



# Chest Pain: A Clinical Assessment

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■ Major pathologies that produce chest pain  
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Chest pain is one of the most common chief complaints in emergency medicine. During the acute presentation of a patient who has chest pain, chest imaging is invaluable, especially in the initial stabilization of a life-threatening cardiac or pulmonary event. The initial approach to evaluating chest pain includes excluding life-threatening causes, such as aortic dissection, pulmonary embolism (PE), pneumothorax, pneumomediastinum, pericarditis, and esophageal perforation.

The evaluation of an unstable patient who has chest pain or shortness of breath begins with a primary medical survey to evaluate airway, breathing, and circulation. In tandem with this rapid assessment, the emergency physician requests radiographic images of the chest, which provide visualization of the thoracic anatomy. The first image obtained is the anteroposterior chest radiograph, using portable radiography or fixed equipment, depending on the patient's presenting clinical appearance. The initial study is invaluable in providing clinically relevant information that directs the patient's care.

Although technologic advances have improved diagnostic accuracy greatly in recent years, a thorough history and physical examination remain the most important components in the evaluation process. It is imperative to obtain as many details about the pain as possible, including its onset,

location, duration, radiation, quality, and exacerbating and relieving factors. A detailed history sets in motion further diagnostic testing and management decisions.

## Major pathologies that produce chest pain

### *Pneumothorax*

Perfect coupling between the visceral and parietal pleura is required for effective ventilation. Patients who have pneumothorax have gas in the intrapleural space. This abnormality uncouples the visceral and parietal pleura and thus elevates the intrapleural pressure, which affects ventilation, gas exchange, and perfusion.

Pneumothorax commonly is divided into two types: primary spontaneous pneumothorax (PSP), which usually occurs without a precipitating event in patients who have no clinical lung disease, and secondary spontaneous pneumothorax, a complication of underlying lung disease. In actuality, most patients who have PSP have underlying lung disease, most commonly rupture of a subpleural bleb [1]. Iatrogenic pneumothorax is difficult to identify; its incidence is increasing due to the more widespread use of mechanical ventilation and interventional procedures such as central line placement and lung biopsy [2]. When pneumo-

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thorax is suspected, correct interpretation of chest radiographs and knowledge of the benefit of more complex imaging techniques are essential. The causes of spontaneous and iatrogenic pneumothorax and of pneumomediastinum are summarized in **Box 1**.

The incidence of PSP (age-adjusted) is 7.4 cases per 100,000 persons per year for men and 1.2 cases per 100,000 persons per year for women [3,4]. The incidence of secondary spontaneous pneumothorax (age-adjusted) is 6.3 cases per 100,000 persons per year for men and 2 cases per 100,000 persons per year for women [3,4]. The incidence of iatrogenic pneumothorax is not known, but it probably occurs more often than do primary and secondary spontaneous pneumothoraces combined. Pneumomediastinum occurs in approximately 1 of 10,000 hospital admissions [5].

#### **Box 1: Causes of pneumothorax**

##### **Spontaneous pneumothorax**

- Rupture of subpleural apical emphysematous blebs
- Smoking (increases the risk of a first spontaneous pneumothorax by more than 20-fold in men and by nearly 10-fold in women, compared with the risks in nonsmokers)
- Physical height (taller patients are at risk because alveoli are subjected to a greater mean distending pressure over time, which leads to subpleural bleb formation; because pleural pressure is more negative at the apex of the lung, blebs are more likely to rupture and cause pneumothorax)

##### **Iatrogenic pneumothorax**

- Transthoracic needle aspiration procedures
- Subclavian and supraclavicular needlestick
- Thoracentesis
- Mechanical ventilation (directly related to peak airway pressures)
- Pleural or transbronchial biopsy
- Cardiopulmonary resuscitation
- Tracheostomy

##### **Pneumomediastinum**

- Acute production of high intrathoracic pressures (usual cause)
- Asthma
- Smoking marijuana
- Inhalation of cocaine
- Athletic competition
- Respiratory tract infection
- Parturition
- Emesis
- Severe cough
- Mechanical ventilation

#### **Pathogenesis**

The pathogenesis of the subpleural blebs that cause PSP is related to airway inflammation that results from cigarette smoking. The risk of PSP is related directly to the level of cigarette smoking (number of pack years) [6].

Pneumothorax occurs with increasing frequency in patients who have Marfan's syndrome and homocystinuria [7]. Catamenial pneumothorax may result from thoracic endometriosis and should be considered in menstruating women who present with spontaneous pneumothorax [8].

#### **Clinical presentation**

PSP usually develops at rest. The peak age is the early 20s. The disorder is rare after age 40. Patients usually complain of the sudden onset of dyspnea and pleuritic chest pain. The severity of symptoms is related to the volume of air in the pleural space; dyspnea is more predominant if the pneumothorax is large. In patients who have a large pneumothorax, the physical findings include decreased chest excursion on the affected side, diminished breath sounds, and hyperresonant lungs. Many affected individuals do not seek medical attention for days after symptoms develop. This sequence is important, because the incidence of re-expansion pulmonary edema increases in patients whose chest tubes were placed 3 or more days after the pneumothorax occurred.

Pneumomediastinum usually occurs when intrathoracic pressures become elevated. This elevation may occur with an exacerbation of asthma, coughing, vomiting, childbirth, seizures, and a Valsalva maneuver. Patients usually complain of a sudden onset of chest pain and dyspnea.

#### **Radiographic features**

The main radiographic abnormality that is indicative of pneumothorax is a white visceral pleural line—straight or convex toward the chest wall—which is separated from the parietal pleura by an avascular collection of air. In most cases, no pulmonary vessels are visible beyond the visceral edge.

The size of a pneumothorax is difficult to estimate. The measurement of the distance between the ribs and the visceral pleura can be used to decide whether to perform a tube thoracostomy. If the distance is greater than 3 cm laterally or 4 cm at the apex, a chest tube may be needed to re-expand the lung. A pneumothorax of less than 10% will reabsorb on its own and does not require placement of a chest tube.

