Aortic aneurysms remain a challenging problem for patients and physicians. There have been major advances in the treatment of large-vessel aneurysms during the past 10 years. The surgical armamentarium used to treat these aneurysms now includes an endovascular approach that allows the insertion of a graft to exclude the aneurysm sac from blood flow. The endovascular repair of abdominal and thoracic aortic aneurysms has become a viable alternative to open repair and is often the approach of choice for high-risk patients. In this review, we examine the endovascular treatment of abdominal and thoracic aortic aneurysms.

Endovascular Repair of Abdominal Aortic Aneurysms

Abdominal aortic aneurysms (AAAs) are a formidable diagnosis for patients. This is a life-threatening condition that mandates consideration of repair. A ruptured AAA has a mortality rate approaching 90%; however, when an AAA is repaired electively, the mortality drops to less than 5%. There is, therefore, a clear advantage to treating these aneurysms before they rupture. Because this disease affects 4% to 7% of adults over the age of 65 years, with a far greater prevalence in males than females, clinicians will encounter this problem more frequently as the population ages.

AAAs usually develop in patients with a history of atherosclerosis or smoking. Patients present for repair when it is discovered that there is a dilation of their abdominal aorta to a diameter 1.5 times normal. The result is a weakened aortic wall that is at increased risk of rupture. The pathogenesis of this aortic wall change likely involves enzymes responsible for elastin and collagen breakdown. Recent research has focused on the role of metalloproteinase-9 (MMP-9). Aneurysm presence and size have been correlated with MMP-9 levels. Other investigators are looking into the inflammatory and autoimmune mechanisms involved in aneurysmal disease. Another area of research is in the molecular genetics of AAAs, because these aneurysms have a well-recognized familial nature. There is also an interest in the analysis of aortic wall stress distribution from a biomechanical approach. Ongoing studies have found that peak wall stress can predict those patients who will experience AAA rupture.

Patients with symptomatic aneurysms should be offered repair, after careful consideration of comorbidities, even if the aneurysm is not of usual elective operative size. For patients with asymptomatic AAAs, there are guidelines to help plan further surveillance or operative repair. The size of the aneurysm is one factor, and the operative approach is another. In the past, the fitness of patients for a transabdominal or retroperitoneal operation was of paramount concern. High-risk patients faced the choice of a morbid operation with a not insignificant mortality rate or the risk of rupture. The treatment option for this population was not satisfactory, and the development of a lower-risk operation was a necessity to adequately treat this population.

Over the past 15 years, the management of AAAs has changed dramatically because of the development of the technique of endovascular aneurysm repair (EVAR; Figures 1 and 2). Patients and physicians have embraced EVAR as the method of choice to treat high-risk patients with AAAs. EVAR has great appeal for this older population because it leads to faster recovery with fewer systemic complications than open repair.

Parodi et al reported the first endovascular repair of an AAA in a human in 1991 using a graft fashioned from prosthetic vascular grafts and expandable stents. Current estimates are that more than 20,000 EVAR procedures take place each year in the United States, which represents ~36% of all AAA repairs. The estimate is that >12% of all procedures in Europe are with EVAR, and expected annual growth is ~15% at this time (Medtronic Marketing Department, personal communication, 2004). EVAR is the method of choice in high-risk older patients because of its minimal incisions, shorter operating time, and reduced blood loss.

Three EVAR devices are available commercially in the United States at this time, and several more are under evaluation in clinical trials. The Food and Drug Administration (FDA), well aware of the many confounding variables in an EVAR-to-surgery comparison, permitted the use of concurrent surgical controls. The large deposit of multicenter, prospectively collected data on the European EVAR experience also provides a great source for analyses: the EUROSTAR (EUROpean collaborators on Stent-graft Techniques for abdominal aortic Aneurysm Repair) Registry (4392 patients, 1996 to 2002). In the present review, we will address EVAR indications, contraindications, devices, outcomes, complications, en-
Indications for AAA Treatment
Most asymptomatic AAAs are discovered serendipitously, often on imaging examinations for other complaints. Increasing evidence indicates that there is value to screening patients for AAAs, and it is likely that screening will be approved in the near future. Once the diagnosis of AAA is made, 2 critical questions need to be answered: when to intervene and how to intervene. The availability of EVAR has made these decisions somewhat more complex while adding a significant treatment option.

Recent studies have questioned whether aneurysms smaller than 5 cm should be treated. However, in general, the clinical recommendation remains to offer treatment for aneurysms between 5 and 5.5 cm, depending on the results of clinical trials. An exception to this guideline is that intervention should be offered despite the size of the aneurysm if symptoms develop or the aneurysm increases in size by 1 cm per year. In addition, if the patient is a woman with smaller native vessels, the relative size that represents aneurysmal disease may be less than the conventional 5 to 5.5 cm range.

