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CHOOSING THE RIGHT DEVICE FOR THE PATIENT

Experts discuss how anatomy and technology affect durability.


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Progressive Aortic Aneurysm Disease

BY PROFESSOR MICHAEL LAWRENCE-BROWN, FRACS

Aortic aneurysm repair is one of the flagships of vascular intervention. Aortic aneurysms were feared in the age of syphilis when affected aortic arches protruded up into the neck, throughout the 19th century and in the earlier half of the 20th. Even when treatment became available in the 1950s with prosthetic grafts it still daunted many in the latter half of the 20th century and, despite the quantum leaps in treatment technologies in the 1990s and first decade of this century, there is still work to be done.

Gone are the days of estimating the size of an abdominal aneurysm by palpation and retreating from treatment if the aneurysm extended above the renal arteries. Technology and knowledge have brought accurate diagnosis of the size and extent of aneurysms. Treatment of general health, with measures against accelerators such as smoking and hypertension, is reducing the prevalence and rate of growth. Technical advances in surgical techniques and prostheses have reduced the risks and increased the efficacy of intervention. However, this does not mean that the challenge of aortic aneurysm disease has been met—rather that the challenges have changed. One of these challenges is not what we can do, but what we should do and when should we do it because now we can replace every segment of the aorta, including the arch. Sometimes, we have to decide whether to treat one segment or multiple segments at the same time or which segment to tackle first. We need to bear foremost in our minds that this is a progressive disease that may require further primary or secondary intervention.

The prevalence of aortic aneurysm rises from 2.5% in white men aged 65 years to 11% in those older than 80 years. The odds ratio of developing this disease increases from 0.4 in those whose ethnicity originates near the equator to 1 in those whose ethnicity comes from northern latitudes, progressively changing toward the pole.[1,2] Clearly, in the modern age, this is a disease that is influenced by genetic and environmental factors with age being the greatest risk factor. Herein lies the dilemma—treatment versus no treatment—from both an economic and a clinical risk-benefit perspective. The multicultural and ethnic mixing of modern societies may strengthen the aorta and behavioral influences may affect the rates of progression; however, with age, disease will progress.

Progression of the disease from the straight, nonbranched segments to the branched segments increases the risk and complexity of intervention by any technique until the risks outweigh the benefits. As the risk-benefit balance is steadily pushed toward a benefit with endovascular techniques in terms of quality life years, the challenge is to treat the entire length of the aorta, including the aortic root, without any or only minor adverse events.

The progression of aortic aneurysms is a reflection of the degenerative process of the aorta as a result of biological aging, constant pressure, and fatiguing pulsating forces—at times, we even feel the hammering to which our arteries are subjected. Mechanical forces weaken the aorta and balloon physics dictates that an aneurysm will expand and extend leading to tortuosity or rupture. Decussating fibers around major branches often hold the extensions back and angulation develops until the ballooning reaches the instability point. Then, the extensions progress, fascinatingly, along the line of major arteries derived from the fetal circulation, which defines those that are predisposed. Treating an aneurysm with a prosthesis demands that the prosthesis acts as a bridge from one secure bridge head to another, that we are not deceived by illusions of secure positioning and constant seal, that the repair will withstand potential worst-case disruptive force and last up to 20 years and that there may be a need for further repair or extension.

In this supplement engineers address the problems of securing and sealing at attachment sites and practicing physicians discuss the difficulties of matching the available prostheses to the pathological changes. The emphasis is on the underlying appreciation that the dynamics that caused the disease will progress along the aorta. Further intervention at the same or another site in the aorta is to be anticipated.

So, the modern challenge is to check all the predisposed arteries whenever examining the cardiovascular system, to make and install prostheses that address all of the problems in the progressively weakening aorta at an optimum time and to provide for more quality life years in a cost-effective and biologically beneficial way. Can we do this with current materials and techniques or do we need another quantum leap, like the one that was made in advancing to endovascular therapy as it is today? While this continues to be tested, every measured improvement takes us further along the way. ■

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1. Norman PE, Jamrozik K, Lawrence-Brown MM, et al. Population based randomized controlled trial on impact of screening on mortality from abdominal aortic aneurysm. Br Med J. 2004;329:1259-1262A.
2. Norman PE, Lawrence-Brown M, Semmens J, Mai Q. The anatomical distribution of iliac aneurysms: is there an embryological basis? Eur J Vasc Endovasc Surg. 2003;25:82-84.

Proximal Abdominal Aortic Aneurysm Necks

The clinical issues and challenges that this anatomy poses for endovascular graft design.

BY DAVID HARTLEY, FIR; MATTHEW EAGLETON, MD; AND BLAYNE ROEDER, PHD

Endovascular aneurysm repair (EVAR) for abdominal aortic aneurysms (AAAs) has revolutionized our approach to treating this disease. For more than a decade, this technology has undergone intense scrutiny, which has allowed for the rapid development and refinement of many generations of stent grafts—with careful attention applied to the mechanics of deliverability, profile, ease of use, and durability. These assessments have not only improved our understanding of the technology, but also caused the vascular community to begin to reevaluate our understanding of the pathobiology of aortic diseases.

It has become clear that not only the technology but also disease progression plays an important role in the durability of endovascular aortic therapy. This is particularly important given the increasing longevity of the elderly population, even after aortic aneurysm repair.[1] One of the key features of EVAR that portends its success is addressing the proximal attachment site. In this article, we examine some of the clinical features that make the proximal neck of AAAs challenging to address, the evolution of EVAR device development that attempted to overcome these issues, and current device designs that may allow us to provide a durable repair in the face of progressive disease.

CLINICAL CHALLENGES WITH THE PROXIMAL AORTIC NECK

The proximal aortic neck is the crux for long-term EVAR durability. Endografts must achieve a seal in this location to ensure exclusion of the aneurysm without developing type I endoleaks, and the device must achieve fixation to prevent migration. There are several morphologic features that can hinder the ability of an endograft to achieve adequate fixation and seal within the proximal aortic neck. These features include alterations in neck composition (such as the presence of thrombus or calcification), neck angulation, and undesirable neck length and diameter. Even as we improve the devices and push the boundaries of what may provide a durable repair, these unfavorable features remain the key challenges that must be overcome. Some of these features lead to mechanical issues that may be adequately addressed through device engineering, whereas others may be hallmarks of impending disease progression that is best managed through appropriate device or patient selection.

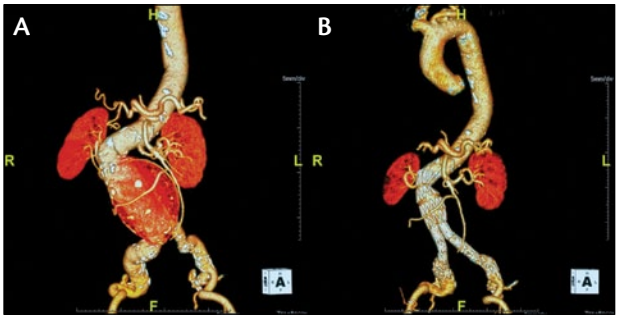

Figure 1. Three-dimensional reconstruction of a patient with an infrarenal AAA with significant angulation in the neck (A). Despite the angulation, the patient had successful placement of an endovascular graft that excluded the aneurysm (B).

