AHA Scientific Statement

Surgical Management of Descending Thoracic Aortic Disease: Open and Endovascular Approaches
A Scientific Statement From the American Heart Association

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Vascular Disease

Recent years have witnessed the emergence of novel technologies that enable less invasive endovascular treatment of descending thoracic aortic disease (TAD). This has occurred against a backdrop of improved identification of various disease processes and better results with open surgical repair. The natural history of the specific acute aortic syndromes that affect the descending thoracic aorta has also been described with more clarity and has become more commonly recognized. This is in part secondary to the widespread availability and application of advanced imaging technologies that permit precise diagnoses. As data are accumulating, these pathological processes involving the descending thoracic aorta are no longer thought of as simply variants of one another but as distinct entities with well-defined clinical behavior. As the technology for endovascular repair continues to mature and its utilization increases, there is a need for a careful assessment of the current state of medical management, traditional open therapy, and evolving endovascular treatment of distinct thoracic aortic pathologies.

The purpose of this scientific statement is to present a contemporary review of the various pathological processes that affect the descending thoracic aorta: Aneurysms, dissections, intramural hematomas (IMHs), penetrating atherosclerotic ulcers (PAUs), and aortic transections. These disorders will be considered in detail, with an exploration of the natural history, available treatment options, and controversies regarding management. Current intervention criteria will be reviewed with respect to both open surgical repair and endovascular treatment.

Our goal is to provide the healthcare professional with a better understanding of the pathophysiology of the various disease processes that involve the descending thoracic aorta and to review current outcomes and technical pitfalls associated with these therapies to facilitate strong, evidence-based decision making in the care of these patients.

General Considerations for Stent Grafting Versus Open Repair for TAD

Treatment of descending TAD involves complex, exigent decision making in an era of evolving technology. Survival data for nonoperative management are often dismal for the majority of the descending TADs discussed in this document.[1,2] For many patients, the gold standard (open surgical repair) provides long-lasting results; however, earlier reports for TAD indicate a perioperative mortality rate that ranges between 12% and 44%, depending on the extent of comorbidity and urgency of the surgical repair.[3,4]

Recent literature suggests a significant improvement in the mortality rate (4% to 9%) and incidence of paraplegia (3%) for descending thoracic aortic resections for thoracic aortic aneurysms (TAAs) at high-volume aortic centers.[5,6] Despite these improvements in outcomes with open repair, a less invasive approach is quite appealing, especially in older patients and in patients with significant concomitant comorbid diseases, many of whom would be unsuitable candidates for conventional open repair.

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Advancing age correlates with higher operative risk after an open repair. The prevalence of TAD appears to have tripled in the 2 most recent decades.[7] Whether this upswing in prevalence represents an increase in the elderly proportion of the population, improved diagnostic capabilities, or an actual increase in incidence is unknown.[7] Nevertheless, TAA rupture is more common in the elderly, and as detailed in the sections that follow, type B aortic dissections, PAUs, and IMHs are more frequently seen in older patients. Because the morbidity and mortality associated with open surgical repair of descending aortic replacement are much greater in the elderly population, there has been increasing interest in treating this group of patients with aortic stent grafting as an alternative to open repair. To date, however, no clinical trial has compared optimum medical management of descending TAD in the elderly with stent grafting. Although the long-term durability of stent grafts remains undefined, the durability of these devices may be of less concern in older patients with degenerative conditions and limited life expectancies. However, there are many younger patients, such as those individuals with posttraumatic transections, for whom the durability of these devices becomes much more relevant. To date, the 3 US Food and Drug Administration–approved thoracic aortic endografts have demonstrated good durability at 5 years of follow-up, but continued follow-up of patients in whom these devices have been placed will be needed.

In addition to patients with advanced age, endovascular stenting may well be a reasonable alternative in patients with advanced pulmonary disease. Chronic obstructive pulmonary disease is associated with TAA growth and rupture and is a significant risk factor for open surgical repair.[8] In these patients, the pathology involving the descending thoracic aorta is likely to be better treated by stent grafting and avoidance of a thoracotomy. Life expectancy of a patient with chronic obstructive pulmonary disease is often <5 years; therefore, concerns about the durability of stent grafting are not as great as with healthier patients.[9]

In making decisions on when to use stent grafting as an alternative to open repair, there is also a paucity of prospective studies in the literature comparing the 2 therapies in a head-to-head fashion. Specifically, there are no prospective randomized studies comparing open and endovascular treatment of descending TAD. Reports in the literature, including the majority of studies summarized in the present statement, primarily include single-center experiences and nonrandomized studies of open and endovascular stent graft procedures with limited follow-up. Most patients undergoing thoracic aortic stent grafting have been treated outside the confines of prospective trials, and the long-term stability of thoracic endografts is unknown. Stent grafts generally have been designed to have a durability of 10 years based on International Standardization Organization stress testing.[10] In the Stanford study of their initial 103 first-generation stent-grafted descending TAA patients, 30±6% required aortic reintervention by 8 years.[11] Therefore, in the young patient especially, stent grafting should be used with circumspection, because the long-term durability of these individual devices is largely unknown. Having made this statement, we should also realize that the Stanford study[11]

primarily used first-generation devices, which have been modified and improved since that time.

Perhaps the most complete and comprehensive report on the results of open repair versus stent grafting for treatment of descending TAAs is a multicenter, comparative, phase II, nonrandomized clinical trial that evaluated the Gore TAG thoracic endograft against an open-surgery control cohort of patients with descending thoracic aneurysms.[10] In that study, 137 of 140 patients had successful implantation of the endograft. The perioperative death rate in the endograft versus the open-surgery control cohort was 2.1% versus 11.7%, respectively (P<0.001). Thirty-day analysis revealed a statistically significant lower incidence of complications in the endovascular cohort than in the open-surgery cohort, including paraplegia/paraparesis, respiratory failure, and renal insufficiency. The overall stroke rate was similar in both the endograft and open-surgery control cohorts. The mean lengths of intensive care unit stay were shorter in the endovascular cohort. Although this study had limited 2-year follow-up, there was a 9% incidence of endoleaks and 3 reinterventions associated with endovascular versus open surgical repair. There was no survival advantage associated with either strategy after 2 years (Figure 1).

Another prospective, comparative, nonrandomized trial of stent grafting versus open surgery of the descending thoracic aorta was performed on 105 patients (68 degenerative aneurysms, 21 penetrating ulcers, 15 pseudoaneurysms, 9 traumatic tears, and 1 acute dissection) versus 93 patients who had open surgical repair for descending thoracic aneurysms. The rate of operative death was halved with stent grafting, despite the overall higher-risk population of patients treated. There was similar late survival for both cohorts. Reinterventions were required at a nearly identical rate for open-repair and stent-graft patients, and both groups experienced similar rates of spinal cord ischemic complications.[12]

Available data from individual-center reports and nonrandomized studies will be presented as they relate to individual disease processes; however, several general concepts regarding endovascular treatment and open repair will be emphasized.

Stent-Grafting Considerations

Thoracic endografting has been shown to be technically feasible in every variety of descending TAD by a number of very sound studies. Early success, however, has been documented primarily in retrospective, single-center experiences. For high-risk groups, stent grafting offers the potential for lower morbidity and mortality than with open repair. The exact role and specific recommendations for stent grafting, however, remain to be defined. This conundrum is in part secondary to the relatively short period of time during which this technology has been used and the relative paucity of centers that have systematically investigated its use. Therefore, there is a trend toward use of stent grafts in patients who have not met standard recommended criteria.[10]

The diameter of the aorta is the single most commonly used criterion for deciding to intervene operatively for a TAA.[13] Aortic growth is also of great importance, as discussed below in the section on TAAs; however, given the relative ease of stent grafting in the descending thoracic aorta,
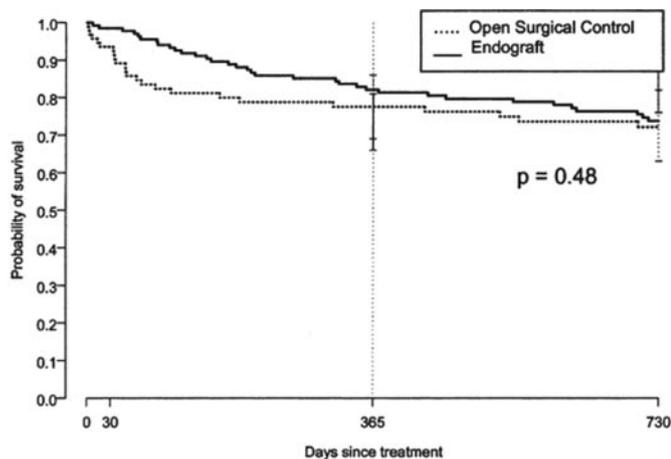
Figure 1. Kaplan–Meier survival curves for the endograft group vs the open surgical cohort. Reprinted from Bavaria et al,[7] with permission from Mosby. Copyright 2007, The American Association for Thoracic Surgery.

the procedure has been used on varying descending aortic pathology at far smaller diameters than those sizes routinely selected for standard open-surgery procedures.[10] This variance in using traditional criteria relates to the lack of long-term follow-up studies and the understanding of the risks and benefits of stent grafting versus either medical management or open surgical replacement of the descending thoracic aorta.[7] It is not clear whether the trend toward more aggressive endovascular management of thoracic aortic disease will influence prognosis, freedom from complications, or survival compared with open surgical management or medical management. Overall, the indication for aortic stent grafting of descending thoracic aortic pathology should be based on a predicted operative risk that is lower than the risk of either conventional open repair or optimal medical management. Use of standard operative criteria is imperative given the lack of adequate long-term outcome data.

Marfan Syndrome and Connective Tissue Disorders
Patients with Marfan syndrome and other connective tissue disorders deserve special consideration because the disease process commonly involves the descending thoracic aorta. There are a few reports of short-term success after endovascular stent grafting of descending thoracic aortic pathology in patients with Marfan syndrome.[14] There is, however, limited information regarding the impact of persistent radial forces of the stent graft in the abnormal aorta in patients with this condition.[10] In chronic aortic dissections, although stent grafting in patients with Marfan syndrome is feasible, postintervention surveillance confirms that the aorta continues to dilate despite satisfactory endograft deployment and false-lumen thrombosis.[15] Therefore, stent grafting may be a viable option in patients for whom open surgery poses prohibitive risks; however, it is not recommended currently in this subset of patients. Marfan syndrome and other connective tissue disorders historically have been strict exclusion criteria in all commercial thoracic aortic stent-graft trials. Patients with these conditions are often young, and the long-term durability of stent grafting in these patients with abnormal connective tissue is unknown.

Extent of Aortic Disease
The extent of descending thoracic aortic involvement is typically not a limiting factor in determining the suitability for stent grafting beyond the need for suitable proximal and distal landing zones.[10] The length and diameter of the landing zones depend on the particular stent graft used. Branch-vessel coverage of the left subclavian artery is feasible and safe when combined with revascularization of the vessel. Coverage of the left subclavian artery without revascularization can be accomplished with safety provided that the cerebellum and posterior cerebrum are not dependent on left vertebral arterial flow. Left upper-extremity claudication can occur postoperatively, but this is not life-threatening and can be treated subsequently with a carotid-subclavian bypass. If the right vertebral artery is dominant, the majority of patients tolerate coverage without serious neurological complications, although EUROSTAR (EURopean collaborators On Stent/graft Techniques for Aortic aneurysm Repair) data suggest that ischemic spinal cord complications may occur more frequently in patients in whom the left subclavian artery is covered without the performance of left subclavian artery revascularization, especially in patients who have extensive coverage of the distal thoracic aorta.[16]

The extent of endovascular coverage does affect the risk of paraplegia or paraparesis. As shown in the TAG multicenter trial, descending thoracic aortic stent grafting may be associated with a lower risk of spinal cord injury than replacement of an equivalent aortic segment with open surgical techniques.[7]

Risk of Cerebrovascular Accidents
The risk of stroke with stent grafting has been high historically, especially with older delivery systems. In the initial Stanford experience, among 103 cases, the stroke rate was $7 \pm 3\%$.[17] This rate is most likely secondary to use of older large, stiff sheath/dilator stent-delivery systems that required manipulation of hardware across the diseased aortic arch of elderly patients.[10] In their initial experience of 171 thoracic endovascular aortic repairs, the University of Pennsylvania reported a perioperative stroke rate of 5.8%.[18] All new perioperative strokes were embolic in nature, and the in-hospital mortality rate associated with perioperative stroke in this patient population was 33%. Risk factors for stroke included prior stroke, severe atherosclerotic disease of the aortic arch, and stent-graft landing zone in the distal aortic

arch or proximal descending thoracic aorta. These findings suggested that cerebral embolism from intravascular instrumentation of the aortic arch in patients with vulnerable atheroma was the major cause of stroke during thoracic endovascular aortic repair procedures. The presence of vulnerable atheroma in the aortic arch can be defined by prior thromboembolic stroke, high-grade atheroma (atheroma that protrudes >5 mm, is ulcerated, or is pedunculated) on computed tomography (CT) scan, or atheroma with mobile elements on echocardiographic examination. On the basis of these findings, the risk of stroke can be reduced by patient selection and minimizing or avoiding instrumentation of the aortic arch. Newer stent-graft systems require less manipulation across the aortic arch. In the more recent Gore TAG phase II trial, the rate of cerebrovascular accidents was approximately 4% in both the TAG stent-graft group and the open surgical repair group, consistent with earlier reports.[7]

Overall Risk Assessment and Complications

In determining suitability for both open and endovascular repair, it is important to consider all of the major factors that influence the risk of descending thoracic aortic complications in the context of procedural risks to determine when and whom to treat. Considerations for an intervention also include expectant results with medical management rather than open operative repair or stent-graft implantation. Models have been developed to assist in risk assessment.[19]

Despite relatively low initial death rate, complications with endovascular stenting include endoleaks, stent fractures, and aortic-related death. Given the ease of aortic stent grafting relative to open surgical repair, it has been applied more liberally to patients with various descending thoracic aortic pathologies, including aortic aneurysms, dissections, penetrating ulcers, IMHs, and aortic transections. Stent-graft patients require serial surveillance CT scans and possible reintervention at a later date. Mitchell[20] has identified 4 different types of endoleaks that may complicate endovascular treatment of descending aortic diseases: Type I, leak at the anastomotic junction of the aorta and the stent graft; type II, collateral vessels that communicate within the aneurysm sac; type III, stent-graft junction leak or fabric disruption that can be treated easily by further stent-graft placement; and type IV, leak through the stent-graft fabric due to graft-material porosity.

