

Chapter 27

CHEST PAIN

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Chest pain in the ICU is a common complaint that demands urgent evaluation. It is also a somewhat different entity from chest pain seen in the office, ward, or emergency department (ED). Although ICU patients typically are sicker and their problems more complex, management is expedited. ICU patients have already been identified as being critically ill, and they are already in the most resource-rich area of the hospital. The keys to proper management of chest pain in the ICU are a rapid and focused assessment of immediate problems, a careful consideration of the differential diagnosis, a logical evaluation plan, and empirical treatment while awaiting a definitive diagnosis.

INITIAL APPROACH

An ICU patient with chest pain should be seen as soon as possible. When performing the initial evaluation (Fig. 27-1), a good policy is to obtain a fresh set of vital signs and determine whether anything else has changed. First, ensure the adequacy of the basic ABCs: airway, breathing, and circulation. Ensure that the patient has intravenous access and is on a cardiac monitor. Next, take a moment to note the patient's cardiac rhythm and arterial oxygen saturation (pulse oximetry). Check the ventilator settings and, if an arterial catheter or pulmonary artery catheter is in place, the systemic arterial or pulmonary arterial pressure waveforms, respectively. Determine whether the patient appears obtunded, dyspneic, mottled, cool, or diaphoretic. Auscultate the chest and precordium, listening for heart murmurs, friction rubs, and the presence and quality of breath sounds. Seek to identify immediate life-threatening problems, such as tension pneumothorax, ventricular arrhythmias, or arterial hypoxemia, before moving on to perform a more detailed assessment. If life-threatening problems are suspected, evaluation and treatment must be performed almost concurrently. Other chapters in this textbook discuss these time-urgent conditions in greater detail.

HISTORY

If the patient is stable after the initial evaluation, obtain a more detailed history. If the patient can communicate, start with an open-ended question, such as "What's going on, Mr. Jones?" Physicians typically interrupt their patients after about 23 seconds,¹ so force yourself to simply listen for at least 1 minute before saying anything else. The most pertinent information will usually come out during those 60 seconds. Next, fully characterize the chest pain. In one study, only 42% of patients with confirmed thoracic aortic

dissections were asked even basic questions about their pain.² Omitting one or more of these basic questions during the initial evaluation was associated with a delayed diagnosis. The mnemonic OLDCAAR can help clinicians avoid this mistake (Table 27-1).

The patient's bedside nurse should also be queried about recent changes in the patient's status (e.g., mental status, respiratory pattern, cardiac rate and rhythm). Last, a quick "chart dissection" should be performed, focusing on the initial history and physical examination, past medical history (paying special attention to cardiac risk factors and prior surgical procedures), reason for ICU admission, and the last few progress notes. Do not waste time asking the patient or nurse questions that can be answered by reading the medical record.

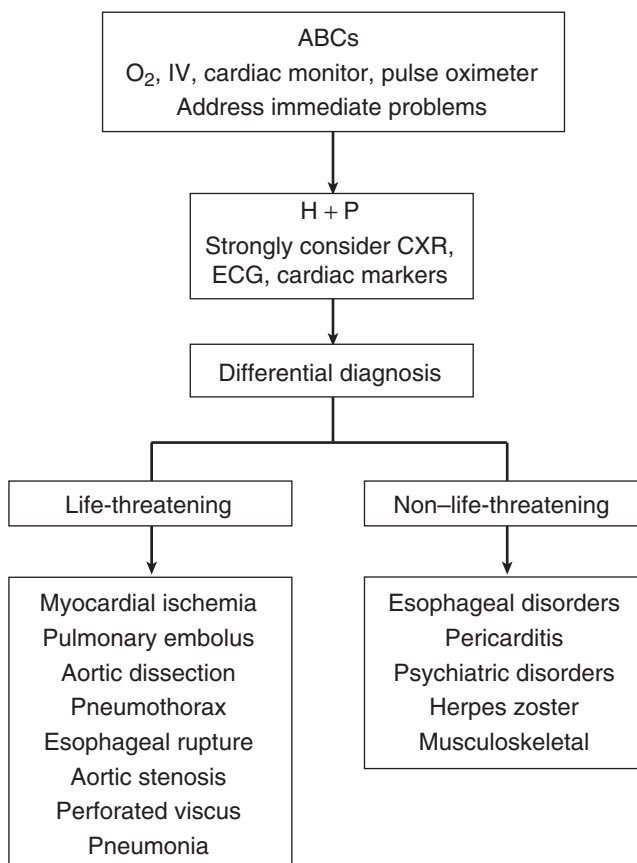


FIGURE 27-1. Approach to chest pain in the ICU. ABC, airway, breathing, circulation; CXR, chest x-ray; ECG, electrocardiogram; H + P, history and physical examination; IV, intravenous access.

