

AORTIC DISSECTION

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Patients with dissection of the thoracic aorta have challenged physicians for centuries. The precise etiology of thoracic aortic dissection is uncertain but the pathogenesis and clinical sequelae have been fairly well documented over the last 50 years. Disruption of the aortic intima and a variable depth of the media permit the force of aortic blood flow to be redirected from the true lumen into a dissection plane within the media. This is often a lethal event but may result in a chronic condition with aneurysmal dilatation of the dissected aorta and the potential for aortic rupture. Currently, diagnostic algorithms and the subsequent management of thoracic aortic dissection are based primarily on dissection location and on the timing of presentation. Newer imaging modalities, improved medical management, and advances in surgical technique have resulted in improved morbidity and mortality.

The earliest descriptions of aortic dissection appeared in the seventeenth and eighteenth centuries, but Maunoir is credited with first referring to the process as aortic “dissection.”^{1,2} Early attempts at surgical treatment met with limited clinical success but certain concepts such as fenestration persisted and are still in use today.^{3,4} During the era of cardiopulmonary bypass, innovators such as DeBakey and Cooley described surgical correction and altered the natural history of this disease.^{5,6} Current surgical procedures for the treatment of aortic dissection have been modified since the pioneering work of these and other earlier investigators, but the concepts introduced continue to guide treatment.

Incidence

Aortic dissection is the most frequently diagnosed lethal condition of the aorta and occurs more frequently than rupture of abdominal aortic aneurysm in the United States.⁷ There is an estimated worldwide prevalence of 0.5 to 2.95 of 100,000 persons/yr; the prevalence ranges from 0.2 to 0.8 of 100,000 persons/yr in the United States, resulting in roughly 2,000 new cases per year. These figures are, however, only an estimate. In one autopsy series, the antemortem diagnosis was made in only 15%, revealing that many immediately fatal events go undiagnosed.⁸

Classification

Classification systems for aortic dissection have been critical to design strategies for diagnosis and the subsequent management of individual patients. Although several classification systems exist, the two variables necessary to categorize patients are the location and the timing of dissection. *Acute dissection* has traditionally been used to describe presentation within the first 2 weeks of an event. The more recently added *subacute* designation is used to describe the period between 2 weeks and 2 months, while the term *chronic dissection* is reserved for those presenting at greater than 2 months following the initial event.

The two classification systems most frequently used in clinical practice are the DeBakey and Stanford systems (Figure 36-1). That proposed by DeBakey is a taxonomic system that differentiates between aortic dissection involving only the ascending aorta and those that additionally involve the descending aorta.⁹ This system provides the greatest opportunity to segregate like patients for subsequent comparative research. In contrast, the Stanford system proposed by Daily is a functional classification system.¹⁰ All dissections that involve the ascending

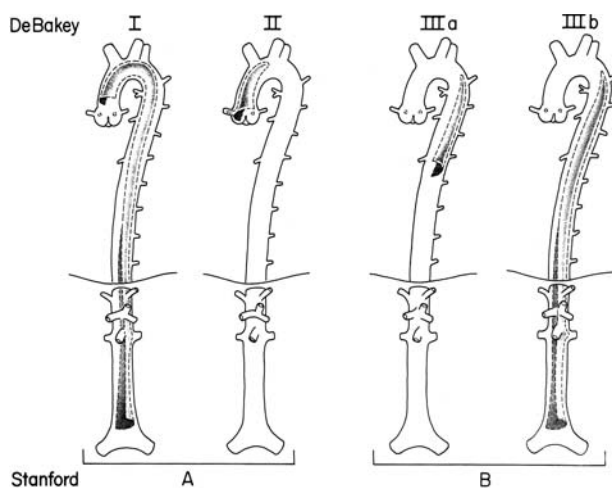


FIGURE 36-1. The DeBakey and Stanford classification systems for thoracic aortic dissection.

aorta are grouped together as type A, regardless of where the primary tear occurs; the clinical behavior of patients with aortic dissection is essentially determined by involvement of the ascending aorta. Critics of the Stanford system suggest that individual patients in the type A classification may be quite different, given the distal extent of dissection. Such a heterogeneous group would not permit rational comparison of similar groups of patients in which the ascending aorta is involved. The descending aortic dissection classifications, DeBakey type III (a and b) and Stanford type B, are equivalent. The Stanford system is used throughout this chapter.

The clinical characteristics of patients suffering acute aortic dissection are somewhat different between the two types (Table 36-1). Type A dissections occur with a greater overall frequency. Both type A and type B dissections occur more frequently in males in the sixth decade of life, while patients with type A dissection are slightly younger. Arterial hypertension is a strong risk factor for type B dissection but less so for type A, whereas the connective tissue disorders are seen more frequently in patients with type A dissection.

Etiology and Pathogenesis

There are several hypotheses regarding the etiology of the intimal disruption (primary tear) that permits aortic blood flow to create a cleavage plane within the media of the aortic wall (false lumen). Originally, this was viewed as a consequence of a biochemical abnormality within the media upon which normal mechanical forces in the aorta acted to create an intimal tear. The link between the abnormal media, termed *cystic medial necrosis* or *degeneration*, and the primary tear has not been scientifically established.

TABLE 36-1. Clinical Characteristics of Patients Presenting with Acute Type A and B Thoracic Aortic Dissection

| | Type A | Type B |
|----------------------------|-----------------------------|-------------------|
| Frequency | 60–75% | 25–40% |
| Sex (M:F) | 1.7–2.6:1 | 2.3–3:1 |
| Age (years) | 50–56 | 60–70 |
| Hypertension | ++ | +++ |
| Connective tissue disorder | ++ | + |
| Pain | | |
| Retrosternal | +++ | +,- |
| Interscapular | +,- | +++ |
| Syncope | ++ | +,- |
| Cerebrovascular accident | + | - |
| Congestive heart failure | + | - |
| Aortic valve regurgitation | ++ | +,- |
| Myocardial infarction | + | - |
| Pericardial effusion | +++ | - |
| Pleural effusion | +,- | +++ |
| Abdominal pain | +,- | +,- |
| Peripheral pulse deficit | Upper and lower extremities | Lower extremities |

+ and - indicate degree of presence or absence, respectively.

In fact, medial degeneration is found in only a minority of patients with acute aortic dissection and most are children.¹¹ This theory has lost support over the years. Alternatively, there are data that support a relationship between aortic dissections and intramural hematoma. Advocates of this theory suggest that bleeding from vasa vasorum into the media creates a mass, which results in localized areas of increased stress in the intima during diastole. These areas then permit intimal disruption. In fact, between 10 and 20% of patients thought to have acute aortic dissection are found to have intramural hematoma, suggesting that it may be a precursor to dissection.¹² Penetrating atherosclerotic ulcers have been implicated as the source of intimal disruption in certain cases, yet support for the concept has waned over the years. The pattern of atherosclerotic involvement of the thoracic aorta resulting in penetrating ulcer and the frequency of dissection throughout the aorta do not support this theory.

Once a cleave plane exists in the media, the aortic wall floating within the lumen is termed the *dissection flap* and is composed of the aortic intima and partial thickness media. The primary tear is usually greater than 50% of the circumference of the aorta, but the full circumference is rarely involved. The primary tear in type A dissection is usually located on the right anterior aspect of the ascending aorta and follows a somewhat predictable course spiraling around the arch and into the descending thoracic and abdominal aorta on the left and posteriorly. The dissection may propagate in a retrograde fashion for a variable distance as well, to involve the coronary ostia; this occurs in roughly 11% of all dissections.¹³ Myocardial ischemia and aortic rupture into the pericardium are the cause of death in as many as 80% of deaths from acute dissection. Often, the distal false lumen communicates with the true lumen through one or more fenestrations within the dissection flap. The false lumen may also end blindly in as many as 4 to 12% of all dissections, in which case blood in the false lumen thromboses. It may also penetrate the adventitia, causing rupture and death. Regardless of whether the true lumen and false lumen communicate, perfusion of aortic side branches may be compromised by the dissection, causing end-organ ischemia. If these acute complications are avoided, the weakened outer aortic wall, composed of partial media and the adventitia, may dilate, resulting in later aneurysm formation. This long-term complication is the reason for operation in the majority of chronic dissections regardless of type.

Although no single disorder is responsible for aortic dissection, several risk factors have been identified that could damage the aortic wall and lead to dissection (Table 36-2). These include direct mechanical forces on the aortic wall (ie, hypertension, hypervolemia, derangements of aortic flow) and forces that affect the composition of the aortic wall (ie, connective tissue disorders or direct chemical destruction). Hypertension is the mechanical force most often associated with dissection and is found in

TABLE 36-2. Risk Factors for Type A and B Thoracic Aortic Dissection

| |
|--------------------------------|
| Hypertension |
| Connective tissue disorders |
| Ehlers-Danlos syndrome |
| Marfan's disease |
| Turner syndrome |
| Cystic medial disease of aorta |
| Aortitis |
| Iatrogenic |
| Atherosclerosis |
| Thoracic aortic aneurysm |
| Bicuspid aortic valve |
| Trauma |
| Pharmacologic |
| Coarctation of aorta |
| Hypervolemia (pregnancy) |
| Congenital aortic stenosis |
| Polycystic kidney disease |
| Pheochromocytoma |
| Sheehan syndrome |
| Cushing syndrome |

greater than 75% of cases. Although increased strain on the aortic wall is intuitive, the mechanism by which hypertension actually leads to dissection is unclear. Similarly, hypervolemia, high cardiac output, and an abnormal hormonal milieu certainly contribute to the increased incidence of dissection in pregnancy, but the mechanism is unclear. Atherosclerosis is not a risk factor for aortic dissection except in pre-existing aneurysms or in the case of atherosclerotic ulceration, which may lead to dissection in the descending thoracic aorta. The fact that distal aortic dissection infrequently accompanies traumatic aortic transection reveals that a primary tear alone is insufficient for subsequent aortic dissection. It should be noted, however, that iatrogenic trauma to the aortic intima may result in dissection. Catheterization procedures, aortic root and femoral artery cannulation for cardiopulmonary bypass, aortic cross-clamping, surgical procedures performed on the aorta (aortic valve replacement and aortocoronary bypass grafting), and placement of intra-aortic balloon pumps have all been reported to result in dissection.

