

03 - 15 Chest Discomfort

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Chest Discomfort Chest discomfort is among the most common reasons for which patients present for medical attention at either an emergency department (ED) or an outpatient clinic. The evaluation of nontraumatic chest discomfort is inherently challenging owing to the broad variety of possible causes, a minority of which are life-threatening conditions that should not be missed. It is helpful to frame the initial diagnostic assessment and triage of patients with acute chest discomfort around three categories: (1) myocardial ischemia; (2) other cardiopulmonary causes (myopericardial disease, aortic emergencies, and pulmonary conditions); and (3) noncardiopulmonary causes. Although rapid identification of high-risk conditions is a priority of the initial assessment, strategies that incorporate routine liberal use of testing carry the potential for adverse effects of unnecessary investigations.

PART 2 Cardinal Manifestations and Presentation of Diseases

EPIDEMIOLOGY AND NATURAL HISTORY Chest discomfort is one of the three most common reason for visits to the ED in the United States, resulting in 6–7 million emergency visits each year. More than 60% of patients with this presentation are hospitalized for further testing, and most of the remainder undergo additional investigation in the ED. Fewer than 15% of evaluated patients are eventually diagnosed with acute coronary syndrome (ACS), with rates of 10–20% in most series of unselected populations, and a rate as low as 5% in some studies. The most common diagnoses are gastrointestinal causes (Fig. 15-1), and as few as 5% are other life-threatening cardiopulmonary conditions. In a large proportion of patients with transient acute chest discomfort, ACS or another acute cardiopulmonary cause is excluded but the cause is not determined. Therefore, the resources and time devoted to the evaluation of chest discomfort in the absence of a severe cause are substantial. Nevertheless, historically, a disconcerting 2–6% of patients with chest discomfort of presumed nonischemic etiology who are discharged from the ED were later deemed to have had a missed myocardial infarction (MI). The natural histories of ACS, myocarditis, acute pericardial diseases, pulmonary embolism, and aortic emergencies are discussed in Chaps. 281, 284, 285, 286, 290, and 291, respectively. The estimated rate of major cardiovascular events through 30 days in patients with acute chest pain who had been stratified as low risk was 2.5% in a large population-based study that excluded patients with ST-segment elevation or definite noncardiac chest pain.

CAUSES OF CHEST DISCOMFORT The major etiologies of chest discomfort are discussed in this section and summarized in Table 15-1. Additional elements of the history, physical examination, and diagnostic testing that aid in

FIGURE 15-1 Distribution of final discharge diagnoses in patients with nontraumatic acute chest pain. (Figure prepared from data in P Fruergaard et al: Eur Heart J 17:1028, 1996.)

distinguishing these causes are discussed in a later section (see “Approach to the Patient”). ■

■ **MYOCARDIAL ISCHEMIA/INJURY** Myocardial ischemia causing chest discomfort, termed angina pectoris, is a primary clinical concern in patients presenting with chest symptoms. Myocardial ischemia is precipitated by an imbalance between myocardial oxygen requirements and myocardial oxygen supply, resulting in insufficient delivery of oxygen to meet the heart’s metabolic demands. Myocardial oxygen consumption may be elevated by increases in heart rate, ventricular wall stress, and myocardial contractility, whereas myocardial oxygen supply is determined by coronary blood flow and coronary arterial oxygen content. When myocardial ischemia is sufficiently severe and prolonged in duration (as little as 20 min), irreversible cellular injury occurs, resulting in MI. The pathobiology of ischemic heart disease is described in Chap. 284. Stable angina is characterized by ischemic episodes that are typically precipitated by a superimposed increase in oxygen demand during physical exertion and relieved upon resting. Unstable ischemic heart disease (Chaps. 285 and 286) is classified clinically by the presence or absence of detectable acute myocardial injury and the presence or absence of ST-segment elevation on the patient’s electrocardiogram (ECG). Unstable ischemic heart disease is classified as unstable angina when there is no detectable acute myocardial injury and either as non-ST elevation MI (NSTEMI; see Chap. 285) or ST-elevation MI (STEMI; see Chap. 286) when there is evidence of acute myocardial injury. Clinicians should be aware that unstable ischemic symptoms may also occur predominantly because of increased myocardial oxygen demand (e.g., during intense psychological stress) or because of decreased oxygen delivery due to anemia, hypoxia, or hypotension. However, the term acute coronary syndrome, which encompasses unstable angina, NSTEMI, and STEMI, is in general reserved for ischemia precipitated by acute coronary atherothrombosis. To guide therapeutic strategies, a standardized system for classification of MI has been expanded to discriminate MI resulting from acute coronary thrombosis (type 1 MI) from MI occurring secondary to other imbalances of myocardial oxygen supply and demand (type 2 MI; see Chap. 285). Conditions associated with extreme myocardial oxygen demand and impaired endocardial blood flow, such as aortic valve disease (Chap. 291), hypertrophic cardiomyopathy, or idiopathic dilated cardiomyopathy (Chaps. 266–270), can precipitate myocardial ischemia in patients with or without underlying obstructive atherosclerosis. Ischemic causes of chest pain and myocardial injury are additionally distinguished from nonischemic causes of acute myocardial injury, such as myocarditis. Characteristics of Ischemic Chest Discomfort The clinical characteristics of angina pectoris, often referred to simply as “angina,” are highly similar in quality and location whether the ischemic discomfort is a manifestation of stable ischemic heart disease, unstable angina, or MI (Table 15-1). Heberden initially described angina as a sense of

