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Acute and Chronic Chest Pain

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Objectives

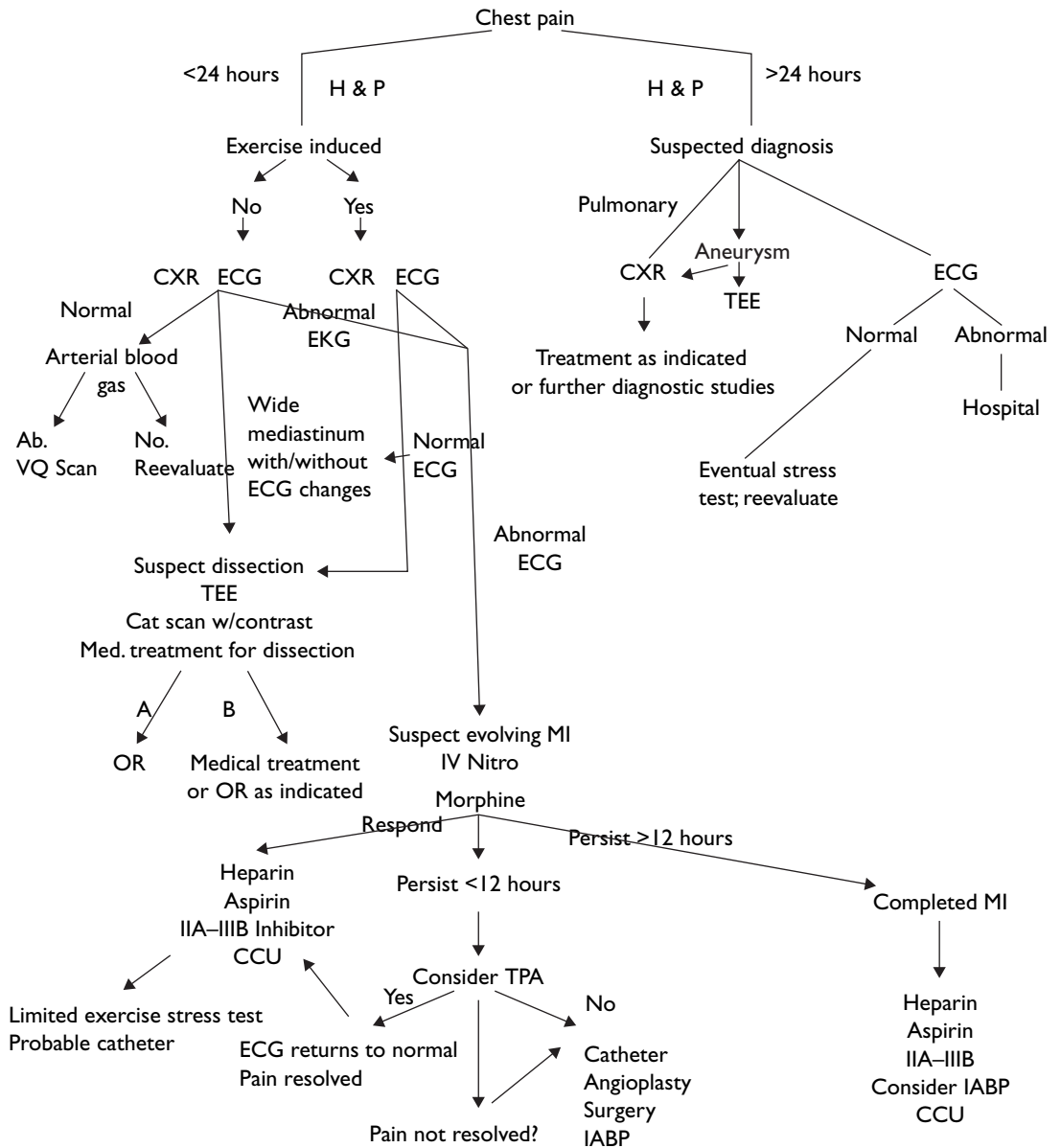
1. To understand the differential diagnosis of chest pain requiring cardiac surgical consultation.
 2. To understand the physiology of and rationality for medical treatment of ischemic coronary artery disease.
 3. To differentiate acute aortic dissection from myocardial infarction in the emergency setting.
 4. To understand initial stabilization of the patient with aortic dissection.
 5. To recognize the risk factors associated with open-heart surgery.
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Case

A 65-year-old man presents to the emergency room complaining of “chest pain.” He drove himself to the hospital. The pain has lasted about an hour and is now going away. He has a history of a hiatus hernia and esophageal reflux. He denies any other associated symptoms. He took an antacid without relief. Because he failed to get any pain relief, he took one of his 87-year-old father’s nitroglycerin tablets, and the pain started to ease. He came to the emergency room. He is now pain free and wants to go home. Vital signs are normal except for a heart rate of 100. How do you proceed?

Introduction

This chapter discusses the causes of chest pain that may require intervention by a cardiac surgeon, distinguishes them from other causes that are of less concern, and provides a systematic approach by which the diagnosis and early treatment of these conditions can be begun before the cardiac surgeon arrives. (See Algorithm 16.1.) Often, these



Algorithm 16.1. Algorithm for initial evaluation and treatment of patients with chest pain. CCU, cardiac care unit; CXR, chest x-ray; ECG, electrocardiogram; H&P, history and physical exam; IABP, intraaortic balloon pump; MI, myocardial infarction; OR, operating room; TEE, transesophageal echocardiography; TPA, tissue plasminogen activator.

emergencies occur at night. **Early notification of the surgical team may save precious minutes** in getting a patient through the necessary diagnostic studies and into the operating room when a lifesaving operation is required.

Differential Diagnosis

The major diagnoses associated with chest pain and treated by cardiac surgeons that should concern both medical personnel and patients include **ischemic heart disease, diseases of the thoracic aorta, diseases of the pericardium, and pulmonary embolism**. There are other diseases treated by general thoracic surgeons that are not discussed here. The case described above is relatively nonspecific, but it could describe a scenario for any of these life-threatening diagnoses.

Ischemic Heart Disease

The term **ischemic heart disease** is descriptive of a broad range of clinically significant diagnoses with a common origin. **The underlying pathogenesis in all of these is the mismatch of oxygen supply and oxygen demand of the myocardium**. The most common descriptor of the chest pain associated with this etiology is **angina pectoris**.