Clinical and Anatomic Selection Factors
Patient selection has emerged as the most important factor related to successful EVAR. The assessment begins with consideration of the body habitus and sex of the patient; small body size and female sex have been associated with a higher
risk of procedure abortion. In addition, the comorbidities of the patients must be assessed, with careful attention to cardiac, pulmonary, and renal conditions. In general, open surgical repair is advocated for younger, lower-risk patients, and EVAR is preferred for older, higher-risk patients. Open surgical repair of AAA has proven long-term durability; EVAR follow-up is now at 10 years. The use of risk stratification to analyze outcomes clearly indicates that survival for those at low to minimal risk is excellent over 10 years; those at highest risk succumb to cardiac disease or cancer, and for those patients, survival is poorest. EVAR has shown a reduction in 30-day mortality relative to that achieved with open repair (1.2% versus 4.6%). Further study is required to determine whether there is a long-term survival advantage. Risk stratification determines survival in general and shows that both open surgery and EVAR decrease the risk of death from AAA rupture.

The characteristics of the aneurysm must be matched to the most suitable device; this has a direct impact on outcomes and the complication profile of the procedure. The aneurysm is evaluated from a 3D reconstruction CT scan or aortography with a calibrated catheter. There are at least 4 important features that must be assessed before one determines a patient’s eligibility for EVAR, and this analysis leads to a list of contraindications (Table 1). Experienced interventionalists can deal with some of these challenges, but morphological features of the aneurysm and access vessels may preclude EVAR.

Currently, we use EVAR primarily to treat patients at high surgical risk. We are confident that EVAR protects patients from aneurysm rupture, but we cannot yet extend the offer of EVAR to young, healthy patients, because we do not have data on long-term durability to support this practice.

### Devices

Endografts have developed from simple tube grafts suitable for a small subset of aneurysms to complex bifurcated grafts with a range of sizing and extensions that can be used to treat the morphological challenges of aneurysmal disease. The language used to describe the devices has also evolved over time.

There are 2 types of device bodies: unibody and modular. The former comes in 1 piece, and although easy to deploy, it requires contralateral occlusion and bypass grafting. Modular devices are composed of more than 1 piece, and the components are deployed through both groins. There is greater flexibility associated with the modular devices, and they dominate the market. The endograft fabric is either a woven polyester or PTFE (polytetrafluoroethylene). The difference between these is the degree of porosity (PTFE > woven polyester), and some believe this affects the rate of type IV endoleaks.

The key features of endovascular repair of AAAs that determine procedural success and long-term outcomes are proximal and distal fixation and sealing. Various device modifications and designs address this need; there are now 3 FDA-approved endografts and several others in various stages of development (Table 2). Early devices were secured proximally and distally with the aid of expandable stents. Now, most of the devices use a metal skeleton throughout the graft that is made from stainless steel, nitinol, or Elgiloy. Attachment is facilitated by the use of hooks or radial force.

Once the graft is inserted through the sheath, it can be deployed by a self-expanding mechanism or balloon expansion. Some grafts attach superior to the renal arteries (suprarenal attachment), whereas most of the devices require at least 15 mm of proximal neck to achieve fixation and sealing in the

### Table 1. Evaluation for EVAR

<table>
<thead>
<tr>
<th>CT scan assessment for EVAR eligibility</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal neck: diameter, length, angle, presence or absence of thrombus</td>
<td></td>
</tr>
<tr>
<td>Distal landing zone: diameter and length</td>
<td></td>
</tr>
<tr>
<td>Iliac arteries: presence of aneurysms and occlusive disease</td>
<td></td>
</tr>
<tr>
<td>Access arteries: diameter, presence of occlusive disease</td>
<td></td>
</tr>
</tbody>
</table>

### Table 2. Endograft Devices

<table>
<thead>
<tr>
<th>Device (Company)/Market</th>
<th>FDA Approval</th>
<th>Main Body Diameter, mm</th>
<th>Fixation Profile</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ancure (Guidant)/off-market 2003</td>
<td>September 1999</td>
<td>20–26</td>
<td>Infrarenal 21F</td>
</tr>
<tr>
<td>AneuRx (Medtronic)/worldwide</td>
<td>September 1999</td>
<td>22–28</td>
<td>Infrarenal 21F</td>
</tr>
<tr>
<td>Excluder (Gore)/worldwide</td>
<td>November 1999</td>
<td>23–31</td>
<td>Infrarenal 18F</td>
</tr>
<tr>
<td>Fortron (Cordis)/Europe</td>
<td>No</td>
<td>26–34</td>
<td>Suprarenal NA</td>
</tr>
<tr>
<td>LifePath (Edwards)/Europe, Australia</td>
<td>No</td>
<td>21–29</td>
<td>Infrarenal 21F</td>
</tr>
<tr>
<td>Powerlink (Endologix)/Europe</td>
<td>No</td>
<td>25–34</td>
<td>Infrarenal; suprarenal 21F</td>
</tr>
<tr>
<td>Stentor/Vanguard (Boston Scientific)/off-market 1999</td>
<td>No</td>
<td>22–26</td>
<td>Infrarenal 21F</td>
</tr>
<tr>
<td>Talent (Medtronic)/Europe, Australia, Asia</td>
<td>No</td>
<td>24–34</td>
<td>Suprarenal 22F/24F</td>
</tr>
<tr>
<td>Trivascular (Boston Scientific)/in research</td>
<td>No</td>
<td>16–26</td>
<td>Infrarenal 16F</td>
</tr>
<tr>
<td>Zenith (Cook)/worldwide</td>
<td>May 2003</td>
<td>22–32</td>
<td>Suprarenal 18F/20F</td>
</tr>
</tbody>
</table>
infrarenal position. The grafts also differ in their “profile” or the size of the delivery system. Low-profile devices permit access through smaller arteries.

