The first surgical management of aortic dissection was reported in 1935 by Gurin et al., who created a distal reentry point in the iliac artery to decompress the false lumen. In 1949, Abbott et al. reported the repair of a chronic dissection by wrapping cellophane around the descending aorta to reinforce it. Despite the efforts of these early pioneers and other investigators, it was not until 1955 that a major therapeutic advance was made; this was the year that DeBakey, Cooley, and Creech introduced a revolutionary surgical treatment that involved excision of the intimal tear, obliteration of the false lumen, and either direct reanastomosis or insertion of a prosthetic graft. The next great milestone in therapy was introduced by Wheat et al. in 1965, when they described medical therapy directed toward lowering blood pressure and dP/dt. Since then, investigators have made significant advances in the detection, characterization, and treatment of aortic dissection; however, the morbidity and mortality of this debilitating disease remain alarmingly high, with an overall in-hospital mortality of 27.4% reported by the International Registry of Aortic Dissection (IRAD).

The latest additions to the armamentarium to treat dissection have been based on percutaneous interventional techniques. The minimally invasive nature of these techniques makes them an attractive alternative to open surgical intervention; however, the exact role and long-term durability of these procedures remain to be proven. The earliest endovascular therapies were directed toward the complications of aortic dissections and included angioplasty of an obstructed aorta, stenting of obstructed branch vessels, and fenestration of the dissection flap to relieve mesenteric ischemia. More recently, the advent of the stent graft has led to a novel endovascular approach aimed at treating the inciting lesion of aortic dissection by obliterating the primary intimal tear. Since the first reports in 1999, which involved treatment of acute and chronic aortic dissections, investigators have extended the application of stent grafts to treat a variety of related pathologies, such as intramural hematoma (IMH), penetrating atherosclerotic ulcer (PAU), and traumatic dissection.

In this report, we begin by reviewing key features of aortic dissection pertinent to endovascular management and review the current literature. Specifically, we will discuss the use of stent grafts, fenestration, and branch-vessel stenting in the treatment of aortic dissection and related pathologies.

### Pertinent Features

#### Classic Dissection

The sine qua non of the classic aortic dissection is a tear in the intima that allows pulsatile blood to penetrate the vessel wall. A cleavage plane develops between the layers of the intima and media and allows a column of blood to form within the intramural space, composing the false lumen. The dissection may propagate in an antegrade or retrograde direction or in both directions. The location of the intimal tear usually occurs in a compromised region of the vessel with underlying mural degeneration. Common causes include long-standing hypertension, connective tissue disorders, and trauma.

It is important to differentiate the primary or entry intimal tear from the secondary or reentry tear(s). Approximately two thirds of primary tears occur in the ascending aorta, with more than half of these located within the first 2 cm of the ascending aorta. The next most common site of the primary tear is the isthmus of the aorta just beyond the ligamentum arteriosum. These regions are presumably subjected to the greatest hemodynamic stress, which makes them more susceptible to injury. In either location, these tears are 5 times more likely to be transverse in orientation rather than longitudinal. Other sites of primary tears include the descending thoracic aorta, aortic arch, and abdominal aorta, with multiple primary tears seen in ~8% of cases.

Secondary tears are identified less frequently than primary tears. The secondary tears tend to occur at the ostia of branch vessels, where often circumferential shearing of the intima from the vessel origin has taken place. When present, they allow blood to communicate between the true lumen and the dissecting column.

Whether a reentry tear is present or not, during the acute phase of a dissection, the local environment of the false lumen is highly thrombogenic owing to the exposed adventitial and medial layers. This, in addition to the morphology of the dissection and underlying hemodynamics, may cause the false channel to become completely or partially thrombosed.
Commonly, the true lumen, which is bound by intima, will course along the inferomedial aspect of the distal arch and descending aorta. Typically, the celiac trunk, superior mesenteric artery, and right renal arteries arise from the true lumen, and the left renal artery arises from the false lumen; however, variation in this pattern is frequent. Although branch-vessel obstruction may occur, more commonly, flow to branch vessels remains unobstructed with supply from the true lumen, false lumen, or both the true and false lumens.

Related Pathologies

Rapid advances in imaging technology have led to a greater understanding of aortic dissection variants. In particular, 2 entities (IMH and PAU) have been recognized and, when diagnosed in symptomatic patients, are categorized under the umbrella term “acute aortic syndromes” alongside classic type A and type B dissection.17,18

IMH is defined as blood within the intramural space without identification of an intimal disruption. In the purest description, IMH is considered a precursor to dissection, originating from a ruptured vasa vasorum within the medial layer. Subsequent aortic wall weakening or infarction may lead to formation of an intimal tear and result in a classic dissection. However, it is possible that numerous cases of IMH actually represent cases of classic dissection in which the intimal tear is occult on imaging studies.19

PAU is defined as focal ulceration of an atherosclerotic plaque that penetrates a variable depth through the intima and may be associated with intramural blood. The hematoma may propagate along the media and lead to aortic rupture or, less frequently, to an aortic dissection with development of a true and false lumen.20,21

Malperfusion Syndrome

A major complication of aortic dissection is obstruction of flow to the aortic branch vessels. This may involve any aortic branch with 1 or more vascular territories threatened. Critical ischemia of the vascular territory is termed “malperfusion syndrome.” It is usually related mechanistically to branch flow obstruction by an intimal flap. Two patterns of branch-vessel involvement are described, dynamic obstruction and static obstruction.22