Gastrointestinal 42% Ischemic heart disease 31% Chest wall syndrome 28% Pericarditis 4% Pleuritis 2% Pulmonary embolism 2% Lung cancer 1.5% Aortic aneurysm 1% Aortic stenosis 1% Herpes zoster 1%

TABLE 15-1 Typical Clinical Features of Major Causes of Acute Chest Discomfort

SYSTEM	CONDITION	ONSET/DURATION	QUALITY	LOCATION	ASSOCIATED FEATURES
Cardiopulmonary	Cardiac Myocardial ischemia	Stable angina: Precipitated by exertion, cold, or stress; 2–10 min	Unstable angina: Increasing pattern or at rest	Myocardial infarction: Usually >30 min	Pericarditis
		Variable; hours to days; may be episodic	Vascular	Acute aortic syndrome	Sudden onset of unrelenting pain
Pulmonary	Pulmonary embolism	Sudden onset	Pleuritic; may manifest as heaviness with massive pulmonary embolism	Pulmonary hypertension	Variable; often exertional
		Pressure	Substernal	Dyspnea, signs of increased venous pressure	Pulmonary
Pneumonia or pleuritis		Variable	Pleuritic	Unilateral, often localized	Dyspnea, cough, fever, rales, occasional rub
		Spontaneous			

pneumothorax Sudden onset Pleuritic Lateral to side of pneumothorax Noncardiopulmonary
 Gastrointestinal Esophageal reflux 10–60 min Burning Substernal, epigastric Worsened by
 postprandial recumbency; relieved by antacids Esophageal spasm 2–30 min Pressure, tightness,
 burning, intense squeezing Esophageal injury Prolonged Intense squeezing Retrosternal Includes
 Mallory-Weiss tear or esophageal rupture (Boerhaave's syndrome) from vomiting Peptic ulcer
 Prolonged; 60–90 min after meals Gallbladder disease, including cholecystitis and biliary colic
 Prolonged; steady, usually hours Pancreatitis Prolonged Typically aching Epigastric, radiates to
 the back Neuromuscular Costochondritis Variable Aching Sternal Sometimes swollen, tender, warm
 over joint; may be reproduced by localized pressure on examination Cervical disk disease
 Variable; may be sudden Aching; may include numbness Trauma or strain Usually constant Aching
 Localized to area of strain Reproduced by movement or palpation Herpes zoster Usually
 prolonged Sharp or burning Dermatomal distribution Vesicular rash in area of discomfort
 Psychological Emotional and psychiatric conditions Variable; may be fleeting or prolonged
 "strangling and anxiety." Chest discomfort characteristic of myocardial ischemia is typically
 described as aching, heavy, squeezing, crushing, or constricting. However, in a substantial minority
 of patients, the quality of discomfort is extremely vague and may be described as a mild tightness,
 or merely an uncomfortable feeling, that sometimes is experienced as numbness or a burning
 sensation. The site of the discomfort is usually retrosternal, but radiation is common and generally
 occurs down the ulnar surface of the left arm; the right arm, both arms, neck,

Pressure, tightness, squeezing, heaviness, burning Retrosternal; often radiation to neck, jaw,
 shoulders, or arms; sometimes epigastric S4 gallop or mitral regurgitation murmur (rare) during
 pain; S3 or rales if severe ischemia or complication of myocardial infarction Chest Discomfort
 CHAPTER 15 Pleuritic, sharp Retrosternal or toward cardiac apex; may radiate to left shoulder May
 be relieved by sitting up and leaning forward; pericardial friction rub Tearing or ripping; knifelike
 Anterior chest, often radiating to back, between shoulder blades Associated with hypertension
 and/or underlying connective tissue disorder; murmur of aortic insufficiency; loss of peripheral
 pulses Often lateral, on the side of the embolism Dyspnea, tachypnea, tachycardia, and
 hypotension Dyspnea, decreased breath sounds on side of pneumothorax Retrosternal Can closely
 mimic angina. May be relieved by nitroglycerin or dihydropyridine calcium channel antagonists
 Burning Epigastric (most common), substernal Relieved with food or antacids Aching or colicky
 Epigastric, right upper quadrant; sometimes to the back, scapula, and lower chest May follow meal
 and may subside spontaneously Arms and shoulders May be exacerbated by movement of neck
 Variable; often manifests as tightness and dyspnea with feeling of panic or doom Variable; may be
 retrosternal Situational factors may precipitate symptoms; history of panic attacks, depression jaw,
 or shoulders may also be involved. These and other characteristics of ischemic chest discomfort
 pertinent to discrimination from other causes of chest pain are discussed later in this chapter (see
 "Approach to the Patient"). Stable angina usually begins gradually and reaches its maximal
 intensity over a period of minutes before dissipating within several minutes with rest or with
 nitroglycerin. The discomfort typically occurs predictably at a characteristic level of exertion or
 psychological

stress. By definition, unstable angina is manifest by anginal chest discomfort that occurs with
 progressively lower intensity of physical activity or even at rest. Chest discomfort associated with
 MI is commonly more severe, is prolonged (usually lasting ≥ 30 min), and is not relieved by rest.