Assessment of these issues, however, is not new, as arguments for and against aggressive EVAR in unfavorable anatomy have been ongoing for well over a decade.[2-5] Instructions for use (IFUs) for EVAR have historically recommended more ideal aortic necks—those lacking thrombus/calcification, with longer (≥ 15 mm), parallel, nondilated walls that are relatively free of angulation. With increased experience, however, these recommendations have been challenged. Outcomes of EVAR when used in more challenging necks (Figure 1) have been inconsistent, with reports highlighting the difficulties of EVAR in hostile necks[6-8] versus successful treatment of patients who are well outside the IFU.[9,10]

Thrombus and Calcification

Most IFUs recommend against EVAR in the setting of significant neck thrombus and calcification. There have been few direct assessments of the durability of stent grafts in these settings. One difficulty in analyzing this morphologic feature is the lack of a universally agreed-upon method of quantifying the degree of calcification and thrombus within the proximal neck. Bastos and colleagues directly assessed outcomes related to the presence of neck thrombus and demonstrated that its presence (in ≥ 50% of neck circumference) was associated with endograft migration of > 10 mm (9.3% vs 2.3%) on univariate analysis.[4] Cox multivariate analysis, however, identified the lack of an active fixation system as the only significant factor for device migration, although nearly 20% of patients with neck thrombus in this series experienced device migration of at least 5 mm. This may become a significant factor, as shorter proximal necks are thought to be permissible.

Wyss et al demonstrated that the presence of neck thrombus may have a protective effect against the development of long-term complications following EVAR, whereas the presence of calcification, particularly when associated with neck angulation, was associated with the development of complications.[5] However, the adverse role of neck calcification has been disputed,[11] with aneurysm sac regression occurring in the presence of less severe aortic neck calcification.[12]

Angulation

Proximal neck angulation has been extensively studied and found to be a significant factor affecting the success of EVAR. Grisafi et al demonstrated that the presence of an infrarenal neck angle > 45° was associated with a significantly increased risk of initial type IA endoleak.[11] Neck angulation can be lessened, however, with device placement. After successful EVAR, the degree of both suprarenal and infrarenal neck angulation decreases, with the angles continuing to "straighten" for up to 3 years postoperatively,[13] which may be independent of the type of device used.[14]

Neck Length

Experimental modeling of proximal fixation strength in the aortic neck demonstrates that, among a variety of graft designs, pull-out forces significantly vary, and these pull-out forces can be lowered by shortening the length of the proximal seal, likely directly related to graft design.[15] Data from the EUROSTAR registry were used to assess outcomes for patients with short infrarenal necks.[16] Patients were categorized into one of three groups according to the neck length: > 15 mm, 11 to 15 mm, and ≤ 10 mm. The rate of type IA endoleaks was significantly greater for patients with neck lengths ≤ 10 mm (11%). At follow-up, freedom from type I endoleak was 97% in those with > 15 mm necks, but only 90% in those with 11- to 15-mm necks, and 89% in those with ≤ 10-mm necks. No differences were observed with respect to device migration, late conversion, aneurysm rupture, or secondary intervention. Some of the current devices have adjusted their IFUs to include treatment of shorter-necked aneurysms.

Neck Diameter and Dilation

Analysis of the EUROSTAR database by Leurs et al demonstrates that 32% of patients experience neck dilation following EVAR, with approximately 10% of these having migration associated with dilation.[17] In this analysis, risk factors for neck dilation included larger device main body diameter and graft oversizing by at least 20%, whereas less frequent neck dilation was observed with larger baseline neck diameters and the absence of a suprarenal bare stent. In contrast, Cao et al reported aortic neck dilation after EVAR was associated with neck circumferential thrombus, large preoperative aortic necks, and large AAA diameters.[18] Post-EVAR neck dilation has been observed at rates as high as 63% in patients who have thrombus-lined proximal necks.[4] Neck dilation, especially in cases of thrombus-lined or large necks, may be representative of underrecognized diseases and dilation secondary to disease progression. This process does not occur quickly, which may explain why problems with stent graft fixation and sealing may not become apparent for several years after the initial EVAR procedure.[19,20]

Hostile Neck

Specific analysis of individual factors is difficult given that most patients without an ideal neck have multiple morphologic features that create a "hostile" neck. In a single-center series of 552 patients, Stather and colleagues demonstrated that the presence of hostile neck anatomy (defined as diameter > 28 mm, angulation > 60°, length < 15 mm, and neck flare and thrombus) was not associated with alterations in technical success, 30-day mortality, 30-day type IA endoleak development, or 30-day reintervention rates.[21] Outcomes after 30 days, however, demonstrate an increased rate of type I endoleaks (9.5% vs 4.5%; $P = .02$) in those with hostile necks, but no differences with regard to device migration, sac expansion, aneurysm rupture, or 5-year mortality. Patients with hostile necks, however, required significantly more reinterventions (23% vs 11%; $P < .01$), as a result of the need to treat type IA endoleaks.

Binary logistic regression showed that reinterventions, technical failure, and late type I endoleak development

were significantly increased in patients with increased neck diameters (> 28 mm). Similar outcomes showing early technical success have been demonstrated in other single-center series,[7] with favorable outcomes in hostile neck anatomy being attributed to the use of suprarenal fixation.[22] Outcomes for more recently available stent graft systems have shown similar early outcomes in patients with hostile neck anatomy, but long-term assessment of their durability is not yet available.[23-25]

Stather et al performed a meta-analysis of EVAR in patients with hostile necks (defined as length < 15 mm, diameter > 28 mm, and angulation > 60°) (n = 3,039) compared to those with a favorable neck anatomy (n = 8,920).[26] In contrast to the single-center series, this analysis demonstrated that the presence of a hostile neck was associated with an increase in 30-day mortality (2.4% vs 3.5%; $P < .01$), intraoperative adjuncts (8.8% vs 15.4%; $P = .01$), and 30-day migration (0.9% vs 1.6%; $P < .01$). When all three hostile neck criteria were present, primary technical success was reduced to 94%. Although those with a hostile neck had a significantly increased risk for early and late type IA endoleaks and required more secondary procedures, there were no differences in long-term aneurysm-related mortality, all-cause mortality, migration, or aortic expansion.

ENDOVASCULAR GRAFT DESIGN FOR A DURABLE PROXIMAL SEAL

Overcoming the aforementioned challenges of the AAA proximal neck has been the primary goal in endovascular graft design since the first placement of an endovascular graft in a human to treat AAA in 1990.[27] For such a novel treatment modality, the baseline that had to be matched was the known performance of the open operation, in which a surgical graft is attached to the vessel wall with sutures. In the open operation, it was recognized that for a satisfactory seal and reliable attachment, it was necessary to suture the proximal anastomosis to a healthy vessel.

Twenty-five years of endovascular graft design has focused on meeting this requirement, with designs evolving from devices built by the implanting physicians for their specific patients in the operating room to highly engineered and extensively tested devices available today from several manufacturers. The methods of seal and attachment have varied, and include balloon-expanded stainless steel stents (the giant Palmaz [Cordis Corporation, Bridgewater, NJ]), self-expanding stents with infrarenal active fixation, self-expanding infrarenal stents with column strength but no active fixation, self-expanding stents with a suprarenal bare stent without active fixation, and steel self-expanding stents with a bare suprarenal stent with active fixation (Figure 2).