It must be remembered that unlike the abdominal aorta, the disease process in the descending thoracic aorta is more diffuse and often involves the entire thoracic aorta. The disease process also evolves over the years and may not become manifest during the immediate posttreatment interval of observation. Therefore, it is likely that disease progression in previously untreated areas of the thoracic aorta will provoke type I endoleaks at previously hemostatic attachment sites.

Results With Traditional Open Surgery

Before we embark on a discussion of the various disease processes that affect the descending thoracic aorta and the treatment options, a generalized understanding of the current state of open aortic replacement is important. Because of the frequency of the disease, most of the outcomes data on descending thoracic aortic replacement are found in studies of TAAs. In reviewing clinical results, careful distinction between thoracic aortic replacement and thoracoabdominal aortic replacement is critical. Although the literature is replete with studies of thoracoabdominal aortic surgery, limited series exist detailing the results of thoracic aortic operations that are performed less frequently.[21] With thoracic aortic replacement, the duration of renal and visceral ischemia is minimal because the aortic anastomosis is often accomplished in <30 minutes. In addition, if distal perfusion techniques are used, distal ischemia may be avoided completely. Therefore, examination of literature that presents results of thoracoabdominal aortic replacement is likely to overestimate the attendant risk of open treatment of the pathologies presented in this document.[21]

In 2008, Minatoya et al[22] reviewed their experience in treating descending aortic aneurysms with open replacement techniques with partial cardiopulmonary bypass. A total of 113 patients underwent graft replacement of the descending aorta for aneurysmal disease. The mean age was 68±12 years. Sixteen cases (14.2%) were emergency cases. All operations were performed through a left thoracotomy with partial cardiopulmonary bypass with segmental clamping. Since 1998, preoperative magnetic resonance angiography has been performed to detect the Adamkiewicz artery in elective cases. Motor evoked potentials were measured intraoperatively. The overall early death rate was 5.3% (1.0% for elective cases and 31.3% for emergency cases). Rates of spinal cord dysfunction were 2.7% overall (1.0% in elective cases and 12.5% in emergency cases). Stroke rates were 7.1% overall (4.1% in elective cases and 25.0% in emergency cases). Rates of respiratory failure were 9.7% overall (9.2% in elective cases and 12.5% in emergency cases). No patient underwent a reoperation for the same lesion as a result of technical problems during the follow-up period. Kaplan–Meier overall survival estimates were 92.2% at 3 years, 90.6% at 5 years, and 70.2% at 10 years. The authors concluded that descending aortic replacement performed with partial cardiopulmonary bypass involves a risk comparable to that associated with thoracic endograft placement.

Articles on stent grafting often refer to open repair of descending TAAs and point out that the mortality rate associated with open repair is >10% and that the risk of spinal ischemia is 4% to 5%.[22] The results of open repair, however, have improved dramatically in recent years. Coselli et al[6] published the largest series that reported results of thoracic aorta surgery for repair of degenerative aneurysms in 2004. This group demonstrated a death rate of 4.4% and a paraplegia rate of 2.6% after open repair of descending thoracic aneurysms.[6] Estrera et al[5] reported a death rate of 8.8% and a paraplegia rate of 2.7% after open repair of descending TAAs with cerebrospinal fluid drainage and distal perfusion. Even with hypothermic circulatory arrest, Patel et al[23] reported a mortality rate of 6.0%, a stroke rate of 6.8%, and a spinal ischemia rate of 4.5%. Their results with open repair are quite comparable to those of endovascular repair reported for descending TAAs. Although open repair is, by definition, more invasive, open surgical treatment is definitive, with long-term follow-up well defined. Clinical results (both open and endovascular) of specific acute aortic syn-

dromes are discussed below within the context of the specific disease processes.

Aneurysms of the Descending Thoracic Aorta

An aortic aneurysm may be defined as a doubling in diameter of the normal aorta for a particular body surface area, age, and gender.[24] In aneurysmal disease, the aortic wall becomes progressively weakened, which leads to complications that include dissection, rupture, and even death. The mean age at the time of diagnosis ranges between 59 and 69 years, with men predominating over women with a ratio of from 2:1 to 4:1. Very often, individuals with an aortic aneurysm have concomitant medical conditions, including hypertension, coronary artery disease, chronic obstructive pulmonary disease, and congestive heart failure.

Aneurysms of the descending thoracic aorta account for approximately 30% to 40% of all TAAs and are now estimated to affect 10 of every 100 000 elderly adults.[7] Their prevalence has appeared to triple in the past 2 decades, and they carry a substantial risk of death.[7] These aneurysms most commonly arise at the level of the left subclavian artery, and unlike ascending aortic aneurysms, they are more often associated with significant gross and microscopic atherosclerosis; however, atherosclerosis as an underlying cause of the arterial dilation is quite controversial. One current theory is that as the aorta dilates, boundary-layer separation occurs along the surface, and hemodynamic forces trigger subintimal proliferative changes that result in plaque formation.[8,25] Therefore, although atherosclerosis occurs commonly in descending aortic aneurysms, atherosclerosis as an etiologic factor may be more of an association than causal.

Other causes of descending aortic aneurysms include cystic medial necrosis, chronic dissections, aortitis, and traumatic transection. Similar to aneurysms of the ascending aorta, patients with descending aortic aneurysms manifest progressive dilation and eventual rupture. Indeed, rupture may account for up to 50% of deaths in patients with atherosclerotic thoracic aneurysms.[26] Additional causes of aneurysmal dilation are related to gradual degenerative changes (loss of elastic fibers) in the medial layer of the aortic wall. These changes within the muscular layers of the aorta may be associated with inherited metabolic derangements (ie, Marfan syndrome and Ehlers-Danlos syndrome), acquired defects in the aortic media, annuloaortic ectasia, trauma, infections, mycotic conditions, syphilis, or idiopathic causes.[27] The natural history of TAA is quite diverse, reflecting the broad spectrum of causes.

As described in detail below, aortic size also has a substantial impact on aortic growth and is considered a major risk factor for complications (dissection and rupture). Aortic size is perhaps the single most important factor in the decision to intervene surgically on a nonemergent basis. Other risk factors important in influencing the risk of aortic growth and complications include significant hypertension, presence of a chronic dissection, and chronic obstructive pulmonary disease.[8,28]

Aortic medial degeneration has been shown to occur in most aneurysms, regardless of cause or location. The media is composed of smooth muscle cells within a matrix of structural proteins (elastin, collagen, and fibrillin) and extracellular matrix proteins, such as laminin, glycosaminoglycans,

proteoglycans, and fibronectin. These proteins are regulated in part by alterations in matrix metalloproteinases and their tissue inhibitors. The elastic fibers in the aortic wall are arranged in the media as circumferential lamellae. The thoracic aorta consists of 45 to 56 lamellar units (concentric elastic lamellae with smooth muscle cells, collagen, and ground substance); the abdominal aorta contains only 28 units.[29] The number of lamellar units is preserved throughout the mammalian species with the exception of the human abdominal aorta, which consists of fewer units than expected given the load it bears. Having fewer lamellar units may be a contributing factor in the prevalence of abdominal aortic aneurysms compared with aneurysms in the thoracic aorta.

Unexpected rupture of aortic aneurysms is almost uniformly fatal.[28] Appreciation for growth rates and risk factors for aneurysm growth and complications (dissection, rupture, and death) analyzed in vigorous statistical models enables the caregiver to better understand the natural history of the disease and to make evidence-based decisions regarding indications for therapeutic interventions and follow-up care.

Aortic Growth Rates

Exponential equations have been developed that permit rigorous, accurate calculation of growth rates from large populations of TAA patients.[30] These estimates were obtained for the entire population and for subgroups with specific risk factors by means of a multivariable regression analysis in which aneurysm growth followed an exponential path. In particular, the natural logarithm of the difference between the last measured size and the first measured size is related to the time interval between the 2 tests and interactions between this time variable and risk factors.

The descending thoracic aorta grows quite slowly, on average approximately 0.19 cm per year. Although slow-growing, descending thoracic aneurysms demonstrate more accelerated growth than seen in the ascending aorta and aortic arch, which grow at approximately 0.07 cm per year.[1] Thus, when TAA disease is discovered incidentally, the natural history of a TAA is one of progression, with slow, steady growth over years. If a complication such as an aortic dissection occurs in the setting of an aortic aneurysm, the aortic growth is significantly accelerated, with yearly growth rates as high as 0.28 cm per year.[10,13] Additional risk factors linked to accelerated aortic growth include advancing age, smoking, and progressive aortic size.[31]

Figure 2 shows overall estimated TAA growth rates in relation to initial aneurysm size. As the schematic demonstrates, annual growth varied from 0.10 cm per year for small (4.0 cm) aneurysms to 0.19 cm per year for large (8.0 cm) aneurysms. As the aortic size increases, the estimated rate of aortic growth is progressively higher. Familiarity with expected growth rates given an aortic size, and knowledge of risk factors influencing aortic growth, facilitates appropriate planning of longitudinal radiological follow-up studies.

Risk of Complications

With gradual dilation, the aortic wall becomes increasingly weakened, which leads to a higher risk of complications (dissection, rupture, and even death). The law of Laplace states that
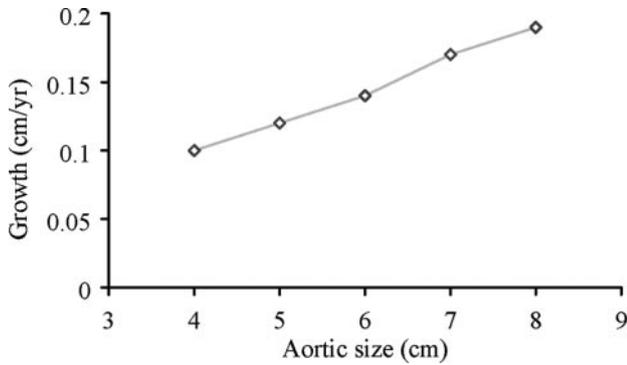
Figure 2. Absolute change in aortic growth rate as a function of size. Reprinted from Coady et al,[13] with permission from Mosby. Copyright 1997, The American Association for Thoracic Surgery.

as a cylinder increases in diameter, the wall tension also increases.[8] It also indicates that wall tension varies directly with the pressure in the lumen. Laplace's law is defined as:

$$T \approx P_t \times R/\mu,$$

where T is the circumferential wall tension, P_t is the transmural wall pressure, R is the radius of the vessel, and μ is the wall thickness. Because hypertension is correlated with larger aortic diameters, it has been considered a risk factor for the development of aneurysm rupture based on this law.

The incidence of adverse events (rupture and dissection) increases with faster growth rates, as illustrated in Figure 3. When the growth rate is less than 0.05 cm per year, the incidence of a negative event is 24% compared with 45% with a growth rate more than 0.20 cm per year.[13]

In an attempt to define the natural history of aortic disease, the Yale group[13] has provided lifetime observational evidence on asymptomatic patients with aortic aneurysms that demonstrates a rising incidence of aortic dissection or rupture with expanding aneurysm size. The median size at the time of rupture or dissection in their series was 6.0 cm for ascending aneurysms and 7.2 cm for descending aneurysms.[13] Multivar-

iate regression analysis to isolate risk factors for acute dissection or rupture revealed that the probability of these events was increased by 32.1 percentage points for ascending aneurysms >6.0 cm in size and by 43.0 percentage points for descending aneurysms >7.0 cm in size (Figure 4). The regression curves in Figure 4 represent the lifetime cumulative risk of aortic rupture or dissection starting at a particular aortic size.

In Figure 4, discrete hinge points are seen in the curves for aneurysm diameter and risk for dissection or rupture. For the ascending aorta, the hinge point occurs at 6 cm. Relative to the 4.0- to 4.9-cm TAA cohort, the probability of incurring a dissection or rupture is 32.1 percentage points higher in the 6.0- to 6.9-cm cohort ($P<0.01$). This difference is a serious statistic, expressing once again the acerbity of this disease over the course of time. Similarly, the rate of dissection or rupture increases by 43 percentage points in descending TAAs ≥7 cm in diameter ($P<0.01$). For the descending aorta, the hinge point occurs at 7 cm.