Dynamic obstruction is a term that characterizes the phenomenon of aortic true-lumen collapse or obliteration. Imaging in the setting of dynamic branch-vessel obstruction shows a paper-thin, crescent-shaped true aortic lumen dwarfed by a larger false lumen. The aortic flap has a convex contour that appears to flatten or efface the true lumen. Consequently, flow to all downstream abdominal branches supplied by the true lumen may be compromised. Further obstruction to a particular branch vessel may occur as the dissection flap prolapses over the branch-vessel ostium. Because of the constantly changing position of the intimal flap, particularly in the acute phase, these obstructions can be total or subtotal with persistent or intermittent features and thus are referred to as dynamic obstructions. A clear understanding of the pathophysiology responsible for dynamic obstruction is not apparent; however, large proximal entry tears are frequently observed.22

In distinction, static obstruction refers to the effects of direct extension of the dissection process into an individual branch. Typically, flap progression into a branch is tolerated. This occurs when the false lumen within the branch vessel develops a distal reentry tear that allows double-barrelled flow to the vascular bed with both true- and false-lumen contribution. In most cases, biluminal perfusion of the branch-vessel tissue bed is sufficient and is not associated with serious ischemia or the risk of necrosis. However, despite the “reentry” branch configuration described, the physical presence of the flap may cause various degrees of static obstruction depending on the artery involved, the adequacy of flow, and the presence of underlying lesions.22

The potential for profound malperfusion exists when the false lumen within the branch does not have a reentry point in the vessel. As a result, the false lumen is a blind cul-de-sac that enlarges and compresses the true lumen. The severity of true lumen obstruction is maximum at the distal margin of false-lumen extension, where a prominent bulbous contour is frequently evident. This “no reentry” branch configuration of static obstruction is often associated with severe ischemia related to the absence of false-lumen flow within the branch and a severely compromised true lumen. Fortunately, static obstruction with no reentry of the false lumen occurs relatively infrequently compared with the more common pattern of false-lumen reentry.22

In addition, it is possible for a combination of dynamic and static obstruction with or without branch-vessel reentry to coexist within an individual patient, which creates an interesting “signature” profile depending on the particular branch vessels affected and mechanism(s) of involvement by the process. As we will see, differentiating between a dynamic or static obstruction has significant implications for endovascular management.9,22

Classification Systems

The 2 most common anatomic classifications of aortic dissection are the DeBakey and Stanford classifications. Under the DeBakey system, type I dissection begins in the proximal aorta and involves both the ascending and descending thoracic aorta, type II dissection is confined to the ascending aorta, and type III is confined to the descending aorta. Under the Stanford system, type A dissection involves the ascending aorta, whereas type B dissection does not.

The convenience and prognostic value of the Stanford system has resulted in its popular use; however, an important feature that is not distinguished in the Stanford system is the location of the primary tear. In a typical type A dissection, the primary tear is located in the ascending aorta, whereas in a retrograde type A dissection, the tear is located in the descending aorta. This detail has a profound influence on the current feasibility of stent-graft management.

Aortic dissections are temporally classified as acute when identified ≤2 weeks from the onset of symptoms and chronic when identified >2 weeks from the onset of symptoms, with the highest morbidity and mortality occurring during the acute phase. Some investigators further classify the acute phase as early (<24 hours) and late (≥24 hours to 2 weeks) from the onset of symptoms.
In addition, type B dissections are often classified as complicated or uncomplicated. Complicated cases involve rupture, lesion progression, impending rupture, refractory hypertension, localized large false aneurysm, continued pain, or malperfusion syndrome and are associated with worse outcomes.23,24

Natural History and Conventional Management

Conventional treatment of both type A and B dissection involves prompt management of systemic blood pressure and dP/dt to stabilize the extent of dissection, avoid false-lumen dilatation, reduce pain, and decrease risk of rupture. In type A dissection, concomitant emergent surgical graft replacement with or without aortic valve repair or replacement is mandated to reduce the risk of sudden death associated with aortic rupture, aortic regurgitation, pericardial tamponade, coronary artery involvement, and malperfusion of the brain. In uncomplicated type B dissection, surgical therapy has shown no superiority over medical management alone and is reserved for complicated cases.25

Acute Type A Dissection

Although shown to be superior to medical management alone, surgical management is still associated with alarmingly high rates of morbidity and mortality. In a review of 547 type A dissections, IRAD investigators demonstrated a hospital mortality rate of 27% for those patients treated surgically.26 In another IRAD report, surgical repair was associated with in-hospital mortality rates of 10% by 24 hours, 16% by 7 days, and nearly 20% by 14 days.3 Long-term survival for patients treated with surgery who were discharged alive has been shown to be 96% at 1 year and 91% at 3 years.27

Patients with retrograde type A dissection (DeBakey type IIId) represent an important subgroup that comprises 4% to 20% of all type A cases.16,23,28,29 In these individuals, the inciting primary tear is typically positioned in the distal arch, with retrograde extension of the dissection process to the ascending aorta. This poses a dilemma for surgical repair that involves either excision of the entry tear with replacement of both the ascending aorta and aortic arch, which is associated with high morbidity and mortality, or graft replacement of the ascending aorta alone without excision of the primary tear, which leaves the patient at risk of postoperative aortic rupture.16,23,28–30 Regardless, conventional therapy mandates immediate surgical repair in this patient subgroup.