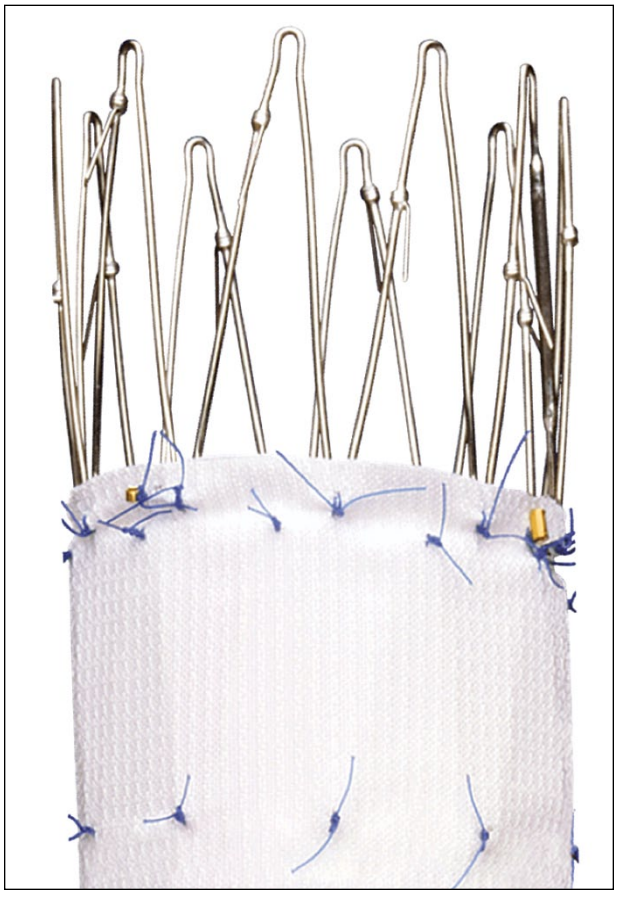

Figure 2. Proximal design of an endovascular graft including a self-expanding stent with suprarenal fixation and an internal self-expanding stent for sealing.

Until the analysis by Liffman et al,[28] originally presented in 1999, there had been little appreciation for the nature of the forces being applied to the proximal attachment stents. There was even less appreciation for the extent by which the relentless pulsation forces could bring about fatigue failure of the metallic and fiber components on the most proximal aspect of the endovascular graft, which can cause migration, with and without component failure. Some of the robust designs of this early phase, enhanced by detailed improvements, have survived to the present day and have been the platform for further development of specialized devices to address the hostile infrarenal neck.

Today, alternative infrarenal stent graft designs with unique means of excluding the aneurysm and achieving proximal seal and attachment are also being conceived and evaluated in clinical trials.[29,30] The surgical practice, in the absence of a satisfactory infrarenal neck, was to suture the graft to the healthy suprarenal aorta (and provide flow to the renal and any visceral vessels other-

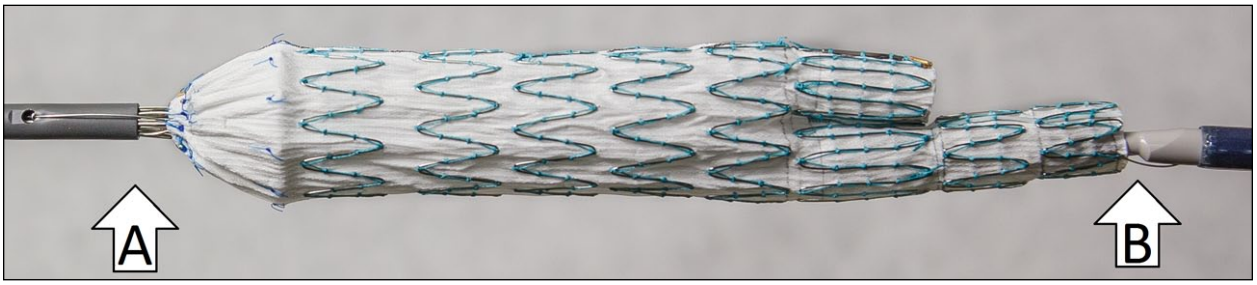

Figure 3. Multistage, controlled delivery of an endovascular graft is accomplished with multiple constraints. Retraction of the delivery sheath demonstrates proximal constraints (bare stent inside cap) (A) and a distal constraining wire (B).

wise occluded) by implantation or bypass procedures. As such, the alternative endovascular strategy is also to move more proximal into the visceral aorta for better seal and attachment in healthy vessels. Again mimicking the surgical approach, endovascular devices were developed, beginning as early as 1997, with the intent to place the sealing component above the renal arteries and supply flow to the renal and the mesenteric vessels with fenestrations and/or side branches.[31] Continued development of devices targeted toward a more proximal seal continues today.[32-34]

Similar to open repair, the primary design objective of AAA endovascular grafts is simply to prevent aneurysm rupture and subsequent patient death. However, durable exclusion of the aneurysm sac from hemodynamic pressure requires that several interrelated design functions and specific performance goals be achieved to meet this primary design objective. First, the endovascular graft delivery system must have the ability to accurately deploy the graft in its intended location. Once placed in its intended landing site, the endovascular graft must provide a proximal seal and prevent its migration. Most importantly, the graft must provide these functions for the life of the patient; structural durability of the device is paramount. Herein is a discussion of these fundamental design features and the performance criteria required to achieve these design functions.

Deployment Accuracy

Achieving reliable and accurate deployment is critical to the long-term success of the repair. Failure of the endovascular graft to deploy and subsequent need for conversion to open repair puts the patient at high risk. Buth et al reported a perioperative mortality rate of 22% for patients who were converted to open repair in the EUROSTAR study.[35] Although the majority of the deployment failures in this study were related to early device designs, they underscore the importance of deployment reliability. In addition to reliability, deployment accuracy of the endovascular graft system has a significant effect on the success of the repair, specifically the ability to attain adequate proximal seal. Consider the AAA with a 15-mm-long proximal neck. If deployment accuracy can only be expected to be within 5 mm, the resulting seal zone may only be 10 mm in length, or worse yet, a renal artery may be covered.

Deployment accuracy is most critical when the neck available for seal is complicated with a short length, angulation, calcification, and/or thrombus. Multistaged, controlled delivery facilitates accurate placement of the endograft,[36,37] which in turn can maximize the amount of healthy aorta available for seal (Figure 3). However, it is important to note that specific aortic features (eg, a short neck, angulation, calcification, and/or thrombus) that require a precise landing zone may also make accurate endograft placement more difficult and result in an increased number of procedural complications.[38]

Radial Force and Proximal Seal

Once placed in a stable position, the endovascular graft must inhibit blood from leaking around the proximal seal (type IA endoleaks). Stents at the proximal end of the graft must exert adequate radial force, or sealing pressure, to keep the graft against the aortic wall throughout the cardiac cycle and potentially other biomechanical motions to prevent type I endoleaks. The radial force produced by stents varies based on the extent of oversizing, and thus proper oversizing is critical in maintaining a seal in the short- and long-term. Endovascular grafts are designed and tested to maintain adequate radial pressure over a specified range of oversizing. These oversizing recommendations are explicitly defined in the IFU, and oversizing outside these bounds risks complications such as endoleaks,[39] continued aneurysm growth and/or migration,[40] or endovascular graft collapse.[41]

The mechanical properties and long-term stability of the aorta in the seal zone must also be considered in selecting an appropriate seal, so that the proximal endo-

Figure 4. An endovascular graft incorporating fenestrations and scallops to accommodate visceral vessels and allow sealing in the suprarenal aorta (A). Alternative designs incorporate features such as pivoting fenestrations to allow for variability in visceral vessel location (B).

vascular graft design can take advantage of that sealing zone. As previously stated, short-length seal zones, large neck diameters, significant angulation, the presence of thrombus, and calcification may increase the risks for type I endoleaks and sac expansion. These increased risks may not be a result of limitations in endograft design, but rather limitations in the durability of aortic seal zones with these features.