In upright patients who have pneumothorax, gas accumulates primarily in an apicolateral location.

As little as 50 mL of pleural gas can be seen on chest film. A lateral chest film with a 1-cm intrapleural space corresponds to a 10% pneumothorax. The size of the pneumothorax is accounted for by the collapsed lung and, to a lesser degree, the expanding chest cage.

The value of expiratory chest radiographs in detecting pneumothoraces has been overstated. In a study of 85 patients who had pneumothoraces and 93 controls, inspiratory and expiratory upright chest radiographs had equal sensitivity for pneumothorax detection [9]. Because expiratory films provide no added benefit, only inspiratory films are recommended as the initial radiograph of choice for pneumothorax.

In the supine patient, approximately 500 mL of pleural air is needed for definitive diagnosis of pneumothorax [10]. The pleural gas accumulates in the subpulmonic location and outlines the anterior pleural reflection, the costophrenic sulcus, and the anterolateral border of the mediastinum. The overall transradiancy of the entire affected hemithorax can be increased on the side of a pneumothorax in the recumbent patient.

Small pneumothoraces can be visualized more easily in the lateral decubitus view. In this position, as little as 5 mL of pleural gas is visible on the nondependent side [10].

### Ultrasound detection of pneumothorax

Bedside ultrasound has become standard in most emergency departments. Focused abdominal sonography for trauma has been integrated into the assessment of the unstable patient. A key element in ultrasound assessment of the chest for pneumothorax is the presence or absence of the “sliding lung sign.” On ultrasound of the normal chest, the lung surface can be seen sliding along the chest wall during inspiration and expiration. In a patient who has pneumothorax, this sign is absent, which suggests that the air adjacent to the chest wall is not contained within the lung.

Ultrasound has proven to be more sensitive than flat anteroposterior chest radiography in the diagnosis of trauma-induced pneumothorax. Ultrasound provides added benefit by allowing sonologists to differentiate between small, medium, and large pneumothoraces, with good agreement with CT results [11].

### Tension pneumothorax

Tension pneumothorax shows a distinct shift of the mediastinum to the contralateral side and flattening or inversion of the ipsilateral hemidiaphragm. This is the result of accumulation of air under pressure in the pleural space. This emergent condi-

tion develops when injured tissue forms a one-way valve and allows air to enter the pleural space but prevents it from escaping naturally. Arising from numerous causes, this condition progresses rapidly to respiratory insufficiency, cardiovascular collapse, and, ultimately, death if it is unrecognized and untreated. Favorable patient outcomes require urgent clinical diagnosis and immediate management.

### Conditions that mimic pneumothorax

Large subpleural bullae can mimic a loculated pneumothorax. In most cases, the medial border of the bulla is concave toward the chest wall, whereas a visceral pleural contour is straight or convex laterally. Skin folds can be differentiated from a pneumothorax by density profile: they form a negative black Mach band instead of the white visceral pleural line. Skin folds increase gradually in opacity, with an abrupt drop-off at the edge, and usually extend beyond the ribcage or stop short of the ribs.

Bilateral pneumothoraces may be seen after heart/lung transplant surgery. Replacement of the heart and lungs leaves an open communication between the two sides of the thorax, which may allow air or fluid to shift from one side to the other. Extensive mediastinal dissection can disrupt the anterior junction line, allowing a unilateral pneumothorax to propagate to the contralateral hemithorax. Placement of a single thoracotomy tube decompresses and evacuates both pleural cavities.

### Treatment of pneumothorax

The treatment of pneumothorax is based on its classification. A tension pneumothorax usually results in cardiopulmonary compromise (shock, bradycardia, hypoxia) and requires immediate needle decompression (thoracocentesis), which can be accomplished by inserting a large-bore (16- or 18-gauge) needle (smaller needles are satisfactory for premature infants, newborns, and infants) through the second or third interspace (near the apex of the lung) in the midclavicular line. *Immediate decompression cannot wait for radiographic confirmation.* Tube thoracostomy may be required after the initial decompression if the pneumothorax reaccumulates.

Management of a simple pneumothorax depends on its size and cause. A clinically stable patient who has a small PSP (occupying <15% of the hemithorax) should be observed in the emergency department for 3 to 6 hours and discharged home if a repeat chest film demonstrates no progression of the pneumothorax. If the patient is to be admitted to the hospital, oxygen therapy may be initiated to hasten absorption of the pneumothorax. Clinically stable patients who have a large PSP should be admitted to the hospital for tube thoracostomy.

## Pneumonia

Despite advances in diagnosis and treatment, pulmonary infections remain a major cause of morbidity and mortality in adult patients. An estimated 4 to 6 million cases of community-acquired pneumonia occur each year [12]. The spectrum of organisms known to cause respiratory infections is broad and constantly increasing as new pathogens are identified and the host immune response is altered by medications or other diseases or responses. In the United States, it is estimated that 1.1 million cases of community-acquired pneumonia require hospitalization each year, at an estimated cost of \$8 billion [13]. Pneumonia is responsible for more than 64 million days of restricted activity from work and is the seventh leading cause of death in this country, with a mortality rate of 22.4 per 100,000 [14].

Among hospital-acquired infections, nosocomial pneumonia has the highest mortality [15]. Moreover, since the beginning of the AIDS epidemic, the lungs are identified increasingly as the source of infection.

Radiology plays a prominent role in the evaluation of pneumonia. Chest radiography is the most commonly used imaging tool in pneumonias, because of its availability and excellent cost/benefit ratio. CT should be used in unresolved cases or when complications of pneumonia are suspected [16]. The main applications of radiology in pneumonia are oriented toward detection, characterization, and follow-up, especially regarding complications.