Note that the descending aorta does not rupture or dissect until a somewhat large size is attained. This finding is not intuitive, because the descending aorta has fewer lamellae than the ascending aorta and is normally smaller in its nonaneurysmal state.[32] We would expect the descending aorta to rupture earlier, but this expectation does not appear to be the case. Fluid dynamics, flow patterns, wall stress, and structural differences may underlie this observation.

With increasingly robust data, predictions have been made of specific yearly dissection, rupture, and mortality rates for each size of the thoracic aorta. Figure 5 demonstrates that the risk of rupture, dissection, and death increases in a stepwise fashion as the aorta grows, with the biggest step-up in risk being seen at the critical aortic dimension of 6 cm. Beyond this size, the rate of complications increases exponentially. At 6 cm, the yearly risk of rupture and dissection is approximately 4%; the rate of death is approximately 12%, and the combined risk of rupture, dissection, or death is approximately 16% annually. These data should be taken into account when a patient with an aneurysm is being followed up.
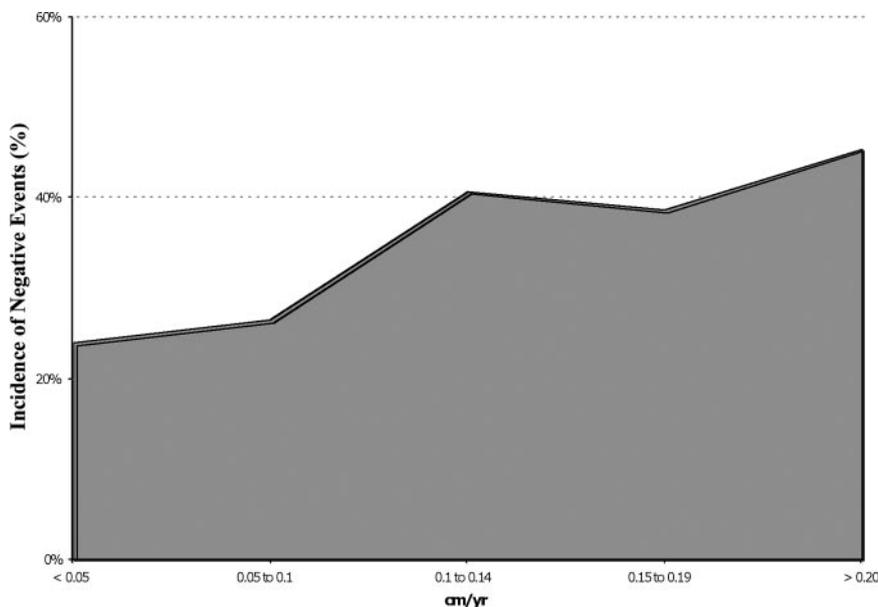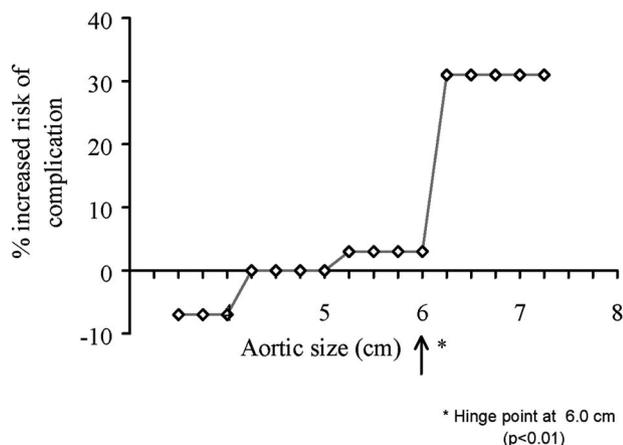

Figure 3. The incidence of negative events (rupture, acute dissection, and death) as a function of growth rate. Reprinted from Coady et al,[31] with permission from Informa Healthcare.

A Regression Analysis for the Ascending Aorta

B Regression Analysis for the Descending Aorta

* Hinge point at 6.0 cm
(p<0.01)

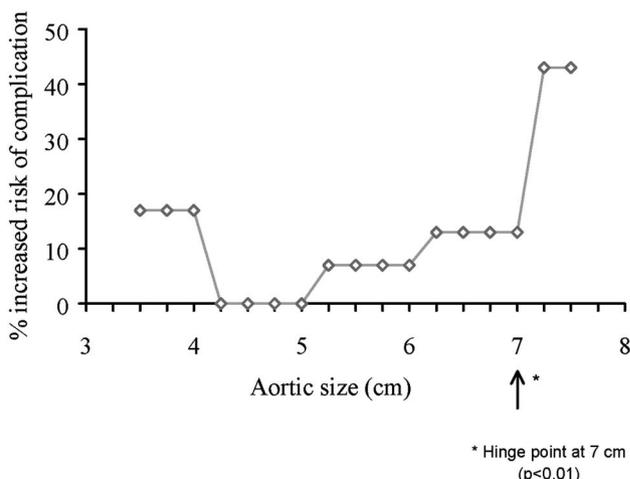* Hinge point at 7 cm
(p<0.01)

Figure 4. Regression analysis for the ascending and descending aorta. Reprinted from Coady et al,[13] with permission from Mosby. Copyright 1997, The American Association for Thoracic Surgery.

Defining Surgical Intervention Criteria

In the development of management protocols for appropriate patient selection for surgery, it is essential to study risk factors that may influence the natural history of the disease. The specific objective is to select patients for whom the operative risks are justified. The hinge points illustrated in Figure 4 isolate critical size thresholds for aortic aneurysms by location in a large observational series of patients. These data strongly support size criteria for preemptive surgical replacement of the aneurysmal aorta to prevent the complications of rupture or dissection. Intervention criteria that select a safer aortic size below these hinge points facilitated size recommendations. For the descending thoracic aorta, 6.5 cm will preempt most ruptures or dissections. Smaller size criteria are applied to patients with a family history of connective tissue disorders such as Marfan syndrome, because aortic dissections in these individuals are likely to present at smaller aortic diameters.

Figure 6 summarizes current recommendations for surgical intervention in patients with TAA. Contained rupture and symptomatic states are clear-cut indications for intervention, as are documented aneurysmal growth of >1 cm per year and selected cases of aortic dissection.[13,33] Symptomatic patients should be treated regardless of aortic size if there are no contraindications, because symptoms often portend rupture.[13] Clearly, however, the decision for surgery must carefully balance the risks with the potential benefits.

Aortic Size

Female sex has been identified as a significant predictor of negative events, in particular the combined end point of rupture or dissection.[34] This observation may well be due in part to differences in mean body size between males and females, with a given aortic dimension representing a proportionally greater diameter in smaller women. In 2006, Davies et al[34] proposed an aortic size index that takes into account both the aortic diameter

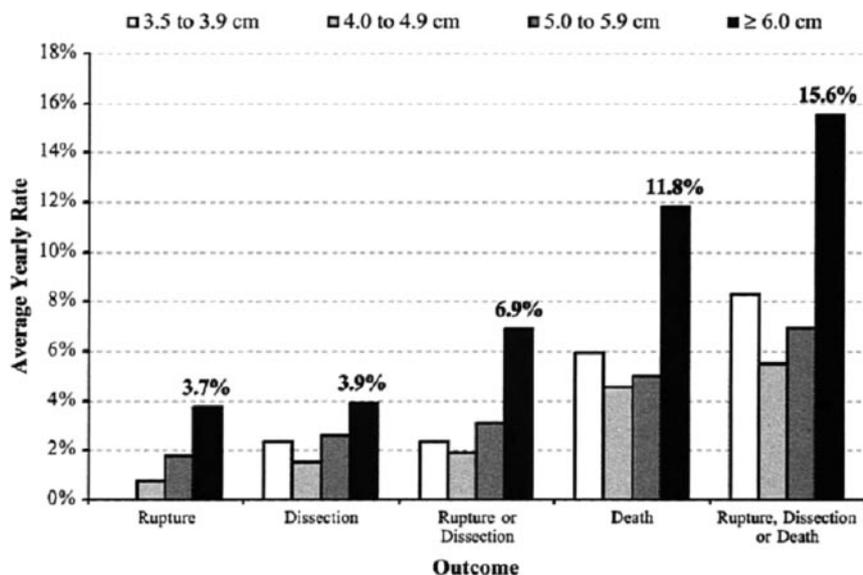□ 3.5 to 3.9 cm   ▨ 4.0 to 4.9 cm   ▨ 5.0 to 5.9 cm   ■ ≥ 6.0 cm

Figure 5. Yearly risk of complications. Average yearly rates of negative outcomes (rupture, dissection, and death). These estimates represent the average rate during the first 5 years after presentation. Reprinted from Davies et al,[1] with permission from Elsevier. Copyright 2006, The Society of Thoracic Surgeons.

1. Rupture

2. Symptomatic states

    a. Pain consistent with rupture and unexplained by other causes.

    b. Compression of adjacent organs, especially trachea, esophagus, and

       left main stem bronchus.

    c. Significant aortic insufficiency in conjunction with ascending aortic aneurysm

3. Absolute size

	Marfan's syndrome patients*	Non-Marfan's syndrome patients
Ascending	5.0 cm	5.5 cm
Descending	6.0 cm	6.5 cm

Figure 6. Recommended surgical intervention criteria for TAAs. *The Marfan intervention criteria should also apply if there exists a family history of aortic disease other than Marfan syndrome. Reprinted from Coady et al,[13] with permission from Mosby. Copyright 1997, The American Association for Thoracic Surgery.

4. Documented enlargement

    a. Growth ≥ 1 cm/yr or substantial growth, and aneurysm is rapidly

       approaching criteria in number 3 above.

5. Acute aortic dissection

    a. Ascending requires urgent operation

    b. Descending requires "complication-specific approach"[33]

and body surface area (Figure 7). The aortic size index has been shown to be a better predictor of negative events than maximal aortic diameter. Figure 7 stratifies patients into 3 categories of risk based on aortic size index. This risk stratification is useful in counseling individual patients regarding the risks of nonoperative management of aneurysms. Surgery is advocated before a patient enters the zone of moderate risk with an aortic size index >2.75 cm/m^2.

Implications for Stent Grafting

There is general agreement regarding anatomic constraints for stent grafting in descending TAAs. These limitations include landing (sealing) zones of aorta of ≥1.5 cm in length distal to the left subclavian artery (or left common carotid artery if the left subclavian artery is to be euthanized) and proximal to the celiac axis.[18] Straighter segments of aorta are

stented more successfully than acutely angled segments, especially if the angle of curvature around the aortic arch exceeds 60°. Furthermore, access through the femoral and iliac arteries is facilitated by a vessel diameter >7 mm, depending on the specific device used.[20]

Figure 6 reviews the recommended size criterion for open resection of TAAs. As mentioned previously, although the feasibility of stent grafting for descending TAA has been firmly established, the indications for intervention remain to be fully defined. The literature is replete with small individual series. The summarized results of endovascular repair are condensed in Table 1.[11,35–40] Note that these studies included limited numbers of patients and relatively modest follow-up periods.

The initial report of stent grafts for treatment of TAAs was pioneered in 1994 by Dake et al[35] at Stanford. This group used custom-designed, homemade grafts for each patient

	Aortic Size (cm)									
	3.5	4.0	4.5	5.0	5.5	6.0	6.5	7.0	7.5	8.0
BSA										
1.30	2.69	3.08	3.46	3.85	4.23	4.62	5.00	5.38	5.77	6.15
1.40	2.50	2.86	3.21	3.57	3.93	4.29	4.64	5.00	5.36	5.71
1.50	2.33	2.67	3.00	3.33	3.67	4.00	4.33	4.67	5.00	5.33
1.60	2.19	2.50	2.80	3.13	3.44	3.75	4.06	4.38	4.69	5.00
1.70	2.05	2.35	2.65	2.94	3.24	3.53	3.82	4.12	4.41	4.71
1.80	1.94	2.22	2.50	2.78	3.06	3.33	3.61	3.89	4.17	4.44
1.90	1.84	2.11	2.37	2.63	2.89	3.16	3.42	3.68	3.95	4.22
2.00	1.75	2.00	2.25	2.50	2.75	3.00	3.25	3.50	3.75	4.00
2.10	1.67	1.90	2.14	2.38	2.62	2.86	3.10	3.33	3.57	3.80
2.20	1.59	1.82	2.05	2.27	2.50	2.72	2.95	3.18	3.41	3.64
2.30	1.52	1.74	1.96	2.17	2.39	2.61	2.83	3.04	3.26	3.48
2.40	1.46	1.67	1.88	2.08	2.29	2.50	2.71	2.92	3.13	3.33
2.50	1.40	1.60	1.80	2.00	2.20	2.40	2.60	2.80	3.00	3.20

Figure 7. Risk of complications by aortic diameter and body surface area (BSA), with aortic size index given within chart. The white area indicates low risk (≈4%/ y); light gray area, moderate risk (≈8%/ y); and dark gray area, severe risk (≈20%/y). Reprinted from Davies et al,[34] with permission from Elsevier. Copyright 2006, The Society of Thoracic Surgeons.