The adventitia provides most of the tensile strength of the aortic wall, with little contribution from the media. The media is composed of concentrically arranged smooth muscle interposed with connective tissue proteins such as collagen, elastin, and fibrillin within the ground substance. Abnormal constituents of the media as in certain connective tissue disorders such as Marfan's disease and Ehlers-Danlos syndrome are associated with aortic dissection. Marfan syndrome is an autosomal dominant inherited disorder in which a point mutation of the fibrillin-1 gene (*FBNI*), located on the long arm of chromosome 15, results in an abnormal media. The incidence of Marfan syndrome is approximately 1 per 5,000 live births. There are, however, many incomplete forms of the disease and as many as 25% of cases may be sporadic, in which no

fibrillin abnormalities are observed. Type IV Ehlers-Danlos syndrome is a connective tissue disorder of the pro α 1(III) chain of type III. The structurally abnormal media is susceptible to dissection. There are also familial aggregations of dissection without discernible biochemical or genetic abnormalities. Congenital abnormalities of the aortic valve, including bicuspid aortic valve, were found in nearly 10% of fatal dissections in one series. These are usually associated with a pre-existing aortic aneurysm.

Acute Dissection of the Aorta

Clinical Presentation

As many as 40% of patients suffering acute aortic dissection die immediately. Those surviving the initial event may be stabilized with medical management, and it is these patients in whom surgical treatment of aortic dissection has altered the natural history of the disease. The diagnosis of aortic dissection requires a high level of suspicion and should be considered in the setting of severe, unrelenting chest pain that is present in greater than 90% of such patients. Up to 30% of patients ultimately diagnosed with acute dissection are first thought to have another diagnosis. Patients often have no previous episodes of similar pain and are often quite anxious. Pain is located in the midsternum in dissection of the ascending aorta and in the interscapular region for dissection of the descending thoracic aorta (see Table 36-1). It is not unusual for the location of maximum pain to change as the dissection extends in an antegrade or retrograde direction. The character of the pain is often described as "ripping" or "tearing" and is constant with greatest intensity at the onset. Painless dissection has been described and usually occurs in the setting of an existing aneurysm where the pain of a new dissection may not be differentiated from chronic aneurysm pain. Patients may also have signs or symptoms related to malperfusion of the brain, limbs, or visceral organs. These findings may even dominate the presentation following the initial episode of pain. Elements of the past medical history such as primary hypertension, presence of aneurysmal disease of the aorta, or familial connective tissue disorders help to make the diagnosis. Illicit drug use is an increasingly important predisposing factor to dissection that must be investigated.

Patients suffering acute dissection appear ill. Tachycardia may be accompanied by either hypertension in the setting of baseline essential hypertension or hypotension secondary to aortic rupture, pericardial tamponade, acute aortic valve regurgitation, and even acute myocardial ischemia following involvement of the coronary ostia. An abnormal peripheral vascular examination is present in less than 20% of patients with acute aortic dissection, but when present may indicate the type of dissection. Absence of pulses in the upper extremity suggests ascending aortic involvement, whereas pulse deficits in the lower

extremities speak to involvement of the distal aorta. These findings are subject to change as the dissection progresses or reentry into the true lumen occurs. Auscultation of the heart may reveal a diastolic murmur or an S3 indicating left-heart volume overload consistent with acute aortic valve insufficiency. Physical exam findings such as jugular venous distension and a pulsus paradoxus are signs of pericardial tamponade that should be identified in any unstable patient to initiate the correct diagnostic and treatment algorithms. Unilateral loss of breath sounds in one thorax, usually the left, may indicate hemothorax as a result of aortic leak or rupture with hemothorax. Alternatively, a pleural effusion may exist secondary to pleural inflammation related to the dissection. This finding requires additional evaluation prior to treatment.

A complete central and peripheral neurologic exam is critical in that abnormalities are present in up to 40% of acute type A dissections. Involvement of the brachiocephalic vessels with loss of brain perfusion may result in transient syncope or stroke. Stroke rarely improves with restoration of blood flow and may even cause hemorrhage and brain death, yet surgery is indicated in such patients. Loss of perfusion to intercostal or lumbar arteries may result in spinal cord ischemia and paraplegia. Peripheral nerve ischemia as a result of malperfusion may yield findings similar to spinal cord malperfusion and should be discerned as these patients often improve with restoration of blood flow. Acute aortic dissection may also cause superior vena cava syndrome, vocal cord paralysis, hematemesis, Horner syndrome, hemoptysis, and airway compression as a result of local compression and mass effects.

Diagnostic Studies

No routine diagnostic study will reliably yield the diagnosis of acute aortic dissection. An electrocardiogram (ECG) is routinely performed and reveals no ischemic changes in most cases. Obvious ischemic changes are present in as many as 20% of acute type A dissections, yet only nonspecific repolarization abnormalities alone are present in up to one-third of patients with coronary ostial involvement. The chest x-ray is abnormal in 60 to 90% of patients with acute dissection (Table 36-3). Although most patients have at least one, if not several, abnormal finding(s), a normal chest x-ray does not rule out the diagnosis. Blood tests obtained at the time of initial obser-

TABLE 36-3. Abnormalities Identified on Chest Radiography in Acute Aortic Dissection

| |
|--------------------------------------|
| Widened mediastinum |
| Irregular aortic contour |
| Aortic apical cap |
| Tracheal displacement |
| Depression of left mainstem bronchus |
| Esophageal displacement |
| Obscure aortic-pulmonary window |
| Pleural effusion |

vation are usually unremarkable. There is frequently a mild to moderate leukocytosis. Anemia may result from sequestration of blood or hemolysis. Liver function tests, serum creatinine, myoglobin, and lactic acid may all be abnormal in the setting of certain malperfusion syndromes, depending on duration.

Diagnostic Imaging

The goal of diagnostic imaging is to rapidly demonstrate dissection with minimal distress for the patient. Five advanced imaging modalities are used clinically to diagnose acute aortic dissection: computerized tomography, echocardiography, magnetic resonance imaging, aortography, and intravascular ultrasound. The relative benefits, disadvantages, and diagnostic accuracy of each must be considered in choosing the most appropriate study for a particular clinical situation (Table 36-4). In addition to the essential diagnostic information, certain tests may provide additional important information such as the site of intimal disruption, reentry points, whether there is flow or thrombus in the false lumen, status of the aortic valve, presence and nature of myocardial ischemia, and brachiocephalic and aortic branch vessel involvement.

Aortography was first used to diagnose acute dissection in 1939 and, until recently, was considered the gold standard for diagnosis. It is an invasive test requiring nephrotoxic contrast media in which the aorta is visualized in multiple two-dimensional projections. The diagnosis of dissection depends upon visualization of the intimal flap, two distinct lumen or compression of the true lumen by flow through an adjacent false lumen (Figure 36-2). Indirect signs of dissection include the presence of branch vessel abnormalities and an abnormal intimal contour on injection of the false lumen. The status of the aortic valve may be evaluated, and coronary angiography in the setting of type A dissections is possible only with this diagnostic test. In reality, the coronary ostia are involved in only 10 to 20% of acute type A dissections, and coronary atherosclerosis is present in 25% of all patients with acute aortic dissection. Taking time to perform this test is debatable given that the coronary ostia are easily evaluated at the time of surgery. Aortography is especially useful in type B dissections with evidence of mesenteric ischemia or oliguria, and in type A dissections

TABLE 36-4. Sensitivity and Specificity of Various Imaging Modalities Useful for the Diagnosis of Thoracic Aortic Dissection

| <i>Imaging Study</i> | <i>Sensitivity (%)</i> | <i>Specificity (%)</i> |
|--------------------------------------|------------------------|------------------------|
| Aortography | 80–90 | 88–95 |
| Computerized tomography (CT) | 90–100 | 90–100 |
| Intravascular ultrasonography (IVUS) | 94–100 | 97–100 |
| Echocardiogram | | |
| Transthoracic | 60–80 | 80–96 |
| Transesophageal | 90–99 | 85–98 |
| Magnetic resonance imaging (MRI) | 98–100 | 98–100 |

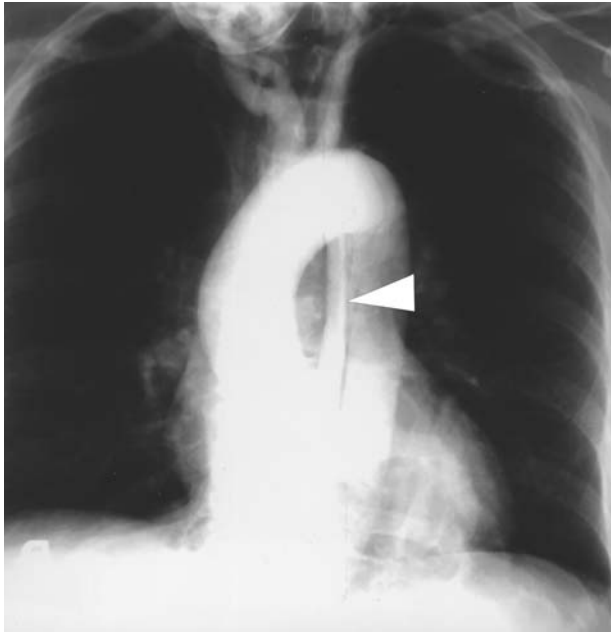


FIGURE 36-2. Aortogram of acute type B dissection illustrating differential contrast enhancement of the true and false lumen in the descending thoracic aorta. The intimal flap (arrowhead) can be seen separating the two lumens.

with signs of malperfusion, because catheter-based intervention may be possible. Aortography may yield false-negative results with thrombosis of one lumen or when contrast equally opacifies each lumen, impairing distinction of a separate true and false lumen. The diagnosis of intramural hematoma is also difficult given the absence of intimal disruption. Aortography requires the presence of skilled personnel whose availability varies with institution. The time required to assemble this team is a detriment when compared to other immediately available diagnostic tests given the high early mortality following acute dissection.