The classic classification of pneumonias into lobar and bronchial types has been abandoned for a more clinical classification. Thus, bacterial pneumonias are divided into three main groups: community-acquired pneumonia, aspiration pneumonia, and nosocomial pneumonia. The usual pattern of community-acquired pneumonia is that of lobar pneumonia: an air-space consolidation that is limited to one lobe or segment. Nevertheless, the radiographic patterns of community-acquired pneumonia may be variable and often are related to the causative agent.

Aspiration pneumonia generally involves the lower lobes, with bilateral multicentric opacities. The most valuable information is obtained when the chest radiographs are negative and exclude pneumonia.

The criterion standard test for the diagnosis of pneumonia has been the two-view plain chest film. In a study by Courtoy and colleagues [17], however, radiologists who were blinded to culture results could not differentiate viral pneumonia from bacterial pneumonia by reviewing the chest films. Several investigative teams have concluded that no radiologic features exist that can be used

to differentiate between these two major etiologic classes [17,18].

### Radiographic findings that are suggestive of specific etiologic agents

**Pneumococcal pneumonia** Lobar consolidation, involving single or multiple lobes, is the most common radiographic pattern of community-acquired pneumococcal pneumonia in patients who require hospitalization [19]. Pleural effusions also are a common finding in pneumococcal pneumonia. The pattern of consolidation is not influenced by bacteremia or HIV status. The presence of a pneumonic process on radiography correlates with identifiable clinical signs [20,21]. Normal findings on a chest radiograph virtually exclude a diagnosis of pneumonia other than in HIV-infected patients who have *Pneumocystis carinii* or, rarely, in dehydrated, elderly, or neutropenic patients and those who were examined within 24 hours of the onset of symptoms.

**Mycoplasma pneumoniae pneumonia** The radiographic findings in patients who are infected with *Mycoplasma pneumoniae* also are nonspecific, and in some cases, closely resemble those seen in children who have viral infections of the lower respiratory tract. Focal reticulonodular opacification confined to a single lobe is a radiographic pattern that seems to be associated more closely with *Mycoplasma* infection than with other types of pediatric respiratory illnesses. The diagnosis of *Mycoplasma* pneumonia should be considered whenever focal or bilateral reticulonodular opacification is seen. Hazy or ground-glass consolidations occur frequently; however, dense homogeneous consolidations like those seen with bacterial pneumonias are uncommon. Often, atelectasis or transient pseudo-consolidations that produce confluent interstitial shadows are seen. Radiographic findings alone are not sufficient for the definitive diagnosis of *Mycoplasma* pneumonia, but in combination with clinical findings, they can improve the accuracy of diagnosis of this disease significantly.

Guckel and colleagues [22] described three patterns of infiltration in children who have mycoplasma pneumoniae pneumonia, which occur with equal frequency: peribronchial and perivascular interstitial infiltrates, patchy consolidations, and homogeneous acinar consolidations like ground glass. The infiltrates were seen primarily in the lower lungs. Enlargement of the hilar glands was a common finding among the 23 children in their series. Pleural effusion was rare. Diffuse interstitial and bilateral parahilar peribronchial patterns are common in *Mycoplasma* respiratory infections.

**Viral pneumonias** Viral pneumonias are located predominantly in spaces along and around the alveoli. Therefore, these pneumonias appear reticular on plain radiograph and often are bilateral and diffuse in distribution. Associated with thickening of the interlobular septa, viral pneumonias can be associated with Kerly B lines on chest radiograph. Rarely associated with complications or even pleural effusion, viral pneumonia can lead to secondary bacterial pneumonias. In viral pneumonia, four radiographic findings are common: parahilar peribronchial infiltrates, hyperexpansion, segmental or lobar atelectasis, and hilar adenopathy.

***Pneumocystis carinii* pneumonia** Although the radiographic findings in patients who have *Pneumocystis carinii* pneumonia (PCP) vary, most chest radiographs reveal bilateral, symmetric, fine to medium reticular heterogeneous opacities [23–25]. As the disease worsens, the opacities coalesce and eventually appear as a bilateral homogeneous consolidation. Uncommonly, a more coarse reticular pattern or a miliary pattern may be noted [25,26].

Unilateral or unilobar involvement may occur, but the radiographic pattern remains fine reticular opacities. Predominant upper lobe involvement occurs with increased frequency in patients who have used aerosolized pentamidine for prophylaxis [21,27,28]; however, because the use of this form of prophylaxis has waned, the incidence of this appearance has decreased. The presence of hilar or mediastinal adenopathy as well as pleural fluid is rare and suggests another disease process. Usually, these findings are seen in patients who have been taking aerosolized pentamidine and have developed disseminated pneumocystosis [29,30]. Cases of pneumocystosis following the use of dapsone prophylaxis have been reported [31]. Calcified hilar and mediastinal lymph nodes have been reported but are rare [32]. Approximately 10% of patients who have HIV disease and subsequently proven PCP have had normal chest radiographs. In some circumstances, gallium scanning or high-resolution CT may demonstrate lung abnormalities, particularly ground-glass opacities [33]. In many institutions, however, treatment is recommended empirically, without a request for further imaging [34,35].

***Legionella*** Virtually all patients who have Legionnaire's disease have abnormal chest radiography that shows pulmonary infiltrates at the time of clinical presentation. In a few cases of nosocomial disease, fever and respiratory tract symptoms have preceded the appearance of the infiltrate on chest radiography. Findings on chest films are nonspecific and do not distinguish *Legionella* from causes

of pneumonia. Pleural effusion is evident in one third of cases.

In immunosuppressed patients, distinctive, rounded, nodular opacities may be seen; these lesions may expand or cavitate. Pulmonary abscesses may occur in the immunosuppressed host. Infiltration that progresses on chest radiography, despite appropriate antibiotic therapy, is common, and radiographic improvement lags behind clinical improvement by several days. Complete clearing of infiltrates requires 1 to 4 months.

### ***Acute coronary syndrome***

Acute coronary syndrome (ACS) is a spectrum of acute myocardial ischemia that spans acute myocardial infarction (AMI) and unstable angina [36]. Less than 25% of patients who are admitted with suspicion of ACS still have this diagnosis at discharge [37].