The most commonly recognized cause of ischemic heart disease is the occlusion of large epicardial vessels in the heart by atherosclerotic cardiovascular disease. There are other etiologies, however, including valvular heart disease, vasculitis, congenital coronary artery anomalies, episodes of coronary artery spasm related to cocaine use or other causes (Prinzmetal's angina), and dissection of the thoracic aorta when the ostia of a coronary artery are involved.

Classically, angina pectoris is characterized by substernal chest pain or pressure that may radiate down the arm. However, these symptoms may be present in less than 25% of patients with ischemic heart disease. **Other "anginal equivalents" include jaw pain, throat pain, arm pain, dyspnea on exertion, and frank pulmonary edema**. Patients with diabetes are notorious for having "silent ischemia." This means that they do not develop any symptoms of pain when ischemia occurs. They may present with previously unrecognized myocardial infarction or with an episode of acute pulmonary edema.

At one extreme of the spectrum of patients with ischemic heart disease are those with chronic stable angina. Evaluation of previous anginal episodes has resulted in this diagnosis, and the presenting problem is just another manifestation of the underlying cause. **At the other extreme are patients who die suddenly or present with an acute evolving myocardial infarction in cardiogenic shock**. In between these extremes are the patients with **new-onset angina, unstable angina, preinfarction angina, non-Q-wave myocardial infarction, and myocardial infarction without shock**.

In **chronic stable angina pectoris**, short episodes leading to ischemia of the heart muscle occur, but they reverse rapidly without significant damage to the myocardium. This pattern may go on for years, but a

progressive course is more likely. **Unstable angina** is descriptive of a scenario in which new symptoms occur in a previously asymptomatic individual or in which a chronic pattern of pain associated with certain activities becomes more frequent or severe. **Rest angina**, as the term implies, **occurs in the absence of stressful activity and is an ominous sign**. **Myocardial infarction** is the end result of this process. The ischemia lasts so long that actual tissue necrosis occurs. **Q-wave myocardial infarction** is descriptive of the damage that occurs when transmural (all layers of the myocardial wall) infarction occurs and is manifest by Q-waves on the electrocardiogram (ECG). **Cardiogenic shock** results when the amount of myocardium that becomes dysfunctional due to ischemia or infarction is so large that the remaining myocardium cannot adequately maintain the systemic circulation.

To quantify the different levels of ischemia that occur before infarction and to suggest the resulting levels of risk for the patient, the **New York Heart Association Classification of Angina** (or other cardiac symptoms) was developed. It is far from accurate and is purely qualitative, but it does provide some frame of reference (**Table 16.1**).

Atherosclerosis is recognized as the major cause of coronary artery disease. It is a progressive disease that can appear microscopically even in infants. Many factors, including **genetic factors, hypertension, dietary indiscretion (including early in life), and diabetes**, have been recognized as contributing to the development of atherosclerotic cardiovascular disease. These cannot be altered. Other factors, such as **obesity, hypercholesterolemia, and smoking** (all major contributors to atherosclerotic disease), can be modified. **This modification can significantly alter the progressive course of the disease**. In the heart, atherosclerotic lesions tend to develop in the proximal portions of the coronary arteries and at major branch points. Patients with diabetes are

Table 16.1. New York Heart Association functional classification of angina.

Class	Description
I	Patients with cardiac disease but without resulting limitations of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea, or anginal pain.
II	Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain.
III	Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary physical activity causes fatigue, palpitation, dyspnea, or anginal pain.
IV	Patient with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency or of the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.

Source: Reprinted from The Criteria Committee of the New York Heart Association. *Nomenclature and Criteria for Diagnosis*, 9th ed. Boston: Little, Brown, 1994. With permission from Lippincott Williams & Wilkins.

likely to have more diffuse disease. Once significant stenoses have developed, symptoms may begin to manifest themselves, especially at times of stress and increased oxygen demand in the heart.

Referring to our case scenario, this patient's presentation certainly could be that of ischemic heart disease. There is a family history, he received some mild relief from nitroglycerin, and he is at the age at which atherosclerotic heart disease has a high incidence. Were you to encounter such a patient and were you to believe his pain was from acute myocardial ischemia, a **physical examination** needs to be done and **further history** needs to be obtained (as described below). Appropriate laboratory studies need to be initiated. These should include an **electrocardiogram, a chest x-ray, and cardiac enzyme screen**. Electrocardiogram may be normal at this point, but it also may be suggestive of some ongoing ischemia or previous injury to the heart. A chest x-ray might reveal signs of cardiac enlargement and may be suggestive of signs of heart failure, should there be any. Finally, cardiac enzymes drawn at this point may or may not be positive, even in the presence of ischemic disease. At the present time, **troponin levels are the most sensitive laboratory study to do for signs of myocardial injury. Creatinine phosphokinase (CPK-MB) levels specifically attributed to the heart also should be obtained.** Based on this information, the history, and the physical examination, a determination should be reached as to the likelihood of ischemic disease and how to proceed further.

Diseases of the Thoracic Aorta

Diseases of the thoracic aorta are **far more prevalent than commonly is recognized and should be considered in the differential diagnosis of any patient complaining of significant chest pain.** Involvement of the thoracic aorta takes two forms: **aneurysmal disease alone or aortic dissection that can occur with or without the presence of aneurysmal disease** and has potentially catastrophic consequences.

Aneurysms of the thoracic aorta are of two types: **saccular** and **fusiform**. The former occurs as an outpouching off the side of the vessel, while the latter consists of a dilatation of a segment of the aorta. **The definition of a true aneurysm requires that all three layers of the normal wall are present (intima, media, and adventitia) and that the diameter of the aorta in the diseased segment is at least twice its normal diameter.** Atherosclerosis frequently is associated with or is the etiology of these aneurysms. Others may be **idiopathic** in origin or be a manifestation of a **connective tissue disorder, the most common of which is Marfan's syndrome.**

Symptoms of aneurysms frequently are related to pressure on adjacent structures from the enlarging aorta. Chest pain may be related to the enlarging aorta itself. Complaints of back pain, hoarseness, cough, shortness of breath, and dysphagia may be present from encroachment on the thoracic spine, recurrent laryngeal nerve, trachea or bronchus, or esophagus, respectively. Often, an aneurysm first may be discovered on a routine chest x-ray or computed axial

tomography (CAT) scan obtained for some other reason. There may be no associated symptoms. Surgical repair of these aneurysms usually is recommended when they become large because of the risk of rupture and sudden death. **Once rupture has occurred, the likelihood of survival is low.**