Acute Type B Dissection

Owing to the small risk of aortic rupture and sudden death and the high morbidity and mortality associated with surgical repair of the descending aorta, medical treatment alone is advocated for uncomplicated type B dissection. Surgery is typically reserved for complicated type B dissections. IRAD investigators reviewed 175 patients treated according to this complication-specific approach and identified in-house mortality rates of 11% and 31% for medical and surgical treatment, respectively.5 Patients who undergo surgery also have a high rate of morbidity; in particular, paraplegia has been reported in 1.5% to 19% despite advances in surgical technique.31 IRAD investigators also performed long-term evaluation of outcomes for patients discharged after hospital management of type B dissection. This analysis identified 3-year survival rates of 78% and 83% for patients treated medically and surgically, respectively.32

Because medical therapy alone does not stop flow within the false lumen, 20% to 50% of patients who survive the acute phase develop aneurysmal dilatation of the false lumen within 1 to 5 years after onset.15,33,34 In this regard, the majority of late deaths that occur in patients with type B dissection initially managed by medical therapy are due to rupture, extension of dissection, and perioperative mortality of subsequent aortic or vascular surgeries. In fact, the long-term survival of patients with type B dissection remains worse than that of patients with type A dissection.27,32

Aortic Dissection Variants (IMH and PAU)

Intramural Hematoma

The natural history of IMH is not well understood; however, it accounts for 5% to 20% of acute aortic syndrome cases.13,21,35,36 IRAD investigators demonstrated an association between increased hospital mortality and proximity of IMH to the aortic valve, regardless of medical or surgical treatment.18 A meta-analysis of 11 studies identified an overall mortality of 34% for type A IMH, 24% for those treated surgically and 47% for those treated medically.19 Overall mortality for type B IMH was 14%, with little difference between surgical (15%) and medical (13%) groups. Thus, the treatment paradigm for IMH parallels that of classic aortic dissection, with surgical repair favored for type A IMH and medical management preferred for type B IMH.

Penetrating Atherosclerotic Ulcer

Although PAU is poorly understood, its prognosis is thought to be much poorer than that of classic aortic dissection.37 Coady et al20 reported the risk of aortic rupture in patients with PAU and acute symptoms to be 40% compared with patients with type A or type B dissection, for whom the rupture risks were 7% and 3.6%, respectively. Although no consensus treatment strategy exists, early surgical graft replacement of the aorta has been advocated in symptomatic patients.38,39

Chronic Dissection

Those patients who survive the acute stage of aortic dissection, which is associated with the greatest mortality, by definition have chronic dissection. The 30-day survival rate for this population is high at 90%, independent of whether they were managed medically or surgically.40 Medical therapy is therefore recommended for patients with both type A and type B chronic dissection. Surgery is reserved for those who develop an aneurysm, rupture, peripheral branch-vessel compromise, or other complications.41,42

Malperfusion Syndrome

It is estimated that branch-vessel involvement complicates approximately one third to one half of all aortic dissections.24,43 With control for both age and gender, branch-vessel
involve in-hospital mortality in acute type B dissections.44
Primary surgical aortic repair results in successful revascularization in the vast majority of patients with type B dissection; however, the presence of renal or mesenteric ischemia has been correlated with especially high surgical mortality rates of 70% and 87%, respectively.24,43,45,46 For this reason, alternative surgical procedures have been explored to address organ ischemia specifically. In particular, surgical aortic fenestration has been shown to relieve malperfusion syndrome in 93% to 100% of cases, with an in-hospital mortality rate of 0% to 43%.47–49

**Endovascular Management**

Endovascular management of dissection comprises 3 major treatments: (1) aortic stent-graft placement, (2) dissection flap fenestration, and (3) branch-vessel stenting. Typically, 1 or more of these techniques is used to treat aortic dissection. In some cases, endovascular techniques may obviate the need for surgical management, whereas in other cases, endovascular techniques are complementary to surgical repair.

**Stent-Graft Technology**

The development of thoracic aortic stent grafts has largely followed in the footsteps of abdominal aortic stent-graft technology used primarily to treat abdominal aortic aneurysms. The earliest feasibility and safety studies of thoracic stent grafts were performed in 1992 to treat thoracic aortic aneurysms. Since then, the number of applications of this technology has grown rapidly to include the management of aortic dissection and dissection variants. Although treatment of descending thoracic aneurysm with a stent graft (TAG; WL Gore and Associates, Flagstaff, Ariz) is currently approved by the US Food and Drug Administration, the use of stent grafts to treat other indications such as aortic dissection and its related pathologies remains off-label.

The first-generation stent grafts were primarily homemade devices that married graft materials such as polyester or polytetrafluoroethylene to modified self-expanding stents such as the Gianturco Z stent. Most delivery systems were large (24F to 27F), relatively rigid, and difficult to deploy smoothly and accurately owing to extensive frictional resistance. Significant improvements were made in the second generation of devices, which are largely manufactured commercially. However, as we will demonstrate, numerous design issues remain. Current devices include the TAG (WL Gore and Associates), Talent (Medtronic Inc, Santa Rosa, Calif), TX-2 (Cook Inc, Bloomington, Ind), Relay (Bolton Medical, Inc, Sunrise, Fla), and Valiant (Medtronic Inc).