TABLE 27-1. OLDCAAR MNEMONIC FOR EVALUATING PAIN

Domain	Suggested Questions
Onset	Sudden vs gradual? Maximal pain at onset?
Location	Generalized or localized? Can you point with one finger to where it hurts?
Duration	When did it start? Just now, or did the pain occur earlier, but you didn't want to bother anyone? Is it constant or intermittent? If intermittent, is there a trigger, or is it random?
Character	Sharp? Dull? Ache? Indigestion? Pressure? Tearing? Ripping?
Associated symptoms	"Dizzy"—vertiginous or presyncopal? Diaphoresis? Palpitations? Dyspnea? Nausea or vomiting?
Alleviating/Aggravating	Position? Belching? Exertion? Deep breathing? Coughing?
Radiation	To the back? Jaw? Throat? Arm? Neck? Abdomen?

PHYSICAL EXAMINATION

Disrobe the patient to ensure optimal visualization, looking particularly for obvious chest wall asymmetry or deformities. Seek to identify areas of point tenderness or crepitus. Next, focus on the cardiac, pulmonary, and abdominal examinations. Check the blood pressure in both arms as you talk to the patient. Assess for asymmetry in pulse quality of the carotid, femoral, and radial pulses. There is a difference in the blood pressure recorded from the right and left upper extremities in about one third of patients with aortic dissection.³ Check for pulsus paradoxus and jugular venous distention. Listen for asymmetry and quality of breath sounds in conjunction with a review of ventilator settings, if applicable. Evaluate the heart for diminished heart sounds, new murmurs, friction rubs, or gallops. Examine the abdomen for tenderness, pulsatile masses, and absent or abnormal (i.e., high-pitched) bowel sounds. Last, palpate and inspect the lower extremities for tenderness or size differential. Unfortunately, the physical examination is relatively insensitive, and supplemental tests are frequently necessary.³

DIAGNOSTIC ADJUNCTS

Unless the cause of new chest pain is obvious (e.g., tension pneumothorax, herpes zoster with visible lesions), a portable chest x-ray (CXR) and 12-lead electrocardiogram (ECG) and rhythm strip should always be obtained. In addition, serial measurements of circulating levels of creatinine phosphokinase MB or, preferably, troponin T or troponin I should be measured to exclude a myocardial infarction (MI).

The CXR should be examined for pneumothorax; a widened mediastinum; new infiltrates; effusions; free subdiaphragmatic air; rib fractures; subcutaneous emphysema; malpositioned endotracheal, nasogastric, orogastric, or chest tube; and aortic silhouette abnormalities. Both the ECG and the CXR should be compared with the most recent study before the onset of chest pain.

The ECG and rhythm strip should be evaluated principally for arrhythmias and signs of ischemia, such as inverted T waves, ST segment depression or elevation, and new Q waves. More subtle ECG findings relevant to specific causes of chest pain are discussed in the next section.

An intravenous contrast-enhanced spiral computed tomography (CT) scan is helpful for excluding the diagnosis

of pulmonary embolism and may detect other pathologic findings as well. In many centers, it is the diagnostic test of choice for pulmonary embolism. The ventilation-perfusion (\dot{V}/\dot{Q}) radionuclide lung scan is an alternative method of diagnosing pulmonary embolism. The \dot{V}/\dot{Q} scan can be a useful alternative to spiral CT in patients with a history of allergic reaction to intravenous contrast material or those at high risk for contrast-induced nephropathy. Pulmonary angiography remains the gold standard for detecting pulmonary embolism, but it is an invasive procedure with a low but real risk of iatrogenic complications.

Echocardiography can be useful for assessing not only left and right ventricular function but also regional wall motion abnormalities, pulmonary hypertension, valvular disease, pericardial effusion, cardiac tamponade, and aortic dissection. Transthoracic echocardiography is usually the first step, followed by transesophageal echocardiography, if necessary. However, transthoracic echocardiography does not visualize the aorta well and can be limited by obesity, emphysema, and chest deformity. For patients in urgent need of aortic visualization, transesophageal echocardiography may be indicated as the initial choice.