Intravascular ultrasonography (IVUS) is a catheter-based imaging tool that provides dynamic imaging of the aortic wall and a pulsatile intimal flap in patients with aortic dissection. It is particularly useful in delineating the distal extent of dissection and for identifying the true and false lumen in questionable cases during aortography. High-resolution images of the normal three-layered aortic wall are differentiated from the abnormally thin wall adjacent to the false lumen. Because the aortic wall itself is imaged, intramural hematoma and penetrating atherosclerotic ulcers may also be identified. Currently, as an isolated imaging study, it is time-consuming and requires skilled personnel and therefore may not be useful as an initial imaging study. It may be most useful in combination with aortography in the setting of negative imaging studies when a high clinical suspicion remains.

Helical computerized tomography (CT) scanning has become widely available over the last 20 years and is now

used most frequently to make the diagnosis of acute aortic dissection. It requires intravenous contrast medium that may limit its use in certain clinical situations but generates images, familiar to most practitioners, that result in a high sensitivity and specificity. This technique can be performed quickly, fulfilling the requirements for use in the early management of acute dissection. Additional structures such as the pleural and pericardial spaces are imaged. The aortic branch vessels are also evaluated with CT scanning; involvement of the brachiocephalic vessels is identified with nearly 96% accuracy. The diagnosis of dissection requires two or more channels separated by a dissection flap (Figure 36-3). Transaxial two-dimensional images can be reconstructed to display three-dimensional reconstructions of the aorta that aid in diagnosis and also are useful for operative planning.

Conventional magnetic resonance imaging (MRI) and the newer contrast-enhanced magnetic resonance angiography generate superior images reliably demonstrating aortic dissection (Figure 36-4). In fact, some consider this the new gold-standard imaging study given the published diagnostic accuracy. Dissection is identified as an intraluminal membrane separating two or more channels. It provides detailed images of the entire aorta, the pericardium, and pleural spaces. Cine imaging may also be used to evaluate left ventricular function, the status of the aortic valve, and flow in aortic branch vessels, as well as flow in the false lumen. It is, however, time-consuming and not widely available, and the presence of ferromagnetic metal contraindicates its use. Another disadvantage of MRI is that artifact is identified in up to 64% of studies, which underscores the need for expert radiologic interpretation of the images. These factors obviously limit its use in the acute setting, but it is a reliable, noninvasive imaging study that is perhaps best used to follow chronic dissections (Figure 36-5).



FIGURE 36-3. Axial CT arteriogram image of acute type B dissection. The dissection flap divides the aorta into two distinct lumina within the descending thoracic segment.

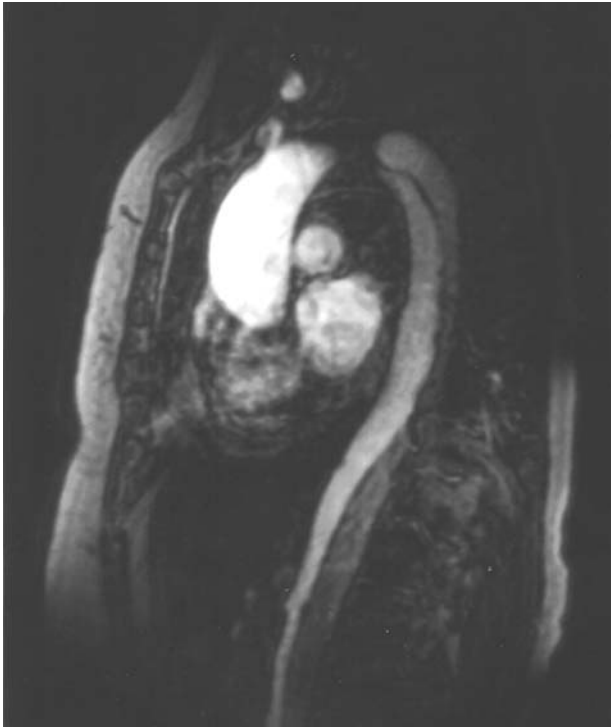


FIGURE 36-4. Sagittal contrast-enhanced magnetic resonance image of acute type B dissection. The dissection extends the entire length of the thoracic and abdominal aorta.

Transesophageal echocardiography (TEE) is currently a preferred method for making the diagnosis of acute aortic dissection. It is widely available, requires no intravenous contrast, and generates dynamic images of the aorta and its branches from which the diagnosis can be made. Criteria for making the diagnosis include visualization of an echogenic surface separating two distinct lumen, repeatedly, in more than one view, and which can be differentiated from normal surrounding cardiac structures (Figure 36-6). The true lumen is identified by expansion during systole and collapse in diastole. Communication between the true lumen and false lumen may be visualized as flow across tears in the dissection flap using color Doppler; similarly the absence of flow indicates false lumen thrombosis. TEE additionally may provide high-quality images of the aortic valve and pericardial space. The coronary ostia and left ventricular function may be assessed to provide information regarding regional wall motion abnormalities to rule out myocardial ischemia as part of the differential diagnosis or as complicating type A dissection. The area surrounding the aorta is also visualized, and therefore, periaortic hematoma indicating leak may be identified. Although the safest setting in which to perform TEE is the operating room under general anesthesia, it can be performed using local anesthesia and minimal sedation in a monitored setting. Relative contraindications to TEE

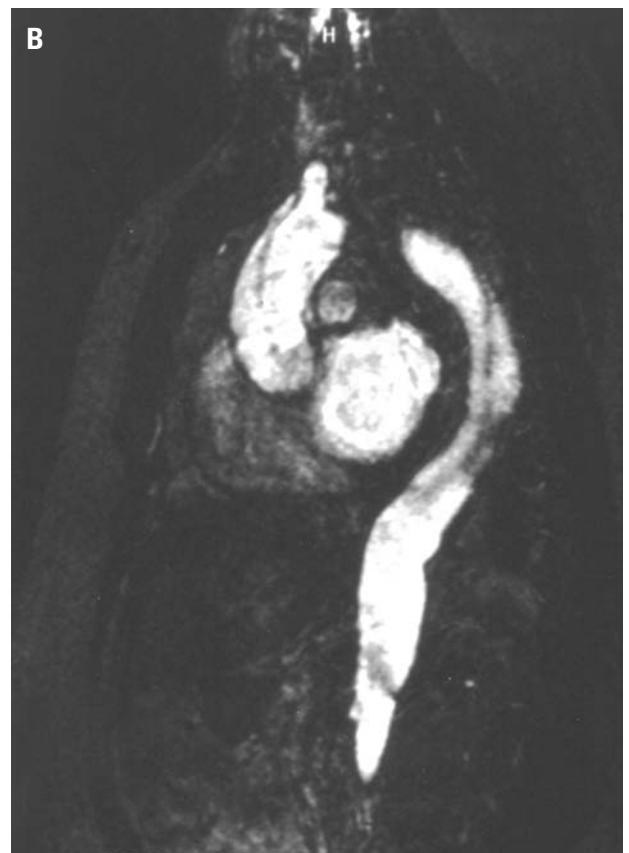


FIGURE 36-5. Axial (A) and sagittal (B) contrast-enhanced magnetic resonance images of a chronic type A dissection.

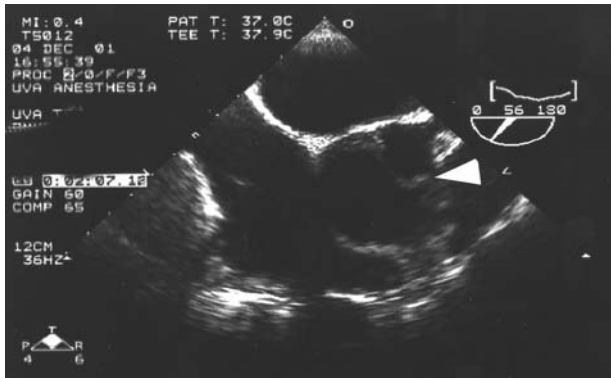


FIGURE 36-6. Transesophageal echocardiographic image of an acute type A dissection. The dissection flap (arrowhead) within the lumen of the aortic root included one aortic valve commissure and freely prolapsed through the aortic valve orifice during diastole.

include esophageal abnormalities and a full stomach, but recognition of these conditions permits safe examination with few complications in the vast majority of patients. Transthoracic echocardiography provides images of the ascending aorta and sections of the aortic arch that may yield the diagnosis but with much less sensitivity than transesophageal imaging (Figure 36-7). As such, transthoracic imaging may prove useful but is generally insufficient to reliably make the diagnosis. Transthoracic evaluation is additionally limited by patient-related factors, including body habitus and emphysema, as well as by operator experience. A negative transthoracic study should be complemented by a transesophageal study that provides greater detail of the entire aorta. To exclude the diagnosis of aortic dissection by TEE, a competent operator must visualize the entire thoracic aorta in a detailed examination.

