Acute aortic dissection is a relatively uncommon but highly lethal condition. Without proper treatment, devastating consequences can occur due to aortic rupture, cardiac tamponade, or irreversible ischemia involving the spinal cord or the visceral organs. The treatment strategy of this condition is in part influenced by the location and the severity of aortic dissection as immediate surgical intervention is necessary in acute ascending aortic dissection, whereas medical therapy is the initial treatment approach in uncomplicated descending aortic dissection. Recent advances of endovascular technology have broadened the potential application of this catheter-based therapy in aortic pathologies, including descending thoracic aortic dissection. In this article, the etiology, pathogenesis, and classification of this condition are discussed. The diagnostic benefits of various imaging modalities for descending aortic dissection are also discussed. Current treatment strategies, including medical, surgical, and catheter-based interventions, are reviewed. Lastly, clinical experiences of endovascular treatment for descending aortic dissection and various endovascular devices potentially applicable for this condition are discussed.

Keywords: aortic dissection; endovascular thoracic aortic aneurysm repair; endograft; malperfusion; false lumen; endovascular aortic aneurysm
instances, endovascular treatment has gained greater preferences by both physicians and patients when compared with open surgical repair. Physicians who provide cardiovascular health care must possess a thorough understanding regarding the available treatment strategies, including both open and endovascular modalities, of aortic dissection. This article discusses the etiology, pathophysiology, classification, diagnosis, and treatment strategies of this condition.

Etiology

Aortic dissection usually presents in the older population, with a male predominance, as men are affected approximately 2 to 5 times as often as women.7,9-14 The peak incidence of aortic dissection occurs in the sixth and seventh decades of life.13 A variety of congenital and acquired conditions can predispose a person to aortic dissection (Table 1). Approximately 90% of patients with aortic dissection have either poorly controlled hypertension or a history of chronic hypertension, which is the most common predisposing factor for aortic dissection.11,13 In the International Registry of Acute Aortic Dissection (IRAD) study, a history of systemic hypertension was noted in 72% of patients with aortic dissection.11 Hypertension causes excessive mechanical and metabolic strain on the media, which opposes arterial pressure within the aortic wall. Atherosclerosis is an associated risk factor in older patients with aortic dissection. These patients typically develop an aortic dissection secondary to an atherosclerotic penetrating ulcer, and they also frequently have underlying hypertension. The initial tear in the aorta commonly occurs in the descending thoracic aorta within a few centimeters of the left subclavian artery; propagation of the dissection can result in multiple entry and reentry between the true and false channels within the aortic wall.15-18

In young patients, aortic dissection can be caused by a variety of congenital disorders affecting the aortic elastic tissues. These include Marfan, Loeys-Dietz, Turner, Noonan, and Ehlers-Danlos syndromes. Marfan syndrome is a congenital disorder with autosomal dominant transmission. Patients typically present with characteristic cardiovascular, musculoskeletal, and ocular abnormalities. A fundamental genetic derangement is known in the fibrillin-1 gene, which results in defective production of fibrillin in the extracellular matrix of several organ systems.19-21 Recent data suggest that the mechanism by which the abnormal fibrillin causes aortic degeneration through its role on transforming growth factor-β (TGF-β) regulation.22,23 Transforming growth factor-β also plays a central role in the vascular manifestations of Loeys-Dietz syndrome, which is caused by mutations involving TGF-β receptors and is associated with severe dilatation, tortuosity, and dissection involving the aorta and its branch vessels.24,25 Ehlers-Danlos syndrome is another inherited disorder with connective tissue abnormality, which is characterized by hypermobility of joints and hyperelasticity of the skin. As a result of the structural connective tissue defect, this condition can trigger an inflammatory response, which destroys the medial layers of the aortic wall with resultant aortic dissection.

Adult patients may develop aortic dissection due to various acquired conditions including iatrogenic retrograde dissection from catheter-related aortic intimal injury and previous valvular or coronary

<table>
<thead>
<tr>
<th>Table 1. Predisposing Factors for Aortic Dissection</th>
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<td>Adulthood-related conditions</td>
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<tr>
<td>Chronic hypertension</td>
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<td>Cigarette smoking</td>
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<td>Dyslipidemia</td>
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<td>Cocaine/crack abuse</td>
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<td>Hereditary conditions</td>
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<td>Bicuspid aortic valve</td>
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<td>Coarctation</td>
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<td>Turner’s syndrome</td>
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<td>Connective tissue disorders</td>
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<td>Marfan syndrome</td>
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<td>Loeys-Dietz syndrome</td>
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<td>Ehlers-Danlos syndrome</td>
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<tr>
<td>Hereditary fibrillinopathies</td>
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<tr>
<td>Vascular inflammatory conditions</td>
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<td>Giant cell arteritis</td>
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<td>Takayasu arteritis</td>
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<td>Behcet’s disease</td>
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<td>Syphilis</td>
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<td>Ormond’s disease</td>
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<tr>
<td>Traumatic conditions</td>
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<tr>
<td>Blunt deceleration thoracic injury</td>
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<td>Iatrogenic catheter-based aortic injury/instrumentation</td>
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<tr>
<td>Operative factors</td>
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<tr>
<td>Aortic valvular surgery</td>
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<tr>
<td>Aortic clamping placement/aortotomy</td>
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<td>Aortic graft anastomosis</td>
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<tr>
<td>Aortic patch aortoplasty</td>
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<td>Aortic cannulation for cardiopulmonary bypass</td>
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bypass or aortic surgery. Cocaine use has been postulated as a predisposing factor for aortic dissection due in part to the sudden increase in aortic wall stress caused by catecholamine surge. Weight-lifting has also been linked with acute aortic dissection, although it primarily involves the ascending aorta. Lastly, blunt trauma occasionally causes aortic dissection due to an intimal rupture and subsequent dissecting hematoma. It should be noted, however, that most blunt aortic injuries remain localized and do not result in dissection; true traumatic aortic dissection, with lengthwise separation of the media extending distally along the aorta, is very rare.