Aortic dissection is far more likely to present as an emergency than is a thoracic aortic aneurysm. Aortic dissection is the cause of acute mortality almost twice as often as acute rupture of an abdominal aortic aneurysm. Although it does not fit the definition of an aneurysm as given above, the term **dissecting aortic aneurysm** frequently is applied. The underlying aorta may be normal in character or aneurysmal prior to the onset of the dissection. **Aortic dissection always must be considered in the emergency setting, as it can be difficult to distinguish from a myocardial infarction. Thrombolytic agents used to treat a myocardial infarction may lead to death when the etiology is aortic dissection.**

A dissecting aortic aneurysm frequently presents with the acute onset of severe pain. The pain may be similar to angina with a **crushing type of pressure or pain**. If the ascending aorta is involved and the dissection continues distally, the pain may migrate to the back. Some patients may describe a **tearing pain between the scapulae** and refer to it as the worst pain they have ever felt. **The pain may be localized to the upper abdomen at times and may be confused with an abdominal aneurysm rupture, perforated ulcer, or cholecystitis.** Because of the nature of the problem, associated symptoms can be multiple and often are related to the loss of blood supply to major organs due to the shearing off or occlusion of major side branches. Patients may present with signs of stroke, renal failure, bowel ischemia, or limb ischemia. If the ostium of a main coronary artery is involved, there may be signs of ischemia present on the ECG. This is most common involving the right coronary. New-onset aortic insufficiency is the final sign that should not be overlooked in a patient suspected of aortic dissection. The association of chest pain with loss of one or more peripheral pulses or new-onset aortic insufficiency murmur is the *sine qua non* of aortic dissection.

There are two classifications of aortic dissections commonly used. They are descriptive of that segment of the aorta involved. As the treatment of aortic dissections has evolved, the newer Stanford classification was developed as an additional aid in determining the type of treatment required. In **type A dissections, the ascending aorta is involved**. The dissection may be isolated to this segment or extend into the aortic arch and the descending thoracic aorta or even the abdominal aorta. **In type B dissections, only the descending aorta is involved.** Unless contraindicated, **all type A dissections should undergo emergency surgical repair**, as the mortality with medical treatment alone is extremely high. More than 70% of patients with a type A dissection who arrive in the emergency room die in the first 48 hours if not operated on. **Usually, type B dissections are treated medically unless a specific complication or sign develops.** Some of these complications are listed in Table 16.2.

Table 16.2. Relative indications for surgery in type B aortic dissection.

Evidence of free rupture into pleural space (hemothorax)
Evidence of increasing mediastinal hematoma
Ischemia of a significant vital organ
Recurrent pain after 24 hours of onset of the original episode
Inability to control pain within 24 hours
Inability to control blood pressure within 24 hours, especially if once symptoms persist

Referring again to our case, aneurysmal disease or, more likely, aortic dissection could be a tentative diagnosis in this patient. Age, once again, is appropriate, and male gender is appropriate: aortic dissection is somewhat more common in males than females. If this is one suspected diagnosis, **while history and physical examination are being completed, electrocardiogram and chest X-ray** are the initial diagnostic studies. If a dissection truly is considered, the team that performs **transesophageal echocardiography** needs to be notified immediately so that it can begin to mobilize the equipment required to perform the study (see Diagnostic and Confirmatory Studies, below). Electrocardiogram is likely to be normal, but it may show signs of ischemia, especially in the distribution of the right coronary artery. Chest x-ray could be normal in the presence of a dissection, but more likely one may see significant widening of the mediastinum, a straightening of the mediastinal stripe on the left side, and even signs of left pleural effusion or hemothorax.

Pericardial Disease

Diseases of the pericardium may present with a broad spectrum of symptoms and etiologies. These range from **simple, nonspecific acute pericarditis to larger pericardial effusions, tamponade, or constrictive pericarditis**. Constrictive pericarditis may be the ultimate sequela to acute pericarditis and appear months to years after the acute episode. This is especially true of tuberculous pericarditis.

Etiologies of pericarditis are numerous. **A nonspecific viral infection** is the most common cause in the adult, but **significant purulent pericarditis of a bacterial origin** can occur, especially in children. Other etiologies include **renal failure, dialysis, postcardiac surgery, following irradiation to the mediastinum, rheumatoid disease, sarcoidosis on rare occasions, and classically, with previous tuberculous pericarditis**. Simple pericarditis represents as an inflammatory process involving the pericardium. Pericardial pain can be quite disabling, sharp, and pleuritic in nature. It usually is retrosternal, may radiate to the neck or left shoulder, and often may be relieved by the patient leaning forward.

Significant pericardial effusions can occur from any of the etiologies described above. As fluid gradually accumulates, the pericardial sac can expand without hemodynamic compromise and accumulate **up to**

3 or 4 L of fluid. In the presence of a febrile illness, significant effusion should raise concerns about an infectious nature. **Bacterial, tuberculous, and fungal etiologies all have been recognized and may require fluid aspiration or pericardial biopsy for diagnosis.** When the rate of fluid accumulation exceeds the ability of the pericardium to expand, **tamponade will develop.** Characteristically, patients with tamponade present with chest fullness and may be in extremis with tachycardia, tachypnea, and agitation. **Beck's triad is classically descriptive of those patients with acute tamponade; venous distention, hypotension, and a small quiet heart are characteristic on exam.** **Pulsus paradoxus** is a classic finding associated with tamponade, either acute or chronic. Its etiology is complex and not fully understood. It is thought to be due to hemodynamic changes secondary to external pressure on the heart. This results in a leftward shift of the ventricular septum that, in turn, prevents adequate filling of the left ventricle during diastole and leads to a decrease in systolic blood pressure. **Clinically, pulsus paradoxus is characterized by at least a 10 mmHg drop in systolic pressure associated with normal inspiration.** An asthmatic may show similar alteration in blood pressure that should not be confused with the pulsus paradoxus of cardiac tamponade.

Chronic constrictive pericarditis is the end stage of the spectrum of pericardial disease. The pericardium can become quite **thickened, rigid, and may be calcified.** Patients with constrictive pericarditis can present in what appears to be late stages of profound heart failure with low cardiac output. These end-stage patients have a potentially high mortality with or without surgical intervention.