### ***History and physical examination***

Chest pain or discomfort is the most common presenting complaint in patients who have ACS [37]. The character and radiation of the pain are important for the diagnosis [38]. The pain usually is described as a deep visceral discomfort and may be difficult to localize to one region of the chest [38]. The character of the pain often is described as pressure, a weight on the chest, tightness, constriction about the throat, or an aching feeling. The pain is not affected by respiration or movement. Beginning gradually and reaching maximum severity after 2 or 3 minutes, the pain lasts for minutes or longer [38]. Physical exertion or emotional stress may be associated with the onset of pain, and the pain may subside with rest [36]. Radiation of the pain to the arm or neck increases the likelihood of AMI [38]. The patient may have associated symptoms of shortness of breath, nausea, vomiting, profound weakness, dizziness, palpitations, and diaphoresis [36].

Chest pain is absent in up to 6.2% of patients who have ACS and 9.8% of patients who have AMI [39]. Atypical presentations are more likely in elderly patients and diabetics, who have an altered ability to localize symptoms [38], and in women and younger people [36]. Atypical symptoms include epigastric pain, indigestion, stabbing chest pain, pleuritic chest pain, chest pain that is reproducible on palpation, and isolated dyspnea [36].

Risk factors for cardiac disease are elicited during the history. Traditional risk factors for coronary artery disease (CAD) include hypertension, hypercholesterolemia, cigarette smoking, diabetes, peripheral vascular disease, family history of CAD, personal history of CAD, male gender, and increasing age [36–38]. These are long-term risk factors for



CAD; the absence of risk factors for CAD should not be used to exclude the diagnosis of ACS [37,38].

The physical examination of a patient in whom ACS is suspected generally is not helpful unless it reveals an alternate diagnosis [37]. Thus, the physical examination should focus on excluding other diagnoses; identifying causes of myocardial ischemia, such as uncontrolled hypertension or thyroid disease; and searching for signs of hemodynamic instability [36]. Caution should be taken in automatically attributing chest pain that is reproducible on examination to musculoskeletal causes, because 11% of cases of partially or fully reproducible chest pain may be attributable to ACS [37]. Any degree of pulmonary rales on examination is associated with ACS; however, an S3 gallop on cardiac auscultation is nonspecific [37].

Pope and colleagues [37] found that patients who had a final diagnosis of ACS were more likely to have a lower pulse rate and higher blood pressure than patients who had other diagnoses; this probably is associated with adrenergic excess or lower compliance of an ischemic left ventricle. The clinician would need to know the patient's baseline vital signs; usually, that information is not available in the emergency department setting, which limits the usefulness of this observation [38]. The probability of AMI is increased if the patient is diaphoretic and is decreased if the respiratory rate is normal [37].

### **Laboratory studies**

Because myocytes lose their membrane integrity in response to ischemia, they release molecules into the peripheral circulation [36]. These molecules, known as cardiac biomarkers, are useful in the diagnosis of AMI. The biomarkers that can be detected do not aid in the diagnosis of unstable angina, which accounts for roughly half of all cases of ACS [38,40,41]. The cardiac biomarkers that are in widespread use are creatinine kinase (CK), creatinine kinase MB fraction (CK-MB), myoglobin, cardiac troponin I (cTnI), and cardiac troponin T (cTnT).

**Creatinine kinase and creatinine kinase MB fraction** CK and CK-MB are nonspecific biomarkers that can be found in any case of muscle damage [36]. Until recently, CK-MB had been the principal serum marker of cardiac myocyte damage [36]. The sensitivity of serum CK and CK-MB concentrations for detection of ischemia increases with the duration of the patient's symptoms [38]. Serial measurements of both biomarkers increase sensitivity and specificity when performed over 4 to 9 hours [38]. Serial CK-MB has a sensitivity of

87% and a specificity of 96% for AMI [38]. These serial tests should be performed over 4 to 9 hours.

**Myoglobin** Serum myoglobin is another non-specific biomarker that appears in the peripheral circulation as early as 1 to 2 hours after muscle damage [36]. Again, the sensitivity of myoglobin measurements in the diagnosis of AMI increases with serial measurements [38,41]. Serum myoglobin levels should not be used in isolation for the diagnosis of ACS [36]; however, there is some evidence that a normal myoglobin concentration 2 hours after presentation can exclude AMI [38,42].

**Troponin** cTnI and cTnT are specific for myocardial damage and have supplanted CK-MB as the preferred biomarker for myocardial ischemia [36]. These biomarkers are not found in the blood of healthy individuals [36]. As with the other cardiac biomarkers, the sensitivity of cTnI and cTnT increases with serial measurements and with duration of symptoms [38]. Elevated levels of cTnI and cTnT are associated with increased mortality, even when the ECG is inconclusive for ACS and CK-MB concentrations are normal [36,42].

### **Electrocardiography**

Electrocardiography is a safe, inexpensive, and readily available bedside test that represents the standard of care for patients who have expected ACS. When possible, the ECG should be obtained while the patient is symptomatic [36]. Although the ECG is highly sensitive for AMI, it is neither highly sensitive nor specific for ACS in general [38]. Pope and colleagues [37] found that up to 20% of patients who had AMI and 37% of patients who had the diagnosis of unstable angina had a normal ECG at presentation. The ECG should be interpreted with consideration of the patient's presentation. Thus, in a patient with a clinical picture that is consistent with ACS and a normal ECG, the probability of ischemia is not reduced substantially [38].

ST-segment and T-wave abnormalities are the quintessential electrocardiographic abnormalities in the diagnosis of ACS [37,38]. ST-segment elevation indicates transmural ischemia [36], whereas ST-segment depression indicates subendocardial ischemia [38]. Inverted T waves indicate acute ischemia [38]. Q waves are diagnostic of infarction but could represent previous infarction [38]. Obtaining an old ECG can aid in determining if any abnormalities have developed acutely.