Another factor that has been associated with aortic dissection is pregnancy, with dissection and aortic rupture commonly occurring in these patients during the third trimester or the first stage of labor. Aortic dissection in pregnant patients has been attributed to a variety of factors, including hypercirculation during the late gestational period, hypertension, and the loosened connective tissue owing to hormonal changes. Recent literature, however, has demonstrated that the dissection is extremely rare during pregnancy and has suggested that the commonly regarded association between these events may be an artifact of selective reporting.

In an analysis of the IRAD data, Nienaber et al found that only 2 of 346 women (0.6%) in the registry were pregnant at the time of their acute dissection. Although pregnancy has been described as being responsible for nearly half of aortic dissections in women below the age of 40 years, the prevalence of pregnancy in this subgroup of IRAD patients was only 12.5% (2 of 16).

Pathogenesis

The predominate pathologic mechanism of an aortic dissection begins with a tear in the intimal layer of the aorta, through which blood penetrates and subsequently dissects distally into the plane of the medial layer with eventual hemorrhage into the underlying diseased aortic media, thereby creating a blood-filled channel in the aortic wall. This process is primarily responsible for the creation of true and false lumen separated by the intimal flap.

Spontaneous hypertension-induced rupture of the vasa vasorum of the aorta may cause intramural hematoma (IMH) with subsequent weakening of the medial layer, which eventually leads to an intimal tear. A variety of mechanical forces have been reported, which can contribute to aortic dissection, and they include flexion forces of the vessel at fixed sites, the radial effect of the pressure pulse, and the shear stress of the blood. Persistent high blood pressure can be a compounding mechanical strain on the aortic wall as it creates a shearing force exerting a longitudinal stress along the aortic wall. A combination of these mechanical factors can result in the propagation of the intimal tear in the aortic media, particularly in patients with medial degeneration or weakening of the aortic wall due to underlying atherosclerosis.

Because an aortic dissection predominately originates from an intimal tear, it is postulated that an intimal tear typically occurs in areas subjected to the greatest change of blood pressure pulsatility or rate of ventricular or aortic pressure change (dP/dT). Blood pressure and the rate of acceleration of the pulsatile flow are critical factors in the propagation of an aortic dissection. Intraluminal pressures serve as a driving force, which propagates a dissection, and this can occur either in an antegrade or retrograde fashion. Due in part to the intraluminal pressure differences between the true and false aortic channels, the false lumen may compress or collapse the true lumen because of higher intraluminal pressure. The dissection space may remain patent as a false lumen, become obliterated by thrombosis, or rupture into potential cavities, such as the pericardial, pleural, or peritoneal space. The typical pattern of a descending thoracic aortic dissection plane involves the left posterolateral portion of the aorta. Certain visceral vessels including the celiac, superior mesenteric, and right renal arteries usually arise from the true lumen, whereas the left renal artery emanates from the false lumen, but various patterns of visceral vessel origin have been reported.

Propagation of the dissection plane with pressurization of the false aortic lumen, either in an antegrade or retrograde fashion, can lead to compromised flow of side branch vessels, resulting in cerebral, coronary, mesenteric, renal, spinal, or limb ischemia.

Classification

Aortic dissections can be classified based on the duration from the onset of symptoms to medical evaluation or anatomic location regarding the extent of aortic involvement. Aortic dissection is considered acute when the onset of symptoms is less than
2 weeks in duration at the time of diagnosis, whereas chronic dissection is defined by the onset of symptoms greater than 2 weeks in duration. A majority of patients are diagnosed with acute aortic dissection when they first seek medical evaluation, whereas only one third have chronic dissection when a diagnosis is made. The distinction between the acute and the chronic aortic dissection is of therapeutic importance because acute dissection is associated with a significantly greater risk of life-threatening complications. Studies have shown that approximately 75% of aortic dissection-related deaths occur in the first 2 weeks from symptom onset when appropriate treatment is not given in a timely fashion.

From an anatomical perspective, it is estimated that 60% of dissections originate in the ascending aorta, 10% in the aortic arch, and 30% in the descending thoracic aorta. The 2 most common locations for intimal tear include the regions just above the aortic valve and just distal to the left subclavian artery. The classification scheme proposed by DeBakey et al in 1964 remains the most widely used and specifically delineates the anatomical extent of aortic dissection (Figure 1). This classification categorizes dissection into 3 types. Briefly, they include (1) type I dissection involving the ascending aorta, aortic arch, and descending aorta, (2) type II dissection involving the ascending aorta, and (3) type III dissection involving the descending aorta distal to the left subclavian artery. Additionally, type III dissection is further divided to either type IIIa, which refers to dissections that originate distal to the left subclavian artery but extend distally above the diaphragm or type IIIb, which refers to dissections that originate distal to the left subclavian artery and extend distally below the diaphragm.

