Aortic dissection is a longitudinal cleavage of the aortic media created by a dissecting column of blood. The term “dissecting aortic aneurysm” has been inaccurately applied to this entity since 1819, when Laënnec first used the term *aneurysme dissequant*. The term *aortic dissection* is preferred to *dissecting aortic aneurysm* because the affected aorta is only rarely aneurysmal. In 1955, DeBakey outlined the principles that remain the basis for the surgical treatment of this entity. Medical treatment of aortic dissection was first advocated in the 1960s and is indicated for certain types of dissections.1-5 The in-hospital mortality rate for patients treated for aortic dissection is 27%.5

**Epidemiology**

Aortic dissection occurs more often in men and increases with age.5,6 The incidence and prevalence are difficult to determine because of under-reporting of this condition. Mortality is 1 to 5 per 100,000 population per year. Hypertension is the most common risk factor associated with aortic dissection and is seen in most patients.5-7 A history of cardiac surgery is present in 18% and a bicuspid aortic valve in 14% of all patients with aortic dissections but more often in proximal dissections.5,7 Atherosclerosis is rarely involved at the site of dissection. Patients with aortic dissection may have a positive family history.5

Aortic dissection is uncommon before age 40 except in association with congenital heart disease, connective tissue disease, or inflammatory vasculitides. As many as 44% of patients with Marfan syndrome develop aortic dissection and account for about 5% of cases.5-7 Women with Marfan syndrome are at particular risk during pregnancy.10 In patients without connective tissue disease and with an aortic root size of less than 40 mm, pregnancy does not appear to be an independent risk factor.11,12 Loeys-Dietz syndrome is an autosomal dominant genetic syndrome associated with aortic aneurysms and skeletal features similar to Marfan syndrome. The vascular disease in these patients is aggressive, and the mean age of death is 26 years.13 Inflammatory vasculitides associated with thoracic aortic disease include Takayasu’s arteritis, giant cell arteritis, and Behçet’s disease.1 Acute aortic dissection also occurs with stimulant use, exertion, cardiac surgery, or intra-aortic balloon pump insertion.5

Blunt trauma from a high-speed deceleration injury usually causes traumatic aortic rupture, which is an entity distinctly different from aortic dissection (see Chapter 45).
which rupture and hemorrhage into the aortic media, which may explain the absence of an intimal tear in some cases of dissection. Regardless of which of these theories is correct, the depth of penetration into the media and the distance and direction of dissection are at least partially determined by the degree of medial degeneration.

Once a dissecting hematoma is established in the media, migration of the hematoma occurs in an antegrade or retrograde fashion, or both, forming a “false lumen.” The false lumen forms in the outer half of the media and propagates until it ruptures back into the “true lumen” of the aorta, resulting in a rare “spontaneous cure,” or through the adventitia into the pericardial sac or pleural cavity. Because the outer wall of the aorta that contains the hematoma is thin, rupture is much more likely to occur to the outside. The most important factors favoring continued dissection of the aorta are (1) the degree of elevation of blood pressure and (2) the steepness (slope) of the pulse wave (upstroke pattern on apex cardiogram, dP/dt).

**Classification**

Anatomic classification is important for diagnosis and therapy. The Stanford classification is based on the involvement of the ascending aorta. Type A dissections involve the ascending aorta; type B dissections do not (Fig. 85-1). Dissections that involve the ascending aorta are more often lethal than those limited to the distal aorta and call for a different therapeutic approach. In the International Registry of Acute Aortic Dissection (IRAD), 62% of dissections are type A and 38% are type B. Patients with distal dissections tend to be older, heavy smokers with chronic lung disease and more often have generalized atherosclerosis and hypertension compared with patients who have proximal aortic dissections.