### **Radiology**

**Chest radiography** A chest film usually is obtained during the initial assessment of the patient who has ACS. This imaging study is used to search

for other causes of the patient's symptoms and to assess for contraindications to heparin therapy (eg, aortic dissection). The presence of pulmonary edema, which could indicate acute heart failure, also is evaluated using plain chest radiography.

**Echocardiography** For the patient who has a low risk for ACS, resting echocardiography has a high sensitivity (93%), although only a moderate specificity (66%), for diagnosing AMI [43]. Echocardiography does not distinguish between acute and chronic abnormalities and requires skilled technicians and interpreters, which often limits its use in the acute setting [40]. Echocardiography is useful in providing information about the patient's hemodynamic status and may help to identify other causes of disease, such as PE and pericarditis [40].

**Nuclear imaging** Thallium-201 ( $^{201}\text{Tl}$ ) and technetium-99m sestamibi ( $^{99\text{m}}\text{Tc}$ -sestamibi) are radionuclides used commonly in nuclear cardiac imaging. Noninvasive tests based on those isotopes detect ischemic or infarcted myocardium. Both imaging modalities can detect perfusion abnormalities for several hours after the last symptomatic episode of chest pain [36]. Abnormal results of myocardial perfusion imaging studies done with the patient at rest indicate risk for AMI and death and the need for revascularization, whereas normal images at rest indicate the patient has a low risk for cardiac complications [36].  $^{201}\text{Tl}$  images must be taken within 15 to 20 minutes of injection, which limits the usefulness of this modality in the acute setting [40]. Imaging with  $^{99\text{m}}\text{Tc}$  sestamibi is advantageous because serial imaging can be done, and left ventricular wall motion abnormalities can be evaluated using gated single photon emission CT (SPECT) imaging [36]. Nuclear cardiac imaging is most useful in patients who have low to moderate risk for ACS and no acute ECG changes [43].

### **Pulmonary embolism**

PE must be considered in every patient who has chest pain and dyspnea. PE is the third most common cause of cardiovascular death among Americans, accounting for 50,000 to 100,000 deaths per year [44,45]. Only 30% of PE are diagnosed before death [46]. Alternatively, less than 35% of patients who are suspected of having a PE actually have one [45,47–49]. PE is a challenging diagnosis to reach, it often is missed, and it often is sought but not found.

### **History and physical examination**

The history and physical examination are notoriously insensitive for PE. The classic presentation of PE is chest pain, dyspnea, and hemoptysis; however, this triad is present in less than 20% of

patients [48]. Patients who have significant PE may remain asymptomatic if the obstruction of pulmonary circulation is less than 50% [50].

The Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study found that in patients who were diagnosed with PE, one or more risk factors for PE were likely to be present [47]. Risk factors for venous thromboembolism are listed in Box 2.

The most common symptom of acute PE is unexplained dyspnea of acute onset [48,51]. Dyspnea is present in more than 70% of patients who are diagnosed with PE [44]. Palpitations, cough, anxiety, lightheadedness, abdominal pain, back pain, atrial fibrillation, and hiccoughs are nonspecific symptoms [48,51]. Syncope occurs in 8% to 13% of patients who have PE [52].

The presentation of the patient who has PE depends on the degree of obstruction of the pulmonary circulation, the speed of accumulation of the clot burden, and the patient's underlying health [50]. Three clinical syndromes have been described in the patient who has PE: pulmonary infarction, isolated dyspnea, and circulatory collapse [48,53]. Signs and symptoms of PE vary according to the clinical syndrome that is present. For patients who have pulmonary infarction, pleuritic chest pain and hemoptysis may predominate [53]. Patients who have underlying cardiovascular disease, such as elderly patients, are more likely to have pulmonary infarction [48].

### **Box 2: Risk factors for pulmonary embolism**

#### **Inherited hematologic risk factors**

- Antithrombin III deficiency
- Factor V Leiden mutation
- Proteins C and S deficiency
- Lupus anticoagulant
- Abnormalities in fibrinolysis

#### **Acquired risk factors**

- Advanced age
- Smoking
- Immobilization
- Surgery
- Malignancy
- Trauma
- Oral contraceptives/hormone replacement
- Pregnancy
- Central venous catheters
- Obesity
- Myocardial infarction
- Congestive heart failure

Data from Refs. [44,48,53].

For patients who have isolated dyspnea, the degree of dyspnea varies with the degree of pulmonary vascular infarction [48]. For patients who have no underlying cardiovascular disease, the extent of embolism correlates with the degree of arterial hypoxemia [50]. Patients who have circulatory collapse may present following syncope, be hemodynamically unstable, or present in cardiac arrest [53].

No specific or sensitive physical examination finding is indicative of PE [53]. The most common signs of PE on examination are tachypnea and tachycardia [51]; however, normal vital signs should not discourage the physician from searching for a PE [53]. Fever, wheezing, rales, pleural rub, a loud pulmonic component of the second heart sound, right ventricular lift, right-sided fourth heart sound, cyanosis, and evidence of phlebitis may be present [45,51,53].

### Clinical scoring systems

Clinical scoring systems attempt to help the clinician estimate the probability of PE. The best known of the clinical scoring systems is Well's criteria for prediction of PE [Table 1]. This scoring system combines the assessment of risk factors, presenting signs and symptoms, as well as the clinician's suspicion of an alternate diagnosis [54]. This scoring system is vulnerable because it relies heavily on the subjective judgment of the clinician as to the presence of alternate diagnoses [53].

### Laboratory studies

Although arterial blood gas analysis (ABG) is a widely available and rapid laboratory study, it lacks the sensitivity to diagnose or exclude PE [53,55]. Patients who do not have underlying cardiopulmonary disease may have normal PaO<sub>2</sub>, nor-

mal PaCO<sub>2</sub>, and normal P(Aa)O<sub>2</sub> gradients in the face of angiographically proven PE [55].