Physical examination is usually nonspecific in the absence of tamponade. Frequently, **a pericardial friction rub may be heard, which is classically diagnostic of the problem, and neck vein distention may be present.** Referring to the case, the description is so nonspecific that it could be related to an episode of pericarditis. **Electrocardiogram, chest x-ray, and complete blood count (CBC)** are appropriate as initial screenings. Electrocardiogram may show significant **ST segment elevations throughout all leads of the ECG but without the T waves being affected significantly.** One must be careful if the ST segment elevations are limited only to regional myocardial coronary distribution. Suspicion of myocardial ischemia rather than pericarditis should be raised if this is the case. Chest x-ray is likely to be normal in the early stages of pericarditis. However, if large amounts of pericardial fluid have accumulated, increases in the cardiac silhouette may occur. (It often is estimated that at least 500 cc of pericardial fluid must be present for enlargement of the cardiac silhouette to be noted.) The result of the CBC will give any suggestion of infection or inflammatory processes.

Pulmonary Embolism

Pulmonary embolism is another major concern in the differential diagnosis of patients with new onset of chest pain. The embolus to the lung, however, is always a **consequence of disease elsewhere in the body.** Usually, this represents venous thrombosis involving the inferior vena

cava, the pelvic veins in women, or the ileofemoral and deep veins of the leg. (See the chapter on venous disease.) Embolization can occur from upper extremities thrombosis, but it is rare. Tumor embolization also can occur from tumors involving the inferior vena cava or the right side of the heart. Multiple septic emboli from patients with tricuspid valve endocarditis also are causes of this problem.

The presentation can be variable. Classically, a patient presents with **tachycardia, tachypnea, pleuritic chest pain, hemoptysis, cyanosis, elevated venous pressure, or total cardiovascular collapse**. New-onset atrial fibrillation may be present and accompany the onset of symptoms. **Any of these findings** in a postoperative patient, a patient with prolonged bed rest, or others susceptible to deep vein thrombosis **should raise the possibility of pulmonary embolus**.

Again, look back to the case cited at the beginning of the chapter. Although, less likely with the presenting signs and symptoms, pulmonary embolism is certainly a possibility, though low on the differential diagnosis scale. Suspicion, however, especially if the patient complains of shortness of breath, should be raised. **The ECG is likely to be normal, but it may show signs of right ventricular strain with a new S wave in lead 1 and a new Q wave in lead 3. Chest x-ray is likely to show little significant changes, but it could show a wedge-type infiltrate or even signs of decreased perfusion to one lung or one portion of the lung. In a patient in whom the diagnosis of pulmonary embolism has been raised, especially in the presence of shortness of breath, a room air blood gas should be obtained, supplemental oxygen applied, and further diagnostic studies performed, such as ventilation/perfusion scanning, pulmonary angiography, and spiral CAT scanning.**

Diagnostic Methods

History and Physical Examination

The history and physical examination are crucial to the differential diagnosis and initial treatment of patients with chest pain. In the emergency setting, time is of the essence, and the initial diagnostic and therapeutic interventions must be begun based on this information. Without *question*, **the history remains the most valuable mode of evaluation**. The history is designed to elicit essential positive and negative information relevant to the diagnosis of the underlying cause of the patient's chest discomfort. In obtaining the history of a patient with chest pain, it is helpful to have a **mental checklist** and to ask the patient to **describe the location, radiation, and character of the discomfort; what causes and relieves it; time relationships, including the duration, frequency, and pattern of recurrence of the discomfort; the setting in which it occurs; and associated symptoms**.

Because of the nonspecific presentations of the various pathophysiologies described, care must be taken in obtaining a history. Asking a patient "Do you have chest pain?" may result in a negative answer when a patient, in fact, is having significant chest discomfort. More

generalized descriptions may be required such as chest pressure, chest discomfort, respiratory pain, etc. Similarly, activities that relieve the symptoms, such as resting, changing position, taking a deep breath while leaning forward, etc., must be documented. Angina must be differentiated from other causes that may mimic its symptoms as listed in **Table 16.3**. It can be confused with the epigastric discomfort of “heartburn,” the chest pain of pericarditis or pleuritis, or the discomfort of episodes of bursitis or inflammatory problems in the chest wall. Association of nausea, diaphoresis, shortness of breath, or syncope may be important clues as to etiology. Additional aspects of history should include but not be limited to **inquiries into family history; a history of prior myocardial infarction or heart murmurs; the presence of hypertension, diabetes, or connective tissue disorders; smoking, exercise, dietary habits, and other factors that might predispose the patient to one diagnosis or another or play a significant role in decisions about diagnostic studies and therapeutic interventions.**

The initial physical examination is directed toward eliciting findings consistent with or excluding a diagnosis suggested by the initial history. The **vital signs and general appearance** of the patient are major clues to the severity of the problem. **Cyanosis, agitation, and the level of pain and anxiety** in the patient are easy observational signs, as is obesity. **Performing the exam in a standard way to avoid missing relevant findings is crucial.** One way is to start at the head and work your way to the extremities in a systematic way. Quality of the **pulse, diaphoresis, warm or cold skin** are surmised in seconds as the history is taken. **Noting neck vein** distention, the position of the **trachea**, and the quality of the **carotid pulse**, and listening for carotid bruits should be next. Listening and quantifying **heart and breath sounds**, as a baseline, are important in what can be a rapidly changing physical exam. The **cardiac exam** needs to be complete and is directed toward signs of increased cardiac size, the presence of abnormal heart sounds suggestive of heart failure, and the existence of any cardiac murmurs. **Palpation for an abdominal aneurysm** is done rapidly, if possible, as is checking for the presence of **bowel sounds** or the presence of **hepatomegaly**. **Peripheral pulses** are checked and **signs of chronic or acute ischemia** are sought. Any **swelling**, either bilateral (congestive heart failure) or unilateral (possible deep vein thrombosis), is checked for. An experienced physician can complete this examination in 2 or 3 minutes.

Chest X-Ray

The chest x-ray is one of the initial studies that **should be completed and often is overlooked** as a means of rapidly differentiating the significant causes of chest pain. Findings of congestive heart failure, pleural effusion, or pneumothorax may be noted; enlarged cardiac silhouette consistent with cardiomegaly or large pericardial effusion may be present. Signs of aortic aneurysm or dissection may be present. Large pulmonary emboli may be diagnosed by the absence of pulmonary markings on the chest x-ray.

