Evaluation of the adult with chest pain in the emergency department

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INTRODUCTION

Chest pain accounts for approximately 7.6 million annual visits to emergency departments (ED) in the United States, making chest pain the second most common complaint [1]. Patients present with a spectrum of signs and symptoms reflecting the many potential etiologies of chest pain. Diseases of the heart, aorta, lungs, esophagus, stomach, mediastinum, pleura, and abdominal viscera may all cause chest discomfort.

Clinicians in the ED focus on the immediate recognition and exclusion of life-threatening causes of chest pain. Patients with life-threatening etiologies for chest pain may appear deceptively well, manifesting neither vital sign nor physical examination abnormalities.

This topic review will discuss life-threatening and common causes of chest pain and provide an approach to the evaluation of chest pain patients in the ED. Detailed discussions of specific causes of chest pain, including the management of a suspected acute coronary syndrome in the ED are found separately. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department" and "Epidemiology and pathogenesis of acute pulmonary embolism in adults" and "Management of acute type B aortic dissection" and "Pneumothorax in adults: Epidemiology and etiology" and "Treatment of secondary spontaneous pneumothorax in adults" and "Cardiac tamponade" and "Boerhaave syndrome: Effort rupture of the esophagus".)

DIFFERENTIAL DIAGNOSIS

Life-threatening conditions — Causes of chest pain that pose an immediate threat to life include are listed and described briefly below.

- Acute coronary syndrome
- Acute aortic dissection
- Pulmonary embolism
- Tension pneumothorax
- Pericardial tamponade
- Mediastinitis (eg, esophageal rupture)

- **Acute coronary syndrome** – Coronary vascular disease remains the leading killer of adults in developed countries. The 28 day case mortality rate for an acute coronary syndrome (ACS) among patients in developed nations is approximately 10 percent, but varies with the severity of disease and the treatment provided. Less than 15 to 30 percent of patients who present to the emergency department (ED) with nontraumatic chest pain have ACS, which includes myocardial infarction and unstable angina [2,3]. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department" and "Evaluation of emergency department patients with chest pain at low or intermediate risk for acute coronary syndrome" and "Overview of the acute management of ST-elevation myocardial infarction" and "Overview of the acute management of non-ST-elevation acute coronary syndromes".)

  ACS results from atherosclerotic plaque rupture and thrombus formation via the adhesion, activation, and aggregation of platelets. Coronary blood flow is reduced and myocardial ischemia occurs. The degree and duration of the oxygen supply-demand mismatch determines whether the patient develops reversible myocardial ischemia without injury (unstable angina) or myocardial ischemia with injury (myocardial infarction). (See "The role of platelets in coronary heart disease".)

- **Acute aortic dissection** – The incidence of aortic dissection is estimated at 3 per 100,000 patients per year. This number may be a gross underestimation of the true incidence as many patients die prior to diagnosis. Aortic dissection most commonly affects patients with systemic hypertension in their seventh decade of life, but it can affect younger individuals, particularly those with known aortic valve or connective tissue abnormalities. Dissection typically begins with a tear in the inner layer of the aortic wall allowing blood to track between the intima (inner layer) and media (middle layer). Pulsatile blood flow

causes propagation of the dissection with subsequent obstruction of branch arteries (eg, coronary, carotid, mesenteric) leading to ischemic injury to areas perfused by those vessels. In approximately 13 percent of cases, no intimal tear is identified. Such patients have an acute intramural hematoma likely caused by bleeding of the vasa vasorum with intramural hematoma formation in the wall of the aorta. The clinical picture of aortic intramural hematoma and other acute aortic syndrome (eg, penetrating aortic ulcer, aortic rupture) is similar to classic acute aortic dissection. (See "Clinical features and diagnosis of acute aortic dissection" and "Overview of acute aortic dissection and other acute aortic syndromes".)

- **Pulmonary embolism** – The incidence of pulmonary embolism (PE) is estimated at over 1 in 1000 patients, but the diagnosis is often missed and the incidence may be higher. Mortality rates vary widely based upon comorbid conditions and the size of the embolus. Early diagnosis and treatment reduce mortality for large hemodynamically unstable pulmonary emboli. Pulmonary embolism occurs when a dislodged venous clot migrates through the right side of the heart and becomes lodged at the branch point of the pulmonary arteries (saddle embolus) or more distally. Occlusion of pulmonary blood flow results in pulmonary hypertension, right ventricular dysfunction, poor gas exchange, and ultimately parenchymal infarction. (See "Epidemiology and pathogenesis of acute pulmonary embolism in adults".)

- **Pneumothorax** – Pneumothorax can occur following trauma or pulmonary procedures. It also occurs spontaneously in patients with underlying lung disease (secondary pneumothorax) and without (primary pneumothorax) (image 1). Patients with primary spontaneous pneumothorax tend to be younger males who are tall and thin. Secondary spontaneous pneumothorax occurs with greatest frequency in patients with chronic obstructive pulmonary disease, cystic fibrosis, and asthma. Regardless of etiology, the accumulation of air in the pleural space can lead to tension pneumothorax with compression of the mediastinum, causing rapid clinical deterioration and death if unrecognized. (See "Pneumothorax in adults: Epidemiology and etiology".)

- **Mediastinitis** – Common causes of mediastinitis include odontogenic infections, esophageal perforation, and iatrogenic complications of cardiac surgery or upper gastrointestinal and airway procedures. Mortality for patients with mediastinitis remains high (14 to 42 percent), even when treated with operative debridement and antibiotics [4-7]. Delays in diagnosis further increase mortality. (See "Boerhaave syndrome: Effort rupture of the esophagus" and "Postoperative mediastinitis after cardiac surgery".)

- **Pericardial tamponade** – Pericardial tamponade occurs when there is accumulation of pericardial fluid under pressure, leading to impaired cardiac filling. Tamponade covers a spectrum of clinical severity. Some patients have mild compromise, while others develop a severe compromise in cardiac filling, producing a picture resembling cardiogenic shock that requires immediate reduction in pericardial pressure by pericardiocentesis. Tamponade may occur with aortic dissection, after thoracic trauma, or as a consequence of acute pericarditis from infection, malignancy, uremia, or some other cause. (See "Cardiac tamponade".)

Common conditions — Below is a brief description of diseases that commonly occur among emergency department (ED) patients complaining of chest pain. Gastrointestinal problems, such as gastroesophageal reflux disease, comprise a significant number of such patients [2]. Common causes of chest pain that are not life-threatening are discussed in greater detail separately.

- **Cardiac causes** – Acute heart failure is frequently associated with chest discomfort. Patients with stable angina can usually identify their anginal chest pain and relay a history of exertional triggers. Valvular heart disease, such as aortic stenosis, may cause chest discomfort, which may signify worsening valvular function. Infectious or inflammatory causes of chest discomfort include pericarditis, myocarditis, and endocarditis. Accumulation of pericardial fluid can result in chest discomfort as can cardiac arrhythmias, especially if coronary blood flow is impaired. (See "Approach to diagnosis and evaluation of acute decompensated heart failure in adults" and "Chronic coronary syndrome: Overview of care" and "Clinical manifestations and diagnosis of aortic stenosis in adults" and "Clinical manifestations and evaluation of adults with suspected left-sided native valve endocarditis" and "Acute pericarditis: Clinical presentation and diagnosis" and "Clinical manifestations and diagnosis of myocarditis in adults".)

- **Pulmonary/pleural causes** – Respiratory infections, such as pneumonia, tracheitis, and bronchitis, are frequently accompanied by chest discomfort and cough. Chest tightness is a common complaint with asthma exacerbations. A number of disease processes can result in increased pulmonary arterial pressures and resultant right sided heart dysfunction (cor pulmonale). Pulmonary malignancy can cause chest pain particularly if there is pleural involvement. Chest heaviness or discomfort may be noted with pleural effusions. (See "Clinical evaluation and diagnostic testing for community-acquired pneumonia in adults" and "Acute exacerbations of asthma in adults: Home and office management" and "Pulmonary hypertension due to lung disease and/or hypoxemia

(group 3 pulmonary hypertension): Epidemiology, pathogenesis, and diagnostic evaluation in adults" and "Pleural fluid analysis in adults with a pleural effusion".)

- **Gastrointestinal causes** – Gastroesophageal reflux and esophageal spasm, rupture (Boerhaave syndrome), or inflammation can all present as chest discomfort. A sliding hiatal hernia may result in chest pain. Pain from pancreatitis can be referred to the chest. Gastrointestinal causes account for the symptoms of a sizable number of patients who complain of chest pain and do not have an acute coronary syndrome. (See "Clinical manifestations and diagnosis of gastroesophageal reflux in adults" and "Evaluation of the adult with chest pain of esophageal origin" and "Boerhaave syndrome: Effort rupture of the esophagus" and "Clinical manifestations and diagnosis of acute pancreatitis".)

- **Musculoskeletal causes** – Musculoskeletal causes of chest pain include rib contusions and fractures, intercostal muscle strains, and costochondritis. (See "Major causes of musculoskeletal chest pain in adults".)

- **Psychiatric causes** – Patients with panic attack often complain of chest tightness and a sense of impending doom. This remains a diagnosis of exclusion in the ED. (See "Panic disorder in adults: Treatment overview".)

- **Other conditions** – Less commonly encountered conditions that may manifest as chest pain include: herpes zoster, referred pain, and pain associated with various inflammatory conditions and collagen vascular diseases, including lupus, sarcoid, scleroderma, Kawasaki disease, polyarteritis nodosa, and Takayasu arteritis. (See "Postherpetic neuralgia".)

HISTORY

Thoracic organs share afferent nervous system pathways. This creates significant overlap in the symptoms patients experience when thoracic organs develop disease, and makes it difficult to distinguish which organ system is involved purely on the basis of history. Patient descriptions of their symptoms can be helpful in some instances, but emergency clinicians must guard against premature diagnostic closure based upon history. Several studies demonstrate that so-called "atypical" presentations occur more often than was previously thought and misinterpretation of such presentations increases the risk for misdiagnosis and adverse outcomes [8,9].

General approach — Obtain a detailed history of the patient's chest pain, including:

- Onset of pain (eg, abrupt or gradual)
- Provocation/Palliation (which activities provoke pain; which alleviate pain)

- Quality of pain (eg, sharp, squeezing, pleuritic)
- Radiation (eg, shoulder, jaw, back)
- Site of pain (eg, substernal, chest wall, back, diffuse, localized)
- Timing (eg, constant or episodic, duration of episodes, when pain began)

Ask about prior diagnostic studies (eg, stress test or coronary CT angiography) for similar symptoms or prior procedures (eg, cardiac catheterization). Ask whether the discomfort is similar to prior illness. Associated symptoms, such as nausea, vomiting, diaphoresis, dyspnea, syncope, and palpitations, can be helpful. Preceding or concomitant symptoms, such as fever or peripheral edema, may point to a diagnosis. Ask about risk factors for life-threatening illness, especially known risk factors for acute coronary syndrome, acute aortic dissection, and pulmonary embolus. These include the following and others discussed below: (See 'Risk factors' below.)

- Comorbidities: hypertension, diabetes mellitus, peripheral artery disease, malignancy, connective tissue disorders, bicuspid aortic valve, recent pregnancy

- Recent events: trauma, major surgery or medical procedures (eg, endoscopy, aortic catheterization), periods of immobilization (eg, long plane ride)

- Other factors: cocaine use, cigarette use, family history

Onset of pain — The timing of the onset of chest pain can help to narrow the differential diagnosis. Pain that starts suddenly and is severe at onset is associated with acute aortic dissection, pneumothorax, and pulmonary embolism. Abrupt onset of pain was reported in 85 percent of patients in one registry of patients with acute aortic dissection [10]. Chest pain associated with pulmonary embolism can begin suddenly, but may worsen over time. Nontraumatic pneumothorax most often occurs suddenly at rest, without any precipitating event. A history of forceful vomiting preceding symptoms in a toxic appearing patient raises concern for a ruptured esophagus and mediastinitis. However, a significant portion of patients who rupture their esophagus give no history of vomiting and presentations vary [11-13].

Conversely, discomfort from an acute coronary syndrome typically starts gradually and may worsen with exertion. With stable angina, discomfort occurs only when activity creates an oxygen demand that outstrips supply limitations imposed by a fixed atherosclerotic lesion. This occurs at relatively predictable points and changes slowly over time. Unstable angina represents an abrupt change from baseline functioning, which may manifest as discomfort that begins at lower levels of exercise or at rest.