The importance of choosing the appropriate device to fit the characteristics of the aneurysm cannot be overemphasized. With a short proximal neck, the suprarenal Talent device proves useful, and when small access arteries are encountered (especially in females), the low-profile Excluder endograft is appealing. The Lifepath device has been used with some success in the difficult angulated neck.

Outcomes

Table 3 describes the published literature from large device-specific series. Some of the series compare the EVAR data with that obtained from open surgical repair. Recently, a prospective, randomized trial of EVAR and open repair demonstrated a two-thirds reduction in early mortality with EVAR. All studies have shown excellent rupture-free survival and significant reduction in blood loss, cardiovascular events, and intensive care unit stay. High-volume institutions with experience using multiple types of endografts are able to optimize the matching of the device to the characteristics of the aneurysm.

Complications

Most of the complications associated with EVAR are minor and can be watched carefully or treated easily with additional interventional procedures. Some complications occur during or soon after the procedure, whereas others are only noticed during graft surveillance (Table 4). A study by Ohki et al analyzed complication and death rates within 30 days of EVAR and found the major complication and death rates were 17.6% and 8.5%, respectively. This remains an active and important area of EVAR research, and standards have been developed to facilitate reporting of endovascular abdominal aortic repair complications.

Endoleaks can have substantial clinical significance because of an increased risk of becoming symptomatic or leading to aneurysm rupture. Endoleak describes the continuation of blood flow into the extragraft portion of the aneurysm; this flow increases the size of the aneurysmal sac. Endoleaks occur in either the acute setting during graft implantation or during the postoperative surveillance period. The majority of procedural endoleaks will disappear without intervention.

Endoleaks are either graft-related or nongraft-related, and a classification system has been developed (Table 5). Type I endoleaks occur when the attachment is not complete, either proximally or distally; blood is able to flow into the aneurysmal sac and is not completely occluded by endograft attachment to the arterial wall. Type II endoleaks result from continued back flow from aortic branches such as the inferior mesenteric artery and lumbar arteries. This flow occurs retrograde into the aneurysmal sac around the endograft. Type III endoleaks are caused by defects in the endograft structure that lead to leakage of blood flow from inside the endograft to the aneurysmal sac. Finally, type IV endoleaks are noted early after endograft placement and resolve when the fabric porosity is decreased by clotted blood. Because endovascular repair uses a relatively new technology, graft surveillance for complications such as endoleaks is essential. Endoleaks are diagnosed by a variety of techniques: arteriography, pressure monitoring during or after the procedure, CT scan, and duplex scanning. The preferred method of detecting
endoleaks is by CT scanning. An analysis of 2463 patients from the EUROSTAR registry revealed that 171 had an endoleak by the time of their first-month postoperative evaluation, and 317 patients developed one at a later date. Of these, 7.8% had a type II endoleak, and 12% had a type I, type III, or combination of endoleaks.

There are many different ways to treat endoleaks, including coil embolization, placement of stent-graft cuffs and extensions, laparoscopic ligation of inferior mesenteric and lumbar arteries, open surgical repair, and EVAR redo procedures. Type I and III endoleaks require fairly urgent intervention, because blood flow and sac pressure will continue to increase and lead to rupture. Type IV endoleaks usually resolve on their own.

The management of type II endoleaks is more controversial, because some of them will thrombose on their own, whereas others will lead to sac enlargement. There are 2 critical questions that must be addressed at this juncture: (1) Should an intervention be entertained, and (2) when should the intervention be performed? One approach is to monitor the patient with a postprocedure 6-month CT scan, and if the aneurysm has increased in size, then a plan for intervention is formulated. There are at least 3 approaches to manage type II endoleaks: transarterial, translumbar embolization, and laparoscopic ligation. Another approach to eliminate endoleaks is to perform preoperative embolization of feeding arteries. A study by Bonvini et al analyzed 23 consecutive patients who underwent preoperative embolization to reduce type II endoleaks. They monitored the patients for a mean of 17 months and found 1 type II endoleak (4.5%). During a consensus conference in 2000, 23 of 26 international leaders in EVAR disagreed with the preoperative approach to endoleaks. Surveillance

Endograft surveillance is important to document normal and abnormal morphological changes in the repair and the involved vessels. This process is vital for the detection of endoleaks, increased aneurysm diameter, and possible device migration. The study by Corriere et al showed that 24% of the patients had an endoleak detected during surveillance that averaged 19 months. In that study, 3 endoleaks were detected 2 years after repair, with 1 at 7 years. Other findings, including periendograft air, accompanied by leukocytosis and fever, can be detected after repair and have been shown to be nonspecific and not indicative of graft infection.