Architects and civil engineers have understood for thousands of years that there are specific requirements for designing foundations so that a structure is stable and durable for centuries to come. These requirements have less to do with the structural design capabilities of concrete, steel, or wood, but the ability of the earth to be stable under the weight of a building. We are only just beginning to understand these tradeoffs for endovascular grafts, especially in terms of how the seal zone of an endovascular graft interacts with a hostile neck. Rather than pushing the limits of infrarenal EVAR into a less-than-adequate seal zone, branched and fenestrated endovascular grafts were developed to take advantage of the additional suprarenal aortic segment, effectively increasing the amount of sealing zone available (Figure 4).[31]

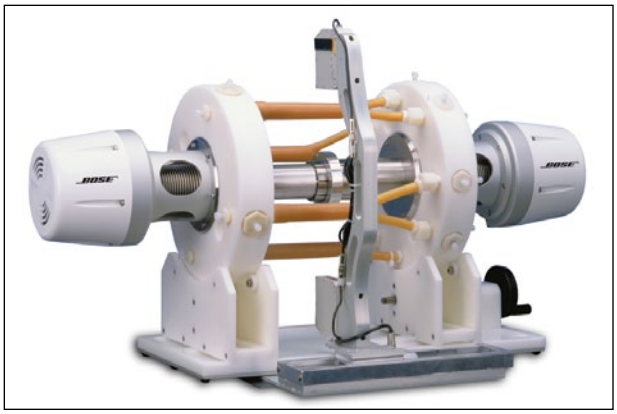

Figure 5. Test setup used to evaluate fatigue durability of a bifurcated endovascular graft to treat AAAs. Devices sit inside the bifurcated tubes as the tubes are subjected to physiologic motion. Laser measurement tools are used to measure the motion of the tubes.

Migration Resistance

In order to maintain a durable seal and exclude the aneurysm for the life of the patient, the endovascular graft must maintain its position relative to the aorta. Endograft migration can lead to late failure of the repair, specifically, type I endoleak, aneurysm rupture, and death. Endovascular grafts are subject to a hemodynamically challenging environment in which they must resist the physiologic forces associated with blood flow. Fluid mechanics analyses show that bifurcated aortic endovascular grafts are subject to cyclic forces on the order of 10 N, acting to displace the graft in a caudal direction for the life of the patient.[28] As previously described, many means of fixation have been utilized in commercially available endovascular grafts, including columnar strength, iliac fixation, bare stents, and active fixation (eg, hooks or barbs) (Figure 2). Nonclinical studies comparing grafts with and without active fixation have demonstrated that endovascular grafts with active fixation have higher migration resistance (ie, force required to displace them from the aorta) than those without active fixation.[42-44] These findings have been supported by lower migration rates of devices with active suprarenal fixation in clinical use.[40,45-48]

Fatigue Durability

Finally, the endovascular graft must be durable in order to maintain its function for the life of the patient. Endovascular grafts must be evaluated in all modes where cyclic (fatigue) loads are expected. Primary cyclic loads are a result of pulsatile blood flow. However, the mechanical loads and arterial motions from other sources, such as respiration or other bodily motions, also need to be considered. The aggregate effects of these loads on all

components of the endovascular graft (eg, stents, graft, sutures, etc.) need to be thoroughly evaluated. Clinical use of early endovascular grafts has elucidated many potential failure modes. These failures provided the opportunity to develop new graft designs and, in parallel, new test methods to evaluate for potential failure modes. The result is mature testing equipment (Figure 5) and standards for endovascular testing.[49] Standards typically require testing to be completed for a 10-year equivalent of 400 million cycles.

SUMMARY

The key challenges in achieving a stable and durable proximal seal in EVAR include inadequate length of healthy aorta for sealing, large neck diameters, and the presence of thrombus or calcification. These challenges have become increasingly critical as EVAR is disseminated to more patients, especially those whose proximal neck anatomy challenges IFU recommendations. These complex anatomies present key challenges to endovascular graft design. Engineering requirements for device deployment, proximal sealing, migration resistance, and durability were reviewed relative to these key challenges. Although advancements in endovascular graft design continue to push the indications for EVAR, it remains clear that healthy aorta is required for adequate fixation of the endograft to prevent migration and to maintain a durable seal without endoleaks. ■