Pain quality and location — Patients often describe the symptoms of an **acute coronary syndrome** (ACS) as discomfort rather than pain. The discomfort may be a pressure, heaviness, tightness, fullness, or squeezing. Ischemia is less likely if the discomfort is knifelike, sharp, pleuritic, or positional. The classic location is substernal or in the left chest, and radiation to the arm, neck, jaw, back, abdomen, or shoulders may occur. Pain that radiates to the shoulders or occurs with exertion significantly increases the relative risk for ACS.

Relief of pain following the administration of sublingual nitroglycerin does **not** reliably distinguish between cardiac ischemia and noncardiac causes of chest pain [14,15]. Beware of "atypical" presentations of ACS, which are common and occur more often in the elderly, diabetics, and women. Patients with "atypical" symptoms (eg, dyspnea, weakness) associated with their myocardial infarction fare worse than patients who experience typical symptoms, likely due to delays in diagnosis and treatment. These issues are discussed in greater detail separately. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department", section on 'Clinical presentation' and "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department", section on 'Atypical presentations'.)

An acute aortic dissection most often presents with the sudden onset of sharp, severe pain [10,16]. Patients may describe the pain as tearing, or ripping. However, according to the International Registry of Acute Aortic Dissection (IRAD), presentations can be diverse and classic findings absent. The pain most often occurs in the chest, but can begin in the back, and may migrate or radiate into other areas of the chest, back, or abdomen, depending upon the portion of the aorta involved and the extent of the dissection. Sharp pain may also accompany pulmonary embolism, pneumothorax, or pericarditis.

A **pulmonary embolism** (PE) can create different kinds of pain, or painless dyspnea. Pain associated with PE may worsen with deep inspiration, and may localize to the chest wall. Patients with **pneumothorax** report ipsilateral chest pain which may initially be sharp and pleuritic but may become dull or achy over time. The discomfort of **pericarditis** is classically positional: worse when lying supine and relieved somewhat when leaning forward. It may also worsen with deep inspiration.

Sharp, well-localized pain reproduced with movement or palpation of the chest wall is characteristic of **musculoskeletal** causes. Often the patient relates a history of trauma or strenuous activity prior to developing pain.

Burning pain in the chest and epigastrium is commonly associated with gastrointestinal causes. **Esophageal rupture** can cause chest and/or abdominal pain. Cardiac disease can cause identical symptoms, however, and emergency clinicians must avoid prematurely attributing such symptoms to gastrointestinal disease.

Associated symptoms — Diaphoresis, nausea, and vomiting frequently accompany chest discomfort associated with acute coronary syndrome (ACS), but are not predictive of ACS [17]. Elderly patients with ACS may only complain of symptoms other than chest pain, such as dyspnea, weakness, altered mental status, or syncope. Symptoms, such as diaphoresis and nausea, may also occur with nonischemic chest pain, including aortic dissection, pulmonary embolus, acute heart failure, and esophageal spasm.

Acute aortic dissection has a wide range of potential associated symptoms, depending on the arterial branches involved, which may confound the diagnosis (table 1). According to one review, syncope accompanies 13 percent of dissections involving the ascending aorta [18]. Neurological symptoms, ranging from hoarseness to paraplegia and altered mental status, occur in 18 to 30 percent of patients with aortic dissection [18]. ACS can occur when the dissection involves the coronary arteries.

Shortness of breath frequently accompanies pulmonary causes of chest pain and may be the predominant symptom in pulmonary embolus, pneumothorax, and pneumonia. Tachypnea is common with PE and may be accompanied by wheezing and fever. Young healthy patients may manifest only a relative tachypnea or tachycardia despite the presence of pneumonia or pulmonary embolism. Dyspnea is often the only complaint among elderly patients with ACS.

Cough, syncope, and hemoptysis may occur with pulmonary embolism or valvular heart disease (particularly mitral stenosis), although cough and hemoptysis are more common with bronchitis, pharyngitis, or exacerbations of COPD. Dyspnea and cough may accompany pericardial and pleural effusions regardless of etiology.

Preceding or concomitant pain and swelling in an extremity suggests deep venous thrombosis (DVT) complicated by pulmonary embolism. DVT occurs most often in the lower extremities but clots may also originate in the upper extremities and the large veins of the pelvis, where they may produce bilateral lower extremity swelling if the inferior vena cava becomes occluded.

Nausea and belching frequently accompany gastrointestinal causes of chest pain, but can also occur in patients with inferior myocardial infarction. Fever raises concern for infectious causes, but is also associated with pericarditis, myocarditis, and rarely acute myocardial infarction. A low-grade fever may accompany pulmonary embolus.

Risk factors — Risk factors for **acute coronary syndrome** (ACS) include: male sex, age over 55 years, family history of coronary artery disease, diabetes mellitus, hypercholesterolemia, hypertension, and tobacco use. Cardiac risk factors are poor predictors of acute risk in symptomatic emergency department (ED) patients, as the presence of chest pain outweighs their predictive value [19]. The absence of cardiac risk factors does not identify patients that can safely be discharged from the ED. Cocaine or amphetamine use raises concern for ACS regardless of other risk factors. Cocaine increases the metabolic demands of the heart via its stimulant effects, and also causes coronary artery vasoconstriction and promotes thrombus formation in patients who may otherwise be at low risk for ACS. (See "Overview of established risk factors for cardiovascular disease" and "Cocaine: Acute intoxication".)

Aortic dissection occurs most often in older patients with systemic hypertension and atherosclerotic disease, but this description alone is not helpful for distinguishing it from other life-threatening conditions. The possibility of **aortic dissection** should be considered in patients with acquired or congenital conditions that weaken the structural architecture of the aortic wall. Patients younger than 40 years of age with connective tissue disorders, such as Marfan's syndrome, bicuspid aortic valve, cocaine use, or pregnancy (particularly in combination with bicuspid aortic valve or connective tissue disease) are at risk for aortic dissection. Other factors that predispose to aortic dissection include previous aortic surgery and recent cardiac surgery or aortic instrumentation. (See "Clinical features and diagnosis of acute aortic dissection", section on 'High-risk conditions' and "Overview of acute aortic dissection and other acute aortic syndromes".)

An increased risk for deep vein thrombosis and subsequent **pulmonary embolus** exists among patients with a recent history of prolonged immobilization (eg, long distance travel), surgery (particularly an orthopedic procedure of the lower extremity lasting more than 30 minutes), central venous catheterization, or trauma. Also at risk are pregnant patients, patients with cancer, lung, or chronic heart disease, and those with a personal or family history of hypercoagulability. Use of certain hormonal contraceptives [20] or chemotherapeutic agents that raise serum levels of estrogen and, to a lesser extent, progestin also confer increased risk. A significant number of patients with PE may have no identifiable risk factor at the time of diagnosis, but subsequent evaluation reveals a hematologic predisposition for venous thrombosis (eg, Factor V Leiden mutation) or anatomic predisposition (eg, thoracic outlet syndrome). (See "Epidemiology and pathogenesis of acute pulmonary embolism in adults", section on 'Pathogenesis and pathophysiology'.)

Tobacco use raises patient risk for cardiovascular and pulmonary disease. Smoking is also an independent risk factor for spontaneous **pneumothorax**, regardless of underlying lung

disease. A high prevalence of spontaneous pneumothorax exists among HIV infected patients with pneumocystis carinii (*P. jirovecii*) pneumonia. Young females with endometriosis may experience menses-related pneumothoraces, also referred to as catamenial pneumothorax, if pleural involvement exists [21]. Activities, such as SCUBA diving, can precipitate a spontaneous pneumothorax and air travel may precipitate recurrence in patients with an incompletely healed pneumothorax [22]. (See "Complications of SCUBA diving" and "Pneumothorax and air travel" and "Pneumothorax in adults: Epidemiology and etiology", section on 'Subpleural blebs' and "Clinical presentation and diagnosis of pneumothorax".)

Prior testing — Many patients with chest pain have undergone diagnostic testing for prior episodes, the results of which may be useful. As an example, a recent cardiac catheterization or coronary CT angiogram with normal or minimally diseased vessels virtually eliminates the possibility of an acute coronary syndrome (ACS). An observational study of 1977 consecutive patients with coronary arteriograms documenting minimal (ie, less than 25 percent) stenosis or normal coronary arteries found that 98 percent of these patients were free of myocardial infarction 10 years later [23]. Other studies confirm this data [24]. Conversely, a prior negative stress test is not useful to rule out ACS in patients with active chest pain in the ED.

PHYSICAL EXAMINATION

Most often the physical examination is not helpful in distinguishing patients with acute coronary syndromes (ACS) from those with noncardiac chest pain. In some instances, physical findings suggest a specific noncardiac diagnosis. Patients with an immediately life-threatening cause for their chest pain tend to appear anxious and distressed and may be diaphoretic and dyspneic.

Physical examination findings in patients with acute aortic dissection may be absent or suggestive of end-organ ischemia due to aortic branch vessel occlusion, including myocardial infarction, stroke, acute intestinal ischemia, or extremity ischemia depending on the affected arteries (table 1). Discrepancies in pulses or blood pressure are notable findings when present, but occur infrequently. In the International Registry of Acute Aortic Dissection (IRAD), signs of dissection included: murmur of aortic insufficiency (32 percent) pulse deficit (15 percent), signs of shock or cardiac tamponade (8 percent), acute heart failure (7 percent), and cerebrovascular accident (5 percent) [10]. Up to 30 percent of patients may have neurologic findings (eg, Horner syndrome, paraparesis, paraplegia).

Chest pain associated with focal wheezing or asymmetric extremity swelling raises concern for pulmonary embolus (PE). Most often patients with PE have a normal extremity examination.

Unilateral decreased breath sounds may be noted with pneumothorax; subcutaneous emphysema is uncommon.

The presence of pulmonary crackles, with or without an S3 gallop, is associated with left ventricular dysfunction and left-sided heart failure, possibly due to ACS. Jugular venous distention (movie 1), hepatojugular reflux, and peripheral edema suggest right-sided heart failure, possibly due to ACS or PE. A new systolic murmur is an ominous sign, which may signify papillary muscle dysfunction or a ventricular septal defect. Clinicians may hear a pericardial friction rub in patients with pericarditis. Hamman's crunch is a crackling sound (movie 2) similar to a pericardial friction rub heard over the mediastinum in patients with mediastinal emphysema.

Epigastric tenderness and heme positive stool suggest a possible gastrointestinal source for pain.

ANCILLARY STUDIES

Electrocardiogram

- Acute coronary syndrome – A standard 12-lead electrocardiogram (ECG) is obtained for all emergency department (ED) patients presenting with chest pain that may be from an acute coronary syndrome (ACS). Guidelines from the American College of Cardiology and American Heart Association (ACC/AHA) suggest the ECG be obtained and interpreted within 10 minutes of patient presentation in the ED.

  Although the ECG remains the best immediately available test for detecting ACS, its sensitivity for acute myocardial infarction (AMI) is low. A single ECG performed during the patient's initial presentation detects fewer than 50 percent of AMIs. Patients with normal or nonspecific ECGs have a 1 to 5 percent incidence of AMI and a 4 to 23 percent incidence of unstable angina [25-29]. The ECG can be repeated if the initial ECG is not diagnostic but the patient remains symptomatic and there remains high clinical suspicion for AMI. Prior ECGs are important for determining whether abnormalities are new. The presence of a left bundle branch block makes it difficult to determine the presence of ischemic ECG changes. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department".)

  ECG interpretation in ACS is discussed in detail separately. (See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction" and "Electrocardiogram in the prognosis of myocardial infarction or unstable angina".)

- Pulmonary embolus – The ECG is of limited value in patients with pulmonary embolism (PE). The most common finding is sinus tachycardia. The classically described finding "S1Q3T3" (ie, prominent S wave in lead I, Q wave in lead III, and inverted T wave in lead III (waveform 1)) reflects right heart strain but is neither sensitive nor specific for PE. Patients with acute PE rarely have a normal ECG, but a wide range of abnormalities is possible and most are equally likely to be seen in patients without PE [30]. If the clinical scenario suggests PE, evidence of right heart strain further increases suspicion. Right axis deviation, right bundle branch block, right atrial enlargement (ie, "P pulmonale"), and atrial fibrillation can occur. (See "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism", section on 'Electrocardiography'.)