Daily et al proposed a different classification scheme, which simplifies aortic dissection into Stanford type A and B. Stanford type A dissection originates in the ascending aorta, typically within a few centimeters of the aortic valve and may extend around the aortic arch and into the descending aorta. Type A dissection includes any dissection involving the ascending aorta, which is equivalent to
Table 2. The Classification Systems of Aortic Dissection

<table>
<thead>
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<th>Stanford classification</th>
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<tr>
<td>Type A: dissection involving ascending aorta, may extend distally</td>
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<tr>
<td>Type B: dissection involving only the descending aorta</td>
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<thead>
<tr>
<th>DeBakey classification</th>
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<tr>
<td>Type I: dissection of the ascending and descending aorta</td>
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<tr>
<td>Type II: dissection of the ascending aorta only</td>
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<tr>
<td>Type IIIa: dissection of the descending aorta only</td>
</tr>
<tr>
<td>Type IIIb: dissection of the descending thoracic and abdominal aorta</td>
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<th>European Society of Cardiology classification</th>
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<tr>
<td>Class 1: classic aortic dissection with intimal flap</td>
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<tr>
<td>Class 2: intramural hematoma/hemorrhage</td>
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<tr>
<td>Class 3: discrete dissection without hematoma</td>
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<tr>
<td>Class 4: penetrating atherosclerotic ulcer</td>
</tr>
<tr>
<td>Class 5: iatrogenic or traumatic dissection</td>
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Can further divide into communicating and noncommunicating dissection. Communicating dissection has an intimal tear with unidirectional or multidirectional flow between the true and false lumen. In contrast, in a noncommunicating aortic dissection, no flow and no intimal tear can be detected.

- Class 2: IMH/hemorrhage. This may be the result of ruptured vasa vasorum and may be the initial lesion in cases of severe medial degeneration. It often coexists with or progresses to class 1 dissection.
- Class 3: Subtle-discrete aortic dissection. This is characterized by a stellate or linear intimal tear with the exposure of the underlying media and adventitia but without progression to separation of medial layers. This type of dissection was first described by Svensson et al. who reported the presence of this rare type of dissection in 9 patients with suspected aortic dissection. Because multiple diagnostic tests, including transesophageal echocardiography (TEE), computed tomography (CT), and magnetic resonance imaging (MRI) all failed to make the diagnosis, the authors recommended that aortography should be performed if dissection is still a highly likely diagnosis, but other imaging modalities were negative. A typical angiographic appearance of a class 3 dissection is an eccentric bulge on the lateral border of the aorta (Figure 2).
- Class 4: Plaque rupture and ulceration. This aortic lesion can lead to aortic dissection or aortic rupture. The natural history and progression of aortic ulcer and the treatment remain a subject of debate. In patients with symptomatic aortic dissection, this may lead to a similar clinical presentation to other causes of acute chest pain. Frequently, this lesion is an incidental diagnosis in asymptomatic patients with chronic aortic dissection (Figure 3).
- Class 5: Traumatic and iatrogenic aortic dissection. This typically occurs due to intimal disruption at the level of the aortic isthmus. On the other hand, catheter-related iatrogenic dissection may be seen after aortic angioplasty for aortic coarctation, coronary angiography, and other catheter-based procedures. Operative iatrogenic injury following aortic cross-clamp placement has also been linked to the development of aortic dissection.

**Diagnostic Evaluation**

Commonly used imaging modalities to establish a diagnosis of aortic dissection include CT, MRI, TEE, or transthoracic echocardiography (TTE). Aortography is an invasive diagnostic modality with
limited clinical utility, particularly in critically ill or unstable patients. Nonetheless, it remains the diagnostic study of choice in diagnosing class 3 dissection based on the new European Society of Cardiology classification. Clearly, each diagnostic test has its own unique advantages and limitations. When determining the diagnostic study for aortic dissection, physicians must consider the overall context of the patient's clinical condition, degree of diagnostic invasiveness, and diagnostic cost-effectiveness.

In a decision analysis study analyzing the risks of performing one or more diagnostic procedures in patients suspected of having aortic dissection, Sarasmin et al assessed the test’s accuracy, the risks and benefits of treatment, and time-dependent mortality rate in patients with aortic dissection who did not receive treatment. The authors noted that in patients with a high clinical suspicion of aortic dissection, a delay in appropriate imaging studies affected mortality. For instance, a TEE performed within 6 hours or CT scan obtained within 2 hours yielded greater survival than MRI obtained 9 hours following the onset of symptoms. The challenge in making prompt diagnosis of aortic dissection using these various imaging modalities was highlighted by the Task Force on Aortic Dissection study of the European Society of Cardiology. The study reported that most centers use an average of 1.8 diagnostic methods to diagnose aortic dissection, revealing the uncertainty of this potentially life-threatening disease.

Chest Radiography

Routine chest radiography is neither sensitive nor specific for the diagnosis of aortic dissection. Several nonspecific radiographic signs suggestive of aortic dissection include mediastinal widening and tortuous aortic contour, displaced calcification, and opacification of the aorticopulmonary window. Von Kodolitsch et al evaluated the utility of chest radiography in 216 patients suspected of having acute aortic dissection during a 6-year period and reported a sensitivity of 67% for overt aortic dissection and of 63% for IMH or penetrating ulcer. Additionally, the sensitivity for proximal aorta was 47% and for distal aortic dissection was 77%. These findings underscored the low diagnostic value of chest radiography in patients with acute aortic dissection.