Table 16.3. Differential diagnosis of episodic chest pain resembling angina pectoris.

	Duration	Quality	Provocation	Relief	Location	Comment
Effort angina	5–15 minutes	Visceral (pressure)	During effort or emotion	Rest, nitroglycerin	Substernal, radiates	First episode vivid
Rest angina	5–45 minutes	Visceral (pressure)	Spontaneous (? with exercise)	Nitroglycerin	Substernal, radiates	Often nocturnal
Mitral prolapse	Minutes to hours	Superficial (rarely visceral)	Spontaneous (no pattern)	Time	Left anterior	No pattern, variable character
Esophageal reflux	10 minutes to 1 hour	Visceral	Recumbency, lack of food	Food, antacid	Substernal, epigastric	Rarely radiates
Esophageal spasm	5–60 minutes	Visceral	Spontaneous, cold liquids, exercise	Nitroglycerin	Substernal, radiates	Mimics angina
Peptic ulcer	Hours	Visceral, burning	Lack of food, “acid” foods	Foods, antacids	Epigastric, substernal	
Biliary disease	Hours	Visceral (waxes and wanes)	Spontaneous, food	Time, analgesia	Epigastric, ? radiates	Colic
Cervical disk	Variable (gradually subsides)	Superficial	Head and neck movement, palpation	Time, analgesia	Arm, neck	Not relieved by rest
Hyperventilation	2–3 minutes	Visceral	Emotion, tachypnea	Stimulus removal	Substernal	Facial paresthesia
Musculoskeletal	Variable	Superficial	Movement, palpation	Time, analgesia	Multiple	Tenderness
Pulmonary	30 minutes +	Visceral (pressure)	Often spontaneous	Rest, time, bronchodilator	Substernal	Dyspneic

Source: Reprinted from Christie LG Jr, Conti CR. Systematic approach to the evaluation of angina-like chest pain. Am Heart J 1981;102:897. Copyright © 1981 Mosby. With permission from Elsevier.

Electrocardiogram

The ECG is the major initial tool used to differentiate ischemic heart disease from other etiologies of chest pain. Acute ECG changes frequently are present, especially when there is ongoing pain. The presence of **Q waves** indicates myocardial cellular necrosis and cell death. These are present when chronic scarring has preceded the current event or may document an acute event in which recovery of function of the myocardium is unlikely. **ST segment elevations** are present during episodes of acute ischemia and represent ongoing cellular injury. **T-wave inversions** are related to ongoing ischemia. At times, **ST segment depression** is seen and must be differentiated as subendocardial ischemia or reciprocal changes to ST segment elevation elsewhere. **ST segment elevations** in all leads may be more suggestive of pericarditis.

Other Laboratory Studies

Additional lab work (blood work) is needed on these patients depending on the suspected etiology of the chest pain. It is probably appropriate that **cardiac enzymes, especially troponin levels**, be drawn on all of these patients. Depending on findings and history, **CBC and differential** may be performed. A **chemistry profile** may be required and, perhaps, **arterial blood gases** obtained to help make and confirm diagnosis.

Diagnostic and Confirmatory Studies

Diagnostic and confirmatory studies are now required as the list of diagnoses is developed. They are ordered on the basis of presumptive diagnosis from the initial history, physical exam, chest x-ray, ECG, and laboratory studies. In reality, findings from the history often establish the subsequent diagnostic path.

Echocardiography

Echocardiography is a superb diagnostic study performed early in the diagnostic sequence. It is simple to perform by an experienced technician. It is performed in two different methods. **Transthoracically**, this is a completely benign study requiring nothing from the patient except cooperation in positioning (perhaps a problem when severe pain or dyspnea are present). **Transesophageal echocardiography (TEE)** is more complicated, but it will often provide more detailed and accurate information, especially if the transthoracic study is insufficient due to technical reasons. The TEE can be difficult and stressful on some patients because of the need to pass the probe through the pharynx and down the esophagus. Despite local anesthesia, the gag reflex can make this impossible at times. Sedation often is used, and a cardiologist is present. Once the probe is in position, diagnostic information is readily available. In the presence of ischemia or myocardial infarction, rapid quantitation of ventricular function can be established. Any mechanical complication of myocardial infarction (ventricular septal defect, ruptured papillary muscle, ruptured free myocardial wall) should be

diagnosed. Transesophageal echocardiography has the highest accuracy and specificity of any study for the diagnosis of aortic aneurysms and aortic dissections. It can be done rapidly in the emergency room and does not require transporting the patient, as other diagnostic studies do.

CAT Scan

The CAT scan is another **noninterventional study** that helps differentiate the causes of significant chest pain. Especially when contrast agents can be used (in normal renal function), aortic aneurysm and dissections can be diagnosed (**Fig. 16.1**), pericardial effusions and the