DIFFERENTIAL DIAGNOSES

There are three rules to live by:

1. Do not assume that the admission diagnosis is necessarily correct or inclusive. MI can present as gastrointestinal complaints, especially among African Americans.⁴ Conversely, the actual diagnosis among patients admitted with presumed (but unconfirmed) MI includes pneumonia, perforated duodenal ulcer, or acute cholecystitis, among myriad other possibilities.
2. Do not be biased by the type of ICU the patient happens to be in. For example, aortic dissection can present as a stroke, prompting admission to a neurologic ICU. Acute serious abdominal problems can occur in medical ICU patients. Indeed, a recent retrospective review of abdominal catastrophes in a medical ICU concluded, "delays in surgical evaluation and intervention are critical contributors to mortality rate in patients who develop acute abdominal complications in a medical ICU."⁵
3. Do not close your mind to alternative diagnoses, even if the diagnosis seems obvious.

ACUTE LIFE-THREATENING PROBLEMS

Myocardial Ischemia

The spectrum of myocardial ischemia ranges from angina to frank MI. Because coronary artery disease is highly prevalent in ICU patients, whether previously diagnosed or occult, a high index of suspicion for ischemia is mandatory. Enumeration of the patient's risk factors (hypercholesterolemia, hypertension, smoking history, family history, age, diabetes mellitus) is useful. The classic signs of myocardial ischemia include chest pain, diaphoresis, palpitations, nausea, syncope or near syncope, vomiting, and dyspnea. Pain often radiates to the neck, arm, or jaw. Unfortunately, myocardial ischemia can also present in much more subtle ways. The type of chest pain is variable and has been described as sharp, dull, tearing, or crushing. Many patients do not even report pain but describe only pressure or simply

an odd feeling. Importantly, MI can often present as gastrointestinal symptoms alone, such as “gas,” “heartburn,” or simply nausea. A retrospective review of 434,877 patients with confirmed MI found that 33% did not have chest pain.⁶ Further, patients without chest pain had higher in-hospital mortality rates, possibly due to delays in care. These atypical presentations are more common in patients with heart failure, a previous stroke, or diabetes and in the elderly, women, and minorities.^{4,6}

An ECG should be obtained, and supplemental oxygen and pain relief should be provided, if myocardial ischemia is deemed possible. Unless contraindicated, antiplatelet therapy in the form of aspirin 162 to 325 mg p.o or clopidogrel 75 mg p.o. (if aspirin allergy is present) should also be administered. The ECG should be compared with the most recent previous one and examined for ST segment elevation or depression, new Q waves, and T wave inversion. Unfortunately, many MIs are associated with equivocal ECG findings,⁷ in which case serial cardiac enzymes and serial ECGs are necessary for diagnosis. Nitroglycerin and morphine should be used to relieve pain, checking the blood pressure before and after each dose. If pain is not relieved with these measures, alternative diagnoses such as aortic dissection should be considered. However, if the diagnosis of MI is strongly suspected, an interventional cardiology consultation should be obtained, because persistent chest pain is an indication for urgent cardiac catheterization.⁸

Pulmonary Embolus

Most ICU patients have at least one risk factor for pulmonary embolus (prolonged bed rest, postoperative state, hypercoagulable state, trauma, burns, heart failure); therefore, pulmonary embolus, like MI, should be strongly considered in this population. Pulmonary embolus can present in multiple ways, but most frequently as pleuritic chest pain and dyspnea or tachypnea. Other presentations include syncope, hemoptysis, diaphoresis, cough, and hypoxia. Although pulmonary embolus is often associated with a widened alveolar-arterial (A-a) gradient, this finding is not very useful among ICU patients, as it is neither specific (ICU patients often have many other reasons for hypoxia) nor sensitive (the A-a gradient is normal in approximately 25% of patients with pulmonary embolus).⁹ Large pulmonary emboli that significantly occlude the pulmonary circulation present with obstructive cardiogenic shock, hypotension, and a sudden rise in central venous, right ventricular, and pulmonary arterial pressures. Echocardiography can be useful in this setting to confirm the diagnosis by demonstrating right heart failure and right ventricular dilatation with septal shift and subsequent left ventricular outflow obstruction.

The CXR is insensitive for diagnosing pulmonary embolus, so more advanced studies are typically required (CT, V/Q scan, pulmonary angiography). For each test, the risks of iatrogenic complications and complications during transport must be taken into account.