Aortography

Contrast aortography was once considered the standard diagnostic study of choice for aortic dissection.
Accurate diagnosis based on aortography requires the identification of both true and false lumens or the presence of an intimal flap (Figure 4). This invasive catheter-based diagnostic modality has a high specificity of 94%, but its sensitivity may be lower than that of other techniques, which has been reported to be as low as 88%. False-negative results are because of difficulties in identifying IMH, thrombosed false lumen, and simultaneous opacification of the true and false lumens when the imaging plane renders the intimal flap invisible. This modality may provide diagnostic value in identifying aortic branch vessel involvement, aortic regurgitation, and coronary involvement. However, aortography has disadvantages, including the invasive nature of transarterial catheterization, potentially long procedural time, the risks of contrast agent, and limited diagnostic accuracy in detecting IMH. These factors along with the improvement of other noninvasive imaging modalities have decreased the use of contrast aortography in the diagnosis of aortic dissection.

CT Scan

Contrast-enhanced CT imaging is an accurate, noninvasive, and fast diagnostic study, which is available in most hospitals. Continual refinement of this imaging technology has led to the development of CT angiography, as well as helical and multislice CT scanning, which have dramatically enhanced the resolution and permitted 3-dimensional reconstruction of the aorta and its branch vessels. In the IRAD study, contrast-enhanced CT scan was the most commonly performed initial diagnostic modality. The sensitivity of CT scan for the diagnosis of acute aortic dissection ranged from 83% to 100%, whereas the specificity ranged from 87% to 100%. Multislice CT provides more rapid scanning times with enhanced spatial resolution and decreased potential motion artifacts. In patients with suspected acute aortic dissection, contrast-enhanced multislice helical CT scanning of both chest and abdomen should be performed because it is particularly useful in detecting intimal flap, the extent of aortic dissection, branch vessel involvement, patency of true and false lumens, potential pericardial effusion, and even the luminal patency of proximal coronary arteries. In this imaging modality, an initial noncontrast CT scan should be performed to rule out IMH because intravenous contrast may obscure this diagnosis due to uniform opacification of luminal structures. Intramural hematoma may appear as localized thickening of the aortic wall with internal displacement of intimal calcifications. Alternatively, it may present as a crescent shaped, high-attenuation signal within the aortic wall. The diagnosis of aortic dissection is established by the visualization of the intimal flap separating the true and false lumen (Figure 5). Additional CT signs that are suggestive of aortic dissection include compression of the true lumen by the false lumen and widening of aortic lumen displaced intimal calcification. Limitations of CT scanning include inability to identify the location of intimal tear, necessity of administering iodinated contrast agents, and difficulty in assessing aortic insufficiency.

Magnetic Resonance Imaging

The MRI is a dynamic noninvasive imaging modality that provides high-resolution structural and functional information. The sensitivity and specificity of MRI for diagnosing acute aortic dissection range from 95% to 100%. The MRI has the highest
sensitivity and specificity for detecting all classes of aortic dissection with the exception of class III lesions. This modality produces high-contrast images and allows for evaluation of the entire aorta, branch vessel involvement, and associated complications of aortic dissection. The MRI is particularly helpful in identifying aortic dissection in patients with preexisting aortic disease.

Magnetic resonance imaging angiography (MRA) is a promising technique that may have the capability to produce image quality similar to the conventional contrast angiography. The MRA takes advantage of the inherent properties of unsaturated protons in the blood, thus obviating the need for contrast dye administration. In current clinical practice, 3-dimensional gadolinium-enhanced MRA is a commonly used imaging modality in most hospitals. Compared with CT scan, the gadolinium-based MRA has greater resolution, faster examination time, and reduced motion artifacts. Recent advances including the use of multiple radiofrequency amplifiers, increases in imaging gradient strength and speed, and the development of magnets with 3.0 Tesla strength are likely to increase the diagnostic accuracy for aortic dissection.

There are several limitations related to the MRI. This modality is contraindicated in patients with metallic hardware and may be further restricted by its lack of availability in emergent situations. Furthermore, critically ill patients who required mechanical ventilation or hemodynamic monitoring may not be suited for MRI due to equipment-related metallic components. Finally, because gadolinium contrast was generally considered to have less nephrotoxic effects compared with the iodinated contrast used in CT angiography, MRI was often used preferentially in patients with impaired renal function. Unfortunately, recent reports have indicated that gadolinium is associated with the development of nephrogenic systemic fibrosis in patients with chronic renal impairment, so gadolinium administration should be avoided in such patients.

**Principles of Treatment**

As the most lethal catastrophe involving the aorta, aortic dissection can be fatal if prompt diagnosis and proper treatment are not established in a timely fashion. Immediate administration of intravenous antihypertensive medication to reduce pulse rate and systemic blood pressure (dP/dT) is a critical initial medical management in all patients with acute aortic dissection. The underlying treatment objectives are to stabilize the degree of dissection, reduce dynamic aortic vessel obstruction, minimize intimal flap mobility, and lower the risk of aortic rupture. Medical treatment of aortic dissection is based upon generally well-accepted practice guidelines rather than evidenced-based clinical trials. General treatment considerations for medical, surgical, and endovascular modalities are shown in Table 3.