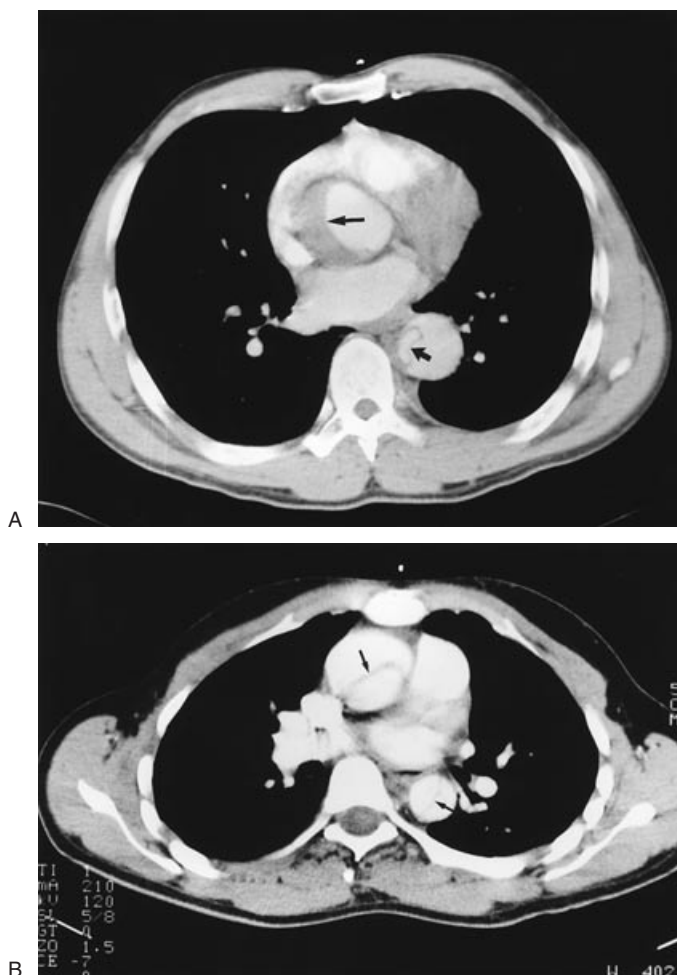


Figure 16.1. Computed tomography of acute dissection. (A) Thrombosis of the false lumen in the ascending aorta (thin arrow) and compression of the true lumen in the descending aorta (thick arrow). (B) Patency of true and false lumen with an intimal flap visible in both the ascending and descending aorta (arrows). (Reprinted from Sundt TM, Thompson RW. Diseases of the thoracic aorta and great vessels. In: Norton JA, Bollinger RR, Chang AE, et al, eds. Surgery: Basic Science and Clinical Evidence. New York: Springer-Verlag, 2001, with permission.)

thickness of the pericardium recognized, large pulmonary emboli identified, and the diagnosis of many of the other causes of chest pain not discussed in this chapter made. Because of the **simplicity and reproducibility of the study, CAT scanning often is used as the method by which patients are followed once a diagnosis of aneurysm or dissection is made or as follow-up after surgery.** The size and other characteristics of the aneurysm can be followed, and the development of false aneurysm or other complications can be recognized.

Magnetic Resonance Imaging

Magnetic Resonance Imaging (MRI) is the final diagnostic tool of a **non-invasive type** that can be very helpful in the differential diagnosis of the multiple etiologies of chest pain. However, **its very nature and the time required to obtain the study tend to make it inappropriate for the acute situation. In more chronic situations, however, especially with complex aortic dissections, it can be quite helpful in characterizing the anatomy and etiologies for chest pain.**

Cardiac Catheterization and Coronary Angiography

Cardiac catheterization and coronary angiography are the only ways presently available for the accurate diagnosis of coronary artery disease and the only way to obtain information for its definitive treatment. Certain situations may require emergency catheterization and interventional studies, primarily when the diagnosis of ischemic heart disease is made in the emergency setting and symptoms cannot be relieved easily by standard treatment methods using nitrates, beta-blockers, oxygen, and pain medication. In the early stages of an evolving myocardial infarction, acute intervention with fibrinolytic agents in the emergency room or early intervention using percutaneous techniques (angioplasty with or without stenting) or even emergency surgery may be required. An intraaortic balloon pump may be required to maximize stabilization during, prior, or even following these interventions.

In most other patients in whom the diagnosis of coronary artery disease is suspected, **exercise stress testing** is the initial diagnostic study, especially when an abnormal ECG has been obtained. The patient exercises progressively at quantified increased levels of work, usually on a treadmill, to gradually increase the heart rate and level of myocardial work. Blood pressure and electrocardiogram are monitored. The patient is questioned as to the presence of any anginal symptoms as the exercise levels increase. **Onset of symptoms, especially when associated with significant ECG changes, is considered a positive test for ischemia. The development of hypotension associated with a stress test is an ominous sign and highly suggestive of left main or critical triple vessel disease.** The accuracy of a stress test alone, however, is only about 70%. This can be improved to approximately 90% by **combining the stress test with radionuclear imaging,** where myocardial perfusion and metabolic function are evaluated at rest and exercise (**an exercise stress thallium study**).

The presence of a positive stress test or continued high suspicion for coronary artery disease even after a negative stress test usually

results in cardiac catheterization. All patients in whom ischemic heart disease is suspected ultimately undergo this test to determine the presence or absence of coronary artery or valvular heart disease. It remains the sine qua non in the diagnosis of ischemic heart disease.

Therapeutic Intervention and Results

Ischemic Heart Disease

At the present time, there are **three classifications of therapy** available to patients with ischemic heart disease. These are referred to as **medical therapy, percutaneous angioplasty, and coronary bypass surgery.** Decisions regarding treatment **must be individualized and based on symptoms, anatomy, and risks of the selected therapy.** Dietary management, weight loss, cessation of smoking, etc., are changes in habits and lifestyle that the patient can make; these changes contribute to both short- and long-term benefits of whichever treatment modality is selected.

The classic approach to **medical therapy** for ischemic heart disease is a **three-pronged approach to decrease oxygen demand by the heart and includes beta-blockers, nitrates, and calcium channel blockers.** As noted earlier, the prime cause of angina pectoris is the **mismatch of oxygen demand and oxygen supply** to the heart. **Oxygen demand** of the heart is determined by three major factors: (1) **heart rate**; (2) **wall tension**; and (3) **to a lesser extent, the level of contractility of the heart.** Wall tension is determined by **Laplace's law** of the heart, in which wall tension is directly related to pressure and volume and inversely related to the wall thickness of the chamber involved:

$$T = P \times R/2h$$

where T is the wall tension, P is the chamber pressure, R is the chamber radius, and h is the wall thickness. **Beta-blockers** are the first line of treatment. The goal of their use is first to **minimize increases in heart rate** due to response to physical and emotional demands and second to **decrease myocardial contractility.** **Nitrates decrease the preload** through venous dilatation and relaxation of the capacitance vessels. They also **dilate the epicardial vessels supplying the ischemic coronary beds.** Sublingual nitroglycerin, nitroglycerin paste, and other longer acting nitrates are included in this category. **Calcium channel blockers** provide **afterload reduction** (and thus, decreased wall tension) by relaxing the smooth muscle of peripheral vessels and **preventing coronary spasm.** In addition, **the oxygen-carrying capacity of the blood must be optimized.** If anemia is present, it must be corrected. In theory, **only after a patient fails to respond to the simultaneous use of all three modes of therapy at maximal tolerated doses is a patient considered to have "failed medical therapy."** The patient then becomes a candidate for another mode of treatment requiring increasing levels of intervention and risk.