- Pericardial tamponade and pericarditis – ECG findings suggestive of tamponade include low voltage and electrical alternans (waveform 2). ECG findings in patients with pericarditis may mimic AMI and may vary as the disease progresses. Findings include PR segment depression, ST segment elevation, and T wave inversions (waveform 3). These findings are typically more diffuse than is found in patients with focal anatomic changes from myocardial ischemia. (See "Acute pericarditis: Clinical presentation and diagnosis", section on 'Electrocardiogram'.)

- Acute aortic dissection – The ECG tracing in aortic dissection can range from completely normal to ST segment elevation if the dissection involves the origin of a coronary artery. In the IRAD review of 464 patients with aortic dissection, the ECG was normal in 31 percent, showed nonspecific ST and T wave changes in 42 percent, and showed ischemic changes in 15 percent. (See "Clinical features and diagnosis of acute aortic dissection", section on 'Electrocardiogram'.)

Laboratory studies

- **Cardiac biomarkers** – In the setting of acute myocardial infarction (AMI), advanced assays for cardiac troponin I and T detect elevations within 3 hours, peak at 12 hours, and remain elevated for 7 to 10 days. Troponins are the preferred test for the diagnosis of AMI. Highly sensitive troponin assays become elevated more rapidly and elevations are even found in patients with what was classically considered to be unstable angina.

  In the majority of cases, a single set of negative cardiac biomarkers is **NOT** sufficient to rule out myocardial infarction; however, using the high-sensitivity troponin T assay, this approach is now possible in select patients. If patients have symptoms for more than 2 hours and the initial value of troponin T is below the level of detection (<6 ng/L), these

patients can safely have AMI ruled out with just the single value [31]. Initial cardiac biomarker determinations above the level of detection cannot be used to determine discharge but abbreviated interval testing (at 0 and 1, 2 or 3 hours) using high-sensitivity troponins may allow for safe discharge and reduce additional testing in patients otherwise at low risk for ACS [32-34]. (See "Troponin testing: Clinical use" and "Elevated cardiac troponin concentration in the absence of an acute coronary syndrome" and "Evaluation of emergency department patients with chest pain at low or intermediate risk for acute coronary syndrome", section on 'Initial evaluation'.)

- **D-dimer** – Among patients with a low-pretest probability for pulmonary embolus (PE), a D-dimer test with high sensitivity can rule out the diagnosis, obviating the need for further testing. The utility of the D-dimer test depends upon both patient baseline characteristics and the sensitivity and specificity of the test employed. Patients likely to have an elevated D-dimer at baseline are the elderly and those with malignancy, sepsis, recent major surgery or trauma, or pregnancy (table 2).

  Incorporating D-dimer results into decision-making for patients with possible PE requires knowledge of the diagnostic characteristics of the test employed and a predetermined algorithm for management in light of those results. (See "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism", section on 'Hemodynamically stable patients'.)

  D-dimer in combination with the Aortic Dissection Detection Risk Score (ADD-RS) may be useful to rule out suspected aortic dissection. (See "Clinical features and diagnosis of acute aortic dissection", section on 'D-dimer' and "Clinical features and diagnosis of acute aortic dissection", section on 'High-risk clinical features' and "Overview of acute aortic dissection and other acute aortic syndromes".)

- **Complete blood count** – The white blood cell count may be elevated in any of the inflammatory or infectious etiologies of chest pain, such as myocarditis and pericarditis, mediastinitis, and pneumonia. Anemia in a patient with exertional chest pain is suggestive of myocardial ischemia, but also consistent with aortic rupture.

- **B-type natriuretic peptide (BNP) and NT-proBNP** – A number of conditions can elevate the plasma concentrations of natriuretic peptides, but BNP levels above 100 pg/mL are highly sensitive for acute heart failure (HF), while levels below 50 pg/mL have an extremely high negative predictive value for HF. When used in conjunction with other clinical information, natriuretic peptide concentrations can help to identify or exclude acute HF as

the cause of dyspnea and chest pain. (See "Heart failure: Clinical manifestations and diagnosis in adults", section on 'Natriuretic peptide'.)

- **Arterial blood gas** – The arterial-alveolar oxygen gradient provides little help in diagnosing or excluding pulmonary embolism (PE), or in distinguishing PE from other causes of ventilation-perfusion mismatch. An arterial blood gas is not routinely indicated for patients with chest pain, even when pulmonary embolism is suspected. (See "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism", section on 'Laboratory tests'.)

- Other tests – Several biomarkers are being studied for use in early diagnosis of aortic dissection, but their role remains unclear. (See "Clinical features and diagnosis of acute aortic dissection".)

Chest radiograph — A plain chest radiograph (CXR) is obtained in all chest pain patients with hemodynamic instability or a potentially life-threatening diagnosis. A nondiagnostic CXR is typical in patients with **ACS**.

Approximately 90 percent of patients with **acute aortic dissection** will have some CXR abnormality [35]. The classic findings of a widened mediastinum or aortic knob occur in up to 76 percent of patients. If clinical suspicion is high, these findings are associated with an odds ratio of 11 for aortic dissection (95% CI 6.1-19.8). Displacement of the aorta and pleural effusion may also be seen. Further imaging is obtained in patients with intermediate or high risk for aortic dissection based on clinical features. (See "Clinical features and diagnosis of acute aortic dissection", section on 'Chest radiograph' and 'Imaging for aortic dissection or pulmonary embolism' below.)

The vast majority of patients with **pulmonary embolus** (PE) have a normal or nonspecific CXR. Nevertheless, several abnormalities may suggest this diagnosis, including: atelectasis, elevated hemidiaphragm, and pleural effusion. Classically described but rare findings include: pleural-based wedge-shaped defect (representing infarcted lung parenchyma, so-called Hamptons hump (image 2)) or paucity of vascular markings distal to the site of embolus (Westermark sign (image 3)). (See "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism", section on 'Hemodynamically stable patients'.)

Pneumonia and pneumothorax are often diagnosed by CXR. A CXR taken with the patient in a lateral decubitus position may detect pneumothorax or pleural effusion when standard views are unrevealing (image 4). Acute heart failure is suggested by pulmonary vascular congestion and cardiomegaly. In patients with severe vomiting or recent instrumentation of the

esophagus, mediastinal emphysema and pleural effusion suggest **esophageal rupture**. A hiatal hernia, pleural effusion, or mass may also explain patient symptoms. (See "Clinical evaluation and diagnostic testing for community-acquired pneumonia in adults", section on 'Chest imaging findings' and "Approach to diagnosis and evaluation of acute decompensated heart failure in adults", section on 'Chest radiograph'.)

Imaging for aortic dissection or pulmonary embolism

- **Acute aortic dissection** – Several modalities diagnose aortic dissection with high sensitivity, including computed tomography (CT) (98 percent), magnetic resonance imaging (MRI) (98 percent), and transesophageal echocardiography (TEE) (94 percent).

  The patient's clinical presentation (likelihood of ascending aortic dissection, hemodynamic status) and the availability and institutional expertise with particular imaging modalities determine the approach to diagnostic imaging. TEE allows for rapid beside diagnosis of the hemodynamically unstable patient but requires an experienced echocardiographer. CT angiography is the most common initial study given that it is widely available in emergency departments (ED) and enables prompt diagnosis of aortic dissection, but may be contraindicated in patients with a contrast allergy or renal insufficiency. The decision to give contrast and risk permanent kidney injury must be balanced against the need for immediate diagnosis, weighing the clinical index of suspicion, the hemodynamic stability of the patient, and the availability of other modalities such as MRI. MRI may provide the greatest anatomic detail about the site of the intimal tear and branch vessel involvement, but it is not universally available in EDs, requires more time to perform than CT, and cannot accommodate patients with indwelling metallic hardware. (See "Clinical features and diagnosis of acute aortic dissection", section on 'Diagnosis'.)

- **Pulmonary embolism** – Pulmonary embolism (PE) can be diagnosed by computed tomography (CT), nuclear imaging, or pulmonary angiography. Bedside ultrasound or echocardiography can provide important confirmatory findings, particularly in hemodynamically unstable patients.

  CT is the most widely used study for the diagnosis of PE. CT provides information about alternative etiologies of chest pain, but exposes patients to radiation and contrast dye, which can limit its use. Improved imaging with multidetector CT scanners allows for visualization of pulmonary emboli in the subsegmental pulmonary arteries, although smaller emboli are of questionable clinical significance. CT pulmonary angiography combined with venography can detect a deep vein thrombosis (DVT) using a single dose of contrast agent. Duplex ultrasonography can be helpful in patients at risk for PE with

physical findings suggestive of DVT. Pulmonary angiography, once the gold standard, is rarely used. Nuclear ventilation/perfusion lung scanning is still used, but frequently results in subsequent imaging because of the high number of indeterminant studies. (See "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism".)

Bedside cardiac ultrasound may be useful in patients with suspected pulmonary embolism and unstable vital signs. Transthoracic echocardiography demonstrating right ventricular strain and wall motion abnormalities, such as McConnell's sign (normal motion at the right ventricular apex relative to akinesis or hypokinesis of the right ventricle free wall), have high specificity but low sensitivity in such settings [36].

Other imaging methods

- Computed tomography (CT) – Computed tomography (CT) technology continues to evolve. Studies for pulmonary embolism or acute aortic dissection can now be performed with a single injection of contrast. CT coronary angiography allows for quantification of coronary artery stenosis and studies suggest excellent correlation of CT coronary angiography with cardiac catheterization. Randomized controlled trials show that CT coronary angiography safely facilitates higher rates of discharge from the ED [37,38]. CT can distinguish a pulmonary bleb from true pneumothorax and can determine the extent of mediastinal soilage in the setting of esophageal rupture. Triple rule out CT scans can be performed to evaluate simultaneously aortic dissection, coronary artery disease, and pulmonary embolism; although these scans have compared well to dedicated coronary CT angiography, results compared to dedicated acute aortic dissection or pulmonary embolism scans are not known. Additionally, triple rule out scans have been associated with increased radiation exposure [39]. (See "Cardiac imaging with computed tomography and magnetic resonance in the adult".)

- Nuclear cardiac imaging – Exercise stress testing has become commonplace in emergency departments (ED) with chest pain observation units. Exercise treadmill testing with or without nuclear imaging, as well as stress echocardiography, can assist in the risk stratification of ED patients with suspected acute coronary syndrome. (See "Stress testing for the diagnosis of obstructive coronary heart disease" and "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department", section on 'Rest and stress imaging studies'.)

- Bedside ultrasonography – Bedside ultrasonography is used with increasing frequency and expertise by emergency clinicians and often helps to exclude or support certain

diagnoses. It is used to assess patients with blunt trauma as part of the extended FAST exam to identify traumatic pneumothorax. In addition, ultrasound can identify pericardial effusions and tamponade, wall motion abnormalities, valvular and septal abnormalities, right ventricular strain, and pleural effusions. However, echocardiography cannot distinguish old from new myocardial infarcts. (See "Emergency ultrasound in adults with abdominal and thoracic trauma".)

APPROACH TO DIAGNOSIS

The emergency clinician assesses all patients with acute chest pain for life-threatening causes. Often a definitive diagnosis cannot be made initially and additional testing is performed in parallel with management. The patient's history, comorbidities, and description of symptoms help to narrow the scope of potential diagnoses and to stratify the patient's risk for life-threatening disease. The physical examination focuses on vital sign abnormalities and cardiac or pulmonary findings, and may support a diagnosis. An electrocardiogram (ECG) and chest x-ray (CXR) are reviewed. An algorithm outlining an approach to the emergency department patient with chest pain and a table allowing for quick comparison of findings in life-threatening causes of chest pain are provided (algorithm 1 and table 3). (See 'History' above and 'Ancillary studies' above.)

