CHAPTER 18 Chest Pain

James E. Brown and Glenn C. Hamilton

PERSPECTIVE

Nearly 6 million patients present to the emergency department (ED) each year with complaints of chest pain, constituting 5% of all patients seen in EDs in the United States.[1] Chest pain is a symptom caused by several life-threatening diseases and has a broad differential diagnosis. It is complicated by a frequent disassociation between intensity of symptoms and signs and seriousness of underlying pathology.

Epidemiology

The epidemiology of the critical diagnoses causing chest pain varies widely. Acute coronary syndromes (ACS), aortic dissection, pulmonary embolism (PE), pneumothorax, pericarditis with tamponade, and esophageal rupture are potentially catastrophic causes of chest pain. Due to its high incidence and potential lethality, ACS is the most significant potential diagnosis in the ED. Of all deaths in the United States, 36% are attributed to cardiovascular diseases; these account for approximately 870,000 deaths per year.[2] Historically, emergency physicians misdiagnose 3 to 5% of myocardial infarctions (MIs), accounting for 25% of malpractice losses in emergency medicine.[3,4] Thoracic aortic dissection has an incidence of 0.5 to 1 per 100,000 population with a mortality rate exceeding 90% if misdiagnosed. The true incidence of PE is unclear, with estimates of 70 per 100,000. This equates to approximately 100,000 PE cases per year in the United States.[5] Although the incidence of tension pneumothorax is also unclear, the incidence of spontaneous pneumothorax ranges from 2.5 to 18 per 100,000 total patients. The total incidence of esophageal rupture is 12.5 cases per 100,000 persons. The true incidence of pericarditis is unknown, but is diagnosed in 1 of every 1000 hospital admissions.[6] Up to 5% of ED chest pain patients without acute ST elevation MI may have pericarditis.[7]

Pathophysiology

Afferent fibers from the heart, lungs, great vessels, and esophagus enter the same thoracic dorsal ganglia. Through these visceral fibers, each organ produces the same indistinct quality and location of pain. The quality of visceral chest pain varies widely and is described as "burning," "aching," "stabbing," or "pressure." Since dorsal segments overlap three segments above and below a level, disease of a thoracic origin can produce pain anywhere from the jaw to the epigastrium. Radiation of pain is caused by somatic afferent fibers synapsing in the same dorsal root ganglia as the thoracic viscera. This stimulation can "confuse" the patient's central nervous system into misperceiving that the pain originates in the arms or shoulders.

DIAGNOSTIC APPROACH

Differential Considerations

Due to the indistinct nature of visceral pain, the differential diagnosis of chest pain is broad and includes many of the most critical diagnoses in medicine and many nonemergent conditions (Table 18-1).

Rapid Stabilization and Assessment

The initial questions are, "Must I intervene immediately?", and "What are the life-threatening possibilities in this patient?" The answers are usually apparent within the first few minutes after assessing the patient's appearance and vital signs. One of the critical diagnoses is tension pneumothorax. If a patient presents with chest pain, respiratory distress, shock, and unilateral reduction or absence of breath sounds, immediate intervention with needle or tube thoracostomy is required. Additionally, patients with severe derangements in vital signs require stabilizing treatment during a search for the precipitating cause. Patients who present with respiratory distress require immediate intervention and lead the emergency physician to consider a more serious cause of the pain (Fig. 18-1; also see Chapter 17).

All patients, except those with obvious benign causes of chest pain, must have an electrocardiogram (ECG) within minutes of reporting their pain. This ECG should be read for acute MI by the emergency physician as soon as it is completed. Patients with positive ECG findings and those considered at high risk are triaged directly to the treatment area and monitored. Symptomatic derangements in vital signs are addressed. If vital signs are stable, a focused history and physical examination are performed. Most patients also require a chest radiograph to evaluate the chest pain. If a cardiac cause is suggested and vital signs are stable, pain relief with nitroglycerin (0.4 mg sublingual every 3–5 minutes) may be appropriate. Aspirin (81–325 mg) is a consideration for patients without hemorrhagic disorders, known allergies, or vascular dissections. Clopidogrel (loading dose 300 mg) or other anti-

Table 18-1 Differential Diagnosis of Chest Pain

ORGAN SYSTEM	CRITICAL DIAGNOSES	EMERGENT DIAGNOSES	NONEMERGENT DIAGNOSES
Cardiovascular	Acute myocardial infarction Acute coronary ischemia Aortic dissection Cardiac tamponade	Unstable angina Coronary spasm Prinzmetal's angina Cocaine-induced pericarditis or myocarditis	Valvular heart disease Aortic stenosis Mitral valve prolapse Hypertrophic cardiomyopathy
Pulmonary	Pulmonary embolus Tension pneumothorax	Pneumothorax Mediastinitis	Pneumonia Pleuritis Tumor Pneumomediastinum
Gastrointestinal	Esophageal rupture (Boerhaave)	Esophageal tear (Mallory-Weiss) Cholecystitis Pancreatitis	Esophageal spasm Esophageal reflux Peptic ulcer Biliary colic
Musculoskeletal			Muscle strain Rib fracture Arthritis Tumor Costochondritis Nonspecific chest wall pain
Neurologic			Spinal root compression Thoracic outlet Herpes zoster Postherpetic neuralgia
Other			Psychologic Hyperventilation