The next treatment option of increasing complexity and risk to the patient is **percutaneous transluminal coronary angioplasty (PTCA).**

Developed by Andreas Grundzig¹ of Switzerland in the 1970s, PTCA has led to the development of many interventional procedures performed in the cardiac catheterization laboratory to open partially occluded coronary vessels using percutaneous techniques. These include **PTCA alone, laser angioplasty, directed atherectomy**, and, most recently, **PTCA with stenting**. Using techniques similar to cardiac catheterization, a guidewire is directed across and through the coronary lesion under fluoroscopic control. A PTCA balloon or atherectomy catheter is then passed over the guidewire and across the lesion. The balloon is inflated, compressing the lesion against the walls of the vessel, or an atherectomy is performed with actual removal of material from the wall of the vessel.

The advantage of these procedures (when they are appropriate) is that the patient suffers little in the way of disability and the hospitalization usually is quite short. Return to normal activities within a week or two is not uncommon. There are some potential disadvantages, however. Recurrence rates of 25% to 30% are not uncommon within 6 months of the procedure. In many situations, PTCA can be repeated. In 1% to 3% of all PTCAs or atherectomies, the patient requires emergency surgery due to a complication. In these situations, the surgical results are not as good as for elective surgery; perioperative myocardial infarction and mortalities both are higher. Recently, **intracoronary stents** made of fine metal mesh have been developed, and, based on limited results to date, **seem to increase the likelihood of longer patencies following angioplasty as well as to lower the risk for emergency surgery at the time of the procedure**. **Irradiated and drug-eluting stents** are now being tested and seem to prolong the patency even further.

Decisions on the use of **coronary artery bypass grafting (CABG)** to treat patients with ischemic heart disease are based on anatomy, symptoms, and the potential risks to the patient as well as the long-term benefits of the operation. **Certain anatomic situations (left main disease, left main equivalent, and three-vessel disease with decreased ventricular function) may warrant surgery even in the absence of symptoms** because of the large amount of myocardium in jeopardy and the recognized high mortality risk without treatment (including sudden death). **Currently, additional acceptable indications for CABG are stable angina unresponsive to medical therapy; unstable angina such as pain at rest; preinfarction or postinfarction angina; and patients with double- or triple-vessel disease with diminished left ventricular function.**² All patients with these conditions are likely to benefit from surgery either with **relief of symptoms, prevention of myocardial infarction, or prolongation of life**. Diabetics with two-vessel disease get better long-term results with CABG than with angioplasty. In addition, concomitant CABG may be indicated for patients under-

¹ Presented at the American Heart Association meeting, 1976. Transluminal dilatation of coronary artery stenosis. Letter to the editor. *Lancet* 1978;1:263.

² ACC/AHA. Guidelines for coronary artery bypass surgery, executive summary and recommendations. *Circulation* 1999;100:1464–1480.

going surgery for complications of myocardial infarction (acute mitral regurgitation, ventricular septal defect, or free rupture of the heart) or for patients undergoing elective valve replacement procedures with critical vessel occlusions.

The risks of surgery to certain patients may far outweigh the benefits to them. Patients with **limited life expectancy from other diseases (especially malignancies), the very elderly, or the physically impaired** might not be considered surgical candidates based on associated physical conditions. In these situations, further medical treatment or attempts at a partial revascularization utilizing PTCA for symptomatic relief may be more appropriate. Although the benefits of CABG to patients with decreased ventricular function and double- or triple-vessel disease are well recognized, **poor ventricular function adds to the mortality of patients undergoing the operation.** Other factors that increase the mortality and morbidity of CABG (as well as other open heart procedures) include **age greater than 70, morbid obesity, diabetes, chronic obstructive pulmonary disease, hypertension, history of myocardial infarction, reoperation, chronic renal failure, peripheral vascular disease, and, possibly, female gender.**

The results of CABG are excellent, but, as noted, many factors contribute to operative mortality and morbidity. Mortality at most major centers for all patients undergoing CABG ranges from 2% to 4%. **Figure 16.2** cites survival benefits of patients undergoing CABG or PTCA.

Diseases of the Thoracic Aorta

Decisions regarding treatment of patients with aortic aneurysms are dependent on the **risk/benefit ratio to the patient.** Symptomatic patients have a mean survival of approximately 2 years following onset of the symptoms. The majority of time, however, the surgeon is confronted with a patient without symptoms found to have an aneurysm on a routine chest x-ray or other study. Here, **the greatest risk to the patient is rupture of the aorta, which is more likely to occur the greater the size of the aorta.** The risks of surgery are lowest when the ascending aorta needs to be approached. Surgery in the descending thoracic aorta is next in risk. The highest risk is associated with repair of the aortic arch.

Aortic dissection is treated in a different manner because of the acuteness of the situation. **Regardless of the type of dissection (Stanford A or B), initial emergent therapy is medical, with a goal of controlling the patient's symptoms, heart rate, and blood pressure.** Intravenous narcotics for pain control are used. The heart rate is controlled by intravenous beta-blockade to lower it below 70. Following beta-blockade, blood pressure control is obtained using intravenous nitroprusside or nitroglycerin. Constant blood pressure monitoring is crucial for these patients, preferably with an arterial line in a radial artery. The extremity with the highest initial blood pressure is utilized to avoid inaccurate readings from a blocked vessel.

All patients with aortic dissection **should be admitted to the surgical service for close observation and management in consultation with cardiology or hypertension specialists.** Transesophageal