The only published treatment guidelines regarding medical management of aortic dissection were reported by the European Society of Cardiology in 2001, which provided specific therapeutic strategies in pharmacological interventions. In this treatment guideline, all patients with acute aortic dissection must be monitored in the intensive care unit. Initial treatment principle is focused on stabilizing the patient prior to conducting diagnostic evaluations. Systemic blood pressure control and analgesic for pain relief are key elements of medical therapy. Intravenous morphine sulfate is recommended for pain control. Invasive monitoring of blood pressure via arterial line or pulmonary capillary wedged pressure catheter may be necessary in critically ill patients. Treatment principle is to lower the aortic wall stress by reducing the systolic blood pressure and the force of left ventricular ejection. Reducing the left ventricular contractile force can correspondingly lower the dP/dT, which consequently should
diminish the propagation of aortic dissection. Intravenous β-blockers should be administered to lower the heart rate to less than 60 beats per minute. Intravenous vasodilators, such as nitroprusside, can provide strict blood pressure control if titrated appropriately. Other direct vasodilators, such as hydralazine, are less desirable because they increase aortic shear stress and have less predictable control of blood pressure. It is noteworthy that vasodilators should not be used without β-blockers because the vasodilation may result in paradoxical activation of the sympathetic nervous system resulting in an increased ventricular contraction and aortic shear stress. In the event that β-blockers are contraindicated, intravenous calcium channel blockers may be used. This published guideline also recommends that systolic blood pressure be maintained between 100 to 120 mm Hg. Blood pressure must be adjusted accordingly in the event that oliguria, spinal ischemia, or neurological changes is evident.

**Ascending Aortic Dissection**

Ascending aortic dissection (Stanford type A or DeBakey type I or II; Figure 6) is considered a surgical emergency because of the high risk of life-threatening complications including myocardial infarction, cardiac tamponade, and acute aortic

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**Table 3. Treatment Considerations in Aortic Dissection**

<table>
<thead>
<tr>
<th>Medical therapy</th>
<th>Treatment of choice in uncomplicated descending thoracic aortic dissection</th>
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<tr>
<td></td>
<td>Stable, isolated arch dissection</td>
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<td></td>
<td>Stable type B dissection (chronic, &gt;2 wk of onset)</td>
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<tr>
<td>Surgical treatment</td>
<td>Treatment of choice in acute ascending aortic dissection</td>
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<td></td>
<td>Possible operative strategies in ascending aortic dissection</td>
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<tr>
<td></td>
<td>Graft replacement of the ascending aorta</td>
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<tr>
<td></td>
<td>With aortic arch replacement if arch contains primary tear, is aneurysmal, or is ruptured</td>
</tr>
<tr>
<td></td>
<td>With aortic valve repair (resuspension) or replacement if valve is involved in dissection or diseased</td>
</tr>
<tr>
<td></td>
<td>With aortic root replacement (using composite valve graft, root bioprosthesis, or valve-sparing technique) if patient has Marfan syndrome or a dilated or severely damaged aortic root (sinuses)</td>
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</table>

Acute descending thoracic aortic dissection complicated by the following conditions:

- Retrograde extension into the ascending aorta
- Dissection superimposed on a chronic degenerative aneurysm
- Rupture or impending rupture
- Persistent or intractable pain
- Progression with end-organ ischemia

Endovascular repair with stent-graft placement

- Treatment objective to exclude entry site to the false lumen and to reperfuse the true lumen
- Potential role in the following conditions:
  - Unstable descending thoracic aortic dissection
  - Malperfusion syndrome
  - Stable descending thoracic aortic dissection (currently under clinical trial)

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**Figure 6.** Computed tomography scan of the aorta demonstrates an intimal flap (arrow) separating the true and false lumens in the chronic ascending aortic dissection.
regurgitation. Approximately 90% of patients treated nonsurgically die within 2 weeks. The objective of operative intervention is to prevent fatal complications, such as aortic rupture, stroke, visceral ischemia, cardiac tamponade, and circulatory failure. The primary surgical approach is to excise the intimal tear, to obliterate the false lumen, and to replace the aorta with prosthetic interposition graft with or without reimplantation of the coronary arteries. Additionally, restoration of aortic valve competence may be necessary in patients with aortic insufficiency by resuspension of the native aortic valve or valve replacement. Various surgical treatment techniques in repairing an ascending aortic dissection are listed in Table 3.

In a clinical series of patients who had ascending aortic dissection and who did not undergo surgical repair, the mortality reported was 38% within 1 day, 50% by 2 days, and 70% within 1 week. With numerous clinical studies reporting the operative mortality rates of 15% to 35%, operative repair has a markedly prognostic advantage compared with medical treatment. It is noteworthy that stroke is considered a relative contraindication for surgical repair due to the concern that anticoagulation and reperfusion may convert ischemic stroke to hemorrhagic stroke. In the initial IRAD study, there were a total of 289 patients with ascending aortic dissection. Patients who underwent surgical repair had a significantly better outcome compared with those treated with medical therapy, with a mortality rate of 26% and 58%, respectively.

**Descending Aortic Dissection**

It is a widely accepted clinical practice that stable patients with descending aortic dissection be treated medically due in part to the high mortality rates associated with surgical intervention, which range from 30% to 45%. Operative interventions for descending aortic dissection include rapid progression, intractable chest or back pain, aortic branch vessel involvement with compromising perfusion to vital organs, and impending aortic rupture. Patients with acute dissection superimposed upon a chronic degenerative descending or thoracoabdominal aortic aneurysm are at particularly high risk of early rupture and are also treated surgically. Nienaber et al published the first comparison study between open repair and stent grafting of patients with subacute or chronic descending thoracic aortic dissection. Among the 12 patients who were treated with endovascular devices, there was no morbidity or mortality. In contrast, open repair was performed in 12 patients, which was associated with 4 deaths (33%) and 5 serious adverse events (42%) within 12 months. Although this outcome seemed to suggest a improved treatment outcome with endovascular vascular, it is noteworthy that the outcome may be influenced by a potential patient selection bias because the sample size was relatively small and the comparison was not randomized.

