Evaluation of the adult with chest pain in the emergency department

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INTRODUCTION — Chest pain accounts for approximately six 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 elsewhere. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department" and "Overview of acute pulmonary embolism in adults" and "Management of aortic dissection" and "Primary spontaneous pneumothorax in adults" and "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:

- Acute coronary syndrome
- 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 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 unstable angina and non-ST elevation myocardial infarction".)

  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".)

- 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. 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 and ischemic injury to areas perfused by those vessels. In approximately 13 percent of cases, no intimal tear is identified. Such patients have an acute aortic syndrome likely caused by bleeding of the vasa vasorum with intramural hematoma formation in the wall of the aorta. The clinical picture is similar to classic aortic dissection. (See "Clinical features and diagnosis of acute aortic dissection".)

- 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 "Overview 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). 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 "Primary spontaneous pneumothorax in adults".)

- 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" and "Deep neck space infections", section on 'Complications'.)

- 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 emergent 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 elsewhere. (See "Differential diagnosis of chest pain in adults".)

- 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 mitral valve prolapse and 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 "Evaluation of acute decompensated heart failure" and "Stable ischemic heart disease: Overview of care" and "Clinical manifestations and diagnosis of aortic stenosis in adults" and "Mitral valve prolapse syndrome" and "Clinical manifestations and diagnosis of infective endocarditis" and "Clinical presentation and diagnostic evaluation of acute pericarditis" 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 "Diagnostic approach to community-acquired pneumonia in adults" and "Treatment of acute exacerbations of asthma in adults" and "Cor pulmonale" and "Diagnostic evaluation of a pleural effusion in adults: Initial testing".)

- Gastrointestinal causes – Gastroesophageal reflux and esophageal spasm, rupture (Boerhaave's 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 "Pharmacotherapy for panic disorder in adults".)

- 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's disease, polyarteritis nodosa, and Takayasu's 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, 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 acute coronary syndrome, aortic dissection, and pulmonary embolus, including:

- Comorbidities: hypertension, diabetes mellitus, peripheral vascular disease, malignancy
- Recent events: trauma, major surgery or medical procedures (eg, endoscopy), 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 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 symptoms'.)

An 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.

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 the risk equivalents and established risk factors for cardiovascular disease" and "Cocaine: Acute intoxication".)

The diagnosis of **aortic dissection** must be considered in patients with inherited connective tissue diseases and other diseases that weaken the structural architecture of the aortic wall. Patients younger than 40 years of age with Marfan's syndrome, bicuspid aortic valve, cocaine use, or pregnancy are at risk for aortic dissection. Other factors that predispose to aortic dissection include: a history of previous aortic surgery and recent cardiac surgery or catheterization. Aortic dissection occurs most often in patients with systemic hypertension and advanced age, but so do many other causes of chest pain. (See "Clinical features and diagnosis of acute aortic dissection", section on 'High-risk conditions' and "Aortic intramural hematoma".)

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 oral contraceptives or chemotherapeutic agents that raise serum levels of estrogen or progestin also confers increased risk. A significant number of patients with PE may have no identifiable risk factor at the time of diagnosis, but subsequent testing reveals a predisposition for venous thrombosis (eg, Factor V Leiden mutation). (See "Overview 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. jiroveci) pneumonia. Young females with endometriosis may experience menses-related pneumothoraces, also referred to as catamenial pneumothorax, if pleural involvement exists [20]. Activities, such as SCUBA diving, can precipitate a spontaneous pneumothorax and air travel may precipitate recurrence in patients with an incompletely healed pneumothorax [21]. (See "Primary spontaneous pneumothorax in adults", section on 'Risk factors' and "Pneumothorax in HIV-infected patients" and "Complications of SCUBA diving" and "Pneumothorax and air travel".)

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 [22]. Other studies confirm this data [23]. 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 aortic dissection may be absent or suggestive of other entities including stroke, myocardial infarction, and intraabdominal catastrophe, depending on the affected arteries. Discrepancies in pulses or blood pressure are notable findings. 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.

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 rales, with or without an S3 gallop, is associated with left ventricular dysfunction and left-sided heart failure, possibly due to ACS. Jugular venous distention, 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 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 [24-28]. The ECG should be repeated as frequently as every 10 minutes 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 elsewhere. (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) 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 [29]. 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 adult with suspected acute pulmonary embolism", section on 'Electrocardiography'.)