- **Acute coronary syndrome** – Acute coronary syndrome (ACS) is the most common potentially life-threatening cause of chest pain encountered in the ED and is characterized by a paucity of examination findings. Any patient without a clear explanation for their chest pain after the initial workup, including electrocardiogram (ECG) and chest x-ray (CXR), is completed is assumed to have ACS until proven otherwise. Serial ECGs, risk assessment using a validated instrument (eg, Thrombolysis in Myocardial Infarction [TIMI] score; HEART score (table 4)), and troponin testing can be used for rapid risk stratification of patients without ST elevation myocardial infarction (STEMI) [40-44]. (See "Evaluation of emergency department patients with chest pain at low or intermediate risk for acute coronary syndrome", section on 'Risk scores' and "Evaluation of emergency department patients with chest pain at low or intermediate risk for acute coronary syndrome", section on 'Troponin testing'.)

  Patients with ST elevation myocardial infarction (STEMI) or at high risk for ACS are managed accordingly. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department", section on 'Immediate emergency department interventions' and "Acute ST-elevation myocardial infarction: Selecting a reperfusion strategy".)

Risk stratification of patients with potential ACS includes two parallel strategies: identify patients at such low risk that they can be safely discharged home with follow-up and identify patients at sufficiently high risk to require admission and acute management. (See "Evaluation of emergency department patients with chest pain at low or intermediate risk for acute coronary syndrome".)

Keep the following guidance in mind when considering the diagnosis of ACS for a patient with acute chest pain. Assume that any patient who presents with symptoms of an acute coronary syndrome within a few days or weeks following percutaneous coronary interventions (eg, angioplasty or stent placement) or coronary artery bypass grafting has an abruptly occluded coronary artery or graft until proven otherwise. Remain cautious when assessing the elderly, diabetics, and women, who are more likely to manifest "atypical" symptoms with ACS. Never rely on a single ECG or a single set of cardiac biomarkers to rule out ACS, unless symptoms have been continuous and prolonged (ie, over six to eight hours). (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department", section on 'Disposition of patient without STEMI'.)

- **Acute aortic dissection** – According to one prospective observational study, the probability of aortic dissection increases significantly with the presence of the following findings:

  - History: Abrupt onset of thoracic or abdominal pain with a sharp, tearing and/or ripping character

  - Examination: A variation in pulse (absence of a proximal extremity or carotid pulse) and/or blood pressure (>20 mmHg difference between the right and left arm)

  - Chest radiograph (CXR): Mediastinal and/or aortic widening [35].

  The emergency clinician should look for these examination and radiographic findings in any patient with a history suggestive of aortic dissection. According to this study, aortic dissection occurs in approximately 83 percent of patients with classic aortic dissection pain and suggestive CXR findings, and approximately 92 percent of patients with classic pain and an absent pulse or significant difference in blood pressure. When all three variables coexist, aortic dissection is present in all patients; when no variable is present approximately 7 percent of patients have aortic dissection.

  The Aortic Dissection Detection Risk Score (ADD-RS) may also be used to assess patients based on the presence of one or more of three categories of findings:

- High-risk condition such as Marfan syndrome, family history of aortic disease, known aortic valve disease, known thoracic aortic aneurysm, or previous aortic manipulation, including cardiac surgery.
- Pain in the chest, back, or abdomen described as abrupt, of severe intensity, or a ripping/tearing sensation.
- Physical examination findings of perfusion deficit, including pulse deficit, systolic blood pressure difference, or of focal neurologic deficit, or of aortic diastolic murmur and hypotension or shock.

The use of the ADD-RS with or without D-dimer testing is discussed in detail separately. (See "Clinical features and diagnosis of acute aortic dissection", section on 'Diagnosis'.)

Definitive diagnostic testing is determined by the patient's hemodynamic stability and the imaging modalities available. (See "Clinical features and diagnosis of acute aortic dissection", section on 'Diagnosis'.)

- **Pulmonary embolism** – Pulmonary embolism (PE) is a common and potentially life-threatening disease frequently missed by emergency clinicians because of its wide range of presentations and nonspecific findings on examination, electrocardiogram, and chest x-ray. Often the biggest problem with PE is failure to consider the diagnosis. Emergency clinicians must consider PE a potential diagnosis in any patient with acute chest discomfort or dyspnea who lacks a firm alternative diagnosis (eg, myocardial infarction diagnosed by history and elevated ST segments, pericardial tamponade diagnosed by ultrasound).

  The approach to patients with potential PE focuses on risk stratification. Patients with symptoms suggestive of PE and right ventricular heart dysfunction or hemodynamic instability are at high risk and may benefit from emergency thrombolysis or embolectomy. For all other patients, risk stratification depends on the pretest probability for PE.

  Several scoring systems exist to characterize patient risk for PE, including the Wells score (table 5) (calculator 1), the Charlotte criteria, the revised Geneva score, and the PERC rule (table 6) (calculator 2). For patients at low-clinical risk it is generally reasonable to withhold anticoagulant therapy while a D-dimer test is performed. In patients at low risk, PE can be ruled out with a negative D-dimer test, provided the test is of high sensitivity. Patients at low risk but whose D-dimer test is positive and those at higher risk require further testing. The PERC rule identifies patients at sufficiently low risk for PE that even D-dimer testing may be unnecessary. Detailed discussions of risk stratification, diagnosis,

and management are found separately. (See "Epidemiology and pathogenesis of acute pulmonary embolism in adults", section on 'Clinical presentation, evaluation, and diagnosis' and "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism" and "Treatment, prognosis, and follow-up of acute pulmonary embolism in adults".)

- **Pericardial tamponade** – Bedside ultrasound is an ideal tool to diagnose or rule out cardiac tamponade in any patient with suggestive historical, examination, or electrocardiogram findings. ED clinicians should perform this study in every patient with acute chest pain and signs of shock. (See "Cardiac tamponade".)

- **Pneumothorax** – Tension pneumothorax is diagnosed clinically and treated with immediate needle thoracostomy, followed by tube thoracostomy. A suggestive history combined with hemodynamic compromise and unilateral diminished breath sounds is the usual presentation. Treatment should not be delayed for confirmation by chest x-ray (CXR). A CXR or bedside ultrasound may be used to make the diagnosis in patients without signs of tension. (See "Pneumothorax in adults: Epidemiology and etiology".)

- **Mediastinitis** – The initial plain chest radiograph is almost always abnormal in patients with esophageal perforation and mediastinitis, and usually reveals mediastinal or free peritoneal air as the initial radiologic manifestation. CT scan may show extraesophageal air, periesophageal fluid, mediastinal widening, and air and fluid in the pleural spaces, retroperitoneum or lesser sac. The diagnosis is confirmed with the oral administration of a water soluble contrast agent followed by chest radiography looking for extravasation of contrast. (See "Boerhaave syndrome: Effort rupture of the esophagus", section on 'Diagnosis'.)

MANAGEMENT

Evaluation of the chest pain patient in the emergency department (ED) begins with assessment and stabilization of the airway, breathing, and circulation. Life-threatening problems are treated immediately, without delay for confirmatory testing. Any patient with acute chest pain at risk for a life-threatening disease is placed on a cardiac monitor and given supplemental oxygen if necessary while intravenous access is established. An electrocardiogram (ECG) and chest x-ray (CXR) are obtained.

- **Acute coronary syndrome** – Management of acute coronary syndrome (ACS) is determined largely by electrocardiogram (ECG) findings. Serial ECGs dramatically increase

the sensitivity for detecting ACS, compared with a single initial ECG. Patients with ST elevation myocardial infarction (STEMI) require emergency revascularization via percutaneous intervention or fibrinolysis. The management of ACS is discussed in detail separately. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department", section on 'Immediate emergency department interventions' and "Acute ST-elevation myocardial infarction: Selecting a reperfusion strategy" and "Overview of the acute management of ST-elevation myocardial infarction" and "Overview of the acute management of non-ST-elevation acute coronary syndromes".)

- **Aortic dissection** – Emergency treatment for a suspected aortic dissection involves blood pressure and heart rate control to reduce shearing forces and intensity of pulsatile cardiac flow (table 7). This is best achieved via a combination of beta blockers (eg, esmolol) and sodium nitroprusside (or nitroglycerin). Beta blockers should be started first to prevent potential rebound tachycardia associated with the vasodilatory effects of sodium nitroprusside. Labetalol, which has both beta and alpha blocking effects, can also be used to manage blood pressure. Emergency vascular imaging and cardiac and/or vascular surgery consultation is obtained. Aortic dissection affecting the ascending thoracic aorta is a cardiac surgical emergency. (See "Management of acute type B aortic dissection".)

- **Pulmonary embolus** – Initial management for confirmed pulmonary embolus involves anticoagulation. There is no evidence demonstrating that empiric anticoagulation should be initiated before test results are obtained. Patients with massive or submassive emboli may require more aggressive therapy with thrombolytics or embolectomy. (See "Treatment, prognosis, and follow-up of acute pulmonary embolism in adults".)

- **Pneumothorax** – Tension pneumothorax is treated with immediate tube thoracostomy or immediate needle thoracostomy followed by tube thoracostomy. Treatment should not be delayed for confirmation by chest x-ray (CXR). Symptomatic pneumothoraces not under tension are treated most often with tube thoracostomy, but smaller pneumothoraces may be amenable to aspiration and observation. (See "Pneumothorax in adults: Epidemiology and etiology" and "Thoracostomy tubes and catheters: Indications and tube selection in adults and children".)

- **Pericardial tamponade** – Tamponade with overt hemodynamic compromise requires immediate removal of pericardial fluid, which produces a rapid and dramatic improvement in cardiac and systemic hemodynamics. Early tamponade with only mild hemodynamic compromise may be treated conservatively, with careful monitoring, serial echocardiographic studies, avoidance of volume depletion, and therapy aimed at the

underlying cause. The decision to drain an effusion must take into account the clinical assessment, echocardiographic findings, and the risk of the procedure. (See "Pericardial effusion: Approach to management".)

- **Mediastinitis** – Broad spectrum antibiotics are given early in suspected mediastinitis. Consultation with cardiothoracic surgery is obtained for surgical debridement and possible repair. (See "Boerhaave syndrome: Effort rupture of the esophagus", section on 'Management'.)

DISPOSITION

Any patient with hemodynamic instability or significant respiratory distress is admitted to the intensive care unit (ICU). Patients with acute aortic dissection, pneumothorax, cardiac tamponade, and mediastinitis require admission and appropriate consultation. (See "Management of acute type B aortic dissection" and "Pneumothorax in adults: Epidemiology and etiology" and "Cardiac tamponade" and "Boerhaave syndrome: Effort rupture of the esophagus".).

Patients with pulmonary emboli with hemodynamic instability or significant hypoxia are admitted to the ICU. Stable patients with pulmonary embolism do not require admission to an ICU or telemetry monitoring. Some low-risk patients may even be treated as an outpatient. (See "Treatment, prognosis, and follow-up of acute pulmonary embolism in adults".)

Patients with STEMI receive reperfusion therapy via fibrinolytics or percutaneous coronary intervention and are admitted to the ICU. Patients at high risk for acute coronary syndrome (ACS) or death are admitted to an ICU; patients at moderate risk are admitted to a non-ICU monitored setting. (See "Overview of the acute management of ST-elevation myocardial infarction" and "Acute ST-elevation myocardial infarction: Selecting a reperfusion strategy" and "Overview of the acute management of non-ST-elevation acute coronary syndromes".)

Patients not at low risk for ACS, but without known coronary artery disease or obvious signs of myocardial infarction, and without a clear alternative diagnosis, should be observed further, and myocardial ischemia ruled out using serial cardiac biomarkers, electrocardiogram (ECG) testing, and possibly further testing. Patients at low risk with normal ECGs are managed in a non-ICU monitored setting, floor bed, or a chest pain observation unit, unless high-sensitivity troponin testing identifies them as safe for discharge. Exercise treadmill or pharmacological testing with or without nuclear imaging, as well as stress echocardiography and CT coronary angiography, can assist cardiovascular risk stratification in the ED. Patients with an uneventful

observation period, negative serial cardiac markers, and a normal stress test can be safely discharged with a referral for follow-up. If released without provocative testing, low-risk patients should have clear follow-up arranged, ideally within a few days of discharge. Follow-up within 72 hours is safe [45]. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department" and "Evaluation of emergency department patients with chest pain at low or intermediate risk for acute coronary syndrome" and "Cardiac imaging with computed tomography and magnetic resonance in the adult".)