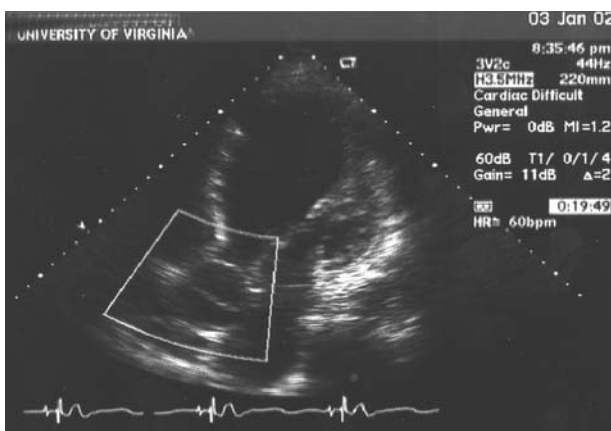


FIGURE 36-7. Transthoracic echocardiographic image of acute type A dissection. Occasionally a dissection flap is identified in the ascending aorta (inset) by using this comparatively insensitive imaging tool. If possible, the diagnosis of aortic dissection should be confirmed by an additional imaging study prior to initiating a particular management algorithm.

Initial Management

Fifty percent of patients suffering acute aortic dissection are dead within 48 hours. A conventional wisdom has evolved that acute aortic dissection carries a “1% per hour” mortality. This demonstrates that patients surviving a dissection must be quickly and aggressively diagnosed and managed.

The initial evaluation of a stable patient suspected of having aortic dissection includes a detailed history and physical examination focusing on those elements likely to rule in the diagnosis. Blood pressure is measured in both arms and immediately treated to achieve a target systolic blood pressure between 90 and 110 mm Hg. During this time, peripheral, and sometimes central, venous and arterial access is obtained; blood is sent for complete blood count, serum electrolytes, creatine kinase with myocardial isoenzymes, and troponin, and a blood type and screen is obtained. The unstable patient may require intubation and mechanical ventilation and possibly placement of a pulmonary artery catheter if necessary. The site of this initial evaluation and resuscitation is determined primarily by the hemodynamic stability of the patient. The unstable patient belongs in the operating room, whereas the stable patient permits a more detailed diagnostic approach from which management follows on an urgent basis. Therefore, the hypotensive patient who may be hypovolemic as a result of blood loss into the thorax or pericardium undergoes the aforementioned evaluation and resuscitation on transfer to the operating room. It is preferable to avoid procedures such as transesophageal echocardiography or central line placement on an awake patient outside the operating room because hypertension resulting from patient discomfort could precipitate aortic rupture or propagation of dissection.

Recognizing the natural history of patients with aortic dissection dictates that management occurs as part of the initial diagnostic evaluation. Blood pressure control in hypertensive patients with pain should first be treated with narcotic analgesics. In general, the goals of hypertension management in acute aortic dissection are twofold.¹⁴ First, aortic wall stress is lowered by decreasing the systolic blood pressure, which reduces the possibility of rupture. Second, shear stress on the aorta is decreased by minimizing the rate of rise of aortic pressure to decrease the likelihood of dissection propagation, so-called anti-impulse therapy. The drugs most commonly used for these purposes are sodium nitroprusside and esmolol. Sodium nitroprusside is a direct arterial vasodilator with a fast onset and short duration of action, which make it ideal to achieve a target systolic blood pressure between 90 and 110 mm Hg. The rate of rise of aortic pressure, however, is increased when sodium nitroprusside is used alone. Esmolol is added to decrease the inotropic state of the myocardium and to decrease the heart rate. This drug is a β_1 selective blocking agent with a short half-life, which can easily be titrated to achieve the target blood pressure.

Loading doses for esmolol and sodium nitroprusside should be avoided to prevent hypotension. Alternative β_1 -blocking drugs, such as propranolol or metoprolol, and the combined α - and β -blocker labetalol, are appropriate in the subacute phase. Alternatively, calcium channel blockers may be necessary to reduce systolic blood pressure in those patients with a contraindication to β -blocker use. There are, however, no compelling data supporting their efficacy in acute dissection.

Diagnostic Strategy

The evaluation of suspected acute aortic dissection begins with determination of the likelihood of the diagnosis being correct and the hemodynamic stability of the patient. Unstable patients should undergo ECG and chest x-ray and be transferred to the operating room immediately. These patients should be intubated, mechanically ventilated, and have monitoring lines placed, at which time TEE is performed. Initial medical management is initiated as soon as the diagnosis is suspected prior to transfer to the operating room for subsequent evaluation and management. If TEE fails to yield a diagnosis, then a hemodynamically unstable patient will at that point have a protected airway and invasive monitoring lines placed for subsequent evaluation of alternate diagnoses. If dissection is suspected even in the setting of a negative TEE, CT or aortography (potentially with IVUS) are the next studies of choice.

At the University of Virginia, clinically and hemodynamically stable patients are first evaluated with CT scanning. With a CT scanner in the emergency room, it takes less than 15 min to obtain these data. If that study is negative, yet the diagnosis is still entertained, a transesophageal echocardiogram is obtained. In a recent review, an average of 1.8 imaging studies were used to correctly diagnose acute aortic dissection.⁸

Surgical Indications

The presence of ascending aortic involvement is generally an indication for operative management given that older data indicate a > 90% mortality without surgery in 2 weeks (Table 36-5). The goals are to prevent aortic rupture into the pericardium or pleural space, and to prevent potential coronary ostial or aortic valve involvement. It is important to note that neurologic complications at the time of presentation, including stroke or paraplegia, are not contraindications to surgical correction, as some patients will improve. Thrombosis of either lumen is also not a contraindication to surgery. Although immediate surgical correction of type A dissection is recommended, there are data suggesting that patients who survive type A dissection and who present at greater than 2 weeks may safely undergo elective operation. In fact, poor-risk patients in that same cohort had an acceptable early survival and short-term outcome with medical management alone.¹⁵ In another recent study, type A dissection was

TABLE 36-5. Operative Indications for Types A and B Thoracic Aortic Dissections

| <i>Dissection Type</i> | <i>Operative Indication</i> |
|------------------------|---|
| Acute | |
| Type A | Presence |
| Type B | Rupture Malperfusion Progressive dissection Failure of medical management |
| Chronic | |
| Type A | Symptoms related to dissection (congestive failure, angina, aortic regurgitation, stroke, pain) Malperfusion Aneurysm |
| Type B | Symptoms related to dissection Malperfusion Aneurysm |

managed medically in 28% of patients for various reasons, with a 58% in-hospital mortality for that group.¹⁶ Despite this recent recognition that early mortality following type A dissection may be lower than expected, immediate surgical intervention is indicated in the majority of patients.

Aortic rupture and visceral malperfusion are the most frequent causes of death in acute type B dissection. These, however, occur much less frequently when compared with type A dissection, and between 70 and 80% survive the acute and subacute phases with medical management alone. Such success with medical management has traditionally created surgical indications for acute type B dissections, which include complications of medical management or progression of the disease. Specifically included are contained or free aortic rupture, acute aortic expansion, malperfusion syndrome, pain or progression of dissection despite maximal medical management, and failure of medical management to control hypertension. Although medical management of acute type B dissection is the rule in most centers, some centers advocate immediate surgical intervention in selected patients with uncomplicated acute type B dissection. Other factors that may indicate early operation in acute type B dissection are the presence of Marfan syndrome, a large false aneurysm, arch involvement, and presumed medical compliance issues.¹⁷ As in acute type A dissection, acute paralysis does not contraindicate surgery because patients can have remarkable improvement following revascularization.

There is some debate over the treatment of patients diagnosed with intramural hematoma and penetrating atherosclerotic ulcer. The natural history of these so-called dissection variants has made the issue less confusing. Intramural hematoma may lead to acute rupture in up to 35% of patients, whereas regression or no change in the hematoma is seen in the majority of medically managed patients surviving the initial period.¹⁸ Those patients with penetrating ulcer were found to have a rate of acute

rupture of 42%. The current recommendations from the Yale group are early operative intervention for intramural hematoma and penetrating ulcer involving the ascending aorta. In the descending aorta, medical management with anti-impulse therapy and a low threshold for operative intervention result in the lowest mortality. These patients require continuous observation and repeat diagnostic imaging after 3 to 5 days in the hospital to monitor the lesion.

Surgical Management

The surgical management of acute aortic dissection must be tailored to the type of dissection and modified by specific patient-related factors, but several general rules apply. In acute type A dissection with involvement of the aortic valve, preservation of the native valve is preferred and accomplished in nearly 85% of cases. Preservation of the aortic valve in patients with connective tissue disorders is performed by some, but long-term data regarding longevity of valve competence lacking. If the valve is preserved in these patients, the sinuses must be replaced or re-created by using various surgical techniques or grafts to prevent aneurysmal dilatation. Preservation of the aortic valve in patients with congenitally bicuspid aortic valves is a matter of debate. Use of an aortic homograft is an alternative surgical strategy for those requiring replacement of the valve and ascending aorta. This is an ideal solution in individuals who have a contraindication to anticoagulation or in young reproductive females.