■ ■ OTHER CARDIOPULMONARY CAUSES Pericardial and Other Myocardial Diseases (See also Chap. 281) Inflammation of the pericardium due to infectious or noninfectious causes can be responsible for acute or chronic chest discomfort. The visceral surface and most of the parietal surface of the pericardium are insensitive to pain. Therefore, the pain of pericarditis is thought to arise principally from associated pleural inflammation. Because of this pleural association, the discomfort of pericarditis is usually pleuritic pain that is exacerbated by breathing, coughing, or changes in position. Moreover, owing to the overlapping sensory supply of the central diaphragm via the phrenic nerve with somatic sensory fibers originating in the third to fifth cervical segments, the pain of pleural and pericardial inflammation is often referred to the shoulder and neck. Involvement of the pleural surface of the lateral diaphragm can lead to pain in the upper abdomen.

PART 2
Cardinal Manifestations and Presentation of Diseases Acute inflammatory and other nonischemic myocardial diseases can also produce chest discomfort. The symptoms of acute myocarditis are highly varied. Chest discomfort may either originate with inflammatory injury of the myocardium or be due to severe increases in wall stress related to poor ventricular performance. The symptoms of Takotsubo (stress-related) cardiomyopathy often start abruptly with chest pain and shortness of breath. This form of cardiomyopathy, in its most recognizable form, is triggered by an emotionally or physically stressful event and may mimic acute MI because of its commonly associated ECG abnormalities, including ST-segment elevation, and elevated biomarkers of myocardial injury.

Diseases of the Aorta (See also Chap. 291) Acute aortic dissection (Fig. 15-1) is a less common cause of chest discomfort but is important because of the catastrophic natural history of certain subsets of cases when recognized late or left untreated. Acute aortic syndromes encompass a spectrum of acute aortic diseases related to disruption of the media of the aortic wall, including penetrating ulcer and intramural hematoma (see Chap. 291). Each of these subtypes of acute aortic syndrome typically presents with chest discomfort that is often severe, sudden in onset, and some times described as “tearing” in quality. Acute aortic syndromes involving the ascending aorta tend to cause pain in the midline of the anterior chest, whereas descending aortic syndromes most often present with pain in the back. Therefore, dissections that begin in the ascending aorta and extend to the descending aorta tend to cause pain in the front of the chest that extends toward the back, between the shoulder blades. Nontraumatic aortic dissections are very rare in the absence of hypertension or conditions associated with deterioration of the elastic or muscular components of the aortic media, including pregnancy, bicuspid aortic disease, or inherited connective tissue diseases, such as Marfan and Ehlers-Danlos syndromes. Aortitis, whether of noninfectious or infectious etiology, in the absence of aortic dissection is a rare cause of chest or back discomfort.

Pulmonary Conditions Pulmonary and pulmonary-vascular conditions that cause chest discomfort usually do so in conjunction with dyspnea and often produce symptoms that have a pleuritic nature.

PULMONARY EMBOLISM (SEE ALSO CHAP. 290) Pulmonary emboli can produce dyspnea and chest discomfort that is sudden in onset. Typically pleuritic in pattern, the chest discomfort associated with pulmonary embolism may result from (1) involvement of the pleural surface of the lung adjacent to a resultant pulmonary infarction; (2) distention of the pulmonary artery; or (3) possibly, right ventricular wall stress and/or subendocardial ischemia related to acute pulmonary hypertension. The pain associated with small pulmonary emboli is often lateral and pleuritic and is believed to be related to the first of these three possible mechanisms. In contrast, massive pulmonary emboli may cause

severe substernal pain that may mimic an MI and that is plausibly attributed to the second and third of these potential mechanisms. Massive or submassive pulmonary embolism may also be

associated with syncope, hypotension, and signs of right heart failure. Other typical characteristics that aid in the recognition of pulmonary embolism are discussed later in this chapter (see “Approach to the Patient”).

PNEUMOTHORAX (SEE ALSO CHAP. 305) Primary spontaneous pneumothorax is a rare cause of chest discomfort. Risk factors include male sex, smoking, family history, and Marfan syndrome. The symptoms are usually sudden in onset, and dyspnea may be mild; thus, presentation to medical attention is sometimes delayed. Secondary spontaneous pneumothorax may occur in patients with underlying lung disorders, such as chronic obstructive pulmonary disease, asthma, or cystic fibrosis, and usually produces symptoms that are more severe. Tension pneumothorax is a medical emergency caused by trapped intrathoracic air that precipitates hemodynamic collapse.