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1. Mani K, Bjorck M, Lundkvist J, Wanhainen A. Improved long-term survival after abdominal aortic aneurysm repair. Circulation. 2009;120:201-211.
2. Albertini JN, Kalliafas S, Travis S, et al. Anatomical risk factors for proximal perigraft endoleak and graft migration following endovascular repair of abdominal aortic aneurysms. Eur J Endovasc Surg. 2000;19:308-312.
3. Gitlitz DB, Ramaswami G, Kaplan D, et al. Endovascular stent grafting in the presence of aortic neck filling defects: Early clinical experience. J Vasc Surg. 2001;33:340-344.
4. Bastos Goncalves F, Verhagen HJ, Chinsakchai K, et al. The influence of neck thrombus on clinical outcome and aneurysm morphology after endovascular aneurysm repair. J Vasc Surg. 2012;56:36-44.
5. Wyss TR, Dick F, Brown LC, Greenhalgh RM. The influence of thrombus, calcification, angulation, and tortuosity of attachment sites on the time to the first graft-related complication after endovascular aneurysm repair. J Vasc Surg. 2011;54:965-971.
6. Sternbergh WC 3rd, Carter G, York JW, et al. Aortic neck angulation predicts adverse outcome with endovascular abdominal aortic aneurysm repair. J Vasc Surg. 2002;35:482-486.
7. Aburahma AF, Campbell JE, Mousa AY, et al. Clinical outcomes for hostile versus favorable aortic neck anatomy in endovascular aortic aneurysm repair using modular devices. J Vasc Surg. 2011;54:13-21.
8. Fulton JJ, Farber MA, Sanchez LA, et al. Effect of challenging neck anatomy on mid-term migration rates in AneuRx endografts. J Vasc Surg. 2006;44:932-937; discussion 937.
9. Torsello G, Troisi N, Donas KP, Austermann M. Evaluation of the Endurant stent graft under instructions for use vs off-label conditions for endovascular aortic aneurysm repair. J Vasc Surg. 2011;54:300-306.
10. Fairman RM, Velazquez OC, Carpenter JP, et al. Midterm pivotal trial results of the Talent low profile system for repair of abdominal aortic aneurysm: analysis of complicated versus uncomplicated aortic necks. J Vasc Surg. 2004;40:1074-1082.
11. Grisafi JL, Rahbar R, Nelms J, et al. Challenging neck anatomy is associated with need for intraoperative endovascular adjuncts during endovascular aortic aneurysm repair (EVAR). Ann Vasc Surg. 2011;25:729-734.
12. Kaladji A, Cardon A, Abouliatim I, et al. Preoperative predictive factors of aneurysmal regression using the reporting standards for endovascular aortic aneurysm repair. J Vasc Surg. 2012;55:1287-1295.
13. van Keulen JW, Moll FL, Arts J, et al. Aortic neck angulations decrease during and after endovascular aneurysm repair. J Endovasc Ther. 2010;17:594-598.
14. Hoshina K, Akai T, Takayama T, et al. Outcomes and morphologic changes after endovascular repair for abdominal aortic aneurysms with severely angulated neck: a device specific analysis. Circ J. 2013;77:1996-2002.
15. Bosman WM, Steenhoven TJ, Suarez DR, et al. The proximal fixation strength of modern EVAR grafts in a short aneurysm neck. An in vitro study. Eur J Vasc Endovasc Surg. 2010;39:187-192.
16. Leurs LJ, Kievit J, Dagnelie PC, et al. Influence of infrarenal neck length on outcome of endovascular abdominal aortic aneurysm repair. J Endovasc Ther. 2006;13:640-648.
17. Leurs LJ, Stultiens G, Kievit J, et al. Adverse events at the aneurysmal neck identified at follow-up after endovascular abdominal aortic aneurysm repair: how do they correlate? Vascular. 2005;13:261-267.
18. Cao P, Verzini F, Parlani G, et al. Predictive factors and clinical consequences of proximal aortic neck dilatation in 230 patients undergoing abdominal aorta aneurysm repair with self-expandable stent-grafts. J Vasc Surg. 2003;37:1200-1205.
19. Verhoeven BA, Waasdorp EJ, Gorrepati ML, et al. Long-term results of Talent endografts for endovascular abdominal aortic aneurysm repair. J Vasc Surg. 2011;53:293-298.
20. Espinosa G, Ribeiro Alves M, Ferreira Caramalho M, et al. A 10-year single-center prospective study of endovascular abdominal aortic aneurysm repair with the talent stent-graft. J Endovasc Ther. 2009;16:125-135.
21. Stather PW, Sayers RD, Cheah A, et al. Outcomes of endovascular aneurysm repair in patients with hostile neck anatomy. Eur J Vasc Endovasc Surg. 2012;44:556-561.
22. Lee JT, Ullery BW, Zarins CK, et al. EVAR deployment in anatomically challenging necks outside the IFU. Eur J Vasc Endovasc Surg. 2013;46:65-73.
23. Bachoo P, Verhoeven ELG, Larzon T. Early outcome of endovascular aneurysm repair in challenging aortic neck morphology based on experience from the GREAT C3 registry. J Cardiovasc Surg. 2013;54:573-580.
24. Smeds MR, Jacobs DL, Peterson GJ, Peterson BG. Short-term outcomes of the C3 excluder for patients with abdominal aortic aneurysms and unfavorable proximal aortic seal zones. Ann Vasc Surg. 2013;27:8-15.
25. Mwipatayi BP, Picardo A, Wong J, et al. Endovascular repair of abdominal aortic aneurysms with reverse taper neck anatomy using the Endurant stent-graft: analysis of stent-graft oversizing. J Endovasc Ther. 2013;20:514-522.
26. Stather PW, Wild JB, Sayers RD, et al. Endovascular aortic aneurysm repair in patients with hostile neck anatomy. J Endovasc Ther. 2013;20:623-637.
27. Parodi JC, Palmaz JC, Barone HD. Transfemoral intraluminal graft implantation for abdominal aortic aneurysms. Ann Vasc Surg. 1991;5:491-499.
28. Liffman K, Lawrence-Brown MM, Semmens JB, et al. Analytical modeling and numerical simulation of forces in an endoluminal graft. J Endovasc Ther. 2001;8:358-371.
29. Karthikesalingam A, Cobb RJ, Khoury A, et al. The morphological applicability of a novel Endovascular Aneurysm Sealing (EVAS) System (Nellix) in patients with abdominal aortic aneurysms. Eur J Vasc Endovasc Surg. 2013;46:440-445.
30. Mehta M, Valdés FE, Nolte T et al. One-year outcomes from an international study of the Ovation abdominal stent graft system for endovascular aneurysm repair. J Vasc Surg. 2014;59:65-73.
31. Park JH, Chung JW, Choo IW, et al. Fenestrated stent-grafts for preserving visceral arterial branches in the treatment of abdominal aortic aneurysms: preliminary experience. J Vasc Interv Radiol. 1996;7:819-823.
32. Greenberg RK, Sternbergh WC, Makaroun M, et al. Intermediate results of a United States multicenter trial of fenestrated endograft repair for juxtarenal abdominal aortic aneurysms. J Vasc Surg. 2009;50:730-737.
33. Quiñones-Baldrich WJ, Holden A, Mertens R, et al. Prospective, multicenter experience with the Ventana fenestrated system for juxtarenal and pararenal aortic aneurysm endovascular repair. J Vasc Surg. 2013;58:1-9.
34. Kitagawa A, Greenberg RK, Eagleton MJ, Mastracci TM. Zenith p-Branch standard fenestrated endovascular graft for juxtarenal abdominal aortic aneurysms. J Vasc Surg. 2013;58:291-300.
35. Buth J, Laheij RJF; on behalf of the EUROSTAR Collaborators. Early complications and endoleaks after endovascular abdominal aortic aneurysm repair: report of a multicenter study. J Vasc Surg. 2000;31:134-146.
36. Chuter TA. The choice of stent-graft for endovascular repair of abdominal aortic aneurysm. J Cardiovasc Surg. 2003;44:519-525.
37. Brunkwall J. How to design the optimal stent graft—what have we learnt? Scand J Surg. 2008;97:191-194.
38. Hovsepian DM, Hein AN, Pilgram TK, et al. Endovascular abdominal aortic aneurysm repair in 144 patients: correlation of aneurysm size, proximal aortic neck length, and procedure-related complications. J Vasc Interv Radiol. 2001;12:1373-1382.
39. Mohan IV, Laheij RJ, Harris PL; on behalf of the EUROSTAR Collaborators. Risk factors for endoleak and the evidence for stent-graft oversizing in patients undergoing endovascular aneurysm repair. Eur J Vasc Endovasc Surg. 2001;21:344-349.
40. Sternbergh WC, Money SR, Greenberg RK, et al; for the Zenith Investigators. Influence of endograft oversizing on device migration, endoleak, aneurysm shrinkage, and aortic neck dilation: results from the Zenith multicenter trial. J Vasc Surg 2004;39:20-26.
41. Idu MM, Reekers JA, Balm R, et al. Collapse of a stent-graft following treatment of a traumatic thoracic aortic rupture. J Endovasc Ther. 2005;12:503-507.
42. Veerapen R, Dorandeu A, Serre I, et al. Improvement in proximal aortic endograft fixation: an experimental study using different stent-grafts in human cadaveric aortas. J Endovasc Ther. 2003;10:1101-1109.
43. Resch T, Malina M, Lindblad B, et al. The impact of stent design on proximal stent-graft fixation in the abdominal aorta: an experimental study. Eur J Vasc Endovasc Surg. 2000;20:190-195.
44. Malina M, Lindblad B, Ivancev K, et al. Endovascular AAA exclusion: will stents with hooks and barbs prevent stent-graft migration? J Endovasc Surg. 1998;5:310-317.
45. Zarins CK, Bloch DA, Crabtree T, et al. Stent graft migration after endovascular aneurysm repair: importance of proximal fixation. J Vasc Surg. 2003;38:1264-1272.
46. Tonnessen BH, Sternbergh WC, Money SR. Mid- and long-term device migration after endovascular abdominal aortic aneurysm repair: a comparison of AneuRX and Zenith endografts. J Vasc Surg. 2005;42:392-401.
47. Abbruzzese TA, Kwolek CJ, Brewster DC, et al. Outcomes following endovascular abdominal aortic aneurysm repair (EVAR): an anatomic and device-specific analysis. J Vasc Surg. 2008;48:19-28.
48. Greenberg RK, Chuter TA, Cambria RP, et al. Zenith abdominal aortic aneurysm endovascular graft. J Vasc Surg. 2008;48:1-9.
49. ANSI/AAMI/ISO 25539-1:2003/(R)2009 Cardiovascular implants— Endovascular devices— Part 1: Endovascular prostheses.

The Distal Seal Zone in AAA Repair

A facet of EVAR that is not to be overlooked.