Patients with **stable** angina do not require inpatient evaluation. Patients less than 40 years old with normal ECGs and no prior cardiac history have less than a one percent risk of ACS and less than a one percent risk of death, acute myocardial infarction, or revascularization at 30 days [46]. They can be discharged to home with follow-up. (See "Chronic coronary syndrome: Overview of care".)

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: Adult with chest pain in the emergency department".)

SUMMARY AND RECOMMENDATIONS

- **General guidance and algorithm** – Adults with chest pain account for a large number of emergency department (ED) visits. Patients present with a spectrum of signs and symptoms reflecting the many potential etiologies. Diseases of the heart, aorta, lungs, esophagus, stomach, mediastinum, pleura, and abdominal viscera may all cause chest discomfort. An algorithm outlining an approach to the ED patient with chest pain and a table allowing for quick comparison of findings in life-threatening causes are provided (algorithm 1 and table 3). (See 'Approach to diagnosis' above.)

- **Life-threatening causes** – Clinicians in the ED focus on the immediate recognition and exclusion of life-threatening causes. Patients with life threatening etiologies for chest pain may appear deceptively well, manifesting neither vital sign nor physical examination abnormalities. (See 'Life-threatening conditions' above.)

  Causes of chest pain that pose an immediate threat to life include:

- Acute coronary syndrome (ACS)
- Acute aortic dissection
- Pulmonary embolism
- Tension pneumothorax
- Pericardial tamponade
- Mediastinitis (eg, Esophageal rupture) (see 'Differential diagnosis' above)

- **History** – Significant overlap exists among the symptoms experienced by patients with life-threatening and common causes of chest pain. Emergency clinicians must guard against premature diagnostic closure based upon the history or results of nonspecific diagnostic testing. So-called "atypical" presentations occur often; misinterpretation of such presentations increases the risk for adverse outcomes. (See 'History' above.)

- **Physical examination** – The physical examination is often not helpful in distinguishing ACS from noncardiac chest pain. In some instances, physical findings suggest a specific noncardiac diagnosis. Patients with an immediately life-threatening cause tend to appear anxious and distressed and may be diaphoretic and dyspneic. (See 'Physical examination' above.)

- **Diagnostic testing** – Commonly obtained tests and studies used to help differentiate the cause of chest pain are described in the text. These often include electrocardiogram (ECG), plain chest radiograph, bedside ultrasound, and blood tests (eg, cardiac biomarkers, D-dimer, complete blood count, B-type natriuretic peptide, blood gas). (See 'Ancillary studies' above.)

- **Management and disposition** – A basic approach to the diagnosis and management of patients with chest pain is provided in the text, along with links to more detailed discussions. (See 'Approach to diagnosis' above and 'Management' above and 'Disposition' above.)

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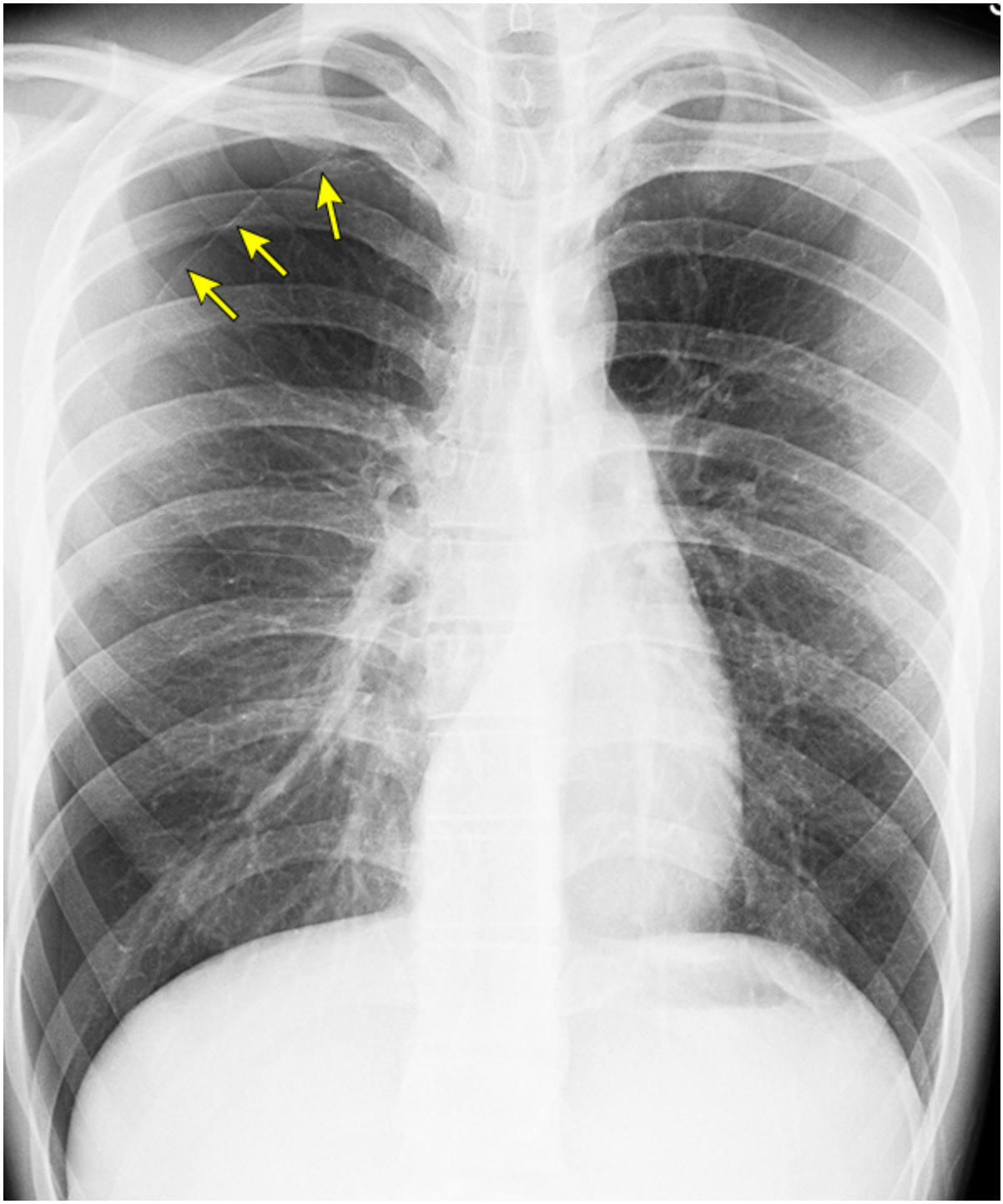
Topic 288 Version 31.0

GRAPHICS

Chest radiograph of spontaneous pneumothorax



Chest radiograph of a 20-year-old male with small spontaneous right pneumothorax demonstrates the characteristic convex right white visceral pleural line (arrows).

Courtesy of Nestor L Muller, MD, PhD.

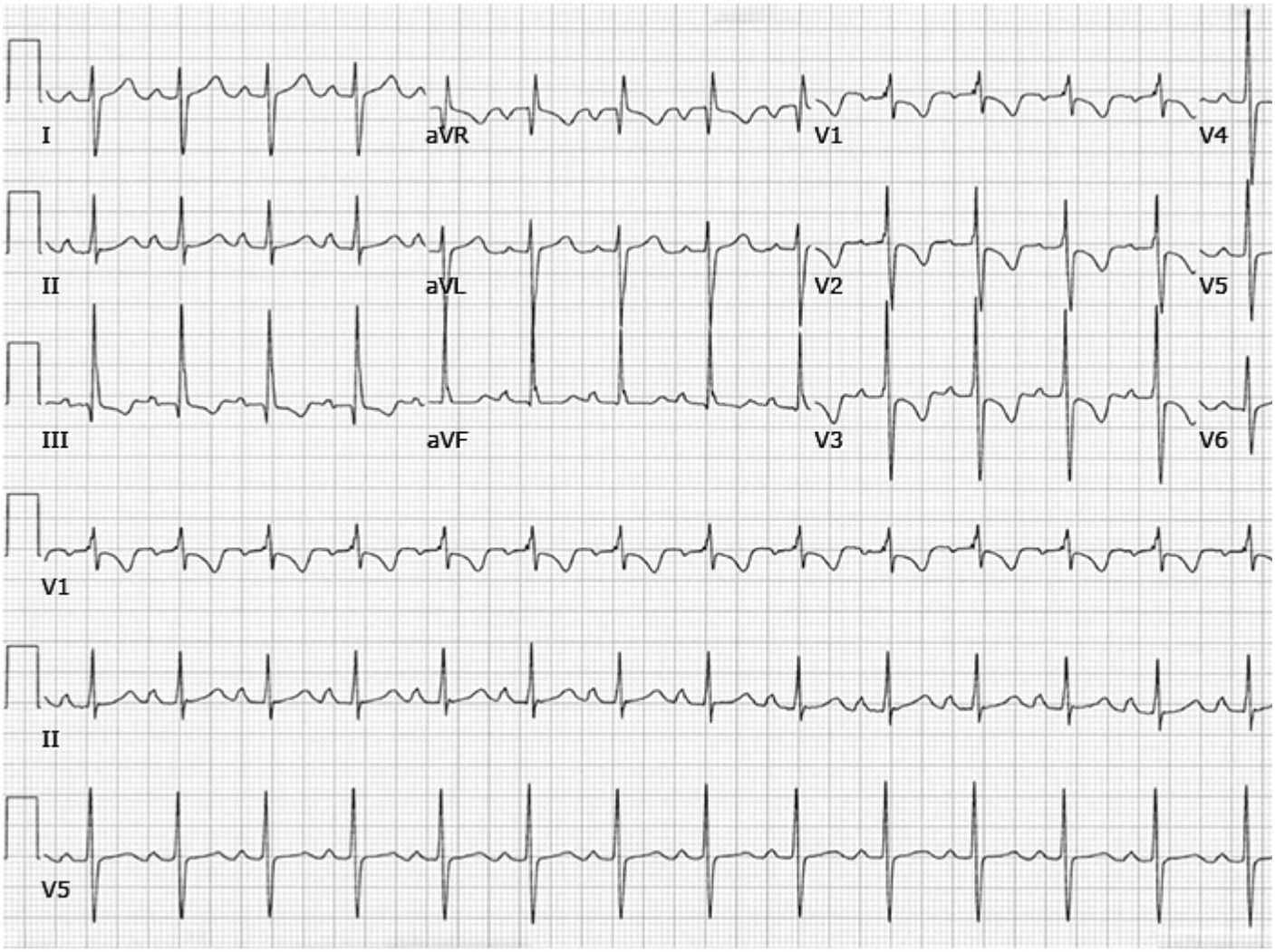
Graphic 139477 Version 1.0

Presentations of aortic dissection based on affected structures

Clinical findings	Artery or structure involved
Aortic insufficiency or heart failure	Aortic valve
Myocardial infarction	Coronary artery (often right)
Cardiac tamponade	Pericardium
Hemothorax	Thorax
Horner syndrome (ptosis, miosis, anhidrosis)	Superior cervical sympathetic ganglion
Stroke or syncope	Brachiocephalic, common carotid, or left subclavian arteries
Upper extremity pulselessness, hypotension pain	Subclavian artery
Paraplegia	Intercostal arteries (give off spinal and vertebral arteries
Back or flank pain; renal failure	Renal artery
Abdominal pain; mesenteric ischemia	Celiac or mesenteric arteries
Lower extremity pain, pulselessness, weakness	Common iliac artery

Graphic 77441 Version 4.0

ECG showing S1Q3T3 and right heart strain

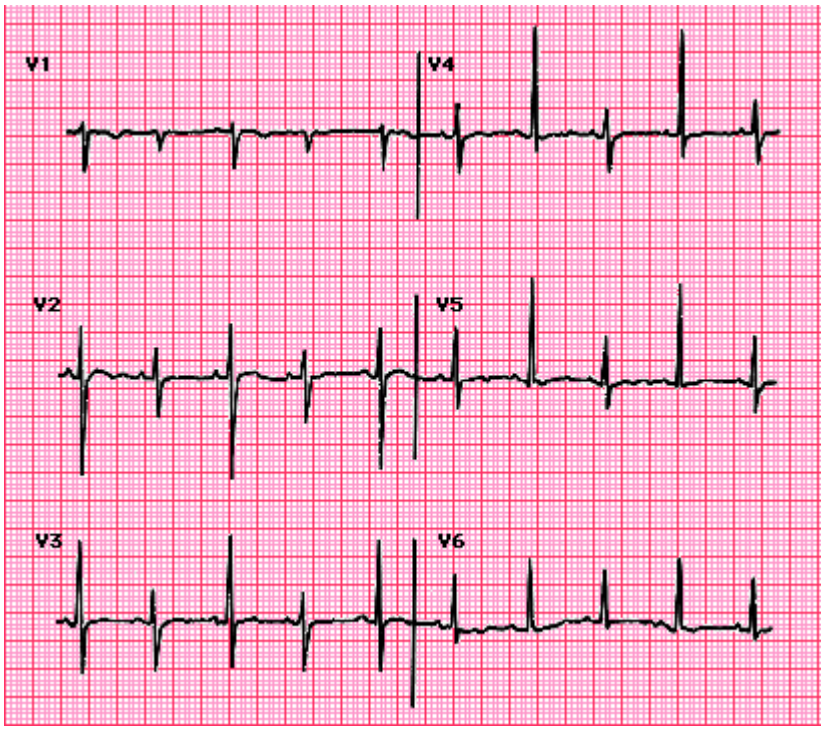


The electrocardiogram above shows an S1 Q3 T3 pattern and inverted T waves in leads V1-V4, which are indicative of right heart strain.