Other Pulmonary Parenchymal, Pleural, or Vascular Disease (See also Chaps. 294, 295, and 305) Most pulmonary diseases that produce chest pain, including pneumonia and malignancy, do so because of involvement of the pleura or surrounding structures. Pleurisy is typically described as a knifelike pain that is worsened by inspiration or coughing. In contrast, chronic pulmonary hypertension can manifest as chest pain that may be very similar to angina in its characteristics, suggesting right ventricular myocardial ischemia in some cases. Reactive airways diseases similarly can cause chest tightness associated with breathlessness rather than pleurisy.

■ ■ **NONCARDIOPULMONARY CAUSES**

Gastrointestinal Conditions (See also Chap. 332) Gastrointestinal disorders are the most common cause of nontraumatic chest discomfort and often produce symptoms that are difficult to discern from more serious causes of chest pain and are described in Table 15-1. Esophageal disorders, in particular, may simulate angina in the character and location of the pain. Gastroesophageal reflux and disorders of esophageal motility are common (Fig. 15-1 and Table 15-1).

Musculoskeletal and Other Causes (See also Chap. 372) Chest discomfort can be produced by any musculoskeletal disorder involving the chest wall or the nerves of the chest wall, neck, or upper limbs. Costochondritis causing tenderness of the costochondral junctions (Tietze’s syndrome) is relatively common. Cervical radiculitis may manifest as a prolonged or constant aching discomfort in the upper chest and limbs. The pain may be exacerbated by motion of the neck. Occasionally, chest pain can be caused by compression of the brachial plexus by the cervical ribs, and tendinitis or bursitis involving the left shoulder may mimic the radiation of angina. Pain in a dermatomal distribution can also be caused by cramping of intercostal muscles or by herpes zoster (Chap. 198).

Emotional and Psychiatric Conditions As many as 10% of patients who present to EDs with acute chest discomfort have a panic disorder or related condition (Table 15-1).

APPROACH TO THE PATIENT Chest Discomfort Given the broad set of potential causes and the heterogeneous risk of serious complications in patients who present with acute nontraumatic chest discomfort, the priorities of the initial clinical encounter include assessment of (1) the patient’s clinical stability and (2) the probability that the patient has an underlying cause of the discomfort that may be life-threatening. The high-risk conditions of principal concern are acute cardiopulmonary processes, including ACS, acute aortic syndrome, pulmonary embolism, tension pneumothorax, and pericarditis with tamponade. Fulminant myocarditis also carries a poor prognosis but is usually also manifested by heart failure symptoms. Among noncardiopulmonary causes

TABLE 15-2 Considerations in the Assessment of the Patient with Chest Discomfort

1. Could the chest discomfort be due to an acute, potentially life-threatening condition that warrants urgent evaluation and management? Unstable ischemic heart disease Aortic dissection Pneumothorax Pulmonary embolism

2. If not, could the discomfort be due to a chronic condition likely to lead to serious complications? Stable angina Aortic stenosis Pulmonary hypertension
 3. If not, could the discomfort be due to an acute condition that warrants specific treatment? Pericarditis Pneumonia/pleuritis Herpes zoster
 4. If not, could the discomfort be due to another treatable chronic condition? Esophageal reflux Cervical disk disease Esophageal spasm Arthritis of the shoulder or spine Peptic ulcer disease Costochondritis Gallbladder disease Other musculoskeletal disorders Other gastrointestinal conditions Anxiety state
- Source: Developed by Dr. Thomas H. Lee for the 18th edition of Harrison's Principles of Internal Medicine.
- of chest pain, esophageal rupture likely holds the greatest urgency for diagnosis. The remaining population with noncardiopulmonary conditions has a more favorable prognosis during completion of the diagnostic workup. A rapid targeted assessment for a serious cardiopulmonary cause is most relevant for patients with acute ongoing pain who have presented for emergency evaluation. Among patients presenting in the outpatient setting with chronic pain or pain that has resolved, a general diagnostic assessment is reasonably undertaken (see "Outpatient Evaluation of Chest Discomfort," below). A series of questions that can be used to structure the clinical evaluation of patients with chest discomfort is shown in Table 15-2.
- INCREASED LIKELIHOOD OF AMI**
- Radiation to right arm or shoulder
 - Radiation to both arms or shoulders
 - Associated with exertion
 - Radiation to left arm
 - Associated with diaphoresis
 - Associated with nausea or vomiting
 - Worse than previous angina or similar to previous MI
 - Described as pressure
- DECREASED LIKELIHOOD OF AMI**
- Inframammary location
 - Reproducible with palpation
 - Described as sharp
 - Described as positional
 - Described as pleuritic

0.5

1.5

2.5

3.5

4.5

FIGURE 15-2 Association of chest pain characteristics with the probability of acute myocardial infarction (AMI). Note that a subsequent larger study showed a nonsignificant association with radiation to the right arm. (Figure prepared from data in CJ Swap, JT Nagurney: JAMA 294:2623, 2005.)