Management of the aortic arch in patients with type A dissection is determined by whether the intima of the arch is intact or violated. If it is intact, the aortic layers can usually be reunited using either Teflon felt or glue and that site of the arch used for distal anastomosis (Figure 36-8). If the intima is fractured, the brachiocephalic vessels may require reimplantation as a Carrel patch or even individually after reuniting the dissected layers. Isolated dissections involving the aortic arch are rare and are treated as type A dissections with resection of the arch at the site of intimal disruption. Acute type A dissections resulting from retrograde extension into the arch are treated by replacing the arch and reuniting the dissected layers proximally or by going beyond the dissection to normal aorta proximally for the proximal anastomosis. In effect, this converts the dissection to type B.

The goals of surgical management of complicated acute type B dissection are the prevention of free rupture and perfusion of all end organs in the absence of symptoms. The particular operation is based on the specific operative indication and the pattern of dissection. Replacement of the descending thoracic aorta usually begins at the left subclavian artery and the distal anastomosis is ideally made to normal distal thoracic aorta. When dissection involves the entire thoracic aorta, the distal aortic layers are reunited with Teflon felt or glue and that site is used for the distal anastomosis. No more than the proximal third of

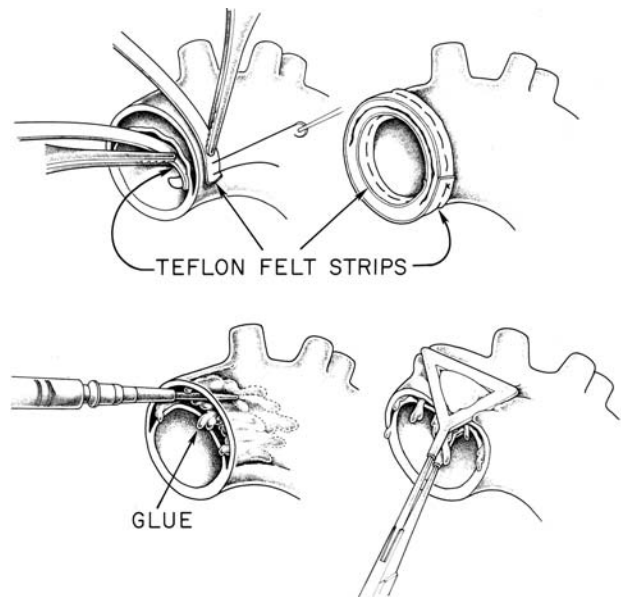


FIGURE 36-8. The intimal flap is reunited with the aortic wall using Teflon felt strips, commercially available glue, or both. Felt strips may be placed on the inside, outside, and/or between the dissected layers of the aorta as the layers are reunited. The specific technique for approximating the layers with glue depends upon the product; fundamentally, glue is applied between the dissected layers, which are then held together manually or with clamps for 3 to 10 min. A seal is formed between the dissected layers that can then be used for primary anastomosis or, if necessary, reinforced with Teflon felt.

the descending thoracic should be replaced if possible. The primary concern when deciding where to place the distal anastomosis in acute type B dissection is preservation of intercostal arteries perfusing the spinal cord. The incidence of paraplegia following surgery for acute type B dissections can be as high as 19%.¹⁹

Endovascular stent-grafting is currently being tested as a definitive form of management in both acute and chronic forms of type B dissections with acceptable results. Long-term data and prospective comparisons to surgery will be necessary before exclusively percutaneous management can be recommended as an alternative to surgery.

Operative Technique

Anesthesia used during the repair of aortic dissections is often narcotic based, with inhalational agents for maintenance. Single-lumen endotracheal tubes are used for procedures performed through a median sternotomy, while double-lumen endotracheal tubes are useful but rarely mandatory for procedures performed through a left thoracotomy. Monitoring lines often include central venous access with a pulmonary artery catheter and arterial pressure monitoring lines specific to the operation performed. One or two radial arterial lines and at least one femoral line are required to ensure perfusion of the upper and lower body when femoral cannulation is used for

cardiopulmonary bypass and during partial left-heart bypass. All patients require a transesophageal echocardiography probe. Core body temperature is monitored in the bladder through the Foley catheter and by using a nasopharyngeal probe. Strict blood conservation is an important aspect of the operation and at least one cell-saver device should be available. Packed red blood cells, platelets, and fresh-frozen plasma should be in the operating room at the start of the operation.

Surgical procedures for aortic dissection can be associated with significant blood loss. Coagulopathy as a result of the preoperative status of the patient, cardiopulmonary bypass, and deep hypothermic circulatory arrest contribute to excessive blood loss. Improvements in vascular graft material have all but eliminated this as a reason for intra- and postoperative blood loss. Antifibrinolytic drugs such as epsilon-aminocaproic acid and aprotinin are useful hemostatic adjuncts. Aprotinin is particularly useful when used in either the full or one-half Hammersmith regimen, and is best administered prior to the operation. In cases in which deep hypothermic circulatory arrest is used, we administer aprotinin only after the period of circulatory arrest. Patients often require transfusion of fresh-frozen plasma, platelets, and possibly cryoprecipitate. Fibrin glues and hemostatic materials such as Surgicel and Gelfoam are useful as systemic coagulopathy is corrected.

There are various options for arterial and venous cannulation sites based upon the type of dissection. Arterial cannulation of the uninvolved aortic arch is preferable in type A dissection. Alternate sites include the right subclavian artery, the innominate artery, or either femoral artery with retrograde aortic perfusion. In any case of retrograde aortic perfusion, it is essential to monitor proximal perfusion with a functioning radial arterial catheter. There is debate over which femoral artery to cannulate in the setting of lower extremity malperfusion with a pulse deficit. Dissection of the abdominal aorta often leaves the left femoral artery originating from the false lumen, and therefore, cannulation of the pulsatile right femoral artery will most often perfuse the true lumen. Potential consequences of false-lumen cannulation are retrograde dissection and malperfusion of aortic branch vessels arising from the true lumen. The solution to this situation begins with prompt recognition of the problem, which requires the appropriate monitoring lines (ie, radial arterial line) and cessation of cardiopulmonary bypass. The goal is then expeditious cannulation of the true lumen and resumption of cardiopulmonary bypass. If the chest has been opened, direct cannulation of the ascending aorta can be successful. An alternative cannulation technique is through the left ventricular apex and aortic valve. The cannula is then held in position with an ascending aortic tourniquet. Venous cannulation is usually through the right atrium by using a two-stage venous cannula while bicaval cannulation is reserved for certain cases in which retrograde cerebral perfusion is used. Because there is often involvement of the aortic valve,

the left ventricle must be vented by a catheter advanced through the right superior pulmonary vein or, rarely, through the left ventricular apex. Cardioplegia is administered retrograde through a coronary sinus catheter.

The formerly popular "clamp-and-sew" technique, which was used for repair of type B dissection, has been supplanted by the use of partial left-heart bypass. Arterial cannulation sites for this technique are either the distal thoracic aorta for limited dissections or the femoral artery for those extending beyond the thorax. Venous drainage of oxygenated blood is from the left inferior pulmonary vein or directly from the left atrium via the appendage. It may be necessary to use deep hypothermic circulatory arrest if a proximal aortic clamp cannot be positioned between the left subclavian and the left common carotid arteries or in the setting of intimal involvement of the distal arch proximal to the clamp. In this case, arterial cannulation of the left femoral artery and vein are recommended and may be complemented by assisted left femoral venous drainage if necessary.

Procedures performed on the dissected aortic arch require disruption of adequate blood flow to the brain. Cerebral protection is achieved by either cessation of electrical activity in the brain through hypothermia or by some form of continued cerebral perfusion. Deep hypothermic circulatory arrest was the first method used to perform operations on the aortic arch and remains an effective method to date for shorter procedures. Generally, periods of circulatory arrest up to 14 min are acceptable at 25°C, and periods up to 31 min appear to result in only transient neurologic sequelae at 15°C in a small number of patients.²⁰ Some warn that cooling to lower than 15°C may result in a form of nonischemic brain injury. The risk of transient neurologic dysfunction on cognitive testing during circulatory arrest is roughly 10% at less than 30 min, but increases to 15% at 40 min, 30% at 50 min, and 60% at 60 min.²¹ It is critical to correctly estimate brain temperature for expected outcome. Nasopharyngeal and tympanic temperature are measured to estimate brain temperature but are imperfect. For that reason, some groups use electroencephalographic silence to determine the appropriate point at which to discontinue cooling and perfusion. This point is reached by slowly cooling on cardiopulmonary bypass maintaining a maximal temperature gradient between perfusate and patient of < 10°C. The head is then packed in ice to maintain a low brain temperature. Methylprednisolone and thiopental administration during cooling are adjunctive measures thought by some to decrease cerebral metabolic requirements during the period of circulatory arrest. Rewarming at the end of the procedure proceeds without exceeding the 10°C temperature gradient to at least 37°C, as core body temperature often falls briefly after cessation of active warming and separation from cardiopulmonary bypass. Furosemide and mannitol are administered to initiate diuresis and as a free radical scavenger following circulatory arrest.

Continued cerebral perfusion during the period of circulatory arrest is a technique used for additional cerebral protection during operations performed on the aortic arch. Flow is delivered in either a retrograde or antegrade fashion. Retrograde cerebral perfusion is useful to flush atherosclerotic material and air from the brachiocephalic vessels. A flow rate to produce a caval pressure of 15 to 25 mm Hg is considered optimal. Recently, selective antegrade cerebral perfusion has become popular. The innominate artery and the left common carotid artery are encircled with vessel occluders and the lumen cannulated with retrograde coronary sinus cannulae. With the left subclavian artery occluded, flow rates are slowly increased to achieve perfusion pressures of 50 to 70 mm Hg at the desired circulatory-arrest temperature.