ECG: electrocardiogram.

Graphic 126265 Version 2.0

Electrical alternans



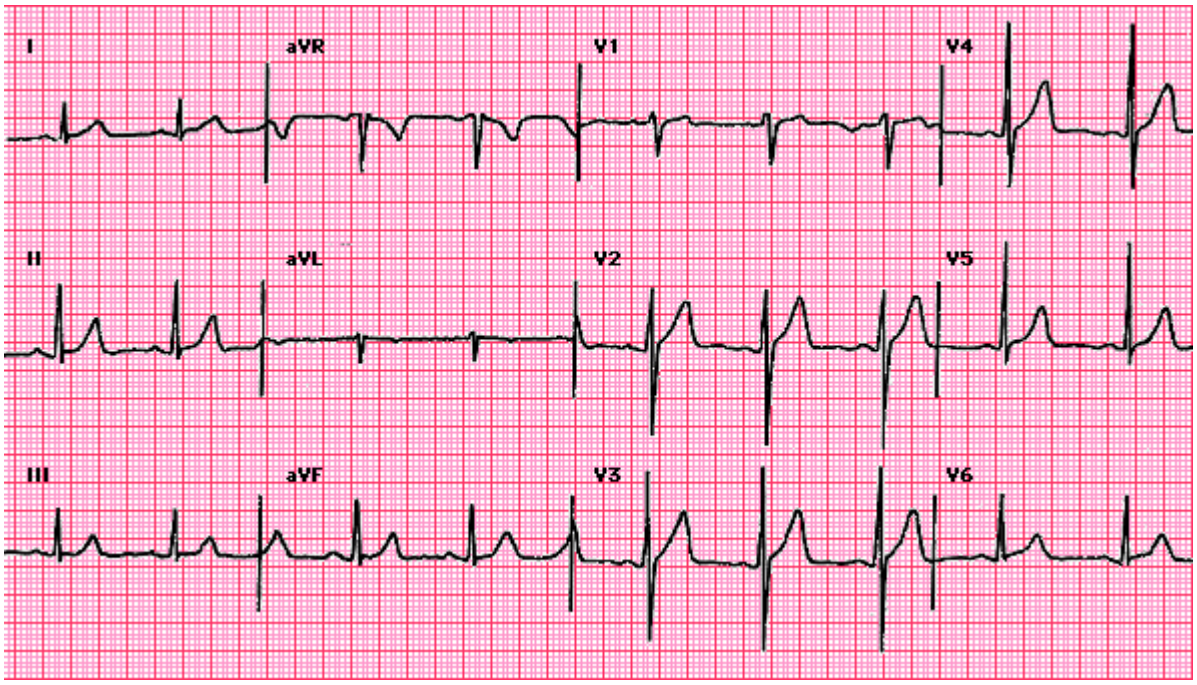
Sinus tachycardia with electrical alternans which is characterized by beat-to-beat alternation in the QRS appearance (best seen in leads V2 to V4). These findings are strongly suggestive of pericardial effusion, usually with cardiac tamponade. The alternating ECG pattern is related to back-and-forth swinging motion of the heart in the pericardial fluid.

ECG: electrocardiogram.

Courtesy of Ary Goldberger, MD.

Graphic 72525 Version 5.0

Normal ECG

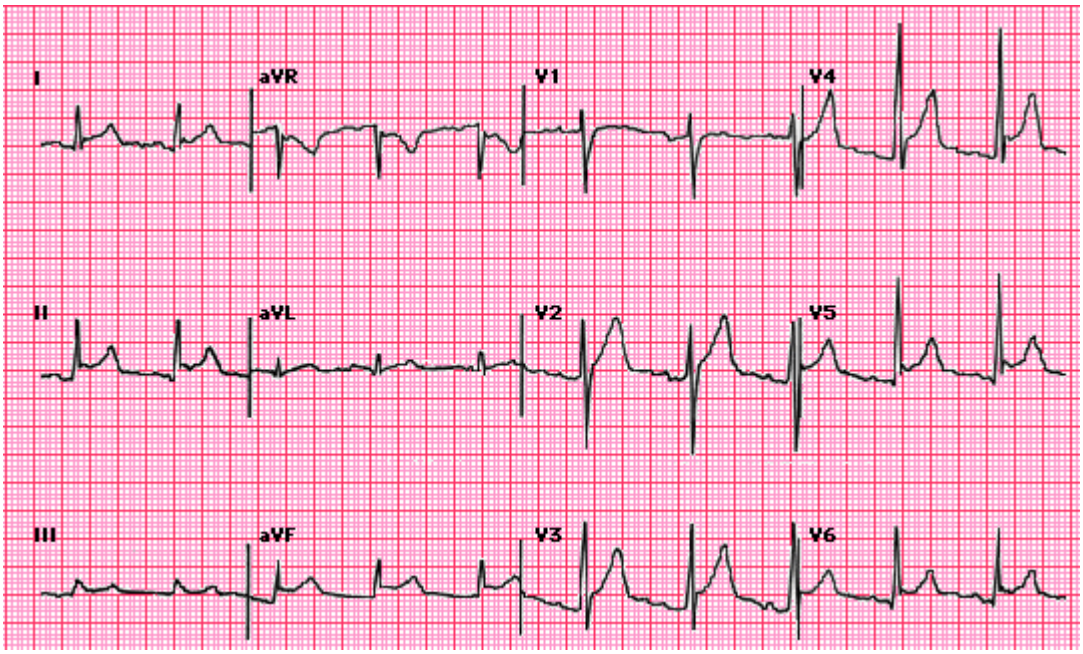


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

Electrocardiogram (ECG) in pericarditis

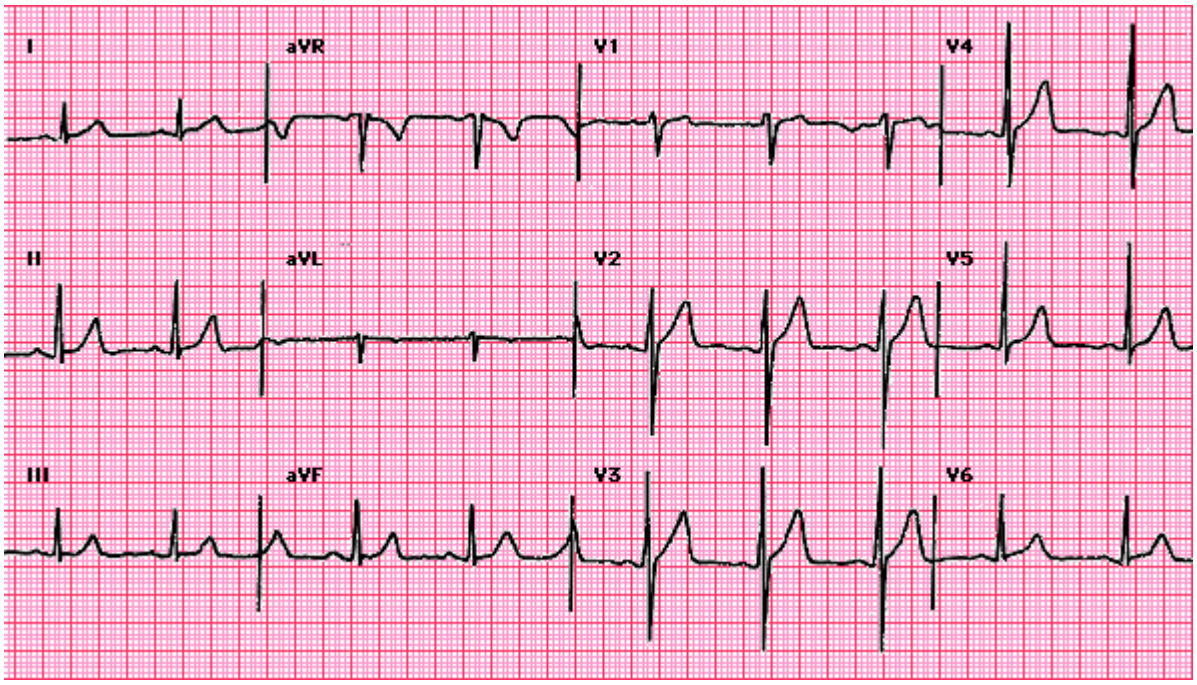


Electrocardiogram in acute pericarditis showing diffuse upsloping (concave up) ST-segment elevations seen best here in leads II, III, aVF, and V2 to V6. There is also subtle PR-segment deviation (positive in aVR, negative in most other leads). ST-segment elevation is due to a ventricular current of injury associated with epicardial inflammation; similarly, the PR-segment changes are due to an atrial current of injury, which, in pericarditis, typically displaces the PR segment upward in lead aVR and downward in most other leads.

Courtesy of Ary Goldberger, MD.

Graphic 77572 Version 5.0

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

Causes of high plasma D-dimer

Condition	Mechanism
Thromboembolism: ▪ Arterial • Myocardial infarction • Stroke • Acute limb ischemia • Intracardiac thrombus ▪ Venous • Deep vein thrombosis • Pulmonary embolism ▪ Disseminated intravascular coagulation (DIC)	Intravascular thrombosis and fibrinolysis
Inflammation: ▪ COVID-19 ▪ Other severe infections ▪ Sepsis ▪ DIC	Activation of the acute inflammatory response and coagulation pathway, intravascular thrombosis and fibrinolysis
Surgery/trauma	Tissue ischemia, tissue necrosis
Liver disease	Reduced clearance of fibrin degradation products
Kidney disease	Multiple, including renal vein thrombosis and nephrotic syndrome
Vascular disorders: ▪ Vascular malformations ▪ Sickle cell disease vaso-occlusion	Intravascular thrombosis and fibrinolysis
Malignancy	Multiple, including vascular abnormalities, cancer procoagulant, and microvascular thrombosis
Thrombolytic therapy	Fibrin breakdown
Pregnancy: ▪ Normal pregnancy ▪ Preeclampsia and eclampsia	Physiologic changes in the coagulation system Microvascular thrombosis and fibrin deposition