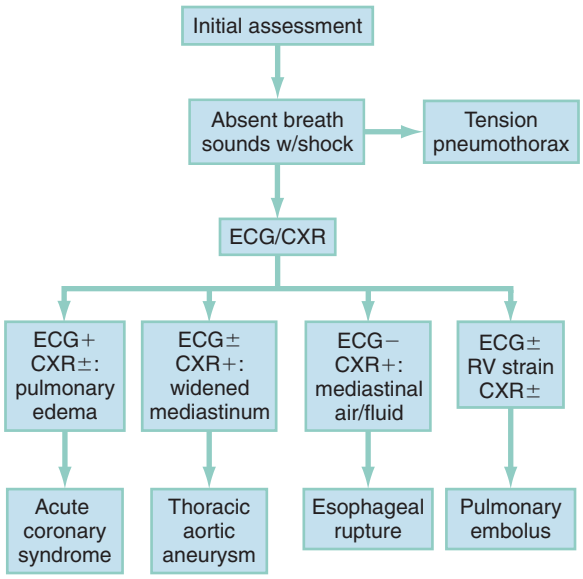

Figure 18-1. Initial assessment of critical diagnoses. CXR, chest x-ray; ECG, electrocardiogram; RV, right ventricular.

platelet agents may also be an alternative. Patients with low voltage on the ECG, diffuse ST segment elevation, elevated jugular venous pressure on examination,[8] and signs of shock should undergo prompt bedside cardiac ultrasound.

Pivotal Findings

The broad and complex nature of chest pain defies application of a simple algorithm. An organized approach to a patient with chest pain is essential, however, to ensure that all causes are evaluated appropriately. The history and physical examination are key to diagnosis. Information pertinent to the differential diagnosis is obtained by the history, physical examination, and ECG in 80 to 90% of patients.

History

1. The patient is asked to describe the character of the pain or discomfort. Descriptions such as "squeezing," "crushing," or "pressure" lead the emergency physician to suspect a cardiac ischemic syndrome, although cardiac ischemia can also be characterized by nonspecific discomfort, such as "bloating" or "indigestion." "Tearing" pain that may migrate from the front to back or back to front is the classic description in aortic dissection. "Sharp" or "stabbing" pain is seen more in pulmonary and musculoskeletal diagnoses. Patients complaining of a "burning" or "indigestion" type of pain may initially be thought to have a gastrointestinal etiology, but due to the visceral nature of chest pain, all causes of pain may present with any of the preceding descriptions. Of note, descriptors may vary among ethnic groups, and, for example, "sharp" may mean "severe."
2. Additional history about the patient's activity at the onset of pain may be helpful. Pain occurring during exertion suggests an ischemic coronary syndrome, whereas progressive onset of pain at rest suggests acute MI. Pain of sudden onset is more typical with aortic dissection, PE, or pneumothorax. Pain after meals is more indicative of a gastrointestinal cause.
3. The severity of pain is commonly quantified using a 1-to-10 pain scale. Alterations in pain severity are documented at times of onset, peak, present, and after intervention.
4. The location of the discomfort is described. Pain that is localized to a small area is more likely to be somatic versus visceral in origin. Pain localized at the periphery of the

chest is more likely with a pulmonary rather than cardiac etiology. Lower chest or upper abdominal pain may be of cardiac or gastrointestinal origin.

5. Any description of radiation of pain should be noted. Transthoracic pain through to the back should suggest aortic dissection or gastrointestinal causes, especially pancreatitis or posterior ulcer. Inferioposterior myocardial ischemia may also present primarily as thoracic back pain. Radiation to the arms, neck, or jaw increases the likelihood of cardiac ischemia.[9,10] Pain located primarily in the back, especially interscapular back pain that migrates to the base of the neck, suggests aortic dissection.[11]
6. Duration of pain is another important historical factor. Pain that lasts a few seconds is rarely of cardiac origin.[12] Pain that is exertional but lasts for only a few minutes after rest may be a manifestation of cardiac ischemia.[9] Pain that is maximal at onset may be due to aortic dissection.[11] Pain that is not severe and persists over the course of days is less likely to be of serious origin than pain that is severe or has a stuttering or fluctuating course.
7. The clinician should consider aggravating or alleviating factors. Pain that worsens with exertion and improves with rest is more likely related to coronary ischemia.[9] Pain related to meals is more suggestive of a gastrointestinal cause. Pain that worsens with respiration is seen more often with pulmonary, pericardial, and musculoskeletal causes.
8. Other associated symptoms may suggest the visceral nature of the pain (Table 18-2). Diaphoresis should lead to an increased clinical suspicion for a serious or visceral cause. Hemoptysis, a classic PE sign, is rarely seen.[13] Near-syncope and syncope lead to higher likelihood of a cardiovascular cause or PE. Dyspnea is seen in cardiovascular and pulmonary disease. Nausea and vomiting may be seen in cardiovascular and gastrointestinal complaints.
9. A history of prior pain and the diagnosis of that episode can facilitate the diagnostic process, but the physician must be wary of prior presumptive diagnoses that may be misleading. A prior history of cardiac testing, such as stress testing, echocardiography, or angiography, may be useful in determining if the current episode is suggestive of cardiac disease. Similarly, patients with previous spontaneous pneumothorax or PE[14] are at increased risk of recurrence.
10. The presence of risk factors for a particular disease is primarily of value as an epidemiologic marker for large population studies (Box 18-1). In the ED, presence of risk factors in an individual patient without established disease has minimal or no effect on the clinical likelihood (pretest probability) of a specific disease process.