Aortic arch involvement cannot always be predicted from preoperative studies; consequently, the need for prolonged circulatory arrest is not always known. In that situation, it is useful to systemically cool to 18°C and stop perfusion for a brief period of circulatory arrest. The intima of the aortic arch is then examined. If the intima is intact, the distal anastomosis is performed and the graft cannulated, de-aired, and clamped for resumption of cardiopulmonary bypass with systemic warming. If the intima of the arch is violated, then a hemiarch reconstruction is performed. Only rarely have we done a complete arch resection for an acute dissection.

The exposure for procedures performed on the ascending aorta and the proximal arch is through a median sternotomy. This can be modified with supraclavicular, cervical, or trapdoor incisions to gain exposure to brachiocephalic vessels or the descending thoracic aorta. When dissecting the distal arch, it is important to identify and protect both the left vagus nerve with its recurrent branch and the left phrenic nerve. Replacement of the ascending aorta in type A dissections is best performed by an open distal anastomosis technique if the arch is involved (30%) or if arch involvement is unknown. The open distal anastomotic technique requires clamping the mid-ascending aorta and cardiac arrest via administration of antegrade and/or retrograde cardioplegic solution. The dissected ascending aorta proximal to the clamp is then resected, leaving 5 to 10 mm of normal aorta distal to the sinotubular ridge. The proximal aorta is then reconstructed by reuniting the dissected aortic layers between one or two strips of Teflon felt by using either 3-0 or 4-0 Prolene suture. There has also been a great deal of enthusiasm for reuniting the dissected layers using gelatin-resorcinol-formalin (GRF) glue or the newer BioGlue (Cryolife International Inc., Kennesaw, Georgia). Evaluation and surgical correction of the aortic valve is ideally performed at this time while systemic cooling continues. Once the temperature reaches 20°C, perfusion is discontinued and the aortic clamp is released. The aortic arch is inspected and repaired. If a complex aortic root procedure is required, it is often useful to repair the aortic

root with one vascular graft and then to use a separate graft to create the distal aortic anastomosis. The two grafts are then measured, cut, and anastomosed to provide the correct length and orientation for aortic replacement.

If the ascending aorta cannot be cross-clamped, the patient is cooled to 20°C with subsequent circulatory arrest. The distal aortic reconstruction is performed first in this circumstance, at which time the graft is cannulated and proximally clamped with resumption of cardiopulmonary bypass and systemic rewarming. Cannulation of the graft for antegrade systemic perfusion and rewarming is associated with improved neurologic outcome, when compared with retrograde perfusion, and should be performed whenever possible. Newly available vascular grafts include 7 to 8 mm Dacron side-arm grafts for easy cannulation to facilitate this technique. Because a cross-clamp is not applied, the left ventricle must be decompressed once fibrillation starts during systemic cooling (~20°C) to prevent distension and irreversible myocardial injury. Proximal ascending aortic repair is completed during the period of rewarming.

An alternative to the open distal technique is possible when dissection is limited to the ascending aorta or to the proximal arch away from the origin of the brachiocephalic vessels. Arterial perfusion is achieved antegrade, through distal arch cannulation, or retrograde, via cannulation of a femoral artery. An aortic cross-clamp is applied tangentially just proximal to the innominate artery. The ascending aorta is resected to include the inferior aspect of the arch. The layers of the dissected aorta proximal to the clamp are then reunited if necessary, and the aorta is replaced with an appropriately sized, beveled vascular graft. The proximal reconstruction and anastomosis may then be performed without the need for deep hypothermia and circulatory arrest.

When aortic root dissection fails to violate the intima of the coronary artery, repair of the ascending aorta at the sinotubular junction is often sufficient to reunite the aortic root layers and provide unimpeded coronary blood flow. Minimal disruption of the coronary ostial intima may be repaired primarily with 5-0 Prolene suture. If, however, the ostium is circumferentially dissected and an aortic root replacement is necessary, an aortic button should be excised and the layers reunited with either running 5-0 Prolene suture or glue (Figure 36-9). The button may then be reimplanted into a vascular graft or to a separate 8 mm vascular graft as part of a Cabral-type repair. Aortocoronary bypass grafting is performed only when the coronary ostium is not reconstructible and as a last resort.

Aortic regurgitation is present in up to 75% of type A dissections and is often caused by the loss of commissural support of the valve leaflets. Resuspension of the valve commissures using pledgeted 4-0 Prolene sutures passed through the commissure and the reconstructed aortic wall is the simplest and most effective surgical solution (Figure 36-10). The valve commissures may also be resuspended

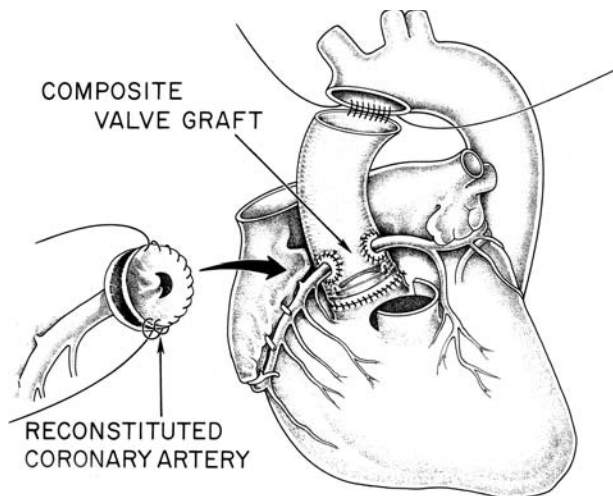


FIGURE 36-9. If the aortic valve cannot be preserved, an aortic root replacement is performed using a composite valve graft. The coronary arteries are excised and the dissected layers repaired with Teflon felt and/or glue as described. The coronary buttons are then reimplanted into a valved conduit as part of a Bentall procedure.

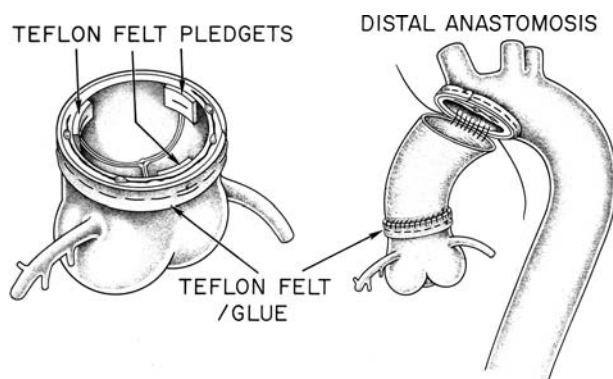


FIGURE 36-10. Reconstruction of the dissected layers of the aortic root is performed using Teflon felt and/or glue. Resuspension of the aortic valve commissures using pledgeted Prolene sutures is often sufficient to achieve aortic valve competence when the intima of the aortic root is intact. The distal ascending aorta is similarly reconstructed and the intervening aorta is replaced with a vascular graft.

into a vascular graft to spare a normal aortic valve in the setting of annuloaortic ectasia.²² Other surgical techniques exist to recreate the sinuses of Valsalva, which may be more important than previously recognized. If the aortic valve cannot be spared, replacement of the ascending aorta and valve should be performed using a composite valve graft or homograft. The composite valve graft is implanted using horizontal mattress 2–0 Tycron sutures to encircle the annulus and to seat the valved conduit. The previously excised and reconstituted coronary buttons are reimplanted into the vascular graft with running 5–0 Prolene suture. The left button is implanted first, at which time the graft is clamped and placed under pressure to define the proper orientation and position of the right

coronary button. The aortic homograft is similarly implanted using horizontal mattress 2–0 Tycron sutures except that a generous margin of aortic root below the coronary buttons is retained for a second hemostatic suture line of running 4–0 Prolene.

Chronic type A dissection, with or without aneurysmal enlargement, is treated by using many of the same operative techniques described for the acute setting. Treatment of the distal anastomosis is controversial because some surgeons advocate obliteration of flow into the false lumen, whereas other surgeons purposely maintain flow into the true lumen and false lumen through distal resection of the intimal flap. Those surgeons who reunite the chronically dissected aortic layers to perfuse only the true lumen maintain that the false lumen remains perfused through distal reentry tears in more than 50% of patients. Alternatively, there is a theoretical and possibly real concern that important side branches arise exclusively from the false lumen and perfusion may be interrupted with this technique. The practice at the University of Virginia is to resect the distal chronic dissection flap to obviate such concerns. The distal anastomosis is made to the outer wall of the aorta, which has nearly the structural integrity of normal aorta. The rate of aortic valve replacement for aortic regurgitation is much higher in patients with chronic dissection. Morphologic changes in the valvular apparatus appear to occur, which renders the valve irreparable in as many as 50% of cases. Two options exist for aortic valve replacement and repair of the ascending aorta: aortic valve replacement with separate ascending aortic replacement or composite valve replacement. We favor the composite valve graft replacement to remove all potentially abnormal aortic tissue from the root and the potential for aneurysmal dilatation or redissection.