BY THEODOSIOS BISDAS, MD, AND GIOVANNI TORSELLO, MD

Compared to the open surgical approach, endovascular aneurysm repair (EVAR) has demonstrated a clear benefit of lower perioperative mortality and morbidity, as well as comparable long-term survival rates in randomized controlled trials.[1] However, the higher reintervention rates remain the Achilles' heel of EVAR (relative risk, 2.54; 95% confidence interval, 1.58–4.08).[1] Hence, the ongoing refinement of the new generation of endografts is now targeting the reduction of device-related reinterventions.[2] Most of the attention regarding secondary procedures has been focused on different anatomic challenges at the proximal sealing zone (angulated or short necks, calcifications or thrombus) and into the aneurysm sac (type II endoleak).[3] However, the distal seal zone (DSZ) also presents a distinct set of challenges but has received less attention in the literature. In this article, we discuss the complexity of the DSZ and present different endovascular solutions to overcome anatomic and technical challenges.

HOSTILE DSZ

There is no clear definition of a hostile DSZ in the literature. In this article, we use the term "hostile DSZ" to describe anatomic challenges at the distal aortic neck and in the common iliac arteries. In contrast to the proximal neck, the DSZ can complicate endograft sealing not only through progression of the aneurysmatic disease but also through the presence of stenotic or tortuous iliac vessels. The following scenarios could lead to poor outcomes after endograft implantation: (1) aneurysmatic degeneration (at the primary procedure or further progression of the disease) at the level of the common or internal iliac arteries, (2) narrow distal neck or stenotic iliac arteries, and (3) tortuous iliac vessels.

ANEURYSMATIC DSZ OR FURTHER DISEASE PROGRESSION POST-EVAR

Epidemiological studies have shown that in 25% of patients older than 65 years who have an abdominal aortic aneurysm (AAA), the disease extends into one or both common iliac arteries, and in 7% of these patients, the disease extends into the internal iliac arteries.[4] External iliac artery aneurysms are extremely rare. It is speculated that the external iliac artery is resistant to aneurysm formation because it arises from the extra-embryonic anlage, in contrast to the common and internal iliac arteries, which arise from the somites.[4] A key procedural issue with aneurysmatic iliac arteries is to achieve a compact DSZ to avoid a type IB endoleak or distal endograft migration in the long run.

Several endovascular approaches have been suggested to achieve a safe landing zone in aortoiliac aneurysms or to repair further aneurysmatic degeneration due to progression of the disease. The first approach is to extend the iliac limb into the external iliac artery by embolizing or overstenting the internal iliac artery.[5] However, occlusion of the internal iliac artery may cause buttock claudication, impotence, and bowel necrosis in up to 55% of patients.[5] An alternative strategy is the "bell-bottom" technique, which has shown encouraging midterm outcomes (2.3% type IB endoleaks in 89 patients with common iliac artery aneurysms < 30 mm).[6] However, common iliac artery aneurysms > 25 mm often cannot be effectively treated with this technique. Lobato et al established the "sandwich" technique to treat complex aortoiliac or isolated iliac aneurysms in 40 patients; the group showed 100% technical success and a primary patency rate of 94% after a mean follow-up period of 12 months.[7] Yet, long-term evaluation of the technique is needed.

Use of an Iliac Side Branch Device for the DSZ

In this context, use of an iliac side branch device (Zenith Branch Iliac Graft*, Cook Medical, Bloomington, IN) provides a safe and effective approach to treat common or internal iliac artery aneurysms either at the primary procedure or due to the progression of the disease post-EVAR (Figure 1). Our early experience with endovascular repair of common iliac artery aneurysms < 26 mm in diameter with the straight iliac side branch device showed very low major morbidity (4.6%) and excellent

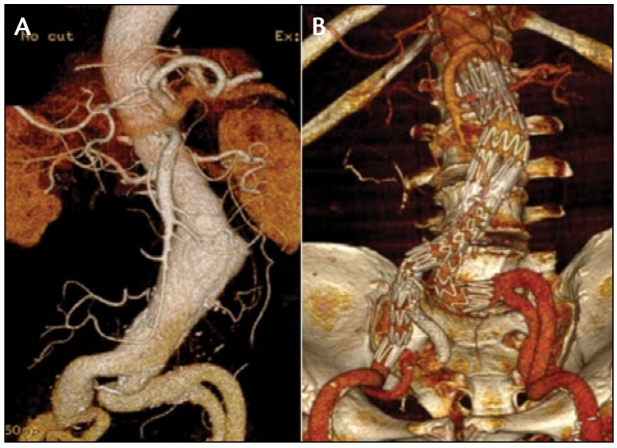

Figure 1. An aorto-mono-iliac aneurysm with a challenging DSZ consisting of an aneurysmatic and tortuous right common iliac artery (A). CT angiography (CTA) 3 years post-EVAR showing the successful exclusion of the aneurysm with a bifurcated endograft (Zenith) supported with an iliac side branch device for the right common iliac artery (B).

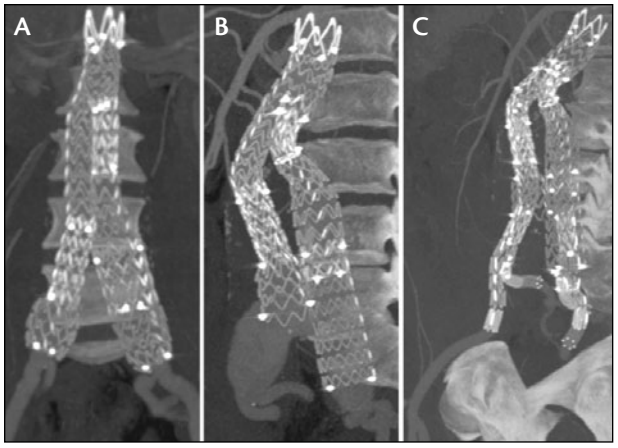

Figure 2. Postoperative CTA showing successful endovascular aneurysm exclusion using the bell-bottom technique for the right common iliac artery (A). Control CTA at 3 years revealing migration of the flexible right limb of the endograft with a type IB endoleak due to progression of aneurysmatic disease at the DSZ (B). Endovascular repair of the distal seal zone with bilateral implantation of iliac branch devices (C).

patency rates (98.4%).[8] Meanwhile, during a period of 8 years (up to November 2013), we treated a total of 176 patients with 211 iliac side branch devices. The types of repaired aneurysms consisted of 60 aorto-bi-iliac aortic aneurysms (34%), 58 aorto-mono-iliac aortic aneurysms (33%), 54 common iliac artery aneurysms (31%, 10 bilateral), and four isolated hypogastric artery aneurysms (2%). Of note, three patients were treated due to progression of the aneurysmatic disease in the iliac arteries after previous EVAR (Figure 2). The reintervention-free survival rates were 83%, 77%, and 71% at 4, 6, and 8 years, respectively. Only six patients (3.4%) showed type I or III endoleaks, and the internal iliac side branch patency amounted to 81% at 5 years. Similarly, Wong et al reported the excellent performance of an iliac side branch device in different branch configurations (helical, bifurcated-bifurcated).[9]

Our Treatment Algorithm for Aneurysmatic Degeneration of DSZ

We have already reported our institution's algorithm for AAA repair with either primary involvement of the common iliac artery or as a result of further progression of the disease.[6,8]

We recommend embolizing and overstenting the internal iliac artery only in cases where neither the bell-bottom technique nor the iliac side branch device are applicable. We always intend to overstent only one internal iliac artery. For common iliac artery aneurysms ≤ 25 mm in diameter, we advocate the bell-bottom technique as the treatment of choice.