Table 18-2 Significant Symptoms of Chest Pain

SYMPTOM	FINDING	DIAGNOSIS
Pain	Severe, crushing, pressure, substernal, exertional, radiation to jaw, neck, shoulder, arm	Acute MI Coronary ischemia Unstable angina Coronary spasm
	Tearing, severe, radiating to or located in back, maximum at onset, may migrate to upper back or neck	Aortic dissection
	Pleuritic	Esophageal rupture Pneumothorax Cholecystitis Pericarditis Myocarditis
	Indigestion or burning	Acute MI Coronary ischemia Esophageal rupture Unstable angina Coronary spasm Esophageal tear Cholecystitis
Associated syncope/ near-syncope		Aortic dissection PE Acute MI Pericarditis Myocarditis
Associated dyspnea (SOB, DOE, PND, orthopnea)		Acute MI Coronary ischemia PE Tension pneumothorax Pneumothorax Unstable angina Pericarditis
Associated hemoptysis		PE
Associated nausea/ vomiting		Esophageal rupture Acute MI Coronary ischemia Unstable angina Coronary spasm Esophageal tear Cholecystitis

DOE, dyspnea on exertion; MI, myocardial infarction; PE, Pulmonary embolism; PND, paroxysmal nocturnal dyspnea; SOB, shortness of breath.

Physical Examination

Specific findings may be found in a variety of causes (Table 18-3).

Ancillary Studies

The two most commonly performed studies in patients with chest pain are the chest radiograph and 12-lead ECG (Table 18-4). An ECG should be performed within 10 minutes of arrival in all patients with chest pain in whom myocardial ischemia is a possibility.[15,16] This generally includes all male patients 33 years old and older and female patients over the age of 39 who complain of pain from the umbilicus to the mandible unless a noncardiac cause is readily apparent. Rapid acquisition of the ECG facilitates the diagnosis of acute MI and expedites the National Heart, Lung, and Blood Institute's recommended "door to treatment" times from arrival to percutaneous coronary intervention (PCI) or thrombolytic therapy in acute MI. Patients with a new injury pattern on ECG (Table 18-5) or new ischemic ECG changes should have appropriate therapy instituted at this point (Fig. 18-2; see also Chapter 77). An ECG showing right ventricular strain pattern, in the appropriate setting, should raise the clinical suspicion for PE. Diffuse ST segment elevation helps make the diagnosis of pericarditis.

A chest radiograph is performed for patients with a possibly serious cause of chest pain. Pneumothorax is definitively diagnosed at this point. A wide mediastinum or ill-defined aortic knob increases the clinical suspicion for acute aortic dissection. Pleural effusion, subcutaneous air, or mediastinal air-fluid