Two other aortic conditions are closely related to aortic dissection: intramural hemorrhage (IMH) and penetrating atherosclerotic ulcer (PAU). Both groups of patients have clinical symptoms and management recommendations similar to those for patients with aortic dissection. An IMH is a contained hematoma within the aortic wall and occurs in about 10% of aortic dissections. Rupture of the vasa vasorum is believed to be the initial event. Penetrating atherosclerotic ulcers of the aorta occur in older hypertensive patients with evidence of coronary artery disease. Computed tomography (CT) shows a focal ulceration without dissection, most commonly in the distal descending aorta. The progression of penetrating ulcers results in progressive aortic enlargement with saccular and fusiform aneurysm formation. Patients can have both an intramural hematoma and a penetrating atherosclerotic ulcer.

A dissection is acute if it is of less than 2 weeks’ duration and chronic if it has been present for more than 2 weeks.

**CLINICAL FEATURES**

**History**

Pain is by far the most common presenting complaint, affecting more than 90% of patients. Most cases of painless aortic dissection are chronic in nature. The pain is usually excruciating, occurs abruptly, is most severe at onset, and is typically described as “sharp” more often than “tearing” or “ripping.” A family history of thoracic aortic disease may be reported.

The location of the pain may help localize the dissection. Anterior chest pain is associated with the ascending aorta, neck and jaw pain with the aortic arch, pain in the interscapular area with the descending thoracic aorta, and pain in the lumbar area or abdomen with involvement below the diaphragm. Migration of the pain consistent with propagation of the dissection suggests aortic dissection but occurs in only 17% of cases. The onset of aortic dissection is often accompanied by visceral pain symptoms, such as diaphoresis, nausea, vomiting, lightheadedness, and severe apprehension.

Syncope occurs early in aortic dissection in approximately 9% of cases and may be the sole presentation in some patients. It often most heralds dissection into the pericardium, causing pericardial tamponade, but may occur from transient interruption of blood flow to the cerebral vasculature. Other causes of syncope from aortic dissection are hypovolemia, excessive vagal tone, and cardiac conduction abnormalities. Patients with aortic dissection and syncope have a higher mortality. Neurologic symptoms such as focal weakness or change in mental status occur in up to 17% of cases.

**Physical Examination**

The presentation varies greatly, depending on the patient and the location and extent of the dissection. In general, the patient appears apprehensive. Most of the patients have a history of chronic hypertension that may be exacerbated by a catecholamine release related to the acute event. Severe hypertension refractory to medical therapy may occur if the dissection involves the renal arteries with subsequent renin release. If hypotension is present, either the dissection has progressed back into the pericardium, with resulting pericardial tamponade, or hypovolemia has occurred from rupture through the adventitia. Pseudohypotension, a condition in which the blood pressure in the arms is low or unobtainable and the central arterial pressure is normal or high, may be present. This results from the interruption of blood flow to the subclavian arteries.

Aortic regurgitation occurs in up to 32% of patients and is more common with type A dissections. The murmur of aortic insufficiency may have a musical, vibrating quality with variable intensity, and congestive heart failure may develop. The patient with presumed aortic dissection should be examined carefully for findings that suggest hemorrhage into the pericardium or tamponade, such as jugular venous distention, muffled heart sounds, tachycardia, and hypotension.

When the integrity of one of the branches of the aorta is compromised, the expected ischemic findings occur. Pulse deficits and discrepancies in blood pressure between limbs can be helpful if present but have a sensitivity of only around 30%. Usually these are present in the upper extremities and result from involvement of one or both of the subclavian arteries.

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*Figure 85-1. Computed tomographic reconstruction of Stanford type B or DeBakey type III dissection distal to the subclavian artery.*
Obstruction of one or both common iliac or superficial femoral arteries may produce pulse deficits in the lower extremities. Arterial obstruction may occur by either of two mechanisms. An intimal flap produced by the dissection may cover the true lumen of a branch vessel, or the dissecting hematoma may compress an adjacent true lumen. Frequent reexamination may detect transient pulse deficits.

Neurologic findings are related to the site of blood flow interruption. Proximal dissections are a more frequent cause of strokes or coma. Stroke treatment with a fibrinolytic agent in the patient with aortic dissection can be fatal. Distal dissections occluding the anterior spinal artery commonly cause ischemic paraparesis or ischemic peripheral neuropathy.