The recommended surveillance routine is for a CT scan at 1, 6, and 12 months and annually thereafter. If an endoleak is detected, the frequency of the scans increases to every 6 months until resolution of the endoleak is detected. Investigators have compared duplex ultrasound with CT scan for surveillance and found that CT scan is superior for endoleak detection. MRI can also be used for graft surveillance, especially in older patients with decreased renal function. A recent development for endograft surveillance is the monitoring of sac pressure within treated aneurysms through an implanted sensor device. Trials with this implanted sensor are currently being conducted.

The use of EVAR technology has led to a greater understanding of the basic science of aneurysmal disease. For example, Curci and Thompson have been studying the relationship between the secretion of matrix metalloproteinases (MMPs) and AAAs. They have measured increased levels in the aneurysmal rather than the normal arterial wall.

### TABLE 4. EVAR Complications

<table>
<thead>
<tr>
<th>Category</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deployment related</td>
<td>Failed deployment, Bleeding, Hematoma, Lymphocele,</td>
</tr>
<tr>
<td></td>
<td>Infection, Embolization, Perforation, Arterial</td>
</tr>
<tr>
<td></td>
<td>rupture, Dissection, Device related, Structural</td>
</tr>
<tr>
<td></td>
<td>failure, Implant leaks</td>
</tr>
<tr>
<td></td>
<td>Limb occlusion/stent-graft kink, Sac enlargement/</td>
</tr>
<tr>
<td></td>
<td>proximal neck dilatation</td>
</tr>
<tr>
<td></td>
<td>Stent migration, AAA rupture, Infection, Buttock/</td>
</tr>
<tr>
<td></td>
<td>leg claudication</td>
</tr>
<tr>
<td>Systemic</td>
<td>Cardiac, Pulmonary, Renal insufficiency,</td>
</tr>
<tr>
<td></td>
<td>Cerebrovascular, Deep vein thrombosis, Pulmonary</td>
</tr>
<tr>
<td></td>
<td>embolus, Coagulopathy, Bowel ischemia, Spinal cord</td>
</tr>
<tr>
<td></td>
<td>ischemia, Erectile dysfunction</td>
</tr>
<tr>
<td>III: Graft defect</td>
<td>A: Junctional leak or modular disconnect, B: Fabric</td>
</tr>
<tr>
<td></td>
<td>disruption (midgraft hole), Minor (&lt;2 mm; eg, suture</td>
</tr>
<tr>
<td></td>
<td>holes), Major (≥2 mm)</td>
</tr>
<tr>
<td>IV: Graft wall (fabric)</td>
<td>porosity (&lt;30 days after graft placement)</td>
</tr>
</tbody>
</table>

### TABLE 5. Classification of Endoleaks

I: Attachment site leaks
   A: Proximal end of endograft
   B: Distal end of endograft
   C: Iliac occluder (plug)

II: Branch leaks (without attachment site connection)
   A: Simple or to-and-fro (from only 1 patent branch)
   B: Complex or flow-through (with 2 or more patent branches)

III: Graft defect
   A: Junctional leak or modular disconnect, Minor (<2 mm; eg, suture holes)
   B: Fabric disruption (midgraft hole)
   Major (≥2 mm)

IV: Graft wall (fabric) porosity (<30 days after graft placement)
This finding may lead to a simple test for increased aneurysm sac growth after endograft repair. Advances in the basic science of aneurysm disease are helping us better manage this disease.

Future Directions
EVAR has clear benefits, especially in high-risk patients. After long-term durability studies have been completed, we will have a better idea of the role of EVAR in young, healthy patients. The future of EVAR is exciting, with many new developments on the horizon. The next generation of endografts will have smaller delivery systems that will permit the application of this technology to a broader group of patients. There are endograft designs in development that will allow us to better treat aneurysms with short proximal necks and tortuous iliac arteries. The results of ongoing clinical trials will help elucidate the role of branched endografts that facilitate treatment of suprarenal aneurysms. A new implantable sac pressure-monitoring device is being studied, and the utility of this device in the surveillance of the endovascular repair may help decrease the frequency of follow-up CT scans. The EVAR story is still being written. This technology is helpful to a segment of the population of patients with AAAs, and more study will only broaden its application.

Endovascular Repair of Thoracic Aortic Aneurysms
Aortic aneurysms, both thoracic and abdominal, generally occur in the elderly and have thus been increasing in incidence as the population ages and diagnostic capabilities advance. With an incidence of 6 to 10 per 100 000 person-years, thoracic aortic aneurysms (TAAs) are less common than AAAs but remain life-threatening. The natural history of TAAs is one of progressive expansion and weakening of the aortic wall, leading to eventual rupture. With an associated mortality rate of 94%, TAA rupture is usually a fatal event. The 5-year survival rate of unoperated TAA patients approximates 13%, whereas 70% to 79% of those who undergo elective surgical intervention are alive at 5 years. The risk of rupture mandates consideration for surgical treatment in all patients who are suitable candidates for operation. Specific indications for operative repair are listed in Table 6. The use of endovascular stent grafts for repair of TAAs is emerging as a promising, less invasive therapeutic alternative to conventional surgical treatment.