Table 18-3 Pivotal Findings in Physical Examination

SIGN	FINDING	DIAGNOSES
Appearance	Acute respiratory distress	PE Tension pneumothorax Acute MI Pneumothorax
	Diaphoresis	Acute MI Aortic dissection Coronary ischemia PE Esophageal rupture Unstable angina Cholecystitis Perforated peptic ulcer
Vital signs	Hypotension	Tension pneumothorax PE Acute MI Aortic dissection (late) Coronary ischemia Esophageal rupture Pericarditis Myocarditis
	Tachycardia	Acute MI PE Aortic dissection Coronary ischemia Tension pneumothorax Esophageal rupture Coronary spasm Pericarditis Myocarditis Mediastinitis Cholecystitis Esophageal tear (Mallory-Weiss)
	Bradycardia	Acute MI Coronary ischemia Unstable angina
	Hypertension	Acute MI Coronary ischemia Aortic dissection (early)
	Fever	PE Esophageal rupture Pericarditis Myocarditis Mediastinitis Cholecystitis
	Hypoxemia	PE Tension pneumothorax Pneumothorax
Cardiovascular examination	Significant difference in upper extremity blood pressures	Aortic dissection
	Narrow pulse pressure	Pericarditis (with effusion)
	New murmur	Acute MI Aortic dissection Coronary ischemia
	S_3/S_4 gallop	Acute MI Coronary ischemia
	Pericardial rub	Pericarditis
	Audible systolic "crunch" on cardiac auscultation (Hamman's sign)	Esophageal rupture Mediastinitis
	JVD	Acute MI Coronary ischemia Tension pneumothorax PE Pericarditis
Pulmonary examination	Unilateral diminished/absent breath sounds	Tension pneumothorax Pneumothorax
	Pleural rub	PE
	Subcutaneous emphysema	Tension pneumothorax Esophageal rupture Pneumothorax Mediastinitis
	Rales	Acute MI Coronary ischemia Unstable angina
Abdominal examination	Epigastric tenderness	Esophageal rupture Esophageal tear Cholecystitis Pancreatitis
	Left upper quadrant tenderness	Pancreatitis
	Right upper quadrant tenderness	Cholecystitis
Extremity examination	Unilateral leg swelling, warmth, pain, tenderness, or erythema	PE
Neurologic examination	Focal findings Stroke	Aortic dissection Acute MI Coronary ischemia Aortic dissection Coronary spasm

JVD, jugular venous distention; MI, myocardial infarction; PE, pulmonary embolism.

BOX 18-1 RISK FACTORS ASSOCIATED WITH POTENTIALLY CATASTROPHIC CAUSES OF CHEST PAIN

- Acute coronary syndromes
  - Past or family history of coronary artery disease
  - Age
    - Men >33 years
    - Women >40 years
  - Diabetes mellitus
  - Hypertension
  - Cigarette use/possible passive exposure
  - Elevated cholesterol (LDL)/triglycerides
  - Sedentary lifestyle
  - Obesity
  - Postmenopausal
  - Left ventricular hypertrophy
  - Cocaine abuse
- Pulmonary embolism
  - Prolonged immobilization
  - Surgery >30 minutes in last 3 mo
  - Prior deep vein thrombosis or pulmonary embolus
  - Pregnancy or recent pregnancy
  - Pelvic or lower extremity trauma
  - Oral contraceptives with cigarette smoking
  - Congestive heart failure
  - Chronic obstructive pulmonary disease
  - Obesity
  - Past medical or family history of hypercoagulability
- Aortic dissection
  - Hypertension
  - Congenital disease of the aorta or aortic valve
  - Inflammatory aortic disease
  - Connective tissue disease
  - Pregnancy
  - Arteriosclerosis
  - Cigarette use
- Pericarditis or myocarditis
  - Infection
  - Autoimmune disease (e.g., systemic lupus erythematosus)
  - Acute rheumatic fever
  - Recent myocardial infarction or cardiac surgery
  - Malignancy
  - Radiation therapy to mediastinum
  - Uremia
  - Drugs
  - Prior pericarditis
- Pneumothorax
  - Prior pneumothorax
  - Valsalva's maneuver
  - Chronic lung disease
  - Cigarette use

Table 18-4 Ancillary Testing of Patients with Chest Pain

TEST	FINDING	DIAGNOSIS
ECG	New injury	Acute MI Aortic dissection
	New ischemia	Coronary ischemia Coronary spasm
	RV strain	PE
	Diffuse ST segment elevation	Pericarditis
CXR	Pneumothorax with mediastinal shift	Tension pneumothorax
	Wide mediastinum	Aortic dissection
	Pneumothorax	Esophageal rupture Pneumothorax
	Effusion	Esophageal rupture
	Increased cardiac silhouette	Pericarditis
	Pneumomediastinum	Esophageal rupture Mediastinitis
ABG	Hypoxemia, A-a gradient	PE
$\dot{V}/\dot{Q}$ scan or spiral CT	High probability or any positive in patient with high clinical suspicion	PE

ABG, arterial blood gas; CT, computed tomography; ECG, electrocardiogram; MI, myocardial infarction; RV, right ventricular.

Table 18-5 Electrocardiogram Findings in Ischemic Chest Pain

Classic myocardial infarction	ST segment elevation (>1 mm) in contiguous leads; new LBBB; Q waves ≥0.04 sec duration
Subendocardial infarction	T wave inversion or ST segment depression in concordant leads
Unstable angina	Most often normal or nonspecific changes; may see T wave inversion
Pericarditis	Diffuse ST segment elevation; PR segment depression

LBBB, left bundle-branch block.

level may be seen in esophageal rupture. Increased cardiac silhouette may indicate pericarditis or cardiomyopathy.

Pneumomediastinum is seen with esophageal rupture and mediastinitis. A serum D-dimer assay may help discriminate patients with PE from those with a possible gastrointestinal cause. A low serum D-dimer in a patient without a high pretest probability of PE effectively excludes the diagnosis.[13,17,18] (see Chapter 87.)