The first IRAD report included 175 patients with descending aortic dissection. Among them, the mortality rate of medical treatment was 11% compared with a 30% mortality rate with surgical repair; this difference partially reflects the fact that surgical treatment is reserved for patients with life-threatening complications. The most common causes of death were aortic rupture and complications related to ischemia, which was 38.5% and 15.4%, respectively. This study further reported that shock or severe hypotension, the absence of chest/back pain, and branch vessel involvement were all significant markers in hospital mortality. Although medical intervention provides improved clinical outcome compared with surgical intervention in uncomplicated descending thoracic aortic dissection, long-term clinical outcomes comparing these 2 treatment modalities remain unclear.

Recent advances in endovascular interventions have prompted several researchers to suggest that asymptomatic acute descending thoracic aortic dissection should be treated with this minimal invasive treatment strategy to prevent acute and late complication. The primary objective of endovascular stent grafting in the setting of a descending thoracic aortic dissection is to occlude the primary and the proximal entry site (Figure 7). Researchers have hypothesized that in an aortic dissection, aortic pressure and perfusion becomes preferentially diverted to the false lumen, which expands and becomes aneurysmal. The expansion of the false lumen can also compress the true lumen, which can lead to inadequate perfusion of vessels arising from the true lumen or compression of visceral, spinal, and limb vessels because of dynamic compromise. This phenomenon of malperfusion is the most common form of complication arising from a descending thoracic aortic dissection requiring intervention. The theoretical benefit of covering the primary entry site by an endograft is to redirect aortic flow through the true lumen, while decompressing the false lumen that would eventually collapse and become thrombosed.
At the present time, the utility of endovascular treatment for acute descending thoracic aortic dissection remains limited, and is generally considered in the treatment of life-threatening complications, such as impending aortic rupture, end organ ischemia, recurrent or persistent intractable pain, progression of dissection, aneurysm expansion, and uncontrolled hypertension. In the treatment guideline released by the European Society of Cardiology Task Force on Acute Aortic Dissection in 2001, the roles of endovascular stent graft and endovascular fenestration are provided based on the type of descending thoracic aortic dissection (Table 4). Moreover, in patients who were deemed high risk for surgical repair due to age or underlying comorbid conditions, endovascular treatment may offer palliative benefit to those with symptomatic lesions who otherwise would have been offered medical treatment.

It is important to note that there is a consensus that stent grafts should be used with great caution, if at all, to repair aortic dissection in patients with Marfan syndrome or other connective tissue disorders. In these genetic conditions, fixation zones for endografts are invariably diseased and consequently subject to future dilatation resulting in endoleaks. When conversion to open repair is required, the need to remove the endograft increases the complexity of the aortic reconstruction and the risk of surgery.

### Table 4. Endovascular Treatment Strategies in Descending Thoracic Aortic Dissection

| 1. | Stenting of obstructed branch origin for static obstruction of branch artery |
| 2. | Balloon fenestration of dissecting membrane plus stenting of aortic true lumen for dynamic obstruction |
| 3. | Aortic stenting to keep fenestration open |
| 4. | Fenestration to provide reentry tear for dead-end false lumen |
| 5. | Aortic stent-graft placement to cover the entry site into the false lumen and reperfuse the true lumen |

At the present time, the utility of endovascular treatment for acute descending thoracic aortic dissection remains limited, and is generally considered in the treatment of life-threatening complications, such as impending aortic rupture, end organ ischemia, recurrent or persistent intractable pain, progression of dissection, aneurysm expansion, and uncontrolled hypertension. In the treatment guideline released by the European Society of Cardiology Task Force on Acute Aortic Dissection in 2001, the roles of endovascular stent graft and endovascular fenestration are provided based on the type of descending thoracic aortic dissection (Table 4). Moreover, in patients who were deemed high risk for surgical repair due to age or underlying comorbid conditions, endovascular treatment may offer palliative benefit to those with symptomatic lesions who otherwise would have been offered medical treatment.

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### Endovascular Stent-Graft Devices

Several endovascular stent-graft devices have been studied in the treatment of descending thoracic aortic dissection. Although some of these devices were originally designed for thoracic aneurysm application, reports of their clinical utility in covering the entry point of an aortic dissection have sparked interests in determining their clinical efficacy in the treatment of thoracic aortic dissection. These devices have various inherent advantages and drawbacks, which undoubtedly require clinical investigation.
to validate their therapeutic role in aortic dissection. A brief summary of these thoracic endograft devices is provided below.

The Zenith Dissection Endovascular System

The Zenith dissection endovascular system (Cook Medical, Bloomington, Indiana) combines a covered proximal Cook Zenith TX2 endograft component with an uncovered, bare-metal stent. The Zenith TX2 endograft is constructed of dacron fabric covered by stainless steel z-stents. In this endograft system, the proximal end is covered and has stainless steel barbs protruding through the graft fabric (Figure 8), which anchor the graft directly to the aortic wall. The distal bare stent component is constructed of stacked z-stents joined by polypropylene sutures, which can be deployed through a 16-French sheath and inserted through the existing Zenith TX2 proximal component sheath (Figure 9). A single stent diameter accommodates aortic luminal diameters ranging from 24 to 38 mm and is available in 82-, 123-, and 164-mm lengths. The z-stents exert a minimal radial force, which allows gradual opposition of the dissection septum and reexpands the true lumen. The large open strut architecture allows maintenance of branch vessel perfusion, so that the stent can be safely deployed.