Endovascular treatment of aortic aneurysms is achieved by transluminal placement of 1 or more stent-graft devices across the longitudinal extent of the lesion (Figure 3). The prosthesis bridges the aneurysmal sac to exclude it from high-pressure aortic blood flow, thereby allowing for sac thrombosis around the endograft and possible remodeling of the aortic wall. Although the advent and ensuing rapid evolution of endovascular aortic repair occurred initially in the abdominal aorta, efforts to adapt this technology for the thoracic aorta are ongoing. As is the case for AAA, a less invasive approach to TAA repair is highly desirable, because the patient population tends to be elderly and harbors multiple comorbidities. Continued development of endovascular therapy for thoracic aneurysms, however, is likely to provide greater benefits in patient outcomes than those observed with AAAs. Conventional surgical treatment of TAA is physio-

<table>
<thead>
<tr>
<th>TABLE 6. Indications for TAA Repair</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥60-mm diameter or &gt;2× transverse diameter of an adjacent normal aortic segment</td>
</tr>
<tr>
<td>Symptomatic regardless of size</td>
</tr>
<tr>
<td>Growth rate of aneurysm to &gt;3 mm/y</td>
</tr>
</tbody>
</table>

Figure 3. Stent-graft repair of thoracic aortic pseudoaneurysm. A, Thoracic aortogram illustrates large aneurysm in a 58-year-old man who had surgery 22 years earlier to repair a traumatic aortic injury from a motor vehicle accident. B, Aortogram after stent-graft placement shows widely patent device without evidence of contrast media endoleak into the aneurysm sac.
Endovascular aneurysm repair of the thoracic aorta is currently focused on the descending portion, defined as being distal to the left subclavian artery. Approximately half of TAAAs are located in the descending thoracic aorta.51,59 Anatomically, this aortic segment provides a substrate more amenable for endovascular stent-graft repair given its avoidance of the great vessels proximally and major visceral branches and aortic bifurcation distally. Despite these anatomic advantages and the ability to draw from early experiences with endovascular AAA repair, the development of stent grafting in the thoracic aorta has progressed more slowly than that of its infrarenal counterpart. The thoracic aorta poses several unique challenges that have impeded simple adaptation of endovascular devices and techniques developed for the abdominal aorta.68 First, the hemodynamic forces of the thoracic aorta are significantly more aggressive and place greater mechanical demands on thoracic endografts. The potential for device migration, kinking, and late structural failures are important concerns. Second, greater flexibility is required of thoracic devices to conform to the natural curvature of the proximal descending aorta and to lesions with tortuous morphology. Third, because larger devices are necessary to accommodate the diameter of the thoracic aorta, arterial access is more problematic. This is an important concern given the greater proportion of TAA patients, relative to AAA patients, who are women, in whom access vessels tend to be smaller. Fourth, as with conventional open TAA repair, paraplegia remains a potential complication in the endovascular approach despite the absence of aortic cross-clamping.69,70 Lastly, the extent of TAAAs can often extend beyond the boundaries of the descending thoracic aorta and involve more proximal or distal aneurysms in the descending thoracic aorta.

Devices

Early clinical experiences with stent grafting of the thoracic aorta were based on the use of first-generation, homemade devices that were rigid and required large delivery systems (24F to 27F).74,75 Fortunately, several commercial manufacturers of abdominal endografts have created derivatives for the thoracic aorta with dramatic improvements over homemade devices. To date, none have been approved for use in the United States, but 3 are undergoing clinical trials for FDA approval (Figure 4; Table 7): TAG (W.L. Gore and Associates, Inc), Talent (Medtronic, Inc), and TX2 (Cook, Inc).

Although each device has unique features, all 3 employ the same basic structural design. The endoprostheses are composed of a stent (nitinol or stainless steel) covered with fabric (polyester or PTFE). The modular design of the TX2 system renders it less flexible than the TAG but more so than the Talent. The TAG device uses a delivery system that is specifically designed to limit distal migration. The design of
the proximal and distal ends varies, and some of the stents are uncovered proximally and distally (TX2), whereas others have an uncovered proximal end (Talent). In addition, the TX2 stent uses bars to facilitate fixation proximally and distally. Ultimately, each device has its advantages and disadvantages.

Requirements for Endovascular Repair

The evaluation of a patient for repair of their TAA considers their overall risk profile, evidence of rapid enlargement of the aneurysm, diameter ≥6 cm, or presence of symptoms. The suitability of the patient for endovascular repair is based on both clinical and anatomic considerations. Clinical parameters indicating a preference for the endovascular approach over the traditional approach remain to be defined but should be based on comparison of respective risk-benefit ratios. Although data from long-term follow-up studies and randomized, controlled trials are pending, endovascular stent grafting is currently reserved for high-surgical-risk and nonoperative patients who have suitable anatomic features.

General consensus has been established for anatomic prerequisites for endovascular repair (Table 8). Thorough preprocedural imaging is essential to fully characterize the lesion and access route. Detailed imaging evaluation can be obtained with spiral CT or MRI; however, without the availability of high resolution 3D renderings of the aorta, catheter-based angiography remains the “gold standard.” Measurements from imaging data are used to select the appropriate device diameter and length.