- Pericardial tamponade and pericarditis – ECG findings suggestive of tamponade include low voltage and electrical alternans. 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. These findings are typically more diffuse than is found in patients with focal

anatomic changes from myocardial ischemia. (See "Clinical presentation and diagnostic evaluation of acute pericarditis", section on 'Electrocardiogram'.)

- 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 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. Creatine kinase MB (CK-MB) isoform levels rise to twice normal at six hours and peak within approximately 24 hours.

  In the majority of cases, a single set of negative cardiac biomarkers is **NOT** sufficient to rule out myocardial infarction. Patients with skeletal muscle disease, acute muscle exertion, chronic kidney disease, and cocaine use often have elevated levels of CK-MB in the absence of infarction. Troponin I has a higher specificity for myocardial necrosis than CK-MB in these patients. Negative initial cardiac biomarker determinations are not used to determine discharge. (See "Troponins as biomarkers of cardiac injury" and "Elevated cardiac troponin concentration in the absence of an acute coronary syndrome" and "Evaluation of 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.

  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 adult with suspected acute pulmonary embolism", section on 'Diagnostic algorithms for hemodynamically stable patients'.)

  A d-dimer may be useful to rule out suspected aortic dissection. (See "Aortic intramural hematoma" and "Clinical features and diagnosis of acute aortic dissection", section on 'D-dimer'.)

- 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.

- B-type natriuretic peptide (BNP) – A number of conditions can elevate the BNP, but levels above 100 pg/mL have a 90 percent sensitivity for acute heart failure (HF) and levels below 50 pg/mL have a 96 percent negative predictive value for HF [30]. When used in conjunction with other clinical information, BNP can help to identify or exclude acute HF as the cause of dyspnea and chest pain. (See "Evaluation of the patient with suspected heart failure".)

- 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 adult with suspected acute pulmonary embolism", section on 'Laboratory tests'.)

- Other tests – Several biomarkers, including smooth muscle myosin heavy chain, 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 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 **aortic dissection** will have some CXR abnormality [31]. 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 nearly all patients with suspected aortic dissection. (See "Clinical features and diagnosis of acute aortic dissection", section on 'Chest radiograph'.)

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 Hampton's hump) or paucity of vascular markings distal to the site of embolus (Westermark's sign). (See "Clinical presentation, evaluation, and diagnosis of the adult with suspected acute pulmonary embolism", section on 'Diagnostic algorithms for 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. 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 "Diagnostic approach to community-acquired pneumonia in adults", section on 'Radiologic evaluation' and "Evaluation of acute decompensated heart failure", section on 'Chest radiography'.)

Other imaging

- 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).

  Availability and institutional expertise determine the modality used. TEE allows for rapid beside diagnosis of the hemodynamically unstable patient but requires an experienced echocardiographer. CT angiography 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. 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. Aortography was once the gold standard for diagnosis of aortic dissection but is now seldom used. (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. 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 adult with suspected acute pulmonary embolism", section on 'Initial approach'.)

- Computed tomography (CT) – Computed tomography (CT) technology continues to evolve. Studies for pulmonary embolism or 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. 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 aortic dissection or pulmonary embolism scans are not known. Additionally, triple rule out scans have been associated with increased radiation exposure [32]. (See "Noninvasive coronary imaging with cardiac computed tomography and cardiovascular magnetic resonance".)

- 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 tests'.)

- 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. Bedside cardiac 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 infarcts. (See "Transthoracic echocardiography for the evaluation of chest pain in the emergency department".)

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 2). (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 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 and formal risk assessment (eg, the TIMI score) are cornerstones of management [33,34]. 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 ED 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 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'.)

- 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 [31].

      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 variables are present approximately 7 percent of patients have aortic dissection. 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 emergent 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, the Charlotte criteria, the revised Geneva score, and the PERC rule. 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 "Overview of acute pulmonary embolism in adults", section on 'Clinical presentation, evaluation, and diagnosis' and "Clinical presentation, evaluation, and diagnosis of the adult with suspected acute pulmonary embolism" and "Overview of the 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 "Primary spontaneous pneumothorax in adults".)