D-dimer, a breakdown product of fibrin, is found in the blood when plasmin acts on a fibrin clot. As a marker of clot lysis, D-dimer is found in any condition in which there is formation or dissolution of clot. Thus, D-dimer can be found in elevated levels in association with PE, trauma, cancer, disseminated intravascular coagulation, myocardial infarction, sepsis, and preeclampsia and following surgery. Therefore, D-dimer is more useful in excluding PE than in diagnosing it [53,55,56,58]. Wells and colleagues [56] concluded that in a patient with a low clinical probability of PE using the Well's clinical scoring system and a negative D-dimer assay, PE can be ruled out safely without any imaging study [55].

### ECG

Most patients who have PE have some abnormality on ECG, but ECG abnormalities in patients who have PE are nonspecific [51]. The ECG is most helpful to exclude other causes of the patient's symptoms, such as myocardial ischemia or pericarditis. The characteristic ECG abnormality of PE is the S1Q3T3 pattern; however, this is found in less than 20% of ECGs from patients who have proven PE [53]. T-wave inversion in the precordial leads is the most common electrocardiographic finding and is present in 68% of patients who have PE [48]. Tachycardia and incomplete right bundle branch block also have been found more often in patients who have PE than in patients who have other diagnoses [45].

### Imaging

**Chest radiography** Like electrocardiography, chest radiography often is abnormal, but nonspecific, and may elucidate other diagnoses. The PIOPED study found that the most sensitive radiographic finding for PE is atelectasis or parenchymal abnormality, with a sensitivity of 68% [47]. Other common abnormalities that are found on chest radiography include pleural effusion, pulmonary infiltrates, mild elevation of the hemidiaphragm, enlargement of the pulmonary artery, and cardiomegaly [51,53]. It is important not to exclude the diagnosis of PE based on radiographic evidence of pneumonia or congestive heart failure, because these entities may coexist with PE [48]. The classic signs of relative oligemia (Westermarck's sign) and wedge-shaped pulmonary opacity (Hampton's hump) are rare [53].

**Ventilation-perfusion scintigraphy** Historically, ventilation-perfusion (V/Q) lung scanning has been the initial imaging modality of choice in

**Table 1: Wells' criteria for assigning pretest probability for pulmonary embolism**

Criteria	Points
Clinical symptoms or signs of DVT	3
PE more likely than other diagnosis	3
Heart rate >100 bpm	1.5
Immobilization or surgery within last 4 wk	1.5
History of DVT or PE	1.5
Hemoptysis	1
Malignancy	1
Clinical probability of PE	Points
Low	<2
Moderate	2–5
High	>6

Data from Refs. [56–58].



patients suspected of having PE. The results of the V/Q scan are interpreted in association with the patient's assigned pretest probability [48,53]. A high-probability V/Q scan in a patient who has a high pretest probability has an 85% to 90% positive predictive value of PE; a normal V/Q scan in a patient who has a low pretest probability essentially excludes the diagnosis of PE [47,59]. Most V/Q scans fall into the category of nondiagnostic, however, which severely limits the usefulness of this imaging modality [45]. Patients who have underlying lung disease also have abnormal baseline studies [48].

**Multidetector CT angiography** Multidetector CT angiography (MDCT-A) is becoming the initial study of choice in the acute setting for the diagnosis of PE, primarily because of its widespread availability, speed, and noninvasive nature. In comparison with V/Q scanning, CT is more accurate [45] and is more likely to show another cause of the patient's symptoms if PE is not present. There has been some question as to the sensitivity of CT for PE. Pooled data show a wide range of sensitivities (53–100%) and specificities (81–100%) [60]; however, for central PE, the sensitivity of CT increases to 94% [45]. Subsegmental emboli and horizontal vessels are not well visualized on CT [48]. Other drawbacks of CT imaging include the use of nephrotoxic contrast and radiation exposure; in addition, the study requires a cooperative patient, because motion artifact limits the quality of the images [45].

**Magnetic resonance angiography** Magnetic resonance angiography (MRA) may be used to visualize PE and lower extremity deep vein thrombosis (DVT) and offers the advantages of safer contrast material, noninvasive nature, and no ionizing radiation [48,53]. MRA is limited in its use by expense and availability. In addition, MR imaging is time consuming and allows only limited access to patients who become unstable [48].

**Pulmonary angiography** Pulmonary angiography is considered the gold standard for the diagnosis of PE [47]. Often, this procedure is not readily available; requires nephrotoxic contrast; and is invasive, time-consuming, and expensive. In addition, the patient must be transported away from the emergency department, and the images rarely elucidate an alternate diagnosis [53].

**Echocardiography** Transthoracic echocardiography (TTE) is noninvasive and can be performed at the bedside. Findings on echocardiography that suggest PE include right-sided thrombus; dilation of the right ventricle, pulmonary artery, or inferior vena cava; decreased right ventricular function; loss of right ventricular contractility; tricuspid regurgitation;

and abnormal septal wall motion [53]. Transesophageal echocardiography (TEE) is more invasive—usually requiring sedation—but is more sensitive than TTE for detection of these hemodynamic abnormalities [51,53].

**Ultrasound** Lower extremity ultrasound imaging for the detection of DVT has the greatest usefulness in the patient who has signs and symptoms of DVT and PE [45]. This test should not be used as an initial imaging modality for the patient who has suspected acute PE [45], but it may be useful as an adjunct test to detect the source of PE.

### **Pericarditis**

Pericarditis is inflammation of the pericardium, the fibrous sac surrounding the heart and great vessels [61,62]. The many causes of pericarditis include collagen vascular disease, renal insufficiency, neoplasm, viral infections, tuberculosis, and bacterial infections [63]. In many cases, the exact etiology remains unknown [63,64]. The diagnosis of pericarditis is suspected in the patient who has chest pain, pericardial rub on physical examination, and characteristic ECG changes [65].