Patients at high pretest probability for PE should undergo diagnostic imaging (multidetector computed tomography [CT], or, less commonly, pulmonary angiography or a ventilation-perfusion lung scan).[19] High pretest probability warrants initiation of anticoagulation (heparin or low-molecular-weight heparin) therapy in the ED before the imaging study, in the absence of a contraindication.

Patients with suspected thoracic aortic dissection may be evaluated by CT angiography, transesophageal echocardiography, or magnetic resonance imaging. Selection of imaging modality depends on patient status and availability of the testing equipment.[20]

CT with a 64 or higher detector scanner has the potential to rule out all of the life-threatening causes of chest pain. Although the "triple rule out" of ACS, PE, and thoracic dissection are the causes most commonly discussed, pneumothorax, mediastinitis, and pericardial effusions are also diagnosed with CT.[21,22]

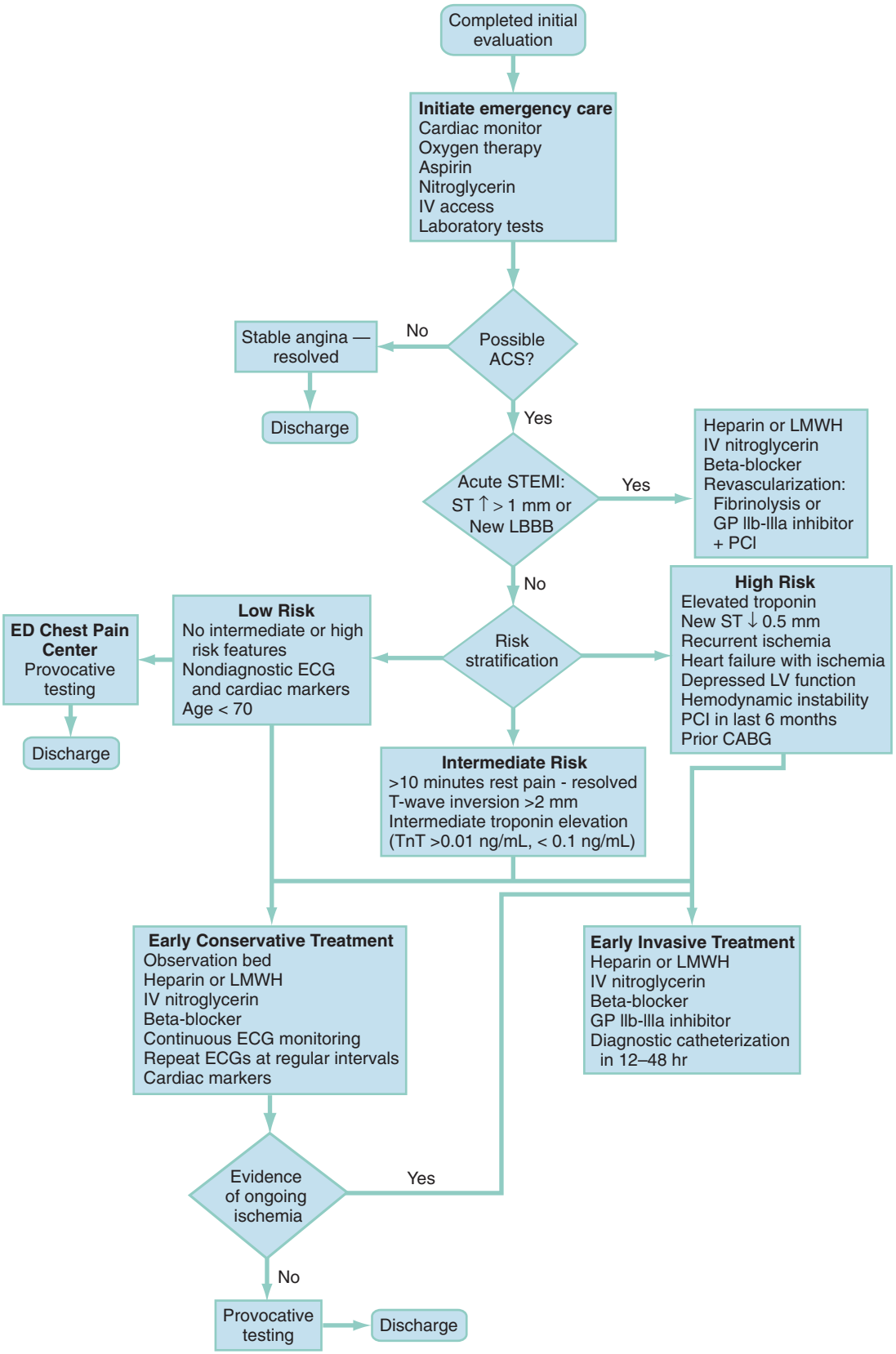

Figure 18-2. Clinical guidelines for emergency department management of chest pain of myocardial ischemic origin. ACS, acute coronary syndrome; CABG, coronary artery bypass graft; ECG, electrocardiogram; GP, glycoprotein; IV, intravenous; LBBB, left bundle-branch block; LMWH, low-molecular-weight heparin; LV, left ventricular; MI, myocardial infarction; PCI, percutaneous coronary intervention; ST, echocardiographic peak; STEMI, ST segment evaluation myocardial infarction; TnT, troponin T. (Adapted from Gibler WB, Cannon CP, Blonikalns AL, et al: Practical implementation of the guidelines for unstable angina/non-ST-segment elevation myocardial infarction in the emergency department: A scientific statement from the American Heart Association Council on Clinical Cardiology (Subcommittee on Acute Cardiac Care), Council on Cardiovascular Nursing, and Quality of Care and Outcomes Research Interdisciplinary Working Group, in Collaboration with the Society of Chest Pain Centers. Circulation 111:2699, 2005.)

Table 18-6 Causes and Differentiation of Potentially Catastrophic Illness Presenting with Central Chest Pain or Discomfort

	PAIN HISTORY	ASSOCIATED SYMPTOMS	SUPPORTING HISTORY	PREVALENCE IN EMERGENCY DEPARTMENT	PHYSICAL EXAMINATION	USEFUL TESTS	ATYPICAL OR ADDITIONAL ASPECTS