Aortic Dissection

The risk factors for aortic dissection overlap considerably with those for myocardial ischemia; therefore, this entity should always be considered among “rule out MI” patients (Table 27-2). Persistent chest pain without ECG changes is a potential clue that aortic dissection may be present.

The basic pathophysiology involves a tear of the aortic intima, leading to a false lumen between the intima

TABLE 27-2. AORTIC DISSECTION RISK FACTORS

Atherosclerosis risk factors (hypertension, diabetes, smoking, age, hypercholesterolemia)
Connective tissue disorders (Marfan's syndrome, Ehlers-Danlos syndrome)
Cocaine
Bicuspid aortic valve
Coarctation of the aorta
Trauma
Previous cardiac surgery (especially aortic valve replacement)
Intra-aortic catheterization
Giant cell arteritis

and adventitia. A recent systematic review noted that the vast majority of patients complain of severe chest pain (90%) of sudden onset (84%).³ The review also noted that 28% have a diastolic murmur (due to aortic regurgitation), 31% have a pulse deficit or blood pressure differential (>20 mm Hg), and 17% have focal neurologic deficits. The physical examination should search for these findings.

Patients with aortic dissection were once thought to experience a tearing or ripping sensation. However, the International Registry of Acute Aortic Dissection reported in its series of 464 patients that pain was most commonly described as “sharp.”¹⁰ Further, only about half the patients described back pain. Therefore, the absence of tearing or ripping pain radiating to the back should not exclude the diagnosis of aortic dissection.

Although a normal CXR does not rule out aortic dissection, the presence of certain findings can be helpful. These findings include a wide mediastinum, separation of intimal calcification from the outer border of the aortic knob by 1 cm or greater, deviation of the trachea to the right, and blurring of the aortic margin. Comparison to the most recent CXR is key. Contrast-enhanced spiral CT is usually the best confirmatory test, but if the risk of transport is too high, bedside transesophageal echocardiography should be performed. Immediate management should focus on blood pressure control, ideally using beta-adrenergic blockade with or without a vasodilator, such as sodium nitroprusside.

Pneumothorax

ICU patients are at high risk for pneumothorax due to iatrogenic complications from central venous catheterization and thoracentesis; preexisting and acquired pulmonary disease, particularly emphysema, asthma, and acute respiratory distress syndrome; and barotrauma secondary to mechanical ventilation. It is absolutely critical to diagnose pneumothorax in patients receiving positive pressure mechanical ventilation, because positive airway pressure can transform a simple pneumothorax into a tension pneumothorax. The cardinal signs of tension pneumothorax are hypotension, jugular venous distention, absence of breath sounds and hyperresonance to percussion on the affected side, and tracheal deviation (away from the affected side). Treatment is immediate needle (14 gauge) decompression, followed by chest tube placement. Needle decompression is quickly accomplished by inserting a large-bore (16 or 18 gauge) needle through the second or third anterior interspace in the midclavicular line of the involved hemithorax.

Simple pneumothorax presents similarly but less dramatically with hypoxia, dyspnea or tachypnea, pleuritic chest pain, decreased breath sounds with hyperresonance, and increased

peak airway pressure. An upright, expiratory CXR should be obtained in cases of suspected pneumothorax. If only a supine film is possible, the deep sulcus sign (hyperlucent, lowered hemidiaphragm with an unusually sharp cardiac border) can help make the diagnosis. Loculated pneumothoraces due to underlying pulmonary adhesions can be difficult to visualize on a CXR. In such cases, chest CT should be obtained promptly; left undiagnosed and untreated, simple pneumothorax can lead to tension pneumothorax. Communication with the radiologist is essential. If the diagnosis of a loculated pneumothorax is confirmed, CT-guided placement of a chest tube or pigtail catheter should be undertaken.

Esophageal Rupture

Prompt recognition is required, because esophageal rupture can lead to potentially lethal mediastinitis. Although usually suggested by a clear history of caustic substance ingestion, forceful vomiting, or iatrogenic trauma (secondary to orogastric lavage, esophageal stricture dilatation, nasogastric tube placement, esophageal intubation, endoscopy), less obvious causes can lead to a delay in diagnosis. Any sudden increase in intra-abdominal pressure can lead to esophageal rupture, and seizures and blunt abdominal trauma have been reported as inciting events. Patients with esophageal disease such as cancer, Barrett's esophagus, and varices are especially vulnerable to rupture.