Determination of aneurysm location in relation to the left subclavian artery and celiac axis is of upmost importance. Successful TAA exclusion requires normal segments of native aorta at both ends of the lesion (the so-called landing zone or neck) of at least 15 to 25 mm to ensure adequate endoprosthesis—aortic wall contact and formation of a tight circumferential seal. Landing zones that are markedly angled or conical or that contain thrombus can result in poor fixation. Devices are oversized by 10% to 20% in diameter to provide sufficient radial force for adequate fixation.

The vascular access route for device introduction and delivery to the pathological target must be of sufficient size and of suitable morphology. Stent grafts are most commonly introduced through the femoral artery. Small-diameter, tortuous, and excessively calcified iliac arteries may preclude such access, requiring more proximal retroperitoneal exposure and direct cannulation of or creation of a graft conduit to the common iliac or abdominal aorta. Additionally, severe stenosis and tortuosity of the abdominal and thoracic aorta distal to the target are contraindications for endovascular repair.

In spite of the use of these criteria, treatment failures can occur, but the specific contributing factors and frequencies are currently unknown, particularly over the long-term. Thus, follow-up surveillance with serial CT scans at 1, 6, and 12 months and annually thereafter is recommended to monitor changes in aneurysm morphology, identify device failures, and detect endoleaks.

Clinical Experience

The literature on thoracic stent grafting consists mostly of small to medium-sized case series with short- to medium-term follow-up (Table 8). Nevertheless, these studies illustrate a consensual pattern of outcomes when viewed in aggregate. Overall, successful device deployment is achieved in 85% to 100% of cases, and periprocedural mortality ranges from 0% to 14%, falling within or below elective surgery mortality rates of 5% to 20%. As expected, outcomes have improved over time with accumulated technical expertise, use of commercially manufactured devices, and improved patient selection criteria. The recently published collective experiences of the EUROSTAR and United Kingdom Thoracic Endograft registries, the largest series to date (n = 249), demonstrate successful deployment in 87% of cases, 30-day mortality of 5% for elective cases, and paraplegia and endoleak rates of 4%. FDA phase II trial data from exclusive deployment of the Gore TAG endograft in 142 TAA patients reveal similar results: technical success in 98%, 30-day mortality of 1.5%, paraplegia in 3.5%, and endoleak in 8.8%.

These results cannot be directly compared with the outcomes of contemporary surgical studies. The majority of TAA patients who were repaired by the endovascular approach in these studies were older and sicker, having been deemed either high risk or not suitable for open surgical repair. For example, 52% of patients in the combined EUROSTAR and United Kingdom registries were preoperatively classified as ASA 3 or above (American Society of Anesthesiologists’ physical status classification that predicts procedural risk: 1 to 2 indicates low risk; 3, intermediate risk; 4 to 5, high risk; and 6, organ donor). In light of these patient substrate differences, endovascular TAA repair will likely play a substantial role in the management of high-risk patients. Furthermore, the applications of this less invasive therapeutic modality may broaden if endograft studies with lower-risk surgical candidates result in favorable mortality and morbidity gains, particularly as devices and techniques continue to evolve.

True comparisons between conventional therapy and the endovascular alternative can only be achieved after the completion of prospective, randomized, controlled trials. Although such trials are under way, a few studies have compared endovascular treatment with anatomically similar open-surgery historic controls. As part of the phase II Gore Excluder study, 19 TAA patients who were candidates for open repair received stent-graft therapy and were compared with a nonrandomized cohort of 10 patients who had under-

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**TABLE 8. Anatomic Requirements for Endovascular Repair of TAA**

<table>
<thead>
<tr>
<th>Requirement</th>
</tr>
</thead>
<tbody>
<tr>
<td>A proximal neck at least 15 to 25 mm from the origin of the left subclavian artery</td>
</tr>
<tr>
<td>A distal neck at least 15 to 25 mm proximal to the origin of the celiac artery</td>
</tr>
<tr>
<td>Adequate vascular access: absence of severe tortuosity, calcification, or atherosclerotic plaque burden involving the aortic or pelvic vasculature</td>
</tr>
<tr>
<td>The transverse diameter of the proximal and distal neck should be within the range that available devices can appropriately accommodate</td>
</tr>
</tbody>
</table>

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*References and further details can be found in the full article.*
TABLE 9. Summary Data on Studies of Endovascular Repair of TAA