Myocardial Infarction	Discomfort is usually moderately severe to severe and rapid in onset. May be more "pressure" than pain. Usually retrosternal, may radiate to neck, jaw, both arms, upper back, epigastrium, and sides of chest (left more than right). Lasts more than 15–30 min and is unrelieved by NTG	Diaphoresis, nausea, vomiting, dyspnea	May be precipitated by emotional stress or exertion. Often comes on at rest. May come on in early awakening period. Prodromal pain pattern often elicited. Previous history of MI or angina. Age >40 years, positive risk factors, and male sex increase possibility	Common	Patients are anxious and uncomfortable. Blood pressure usually is elevated, but normotension and hypotension are seen. The heart rate is usually mildly increased, but bradycardia can be seen. Patients may be diaphoretic and show peripheral poor perfusion. There are no diagnostic examination findings for MI, although S_3 and S_4 heart sounds and new murmur are supportive	ECG changes (new Q waves or ST segment–T wave changes) occur in 80% of patients. CK-MB and troponins are helpful if elevated, but may be normal	Pain may present as "indigestion" or "unable to describe." Other atypical presentations include altered mental status, stroke, angina pattern without extended pain, severe fatigue, syncope. Elderly may present with weakness, congestive heart failure, or chest tightness. 25% of nonfatal MIs are unrecognized by patient. The pain may have resolved by the time of evaluation
Unstable Angina	Changes in pattern of preexisting angina with more severe, prolonged, or frequent pain (crescendo angina). Pain usually lasts >10 min. Angina at rest lasting 15–20 min or new-onset angina (duration <2 mo) with minimal exertion. Pattern of pain change important in gauging risk for AMI. Unpredictable responses to NTG and rest	Often minimal. May have mild diaphoresis, nausea, dyspnea with pain. Increasing pattern of dyspnea on exertion	Not clearly related to precipitating factors. May be a decrease in amount of physical activity that initiates pain. Previous history of MI or angina. Over 40 years old, presence of risk factors, and male sex increase probability	Common	Nonspecific findings of a transient nature, may have similar cardiac findings as in MI, especially intermittent diaphoresis	Often no ECG or enzyme changes. Variant angina (Prinzmetal's) has episodic pain, at rest, often severe, with prominent ST segment elevation	May be pain-free at presentation. Full history is essential. Fewer than 15% of patients hospitalized for unstable angina go on to acute MI. May respond to NTG. May manifest similarly to non–Q wave infarction

