QRS tachycardia
Procainamide IV dose:
20-50 mg/min until arrhythmia suppressed, hypotension ensues.
QRS duration increases >50 percent, or maximum dose 17 mg/kg given.
Maintenance infusion: 1-4 mg/min.
Avoid if prolonged QT or CHF.

Amiodarone IV dose:
First dose: 150 mg over 10 minutes. Repeat as needed if VT recurs.
Follow by maintenance infusion of 0.1 mg/min for first 6 hours.
Total IV dose:
100 mg (1.5 mg/kg) over 5 minutes. Avoid if prolonged QT.

Adult bradycardia algorithm (with pulse): 2010 ACLS guidelines

1. Assess appropriateness for clinical condition. Heart rate typically < 50/min if bradycardia.

2. Identify and treat underlying cause:
   - Maintain patient airway; assist breathing as necessary
   - Oxygen (if hypoxemic)
   - Cardiac monitor to identify rhythm; monitor blood pressure and oximetry
   - IV access
   - 12-lead ECG if available; don't delay therapy

3. Persistent bradycardia causing:
   - Hypothyroidism?
   - Acute altered mental status?
   - Signs of shock?
   - Ischemic chest discomfort?
   - Acute heart failure?

   Yes

   Atropine if atropine ineffective:
   - Transcutaneous pacing
   - Dopamine infusion
   - Epinephrine infusion

   No

4. Monitor and observe

5. Consider:
   - Expert consultation
   - Transvenous pacing

DOSES/DETAILS:
Atropine IV dose:
First dose: 0.5 mg bolus
Repeat every 3-5 minutes
Maximum: 3 mg
Dopamine IV infusion:
2-10 mcg/kg per minute
Epinephrine IV infusion:
2-10 mcg per minute