HISTORY The evaluation of nontraumatic chest discomfort relies heavily on the clinical history and physical examination to direct subsequent diagnostic testing. The evaluating clinician should assess the quality, location (including radiation), and pattern (including onset and duration) of the pain as well as any provoking or alleviating factors. The presence of associated symptoms may also be useful in establishing a diagnosis. **Quality of Pain** The quality of chest discomfort alone is never sufficient to establish a diagnosis. However, the characteristics of the pain are pivotal in formulating an initial clinical impression and assessing the likelihood of a serious cardiopulmonary

process (Table 15-1; Fig. 15-2). Pressure or tightness is consistent with a typical presentation of myocardial ischemic pain. Nevertheless, the clinician must remember that some patients with ischemic chest symptoms deny any “pain” but rather complain of dyspnea or a vague sense of anxiety. The severity of the discomfort has poor diagnostic accuracy. It is often helpful to ask about the similarity of the discomfort to previous definite ischemic symptoms. It is unusual for angina to be sharp, as in knifelike, stabbing, or pleuritic; however, patients sometimes use the word “sharp” to convey the intensity of discomfort rather than the quality. Pleuritic discomfort is suggestive of a process involving the pleura, including pericarditis, pulmonary embolism, or pulmonary parenchymal processes. “Tearing” or “ripping” pain is often described by patients with acute aortic dissection. However, acute aortic emergencies also present commonly with knifelike pain. A burning quality can suggest acid reflux or peptic ulcer disease but may also occur with myocardial ischemia. Esophageal pain, particularly with spasm, can be a severe squeezing discomfort identical to angina. Chest Discomfort CHAPTER 15 Location of Discomfort A substernal location with radiation to the neck, jaw, shoulder, or arms is typical of myocardial ischemic discomfort. Radiation to both arms has a particularly high association with MI as the etiology. Some patients present with aching in sites of radiated pain as their only symptoms of ischemia. However, pain that is highly localized—e.g., that which can be demarcated by the tip of one finger—is highly unusual for angina. A retrosternal Likelihood ratio for AMI

location should prompt consideration of esophageal pain; however, other gastrointestinal conditions usually present with pain that is most intense in the abdomen or epigastrium, with possible radiation into the chest. Angina may also occur in an epigastric location. Pain that occurs solely above the mandible or below the epigastrium is rarely angina. Severe pain radiating to the back, particularly between the shoulder blades, should prompt consideration of acute aortic syndrome. Radiation to the trapezius ridge is characteristic of pericardial pain and does not usually occur with angina. Pattern Myocardial ischemic discomfort usually builds over minutes and is exacerbated by activity and mitigated by rest. In contrast, pain that reaches its peak intensity immediately is more suggestive of aortic dissection, pulmonary embolism, or spontaneous pneumothorax. Pain that is fleeting (lasting only a few seconds) is rarely ischemic in origin. Similarly, pain that is constant in intensity for a prolonged period (many hours to days) is unlikely to represent myocardial ischemia if it occurs in the absence of other clinical consequences, such as abnormalities of the ECG, elevation of cardiac biomarkers, or clinical sequelae (e.g., heart failure or hypotension). Both myocardial ischemia and acid reflux may have their onset in the morning. Provoking and Alleviating Factors Patients with myocardial ischemic pain usually prefer to rest, sit, or stop walking. However, clinicians should be aware of the phenomenon of “warm-up angina” in which some patients experience relief of angina as they continue at the same or even a greater level of exertion (Chap. 284). Alterations in the intensity of pain with changes in position or movement of the upper extremities and neck are less likely with myocardial ischemia and suggest a musculoskeletal etiology. The pain of pericarditis, however, often is worse in the supine position and relieved by sitting upright and leaning forward. Gastroesophageal reflux may be exacerbated by alcohol, some foods, or a reclined position. Relief can occur with sitting. PART 2 Cardinal Manifestations and Presentation of Diseases Exacerbation by eating suggests a gastrointestinal etiology such as peptic ulcer disease, cholecystitis, or pancreatitis. Peptic ulcer disease tends to become symptomatic 60–90 min after meals. However, in the setting of severe coronary atherosclerosis, redistribution of blood flow to the splanchnic vasculature after eating can trigger postprandial angina. The discomfort of acid reflux and peptic ulcer disease is usually diminished

promptly by acid-reducing therapies. In contrast with its impact in some patients with angina, physical exertion is very unlikely to alter symptoms from gastrointestinal causes of chest pain. Relief of chest discomfort within minutes after administration of nitroglycerin is suggestive of but not sufficiently sensitive or specific for a definitive diagnosis of myocardial ischemia. Esophageal spasm may also be relieved promptly with nitroglycerin. A delay of >10 min before relief is obtained after nitroglycerin suggests that the symptoms either are not caused by ischemia or are caused by severe ischemia, such as during acute MI. Associated Symptoms Symptoms that accompany myocardial ischemia may include diaphoresis, dyspnea, nausea, fatigue, faintness, and eructations. In addition, these symptoms may exist in isolation as anginal equivalents, particularly in women and the elderly. Dyspnea may occur with multiple conditions considered in the differential diagnosis of chest pain and thus is not discriminative, but the presence of dyspnea is important because it suggests a cardiopulmonary etiology. Sudden onset of significant respiratory distress should lead to consideration of pulmonary embolism and spontaneous pneumothorax. Hemoptysis may occur with pulmonary embolism or as blood-tinged frothy sputum in severe heart failure but usually points toward a pulmonary parenchymal etiology of chest symptoms. Presentation with syncope or presyncope should prompt consideration of hemodynamically significant pulmonary embolism or aortic dissection as well as ischemic arrhythmias. Although nausea and vomiting suggest a gastrointestinal disorder, these symptoms may occur in the setting of MI (more commonly inferior MI), presumably because of activation of the