Our criteria for the use of iliac side branch devices consist of: (1) diameter of the common iliac artery > 28 mm, (2) length of the common iliac artery > 50 mm, and (3) adequate length of the external iliac artery > 15 mm. Common iliac artery aneurysms with a diameter between 26 and 28 mm still remain a gray zone in terms of the optimal endovascular approach. Our main treatment criteria for CIA aneurysms of this size are the clinical status of the patient and the diameter at the origin of the CIA. The bell-bottom technique is preferred in patients with limited life expectancy and narrow common iliac arteries, as the risk of a distal type I endoleak in such cases is low. Otherwise, the iliac side branch device remains the treatment of choice.

Aneurysmatic degeneration of the internal iliac artery is not an exclusion criterion for an iliac side branch device. We recently published our experience with the novel technique of using the iliac side branch device in combination with the Advanta V12 (Maquet Vascular Systems, Hudson, NH) and Viabahn (Gore & Associates, Flagstaff, AZ) stent grafts supported by a self-expanding stent to adequately seal the posterior trunk and exclude the internal iliac artery aneurysm (Figure 3).[10]

Finally, we recommend the use of balloon-expandable stent grafts (Advanta V12 or BeGraft [Bentley InnoMed GmbH, Hechingen, Germany]) as bridging endografts for the internal iliac artery. However, there has been an open debate about whether a balloon- or a self-expandable covered stent should be used as a bridging endograft for the iliac side branch devices. To inform the debate, we recently performed a pooled analysis of published single-

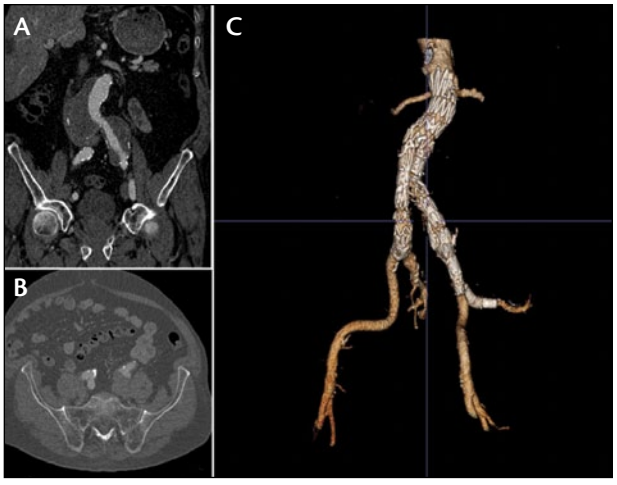

Figure 3. Use of an iliac side branch device to treat an aorto-iliac aneurysm with a concomitant aneurysm of the left internal iliac artery (IIA). Preoperative CTA showing the aortoiliac aneurysm with involvement of the proximal IIA (A, B). Control CTA confirming successful treatment of the IIA aneurysm by combining a balloon-expandable and a self-expanding stent graft and relining them with a self-expanding nitinol stent up to the posterior trunk (C).

center experiences, which revealed better patency rates for the balloon-expandable bridging endografts.[11]

NARROW DISTAL NECKS OR STENOTIC ILIAC ARTERIES

A narrow distal aortic neck is defined by the European Society of Vascular Surgery as one that is ≤ 20 mm in diameter.[12] Stenotic iliac arteries are considered to be < 7 mm in diameter.[3] One technical concern during EVAR in patients with narrow distal aortic necks or stenotic iliac arteries is the risk of limb occlusion. Specifically, in a narrow distal neck, compression of the limb by means of a competition mechanism between the two limbs could be responsible for limb occlusion.[13]

Stent graft kinking has been independently related to the occurrence of graft limb occlusion (odds ratio, 12; 95% confidence interval, 3.4–42.1; $P = .0001$), and approximately 25% to 40% of the described occlusions in recent EVAR series have been correlated to this mechanism.[13] The reported occlusion rates of currently used endografts are controversial between the studies and vary between 0% and 14%.[14-17] Recently, Cieri et al reported the occurrence of 40 occlusions (3%) among 1,450 patients with different endovascular devices for AAA repair (AneuRx [Medtronic, Inc., Santa Rosa, CA], Talent [Medtronic, Inc.], Endurant [Medtronic, Inc.], Zenith [Cook Medical], Excluder [Gore & Associates], Fortron [Cordis Corporation, Bridgewater, NJ], and Anaconda

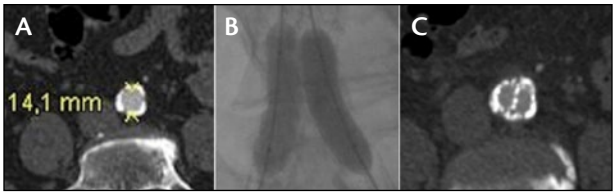

Figure 4. Stenting of both endograft limbs with the kissing-balloon technique in a napkin-ring aorta with a distal diameter of 14 mm. The preoperative diameter of the narrow distal aorta on CTA (A). Stent implantation with the kissing-balloon technique and paving-and-cracking method (B). CTA at 2 years showing the performance of both limbs in the narrowest segment of the distal aorta (C).

[Vascutek Ltd., Inchinnan, UK]) over a mean follow-up of 45 months.[17] In regard to the time of occlusion, van Zeggeren et al reported that 90% of the 20 limb occlusions were diagnosed within 1 year among 496 patients who were treated with the Endurant stent graft.[18]

Our Treatment Algorithm for Narrow Distal Aortas

Generally, the diameter of the distal aorta through which the two limbs must pass should be greater than the sum of their diameters.[13] Failing this, there may be compression of one of the limbs or kinking. At present, there are no bifurcated endografts of which the sum of the diameter of both limbs is < 20 mm.

An adjunctive measure that could overcome the challenge of a narrow distal aorta that is < 20 mm in diameter is the implantation of a balloon-expandable stent in each limb with the kissing-balloon technique (Figure 4). In case of a "napkin-ring" aorta (circumferentially calcified narrow aorta; Figure 4), the use of the so-called paving-and-cracking technique may provide some more room for the deployment of both limbs, but such a maneuver also carries the risk of aortic rupture, which could be a devastating complication, especially in case of endoleaks.[13]

We do not recommend implantation of devices in which the main body artificially elevates the aortic bifurcation with the contralateral gate. In such cases, gate cannulation can be very demanding once the ipsilateral limb is deployed through the narrow distal neck. An alternative endovascular strategy is the use of an aorto-uni-iliac stent graft and a crossover femoro-femoral bypass. We do not recommend this procedure as the first-line treatment in patients with narrow distal aortic necks due to several limitations, such as (1) any limb kinking could lead to a devastating acute aortic occlusion; (2) the procedure requires bilateral groin incisions, which increase the risk of additional morbidity by means of groin or graft infection; and (3) deployment of the contralateral iliac artery occlusion device can be very demanding.

Our Treatment Algorithm for Stenotic Iliac Vessels

The strategy of choice in cases of stenotic iliac vessels is the use of balloon angioplasty in the access vessels prior to aortoiliac endografting and balloon-expandable stenting within the limbs after endografting. According to our experience, use of the paving-and-cracking technique may be required prior to endograft deployment to facilitate endograft advancement. In this scenario, we suggest using the Advanta V12 covered stent in the common iliac artery and the Viabahn stent graft in the external iliac artery. To overcome access issues, ultra-low-profile stent grafts have recently been introduced, but extensive and long-term experience with such devices is still lacking. The use of a balloon-expandable introducer is now available as a recollapsible sheath (SoloPath, Terumo Interventional Systems, Somerset, NJ), and the use of a surgical conduit, mostly an iliofemoral bypass graft, is also recommended.