### **History and physical examination**

By history alone, pericarditis may be difficult to differentiate from myocardial ischemia, because the patient may complain of retrosternal chest pain with a radiation pattern similar to that of myocardial ischemia [65,66]. Classically, pericarditis presents with a retrosternal location of pain, but the patient may complain of pain anywhere in the chest [66]. The pain often is described as sharp or stabbing [61]. A pleuritic component of the pain, including increasing pain with inspiration, an increase in pain with supine position, and some relief with upright posture or sitting forward, is described often [61,65]. The pain may radiate to the neck, arms, or left shoulder [61]. Pain that radiates to either trapezius muscle ridge is likely to be pericarditis secondary to phrenic nerve innervation of the anterior pericardium and both trapezius ridges [61,65,66]. Onset of pain is sudden and progressive over hours to days [61,65].

Fever or other features of a nonspecific prodrome may precede pericarditis of infectious etiology [65,67]. A medical history of renal failure, known neoplasm, collagen vascular disease, or thyroid disorder may aid in the diagnosis, because these are common causes of pericarditis [68]. Temperature higher than 38°C is rare, but when present, may indicate purulent pericarditis [61].

A pericardial friction rub is pathognomonic for pericarditis and is 100% specific for the disease [65,66]. The pericardial friction rub may come

and go with time; thus, the patient should be examined repeatedly [61,66]. The rub is best heard at the left lower sternal border with the patient leaning forward at the end of expiration [5,6,61]. Generally, the rub is described as rasping, creaking, harsh, or high pitched. Classically, it is triphasic, but it may be biphasic or monophasic [62,65]. The stereotypic triphasic rub that corresponds to the motion of the heart during ventricular systole, diastolic ventricular filling, and atrial contraction is present in only half of patients [69].

The presence of muffled heart sounds, tachycardia, distended neck veins, hypotension, and pulsus paradoxus suggests cardiac tamponade. The patient may be in acute respiratory distress, but the lungs generally will be clear [67].

### **Laboratory studies**

Laboratory studies are obtained to exclude other causes of chest pain and to elucidate the possible cause of the pericarditis. Markers of inflammation, such as leukocytosis, elevated C-reactive protein, and elevated erythrocyte sedimentation rate, usually are found in patients who have acute pericarditis [61]. Plasma electrolytes should be measured, and renal function should be evaluated [65].

The patient's clinical picture should guide additional testing, which might include blood cultures, tuberculin skin test, antinuclear antibodies, rheumatoid factor, thyroid function tests, viral throat swabs, and specific viral and bacterial serologies [63–65,68].

Pericardiocentesis should be considered in patients who have tamponade or suspected neoplastic or purulent pericarditis [61,65]. Routine pericardiocentesis for purely diagnostic purposes is not recommended [70].

Cardiac biomarker levels may be abnormal in patients who have pericarditis. Specifically, cTnI is elevated in more than 30% of patients who have acute pericarditis [71–73]. Men and younger patients are more likely to have elevated cTnI levels [71]. Elevation of cTnI is seen only in patients who have elevated ST segment on ECG and indicates myocardial cell damage [72]; however, cTnI levels do not indicate poor prognosis [71,72]. Serum CK and CK-MB levels also may be elevated [61].

### **Electrocardiography**

Diffuse elevation of the ST segments in the precordial and limb leads that is associated with PR segment depression is a classic electrocardiographic indication of acute pericarditis [74]. Historically, electrocardiographic abnormalities of acute pericarditis have been said to evolve over time, with four distinct stages described [75–78]. In stage I, ST elevation is diffuse, with PR segment depression.

Stage II is normalization of the ST and PR segments, whereas stage III is characterized by widespread T-wave inversions. The ECG normalizes again in stage IV. With the exception of purulent pericarditis, if the patient is treated promptly, stage I may be the only electrocardiographic abnormality seen [66]. The diffuse ST segment elevation of pericarditis can be differentiated from myocardial ischemia by the absence of reciprocal ST depression [7] and by the concave shape of the ST-segment elevations [61]. The presence of cardiac tamponade is characterized by low-voltage ECG with electrical alternans [77].

### **Radiography**

Radiologic studies may exclude other causes of chest pain. Chest radiography for pericarditis is aimed primarily at evaluation of the mediastinum and lungs for possible causes of the inflammation [61]. Cardiomegaly may be seen when an effusion of more than 250 mL has accumulated [61,65,67,69].

### **CT and MR imaging**

CT and MR imaging may be used to image the pericardium and pericardial space but are obtained most commonly to exclude other causes of chest pain or shortness of breath. CT and MR imaging evidence of thickened pericardium, enhancement of the pericardium that indicates inflammation, and visualization of pericardial effusion support the diagnosis of pericarditis [79,80].

### **Echocardiography**

Often, TTE is performed in patients who have suspected pericarditis. The presence of an effusion will help to confirm the diagnosis [61]. Evidence of tamponade on echocardiogram indicates the need for pericardiocentesis.

### **Thoracic aortic dissection**

Thoracic aortic dissection (TAD) is the most common aortic emergency that requires immediate surgery [81]. A dissection occurs when there is a tear in the intimal layer of the vessel wall. Blood passes through the tear, separates the intima from the vessel media or adventitia, and results in a false channel. Shear forces lead to dissection propagation as blood continues to flow through this false channel [82].

TAD can be difficult to diagnose. In the patient who presents to the emergency department and has acute chest or back pain, ACSs are 80 times more common than are aortic dissections [83]. Given that TADs occur most commonly in men who are aged 50 to 70 years and have a history of

hypertension, it is not surprising that myocardial ischemia is the most common misdiagnosis [84].