In up to 3% of cases, a proximal dissection can dissect into the ostium of a coronary artery, most frequently the right coronary artery, and cause an acute myocardial infarction (MI), usually inferiorto posterior. Failure to identify the inciting aortic dissection can be fatal. 


table 85-1 characteristics of aortic dissection from the international registry of acute aortic dissection

<table>
<thead>
<tr>
<th>Modality</th>
<th>All (N = 464)</th>
<th>Type A (n = 289)</th>
<th>Type B (n = 175)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain (%)</td>
<td>73</td>
<td>79</td>
<td>63</td>
</tr>
<tr>
<td>Syncope (%)</td>
<td>9</td>
<td>13</td>
<td>4</td>
</tr>
<tr>
<td>Aortic Insufficiency (%)</td>
<td>32</td>
<td>44</td>
<td>12</td>
</tr>
<tr>
<td>Pulse Deficit (%)</td>
<td>15</td>
<td>19</td>
<td>9</td>
</tr>
<tr>
<td>Normal CXR (%)</td>
<td>12</td>
<td>11</td>
<td>16</td>
</tr>
<tr>
<td>Widened Mediastinum (%)</td>
<td>62</td>
<td>63</td>
<td>56</td>
</tr>
<tr>
<td>Normal ECG (%)</td>
<td>31</td>
<td>31</td>
<td>32</td>
</tr>
<tr>
<td>Ischemia (%)</td>
<td>15</td>
<td>17</td>
<td>13</td>
</tr>
<tr>
<td>Hypertrophy (%)</td>
<td>26</td>
<td>25</td>
<td>32</td>
</tr>
</tbody>
</table>

CXR, chest radiographic film; ECG, electrocardiogram.

**DIAGNOSTIC STRATEGIES**

Routine laboratory tests are of little value in the diagnosis of aortic dissection. The hemoglobin usually is normal or unchanged from the patient’s baseline. The leukocyte count is commonly mildly elevated. Recently, there has been increasing interest in the biochemical diagnosis of acute aortic dissection, including C-reactive protein, myosin heavy chain, soluble elastin, and D-dimer levels. D-dimer is the most readily available plasma marker and has the most diagnostic promise. Several authors have suggested that a negative D-dimer testing result makes a diagnosis of aortic dissection unlikely, but there is insufficient evidence to support use of a D-dimer as the sole screening test for aortic dissection, and 2010 guidelines from several major specialty societies recommend against this practice. Studies using a combination of a clinical pretest probability rule and D-dimer testing are needed.

**Electrocardiography**

The electrocardiogram (ECG) is often useful in excluding MI; however, 15% of patients with aortic dissection may have ECG abnormalities suggesting ischemia. Proximal dissections that involve the right coronary artery may show an inferior wall MI, and the constellation of symptoms and signs (pain, diaphoresis, hypotension) may be difficult to distinguish from those associated with primary acute MI. The ECG typically shows left ventricular hypertrophy in 26%, reflecting long-standing hypertension. Other findings include nonspecific ST-T wave changes and prior Q wave infarction. No abnormalities are noted on the ECG in 31% of cases (Table 85-1).

**Chest Radiography**

Routine chest radiographic studies are abnormal in 80 to 90% of patients, but the abnormalities are nonspecific and rarely diagnostic. Mediastinal widening occurs in the majority of cases; may occur in the ascending aorta, the aortic arch, or the descending portion of the thoracic aorta; and may be difficult to differentiate from the aortic tortuosity that is associated with chronic hypertension. A plain chest radiograph is inadequate for ruling out aortic dissection. Up to 12% of patients with aortic dissection have a normal chest radiograph (see Table 85-1).

Other helpful radiographic signs include a double-density appearance of the aorta, suggesting true and false channels, a localized bulge along a normally smooth aortic contour, a disparity in the caliber between the descending and ascending aorta, obliteration of the aortic knob, and displacement of the trachea or nasogastric tube to the right by the dissection. Previous chest radiographs when available, are useful for comparison.