Physical examination may reveal subcutaneous emphysema or the classic finding of mediastinal crackling on auscultation (Hamman's crunch). CXR may show pneumothorax, pneumomediastinum or pneumoperitoneum, pleural effusion, or subcutaneous emphysema. In victims of blunt abdominal trauma, several findings should increase the suspicion of esophageal rupture: left pneumothorax without associated rib fractures, pain or shock out of proportion to the injury, and particulate matter in the chest tube.¹¹ Water-soluble contrast studies or esophagoscopy confirms the diagnosis.

Aortic Stenosis

The main physiologic effect of aortic stenosis is to impede left ventricular ejection, leading ultimately to left ventricular hypertrophy. Critical aortic stenosis results when this compensatory mechanism can no longer overcome the valvular stenosis or when the hypertrophy itself causes diastolic failure or excessive myocardial oxygen demand. The classic symptoms of angina, syncope, and dyspnea result. Clues suggesting critical aortic stenosis on physical examination include narrow pulse pressure, systolic murmur radiating to the carotid, S₄ gallop, and an aortic ejection click. CXR and ECG may show signs of left ventricular hypertrophy, but the definitive test is a Doppler echocardiogram. If positive, cardiac catheterization should be performed to look for concomitant coronary artery disease and to confirm the echo results. The urgency of these tests is determined by the severity of symptoms; once angina, heart failure, or syncope occurs, a prompt workup is required. Aortic valve replacement is the definitive therapy. Temporizing medical management focuses on cautiously decreasing afterload and treating angina with the careful administration of nitrates, angiotensin-converting enzyme inhibitors, and diuretics. Close hemodynamic monitoring is essential if these drugs are given, because decreases in diastolic pressure can worsen myocardial ischemia.

Miscellaneous

A perforated viscus sometimes presents as chest pain, but fortunately, this is usually easily picked up as free subdiaphragmatic air on an upright CXR. However, retroperitoneal perforations do not show up as free air under the diaphragm on CXR.

Pneumonia is often accompanied by pleuritic chest pain. Referred shoulder pain can result from diaphragmatic irritation by lower lobe pneumonia.

NON-LIFE-THREATENING PROBLEMS

All the following entities should be considered diagnoses of exclusion and should be considered only after life-threatening causes have been ruled out.

Esophageal Disorders

Owing to the shared innervation of the heart and esophagus, visceral pain originating from these two organs can be similar in character. Thus, it can be difficult to differentiate between myocardial ischemia and relatively benign esophageal disorders such as gastroesophageal reflux disease and esophageal dysmotility syndromes. The diagnosis of esophageal disease is supported by a history of pain precipitated by lying flat or the ingestion of hot or cold liquids or food. The diagnosis of an esophageal disorder is also supported if the pain is relieved by antacids. Nitroglycerin can relieve pain due to myocardial ischemia or esophageal spasm, so response to this drug is not useful as a diagnostic tool. Confirmatory tests include esophageal manometry and esophageal pH monitoring. Alternatively, an empirical trial of a proton pump inhibitor can be tried first. Last, sometimes a nasogastric tube with the distal tip in the esophagus can produce pain, especially when left on suction.

Musculoskeletal Disorders

Chest wall pain is diagnosed with direct palpation or by asking the patient to press with his or her arms against resistance. Usually these maneuvers elicit pain from the affected area. Costochondritis and myofascial syndromes often have specific trigger points that can stimulate pain. Occult rib fractures should be sought carefully by examining the CXR. According to some reports, up to 15% of patients with MI also have chest wall pain, so unless a very specific, localized, and reproducible area of pain can be found, a cardiac workup should be performed.¹² The insertion points for each chest tube and central line should also be inspected. If chest pain is elicited on physical examination, the clinician should specifically ask the patient whether the pain is the same as the spontaneously occurring pain. A negative reply demands further workup.

Pericarditis

Although pericarditis itself is rarely life-threatening, other entities in the differential diagnosis, such as MI and cardiac tamponade, can be. Pain due to pericarditis is typically pleuritic, sharp or stabbing, and retrosternal or precordial, with radiation to the back, neck, shoulders, or arms. Pain is often relieved by leaning forward and worsened by lying flat. More useful in differentiating pericarditis from ischemia is the presence of a pathognomonic but often transitory triphasic (systole, early diastole, and presystole) friction rub. A pericardial rub sounds similar to hair being rubbed together and has been described as high-pitched. It is best heard with the

diaphragm of the stethoscope at the cardiac apex, with the patient seated and leaning forward.