Aortic Dissection	90% of patients have rapid-onset severe chest pain that is maximal at beginning. Radiates anteriorly in chest to the back interscapular area or into abdomen. Pain often has a "tearing" sensation, and may migrate	Neurologic complications of stroke, peripheral neuropathy, paresis or paraplegia, abdominal and extremity ischemia possible	Median age 59 years. History of hypertension in 70–90% of patients. 3:1 ratio males to females. Marfan's syndrome and congenital bicuspid aortic valves have increased incidence	Rare	Often poorly perfused peripherally but with elevated BP. In 50–60% of cases, there is asymmetrical decrease or absence of peripheral pulses. 50% of proximal dissections cause aortic insufficiency. Other vascular occlusions: coronary (1–2%), mesentery, renal, spinal cord. New-onset pericardial friction rub or aortic insufficiency murmur supportive of diagnosis	ECG usually shows left ventricular hypertrophy, nonspecific changes. Chest film shows abnormal aortic silhouette (90%). Aortic angiography has diagnostic accuracy of 95–99%. Transesophageal echocardiogram, CT, MRI most useful in screening	Rare for patient to present pain-free. May present with neurologic complications. Physical examination findings may be minimal. Dissection into coronary arteries can mimic MI Ascending aortic aneurysms are more often approached surgically. Descending are generally managed medically
Pulmonary Embolism	Pain is more often lateral-pleuritic. Central pain is more consistent with massive embolus. Abrupt in onset and maximal at beginning. May be episodic or intermittent	Dyspnea and apprehension play a prominent role, often more than pain. Cough accompanies about half the cases Hemoptysis occurs in <20%. Angina-like pain may occur in 5%	Often some period of immobilization has occurred, e.g., postoperative. Pregnancy, oral contraceptives, heart disease, and cancer are all risk factors. Previous DVT or PE is the greatest risk factor	Uncommon in ambulatory patients, but common in departments with high volumes of elderly or medically complex patients	Patients are anxious and often have a respiratory rate >16/min. Tachycardia, inspiratory rales, and an increased pulmonic second sound are common. Fever, phlebitis, and diaphoresis are seen in 30–40% of patients. Wheezes and peripheral cyanosis are less common	Arterial blood gases show Po_2 < 80 mm Hg in 90%. Widened A-a gradient is helpful. Chest film is usually normal, although 40% show some volume loss, oligemia, or signs of consolidation due to pulmonary infarction. Lung perfusion scan rules out, if truly negative	Patients may present with dyspnea with or without bronchospasm. Acute mortality rate is 10%. Emboli usually from lower extremities above knee, prostate/pelvis venous plexus, right heart. May be subtle cause of COPD exacerbation

Table 18-6 Causes and Differentiation of Potentially Catastrophic Illness Presenting with Central Chest Pain or Discomfort—cont'd

	PAIN HISTORY	ASSOCIATED SYMPTOMS	SUPPORTING HISTORY	PREVALENCE IN EMERGENCY DEPARTMENT	PHYSICAL EXAMINATION	USEFUL TESTS	ATYPICAL OR ADDITIONAL ASPECTS
Pneumothorax	Pain is usually acute and maximal at onset. Most often lateral-pleuritic, but central pain can occur in large pneumothorax	Dyspnea has a prominent role. Hypotension and altered mental states occur in tension pneumothorax	Chest trauma, previous episode, or asthenic body type	Infrequent	Decreased breath sounds, increased resonance on percussion. Elevated pressure in neck veins occurs in tension pneumothorax	Chest film definitive. Inspiratory and expiratory films may enhance contrast between air and lung parenchyma. Tension pneumothorax should be diagnosed on physical examination	May be subtle in COPD, asthma, cystic fibrosis. Can be complicated by pneumomediastinum
Esophageal Rupture	Pain usually is preceded by vomiting and is abrupt in onset. Pain is persistent and unrelieved, localized along the esophagus, and increased by swallowing and neck flexion	Diaphoresis, dyspnea (late), shock	Older individual with known gastrointestinal problems. History of violent emesis, foreign body, caustic ingestion, blunt trauma, alcoholism, esophageal disease	Rare	Signs of lung consolidation, subcutaneous emphysema may be present	Chest film usually has mediastinal air, a left-sided pleural effusion, pneumothorax, or a widened mediastinum. pH of pleural effusion is <6.0. Diagnosis supported by water-soluble contrast esophagram or esophagoscopy	Patient may present in shock state. This entity often considered late in differential diagnostic process
Pericarditis	Dull, aching recurrent pain unrelated to exercises or meals. Or it may be a sharp, stabbing, pleuritic-type pain that does not change with chest wall motion. May be severe. Not relieved by NTG	Dyspnea, diaphoresis	Pain is often worse when supine, but improves sitting up. Often preceded by viral illness or underlying disease (SLE or uremia)	Rare Tamponade even more rare complication	Friction rub may be heard, often fleeting, position-dependent (50% of patients).	ECG pattern typical for ST segment elevation across the precordial leads. Erythrocyte sedimentation rate may be elevated	More common in 20- to 50-year-olds. May have associated tachycardias, ventricular dysrhythmias. Idiopathic most common (80%). Treated with aspirin, NSAID

AMI, acute myocardial infarction; CK-MB, an isoform of creatine kinase; COPD, chronic obstructive pulmonary disease; CT, computed tomography; DVT, deep vein thrombosis; ECG, electrocardiogram; MRI, magnetic resonance imaging; NSAID, nonsteroidal anti-inflammatory drug; NTG, nitroglycerin; PE, pulmonary embolus; SLE, systemic lupus erythematosus.