- 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 emergent revascularization via percutaneous intervention or fibrinolysis. The management of ACS is discussed in detail elsewhere. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department", section on 'Immediate ED 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 unstable angina and non-ST elevation myocardial infarction".)

- Aortic dissection – Emergent treatment for a suspected aortic dissection involves blood pressure and heart rate control to reduce shearing forces and intensity of pulsatile cardiac flow. 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. Emergent imaging and cardiothoracic surgery consultation is obtained. (See "Management of 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 "Overview of the 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 "Primary spontaneous pneumothorax in adults" and "Placement and management of thoracostomy tubes".)

- Pericardial tamponade – Tamponade with overt hemodynamic compromise requires emergent 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 "Cardiac tamponade", section on 'Treatment'.)

- 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 aortic dissection, pneumothorax, cardiac tamponade, and mediastinitis require admission and appropriate consultation. (See "Management of aortic dissection" and "Primary spontaneous pneumothorax in adults" 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 "Overview of the 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 unstable angina and non-ST elevation myocardial infarction".)

Patients not at low risk for ACS but without known coronary artery disease or obvious signs of myocardial infarction but no clear alternative diagnosis are observed to rule out myocardial ischemia with serial cardiac biomarkers, electrocardiograms (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. 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 [35]. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department" and "Evaluation of patients with chest pain at low or intermediate risk for acute coronary syndrome" and "Noninvasive coronary imaging with cardiac computed tomography and cardiovascular magnetic resonance".)

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 [36]. They can be discharged to home with follow-up.

SUMMARY AND RECOMMENDATIONS

- Chest pain accounts for a large number of emergency department (ED) visits. 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.
- Causes of chest pain that pose an immediate threat to life include:
    - Acute coronary syndrome (ACS)
    - Aortic dissection
    - Pulmonary embolism
    - Tension pneumothorax
    - Pericardial tamponade
    - Mediastinitis (eg, Esophageal rupture) (see 'Differential diagnosis' above)
- Significant overlap exists among the symptoms experienced by patients with life-threatening and common causes of chest pain. Patient descriptions of their symptoms can be helpful in some instances, but emergency clinicians must guard against premature diagnostic closure based upon history. So-called "atypical" presentations occur often; misinterpretation of such presentations increases the risk for adverse outcomes. (See 'History' above.)
- 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 for their chest pain tend to appear anxious and distressed and may be diaphoretic and dyspneic. (See 'Physical examination' above.)
- Commonly obtained tests and studies used to help differentiate the cause of chest pain are described in the text. (See 'Ancillary studies' above.)
- A basic approach to the diagnosis and management of patients with potentially life-threatening causes of 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 15.0