<table>
<thead>
<tr>
<th>First Author and Year of Study Publication</th>
<th>n</th>
<th>Follow-Up, mo</th>
<th>Devices</th>
<th>Technical Success</th>
<th>30-Day Mortality, %</th>
<th>Long-Term Survival, %</th>
<th>Paraplegia, %</th>
<th>Endoleak, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dake 19985</td>
<td>103</td>
<td>22</td>
<td>Homemade</td>
<td>83% Complete thrombosis</td>
<td>9</td>
<td>73 (Actuarial 2 year)</td>
<td>3</td>
<td>24</td>
</tr>
<tr>
<td>Ehrlich 19985</td>
<td>10</td>
<td>NA</td>
<td>Talent</td>
<td>80% Complete thrombosis</td>
<td>10</td>
<td>NA</td>
<td>0</td>
<td>20</td>
</tr>
<tr>
<td>Cartes-Zumezu 200077</td>
<td>32</td>
<td>16</td>
<td>Excluder, Talent</td>
<td>90.6%</td>
<td>9.4</td>
<td>90.6 (32 Months)</td>
<td>3.1</td>
<td>15.4</td>
</tr>
<tr>
<td>Grabenwoger 200079</td>
<td>21</td>
<td>NA</td>
<td>Talent, Prograft</td>
<td>100%</td>
<td>9.5</td>
<td>NA</td>
<td>0</td>
<td>14.3</td>
</tr>
<tr>
<td>Greengberg 200079</td>
<td>25</td>
<td>15.4</td>
<td>Homemade</td>
<td>NA</td>
<td>20 (12.5 for elective, 33 for emergent)</td>
<td>NA</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>Termudom 200080</td>
<td>14</td>
<td>5.5</td>
<td>Homemade, Vanguard, Excluder</td>
<td>78.6%</td>
<td>14.3</td>
<td>NA</td>
<td>7.1</td>
<td>14.3</td>
</tr>
<tr>
<td>Najbi 200221</td>
<td>24</td>
<td>12</td>
<td>Excluder, Talent</td>
<td>94.7%</td>
<td>5.3</td>
<td>89.5 (1 Year)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Heijmen 200242</td>
<td>28</td>
<td>21</td>
<td>Talent, AneuRx, Excluder</td>
<td>96.4%</td>
<td>0</td>
<td>96.4 (Mean 21 months)</td>
<td>0</td>
<td>28.6</td>
</tr>
<tr>
<td>Schoder 20033</td>
<td>28</td>
<td>22.7</td>
<td>Excluder</td>
<td>100%, 89.3% Complete exclusion</td>
<td>0</td>
<td>96.1 (1 Year), 80.2 (3 years)</td>
<td>0</td>
<td>25</td>
</tr>
<tr>
<td>Marin 200324</td>
<td>94</td>
<td>15.4</td>
<td>Excluder, Talent</td>
<td>85.1%</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>24</td>
</tr>
<tr>
<td>Lepore 200341</td>
<td>21</td>
<td>12</td>
<td>Excluder, Talent</td>
<td>100%</td>
<td>9.5</td>
<td>76.2 (1 Year)</td>
<td>4.8</td>
<td>19</td>
</tr>
<tr>
<td>Sunder-Plassman 200335</td>
<td>45</td>
<td>21</td>
<td>Corvita, Stanford, Vanguard, AneuRx, Talent, Excluder</td>
<td>NA</td>
<td>6.7</td>
<td>NA</td>
<td>2.2</td>
<td>22.2</td>
</tr>
<tr>
<td>Oueil 200348</td>
<td>31</td>
<td>6</td>
<td>Excluder, Talent, Other commercial</td>
<td>NA</td>
<td>12.9</td>
<td>81.6 (1 Year)</td>
<td>6.5</td>
<td>32.3</td>
</tr>
<tr>
<td>Bergeron 200337</td>
<td>33</td>
<td>24</td>
<td>Excluder, Talent</td>
<td>NA</td>
<td>9.1</td>
<td>75.8 (Mean 24 months)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Czarny 200446</td>
<td>54</td>
<td>38</td>
<td>Excluder, Talent</td>
<td>94.4%</td>
<td>9.3</td>
<td>63 (3 Years event free)</td>
<td>0</td>
<td>27.8</td>
</tr>
<tr>
<td>Makaroun 200448</td>
<td>142</td>
<td>29.6</td>
<td>TAG</td>
<td>97.9%</td>
<td>1.5</td>
<td>75 (2-Year freedom from death)</td>
<td>3.5</td>
<td>8.8</td>
</tr>
<tr>
<td>Leurs 200450</td>
<td>249</td>
<td>1–60</td>
<td>Excluder, Talent, Zenith, Endofit</td>
<td>87%</td>
<td>10.4 (5.3 for elective, 27.9 for emergent)</td>
<td>80.3 (1 Year)</td>
<td>4</td>
<td>4</td>
</tr>
</tbody>
</table>

Complications

With the avoidance of aortic cross-clamping and prolonged iatrogenic hypotension, endovascular TAA repair was expected to result in lower incidences of paraplegia relative to conventional treatment. Indeed, this has held true, with paraplegia rates generally ranging from 0% to 5% in endovascular studies,68,75–90 whereas paraplegia occurs in 5% to 25% of open repair cases.56,58,64,65 Although low, these rates remain significant, especially because it is impossible to reimplant intercostal arteries in this setting. Some evidence suggests that the occurrence of paraplegia is associated with concomitant or prior surgical AAA repair and increased exclusion length because of the absence of lumbar and hypogastric collateral circulation.79,91 Adjunctive measures to further reduce spinal cord ischemic complication rates in endovascular TAA repair are being investigated.92

Endoleaks are the most prevalent of TAA stent-graft repair.93 Interestingly, the distribution of endoleak types also differs. TAA endoleaks occur more commonly at the proximal or distal attachment sites (type I endoleak), whereas the majority of AAA endoleaks are type II.94 It is generally accepted that type I endoleaks are more serious and require expeditious intervention because they represent direct communications between the aneurysm sac and aortic blood flow.95 Treatment options include transcatheter coil or glue embolization, balloon angioplasty, placement of endovascular graft extensions, and open repair.96,97

Despite the hostile hemodynamic conditions of the thoracic aorta, the anticipated complications of device migration and kinking have occurred infrequently and have been observed primarily with homemade devices and those with unsupported mid-graft segments.70 For both homemade and commercial endografts, however, questions about device durability and stability over the long term remain unanswered.