**Principles and Techniques of Endovascular Management**

**Stent-Graft Management**

The rationale of stent-graft management is 2-fold. First, in the acute phase, the use of stent grafts may prevent imminent aortic rupture and relieve dynamic branch-vessel obstruction. Second, stent-graft management may promote thrombosis of the thoracic false lumen and decrease the long-term morbidity associated with patency of the false lumen, including aneurysmal dilatation, late aortic rupture, and late mortality.9,16

Stent-graft treatment is predicated on the ability to cover the primary intimal tear and create a seal to stop the flow of blood entering the false lumen and prevent the transmission of systemic pressure across the major intimal defect (Figures 1 and 2). If the seal is adequate, cardiac output is redirected into the true lumen and rapid the false lumen simultaneously decompresses (which relieves dynamic obstruction of branches supplied by a diminutive true lumen). As a result, within seconds, the true lumen diameter typically enlarges, with markedly improved flow. The immediate hemodynamic and morphological alterations may prevent imminent rupture and relieve aortic true-lumen collapse and branch-vessel ischemia.9

Furthermore, stent-graft management of retrograde type A dissection and type B dissection has been shown to decrease flow in the false lumen and induce false-lumen thrombosis.50,51 Again, this is a critical point, because natural history studies of type B dissection have shown 20% to 50% of patients who receive medical therapy alone and survive the acute phase ultimately develop aneurysmal dilatation of the false lumen within 1 to 5 years.15,33,34 Even if complete thrombosis of the false lumen does not occur, it is likely that partial thrombosis and decreased flow will limit the progression to aneurysmal dilatation.9

To obliterate the primary tear, an adequate seal zone is required. One of the anatomic requirements is a proximal landing zone (relative to the primary tear) of at least 15 to 20 mm. The ideal landing zone should be uniform in shape and free of significant disease; however, this ideal is rarely met, because the position of the primary tear and its proximity to branch vessels usually requires device deployment within a dissected segment. A common dilemma is selection of the “correct” device dimension, because the true lumen is generally crescentic or elliptical in shape and a fraction of the overall transaortic diameter. Most operators base their selection on more than 1 measurement, the most compelling of which is the diameter of the nondissected aorta immediately proximal to the entry tear. In the setting of a classic entry location just distal to the left subclavian artery origin, the segment between the left carotid and left subclavian arteries is used. This is the best estimate of the original size of the involved aorta before dissection. This measurement is oversized by ~10% and used to select the stent-graft diameter. The oversizing factor ensures secure anchoring and a tight circumferential seal.

Depending on the type and size of the stent graft, currently available devices will require delivery systems that are 20F to 24F in size. The iliofemoral arteries should be assessed routinely to ensure that an adequate intraluminal diameter to accommodate introduction of the device exists. Access usually involves surgical exposure of the common femoral artery. In the case of small or heavily calcified femoral arteries, surgical exposure of the iliac arteries or aorta with or without placement of a graft conduit may be required. Recently, in select patients, stent-graft procedures have been performed entirely percutaneously, with the puncture sites closed by commercially available suture-mediated access-
The obvious benefits of this approach over surgical exposure are the decreased time to recovery and possible reduced risk of infection, lymphocele, seroma, and postoperative scar.

Fenestration

Fenestration of the intimal flap serves as an alternative endovascular treatment to stent-graft management of aortic dissection (Figure 3). Rather than treating the entry tear,
which increases the resistance to false-lumen inflow, fenestration is aimed at artificially creating a distal reentry channel, which decreases the resistance to false-lumen outflow. The reentry channel is not dissimilar to spontaneously formed reentry tears that help to balance pressures within the aortic lumens. The equalization of pressure between the true and false lumen alone may relieve dynamic obstruction of the aorta and branch vessels. In addition, strategic positioning of fenestrations may locally redirect a sufficient amount of blood flow to perfuse compromised end organs. Although beneficial in the acute phase, the continued patency of the false lumen potentially predisposes the patient to less salutary long-term outcomes, such as false-lumen dilatation, aneurysm development, and rupture, compared with those after stent-graft placement and thoracic false-lumen thrombosis.

Because stent-graft treatment generally achieves similar results with a greater likelihood of false-lumen thrombosis, stent-graft management is preferred over fenestration as the primary mode of treatment in patients with malperfusion caused by dynamic obstruction. However, fenestration continues to be a valuable option in poor surgical candidates and in cases in which stent-graft treatment is not available or not feasible owing to anatomic constraints. Fenestration may also serve as a valuable adjunctive treatment in cases in which surgical repair or stent-graft repair inadequately addresses the indications for intervention.

Currently, percutaneous balloon fenestration of the flap is most frequently performed distally in the abdominal aorta at the level of the aortic bifurcation, usually to manage unilateral lower-extremity ischemic symptoms. Several approaches to dissection flap fenestration have been described. The 2 most commonly detailed techniques involve combined intravascular ultrasound and fluoroscopic guidance or fluoroscopic guidance alone.