**Echocardiography**

Transesophageal echocardiography (TEE) is an insensitive tool for detecting aortic dissection because it does not visualize the aortic arch or much of the descending aorta, and imaging quality may not be optimal because of the patient’s body habitus. While more sensitive imaging tests are being scheduled, however, TTE can provide valuable information about pericardial effusion or aortic regurgitation and can help determine whether cardiac tamponade is the cause of hypotension in a patient with aortic dissection. Transthoracic echocardiography (TTE) is highly sensitive (Table 85-2) for the diagnosis of aortic dissection. The proximity of the esophagus to the aorta and the ability to use higher transducer frequencies help the operator to visualize the entire aorta and to detect pericardial effusion and aortic regurgitation. TEE can be quickly performed at the patient’s bedside with sedation or light anesthesia and requires no radiation or contrast agent injection. Visualization of the distal ascending aorta and proximal arch used to be difficult because of the interposition of the air-filled trachea and left mainstem bronchus, but
evaluation of this “blind spot” has been aided by biplane and multiplane probes.\(^35\)

The diagnostic accuracy of TEE depends on the experience and availability of the echocardiographer. It is the primary diagnostic method in many institutions for detecting aortic dissection and is the procedure of choice in unstable patients,\(^2\) in whom it can be performed in the resuscitation area of the ED, or in the operating room coincident with induction of anesthesia.

### Computed Tomography

CT aortography is a reliable test for diagnosing aortic dissection (see Table 85-2) and is the diagnostic test of choice in most institutions.\(^3\) Findings suggestive of aortic dissection include dilation of the aorta, identification of an intimal flap, and the clear demonstration of both the false and true lumina (Fig. 85-2). Dynamic scanning, in which rapid scans are obtained at multiple levels immediately after a bolus injection of an intravenous contrast agent, improves the accuracy of the CT scan in the diagnosis of aortic dissection by allowing detection of differential filling rates in the true and false lumina (see Fig. 85-1). Dynamic scanning performed with helical CT improves sensitivity and specificity.\(^4\) Sixty-four–slice multidetector computed tomography (MDCT) may soon alter the approach to chest pain in the emergency department (ED). MDCT may reliably evaluate for coronary artery disease,\(^5\) pulmonary embolus, and aortic dissection as a “triple” scan.\(^6,41\)

### Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is an appealing option in the detection of low-grade aortic dissection in stable patients in whom the diagnosis is uncertain. Sensitivity and specificity are excellent (see Table 85-2).\(^7\) MRI shows the site of intimal tear, type and extent of dissection, presence of aortic insufficiency, and differential flow velocities in the true and false channels and in the aortic side branches. It requires no contrast material or ionizing radiation and is noninvasive. It is particularly useful in the evaluation of chronic aortic dissection, in the follow-up of postoperative patients, and for monitoring nonoperative patients for progression of the dissection. Its availability, however, is limited, and it is difficult to perform in unstable patients.

### Choice of Diagnostic Test

Although aortic dissection can be suspected on the basis of history and physical examination, diagnostic imaging is necessary for the diagnosis to be established. With a mortality rate in excess of 1% per hour after the onset of aortic dissection, a diagnostic study should be performed as soon as feasible.\(^6\) Frequently, more than one test is required to make the diagnosis and to assess associated complications.

The clinical strategy should consider (1) the technology available at the institution, (2) the institution-specific sensitivities and specificities for the diagnostic tests, (3) the benefits of diagnosing nondissection causes of chest pain, and (4) the ease of performing each test, especially “after hours.”\(^43\) Some tests (e.g., CT, MRI, aortography) may require moving a potentially unstable patient outside the ED. In IRAD, the initial choice of diagnostic test was CT in 61%, TTE or TEE in 33%, aortography in 4%, and MRI in 2% of patients.\(^3\) “Real-world” sensitivities of diagnostic tests in IRAD were CT 93%, TEE 88%, aortography 87%, and MRI 100%; and patients averaged 1.85 imaging studies.\(^44\) A recent meta-analysis suggested that TEE, helical CT, and MRI are of similar diagnostic value in ruling aortic dissection in or out.\(^45\)

Unless institutional circumstances do not permit, contrast CT aortography is recommended as the test of first choice. In patients with renal failure or contrast allergy, or for those considered too unstable to undergo CT scan, TEE is recommended. If the patient has a diagnostic (i.e., positive) TTE, the diagnosis is established, but an additional confirmatory study may or may not be required, depending on the preference of the physician who will be assuming care of the patient. A negative TTE does not exclude dissection, and further imaging is indicated in such cases.