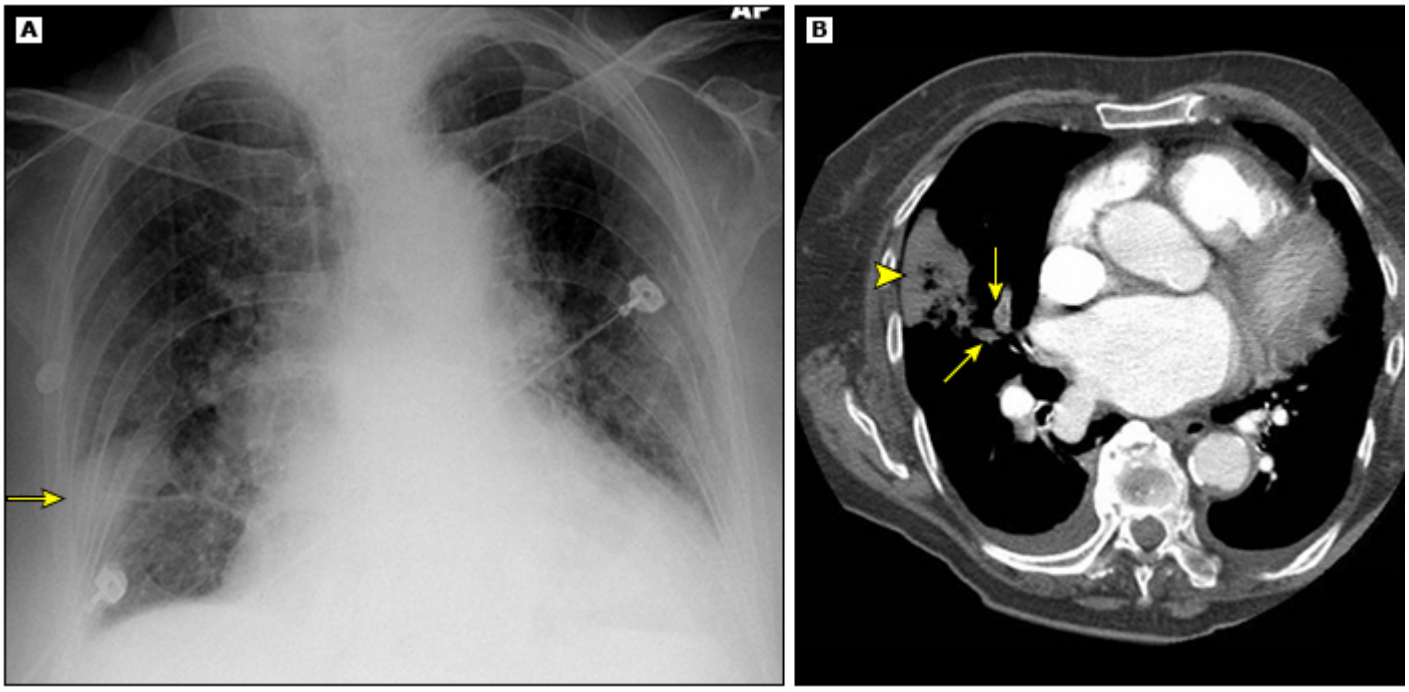
Plasma D-dimer is a product of clot breakdown, released upon degradation of polymerized, crosslinked fibrin (if non-crosslinked fibrinogen was degraded, D-monomers would be released). Elevated plasma D-dimer levels indicate that coagulation has been activated, fibrin clot has formed, and clot degradation by plasmin has occurred. There are many causes of elevated D-dimer; identification of the underlying cause requires correlation with other findings, including the clinical picture and other laboratory results. Refer

to UpToDate for further explanation of fibrinogen domain structure and pathophysiology of the disorders listed here.

COVID-19: coronavirus disease 2019; DIC: disseminated intravascular coagulation.

Graphic 60881 Version 5.0

Hamptons hump on radiograph and CT scan

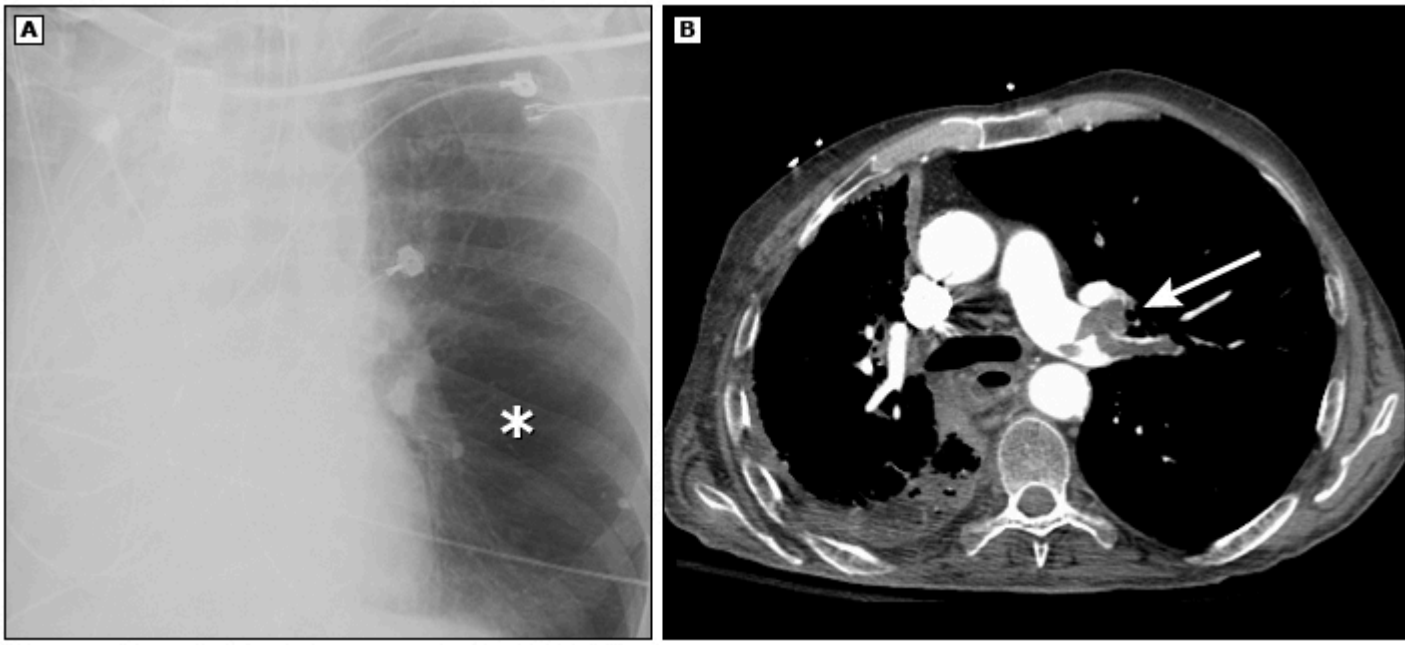


Hamptons hump in a patient with suspected pulmonary embolus. An anterior-posterior chest radiograph (A) shows a wedge-shaped opacity in the lateral segment of the middle lobe (arrow). CT image through the mid-chest shows the corresponding wedge-shaped opacity (arrowhead) and thrombus in the pulmonary arteries (arrows).

CT: computed tomography.

Graphic 97988 Version 3.0

Chest radiograph and CT of the Westermark sign



Westermark sign in a patient with occlusive pulmonary embolism.

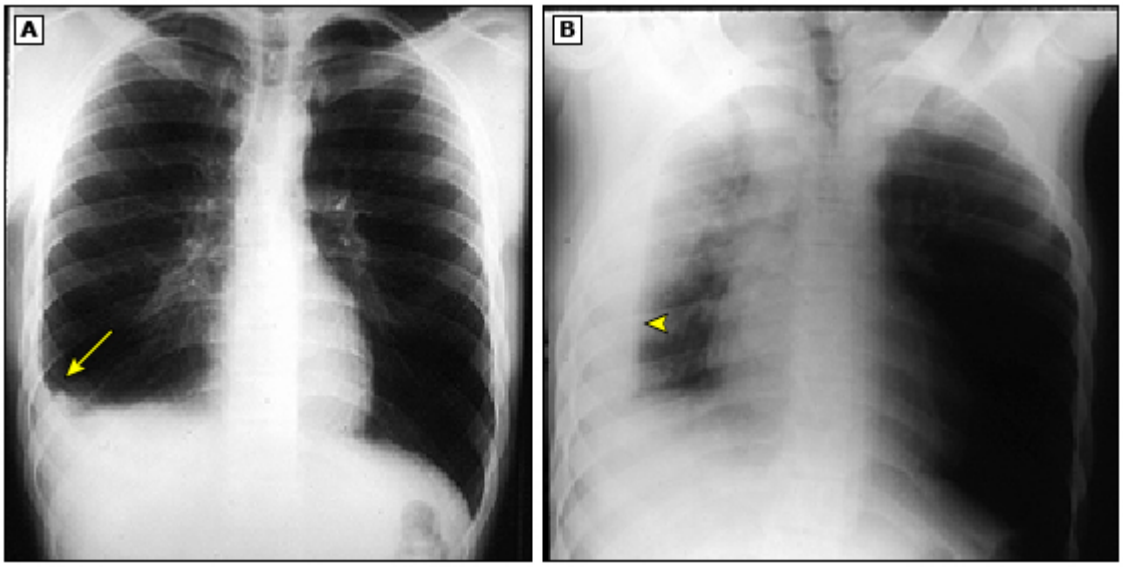
(A) Chest radiograph magnified A-P view shows a region of oligemia in the left lower lung (asterisk).

(B) Chest CT shows a large thrombus in the left main pulmonary artery (arrow).

A-P: anteroposterior; CT: computed tomography.

Graphic 98271 Version 2.0

Free layering pleural effusion

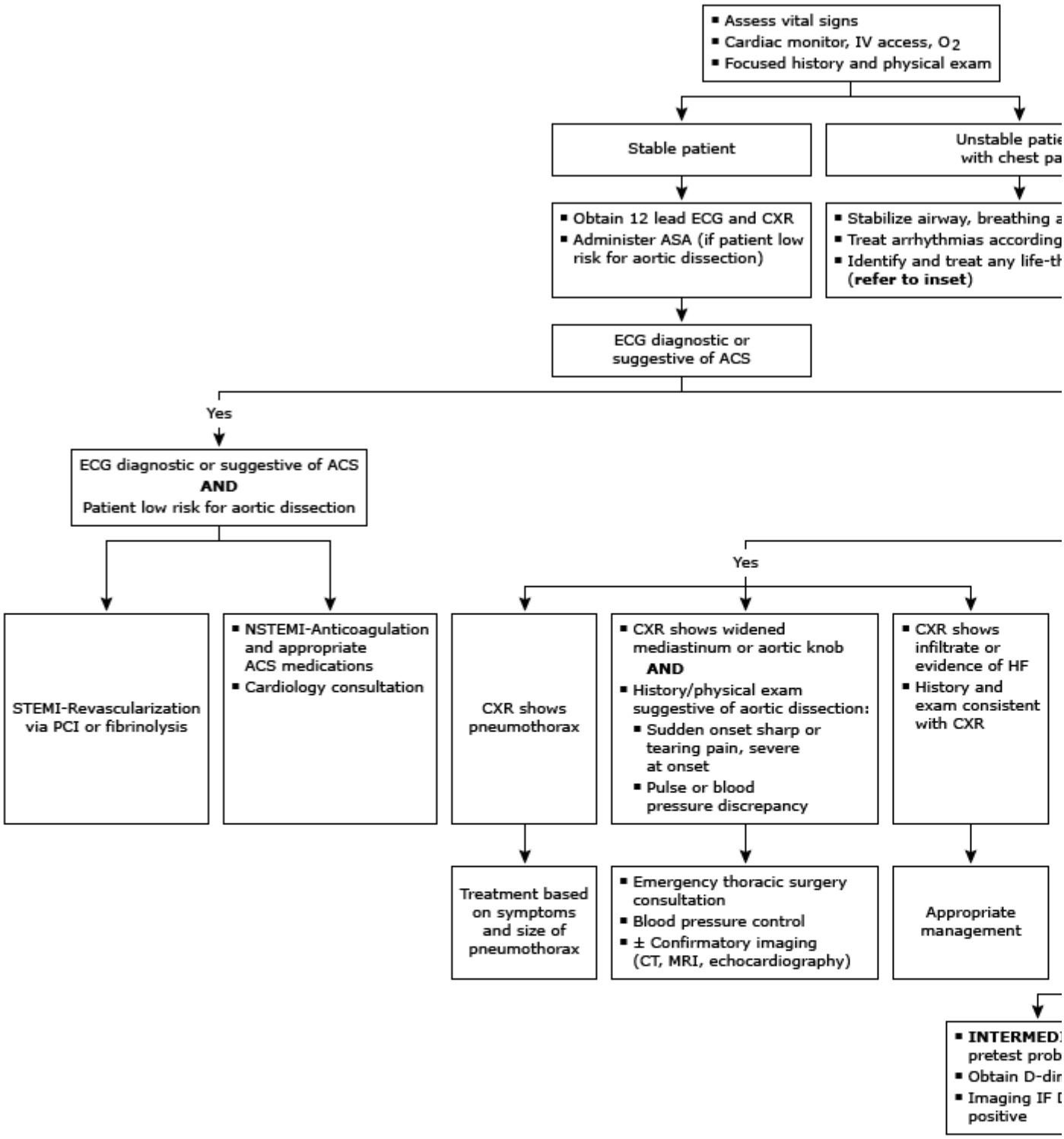


Panel A shows blunting of the right costophrenic sulcus (arrow) on an upright chest radiograph due to the presence of a pleural effusion. Panel B shows a right lateral decubitus radiograph from the same patient, and reveals layering of pleural effusion (arrowhead). Effusions thicker than 1 cm on decubitus views are usually large enough for sampling with thoracentesis.