**Figure 8.** The proximal component of the Zenith TX2 thoracic endograft device, which has stainless steel barbs protruding through the graft fabric for secure aortic attachment. Adapted from Cook Medical, Bloomington, Indiana.

**Figure 9.** The Zenith TX2 distal bare stent component is designed for the treatment of aortic dissections, which is used as a distal extension with the main Zenith TX2 component. Adapted from Cook Medical, Bloomington, Indiana.

**Figure 10.** A glass model depicting the treatment strategy using the TX2 proximal component with the distal bare stent component. Note how the bare stent can be deployed over the intercostal, lumbar, and abdominal visceral vessels in the setting of aortic dissection or malperfusion syndrome. Adapted from Cook Medical, Bloomington, Indiana.
across the origins of intercostal, visceral, and renal arteries. In the scenario of persistent malperfusion due to a dissection flap into or a reentry tear near a vessel origin, the bare z-stent component provides structural scaffolding for placement of a bare or covered peripheral stent from the true lumen into the branch vessel bridging across the false lumen (Figure 10). The Zenith dissection endovascular system is proposed to facilitate remodeling of the aorta with effective closure of the entry site without coverage of a long length of aorta.

The Medtronic Thoracic Endograft System

The Talent thoracic stent-graft system (Medtronic, Santa Rosa, California) is composed of a nitinol stent between layers of polyester graft. A modified form of the Talent endograft system or the Valiant endograft system was introduced with device modification to make it more flexible in device deployment and implantation. The Talent device has a longitudinal support bar throughout the length of the endograft. Individual stents are secured to the graft with suture. Between individual stents is an unsupported graft to allow for flexibility. The proximal end of this stent graft is made in 2 configurations, which include either a serrated (open web configuration) or an open bare stent segment (FreeFlo configuration; Figure 11). The bare stent FreeFlo configuration allows device implantation across the orifice of the left subclavian or common carotid artery, while maintaining antegrade blood flow. In an effort to improve the deployment accuracy and technical ease, the long connecting bar of the Talent device has been removed in the Valiant device, whereas columnar support has been optimized through stent spacing and the exoskeleton. The removal of the connecting bar enables the device to become more flexible. Additionally, it has eliminated the need to orient the device in the aorta due in part to its enhanced flexibility. The Valiant stent graft has a modified proximal FreeFlo configuration with 8-bare peak wires (shown left) compared with the 5-bare peak wires found in the Talent stent graft (shown right). Adapted from Medtronic, Santa Rosa, California.

The Gore TAG Endoprosthesis

The Gore TAG endoprosthesis (WL Gore & Associates, Flagstaff, Arizona) is composed of a symmetrical expanded polytetrafluoroethylene (ePTFE) tube externally reinforced with a layer of ePTFE or fluorinated ethylene propylene (FEP; Figure 13). An exoskeleton consisting of nitinol stents is attached to
the entire external surface of the graft with ePTFE/FEP bonding tape. Both proximal and distal segments of the endograft have scalloped flares that are to facilitate endograft conformity in a tortuous thoracic aorta. Two radiopaque gold bands are attached to the base of the flares serving as a guide during deployment and in graft surveillance. A PTFE sealing cuff, which is affixed to the base of the flares, is attached on 1 end with FEP, while allowing the other end to remain free. This is designed to enhance the device attachment to the aortic wall and potentially reduce type I endoleaks. This device contains a unique deployment mechanism in which the endograft is constrained by an ePTFE/FEP sleeve connected to a deployment knob located at the control end of the delivery catheter (Figure 14). Release of the endograft begins in the midgraft region to reduce distal displacement via a wind-sock effect. Following the device deployment, a unique trilobed balloon, which permits continuous antegrade aortic blood flow during balloon inflation, is used to ensure full device attachment to the aortic wall (Figure 15). The flexible catheter delivery system and the rapid deployment mechanism are potentially beneficial, particularly for deployment in curved segments of aorta within or close to the aortic arch.

Clinical Experience With Endovascular Interventions

The EUROSTAR and the United Kingdom Thoracic Endograft Registries to date remains the largest clinical database of patients treated with thoracic aortic endograft devices. In the latest publications of this combined registry, a total of 131 patients with aortic dissection (5% proximal, 81% distal, 14% not classified) were treated with stent grafts, whereas 57% of these patients presented with symptoms of rupture, aortic expansion, or side-branch occlusion. The primary technical success was achieved in 89%, whereas the 30-day mortality was 8.4%. Spinal cord ischemia with resultant paraplegia occurred in 0.8% in this series. Due to the limitation of this registry database, long-term follow-up data have not been reported. However, among 67 patients with available follow-up records, the 1-year survival rate following endograft implantation was 90%.

A report based on a single institutional experience from the UCLA Harbor Medical Center included 24 high-risk patients with descending thoracic dissection who underwent endovascular stent-graft repair. Among them, there were 16 patients with acute dissections and 8 patients with chronic dissections. There were 3 procedural-related
deaths with an overall perioperative mortality rate of 13% (3 of 24). Among them, 1 patient died because of endoleak-related aortic rupture, whereas 2 other patients died due to retrograde dissection with cardiac tamponade. One patient survived following open conversion with immediate endograft explantation due to retrograde dissection. These 3 cases of retrograde dissection were all associated with an open wire stent-graft design deployed within the thoracic arch. Due in part to the limited device availability in this early clinical experience, the 2-year mortality rate was 17% in this high-risk patient population.93

Researchers from the Arizona Heart Institute recently reported their experience of 40 patients (23 acute patients and 17 chronic patients) who were treated with endovascular stent graft for descending thoracic aortic dissection. Postoperative complications occurred in 15 patients (38%) with the majority being renal and pulmonary adverse events. However, postoperative paraplegia occurred in 1 patient (2%). Among those patients who were able to complete follow-up with CT scan surveillance, 97% of patients showed stabilization of aortic dissection with thrombosis of the false lumen or decreasing of aortic diameter. The 1-year survival rate was 85%. The authors reported a remarkable long-term follow-up outcome with a 4-year survival rate of 89.4%.