Future Directions

The cumulative worldwide experience with endovascular repair of thoracic aneurysms has highlighted not only its potential as an advance in treatment but also its limitations. Efforts are under way to expand the applicability of endovascular stent-graft technology in the thoracic aorta, challenging current anatomic constraints and clinical indications. A variety of exciting strategies are being explored and deserve mention.

Although current anatomic criteria limit thoracic stent-graft exclusion to lesions located at least 15 to 25 mm away from the origin of the left subclavian artery and celiac trunk, it is common for descending TAArs to be located within the proximal or distal neck length necessary for adequate fixa-
tion. At the proximal end, the landing zone can be extended by prophylactic left subclavian to left carotid artery transposition or bypass graft placement. Alternatively, the uncovered proximal portion of the Talent endograft can be placed across the left subclavian origin to achieve fixation without blocking flow. However, because case reports of inadvertent coverage of the left subclavian artery origin found no resulting complications, subsequent studies determined that such maneuvers may not be necessary as long as there is no obstruction of the right vertebral or carotid artery and the left internal mammary artery is not used as a coronary bypass conduit. Complications such as left arm ischemia were found to be rare, possibly owing to collateral blood supply via retrograde left vertebral flow. This is commonly referred to as the “subclavian steal” phenomenon. Most centers now intentionally cover the left subclavian origin when necessary and reserve secondary revascularization procedures for when related symptoms develop.

For even more proximal thoracic aneurysms involving the aortic arch, branched and fenestrated stent grafts are being developed to accommodate perfusion through the great vessels. Although feasibility has been demonstrated, it is already apparent that the required implantation techniques would have to be highly complex and demand considerable technical expertise. Some centers have thus been investigating techniques to create fenestrations intraoperatively after device deployment and coverage of critical branches.

In contrast, there are no easy management strategies to deal with a short distal neck. In this setting as well, fenestrated and branched grafts have been used in isolated cases, but the overall experience is very limited. Intentional coverage of the celiac artery is not recommended given the risk of hepatic and visceral ischemia. Although a normal superior mesenteric artery may provide collateral flow, no methods exist to predetermine whether such collateral supply would be sufficient. Furthermore, the celiac trunk may serve as a prominent source of retrograde endoleak if covered without adjunctive transcatheter occlusion. In distal aneurysms that involve both the descending thoracic and abdominal aorta, combined open AAA repair and endovascular TAA exclusion is a novel treatment approach under investigation.

Stent grafts are also being used to treat patients with diffuse aneurysmal disease involving the entire thoracic aorta. In such patients, the traditional surgical treatment is a 2-staged procedure named the “elephant trunk technique.” In the first stage, the ascending aorta and aortic arch are repaired via a median sternotomy, and an extra-long graft is used for reconstruction, which leaves the excess portion of the graft, the elephant trunk, dangling within the lumen of the remaining diseased aorta. In the second stage, the lesion in the descending aorta is repaired via a left thoracotomy, and the graft replacement is connected to the elephant trunk proximally. To bypass the need for thoracotomy, a few centers have successfully deployed thoracic stent grafts into the elephant trunk extension, altogether replacing the second stage of the traditional elephant trunk procedure.

Following closely on the heels of early clinical experiences with stent grafting for TAA repair, experimental application of this less invasive approach has been extended to a growing number of other pathologies of the thoracic aorta. Most noteworthy among these are aortic dissection, traumatic aortic injury, penetrating atherosclerotic ulcer, and aortic rupture. Some investigators believe that thoracic stent-graft technology may eventually yield the greatest impact on clinical care in the management of aortic dissections, because current treatment standards are far from optimal.

Conclusions

The emergence of endovascular stent grafting as an alternative therapy to open surgical repair of abdominal and thoracic aneurysms is an exciting advance. Although it is apparent that high-operative-risk patients will benefit from this technology, the exact role of stent grafting remains to be defined as we continue to accumulate long-term data and experience and as devices and techniques evolve. Instead of replacing conventional surgical treatment, endovascular repair will likely play a complementary role and offer a less invasive option in our treatment armamentarium. It is clear that the limitations of both approaches are distinct. Although what is considered high risk for surgery is defined by clinical parameters in terms of comorbidities and physiological reserve, contraindications for endovascular stent-graft treatment are defined by anatomical constraints. In this regard, each may fulfill the treatment gaps left by the other.

Disclosures

Dr. Dake has received research grants from Cook and W.L. Gore; has served on the speakers’ bureau of Cook; and has consulted for W.L. Gore. Dr. Katzen has consulted for and received educational grants from Cook, Boston Scientific, W.L. Gore, and Medtronic.

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Endovascular Repair of Abdominal and Thoracic Aortic Aneurysms
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Circulation. 2005;112:1663-1675
doi: 10.1161/CIRCULATIONAHA.105.541284
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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