Table 1.   Summary of Endovascular Repair in TAAs

	Authors						
	Dake et al[35]	Cambria et al[36]	Czerny et al[37]	Demers et al[11]	Midorikawa et al[38]	Marcheix et al[39]	Czerny et al[40]
Year	1994	2002	2004	2004	2005	2006	2007
Patients, n	13	18	54	103	45	45	79
30-Day death, %	0	5.6	3.7	9	0	6.7	6.3
Technical success, %	100	100	94.4	73	96	N/A	100
Paraplegia, %	0	0	0	3	0	4.4	N/A
CVA, %	0					13.3	5
Vascular access comp, %						8.9	
Endoleak, %	30.8	21	28.9	21	13	42	23
Mean follow-up	1.6 mo	17 mo	38 mo	4.5±2.5 y	48 mo	24.7±21.6 mo	42 mo

N/A indicates not applicable; CVA, cerebrovascular accident; and comp, complication.

constructed of self-expanding stainless steel stents covered with woven Dacron grafts. This initial series of 13 patients had 100% technical success, with no death, paraplegia, stroke, or distal embolization. Type I endoleaks were seen in 30.8% of patients, which indicated a poor seal between the aorta and the proximal or distal attachment sites of the stent graft. Because of these promising early results, the authors advocated the use of stent grafts in highly selected patients.[35]

Subsequent studies included a subset of patients with aortic aneurysms and confirmed the feasibility and relative safety of endovascular management of aneurysmal disease.[29–35] These series stressed the importance of patient selection and the necessity of adequate anatomic targets. In the study by Cambria et al,[36] access-artery complications occurred in 21% of patients, and 1 of these was fatal. That report stressed the need for safety precautions during stent-graft placement, including general anesthesia, arterial lines, and an operating room with proper equipment required for an expedient conversion.

In 2004, Demers et al[11] reported their midterm results of endovascular repair of descending TAAs with first-generation stent grafts, which included 103 patients. Sixty percent of these patients were unsuitable candidates for conventional open surgical repair (inoperable). Overall actuarial survival was 82%, 49%, and 27% at 1, 5, and 8 years, respectively. Survival in candidates for open surgical repair was 93% and 78% at 1 and 5 years, respectively, compared with 74% and 31% in those deemed inoperable ($P<0.001$; Figure 8). Independent risk factors for death were older age, previous stroke, and being designated an inoperable candidate. Survival was deemed satisfactory in good-risk candidates but bleak in the inoperable cohort, which raises the question of whether asymptomatic patients should have even been treated. Eleven of the 103 patients had late aortic rupture in the stented segment. Rupture was fatal in 10 of those patients. Endoleaks were common, most of which were type I. The authors concluded that stent grafting of descending TAAs should not be offered to young patients who lack contraindications to open repair. Careful selection of patients for stent grafting is key, with particular emphasis placed on identification of favorable anatomic targets, symptom status, and projected life expectancy. Given the sobering dismal life expectancy of inoperable patients treated with stent grafting, one should consider medical management in these patients.

Aortic aneurysm formation and growth in the descending thoracic aorta is a relatively indolent disease process for smaller aneurysms; however, the natural history of untreated patients with TAAs is characterized by gradual but progressive expansion and eventual rupture. As illustrated in Figure 5, rates of rupture, dissection, and death are low in small-diameter descending thoracic aneurysms, with risks increasing progressively with increasing aortic size. Figure 6 summarizes the recommended surgical intervention criteria for TAA. Open repair remains the gold standard approach for the majority of patients.

The presence of many medical comorbidities in the descending thoracic aneurysm patient population increases the surgical risks, especially in cases of emergency operation for aortic rupture. An endovascular approach to the treatment of descending TAAs was developed in the early 1990s as a less invasive alternative to conventional open-surgery graft replacement. In early reports, stent-graft treatment was typically reserved for poor candidates for open surgical repair (Table 1).[35] There have been encouraging early-term results and increasing enthusiasm for this new technology. A recent report of a multicenter, prospective, phase II clinical trial comparing endovascular treatment of descending thoracic aneurysms with the TAG device with control subjects who underwent standard open surgical repair demonstrated superior results with TAG treatment in anatomically suitable patients after 5 years of follow-up.[41]

A small, asymptomatic TAA should not be stent grafted prophylactically upon identification. Because of the relative ease of stent grafting, descending TAAs have been stent grafted at smaller aortic sizes than the diameter at which open operation has been deemed necessary or indicated conventionally. It is not clear that this trend toward more aggressive stent grafting will influence overall prognosis or provide improved long-term survival and freedom from complications compared with medical management or open surgical repair.[10] Long-term follow-up data are not available currently and will be an important component of decision making for the treatment of these patients.
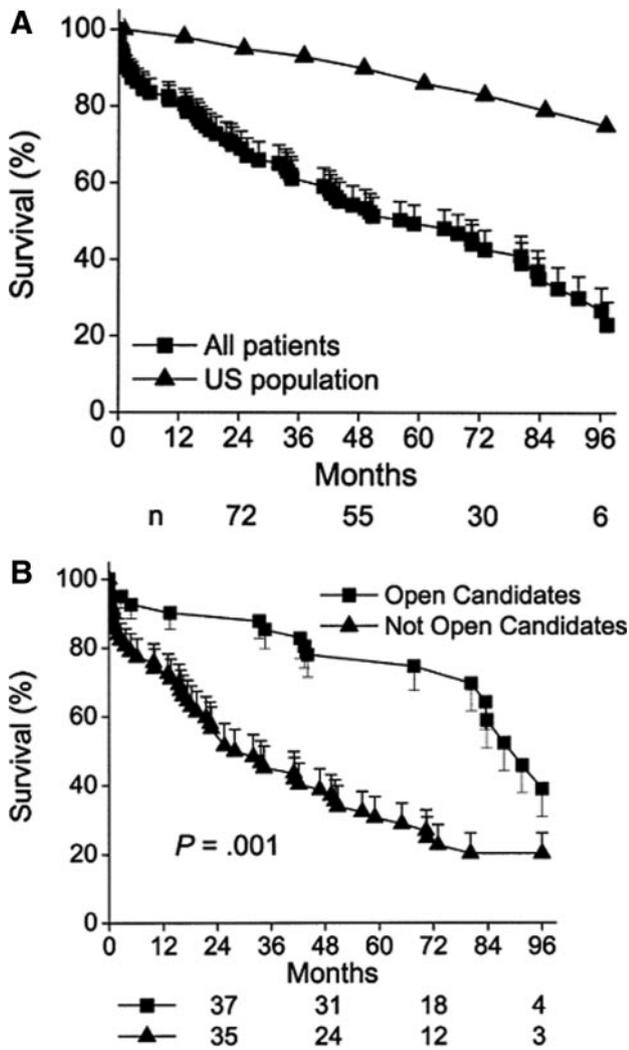
Figure 8. Survival in candidates for open surgical repair compared with those deemed inoperable. Reprinted from Demers et al,[11] with permission from Mosby. Copyright 2004, The American Association for Thoracic Surgery.

Until longer follow-up data in more patients become available to show definitively just how durable stent grafting with new commercial devices is, it is not prudent to offer endovascular stent-graft repair to younger patients who do not have major contraindications to open surgical repair.[11] Careful selection of patients for endovascular stent grafting is key, with particular emphasis placed on the identification of favorable anatomic targets, symptom status, and projected life expectancy. The indication for stent grafting of descend-

ing TAAs should be predicated on a procedural risk that is lower than the risk of conventional open repair or medical management. As mentioned previously, patients with connective tissue disorders, such as Marfan syndrome, should not be treated with endovascular stent grafts because of their thin, weakened aortic walls, which theoretically predispose them to a high likelihood of developing a false aneurysm or endoleak at the proximal or distal landing zones.[11] Lastly, elderly patients with asymptomatic aneurysms who are not candidates for open operative repair should be given serious consideration for medical management because of the very poor reported survival rates after endovascular repair.

Descending Aortic Dissections

Acute Descending Aortic Dissections

Acute aortic dissection is the most common catastrophe of the aorta, with an incidence of 5 to 30 cases per million people per year. Since the first description of aortic dissection in the 1700s, the understanding and treatment of this catastrophic disease has evolved. Men experience acute aortic dissection twice as much as women, with 40% of all dissections presenting as an acute descending thoracic aortic dissection (Stanford type B). A Stanford type B (or DeBakey type III) dissection begins with a laceration of the aortic intima and inner layer of the aortic media just distal to the attachment of the ligamentum arteriosum in the descending aorta, which allows blood to course freely along a false lumen in the outer third of the media. This intimal tear occurs at the area of greatest hydraulic stress and emphasizes the significant impact that flexional forces have on the aortic wall. The result is a dissection flap, which separates the aorta into true and false lumina. The misnomer "dissecting aortic aneurysm" has frequently been applied to this condition, but dilation does not generally occur in the acute setting. Therefore, we prefer the term "aortic dissection" and reserve the term "aortic aneurysm" for those situations in which dilation of the aorta occurs. Clinically, dissections identified within 2 weeks of the onset of symptoms are termed acute; subsequently, they are classified as chronic.

An artist's rendition of a typical aortic dissection is presented in Figure 9, and other aortic pathologies (PAU and IMH) are illustrated for comparison. The artist's rendition corresponds well to the operative findings discovered by the aortic surgeon. The flap traverses the aortic lumen obliquely in cross-sectional views; it is not oriented circumferentially.

Understanding the pathogenesis of aortic dissection requires consideration of the inciting event that causes the intimal-medial tear and the propagation of blood within the aortic media.
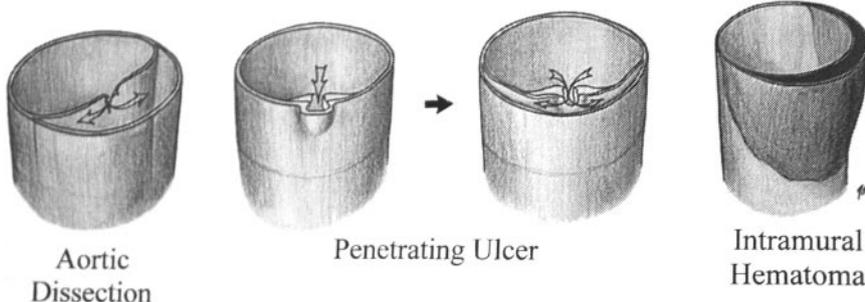
Figure 9. Aortic dissection, IMH, and PAU. Note the distinct intimal flap separating the true and false lumens in aortic dissection. An IMH, possibly caused by rupture of the vasa vasorum, leads to a concentric hematoma within the media and has no intimal tear. PAUs burrow deeply through the intima into the aortic media, precipitating a localized intramedial dissection associated with a variable amount of hematoma. Reprinted from Coady et al,[2] with permission from Elsevier. Copyright 1999, Elsevier.

Although various risk factors that predispose the aorta to dissection have been described well, the precise insult that leads to laceration of the intima and media remains unclear.

Historically, the primary causative event that leads to aortic dissection has been extremely controversial. Cystic medial necrosis associated with Marfan disease and other connective tissue disorders was once believed to contribute to aortic medial degeneration, leading to aortic dissection. Larson and Edwards,[42] however, demonstrated that only a few of their 161 patients with known aortic dissection exhibited medial degeneration. They found that 158 of these patients had intimal aortic tears at autopsy, which supports the theory initially proposed by Murray and Edwards[43] that the intimal tear is the primary event, allowing the blood to spread through the outer two thirds of the aortic media. Cystic medial necrosis is no longer regarded as the common structural disorder underlying aortic dissection.

Degenerative changes within the media itself, however, can affect its elastic constituents. Loss of these elements reduces the resistance of the aortic wall to hemodynamic stress, which leads to significant aneurysmal dilation of the vessel with the subsequent develop of a dissection. Indeed, increasing aortic diameters associated with TAAs have been associated with an increased risk of aortic dissection (Figures 3 and 5). In the dilated aorta, the stress that leads to aortic dissection is composed of a radial and a longitudinal vector. As the aorta becomes increasingly more aneurysmal, the longitudinal vector becomes the more significant force. This explains the typical occurrence of a transverse tear along the course of the aortic wall.

Inheritable disorders of elastic tissues, including Marfan, Turner, Noonan, and Ehlers-Danlos syndromes, also predispose to the development of aortic dissections.[2] Of these, Marfan syndrome is the most common and best understood. Other known risk factors for the development of aortic dissection include pregnancy, inflammatory diseases of the media, blunt chest trauma, and iatrogenic causes, namely, injury during cardiac catheterization or placement of an intra-aortic balloon pump.[45,46]

Significant derangement and loss of elastic tissue are common in younger patients with aortic dissection, especially those with congenital defects; however, this finding is not seen routinely in older patients.[47] Some investigators believe that a loss of smooth muscle cells, often appearing in a laminar pattern, is the predominant factor that leads to aortic dissection in patients older than 40 years of age.[47]

Because degenerative changes do not fully explain the occurrence of a dissection, various mechanical forces have also been considered. These include flexional forces of the vessel at fixed sites, the radial impact of the pressure pulse, and the shear stress of the blood. The heart, ascending aorta, and aortic arch are relatively mobile, whereas the descending aorta is suspended over the spinal column. During the cardiac cycle, the heart and aorta produce rhythmic movements, and this allows all but fixed segments to move. These fixed points are exposed to the most significant flexional forces. Classic type A and B aortic dissections may produce an intimal tear at the areas of greatest hydraulic stress, the right lateral wall of the ascending aorta or the descending thoracic aorta in proximity to the ligamentum arteriosum. These are the most common sites of aortic dissection, which emphasizes the significant impact that flexional forces have on the aortic wall.