When intravascular ultrasound is used, the probe is typically advanced into the larger lumen (usually the false lumen) while a long curved coaxial cannula/needle set, precurved transcatheter needle, or reentry catheter with needle (Outback, Cordis, Miami, Fla; Pioneer, Medtronic, Inc) is advanced into the other lumen. The dissection flap is perforated under ultrasound guidance. Subsequently, a guidewire is advanced through the needle into the opposite lumen. Over the wire, the intimal defect is enlarged with an angioplasty balloon (usually 12-mm to 20-mm diameter). After angioplasty of the fenestration, it is essential to reevaluate the patency of all branch vessels for compromise, because the resultant alteration in blood flow is not always predicted accurately. In the case of inadequate flow through the aorta or branch vessels, an additional fenestration or stenting may be required.

When fluoroscopic guidance is used alone, a balloon catheter is placed in the larger lumen and used as a target when the intimal defect is enlarged with an angioplasty balloon (usually 12-mm to 20-mm diameter). After angioplasty of the fenestration, it is essential to reevaluate the patency of all branch vessels for compromise, because the resultant alteration in blood flow is not always predicted accurately. In the case of inadequate flow through the aorta or branch vessels, an additional fenestration or stenting may be required.

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Regardless of the technique used, successful and safe fenestration re-
quires a high level of interventional skill and intimate familiarity with a wide range of devices, including intravascular ultrasound, angioplasty balloons, stents, snares, occlusion balloons, and transseptal needles.

Stent Placement

The use of uncovered stents in the aorta and side branches may improve flow in aortic dissection (Figure 4). The most common indications include (1) inadequate relief of dynamic obstruction after surgery, stent-graft treatment, or fenestration and (2) static obstruction of abdominal aortic branch vessels, which is typically unaffected by proximal aortic stent-graft treatment and fenestration.9,53,55

In most cases of symptomatic static branch-vessel compromise, attempts are made to restore more normal flow dynamics by placing an uncovered stent via the aortic true lumen into the true lumen of the involved branch. The stent usually covers the length of the dissected branch segment. On stent deployment, the flap is displaced, and a cylindrical true lumen is established. In some instances, stents may be placed from the false lumen of the aorta to the true lumen of the branch vessel to restore flow.53,55

Uncovered aortic stents may also be placed to prop open a severely collapsed true lumen and allow flow to reach distal branches supplied exclusively by the aortic true lumen.53,55 Similarly, uncovered aortic stents may be a useful adjunct to dissection fenestration. After fenestration, the intimal flap

Figure 3. This patient underwent surgical repair for a Stanford type A dissection several years previously and returned complaining of severe lower-extremity claudication. An aortogram (A) demonstrated flow-limiting compression of the true lumen by the false lumen (arrows) between the levels of the celiac trunk and an infrarenal aortic stent as the cause of claudication. The aortic stent seen in panel A was placed soon after surgical repair of the dissection to improve distal flow. With use of an Outback reentry catheter, wire access was obtained from the true lumen to the false lumen (B). Angioplasty alone was insufficient in relieving the obstruction; thus, the fenestration was reinforced with a self-expanding stent (C). The postfenestration aortogram (D) demonstrates improved flow through both the true and false lumens.

Figure 4. An aortogram (A) of a Stanford type B dissection demonstrates extension of the dissection flap into the right renal artery, resulting in a static obstruction. This was treated by placement of 2 overlapping covered stents from the aortic true lumen into the renal artery (B). After stent placement, perfusion was restored to the kidney.
may take on a position that continues to obstruct the aorta and branches fed by the true lumen. In such a case, a stent may be placed across the fenestration to prop open the fenestration and tack down a distal tag of intimal flap.66 A variety of both balloon-expandable and self-expanding stents have been used in the aorta and in renal, iliac, superior mesenteric, and brachiocephalic arteries.53,55,56

### Outcomes of Endovascular Management

#### Acute Type A Dissection

Significant controversy remains about the treatment of retrograde type A dissections in which the primary tear is positioned in the descending aorta with retrograde involvement of the ascending aorta. Kato et al59,60 treated 10 patients with retrograde type A dissection (none of whom showed evidence of cardiac tamponade or severe aortic regurgitation) using endovascular stent grafts. Entry closure and complete thrombosis of the false lumen of the ascending and descending aorta were achieved in all patients. During a mean follow-up of 20 months, all patients were alive and without rupture or aneurysm formation.

Several investigators have reported hybrid surgical and endovascular treatment of acute type A aortic dissections with the primary tear located in the ascending aorta. The combined technique uses traditional open surgical methods to identify and resect the primary tear, followed by stent-graft deployment into the descending aorta under direct, open visualization. The rationale is based on the potential long-term benefits of false-lumen thrombosis and aortic remodeling associated with endograft treatment of the descending aorta; the benefits are thought to include lower rates of aneurysm formation, expansion, and rupture.61–63 Finally, in poor surgical candidates, primary endoluminal stent-graft management of type A dissection with stent-graft placement across an entry tear in the ascending aorta has been described.54–67

### Acute Type B Dissection

#### Meta-Analysis

A meta-analysis by Eggebrecht et al68 reviewed all published data between January 1999 and May 2004 involving stent-graft placement for aortic dissection (96% type B and 4% retrograde type A) with an entry tear located in the descending aorta (Table). Only studies with a minimum of 3 patients were included. Cases of antegrade surgical (“open”) stent-graft placement via the aortic arch were excluded. A total of 39 studies comprising 609 patients were reviewed.