### Differential Considerations

The differential diagnosis for the patient with symptoms suggestive of aortic dissection is extensive. Signs and symptoms associated with aortic dissection vary and depend on the extent of aortic and branch vessel involvement. Patients with the ultimate diagnosis of aortic dissection are often initially thought to have other conditions such as myocardial ischemia, congestive heart failure, or pulmonary embolus.\(^7\) Several clinical syndromes are particularly suggestive of aortic dissection—chest pain that is of sudden onset, migratory pain, chest pain with concomitant neurologic deficits or syncope, and chest pain with pulse deficits.\(^36\)

Although chest pain is the most common symptom of aortic dissection, it is also the most common presenting complaint of at least three other serious and more common clinical entities: acute MI, pulmonary embolus, and pericarditis. An ECG can be helpful in excluding MI, although aortic dissection and MI may coexist as a result of the dissection proceeding retrograde to the ostium of a coronary artery and causing infarction. In cases in which aortic dissection is excluded, CT may reveal other abnormalities that explain a patient’s presentation (e.g., pulmonary embolus). TEE is helpful in identifying causes of chest pain other than aortic dissection (e.g., cardiac ischemia).

When the initial presentation of the aortic dissection is pain or dysfunction in an extremity resulting from disruption of the blood supply, peripheral neurologic diagnoses should be included in the differential diagnosis. An aortic dissection may involve the carotid artery and may initially mimic a primary central nervous system lesion such as a stroke. The diagnosis of aortic dissection should be considered in any patient with a new diagnosis of pericardial effusion, pericardial tamponade, or aortic insufficiency.

### Management

#### Emergency Department

Early therapy for aortic dissection is critical and should be initiated while diagnostic tests are being performed.\(^46\) Opioids should be administered in adequate doses to control pain and decrease
sympathetic tone. Patients with aortic dissections are typically hypertensive. The two goals of medical management are to (1) reduce blood pressure and (2) decrease the rate of rise of the arterial pulse (dP/dt) to diminish shearing forces. A target blood pressure of 100 to 120 mm Hg systolic and a heart rate below 60 beats/min are recommended. Beta-adrenergic blockers are the cornerstone of aortic dissection management and are effective when used as the sole agent (in addition to opioid analgesia for pain). Because vasodilators such as sodium nitroprusside or fenoldopam reflexively increase the heart rate and may also increase the dP/dt, they necessitate concomitant use of a beta-blocker and so are a more complicated form of therapy. Initial therapy with either esmolol or labetalol, as described later, and use of opioid analgesia, such as morphine or fentanyl, for pain are recommended.

**Recommended Therapy**

Esmolol and labetalol are titratable short-acting beta-blockers that can be used as monotherapy for hemodynamic control in aortic dissection. Esmolol is an ultra-short-acting beta-blocker that is given as an initial bolus of 500 µg/kg, followed by an infusion of 50 to 200 µg/kg/min. Labetalol has both alpha- and beta-blocking activity and is given as an initial series of 20-mg intravenous boluses every 5 to 10 minutes, incrementally increased to 80 mg intravenously (IV) until a target heart rate of 60 has been reached or a total of 300 mg has been given. A maintenance infusion of labetalol is then given at 1 to 2 mg/min. If a patient is normotensive, a beta-blocker should still be used to lower the dP/dt and maintain a heart rate of 60. In patients with a history of chronic obstructive pulmonary disease or at risk for bronchospasm, a selective beta-blocker, such as metoprolol or atenolol, should be considered.