The majority of patients who present with an acute type B aortic dissection have hypertension. Hypertension and increased aortic blood pressure alter the slope of the pulse pressure wave and have been shown to add a mechanical strain on the aortic wall, which leads to deposition of additional elastic lamellae.[48] There is also an increased propensity for delamination of the aortic wall because of the shear forces that exert a longitudinal stress along the aortic wall.

At one time, it was also thought that atherosclerosis was the cause of acute aortic dissections. Among the earliest proponents of this theory were Virchow (1851), Girode (1887), Ewald (1890), Rolleston (1893), and von Moller (1906).[49] In a classic paper entitled "Dissecting Aneurysms," Shennan in 1934 began to recognize that although nodular atheromas were present in a large number of aortic dissections and ruptures, only a few displayed a relationship of the atheroma to the exact location of primary rupture or dissection.[49] Only 6 of 218 dissections in his study demonstrated evidence of a dissection at the base of an atheromatous ulcer.

Today, most authorities believe that atherosclerosis does not cause aortic dissections.[50,51] Indeed, the majority are found in the ascending aorta, where atherosclerosis is less common; in the descending aorta, gross atherosclerosis usually limits the dissection because of neighboring fibrosis and calcification. Thus, in the descending aorta, the 2 entities coexist.

Patients with type B aortic dissections are older, are hypertensive, and have a much higher burden of atherosclerotic disease, which can involve all vascular beds, including the coronary arteries.[2] In the acute setting, the majority of patients with uncomplicated type B dissections typically do very well with medical management, unlike those with type A dissections. An uncomplicated acute type B aortic dissection is less frequently lethal, with survival rates of 89% in medically treated patients at 1 month and 84% within 1 year.[52] Initial treatment consists of invasive hemodynamic monitoring, β-blockade, and pulsatile force reduction. Traditional surgical intervention is reserved for complications of the disease.

Medical management of patients with an acute uncomplicated type B dissection can be justified because experience has demonstrated that attentive medical therapy is effective for stabilizing the acute process and preventing rupture in the majority of cases. Operative mortality and complication rates have traditionally been much higher with acute type B aortic dissections.[53] Unfortunately, long-term outcomes with medical therapy alone are suboptimal, with a mortality rate approaching 30% to 50% at 5 years and a delayed expansion of the false lumen in 20% to 50% of patients at 4 years.[52] Therefore, these patients require conscientious long-term follow-up. Once the aorta is dissected, the aortic growth rate increases exponentially, which leads to progressive aneurysmal dilation.[8] The growth rate has been reported to be as high as 0.31 cm per year.[54] Patients with acute or late complications, including renal failure, visceral ischemia, or contained rupture, often require urgent repair, with a mortality rate that rises to 20% at day 2 and 25% to 50% within 1 month.[52]

Traditional indications for surgery in patients with acute type B dissection are limited to prevention or relief of life-threatening complications, such as rupture, ischemia of limbs and end organs, persistent or recurrent intractable pain, progression of the dissection during medical management, and uncontrolled hypertension. Signs of impending rupture include recurrent pain and falling hematocrit or CT evidence of rapid aortic expansions or rapidly accumulating pleural effusion. Spinal cord ischemia occurs in 2% to 10% of patients with type B dissections as a consequence of interruption of intercostal vessels. Acute paralysis is not a contraindication for surgery, particularly if femoral pulses are absent. Rapid relief of distal ischemia often results in a return of motor and sensory function.[55]

In patients presenting with evolving complications such as signs of imminent rupture, expansion, retrograde dissection, or a malperfusion syndrome, surgery for acute type B aortic dissection carries a 14% to 67% risk of irreversible spinal injury or postoperative death.[56]

Open Repair and Implications for Stent Grafting

Treatment goals for both open and endovascular approaches are to reduce the dissection-related risk of death and limit the extent of aorta repaired, to minimize morbidity. The principles of surgical repair are to exclude the proximal primary intimal tear, remove or exclude all aneurysmal disease, and maintain perfusion to all distal organs and major aortic branches.[10] Both open surgical and endovascular stent-graft treatment may slow the disease, but neither reverses its natural history unless the entire extent of dissection is resected or excluded by surgical resection. Regardless of the approach used, patients with residual dissected aorta remain at risk for late aneurysmal degeneration and rupture of the dilated lumen. Keys to therapy include strict blood pressure control, minimization of pulse pressure, and indefinite continuation of medical therapy.

Given the reasonable results with medical management for uncomplicated type B dissections, medical therapy constitutes a gold standard that is difficult to surpass with surgery. Historically, the rate of death for patients with type B dissections has been 10.7% for those undergoing medical treatment and 31.4% for those treated with surgery.[56] In the emergent setting, 25% to 50% of patients have persistent false-lumen flow, and surgeons have had variable success in relieving distal malperfusion. The risk of irreversible spinal cord injury and operative death for acute type B dissections can range from 14% to 67%.[57,58]

Endovascular repair is developing as a strong alternative to surgery and may eventually evolve as a superior method for definitive treatment for patients with appropriate indications (complicated dissections), as discussed above. Intuitive advantages include the ability to obliterate the false lumen by sealing the aortic tear with an aortic endograft. Among patients with acute type B aortic dissection, more than 60% of associated deaths are due to local rupture, usually of the false lumen. Continued patency of the false lumen has been reported to lead to aneurysmal dilatation. Even if partial thrombosis of the false lumen is all that is achieved, the endograft may still protect the false lumen from enlarging over time.[59]

Table 2.   Therapeutic Strategies in Thoracic Aortic Dissections

Medical
Uncomplicated, acute type B aortic dissection
Chronic type B aortic dissection
Stable, isolated aortic arch dissection
Surgery
Type A aortic dissection (involvement of ascending aorta)
Acute type B dissection complicated by:
Retrograde extension into the ascending aorta
Dissection in collagen-vascular disorders (Marfan syndrome; Ehlers-Danlos syndrome)
Interventional
Unstable, acute type B dissection
Malperfusion
Rapid expansion (>1 cm/y)
Critical diameter ($\geq$5.5 cm)
Refractory pain
Stable type B dissection (under current evaluation: INSTEAD and ADSORB trials)
Type B dissection with retrograde extension into the ascending aorta
Hybrid procedure for extended type A aortic dissection

Adapted from Akin et al.[52]

Considerations for Stent Grafting in Type B Dissections

Aside from medical therapy, management of type B aortic dissections is dictated by the presence of complications such as end-organ malperfusion, intractable pain, impending rupture, and chronic events (continued false-lumen expansion >4.5 to 5 cm with risk of delayed rupture). Patients who present with visceral ischemia and undergo a traditional open aortic repair have an associated surgical mortality rate of 50% to 70%.[20] With improved imaging, high-resolution CT scanning allows for identification of the mechanism of malperfusion, which aids in decision making for stent grafting. Dynamic obstruction is cause by compression of the true lumen by the pressurized false lumen; conversely, static obstruction is cause by local intimal tears at branch-vessel orifices.[20] This distinction is critical, because most dynamic obstructions are relieved by stent-graft coverage of the primary intimal tear via a reduction of flow in the false lumen, whereas coverage of the primary intimal tear in static obstructions is successful in only approximately 40% of cases, necessitating further interventional techniques, that is, septal fenestration or orifice stenting.[20] Malperfusion can be assessed immediately in the catheterization laboratory.

The feasibility of stent grafting for dissections of the descending thoracic aorta has been well established since the late 1990s as a supplemental treatment and a true alternative to classic high-risk surgical treatment; however, because there are no randomized, prospective clinical trials with substantial follow-up, the indications for stent grafting are still in the process of evolution. Table 2 categorizes the current therapeutic strategies for treatment of thoracic aortic dissections.

There is clear observational evidence that depressurization and shrinkage of the false lumen are beneficial in acute type B aortic dissections, with the goal of thrombosis of the false

lumen and remodeling of the dissected aorta.[52] Similar to previously accepted indications for surgical intervention, refractory pain, malperfusion, expansion >1 cm per year, and a critical diameter of ≥5.5 cm are increasingly being accepted as indications for stent-graft placement in type B aortic dissections.[52] Stent placement has been used to treat retrograde extension of a type B dissection into the ascending aorta, because coverage of the entry site may enable thrombosis and remodeling of the false lumen, and even healing. If malperfusion of a branch vessel persists, endovascular revascularization by septal fenestration or branch-vessel stenting can be performed. Alternatively, the PETTICOAT (provisional extension to induce complete attachment) technique extends the primary stent graft distally with open bare-metal stents to correct residual distal malperfusion.[60]

Patients who present with an unstable type B aortic dissection manifesting renal or mesenteric ischemia have an operative mortality rate of 50% and 88%, respectively.[52,61] The International Registry of Acute Aortic Dissection currently represents 21 large referral centers from around the world with consecutively enrolled patients presenting with acute type B aortic dissection. Early data from this registry of aortic dissections suggested significant differences with respect to in-hospital death stratified by type of treatment for patients with acute type B aortic dissections. The registry reported an in-hospital mortality rate of 32% for those treated with surgery, 7% for those managed with endovascular techniques, and 10% for those managed with medical therapy alone ($P<0.0001$).[62] These results have been confirmed by subsequent studies.

Fattori et al[63] compared the impact of different treatment strategies on survival in 571 patients with acute type B aortic dissections. Of the 571 patients with acute type B aortic dissection, 390 (68%) were treated medically; among the complicated cases, 59 (10%) underwent standard open surgery, and 66 (12%) were treated with an endovascular approach. In-hospital complications occurred in 20% of patients treated with endovascular techniques and in 40% of patients after open surgical repair. The in-hospital death rate was significantly higher after open surgery (33%) than after endovascular treatment (11%; Figure 10). After propensity and multivariable adjustment, open surgical repair was associated with an independent increased risk of in-hospital death (odds ratio 3.41, 95% confidence interval 1.00 to 11.67, $P=0.05$). Thus, although long-term data are not available, stent-graft repair is emerging as an attractive alternative to open surgical repair for patients with ischemic complications.

In 2008, Parker and Golledge[64] presented a meta-analysis of outcomes data for endovascular treatment of acute type B aortic dissections. The study included 942 patients from 29 independent studies over the previous decade. The in-hospital mortality rate was found to be 9%, and other major complications (stroke 3.1%; paraplegia 1.9%; conversion to type A dissection 2%; bowel infarction 0.9%; and major amputation 0.2%) totaled 8.1%. Aortic rupture was 0.8% over a 20-month period. The study concluded that endovascular treatment of complicated acute type B aortic dissection appears to provide favorable initial outcomes, and although data on long-term outcomes are required, it provides an important treatment option.[64]
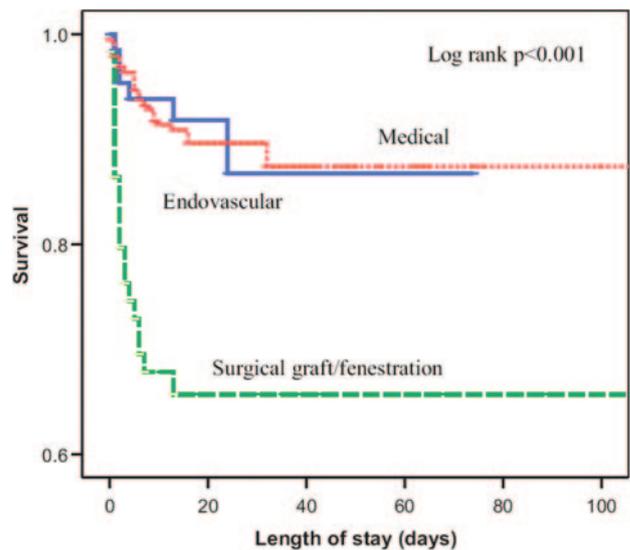

Figure 10. Comparison of various treatments in patients with complicated acute type B aortic dissections. Reprinted from Fattori et al,[63] with permission from Elsevier. Copyright 2008, The American College of Cardiology Foundation.

The initial experience with stent grafting for type B aortic dissections was reported by Dake and colleagues at Stanford University (Table 3).[65] In this early experience, among 15 patients with type B dissections, 3 patients died of procedure-related events. Two had rupture of the false lumen of the distal aorta and later died; 1 patient died of sepsis associated with infarction of the gut. Technical success was achieved in all patients, with complete false-lumen thrombosis in 80% of patients.

Subsequent series are presented in Tables 3 and 4. Nienaber et al[72] published a prospective study contemporaneously with the initial report by Dake et al.[65] Nienaber et al[72] evaluated the safety and efficacy of elective transluminal endovascular stent graft insertion in 12 consecutive patients with type B aortic dissections and compared the results with surgery in 12 matched control subjects. There were 4 deaths in the surgical group (33%) and 5 adverse events (42%) within 12 months. The stent-graft group had no death or major adverse events (deaths, paraplegia, stroke, embolism, side-branch occlusion, or infection). Complete thrombosis of the false lumen was evident on imaging studies at 3 months. The authors demonstrated that endovascular treatment was safe and efficacious in patients for whom surgery was indicated, and they reported a lower overall mortality and morbidity rate than with open repair. Although these results are promising, the authors stressed the importance of a randomized long-term study. Additional results of endovascular repair in complicated acute type B thoracic aortic dissections are presented in Table 3. Note the low risk of paraplegia and death and the overall favorable technical success.