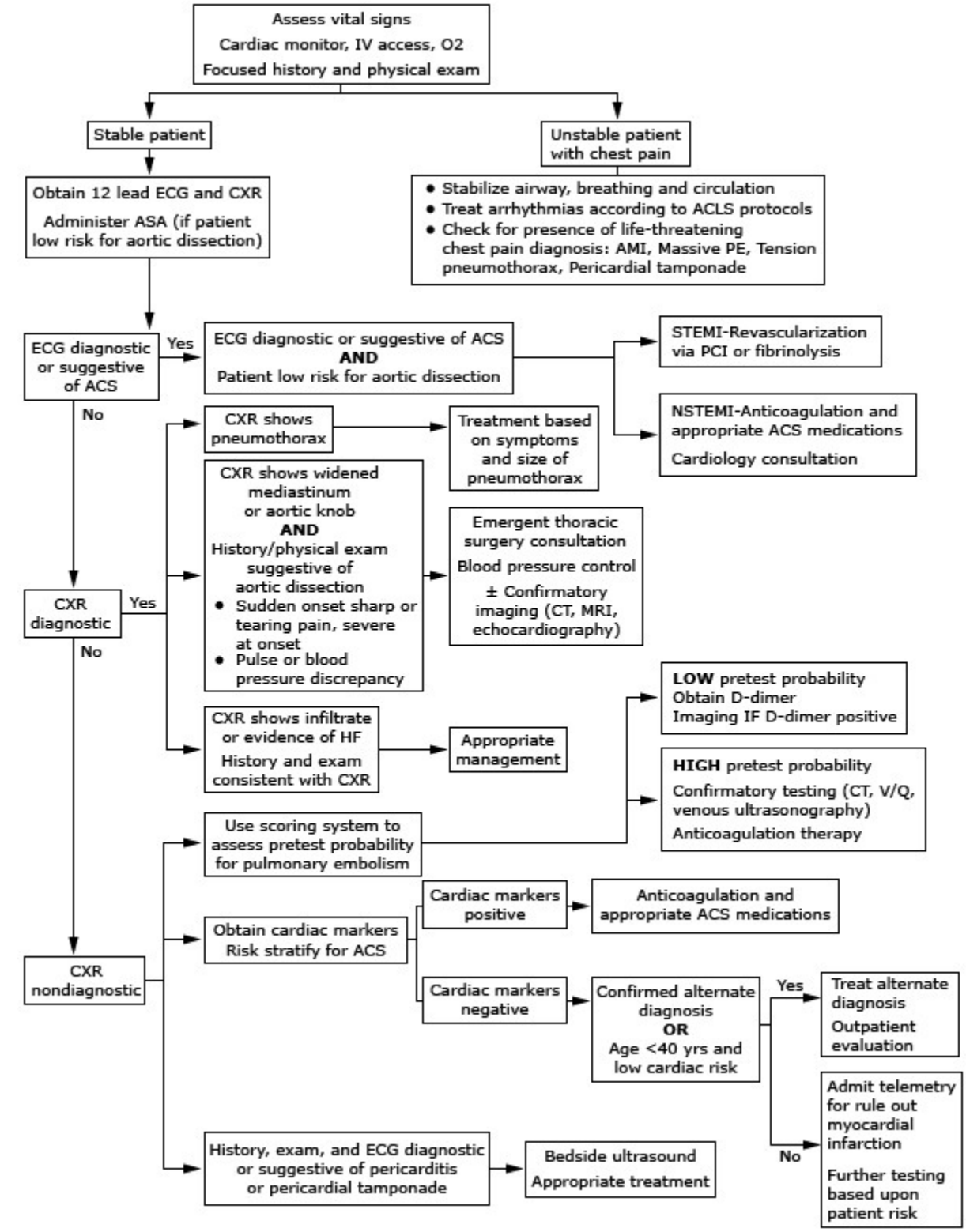
GRAPHICS

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
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)
Lower extremity pain, pulselessness, weakness	Common iliac artery
Abdominal pain; mesenteric ischemia	Celiac or mesenteric arteries
Back or flank pain; renal failure	Renal artery
Horner syndrome (ptosis, miosis, anhidrosis)	Superior cervical sympathetic ganglion

Graphic 77441 Version 3.0

Emergency department approach to chest pain



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

Graphic 66425 Version 3.0

Differentiation of life-threatening causes of chest pain

Diagnosis	Historical features	Examination findings	Electrocardiogram	Chest X-
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 elderly, women, diabetics • Elderly 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 ev of HF
Aortic dissection	• Sudden onset of sharp, tearing, or ripping pain • Maximal severity at onset • Most often begins in chest, can begin in back • Can mimic: stroke, ACS, mesenteric ischemia, kidney stone	• Absent upper extremity or carotid pulse is suggestive • Discrepancy in systolic BP >20 mmHg between right and left upper extremity is suggestive • Up to 30 percent with neurologic findings • Findings vary with arteries affected	• Ischemic changes in 15 percent • Nonspecific ST and T changes in 30 percent	• Wide medias or loss of norm aortic knob co is common (up percent) • 10 percent h normal CXR

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 majori normal • May show: atelectasis, ele hemidiaphragr pleural effusio
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		• Demonstrate 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 enlarged heart
Mediastinitis (esophageal rupture)	• Forceful vomiting often precedes esophageal rupture • Recent upper endoscopy or instrumentation increases risk of perforation • Odontogenic infection is possible cause	• Ill-appearing; shock, fever • May hear (Hamman's) crunch over mediastinum		• Large majori have some abnormality: pneumomedias pleural effusio pneumothorax

		• Coexistent respiratory and gastrointestinal complaints may occur			

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 x-ray; 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.

Graphic 54629 Version 1.0

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