The right lateral decubitus position is optimal for surgical treatment of acute type B dissections requiring operation. The pelvis is canted posteriorly to allow access to both sets of femoral vessels. A posterolateral thoracotomy in the fourth intercostal space provides sufficient access to the aorta; notching the fifth and sixth ribs posteriorly permits visualization of the entire thoracic aorta distally. A thoracoabdominal incision may be required to access the abdominal aorta and is performed either through the abdomen or the retroperitoneum. The left hemidiaphragm is carefully divided in a radial fashion while marking adjacent sites on each side of the division with metal clips. This provides necessary exposure and facilitates subsequent diaphragm approximation.

The operation most frequently performed for acute type B dissection is replacement of the proximal third of the descending thoracic aorta. This includes the site of the primary tear in the majority of cases. After gaining access to the thoracic aorta, the mediastinum between the left subclavian and the left common carotid arteries is dissected and the left subclavian artery encircled with an umbilical tape and Rommel tourniquet. It is essential that

the left vagus and recurrent laryngeal nerves are identified and preserved during the course of the dissection. Ultimately, the entire distal arch must be free enough to place an aortic clamp between the left common carotid and the left subclavian arteries. Next, the proximal descending thoracic aorta is circumferentially mobilized, dividing intercostal and bronchial arteries in the segment to be excised. The left inferior pulmonary vein is then dissected and a 4–0 Prolene purse-string suture placed posteriorly. Following the administration of 100 U/kg of intravenous heparin, 14 French cannulae are inserted into the left inferior pulmonary vein and either the descending thoracic aorta or either femoral artery (Figure 36-11). Partial left-heart bypass is then initiated, with flow rates between 1 and 2 L/min. The left subclavian artery is controlled and vascular clamps are placed on the aorta proximally and distally on the midthoracic aorta. Right radial artery pressure is measured to maintain proximal aortic systolic pressure between 100 and 140 mm Hg and mean femoral artery pressure > 60 mm Hg.²³ The aorta is then opened longitudinally and backbleeding intercostal arteries are oversewn. Proximally, the aorta is transected distal to the origin of the left subclavian artery. The size of the vascular graft is based on the diameter of the distal aorta and beveled proximally. The proximal anastomosis is made to undissected aorta using 3–0 Prolene suture and Teflon felt strips if necessary. This anastomosis may include the origin of the left subclavian to deal with dissection in this vessel. A separate 6 to 8 mm Dacron graft can be used if

there is intimal disruption involving the proximal segment of the left subclavian artery. Once the proximal anastomosis is complete, the proximal clamp is released and repositioned on the vascular graft to inspect the anastomosis. Attention is then turned to repairing the distal aorta with Teflon felt or glue for distal anastomosis. The distal anastomosis is completed, the clamps are released, and partial left-heart bypass is terminated. Decannulation and closure are routine. Percutaneously placed femoral artery cannulae that are 15 French or smaller may be removed without direct repair. When cannulae larger than 15 French are required, surgical repair is indicated.

An alternative surgical strategy for acute type B dissection involving the abdominal aorta requires the use of total cardiopulmonary bypass and deep hypothermic circulatory arrest to prevent potential spinal cord and intra-abdominal ischemia.²⁴ After creation of a thoracoabdominal incision, the thoracic and abdominal aorta is exposed from the left subclavian artery to the aortic bifurcation. The femoral artery and vein are cannulated for cardiopulmonary bypass with systemic cooling and circulatory arrest. Cardiopulmonary bypass is then interrupted and the aorta opened proximally. The arch is repaired if necessary with Teflon felt or glue and the proximal anastomosis created. The graft is clamped distal to the anastomosis and then cannulated for proximal perfusion with resumption of cardiopulmonary bypass. Intercostal arteries to the upper third of the thoracic aorta are divided; larger vessels below T9 are reimplanted into the back of the graft with 4–0 Prolene suture. As vessels are reimplanted, the proximal clamp is moved distally to maintain spinal cord perfusion. Abdominal aortic branch vessels are divided from the wall of the aorta with a 5 mm cuff for reimplantation. Usually the right renal, superior mesenteric, and celiac arteries, as well as several intercostal and lumbar arteries, are removed as a patch and reimplanted into the graft. The left renal artery often originates from a dissected segment of the aorta and is reimplanted after repair. The inferior mesenteric artery and lumbar vessels that bleed are controlled by suture ligation. If the intima of abdominal aortic branch vessels is involved, then repair is carried out with 5–0 Prolene suture. Once all side branches are secured, the distal anastomosis to the aortic bifurcation is performed, reuniting, if necessary, the aortic layers distally. Rewarming should commence as the final aortic branch vessels are being sewn in place.

Two potential situations during repair of acute type B dissection deserve mention. Rupture of the thoracic aorta prior to or during repair often leads to operative death. Successful management requires immediate cannulation of the femoral artery and vein for cardiopulmonary bypass and eventual circulatory arrest, but only if the ruptured area can be locally controlled. While assisted venous drainage through the femoral vein is often adequate, direct cannulation of the right ventricle may also be performed.

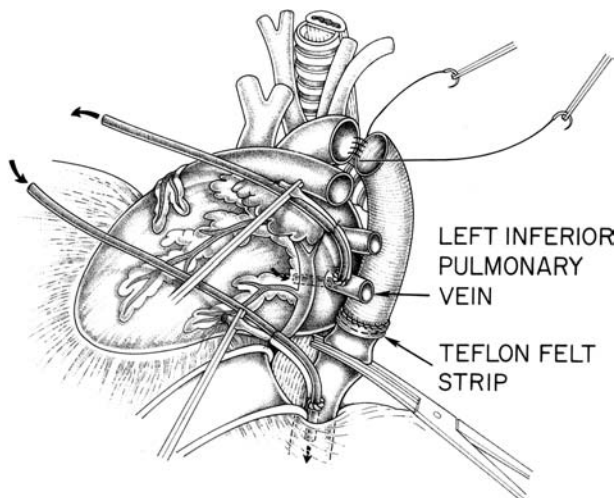


FIGURE 36-11. Partial left-heart bypass is used to repair type B dissections. Cannulation of the normal descending thoracic aorta is possible for limited dissections or aneurysms, but femoral arterial cannulation is sometimes necessary. In the acute setting, repair of the proximal and distal dissected layers with Teflon felt and/or glue and replacement of the proximal third of the descending thoracic aorta are often adequate. Chronic dissections require resection of the intimal flap to maintain perfusion of both lumen and to prevent malperfusion. The chronically thickened outer aortic wall is used in such cases for anastomosis to a vascular graft.

Unexpected dissection of the arch extending proximal to the aortic clamp may also require circulatory arrest and performance of an open proximal anastomosis for repair. The techniques for repair of the arch using Teflon felt or glue are identical to those discussed earlier. It is preferable to avoid this situation through preoperative recognition of arch involvement by using high-quality imaging.

Spinal cord ischemia resulting in paraplegia or paraparesis is a catastrophic complication of dissection repair that may now be partially preventable. Spinal cord ischemia occurs in up to 19% of patients following repair of acute dissection; the incidence is much lower, 2.9%, when the indication is aneurysmal disease resulting from chronic dissection.¹⁹ Various strategies to prevent spinal cord ischemia during repair of chronic dissection secondary to aneurysmal dilatation have been devised. Use of partial left-heart bypass appears sufficient for patients with aneurysmal dilatation of the thoracic aorta above the level of T9 and results in paraplegia in fewer than 5% of those patients.²⁵ Thoracoabdominal aneurysms require additional measures and have a paraplegia rate as high as 10% following surgery. Preoperative identification of the anterior spinal artery origin to preserve its perfusion is one such measure, but its efficacy is controversial. Reimplanting intercostal and lumbar arteries between T9 and L1 while moving the clamp distally has reported benefit in several series.²⁶ Other methods used for spinal cord protection include measurement of sensory- and motor-evoked potentials, spinal cord cooling, cerebrospinal fluid drainage, and the use of a variety of pharmacologic agents for cellular protection. We presently use left-atrial-to-femoral-artery bypass and reimplant key intercostal arteries.

Malperfusion Syndrome

Malperfusion of aortic branch vessels may occur from the coronary ostia to the aortic bifurcation and may dominate the presentation of certain patients. Although autopsy series yield a greater percentage of patients with evidence of malperfusion, clinical series reveal that dissection is often complicated by malperfusion of at least one organ system (Table 36-6).^{13,27} Compression of the true lumen by the false lumen is the mechanism by which aortic branch vessels are occluded in the majority of cases. Branch vessels may also be completely sheared off the true

lumen and perfused to various degrees by the false lumen. Malperfusion is treated in a variety of ways, from primary surgical repair of the dissection to catheter-based or open fenestration.

Percutaneous fenestration and stenting are relatively new adjuncts to the surgical management of malperfusion syndromes. Renewed interest in these procedures grew from the recognition that hospital mortality of the various malperfusion syndromes was as high as 60%.²⁸ Surgical fenestration to treat malperfusion, however, can reduce the mortality to under 20%.^{29,30} Indications for percutaneous fenestration and endovascular stent placement were developed to treat malperfusion syndrome in the hope of improving outcome even further. Direct stenting of obstructed branch vessels and percutaneous fenestration with or without placement of a stent in the true lumen are the procedures most commonly performed. In certain situations, stents may be placed across an existing distal reentry tear to maintain patency and perfusion of the true lumen and the branch vessels. Balloon fenestration may be required to create such a communication between the true lumen and false lumen or to prevent thrombosis of the false lumen from which branch vessels may originate. Early results indicate that this procedure is both safe and effective, with restoration of flow in up to 90% of patients with an average 10 to 25% 30-day mortality.^{31,32} Given that the majority of post-surgical mortality in this group relates to the duration of concomitant malperfusion in patients with acute dissection, one group advocates percutaneous reperfusion followed by surgical repair.³³ Percutaneous treatment of malperfusion, however, is performed subsequent to surgical repair of the dissection in most reports.