Courtesy of Steven E Weinberger, MD.

Graphic 51660 Version 3.0

Emergency department approach to chest pain


▪ Assess vital signs
▪ Cardiac monitor, IV access, O_2
▪ Focused history and physical exam
Stable patient
Unstable patie
with chest pa
▪ Obtain 12 lead ECG and CXR
▪ Administer ASA (if patient low risk for aortic dissection)
▪ Stabilize airway, breathing a
▪ Treat arrhythmias according
▪ Identify and treat any life-th
(refer to inset)
ECG diagnostic or suggestive of ACS
Yes
ECG diagnostic or suggestive of ACS
AND
Patient low risk for aortic dissection
Yes
STEMI-Revascularization via PCI or fibrinolysis
▪ NSTEMI-Anticoagulation and appropriate ACS medications
▪ Cardiology consultation
CXR shows pneumothorax
▪ CXR shows widened mediastinum or aortic knob
AND
▪ History/physical exam suggestive of aortic dissection:
▪ Sudden onset sharp or tearing pain, severe at onset
▪ Pulse or blood pressure discrepancy
▪ CXR shows infiltrate or evidence of HF
▪ History and exam consistent with CXR
Treatment based on symptoms and size of pneumothorax
▪ Emergency thoracic surgery consultation
▪ Blood pressure control
▪ ± Confirmatory imaging (CT, MRI, echocardiography)
Appropriate management
▪ INTERMED
pretest prob
▪ Obtain D-di
▪ Imaging IF
positive

ACS: acute coronary syndrome; ASA: aspirin; CXR: chest x-ray; ECG: electrocardiogram; HF: heart failure; PCI: percutaneous coronary intervention.

Graphic 66425 Version 4.0

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

Diagnosis	Historical features	Examination findings	Electrocardiogram	Chest radiogra
Acute coronary syndrome	▪ 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 ΔMS alone	▪ Nonspecific ▪ May detect signs of HF	▪ 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 V_1 and V_2 strongly suggests posterior AMI	▪ Nonspecific ▪ May show evide HF
Aortic dissection	▪ Sudden onset of sharp, tearing, or ripping pain ▪ Maximal severity at onset ▪ Most often begins in chest,	▪ Absent upper extremity or carotid pulse is suggestive ▪ Discrepancy in systolic BP >20 mmHg between right and left upper	▪ Ischemic changes in 15% ▪ Nonspecific ST and T changes in 30%	▪ Wide mediastinu loss of normal a knob contour is common (up to ▪ 10% have norma

	can begin in back ▪ Can mimic stroke, ACS, mesenteric ischemia, kidney stone	extremity is suggestive ▪ Up to 30% with neurologic findings ▪ Findings vary with arteries affected		
Pulmonary embolism	▪ Many possible presentations, including pleuritic pain and painless dyspnea ▪ Often sudden onset ▪ Dyspnea often dominant feature	▪ No finding is sensitive or specific ▪ Extremity exam generally normal ▪ Lung exam generally nonspecific; focal wheezing may be present; tachypnea is common	▪ Usually abnormal but nonspecific ▪ Signs of right heart strain suggestive (eg, RAD, RBBB, RAE)	▪ Great majority a normal ▪ May show atelec elevated hemidiaphragm, pleural effusion
Tension pneumothorax	▪ Often sudden onset ▪ Initial pain often sharp and pleuritic ▪ Dyspnea often dominant feature	▪ Ipsilateral diminished or absent breath sounds ▪ Subcutaneous emphysema is uncommon		▪ Demonstrates ai pleural space

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 enlar heart
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 h some abnormali pneumomediast 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.

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

Wells criteria and modified Wells criteria: Clinical assessment for pulmonary embolism

▪ Clinical symptoms of DVT (leg swelling, pain with palpation)	3.0
▪ Other diagnosis less likely than pulmonary embolism	3.0
▪ Heart rate >100	1.5
▪ Immobilization (≥3 days) or surgery in the previous four weeks	1.5
▪ Previous DVT/PE	1.5
▪ Hemoptysis	1.0
▪ Malignancy	1.0
Probability	**Score**
Traditional clinical probability assessment (Wells criteria)	
High	>6.0
Moderate	2.0 to 6.0
Low	<2.0
Simplified clinical probability assessment (Modified Wells criteria)	
PE likely	>4.0
PE unlikely	≤4.0

DVT: deep vein thrombosis; PE: pulmonary embolism.

Data from van Belle A, Buller HR, Huisman MV, et al. Effectiveness of managing suspected pulmonary embolism using an algorithm combining clinical probability, D-dimer testing, and computed tomography. JAMA 2006; 295:172.

Graphic 54767 Version 4.0

The pulmonary embolism rule out criteria (PERC rule)*[1]

Age <50 years
Heart rate <100 bpm
Oxyhemoglobin saturation ≥95%
No hemoptysis
No estrogen use
No prior DVT or PE
No unilateral leg swelling
No surgery/trauma requiring hospitalization within the prior four weeks

DVT: deep venous thrombosis; PE: pulmonary embolus; bpm: beats per minute.

* This rule is only valid in patients with a low clinical probability of PE (gestalt estimate <15 percent). In patients with a low probability of PE who fullfil all eight criteria, the likelihood of PE is low and no further testing is required. All other patients should be considered for further testing with sensitive D-dimer or imaging.

Reference:

1. *Kline JA, Courtney DM, Kabrhel C, et al. Prospective multicenter evaluation of the pulmonary embolism rule-out criteria. J Thromb Haemost 2008; 6:772.*

Graphic 94941 Version 2.0

Acute aortic dissection: Rapid overview of emergency management

Treatment of acute aortic dissection depends on the type/location. **Aortic dissection involving the ascending aorta is a cardiac surgical emergency.** Aortic dissection limited to the descending thoracic and/or the abdominal aorta can often be managed medically, unless there is evidence of end-organ ischemia, progression, or rupture.

Clinical features and evaluation

Acute onset of severe, sharp, or knife-like pain in the anterior chest, with radiation to the neck, back, or abdomen. Pain may be migratory.

Assess risk factors for TAAD*.

Palpate carotid, subclavian, and femoral pulses; note any significant differences between sides. Obtain blood pressure in both arms.

Auscultate for diastolic cardiac murmur of aortic regurgitation; assess for tamponade (muffled heart sounds, jugular venous distention, pulsus paradoxus).

Evaluate for signs of ischemic stroke, spinal cord ischemia, ischemic neuropathy, hypoxic encephalopathy.

Findings suggesting involvement of the **ascending aorta** include: back pain, anterior chest pain, hemodynamic instability, diastolic cardiac murmur, tamponade, syncope or stroke (persistent or transient¶; right hemispheric stroke is most common, but bilateral can occur), Horner syndrome (typically partial with ptosis/miosis), weak or absent carotid or subclavian pulse, upper extremity pain/paresthesia/motor deficit.

Findings suggesting involvement of the **descending aorta** include back pain, chest pain, abdominal pain, weak or absent femoral pulses, lower extremity pain/paresthesia/motor deficit, acute paraplegia.

Findings on initial studies

Obtain ECG. Look for signs of ACS; extension of type A dissection to coronary ostia can cause coronary ischemia (right coronary artery most commonly affected).

Obtain D-dimer, CBC, basic electrolytes, LDH, cardiac markers, coagulation parameters, and type and crossmatch. D-dimer <500 ng/dL is less likely to be aortic dissection.

Chest radiograph: Widened mediastinum and/or unexplained pleural effusion are consistent with dissection, particularly if unilateral.

Vascular imaging

For hemodynamically stable patient without suspicion for ascending aortic involvement: Obtain thoraci CT angiography or MR angiography, depending upon resources and speed of acquisition. Dissection is confirmed by presence of intimal flap separating true and false lumen. If these are not readily available or there is a contraindication, obtain transesophageal echocardiogram.

For hemodynamically unstable patient or for strong suspicion of ascending aortic involvement: Obtain transesophageal echocardiogram. If not immediately available, obtain CT angiography. Transthoracic

echocardiography may help identify complications of ascending aortic dissection (eg, aortic valve regurgitation, hemopericardium, inferior ischemia) but is not sensitive for identification of dissection.
Management
Place two large bore IVs; monitor heart rate and blood pressure continuously, preferably using an arterial line.
Control heart rate and blood pressure[Δ]. Maintain heart rate <60 BPM and systolic blood pressure between 100 and 120 mmHg.
Administer esmolol (500 mcg/kg IV loading dose, then infuse at 25 to 50 mcg/kg per minute; titrate to maximum dose of 300 mcg/kg per minute) or labetalol (20 mg IV initially, followed by either 20 to 80 mg IV boluses every 10 minutes to a maximal dose of 300 mg, or an infusion of 0.5 to 2 mg/minute IV). If beta blockers are not tolerated, alternatives are verapamil or diltiazem.
Once heart rate is consistently <60 BPM, give vasodilator therapy. If the systolic blood pressure remains above 120 mmHg, initiate nitroprusside infusion (0.25 to 0.5 mcg/kg per minute titrated to a maximum of 10 mcg/kg per minute) or nicardipine infusion (5 mg/hour increasing every 5 minutes by 2.5 mg/hour to a maximum of 15 mg/hour). Vasodilator therapy (eg, nitroprusside, nicardipine) should **not** be used without first controlling heart rate with beta blockade.
Give IV opioids for analgesia (eg, fentanyl).
Place bladder (Foley) catheter to assess urine output and kidney perfusion.
Surgical consultation
Obtain immediate surgical consultation (cardiothoracic surgery, vascular surgery) as soon as the diagnosis is strongly suspected (particularly for involvement of the ascending aorta) or confirmed.
Aortic dissection involving the ascending aorta is a cardiac surgical emergency. Transesophageal echocardiography should be routinely performed in the operating room to assess aortic valve function, left ventricular function, aortic root and ascending aortic diameter, and evidence of hemopericardium/tamponade.
Aortic dissection involving only the descending thoracic aorta or abdominal aorta **and** with evidence of malperfusion is treated with urgent aortic stent-grafting or surgery.
Aortic dissection involving only the descending thoracic aorta or abdominal aorta **without** evidence for ischemia is admitted to the ICU for medical management of hemodynamics and serial aortic imaging.
If appropriate surgical services[◇] are not available, initiate emergency transfer to nearest available cardiovascular center.

TAAD: thoracic aortic aneurysm/dissection; ECG: electrocardiogram; ACS: acute coronary syndrome; CBC: complete blood count; LDH: lactate dehydrogenase; CT: computed tomography; MR: magnetic resonance; IV: intravenous; BPM: beats per minute; ICU: intensive care unit; AAA: abdominal aortic aneurysm.

* Known history of TAAD, AAA, aortic intramural hematoma, penetrating aortic ulcer, family history of TAAD or AAA, recent aortic instrumentation, known bicuspid aortic valve, known aortic coarctation,

known syndrome associated with TAAD (eg, Marfan, vascular Ehlers-Danlos, Loeys-Dietz, or Turner syndromes).

¶ Amaurosis fugax has been reported.

Δ Patients should be admitted to an intensive care unit as rapidly as possible. Intravenous short-acting agents for control of heart rate and blood pressure should be administered immediately by clinicians who are trained and experienced in their titration using continuous noninvasive electronic monitoring of blood pressure, heart rate, and ECG. The use of non-selective beta blockers alone in patients with acute cocaine intoxication may lead to unopposed alpha stimulation worsening hypertension.

◇ Surgical services should include cardiothoracic/vascular surgery by surgeons experienced in the treatment of aortic dissection, equipment and technical support for cardiopulmonary bypass, and endovascular stent-graft capability.

Graphic 111189 Version 8.0