Procedural success was obtained in 98.2% of cases. Neurological complications were seen in 2.9%, with 1.9% representing periprocedural stroke and 0.8% representing paraplegia. Retrograde extension of dissection into the ascending aorta was seen in 1.9% of cases. The total in-hospital surgical conversion rate was 2.3%. The overall in-hospital mortality rate was 5.2%, and the 30-day mortality rate was 5.3%. Interestingly, this 30-day mortality was compared with IRAD data, which detailed 30-day mortality rates for type B dissection managed medically and surgically to be 10.7% and 31.4%, respectively.5 A composite Kaplan-Meier table including all 3 interventions showed worse survival outcome in the surgical group and similar outcomes in the medical and stent-graft treatment groups.

A mean follow-up time of 19.5 months was available for 561 patients. During follow-up, false-lumen thrombosis was observed in 75.5%, late surgical conversion in 2.5%, and a supplemental endovascular stent-graft procedure in 4.6%. The total reintervention rate was 11.9%. Late death occurred in 2.8%.

#### European Collaborators on Stent Graft Techniques for Thoracic Aortic Aneurysm and Dissection Repair and the United Kingdom Thoracic Endograft Registries

The compilation of cases from the European Collaborators on Stent Graft Techniques for Thoracic Aortic Aneurysm and Dissection Repair (EUROSTAR) and the United Kingdom (UK) Thoracic Endograft registries represents the next-largest reported series to date69 (Table). This series included 131 patients treated for acute dissection, with all cases having an entry tear identified in the descending aorta (81% type B, 5% retrograde type A, and 14% classification unavailable). At the time of the procedure, 43% were symptomatic, whereas 57% of cases were complicated by symptoms of rupture, aortic expansion, or side-branch occlusion. In the series, 42% of patients were at high risk for conventional open surgery, with an American Society of Anesthesiologists score of 3 or 4.

Table. Endovascular Stent-Graft Repair of Type B Aortic Dissection

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<th>Reference, First Author</th>
<th>Year</th>
<th>No. of Patients (Characteristics)</th>
<th>Technical Success, %</th>
<th>Retrograde Dissection, %</th>
<th>Stroke, %</th>
<th>Paraplegia, %</th>
<th>30-Day Mortality, %</th>
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</table>

N/A indicates not applicable.
Procedural success was achieved in 89% of cases, with the remaining 11% having either incomplete coverage of the entry tear, persistent flow with only partial thrombosis of the thoracic false lumen, no expansion of the true lumen, or endoleaks. Periprocedural endoleak was seen in 6.7%. Neurological complications were seen in 2.3%, with 1.5% representing stroke and 0.8% representing paraplegia. The overall 30-day mortality rate was 8.4%.

One-year data were available for 67 patients, with late intervention required in 1.5% and late endoleak identified in 1.5%. Late death occurred in 1.5% of patients, with a 1-year cumulative survival rate of 90%.

Contemporary Studies
A list of more contemporary studies is provided in the Table.68–76 These studies report similar short-term and midterm results compared with the meta-analysis by Eggebrecht et al68 and the EUROSTAR/UK registries.69

This collection of observational data demonstrates that stent-graft treatment of type B dissection is feasible, with high technical success rates and acceptable short-term outcomes. Around the world at aortic-disease–referral centers, stent-graft treatment is currently performed for complicated type B dissection in which conventional surgical repair continues to be associated with strikingly high morbidity and mortality. Endograft management can be used to treat most complications and avoids the major trauma of open surgery. Notably, reports of endograft management detail markedly lower rates of paraplegia than surgical management of the descending aorta.31

In contrast, the role of stent-graft management for uncomplicated type B dissection is uncertain. Currently, no conclusive data exist that demonstrate superiority of either stent-graft or medical management. To address this issue, the INSTEAD (INvestigation of STEnt grafts in patients with type B Aortic Dissection) trial is under way.

INSTEAD Study
The INSTEAD study is a prospective, randomized, controlled clinical trial that compares stent-graft treatment of uncomplicated chronic type B dissection (occurring 2 to 52 weeks before randomization) with optimal medical therapy alone.77 The study design is based on a retrospective comparison of 80 patients with type B dissection treated with a stent graft compared with 80 patients treated with medical management alone. A Kaplan-Meier life-table analysis showed that at 12 months, the risk of death in the stent-graft–treated group was 5.1% versus a historic mortality rate of 27.5% with conventional therapy. The INSTEAD study enrolled 136 patients at 11 European clinical sites. The primary end point is all-cause mortality at 1 year after enrollment. It is anticipated that the forthcoming results will help shed light on the role of stent grafts in chronic uncomplicated type B dissections.

Dissection Variants
Penetrating Atherosclerotic Ulcer
The application of stent grafts to aortic dissection variants has focused more on PAU than other lesions.11,37,78–80 PAU is typically a focal process that involves the descending thoracic aorta, which makes it anatomically well suited for endograft treatment.