A similar study reported by Italian researchers included endovascular repair of 43 patients (24 acute dissections and 19 chronic dissections) with complicated descending thoracic aortic dissections. Three patients suffered immediate retrograde dissection following stent-graft deployment, which resulted in cardiac tamponade and death. It is noteworthy that thoracic endograft with an open stent design was used in all these 3 cases. No cases of spinal cord ischemia were noted. Two patients died during the follow-up period due to continual progression of the dissection despite successful endograft exclusion of the dissection entry site. Remarkably, all remaining
38 patients survived and developed thrombosis of the false lumen during a mean follow-up period of 20 months.96

A recently published Talent Thoracic Registry, which was an industry-sponsored retrospective study, included 457 consecutive patients (113 emergency, 344 elective) who were treated with the Medtronic Talent endograft.97 Among those patients, 180 patients (38%) had aortic dissection, whereas 37 patients (8%) had acute descending thoracic aortic dissection. Technical success was 98%, and 0.7% of patients required direct surgical conversion. Morbidities including stroke and paraplegia occurred in 4% (n = 17) and 1.7% (n = 8) of patients, respectively. In-hospital mortality, including the 113 emergency cases, was 5%, and late mortality was 8.5% within the mean follow-up interval of 24 ± 19 months. Survival rates based on Kaplan-Meier analysis were 91% at 1 year, 85% at 3 years, and 78% at 5 years. Although this study was in part supported by the endograft manufacturer, the outcome of this registry suggested that endografting of descending thoracic dissection can result in relatively low morbidity and mortality rates.

The INSTEAD trial (Investigation of Stent grafts in patients with Type B Aortic Dissection) was one of the first prospective trials designed to compare outcomes of patients randomized to either best medical therapy or endovascular repair with thoracic stent-graft devices. Sponsored by Medtronic and using the Talent endograft device, this study was designed to enroll and randomize a total of 136 patients from various European centers with a follow-up period of 24 months. The study inclusion criteria include age greater than 18 years, uncomplicated type B dissection, and lack of spontaneous thrombosis in the false lumen 14 days after the index event. In designing the trial, a retrospective analysis of 80 patients treated by endovascular approach was compared with 80 patients treated medically; 2-year survival in the endovascular group was 94.9% and in the medical group, 67.5%.82 Preliminary report of the INSTEAD trial revealed that 1-year mortality for the endovascular group was 10% versus 3% for medically treated cohorts.82,88 On the basis of this finding, researchers from these trials noted that elective, prophylactic stent grafting does not appear to be justified in asymptomatic medically controlled patients with subacute or chronic descending thoracic aortic dissection.82,88 However, critical questions remain unanswered that include how to identify those with stable descending thoracic aortic dissection but might require later intervention, and long-term outcome between open surgical and endovascular treatment of this condition. These issues will likely be addressed by a larger clinical trial that will likely be initiated in the near future.

**Conclusion**

Aortic dissection is an uncommon but potentially lethal condition with devastating complications. This condition commonly affects older patients with a history of chronic hypertension. Prompt diagnosis with timely intervention is critical to ensure patient survival. Advances in diagnostic modality have raised the awareness of contributory variants of aortic dissection, including penetrating ulcer and IMH. Our understanding of the pathogenesis of this condition based on these etiological variants has led to the development of more defined classification, which stratifies these risk factors thus potentially allowing better imaging surveillance for disease progression.

Current treatment strategies have been broadened due in part to the advances in endovascular technologies. Medical treatment with blood pressure control has been the mainstay of intervention in patients with uncomplicated descending thoracic aortic dissection. Surgical repair of descending thoracic aortic dissection is indicated in patients with intractable chest or back pain, aortic branch vessel involvement with compromising perfusion to vital organs, impending aortic rupture, and acute dissection superimposed on a preexisting chronic aneurysm. The widely used endovascular technology in infrarenal aortic aneurysm repair has increased the enthusiasm of adopting this treatment strategy in aortic dissection. This less invasive treatment modality may play a therapeutic role in critically ill patients who are deemed high risk for surgical intervention. It is noteworthy that currently there is no approved thoracic stent graft in the United States for the treatment of aortic dissection. All available reports describing endograft treatment of aortic dissections were based on either off-labeled application or emergent usage in critically ill patients who were deemed high risk for surgical repair. Although encouraging outcomes were noted in several clinical series, these published series should not form the basis of indiscriminate adoption of this catheter-based treatment modality in patients with aortic dissection.
Further clinical trials to demonstrate the long-term efficacy of endovascular technology are clearly needed in the treatment of aortic dissection. In the end, comparative studies will also be necessary to delineate the role of endovascular stent graft in patients with aortic dissection compared with either medical intervention or surgical repair.

References

Vascular and Endovascular Surgery / Vol. 43, No. 1, February/March 2009