Dissections likely represent one of the greatest opportunities for the use of stent grafting in patients with complicated type B aortic dissections, as seen in the results compiled in Table 3. It must be remembered, however, that in the acute setting, the dissected vessel is fragile, and injury during stent-graft insertion is an easily imagined complication of unknown magnitude. The

Table 3. Summary of Endovascular Repair in Acute Complicated Type B Thoracic Aortic Dissections

	Authors						
	Dake et al[65]	Bortone et al[66]	Beregi et al[67]	Leurs et al[68]	Eggebrecht et al[69]	Chen et al[70]	Szeto et al[71]
Year	1999	2002	2003	2004	2006	2006	2008
Patients, n	15	7	39	131	609	62	35
30-Day death, %	20	0	17	8.4	11.2	4.8	2.8
Technical success, %	100	100	96	88.6	98	100	97.1
Paraplegia, %	0	0	0	0.8	0.8	0	2.8
Endoleak, %	20	0	10	6		0	5.7
Complete false lumen							
Thrombosis, %	80*	100			75.5±2.4	95.7†	
Partial false lumen							
Thrombosis, %	20			14			40
Mean follow-up, mo	13	9.7±8.5	8	12	19.5±7.1	26.4±14.6	18.3

*100% Complete false lumen thrombosis at 3 months achieved.

†Refers to both complete and partial thrombosis in all patients (acute and chronic).

ability to cover the primary intimal tear is also an issue, especially for large tears adjacent to the left subclavian artery.[20]

Chronic Descending Aortic Dissections

An aortic dissection is classified as chronic after the patient survives a total of 2 weeks after the initial incident. This group includes those treated emergently with surgery for complications and the majority who are managed medically. Some patients present who have never seen a physician and who were undiagnosed and untreated during the initial 2-week period.

Chronic Dissection After Proximal Repair

Most type A dissections (DeBakey types 1 and 2) extend distally beyond the left subclavian artery to involve the descending thoracic and abdominal aorta. A residual distal dissected aorta with flow in the false lumen is detected in 63% of patients and is not predictive of late survival.[10] Once the proximal aorta is repaired, patients who are left with a distal dissection have similar survival to those who initially present with type B dissection. Thus, the management implications are similar to those for patients with chronic type B aortic dissections. These individuals need long-term, careful follow-up.

Chronic Type B Dissections

Most deaths are related to comorbid conditions, but late deaths are linked to the distal aortic dissection and occur in 20% to 50% of patients. Problems include development of a new dissection, rupture of the false lumen, and aneurysmal degeneration of the thinned walls of the false channel, which can lead to rupture and exsanguination. With a chronic aortic dissection, there is progressive thickening of the intimal flap due to fibrosis. The aortic growth in a chronic aortic dissection is also accelerated because of the frequency of patent false lumens. The yearly growth in this setting can be as high as 0.28 cm per year.[10,13]

The long-term outcome with medical therapy alone is suboptimal, with a reported mortality rate of 50% at 5 years and delayed expansion of the false lumen in 20% to 50% of patients at 4 years.[52] This expansion of the false lumen, for which an initial diameter of more than 4 cm and a persistent perfusion of the false lumen have been determined as predictors, predisposes patients to aortic rupture or retrograde progression of the dissection to the ascending aorta, with an increased mortality rate.[75] Table 2 lists considerations for indications for endovascular treatment of chronic aortic dissections as proposed by Akin et al[52]: Diameter ≥5.5 cm and expansion >1 cm per year.

Several reports on stent grafting for chronic type B aortic dissection are present in the literature; however, none have provided long-term results (Table 4). The rationale for treating a chronic dissection is that covering the area of the primary intimal tear promotes false-lumen thrombosis, which eliminates flow in the false lumen. Preliminary success has been achieved; however, success rates for eliminating all flow in the false lumen are lower for chronic than for acute type B

Table 4. Summary of Endovascular Repair in Chronic Type B Thoracic Aortic Dissections

	Authors			
	Nienaber et al[72]	Kato et al[73]	Eggebrecht et al[74]	Chen et al[70]
Year	1999	2001	2005	2006
Patients, n	12	15	28	19
30-Day death, %	0	0	0	5.5
Technical success, %	100	100	100	100
Paraplegia, %	0	0	0	0
Endoleak, %	0	40		5.5
Complete false lumen				
Thrombosis, %	83*	86.6		95.7†
Partial false lumen				
Thrombosis				
Mean follow-up, mo	12	24	12	27±15.3

*100% Complete false lumen thrombosis at 3 months achieved.

†Refers to both complete and partial thrombosis in all patients (acute and chronic).

aortic dissections. Partial thrombosis has been associated with a reduction in aortic diameter.[39]

Whether prophylactic stent grafting should be performed in uncomplicated type B aortic dissections or patients should be managed medically is the subject of debate. An important ongoing prospective, randomized evaluation is the INSTEAD trial (INvestigation of STEnt grafts in patients with type B Aortic Dissections).[76] This study had accrued 125 patients by the end of 2004, which represented 92% of the desired target enrollment. Endoluminal treatment of type B aortic dissection was performed between 2 and 52 weeks after an index dissection. Twenty-four-month outcomes will be measured. The 52-week upper limit from onset of dissection was considered the maximum window of opportunity with respect to aortic plasticity, because the lamella may become too rigid late in chronic dissection to allow for safe stent-induced aortic remodeling. Preliminary unpublished results suggest a 1-year mortality rate of 3% with medical management and 10% with endovascular treatment. Of note, 11% of the patients in the medical treatment arm crossed over to the endovascular group within 12 months. Reasons for medical failure were malperfusion in 2 patients, progression or expansion of the dissection in 4 patients, and rupture in 1 patient. In this group, there were no deaths associated with intervention.

Another ongoing study entitled the ADSORB (Acute uncomplicated aortic Dissection type B: evaluating Stent-graft placement OR Best medical treatment alone) trial began in 2007.[76] This study, in contrast to the INSTEAD trial, will study patients with acute (<14 days) uncomplicated dissection. The trial has a targeted enrollment of 270 patients with a 50:50 randomization (135 stent grafts and 135 medical therapy patients) with a 3-year follow-up. The study promises to clarify the exact role of stent grafting in uncomplicated type B aortic dissections. No preliminary results are available.

There is a continued evolution of endovascular techniques, and the optimal timing of intervention remains to be defined. Results of the INSTEAD and ADSORB trials discussed above will help guide future therapy.

Intramural Hematoma

Although classic aortic dissections begin with an intimal tear that allows blood to course rapidly along a plane in the outer third of an intrinsically diseased media, an IMH is thought to begin with rupture of the vasa vasorum of the aortic wall. This rupturing of the vasa vasorum results in a circumferentially oriented blood-containing space seen within the aortic wall (Figure 9). The IMH is depicted as a blood-filled circumferential space without an overlying ulcer or other intimal disruption or discontinuity. Regional aortic wall thickening is >7 mm on CT scan or magnetic resonance imaging in the absence of an intimal flap and without enhancement after contrast injection.[77] Less commonly, an IMH may develop as a result of a PAU or thoracic trauma.[78]

According to Nienaber et al,[79] Krukenberg first described an IMH in 1920 as a "dissection without intimal tear." This variation of aortic dissection was considered a distinct entity at postmortem examination. On histological analysis, a hematoma is visualized that disrupts the medial layer of the aorta. In 1985, Yamada et al[80] proposed the term "aortic dissection without intimal rupture" to describe IMHs in isolation of intimal disruption or penetrating ulceration. In their series, 6 (43%) of the 14 patients had involvement of the ascending aorta, and the descending aorta was involved in the remaining 8 (57%). All patients were managed medically with serial imaging studies; 3 patients (21%) died within 1 month of diagnosis (2 with involvement of the ascending aorta and 1 with involvement of the descending aorta), and another patient died after a graft replacement. Nine of the 10 survivors showed complete resolution of the aortic IMH within 1 year.

IMH may occur spontaneously in hypertensive patients or after blunt chest trauma.[81] It has been speculated that an aortic IMH may also originate from ulceration in an atherosclerotic aorta; thus, the cause may be multifactorial. Individuals who develop an IMH may have no evidence of an atheromatous ulcer on imaging studies, however, and an intimal tear is not visualized.

Because there is no intimal discontinuity, the space does not communicate directly with the aortic lumen, and unlike the false lumen of classic aortic dissection, it does not generally display enhancement with contrast administration on CT scanning and angiography.[79] On transesophageal echocardiography examinations, an IMH displays an aortic wall hematoma in the absence of an intimal dissection flap or penetrating ulceration.

Often seen as a more focal lesion than an aortic dissection, IMH is misdiagnosed as an aortic dissection approximately 12% of the time.[2] IMH is commonly associated with a pleural effusion and typically does not produce branch-vessel compromise because of its localized nature.

IMH is primarily a disease of the descending aorta in approximately 70.6% of cases.[2] It is a disease of older individuals. Common comorbidities associated with IMH include hypertension, chronic obstructive pulmonary disease, and chronic renal insufficiency.[2] The clinical presentation of a descending thoracic aortic IMH is almost uniformly that of excruciating back pain, similar to a type B aortic dissection. Compared with acute aortic dissections, the aortic rupture rate for IMH has been shown to be higher, occurring in 35.3% of reported cases in 1 series.[2]

In IMH, gross findings in the operating room include a very superficial location of the hematoma in the aortic wall. In contradistinction to a classic dissection, after the external adventitial layer is opened, the inner layer remains convex and appropriate in shape, not falling away from the aortic wall in concave collapse as seen in typical dissection. The IMH pathology uniformly demonstrates a significant degeneration of the aortic media, with hematoma located just cells away from the adventitia. This finding is in contrast to the usual pathological findings in classic dissection, in which the plane of hematoma is routinely found not more than two thirds of the way through the media.

In 1 series, IMH patients had the lowest 1-year survival rate (65.8%) compared with type A and B aortic dissections and PAUs (Figure 11).[2] The authors believe that there may be an underlying pathophysiological process that determines which patient develops a typical dissection and which develops an IMH. In particular, the level of blood collection is
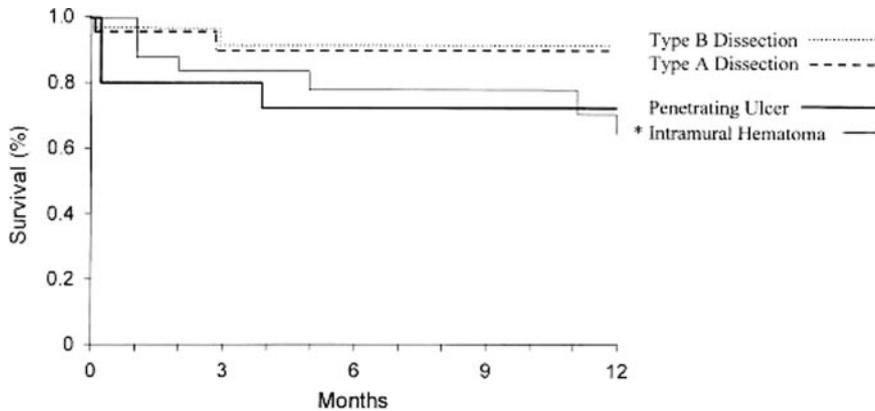
Figure 11. Kaplan–Meier 1-year survival for patients with type A and B aortic dissections, PAUs, and IMHs. *P=0.03. Reprinted from Coady et al,[2] with permission from Elsevier. Copyright 1999, Elsevier.

more superficial and closer to the adventitia in IMH than in typical aortic dissection. This observation may explain why the inner layer does not prolapse into the aorta on imaging studies or at the time of surgery. This more superficial location of an IMH would also explain the high rupture rates compared with classic aortic dissection.

Implications for Stent Grafting

Patients with an IMH in the descending aorta (type B IMH) who present without complications (ie, rupture or impending rupture) can be treated like those with an acute, uncomplicated type B aortic dissection, with anti-impulse therapy, including β-blockade and afterload reduction. This treatment approach is in contradistinction to type A IMH patients, who are treated with emergent surgery, similar to a type A aortic dissection.

These lesions, however, are more serious than classic type B aortic dissection, and a low threshold for surgical or endovascular intervention must be entertained. If radiographic findings are ominous (severely bulging hematoma, extensive subadventitial spread, extra-adventitial blood, significant bloody pleural effusion), surgery or endovascular therapy should be considered preemptively. In the absence of ominous initial radiographic findings, repeat imaging should be performed within 3 to 5 days.[2] In case of any progression in volume or extent of the hematoma or overall size of the aorta, persistent pain, or aortic dilation ($>$5 cm), surgery should be performed preemptively to prevent rupture. Likewise, if symptoms are not controlled or recur on medical treatment, surgery should be performed. This position is supported by most other series.[2,82]

Because IMH primarily affects older individuals with significant comorbidities, however, the above recommendations must be tempered by consideration of the patient's overall condition and general outlook. Some of these patients may not be appropriate candidates for an aggressive surgical strategy.