Eggebrecht et al80 performed a systematic review of 54 patients accumulated from 13 studies. This report detailed complete sealing of the ulcer in 94% of cases, neurological complication in 6%, and an in-hospital mortality rate of 5%.

A more recent study by Demers et al11 evaluated 26 patients with type B PAU (half of whom were deemed inoperable) and found a primary success rate of 92% and perioperative mortality of 12%, with no cases of paraplegia. Estimated survival rates were 81% at 1 year and 65% at 5 years.

Recently, Eggebrecht et al79 presented 22 cases of PAU treated by stent-graft placement with a median follow-up of 27 months. A technical success rate of 96% was achieved. One patient (5%) had a minor stroke, and no other in-hospital complications or deaths occurred. The overall survival rates were 100% at 30 days, 100% at 1 year, 82.5% at 2 years, and 62% at 5 years.

These data suggest high technical success rates with favorable acute and midterm mortality rates compared with the natural history of the disease. Although only limited amounts of data exist, stent-graft treatment of PAU appears to be a viable treatment option.

Intramural Hematoma
The treatment of IMH with stent grafts has been reported; however, the precise definition of its current role in the repair of IMH is still in evolution.9,12,19,78

Malperfusion Syndrome
Endovascular management of malperfusion may involve 1 or more techniques, including stent-graft placement, fenestration, and side-branch stenting. Together, they represent a powerful and promising set of techniques. Yet, only a small collection of data on the outcomes of these techniques exists.

Dake et al12 investigated stent-graft management of malperfusion syndrome in aortic dissection. In this series from the Stanford University School of Medicine, 11 of 19 patients had malperfusion syndrome, which represented 38 of 114 infradiaphragmatic vascular beds examined. Malperfusion syndrome was caused by dynamic obstruction in 22 arteries, dynamic and static obstruction in 15 arteries, and static obstruction only in 1 artery. After stent-graft treatment of the primary tear in the thoracic aorta, all cases of dynamic obstruction resolved, whereas only 6 of 15 arteries with combined dynamic and static obstruction resolved. The remaining vascular beds were treated successfully with placement of uncovered stents in side branches.9 In a smaller series, Duebener et al11 treated 5 cases of malperfusion syndrome in aortic dissection using stent grafts; of these, 2 patients had persistent visceral ischemia that required surgical fenestration and ultimately had fatal courses.

The 2 largest series using fenestration and stenting (without stent-graft management) were from the University of Michigan and Stanford. In the Michigan study, 24 cases of malperfusion syndrome in aortic dissection (13 type A, 8 type B, and 3 atypical) were treated with fenestration and/or stent placement. A total of 92% of the vascular beds were reper-
Complications

With growing worldwide experience and longer follow-up periods, the spectrum of complications associated with stent-graft treatment of aortic dissection is becoming more apparent. Several of these complications that deserve particular attention are discussed briefly.

Two potentially devastating complications of both open surgical repair and endovascular stent-graft repair are stroke and paraplegia. The cause of stroke associated with stent-graft treatment is thought to be primarily atheroembolic, with a stroke rate comparing favorably to that of open surgical repair of the descending aorta. The cause of paraplegia after surgical repair of the thoracic aorta is thought to be multifactorial, influenced by the duration and severity of the ischemic insult, the neuronal metabolic rate during the ischemic period, postoperative hypotension, and subsequent reperfusion injury. Despite improvements in conventional surgical techniques for the descending aorta, paralysis occurs in 1.5% to 19% of cases. The apparent lower risk of paraplegia after endograft treatment, reported to be between 0.8% and 3.6%, is likely attributable to the avoidance of aortic cross clamping and subsequent reperfusion required during open surgery. Although great concern existed over stent-graft coverage of intercostal arteries, it appears to be better tolerated than predicted initially. Nonetheless, extensive coverage of intercostal arteries is discouraged because of the heightened risk of spinal cord ischemia.

Retrograde dissection into the ascending aorta is a serious complication associated with the formation of new intimal tears, aneurysm formation, aortic rupture, tamponade, and death. It typically requires immediate open surgical repair. This complication is identified in approximately 2% of cases during the in-hospital period and 2% of cases during follow-up. The causative mechanism implicated by most reports is a stent-graft–induced intimal injury due to limited flexibility along the outer edges of current devices. The maintenance of meticulous interventional technique is another important consideration that may decrease the frequency of injury to the intima by guidewire and catheter manipulations. It is likely that the rate of retrograde dissection will improve as manufacturers create tapered and precurved stent grafts with less rigid, pliable ends.

Stent-graft collapse has been reported in up to 3% of cases in a series of 87 (Figure 5). In a separate series, 6 cases of stent-graft collapse were reported after treatment of 5 traumatic aortic injuries and 1 dissection with the TAG device. Both reports attributed all cases to oversizing of the stent graft to the aorta. These cases may reflect the inherent difficulty of sizing stent grafts in patients with extremely small true lumens. Furthermore, these cases may reflect the tendency of more experienced operators to push the anatomic limits of stent-graft therapy by treating dissections for which no ideal commercially available device exists. Nonetheless, it is safe to assume that improvements in stent-graft technology will likely offset this phenomenon and ultimately lead to a decrease in the rate of early and late collapse.