The techniques used for surgical treatment of malperfusion are dependent upon location but are quite similar. Malperfusion of the brachiocephalic vessels is repaired acutely by reuniting the dissected aortic layers proximally and reperfusing the true lumen when the intima is intact. If the intima is violated or the dissection extends into the left common carotid or innominate arteries, the vessel should be resected from the arch, the layers reunited, and the vessel reimplanted into the arch, perhaps with an interposition graft if necessary. Extra-anatomic bypass to the carotid arteries is an option in unreconstructable cases. Chronic brachiocephalic vessel malperfusion is usually treated with resection of the dissection flap in the arch. Infrequently, the chronic dissection flap extends into the branch vessels and may present as transient ischemic attacks or stroke. In such cases, it is often necessary to resect the dissection flap into the branch vessel or to reunite the layers distally prior to reimplantation.

Malperfusion of the intra-abdominal viscera may be apparent at presentation but may also complicate repair of either type A or B dissections. Proximal repair of the dissection is standard treatment, but if this fails, or if malperfusion persists despite repair, an additional procedure is

TABLE 36-6. Frequency and Location of Malperfusion in Acute Type A and B Thoracic Aortic Dissection

| Vascular System | Frequency (%) |
|-------------------------------|---------------|
| Renal | 23–75 |
| Extremities (upper and lower) | 25–60 |
| Mesenteric | 10–20 |
| Coronary | 5–11 |
| Cerebral | 3–13 |
| Spinal | 2–9 |

necessary. The options are either open surgical or percutaneous fenestration of the dissection flap. The percutaneous procedure is performed by pulling an inflated balloon through the dissection flap or by using a fenestration knife. A surgical fenestration procedure is performed through a midline laparotomy with exposure of the infrarenal aorta. Proximal and distal control of the aorta is obtained and a transverse aortotomy created. The dissection flap is resected proximally as far as possible but at least to the level of the renal arteries. The distal dissection flap is reunited with the aortic wall by using Teflon felt or glue, and the aorta closed primarily or by using a short interposition vascular graft. Occasionally, fenestration of intra-abdominal aortic branch vessels is required if the intima is violated beyond the ostia. If the dissection flap cannot be completely excised, the distal vessel layers must be reunited. Consideration should be given to closure of these smaller vessels with patch angioplasty to prevent narrowing. In the event that perfusion is not re-established, extra-anatomic bypass may be required.

Obstruction of the terminal aorta or malperfusion of the lower extremities following operative repair is best treated with percutaneous or open fenestration. The most appropriate surgical option if that fails is femoral–femoral bypass grafting in the setting of unilateral malperfusion, or axillofemoral and femoral–femoral bypass grafting if bilateral lower extremity malperfusion exists.

Postoperative Management

Invasive hemodynamic monitoring is used to ensure adequate end-organ perfusion while maintaining systolic blood pressure between 90 and 110 mm Hg. Early on, blood pressure control is achieved by using narcotics and sedative/hypnotic agents. The patient is allowed to emerge from general anesthesia briefly for a gross neurologic examination. The patient is then sedated for a period to ensure continued hemodynamic stability and to eliminate concerns over bleeding. Coagulopathy is aggressively treated with antifibrinolytic agents and blood products as necessary, and by warming the patient. Hematocrit, platelet count, coagulation studies, and serum electrolytes are obtained and corrected as necessary. An ECG and chest radiograph are obtained to assess for abnormalities and to serve as baseline studies. A full physical exam, including a complete peripheral vascular exam, is performed upon arrival in the intensive care unit. Despite adequate repair of the dissection, perfusion of the false lumen may persist and might cause a malperfusion syndrome. If an abdominal malperfusion syndrome is suspected postoperatively, this should be aggressively evaluated with ultrasound and subsequent angiography if positive. A strong clinical suspicion is enough to warrant this evaluation given the consequences of failed recognition. In the morning, if the patient has been hemodynamically stable without excessive bleeding and has a normal neurologic exam, the

patient may be extubated. Management is routine from that point forward.

Long-Term Management

Surviving the operation for acute dissection represents the beginning of a lifelong requirement for meticulous medical management and continued close observation. It has been estimated that replacement of the ascending aorta for type A dissection obliterates flow in the distal false lumen in fewer than 10% of patients. As a result, the natural history of repaired dissection may involve dilatation and potential rupture of the chronically dissected segment of aorta. This occurred and was the reason for late death in nearly 30% of DeBakey's original series in 1982, and is currently the leading cause of late death following surgical repair.³⁴ Often a multidrug antihypertensive regimen that includes β -blocking agents is required to maintain systolic blood pressure below 120 mm Hg. There are some data indicating that blood pressure control within a narrow range may alter the natural history of chronic dissection by diminishing the rate of aneurysmal dilatation. Follow-up diagnostic imaging is required to monitor aortic size in such patients. The current recommendations for follow-up diagnostic imaging are to obtain a baseline study prior to discharge and at a 6-month interval during the first year. If the aorta remains unchanged, this interval is then prolonged to 1 year. If the aorta is enlarging at a rate greater than 0.5 cm per 6 months or becoming more eccentric on comparison of three-dimensional reconstruction images, then the interval is decreased to 3 months, if surgery is not indicated.

Chronic aortic dissection develops in patients who fail to undergo immediate surgical treatment of type A dissection and in those who are successfully treated medically for type B dissection. The natural history of acute dissection rarely involves spontaneous healing with absence of flow in the false lumen and aortic wall thickening. This phenomenon is observed in 4 to 31% of medically treated patients. As complete thrombosis of the false lumen appears necessary for healing, patients with distal communication of the false lumen may go on to develop aneurysmal dilatation of the aorta. The natural history of this process has been examined and reveals that there is an annual rate of expansion of 2 to 3 mm/year in communicating dissections, and the rate is 1 mm/year in those not communicating. Infrequently, chronic dissection may cause pain and result in paralysis/paraplegia from loss of important intercostal arteries or even distal embolization of thrombus or atheroma from the false lumen. The operative indications for chronic dissection include symptoms, rupture, malperfusion, and aneurysm size.

Spiral CT arteriogram and MRI are the imaging studies of choice to use when following patients with repaired or chronic aortic dissection. MRI and ultrasonography

are useful to use with patients with renal insufficiency and for those patients requiring only imaging of the abdominal aorta. Echocardiography is useful for imaging the ascending aorta and provides additional information regarding the aortic valve. It is important to recognize the resolution limitations of each imaging modality and the inherent imprecision of comparing different imaging modalities to evaluate changes. In general, measurements should be made at the same anatomic level with respect to reproducible anatomic structures (ie, the sinotubular ridge, proximal to the innominate or left subclavian arteries or at the diaphragmatic hiatus). It is important to recognize that the false lumen should be included in measurements of aortic diameter whether it is perfused or not. Three-dimensional reconstruction of spiral CT and MRI scans minimize the error introduced by aortic eccentricity when comparing imaging studies and has simplified following this patient population.

The size criteria indicating operative intervention for thoracic aortic aneurysms were recently reviewed by the Yale group and include patients with chronic aortic dissection. These criteria suggest that replacement should be performed for ascending aortic size greater than 5.5 cm, or greater than 5 cm if a connective tissue disorder is present. In the descending thoracic aorta, replacement is indicated at 6.5 cm, or at 6 cm if there is a family history or physical stigmata of a connective tissue disorder.³⁵ Eccentricity of the aorta was also predictive of rupture as was rapid expansion and continued smoking. Such factors should be considered when deciding whether to operate based on aneurysm size.

Despite appropriate medical management and close follow-up, nearly 23% of patients with chronic dissection require operation for aneurysmal dilatation at 10 years. Reoperation is necessary in 10% at 5 years and in up to 40% at 10 years following type A dissection repair. This number is even higher in patients with Marfan's disease. Nearly 20% of patients require reoperation following valve preservation at the initial operation secondary to progressive aortic regurgitation. These operations carry a higher mortality and morbidity, which is made worse in the emergency setting and when the ascending aorta or arch is involved.

Results

The operative mortality for repair of acute aortic dissection has steadily fallen over the years with development of better vascular graft material, more effective hemostatic agents and improvements in the safety of cardiopulmonary bypass. In the last decade, most centers report an operative mortality for acute type A dissection of between 14 and 27.5%.³⁶⁻³⁸ The majority of deaths occur as a result of stroke, myocardial ischemia/heart failure, and malperfusion. The operative mortality of patients suffering acute type B dissection (28 to 65%) is higher than for type A

dissection because the indications for surgery are failure of medical management or complications of dissection, as previously discussed.³⁹ The most recent data from a multicenter international registry, however, reveal that such a disparity in operative mortality between acute type A and B dissections may be disappearing. The mortality in that study was 27% acute type A and 29% acute type B dissection ($p = \text{NS}$).¹⁶

The published results for long-term survival for acute type A dissection surgically treated over the last decade are roughly 55 to 75% at 5 years and between 32 and 65% at 10 years.^{40,41} Following operative repair of acute type B dissection, the 5-year survival averages 48%, with 29% alive at 10 years.⁴¹

The operative mortality for chronic type A dissection is between 4 and 17% and on average is very similar to that reported for chronic type B repair at 10 to 15%.^{40,42} The actuarial survival following operation for chronic type A and B dissections is not different at 5 years (59 to 75%) or at 10 years (45%).⁴¹

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