Endovascular treatment can be performed for a descending thoracic aorta IMH as an alternative to surgery. As mentioned above, IMH occurs in an elderly population, often with significant comorbidities. Although no randomized controlled studies exist, small series have addressed endovascular treatment for IMH (Table 5). Results of these studies reveal a high percentage of technical success with a relatively low mortal-

ity and complication rate. IMH lesions in these studies demonstrate either decreased wall thickness or complete regression in some cases.[83,84]

Penetrating Atherosclerotic Ulcers

In 1934, Shennan[49] was the first to describe penetrating atheromatous ulcers of the thoracic aorta. These atheromatous plaques, characterized further by Stanson et al[85] in 1986, ulcerate and disrupt the internal elastic lamina, burrowing deeply through the intima into the aortic media.[86] The plaque ruptures and precipitates a localized intramedial dissection associated with a variable amount of hematoma within the aortic wall. Blood may then break through into the adventitia, forming a pseudoaneurysm, or may rupture into the right or left hemithorax.

Ulceration of an aortic atheroma occurs in patients with advanced atherosclerosis; however, this process is usually asymptomatic, confined to the intimal layer, and not associated with an IMH. When an atherosclerotic plaque invades the media, it is exposed to pulsatile atrial flow, which causes hemorrhage into the wall without an intimal flap. This PAU involves a localized dissection, and the IMH is prevented from extension by the surrounding transmural inflammation and relative fusion of the aortic wall layers.[87] The adventitial layer is frequently dissected from the media in patients who have penetration through the media.[88] The adventitia may also rupture, and only the surrounding mediastinal tissues then contains the hematoma. Patients with PAU, therefore, have a localized dissection that is limited by areas of severe

Table 5. Summary of Endovascular Repair in IMH

	Authors	
	Grimm et al[83]	Monnin-Bares et al[84]
Year	2008	2009
Patients, n	8	15
30-Day death, %	0	0
Technical success, %	100	93
Endoleak, %	0	7
Decrease in wall thickness, %	100	53
Complete regression, %		47
Complication rate, %	0	20
Mean follow-up, mo	16	21

calcification associated with locally advanced atherosclerotic disease, which represents a different aortopathy than that of a classic aortic dissection.

The clinical presentation of patients with penetrating aortic ulceration is similar to that of classic aortic dissection; however, penetrating ulceration represents a unique pathological event that may have distinct prognostic and therapeutic implications.[89] PAU has become a distinct entity with distinct clinical and pathophysiological manifestations.[85,86,89] Because penetrating ulcer is much less common than classic aortic dissection, it often may be unrecognized and misclassified as an aortic dissection on presentation.

Penetrating ulcers present similarly to typical aortic dissection, with chest or back pain of excruciating intensity. Absent, however, are findings of branch-vessel occlusion or run-off organ ischemia, because PAU does not generally impair luminal blood flow. PAU is a more focal lesion than classic aortic dissection, which frequently propagates for much or the entire extent of the thoracoabdominal aorta. On imaging by CT, magnetic resonance, and transesophageal echocardiography, the cardinal findings for PAU include an ulcer crater similar to the barium appearance of a duodenal ulcer.

An artist's rendition of PAU is presented in Figure 9 that corresponds closely with the images engendered by their verbal designations. PAU is shown as an ulcer crater with varying degrees of surrounding hemorrhage. PAU does not have a flap that traverses the aortic contour and defines a true and false lumen. These renditions correspond well to our current understanding of the pathophysiology of these disorders.

In a retrospective study of 212 patients initially diagnosed as having an aortic dissection, Coady et al[2] found 19 individuals (9%) to have PAU on review of CT, magnetic resonance imaging, angiography, echocardiography imaging studies, intraoperative findings, or pathology results. All patients classified as having PAU showed contrast-filled outpouchings, and an ulcerated crater was seen on angiography, transesophageal echocardiography, CT, or magnetic resonance imaging. Associated IMH and pleural effusions were often identified as well. Severe aortic arteriosclerosis and calcification were seen uniformly.

Similar to IMH, patients with PAU are typically older than patients with classic aortic dissections (Figure 12). Patients with type B aortic dissections tend to be approximately 65 years old on average; PAU patients tend to be a decade older.[2] Not unexpectedly, PAU patients' comorbidities include hypertension, chronic obstructive pulmonary disease, coronary artery disease, chronic renal insufficiency, and diabetes mellitus.

Patients with PAU have also been found to have the largest aortas at presentation (measuring ≈6.2 cm) compared with patients with IMH and type B dissections ($P=0.01$).[2] PAU is almost entirely a descending aortic phenomenon, occurring in 89.5% of cases. The incidence of acute rupture for PAU in the study by Coady et al[2] was as high as 42.1% (8 of 19 patients) as opposed to 35.3% for IMH (6 of 17 patients), 7.5% (6 of 80 patients) for type A dissections, and 4.1% (4 of 98 patients) for type B dissections ($P=0.0001$). The ruptures

occurred with initial presentation or during initial hospitalization. With only 1 exception, all patients with PAU and IMH in the ascending aorta experienced rupture. Three individuals were thought initially to have a type B aortic dissection and died while being treated with standard medical therapy (β-blockade and afterload reduction) in the intensive care unit.

Since its initial description in 1934, the risk and recommended management of PAUs have been a matter of much debate. There is still controversy over the appropriate management of PAU. In addition, surgical resection can be difficult because of the diffuse disease, which makes the extent of surgical resection unclear.[20] The results of the study by Coady et al[2] reaffirm a previous report by Stanson et al,[85] who suggested that PAUs are a particularly malignant lesion with a high risk of perforation and aortic dissection.

Although these data would support more aggressive early therapy,[2,90] Hussain et al[90] presented data on 5 patients (out of 47 patients diagnosed with an aortic dissection) who were found to have PAUs. These patients were managed successfully with medical therapy.

In 2004, the Mayo Clinic group presented 25-year data on 105 patients who were identified as having a penetrating ulcer in the descending thoracic aorta with (n=85) and without (n=20) associated IMH.[91] Among nonoperated patients with follow-up studies, the mean thickness of the IMH decreased at 1 month in 89% and resolved completely at 1 year in 85%. There were 3 deaths (4%) within 30 days among 76 patients treated medically, and 6 deaths (21%) among 29 patients treated surgically ($P<0.05$). Failure of medical therapy, defined as death or need for surgery, was predicted by rupture at presentation and era of treatment (before 1990) but not by aortic diameter, ulcer size, or extent of hematoma. The authors concluded that careful follow-up is necessary and that many PAUs of the descending thoracic aorta can be managed nonoperatively in the acute setting.[91] Given the high frequency of comorbid conditions in elderly patients, an expectant approach is thought to be reasonable in the majority of cases. The associated IMH in patients managed medically appears to resolve with time (Figure 13), and the risk of acute death during the initial hospitalization among medically treated patients appears low.[91]

Still, 1-year survival estimates for IMH and PAU patients fall short of the survival for both type A and type B dissections (Figure 14).[2] Survival in medically and surgically managed patients is remarkably similar, as are late outcomes in symptomatic and asymptomatic patients (Figure 14).[91]

Implications for Stent Grafting

Similar to IMH, PAU lesions in the descending aorta can be observed initially and treated with anti-impulse therapy, including β-blockade and afterload reduction. Mixed clinical presentation, varied patient populations, and data limited to small numbers of retrospectively reviewed patients have made a data-driven algorithm for the surgical treatment of PAUs difficult to construct.[92] Although the natural history of these lesions is still being clarified, most authors believe that a PAU represents a high-risk lesion. A more expectant approach has been advocated, with serial imaging studies[91];
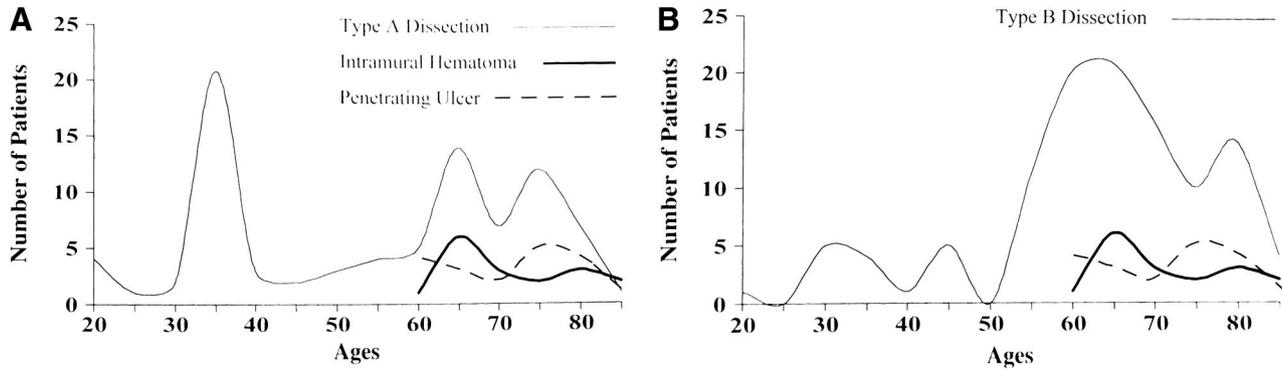
Figure 12. A, Comparison of age distributions for type A dissections, PAU, and IMH. B, Comparison of age distributions for type B dissections, PAU, and IMH. Reprinted from Coady et al,[2] with permission from Elsevier. Copyright 1999, Elsevier.

however, a low threshold for surgical intervention must be maintained. If the radiographic findings are ominous (severely bulging hematoma, extensive subadventitial spread, extra-adventitial blood, increasing bloody pleural effusion, or deeply penetrating ulcer), surgery should be performed preemptively.[2] In the absence of ominous initial radiographic findings, repeat imaging should be performed within 3 to 5 days. In case of any progression in volume or extent of the hematoma or overall size of the aorta, surgery should be performed preemptively to prevent rupture. Likewise, if symptoms are not controlled or recur on medical treatment, surgery is recommended. This position is supported by most other series.[77,82,85,89]

If patients tolerate early medical management without clinical deterioration, they may continue to be followed up conservatively.[2] With long-term follow-up, general size criteria for surgical intervention on the TAAs can be applied.[12] Because PAUs primarily affect an older population, however, the above recommendations must be tempered by consideration of the patient's overall condition and general outlook. Some of these patients may not be appropriate candidates for the aggressive open surgical strategy outlined above. As mentioned previously, many patients have resolution of their associated IMH at 1 year in follow-up.[91]
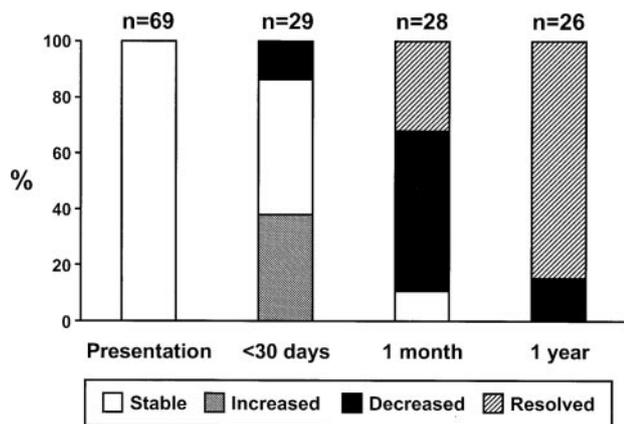
Several small studies have examined the utility of aortic stent grafting in PAU (Table 6).[93–99] Unlike classic aortic dissection, PAU is typically a focal, localized lesion, which represents an ideal anatomic target for stent grafting. In addition, PAU patients have significant atherosclerotic disease, often with other serious comorbidities. In this population, stent grafting presents an attractive treatment option, eliminating the need for a thoracotomy and aortic cross-clamping. Stent grafting excludes the diseased segment, reduces wall stress, and facilitates resolution of associated IMH.

Figure 13. Fate of associated IMH in patients with PAU managed medically. As depicted below, in the vast majority of patients, the IMH resolved by 1 year of follow-up. Reprinted from Cho et al,[91] with permission from Mosby. Copyright 2004, The American Association for Thoracic Surgery.
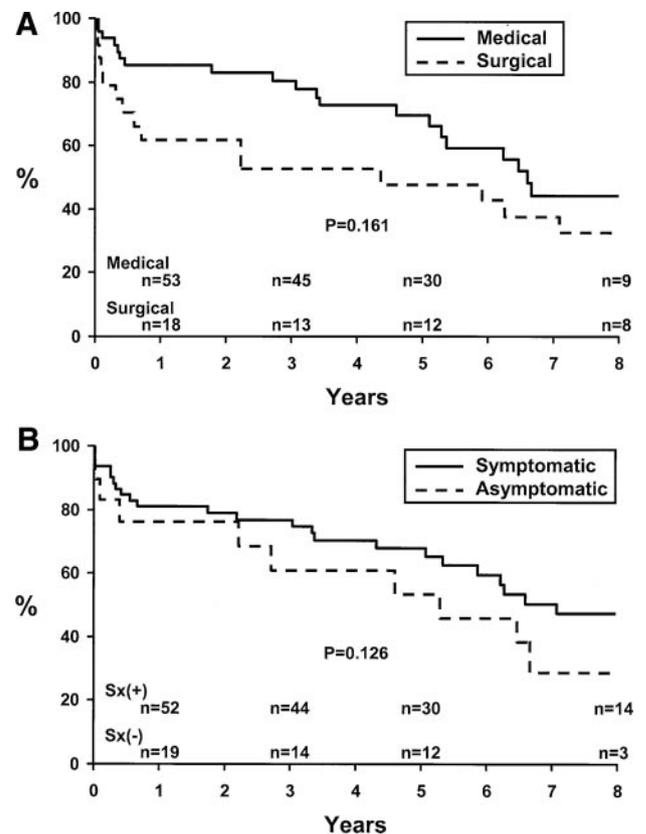
Figure 14. A, Actuarial survival of medically and surgically managed patients. B, Actuarial survival of patients presenting with symptomatic vs asymptomatic ulcers. Sx(+) indicates symptomatic; Sx(-), asymptomatic. Reprinted from Cho et al,[91] with permission from Mosby. Copyright 2004, The American Association for Thoracic Surgery.