### History and physical examination

The acute onset of severe pain, which is maximal at symptom onset, is the most common initial symptom [85]. Usually, the pain is in the midline, may be present in the back, and rarely radiates [85]. A tearing or ripping quality of pain is classic and highly specific for TAD [86]; however, the International Registry of Acute Aortic Dissection (IRAD) found that most often, the pain was described as “sharp” [81]. Migratory pain has been considered classic for TAD, with pain corresponding to the propagation of the dissection, but was found in only 14% of patients in the IRAD [81]. Hypertension is the most common predisposing risk factor for TAD [81]. Inherited disorders, such as Marfan’s syndrome and Ehler-Danlos’ syndrome, associated with abnormal connective tissue structure have high rates of TAD [87,88]. Among women who are younger than 40 years who experience TAD, half are pregnant [85,87]. Cocaine use has been associated with TAD [89]. A history of syncope, with or without chest pain, was documented in 12% of patients who had TAD [81].

Pulse deficits or blood pressure differentials are independent predictive variables for TAD [83]; however, pulse deficits were documented in only 20% of patients in the IRAD [81]. Shear injury of the left carotid artery or compression of the aortic branches that supply the spinal cord may produce focal neurologic deficits [87,88]. When these deficits are present on examination, there is an increased likelihood of TAD [86]. A pulsatile sternoclavicular joint is rare but may indicate dissection [87].

When the dissection is proximal, an aortic regurgitation murmur may be heard. The IRAD study found this murmur in 44% of patients who had proximal TAD [81]. Cardiac tamponade with resultant physical examination findings of muffled heart sounds, elevated jugular venous distention, and narrow pulse pressure may be found when blood fills the pericardium [82].

### Laboratory studies

Generally, routine laboratory studies are not helpful in the diagnosis of TAD [87]. Often, laboratory studies are obtained to assess other causes of chest pain. Recent studies suggest that D-dimer concentration may be useful as a diagnostic tool for the diagnosis of TAD [90]. Serum biomarkers of smooth muscle myosin heavy chain and soluble elastin fragments have been found in higher concentrations in patients who had aortic dissection,

but the means of assessing these markers is not widely available in most clinical settings [91,92].

### Electrocardiography

In the patient who has TAD, the ECG may be normal or show left ventricular hypertrophy from long-standing hypertension [87]. Changes suggestive of myocardial ischemia related to coronary artery involvement in the dissection or occlusion of the artery may be present in up to one third of patients [87,93]. Two thirds of patients who have TAD have nonspecific ECG abnormalities of nonspecific ST-segment or T-wave changes [85].

### Imaging

**Chest radiography** Chest radiography is not specific for aortic dissection but is useful in combination with the history and physical examination. In approximately 50% of cases of dissection, the classic radiographic sign of widened mediastinal shadow is seen [85]. Some type of chest film abnormality [Box 3] is present in 90% of patients [86].

**CT** In the IRAD study, CT often was the initial imaging test for patients who have suspected aortic dissection, likely secondary to its widespread availability and noninvasive nature [81]. CT has a sensitivity of 93.8% and a specificity of 87% for TAD [96]. A positive contrast CT for TAD shows the raised intimal flap of the dissection between the true and false lumens of the aorta [96]. Thrombus also may be visualized within the false lumen [96]. If a dissection is not present, CT images may identify another cause of the patient’s symptoms. Drawbacks of CT imaging include relative difficulty in identifying the origin of the intimal tear, inability to assess involvement of the aortic branch ves-

#### Box 3: Common abnormalities found on chest radiography for aortic dissection

- Widened mediastinal shadow
- Altered configuration of the aorta
- Localized hump on the aortic arch
- Widening of the distal aortic knob past the origin of the left subclavian artery
- Aortic wall thickness indicated by the width of the aortic shadow beyond intimal calcification
- Displacement of the calcification in the aortic knob
- Double aortic shadow
- Disparity in the sizes of the ascending and descending aorta
- Presence of a pleural effusion, most commonly on the left

Data from Refs. [85,94,95].

sels, and lack of information about aortic valve regurgitation [85,96].

**MR imaging** MR imaging has a reported sensitivity and specificity of 98% for TAD [96]. This imaging modality provides quality images of the entire aorta, showing extent of the dissection, site of the tear, involvement of branch vessels, and involvement of the aortic valve [85,97]. However, MR imaging is expensive, time consuming, and not widely available and limits access to the potentially unstable patient [87]. Because of this, MR imaging rarely is obtained in the emergent setting.

**Echocardiography** The sensitivity and specificity of TTE for the detection of TAD vary widely, depending on the location of the dissection [85]. TTE does not visualize the aortic arch well and is virtually useless for the descending aorta [97]; however, TTE is useful for detection of tamponade or aortic insufficiency, which are complications of proximal dissections [87].

TEE is much better than TTE in the detection of TAD. TEE has a reported sensitivity of up to 98%, with a specificity of 77% [96]. A positive TEE may show a double-lumen aorta separated by the dissection membrane, which moves with the differential flow through the lumens [87]. TEE also can identify the site of dissection, sense abnormal flow, visualize a thrombus, assess for involvement of aortic valve and aortic branch vessels, and detect pericardial effusion [85]. The advantages of TEE are that it can be performed at the patient's bedside in a swift manner [85]. In many centers, TEE represents the noninvasive study of choice for the patient suspected of having TAD [85]. TEE requires esophageal intubation, which often necessitates sedation, and may not be available in some centers, especially in the evening and on weekends.

**Aortography** Traditionally, aortography has been considered the gold standard for the diagnosis of TAD, but it has been replaced by less invasive and more readily available radiologic studies. Findings on aortography that are indicative of TAD include distortion of contrast flow, flow reversal, flow stasis, failure of major vessels to fill with contrast, and aortic valve insufficiency [85]. Aortography may underestimate the size of the dissection if a thrombus is present [97]. The procedure is invasive and requires mobilization of an angiographic team. Aortography rarely is obtained for the acute diagnosis of TAD [81].

Despite advances in medical care, cardiopulmonary emergencies remain a major cause of morbidity and mortality in the United States. Rapid bedside radiographic detection of intrathoracic disorders is critical in clinical decision making related to these potentially life-threatening emergencies.

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## Summary

The use of radiographic imaging remains vital in the assessment of patients who present with chest pain.



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