vagal reflex or stimulation of left ventricular receptors as part of the Bezold-Jarisch reflex. Past Medical History The past medical history is useful in assessing the patient for risk factors for coronary atherosclerosis and venous thromboembolism (Chap. 290) as well as for conditions that may predispose the patient to specific disorders. For example, a history of connective tissue diseases such as Marfan syndrome should heighten the clinician's suspicion of acute aortic syndrome or spontaneous pneumothorax. PHYSICAL EXAMINATION In addition to providing an initial assessment of the patient's clinical stability, the physical examination of patients with chest discomfort can provide direct evidence of specific etiologies of chest pain (e.g., unilateral absence of lung sounds) and can identify potential precipitants of acute cardiopulmonary causes of chest pain (e.g., uncontrolled hypertension), relevant comorbid conditions (e.g., obstructive pulmonary disease), and complications of the presenting syndrome (e.g., heart failure). However, because the findings on physical examination may be normal in patients with unstable ischemic heart disease, an unremarkable physical exam is not definitively reassuring. General The patient's general appearance is helpful in establishing an initial impression of the severity of illness. Patients with acute MI or other acute cardiopulmonary disorders often appear anxious, uncomfortable, pale, cyanotic, or diaphoretic. Patients who are massaging or clutching their chests may describe their pain with a clenched fist held against the sternum (Levine's sign). Occasionally, body habitus is helpful—e.g., in patients with Marfan syndrome or the prototypical young, tall, thin man with spontaneous pneumothorax. Vital Signs Significant tachycardia and hypotension are indicative of important hemodynamic consequences of the underlying cause of chest discomfort and should prompt a rapid survey for the most severe conditions, such as acute MI with cardiogenic shock, massive pulmonary embolism, pericarditis with tamponade, or tension pneumothorax. Acute aortic emergencies usually present with severe hypertension but may be associated with profound hypotension when there is coronary arterial compromise or dissection into the pericardium. Sinus tachycardia is an important manifestation of submassive pulmonary embolism. Tachypnea and hypoxemia point toward a pulmonary cause. The presence of low-grade fever is nonspecific

because it may occur with MI and with thromboembolism in addition to infection. Pulmonary Examination of the lungs may localize a primary pulmonary cause of chest discomfort, as in cases of pneumonia, asthma, or pneumothorax. Left ventricular dysfunction from severe ischemia/infarction as well as acute valvular complications of MI or aortic dissection can lead to pulmonary edema, which is an indicator of high risk. Cardiac The jugular venous pulse is often normal in patients with acute myocardial ischemia but may reveal characteristic patterns with pericardial tamponade or acute right ventricular dysfunction (Chaps. 246 and 281). Cardiac auscultation may reveal a third or, more commonly, a fourth heart sound, reflecting myocardial systolic or diastolic dysfunction. Murmurs of mitral regurgitation or a ventricular-septal defect may indicate mechanical complications of STEMI. A murmur of aortic insufficiency may be a complication of ascending aortic dissection. Other murmurs may reveal underlying cardiac disorders contributory to ischemia (e.g., aortic stenosis or hypertrophic cardiomyopathy). Pericardial friction rubs reflect pericardial inflammation. Abdominal Localizing tenderness on the abdominal exam is useful in identifying a gastrointestinal cause of the presenting syndrome. Abdominal findings are infrequent with purely acute cardiopulmonary problems, except in the case of right-sided heart failure leading to hepatic congestion.

Extremities Vascular pulse deficits may reflect underlying chronic atherosclerosis, which increases the likelihood of coronary artery disease. However, evidence of acute limb ischemia with loss of the pulse and pallor, particularly in the upper extremities, can indicate catastrophic consequences of aortic dissection. Unilateral lower-extremity swelling should raise suspicion about venous thromboembolism. Musculoskeletal Pain arising from the costochondral and chondrosternal articulations may be associated with localized swelling, redness, or marked localized tenderness. Pain on palpation of these joints is usually well localized and is a useful clinical sign, although deep palpation may elicit pain in the absence of costochondritis. Although palpation of the chest wall often elicits pain in patients with various musculoskeletal conditions, it should be appreciated that chest wall tenderness does not exclude myocardial ischemia. Sensory deficits in the upper extremities may be indicative of cervical disk disease. ELECTROCARDIOGRAPHY