Table 6.   Summary of Endovascular Repair in PAU

	Authors						
	Murgo et al[93]	Schoder et al[94]	Eggebrecht et al[95]	Demers et al[96]	Brinster et al[97]	Pauls et al[98]	Botta et al[99]
Year	1998	2002	2003	2004	2006	2007	2008
Patients, n	4	8	10	26	21	12	19
30-Day death, %	0	0	0	0	0	0	11.1
Technical success, %	100	100	100	92	100	100	95
Endoleak, %	25	12.5	10	8	0	17	17
Complication rate, %	25	12.5	0	19	5	17	0
Paraplegia, %	25	12.5	0	0	0	0	0
Mean follow-up, mo	6	15	12	51	14±18	27	22

As mentioned previously, traditional open surgical replacement of the descending thoracic aorta carries an associated death risk that ranges between 5% and 20%.[100–102] Reports from high-volume centers have indicated mortality rates specific to descending PAU as high as 21% for those treated with open, traditional surgery.[91]

Technical success in these series has been reported to be high, with low overall mortality and complication rates in selected high-surgical-risk elderly patients with PAU located in the descending thoracic aorta. Because the use of stent grafts is associated with endoleaks and other unique complications, it cannot be considered curative as in the sense of open surgical graft replacement of the aorta.[99] In fact, the diffuse atherosclerotic burden increases the risk of endoleaks in this patient population. The diffuse disease also increases the risk of recognized complications such as aortic dissections that extend into the arch and ascending aorta.[20] In addition, Botta et al[99] reported endoleaks in 17% of patients during follow-up (Table 6). The generalized, systemic atherosclerotic disease also requires careful consideration of the appropriate and safest access for stent grafting in this population. A retroperitoneal exposure of the iliacs or lower abdominal aorta may be prudent.[92]

Acute Aortic Transection

Acute aortic transection is a potentially lethal injury that is second only to head injury as the most common cause of death after blunt trauma.[103] Motor vehicle accidents account for the majority of these cases, and subsequent aortic injury occurs secondary to the effect of vertical forces of deceleration at points of fixation in the descending aorta. The aortic injury typically occurs at the level of the ligamentum arteriosum, proximal to the third intercostal artery, an area commonly referred to as the aortic isthmus. The pathology varies from a simple subintimal hemorrhage, with or without intimal laceration, to complete transection.[104] Transection of the aorta requires a tremendous force of impact, which usually results in death at the scene. In fact, 85% of patients die at the scene of the trauma due to rapid hemorrhage into the mediastinum. Only 15% to 20% of patients are stable enough to arrive at a hospital for definitive treatment. The rate of death over the ensuing 48 hours is approximately 1% per hour among patients who do not receive surgical intervention.[103] Of the patients who present to the hospital, 33% will also become hemodynamically compromised. The mortality rate in this subgroup approaches 100%; among the remaining two thirds who are stable, there is a 25% mortality rate, even in the most expert hands. Rarely is traumatic transection an isolated injury. The majority of patients sustain concomitant orthopedic and abdominal injuries.[105]

Patients who survive to reach the hospital have typically sustained an incomplete noncircumferential lesion limited to the intima and media, in which the rupture is contained by the strength of the tunica adventitia and mediastinal pleura.[106] Chest radiography may or may not reveal a widened mediastinum. Other radiological signs may be present, including rightward shift of the trachea, blurring of the aortic knob or descending thoracic aorta, opacification of the juncture between the aorta and pulmonary trunk, and left hemothorax. CT angiography with at least a 16-detector CT scanner is recommended and is reported to have a sensitivity approaching 100% for diagnosing traumatic transection in patients being investigated for blunt thoracic trauma. Other modalities used for diagnostic purposes include transesophageal echocardiography and angiography. Transesophageal echocardiography lacks the sensitivity and specificity of either the CT angiography or catheter-based angiogram. Currently, catheter-based angiography is only used in cases of indeterminate or equivocal CT angiography scan results.

There are several theories as to why the aortic isthmus is the commonest site of injury. One is that Botallo's ligament (ligamentum arteriosum) fixes the aortic isthmus to the left pulmonary artery and acts like a hinge on which the aortic arch moves, making it vulnerable to shearing forces during injury. In 1964, Lundervall[107] measured the breaking strength of rapidly stretched strips of aortic wall and demonstrated that the isthmic wall was approximately two thirds the strength of the ascending aortic wall, with the descending aorta having an intermediate strength. The water hammer theory is based on the fact that a sudden increase in intravascular hydrostatic pressure at the time of impact can cause a tear in the aorta at its weakest point.[108] Another theory, called the osseous inch theory, explains how compressive force from blunt thoracic trauma may cause the osseous structures of the anterior thorax to rotate posteriorly and inferiorly around the axis of the posterior rib articulations, tearing interposed vascular structures.[104]

The standard approach to acute aortic transections has been an early diagnosis followed by emergent thoracotomy and repair of the aorta. The aim of expeditious surgery is to minimize the risk of impending rupture. This algorithm has been the practice since the highly cited paper by Parmley et al in 1958.[108] That report reviewed 275 cases of traumatic injury to the aorta that resulted in aortic rupture. The initial survival rate was 13.8%. Of the 38 patients who survived to reach the hospital alive, 3 patients died within 1 hour, and 23 died of rupture within 7 days of admission. Parmley et al[108] concluded that prompt diagnosis is essential if surgical treatment is to be performed before fatal sequelae develop.

Despite advances in surgical techniques and options for prevention of spinal cord ischemia, the reported operative mortality rate is in the range of 18% to 28%, with paraplegia rates ranging from 2.3% to 14%.[88] Major contributing factors to the high rate of death in operative cases are the severe associated injuries and the advanced age and preexisting coronary artery disease of these patients.[109]

Because of the significance of concomitant injuries, some authors have explored delayed surgical repair, addressing life-threatening injuries or serious premorbid conditions first. In select cohorts, the risk of emergent aortic repair may outweigh the benefits. These patients may include those with known coronary artery disease, severe head injury (with anticipated thromboplastin release from the brain, which contributes to excessive bleeding), significant pulmonary injury with impaired ventilation, and ongoing coagulopathy.

Minimization of these risks includes management of major injuries first and treatment of medical comorbidities. Camp and Shackford,[109] in an analysis of 395 cases from 14 trauma centers over 11 years, demonstrated that patients with advanced age and preexisting coronary disease may have a higher mortality rate with emergent surgery. Those authors advocated the use of β-blocking agents for blood pressure control in patients older than 55 years of age. Only 5% of pseudoaneurysms in the stable cohort ruptured >4 hours after admission in their series. They proposed that expeditious investigation of all associated injuries and comorbidities be undertaken in stable patients (especially those with injuries >4 hours old). The high-risk elderly patient may be a candidate for nonoperative management or endoluminal graft placement. The safety of delayed surgical repair in patients with advanced age, coronary artery disease, or multiple injuries has been confirmed by other authors.[104]

Clearly, the clinical management of patients with aortic injury, whether to delay surgery or stent grafting, is dominated by an underlying fear of possible aortic rupture. There is no way to differentiate those intimal injuries that will progress to uncontrolled hemorrhage from those with a more benign clinical course.[20] Approximately 10% of stable cases die of rupture before surgical repair.[104] In any delayed-management strategy, improved survival has been clearly demonstrated with a strict antihypertensive strategy that uses β-blockade and afterload reduction in an effort to decrease aortic wall stress.[104] Serial imaging studies are critical in the management of these patients. Hemodynamic instability, an increase in the size of mediastinal hematoma, or worsening pleural effusion may indicate impending rupture, and these patients should be considered for emergent surgical or endovascular intervention.

Implications for Stent Grafting

Despite improvement in surgical outcomes, there is still a substantial mortality and morbidity risk, especially of paraplegia, with emergent open surgical repair of aortic transections. Von Oppell et al[110] performed a meta-analysis of 1742 patients who presented with aortic transection between 1972 and 1992 and arrived at the hospital alive. Approximately one third (32.4%) of these patients died before surgical repair was started. Paraplegia was noted preoperatively in 2.6% of these patients, and paraplegia complicated the surgical repair in 9.9% of 1492 patients who reached the operating room in a relatively stable condition. In 1996, Hunt et al[111] analyzed data from 144 patients who sustained thoracic aortic injury. Sixty-four patients died (44.4%), most commonly in the emergency department (24 patients) or in the operating room (12 patients). This study, again, confirmed the operative risk of approximately 32% once patients underwent emergent open operative repair.

Because of the substantial operative risk and attendant morbidity and mortality associated with open surgical repair, there has been a growing interest in less invasive techniques. Although there are no randomized, prospective series, several small reports have documented the utility of endovascular aortic stent grafting in the treatment of posttraumatic aortic transections with excellent results. Table 7 displays a summary of small series of stent grafting in patients with aortic transections.[112–115] Although the experience is quite limited, these studies demonstrated that stent grafting is feasible and a safe alternative to open surgical repair, especially in high-risk patients. Complications do occur, including endoleaks; however, the overall death and risk of paraplegia in these series are the most compelling reasons for consideration.

From a technical standpoint, it is frequently necessary (>80% of cases) to cover the left subclavian artery to achieve an adequate proximal seal.[20] Because patients are mostly young, with normal-sized aortas, the aorta has not typically

Table 7. Summary of Endovascular Repair in Acute Aortic Transection

	Authors			
	Sam et al[112]	Wellons et al[113]	Mohan et al[114]	Neschis et al[115]
Year	2003	2004	2008	2009
Patients, n	3	9	16	43
30-Day death, %	0	11†	6.3	11.6*
Technical success, %	100	100	100	86
Endoleak, %	0	11	18.7	14.3
Complication rate, %	0	22	18.7	0
Paraplegia, %		0	0	0
Mean follow-up, mo		12	19	7.4

*No death was aorta-related.

†Death secondary to other injuries.

enlarged and unfolded, and thus, the acute angulation of the aortic arch, the narrow angle of curvature of the arch, and the small aortic diameter can often be difficult to accommodate with currently available endografts.[10] Stent grafts used in these cases are obviously of smaller dimensions than those used in patients with TAAs. This issue requires the on-site availability of stent grafts of smaller sizes and lower profile, because often, they must also be introduced through small iliac and femoral arteries.[20]

Additional patients and longer follow-up will be necessary to determine the optimal treatment strategy in select patients. Currently, for treatment of aortic transections, stent grafts are most commonly placed in young patients. In addition to questions about graft durability, uncertainty remains regarding changes to the thoracic aorta and stent grafts as patients age, including long-term complications of vascular access and subclavian artery coverage.[114] These patients also require long-term follow-up and a lifetime of radiological procedures, which results in a large cumulative radiation dose over the patient's lifetime.

Conclusions

Treatment of acute aortic syndromes that affect the descending thoracic aorta continues to evolve with the development of new technologies and management strategies. Although data presented in this summary have highlighted current outcomes of endovascular stenting compared with conventional open repair, it must be stressed that there have been no prospective randomized trials to compare these treatment strategies on a head-to-head basis. In addition, although endovascular stenting offers a minimally invasive method of treatment, its long-term durability is still largely unknown. Ongoing experience and national and international registries will continue to define precise roles for both surgical and endovascular therapy. As technology continues to improve and surgeons continue to make strides along the learning curve in treating these diseases, the long-term complications of endografting may or may not be mitigated. Continued vigilant surveillance of patients treated with an endograft remains important. Finally, the healthcare provider must constantly evaluate and revisit these data and carefully apply the principles of evidence-based medicine to be able to care for these patients appropriately.

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*Modest.

†Significant.

Reviewer Disclosures

Reviewer	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Expert Witness	Ownership Interest	Consultant/Advisory Board	Other
Michael Bacharach	North Central Heart Institute	None	None	None	None	None	None	None
Anthony L. Estrera	University of Texas Houston Medical School	None	None	None	None	None	None	None
Thomas Gleason	University of Pittsburgh Medical Center	None	None	None	None	None	None	None
Larry Hollier	LSU Health Sciences Center	None	None	None	None	None	None	None
James Stephen Jenkins	Ochsner Medical Center	None	None	None	None	None	None	None
Nicholas Kouchoukos	Missouri Baptist Medical Center	None	None	None	None	None	ACC/AHA guidelines document on the diagnosis and management of patients with thoracic aortic disease*	None
Zvonimir Krajcer	Texas Heart Institute	None	None	W.L. Gore*; Endologix†	None	None	W.L. Gore*; Endologix*	None

This table represents the relationships of reviewers that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit. A relationship is considered to be "significant" if (1) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (2) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

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Circulation

Surgical Management of Descending Thoracic Aortic Disease: Open and Endovascular Approaches: A Scientific Statement From the American Heart Association

Michael A. Coady, John S. Ikonomidis, Albert T. Cheung, Alan H. Matsumoto, Michael D. Dake, Elliot L. Chaikof, Richard P. Cambria, Christina T. Mora-Mangano, Thoralf M. Sundt and Frank W. Sellke
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