Characteristic ECG findings also help differentiate pericarditis from MI. Both entities demonstrate ST segment elevation, but with pericarditis, ST segment depression is absent in the reciprocal leads, except occasionally in aV_R and V₁. Absence of Q waves, concave (instead of convex) ST segment elevation, PR depression, and upright T waves also strongly favor pericarditis.¹³ Careful ECG review, auscultation, and history are the key to distinguishing between these two disorders and avoiding potentially fatal complications of contraindicated therapy (administration of a thrombolytic agent to patients with pericarditis can precipitate the development hemotamponade) or missing a diagnosis of life-threatening MI.

Pericarditis can lead to pericardial effusion. If it is large or acute, pericardial effusion can lead to cardiac tamponade. Pericardial effusion can present similarly to pulmonary embolus with dyspnea or tachypnea, tachycardia, and chest pain or pressure. ECG findings of electrical alternans and low voltage, coupled with cardiomegaly on CXR, strongly favor pericardial effusion. Pulsus paradoxus may also be present. Beck's triad (jugular venous distention, hypotension, muffled heart tones) points to a more emergent condition. Note that cardiac tamponade and tension pneumothorax share the first two components of Beck's triad, but the latter condition is characterized by normal heart tones, decreased breath sounds, and hyperresonance of the involved hemithorax. Beck's triad is not always present in patients with tamponade. For instance, if the patient is hypovolemic, jugular venous distention may not be apparent. Tamponade should be suspected when the clinical condition looks like congestive heart failure but breath sounds are clear. The ECG and CXR findings discussed for pericardial effusion are useful, but urgent echocardiography should be ordered to confirm the diagnosis. If the patient is in extremis and the clinical picture strongly suggests tamponade, pericardiocentesis should be performed. Volume loading should be done concurrently, because it can partially overcome the hemodynamic effects of tamponade.

Last, it is important to determine the underlying cause of the pericarditis. Possibilities include infection, malignancy, trauma, autoimmune disorders, and connective tissue disorders; it can also be idiopathic.

Psychiatric Disorders

Anxiety disorders, somatization, and panic attacks can all present with chest pain. Panic attacks, in particular, can be associated with symptoms that closely mimic those of MI. Both conditions are commonly associated with diaphoresis, tachypnea, dyspnea, palpitations, presyncope, and a sense of impending doom. Many patients with panic attacks have had extensive cardiac and gastrointestinal workups in the past, and obtaining these reports is helpful. Nonetheless, the dictum that "psychiatric patients get sick too" should be remembered. Psychiatric patients with real cardiac or pulmonary disease can be especially challenging to diagnose,

and a thorough, empathic history is essential. Depression is often a comorbid psychiatric condition and should be appropriately treated.

Herpes Zoster

Inspection of the patient's thorax usually makes the diagnosis of herpes zoster, although pain precedes skin manifestations by 1 to 3 days. The lesions are limited to a single dermatome and start as a maculopapular rash that quickly changes to the characteristic vesicular lesions. Acyclovir is the treatment.

CONCLUSION

Attention to immediate problems, a thorough history and physical examination, and consideration of each life-threatening possibility are the key steps to managing chest pain in the ICU. The test battery of a CXR, ECG, and serial cardiac enzymes should be used liberally but intelligently. A high index of suspicion for occult disease is necessary for complex ICU patients.

ANNOTATED REFERENCES

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The IRAAD is composed of 12 international referral centers, from which 3 years of data and 464 patients were analyzed. A key finding was that classic presentations such as tearing or ripping chest pain (50.6%), aortic regurgitation (31.6%), and pulse deficit (15.1%) were frequently absent, leading the authors to urge clinicians to maintain a high index of suspicion.
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In this study of 215 patients with confirmed MI, African-American patients attributed their initial symptoms to a gastrointestinal cause 61% of the time, versus 26% in white patients.
- Marvel MK, Epstein RM, Flowers K, Beckman HB: Soliciting the patient's agenda: Have we improved? *JAMA* 1999;281:283-287.
Although this study was conducted in primary care offices and not in an ICU, it emphasizes the importance of the basic history-taking process and listening to patients. It found that physicians interrupted their patients after a mean of only 23.1 seconds and that late-arising patient concerns were more common when physicians did not solicit questions during the interview.