Electrocardiography is crucial in the evaluation of nontraumatic chest discomfort. The ECG is pivotal for identifying patients with ongoing ischemia as the principal reason for their presentation as well as secondary cardiac complications of other disorders. Professional society guidelines recommend that an ECG be obtained within 10 min of presentation, with the primary goal of identifying patients with ST-segment elevation diagnostic of MI who are candidates for immediate interventions to restore flow in the occluded coronary artery. ST-segment depression and symmetric T-wave inversions at least 0.2 mV in depth are useful for detecting myocardial ischemia in the absence of STEMI and are also indicative of higher risk of death or recurrent ischemia. Serial performance of ECGs (every 30–60 min) is recommended early in the ED evaluation of suspected ACS. In addition, an ECG with right-sided lead placement should be considered in patients with clinically suspected ischemia and a nondiagnostic standard 12-lead ECG. Despite the value of the resting ECG, its sensitivity for ischemia is poor—as low as 20% in some studies. Abnormalities of the ST segment and T wave may occur in a variety of conditions, including pulmonary embolism, ventricular hypertrophy, acute and chronic pericarditis, myocarditis, electrolyte imbalance, and metabolic disorders. Notably, hyperventilation associated with panic disorder can also lead to nonspecific ST and T-wave abnormalities. Pulmonary embolism is most often associated with sinus tachycardia but can also lead to rightward shift of the ECG axis, manifesting as an S-wave in lead I, with a Q-wave and T-wave in lead III (Chaps. 247 and 290). In patients with STsegment elevation,

the presence of diffuse lead involvement not corresponding to a specific coronary anatomic distribution and PR-segment depression can aid in distinguishing pericarditis from acute MI. CHEST RADIOGRAPHY (See Chap. A12) Plain radiography of the chest is performed routinely when patients present with acute chest discomfort and selectively when individuals who are being evaluated as outpatients have subacute or chronic pain. The chest radiograph is most useful for identifying pulmonary processes, such as pneumonia or pneumothorax. Findings are often unremarkable in patients with ACS, but pulmonary edema may be evident. Other specific findings include widening of the mediastinum in some patients with aortic dissection, Hampton's hump or Westermark's sign in patients with pulmonary embolism (Chaps. 290 and A12), or pericardial calcification in chronic pericarditis. CARDIAC BIOMARKERS Laboratory testing in patients with acute chest pain is focused on the detection of myocardial injury. Such injury can be detected by the presence of circulating proteins released from damaged

cardiomyocytes. Owing to the time necessary for this release, initial biomarkers of injury may be in the normal range, even in patients with STEMI. Cardiac troponin is the preferred biomarker for the diagnosis of MI and should be measured in all patients with suspected ACS. It is not necessary or advisable to measure troponin in patients without suspicion of ACS unless this test is being used specifically for risk stratification (e.g., in pulmonary embolism or heart failure). The development of cardiac troponin assays with progressively greater analytical sensitivity has facilitated detection of myocardial injury, enhanced the overall accuracy of a diagnosis of MI, and improved risk stratification in suspected ACS. For these reasons, high-sensitivity assays are preferred over prior generation troponin assays. The greater negative predictive value of a negative troponin result with high-sensitivity assays is an advantage in the evaluation of chest pain in the ED. Rapid rule-out protocols that use serial testing and changes in troponin concentration over as short a period as 1–2 h perform well for diagnosis of ACS when using a high-sensitivity troponin assay. Troponin should be measured at presentation and repeated at 1–3 h using high-sensitivity troponin and 3–6 h using conventional troponin assays. Additional troponin measurements may be warranted beyond 3–6 h when the clinical condition still suggests possible ACS or if there is diagnostic uncertainty. In patients presenting more than 2–3 h after symptom onset, a very low concentration of cardiac troponin, at the time of hospital presentation, using a high-sensitivity assay may be sufficient to exclude MI with a negative predictive value >99%. Chest Discomfort CHAPTER 15 With the use of high-sensitivity assays for troponin, myocardial injury is detected in a larger proportion of patients who have nonACS cardiopulmonary conditions than with previous, less sensitive assays. Therefore, other aspects of the clinical evaluation are critical to the practitioner's determination of the probability that the symptoms represent ACS. In addition, observation of a change in cardiac troponin concentration between serial samples is necessary for discriminating acute causes of myocardial injury from chronic elevation due to underlying structural heart disease, end-stage renal disease, or the rare presence of interfering antibodies. The diagnosis of MI is reserved for acute myocardial injury that is marked by a rising and/or falling pattern—with at least one value exceeding the 99th percentile reference limit—and that is caused by ischemia. Other nonischemic insults, such as myocarditis, may result in acute myocardial injury but should not be labeled MI (Fig. 15-3). Other laboratory assessments may include the D-dimer test to aid in exclusion of pulmonary embolism (Chap. 290). Measurement of a B-type natriuretic peptide is useful when considered in conjunction with the clinical history and exam for the diagnosis of heart failure. Elevated cTn Concentration Stable cTn Dynamic cTn (significant rise or fall) Ischemia No ischemia Myocardial infarction Acute myocardial injury Chronic myocardial injury Type 1 MI Type 2 MI

FIGURE 15-3 Clinical classification of patients with elevated cardiac troponin (cTn). MI, myocardial infarction.