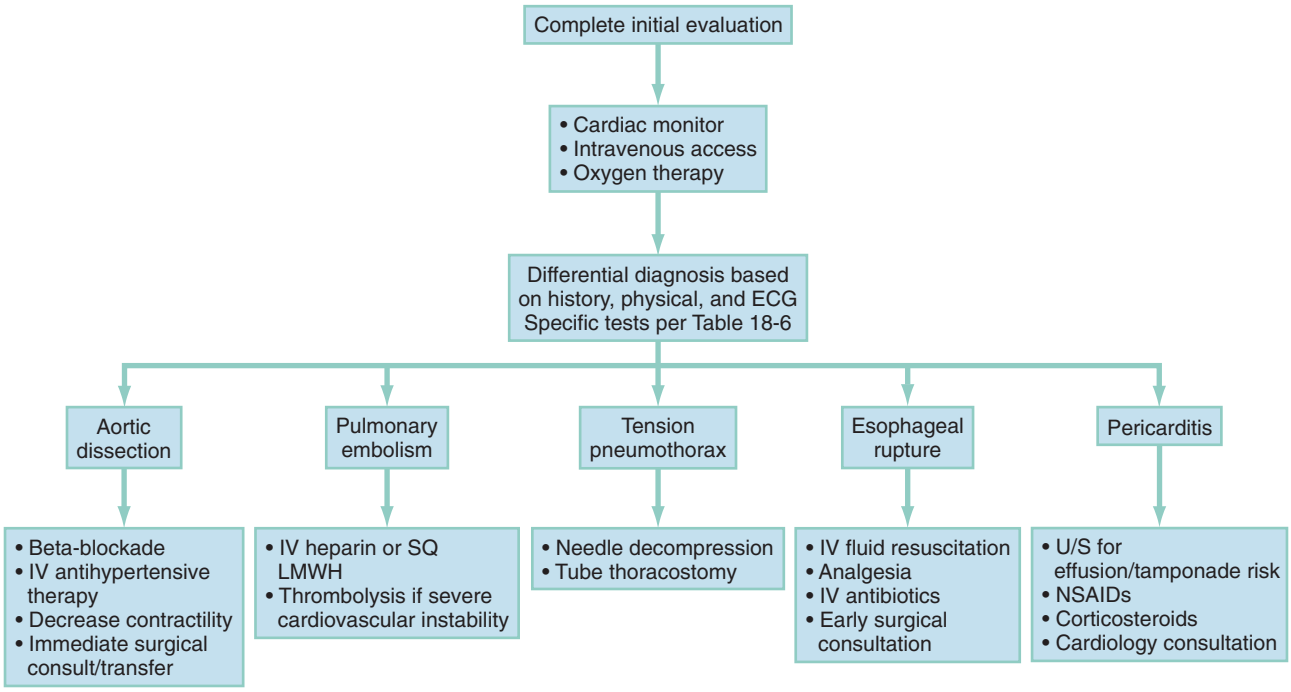

Figure 18-3. Clinical guidelines for emergency department management of chest pain from potentially catastrophic nonmyocardial origins. ECG, electrocardiogram; IV, intravenous; NSAIDs, nonsteroidal anti-inflammatory drugs; SQ, subcutaneous; LMWH, low-molecular-weight heparin; U/S, ultrasound.

Laboratory testing is useful in the evaluation of ACS. Creatine kinase (CK) is associated with multiple false-positive results and has no use in the evaluation of unstable angina. CK-MB, an isoform of CK, is more specific for cardiac ischemia. Evaluating this enzyme produces fewer false-positive results, and peak sensitivity approaches 98%. Sensitivity at 4 hours is, however, only about 60%. CK-MB isoforms improve sensitivity at 4 hours to 80%, approaching 93% at 6 hours. The current universal definition of MI places CK and CK-MB in a secondary role to troponins.[23]

Troponins (I and T), when elevated, identify patients with ACS who have the highest risk for an adverse outcome.[23,24] Sensitivity for acute MI at 4 hours is 60%, rising to nearly 100% by 12 hours.[25,26] Elevated troponin in the correct clinical setting is synonymous with acute MI and is embedded in the universal definition of MI.

DIAGNOSTIC TABLE

After the patient is stabilized and assessment has been completed, the findings are matched to the classic and atypical patterns of the seven potentially critical diseases causing chest pain. This matching process is continual while evaluating the patient and monitoring the response to therapy. Any inconsistency in findings with the primary working diagnoses requires a rapid review of the pivotal findings and the potential diagnoses (Table 18-6).

MANAGEMENT AND DISPOSITION

The management of ACS is discussed in Chapter 76. Figure 18-3 outlines the approach to treatment of critical noncardiac diagnoses. Patients with critical diagnoses generally are admitted to the intensive care unit. Patients with emergent diagnoses typically are admitted to the hospital, most often on telemetry units. Patients with nonemergent diagnoses are most frequently treated as outpatients. Hospitalization is required in certain circumstances, particularly when patients have other comorbid conditions.

Frequently, no definitive diagnosis is established. Any patient with almost any type of chest pain may be having coronary ischemia, PE, or aortic dissection. When a clear pattern does not emerge to allow the emergency physician to make an alternative diagnosis confidently, continued evaluation, hospitalization, or observation admission may be the best course.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.