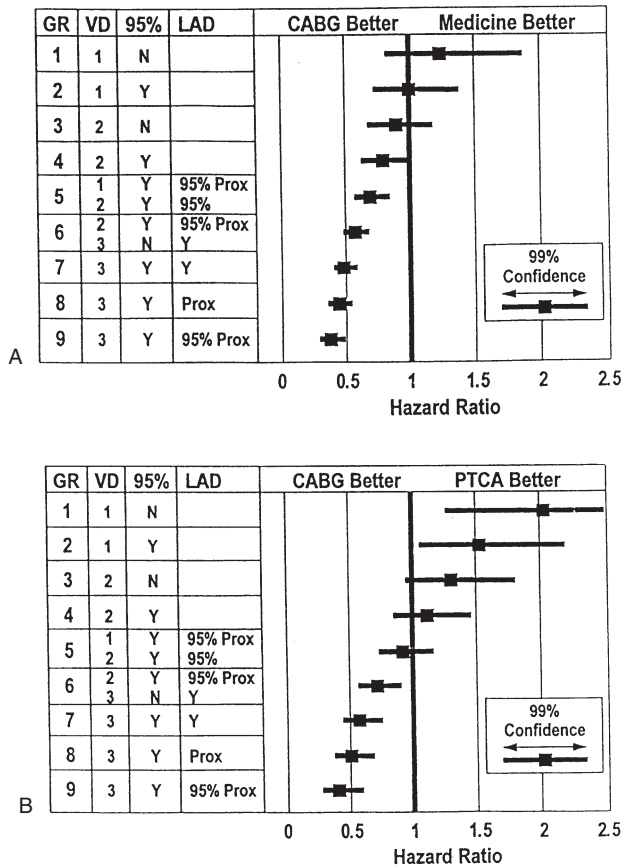


Figure 16.2. Adjusted hazard ratios comparing (A) coronary artery bypass grafting (CABG) and medicine, and (B) CABG and angioplasty, as a function of the extent of coronary artery lesions. PTCA, percutaneous transluminal coronary angioplasty; VD, number of vessels diseased; 95% \geq 95% coronary artery stenosis; Prox, proximal; GR, group. (Reprinted from Jones RH, Kesler K, Phillips HR, et al. Long-term survival benefits of coronary artery bypass grafting and percutaneous transluminal angioplasty in patients with coronary artery disease. *J Thorac Cardiovasc Surg* 1996;111:1013–1025. Copyright © 1996 American Association for Thoracic Surgery. With permission from the American Association for Thoracic Surgery.)

echocardiography is the preferable diagnostic study. In type A dissections, the aortic valve can be evaluated for insufficiency, and the presence or absence of pericardial fluid (suggesting impending rupture into the pericardium and sudden death) can be evaluated. Once a diagnosis of a **type A dissection** is made and the patient is deemed a surgical candidate, **an emergency operation is performed.** If there is any question of the diagnosis or if a type B dissection is identified, then **aortography can be used for additional information.** Aortography can provide information on whether the dissection actually exists, what is involved, the presence of aortic insufficiency, possible identification of associated coronary disease, the site of the tear, and the involvement of major branches off the aorta. **Type B aortic dissections are normally**

treated by continuation of the initial medical therapy. This includes permanent treatment with beta-blockers and antihypertensive agents. There are certain indications, however, that require **surgery for a type B dissection (Table 16.2).** These include **ongoing pain, significant hemothorax, progressive mediastinal enlargement suggesting an expanding mediastinal hematoma, inability to control the blood pressure within 48 hours, and loss of blood supply to a significant branch of the distal aorta.** Loss of distal flow frequently requires surgical intervention for a repair of type B dissection. There are also methods of fenestration of the distal false lumen to permit reentry of blood flow and restoration of adequate distal circulation.

Surgery for aortic aneurysmal disease of the thoracic aorta, whether it is elective (as for most aneurysms) or emergent (as for most dissections), usually is performed in a similar fashion. Replacement of the ascending aorta requires the use of the heart–lung machine. This can be done by cross-clamping the aorta and protecting the heart in the usual techniques of ischemic arrest. The method used, especially if the aortic arch needs replacement, is that of circulatory arrest. Descending thoracic aortic surgery can be performed in many ways through a left posterolateral thoracotomy. Simple cross-clamping is possible, but the likelihood of **paralysis postoperatively is significant**, especially if more than **30 minutes** of ischemia to the spinal cord occurs. Left heart bypass, as will complete bypass and circulatory arrest, may yield some additional protection from prolonged ischemia. Whatever technique is used, there is the risk of paralysis. The artery of Adamkiewicz is thought to provide the majority of blood to the anterior spinal artery, which in turn supplies the anterior aspects of the spinal cord. The greater the extent of aorta resected and the greater the involvement of the areas distal to T6, the greater this risk.

Following treatment of the aneurysm or dissection, **careful follow-up is crucial.** One of the leading causes of death in these patients is redissection or rupture of a new aneurysm or leak from the suture line. Initial follow-up is with CAT scanning at 3 and 6 months, then every 6 months thereafter. If all remains stable after 2 years, then yearly follow-up is appropriate.

Pericardial Disease

The typical case of acute pericarditis can be treated with **antiinflammatory agents, especially salicylates**, and usually will respond rapidly. When this is not the case, concern should be raised about a different etiology other than idiopathic. Since the next level of antiinflammatory treatment for this problem requires **steroid therapies, infectious etiologies should be ruled out before steroid therapy is instituted.** The presence of a significant effusion and an ill patient should lead to **aspiration of the pericardial sac and biopsy.** Definitive therapy to prevent significant reaccumulation of fluid as well as definitive diagnosis is likely to require an **open procedure.** In patients with chronic renal failure and dialysis, initial efforts are to decrease the presence of the effusion by increasing the frequency of the dialysis

episodes. If this does not work, a single simple aspiration should be performed. Repeat accumulation of fluids should lead to a **more permanent drainage procedure**. Finally, patients with chronic constrictive pericarditis require **pericardial stripping for relief of symptoms**. This pericardial stripping may be performed in any of several ways. The safest appears to be through a **median sternotomy**. Should it become necessary, cardiopulmonary bypass may be used as an adjunct for a safe procedure.

Pulmonary Embolism

Once the diagnosis of pulmonary embolus has been confirmed (or if the clinical findings are strong enough to warrant treatment), treatment and further diagnosis requires a dual approach: **treat the embolus and prevent any recurrences**. Most cases require treatment with **heparin anticoagulation and symptomatic support of the patient**. Heparin prevents the formation of additional venous thromboses (the presumed origin of the embolus) and is thought to promote dissolution of the emboli in the pulmonary circulation. Further diagnostic studies are aimed at verifying the origin of the embolus (venous thrombosis, endocarditis, tumor embolus, etc.). If anticoagulants are contraindicated or repeat embolism occurs on anticoagulants, then an **inferior vena cava umbrella** should be placed.

Summary

Several potential life-threatening diseases treated by cardiothoracic surgeons must be rapidly recognized, their pathophysiology understood, and treatment methods recognized. These major entities include coronary artery disease and dissection of and aneurysms of the thoracic aorta. Understanding Laplace's law of the heart is key to understanding both the pathophysiology and therapy for these diseases and the requirements for surgical intervention.

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