INTEGRATIVE DECISION-AIDS Multiple clinical algorithms have been developed to aid in decisionmaking during the evaluation and disposition of patients with acute nontraumatic chest pain. Such decision-aids estimate either of two closely related but not identical probabilities: (1) the probability of a final diagnosis of ACS and (2) the probability of major cardiac events during short-term follow-up. Use of clinical decision pathways (CDPs) is recommended to categorize patients as low, intermediate, and high risk. Evidence-based CDPs may be used to identify patients with a low clinical probability of ACS who are candidates for discharge from the ED without additional noninvasive testing. Two CDPs used commonly in current practice are shown in Fig. 15-4. Elements common across multiple risk stratification tools are (1) symptoms typical for ACS; (2) older age; (3) risk factors for or known atherosclerosis; (4) ischemic ECG abnormalities; and (5) elevated cardiac troponin level. Clinical application of such integrated CDPs incorporating ECGs and serial high-sensitivity cardiac troponin has been reported to achieve overall “miss rates” for ACS of <0.5% and are useful for identifying patients who are reasonable to discharge home without admission or urgent cardiac testing. In some studies, provision of such protocol-driven care in dedicated chest pain units has decreased costs and overall duration of hospital evaluation with no detectable excess of adverse clinical outcomes.

PART 2 Cardinal Manifestations and Presentation of Diseases

Clinicians should differentiate between the algorithms discussed above and risk scores derived for stratification of prognosis (e.g., the TIMI and GRACE risk scores, Chap. 286) in patients who already have an established diagnosis of ACS. The latter risk scores were not designed to be used for diagnostic assessment.

CORONARY AND MYOCARDIAL STRESS IMAGING Among patients for whom other life-threatening causes of chest pain have been reasonably excluded and serial biomarker and HEART Score (without cTn) History Highly suspicious Moderately suspicious Slightly suspicious

ECG Significant ST depression Nonspecific abnormality Normal

Age ≥ 65 y 45–<65 y <45 y

Risk factors ≥ 3 risk factors 1–2 risk factors None

TOTAL Low risk: 0–3 Not low risk: ≥ 4 AND cardiac troponin < the limit of quantification. Captured as low risk (%) 51.8 NPV 99.55

FIGURE 15-4 Examples of decision-aids used in conjunction with serial measurement of cardiac troponin (cTn) for evaluation of acute chest pain. The HEART score was modified by the authors in the presented study and omitting the assignment of 0, 1, or 2 points based on troponin. The negative predictive value (NPV) reported is for the composite endpoint of myocardial infarction (MI), cardiogenic shock, cardiac arrest, and all-cause mortality by 60 days. CABG, coronary artery bypass graft; CAD, coronary artery disease; ECG, electrocardiogram; PCI, percutaneous coronary intervention. (Figure prepared from data in DG Mark et al: J Am Coll Cardiol 13:606, 2018.)

clinical assessment have determined the patient to remain eligible for further testing because of intermediate or undetermined risk, diagnostic coronary imaging with coronary computed tomographic (CT) angiography or functional testing, preferably with nuclear or echocardiographic imaging, is recommended. Patient characteristics (e.g., body habitus and renal function), prior

cardiac testing, history of known coronary artery disease, existing contraindications for a given test modality, and patient preferences are considerations when choosing among these diagnostic tests (Chaps. 248 and A9). CT Angiography (See Chap. 248) CT angiography has emerged as a preferred modality for the evaluation of such patients. Coronary CT angiography is a sensitive technique for detection of obstructive coronary disease. CT appears to enhance the speed to disposition of patients with a low-intermediate probability for ACS, with its major strength being the negative predictive value of a finding of no significant stenosis or coronary plaque. At the same time, CT angiography can exclude aortic dissection, pericardial effusion, and pulmonary embolism. Stress Nuclear Perfusion Imaging or Stress Echocardiography (See Chaps. 248 and A9) Functional testing with stress nuclear perfusion imaging and stress echocardiography are alternatives for the evaluation of patients with acute chest pain who are candidates for further testing and are preferred over coronary CT angiography in patients with known obstructive epicardial disease. The selection of stress test modality may depend on institutional availability and expertise. Stress testing with myocardial imaging, either with nuclear perfusion imaging or echocardiography, offers superior diagnostic performance over exercise ECG. In patients selected for stress myocardial imaging who are able to exercise, exercise stress testing is preferred over pharmacologic testing. When available, EDACS Score Age 86+ y 81–85 y 76–80 y Step down by 5-y increments 46–50 y 18–45 y

(-2)

Known CAD or risk factors Known CAD (prior MI, PCI, or CABG) or ≥ 3 cardiac risk factors in patient aged ≤ 50 y

Sex Male Female

Symptoms Radiation to arm, shoulder, neck, or jaw Diaphoresis Pain with inspiration Reproduced by palpation

-4 -6 TOTAL Low risk: 0–15 Not low risk: ≥ 16 60.6 99.49