**Alternative Agents**

Sodium nitroprusside was widely used before esmolol and labetalol became available and is a reasonable agent to use but necessitates concomitant use of a beta-blocker to mitigate reflex tachycardia and requires more effort to prepare and administer. The initial infusion is 0.5 to 3 µg/kg/min, and the infusion is adjusted to reach the same hemodynamic goals described earlier.

The calcium channel blocker nifedipine is not recommended to treat aortic dissection. Nifedipine has minimal inotropic or chronotropic effects and may reflexively stimulate sympathetic activity and increase shear stress on the aortic wall. Intravenous nitroglycerin is often used initially in patients with hypertensive chest pain and possible or uncertain aortic dissection. Nitroglycerin is a less effective arterial dilator than nitroprusside and less desirable than nitroprusside for the treatment of patients with aortic dissection. Like nitroprusside, nitroglycerin should be accompanied by a beta-blocker. Some physicians prefer an infusion of fenoldopam over nitroprusside, but this has not been specifically studied in patients with aortic dissection.

Patients with hypotension secondary to aortic rupture or pericardial tamponade should be resuscitated with intravenous fluids and immediately transported to the operating room if they are to have a chance to survive. Blood pressure should be measured in all four limbs, if necessary, to ensure that the condition is not a pseudohypotension caused by an intimal flap obstructing the extremity in which the blood pressure is measured. In patients with electromechanical dissociation or marked hypotension, pericardiocentesis may raise the blood pressure pending definitive surgery.

**Surgery**

Type A acute aortic dissections require prompt surgical treatment. The aortic segment containing the original intimal tear is resected when possible, with graft replacement of the ascending aorta to redirect blood into the true lumen. If aortic insufficiency is present, it can be corrected through aortic valve resuspension or replacement. Patients with type A dissections have an in-hospital mortality rate of 27% when treated surgically versus an in-hospital mortality of 56% when treated medically.

Definitive treatment of type B acute aortic dissections is less clear. These patients in general tend to be worse surgical risks. Uncomplicated distal dissections have traditionally been treated with blood pressure control, and patients have an in-hospital mortality of 10% when treated in this manner. Surgery has been reserved for patients who have persistent pain, uncontrolled hypertension, occlusion of a major arterial trunk, frank aortic leaking or rupture, or development of a localized aneurysm. These patients have an in-hospital, 30-day mortality rate of 32%. A “deadly triad” of absence of chest pain, hypotension, and branch vessel involvement is an independent predictor of in-hospital death.

**Interventional Therapy**

Interventional stent-graft and fenestration techniques are replacing surgery for complicated type B dissections in some centers, especially for patients with renal and mesenteric ischemia. Patients treated with interventional therapy have an in-hospital mortality rate of 6.5%. Interventional therapy for stable type B dissections is currently under study, and treatment decisions should rest with the primary treating physician. In general, interventional endovascular techniques are not applicable to type A dissections.

**DISPOSITION**

Patients with chronic aortic dissection have already survived their period of greatest mortality risk and are usually treated by blood pressure control and close monitoring unless complications lead to surgery. Regardless of the type of definitive therapy, all patients who have sustained and survived an aortic dissection require careful long-term treatment. Major complications that may occur with time are rediscission, the development of a localized aneurysm, and progressive aortic insufficiency.

**KEY CONCEPTS**

- Most patients with aortic dissection have chest pain, typically of sudden onset, sharp, and migratory. Chest pain associated with neurologic symptoms or syncope increases the likelihood of aortic disease.
- Physical examination findings may include pulse deficit, aortic insufficiency murmur, or neurologic findings, but often the physical examination is not diagnostic, and imaging is essential to establish or exclude the diagnosis of dissection.
- Of the confirmatory tests, CT aortography is recommended. Transesophageal echocardiography also is an excellent test and can be used when CT is not available or for patients with contrast allergy, renal insufficiency, or critical illness that precludes CT scan.

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


25. Huzii H, et al: Young adult patients with short dissection length and thrombosed false lumen without ulcer-like projections are liable to have false-negative results of D-dimer testing for acute aortic dissection based on a study of 113 cases. *Circ J* 2006; 70:1598-1601.