Overall, periprocedural endoleaks are encountered in approximately 6.7% of cases, and late endoleak is identified in 1.5%. Most type I endoleaks are identified in the periprocedural...
period, whereas most type II endoleaks are identified during follow-up. Extrapolating from stent-graft treatment of abdominal aortic aneurysm, type I endoleaks require immediate repair, usually involving placement of an additional stent extension, and type II endoleaks may be monitored. The consequence of endoleaks in stent-graft management of aortic dissection is not well known, although such leaks are associated with persistent flow in the false lumen and may predispose to complications of false-lumen expansion or rupture.

Other reported complications include stent-graft migration, stent-graft torsion, aortoesophageal fistula, and mobile thrombus within the stent-graft lumen. These complications, in addition to retrograde dissection, stent-graft collapse, endoleak, aneurysm formation, and late rupture, which are described above, underscore the importance of close and lifelong imaging follow-up of these patients.

**Treatment Considerations**

With the increasing use of stent-graft management for aortic dissection, a need exists to better understand the specific determinants of short- and long-term outcomes. This is essential to improve the process of patient selection and allow practitioners to better tailor procedures on the basis of patient characteristics.

Several studies have demonstrated higher in-hospital mortality rates and major complication rates for patients treated with stent-graft placement for type B dissection in the acute phase compared with the chronic phase. Kato et al speculated that worse outcomes in the acute phase after endograft treatment may reflect sequelae caused by unfavorable morphological alterations of a fragile dissection membrane compared with manipulation of a more fibrotic and stable flap in the chronic phase. This led some investigators to favor treatment in the chronic phase. However, Egg-ebrecht et al suggested that a more important factor was the preprocedural clinical status of the patient. They documented that patients with poor clinical health status (American Society of Anesthesiologists class ≥3) had a reduced life expectancy after stent-graft therapy compared with patients with moderate comorbidities (American Society of Anesthesiologists class ≤3). This may explain the poorer outcome in the acute-phase treatment groups described in several case series, which are often composed of sicker patients. In particular, both age and renal failure have been identified as independent determinants of postintervention mortality. Interestingly, the importance of preprocedural clinical status was supported by a study by Chen et al in which an opposite trend was identified, with a lower 30-day mortality rate after stent-graft management in the acute-phase treatment group compared with the rate for the chronic-phase treatment group; the authors attributed the difference to the fact that patients in the acute-treatment group of that study were healthier.

The potential long-term benefit of stent-graft management of aortic dissection is based on the induction of false-lumen thrombosis and remodeling. The overall rate of complete thrombosis of the thoracic false lumen is ~75%. Identification of the determinants of false-lumen thrombosis and strategies to improve false-lumen thrombosis rates have been topics of interest. Kusagawa et al and Resch et al have both shown that thoracic false-lumen thrombosis rates and shrinkage of the false lumen occur more often in patients treated in the acute phase than in the chronic phase.

Whereas false-lumen thrombosis is consistently seen at the level of the implanted stent graft, thrombosis distal to the implant, particularly in the abdominal aorta, is less common. This may relate to the to-and-fro movement of the intimal flap along uncovered segments of the aorta, with retrograde flow through distal flap fenestrations or secondary distal tears. This has implications for the selection of device...
length. Most investigators use stent grafts longer than needed to cover the primary tear alone, usually 15 to 20 cm. The additional distal coverage provides a more normal anatomic configuration by placing the distal device margin within the midthoracic segment, where the aorta usually becomes less curved. Further distal extension may expedite the rate of false-lumen thrombosis; however, extension of the stent graft into the distal third of the thoracic aorta may increase the risk of spinal cord ischemia and paraplegia. In light of this, some investigators have advocated the placement of bare stents into the distal thoracic aorta to provide structural stability without risking occlusion of intercostal arteries.91

Finally, it is becoming clear that adoption of this relatively new treatment modality involves an inherent learning curve that affects not only physicians who perform endovascular procedures but also equipment designers and manufacturers. Eggebrecht et al.68 in their meta-analysis of stent-graft management of type B aortic dissection, reported improved technical success rates in studies published between 2002 and 2004 compared with those published between 1999 and 2001. Additionally, fewer overall complications occurred in centers that performed a total number of cases above the median number of cases performed by all centers in the analysis. The findings likely reflect improvements in device technology and increasing operator experience associated with stent-graft management of aortic dissections over the last decade.68

Conclusions

The role of endovascular techniques in the treatment of aortic dissection and dissection variants continues to evolve. At most large aortic-dissection—referral centers, the primary indication for stent-graft management remains complicated type B aortic dissection for which acceptable early and midterm results have been established. Stent-graft treatment has also been shown to be promising in the treatment of retrograde type A dissection, PAU, and chronic dissection. Further investigation is needed to identify the role of aortic endograft in the treatment of classic type A dissection and IMH. The use of dissection flap fenestration and branch-vein stenting appears to be best suited for patients unable to be treated surgically or with a stent graft, or as adjunctive therapy. With rapidly accumulating data, a greater understanding has been reached of the treatment considerations and complications of endovascular techniques for aortic dissection. It is certain that advances in graft design will address these issues and lead to greater applicability of stent grafts in the management of dissection; however, randomized, controlled clinical trials are needed to demonstrate any advantage of endovascular treatment over conventional management.

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References


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