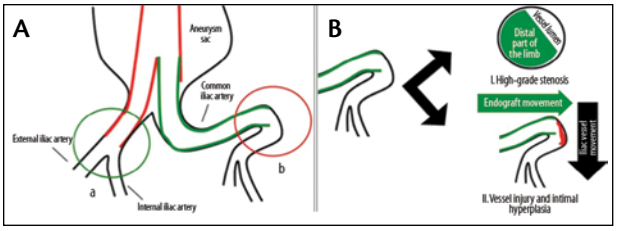

Figure 5. Our assumption about the possible mechanism of limb occlusion post-EVAR due to suboptimal sitting of the distal end of the limb in a tortuous common iliac artery. Optimal position of the right limb (green circle) in a straight right common iliac artery and suboptimal sitting of the left limb (red circle) in the tortuous left common iliac artery (A). Possible mechanisms leading to limb occlusion (I = high-grade stenosis due to reduction of the vessel lumen; II = vessel injury and intimal hyperplasia [red area] associated with the different directions of endograft and artery movement during the cardiac cycle) (B).

TORTUOUS ILIAC VESSELS

Although the flexibility of the current endografts has been much improved, tortuous iliac vessels still remain an anatomic challenge, not only during endograft insertion but also when they are used in the DSZ. To the best of our knowledge, the literature lacks comparable data about the impact of tortuosity on EVAR outcomes, due to the subjective grading of iliac artery tortuosity.[11] According to EVAR reporting standards, iliac artery tortuosity can be measured with an iliac artery tortuosity index.[19] The index is defined as the ratio between the distance along the central lumen line between the aortic bifurcation and the common femoral artery and the straight line between the same landmarks.[20] Despite its accuracy, the use of this index remains complex and time-consuming, and measurement should be performed in different projections.[11]

In view of the DSZ, severe iliac artery tortuosity may influence limb patency in the long run. Possible mechanisms of limb occlusion in cases of tortuous iliac arteries are limb kinking and suboptimal apposition of the distal end of the limb to the iliac vessel wall. The association between limb kinking and occlusion was previously described. The second mechanism remains a hypothesis of the authors and requires further investigation to be proven. According to our assumption, suboptimal wall apposition of the distal end of the endograft's limb may cause either a high-grade local stenosis or lead to subintimal hyperplasia due to continuous intimal injuries of the iliac vessel (Figure 5).

Our Treatment Algorithm for Tortuous Iliac Arteries

An essential step during EVAR implantation in a tortuous iliac system is the performance of control angiography after removing the extra-stiff wire, with either a soft wire or an angiographic catheter (eg, vertebral or pigtail catheter) kept in place. In our experience, this maneuver will unmask possible graft kinking or suboptimal sitting of the distal end of the endografts, which could not otherwise be identified when the extra-stiff wire is in place.

For graft kinking, our recommended measure is the implantation of an additional bare-metal stent, which will optimize the anatomy of the limb. In cases of suboptimal wall apposition of the distal end of the endograft's limb, we recommend the creation of a composite distal end by supporting the endograft with a self-expanding stent up to a straight part of the common or external iliac artery. Of note, in some cases, overstenting of the internal iliac artery is unavoidable. The substitution of a nitinol stent appears to provide a more gradual transition into the tortuous iliac system.

SUMMARY

A hostile DSZ could include one or more of the following anatomic scenarios: aneurysmatic iliac vessels, a narrow distal aortic neck, and a stenotic or tortuous iliac system. The pathogenic mechanism of DSZ-associated poor outcomes and the influence of a hostile DSZ on the reintervention rates after EVAR are not well investigated. However, limb migration and occlusion are known leading causes for reinterventions after EVAR. There are several endovascular solutions to overcome those challenges, but comparative data are missing to draw robust conclusions. In cases of aneurysmatic DSZ, the use of an iliac side branch device is a well-established method with excellent long-term results and provides an effective solution for aneurysmatic degeneration of the DSZ due

to further progression of the disease. In any case, DSZ has an essential impact on a reintervention-free survival after EVAR and is a factor that should not be overlooked. ■

**The Zenith Branch Iliac Graft is an investigational device in the United States. Limited by United States law to investigational use. It is CE Mark approved with indications for use in the endovascular treatment of patients with an aortoiliac or iliac aneurysm, an insufficient distal sealing site within the common iliac artery, and having morphology suitable for endovascular repair.*

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1. Dangas G, O' Connor D, Firwana B, et al. Open versus endovascular stent graft repair of abdominal aortic aneurysms: a meta-analysis of randomized trials. JACC Cardiovasc Interv. 2012;10:1071-1080.
2. Donas KP, Torsello G, Bisdas T. New EVAR devices: pros and cons. J Cardiovasc Surg (Tor). 2012;53:559-569.
3. Schanzer A, Greenberg RK, Hevelone N, et al. Predictors of abdominal aortic aneurysm sac enlargement after endovascular repair. Circulation. 2011;123:2848-2855.
4. Norman PE, Powell JT. Site specificity of aneurysmal disease. Circulation. 2010;121:560-568.
5. Rayt HS, Bown MJ, Lambert KV, et al. Buttock claudication and erectile dysfunction after internal iliac artery embolization in patients prior to endovascular aortic aneurysm repair. Cardiovasc Intervent Radiol. 2008;31:728-734.
6. Torsello G, Schönefeld E, Osada N, et al. Endovascular treatment of common iliac artery aneurysms using the bell-bottom technique: long-term results. J Endovasc Ther. 2010;17:504-509.
7. Lobato AC, Camacho-Lobato L. The sandwich technique to treat complex aortoiliac or isolated iliac aneurysms: results of midterm follow-up. J Vasc Surg. 2013;57:26S-34S.
8. Donas KP, Torsello G, Pitoulias GA, et al. Surgical versus endovascular repair by iliac branch device of aneurysms involving the iliac bifurcation. J Vasc Surg. 2011;53:1223-1229.
9. Wong S, Greenberg RK, Brown CR, et al. Endovascular repair of aortoiliac aneurysmal disease with the helical iliac bifurcation device and the bifurcated-bifurcated iliac bifurcation device. J Vasc Surg. 2013;58:861-869.
10. Austermann M, Bisdas T, Torsello G, et al. Outcomes of a novel technique of endovascular repair of aneurysmal internal iliac arteries using iliac branch devices. J Vasc Surg. 2013;58:1186-1191.
11. Donas KP, Bisdas T, Torsello G, Austermann M. Technical considerations and performance of bridging stent-grafts for iliac side branched devices based on a pooled analysis of single-center experience. J Endovasc Ther. 2012;19:667-671.
12. Harris PL, Vallabhaneni SR, Desgranges P, et al. Incidence and risk factors of late rupture, conversion, and death after endovascular repair of infrarenal aortic aneurysms: the EUROSTAR experience. European collaborators on stent/graft techniques for aortic aneurysm repair. J Vasc Surg. 2000;32:739-749.
13. Nelson PR, Kansal N. The narrow distal aorta. Endovasc Today. 2012;10:79-83.
14. Cochennec F, Becquemin JP, Desgranges P, et al. Limb graft occlusion following EVAR: clinical pattern, outcomes and predictive factors of occurrence. Eur J Vasc Endovasc Surg. 2007;34:59-65.
15. Bos WT, Tielliu IF, van den Dungen JJ et al. Results of endovascular abdominal aortic aneurysm repair with selective use of the Gore Excluder. J Cardiovasc Surg (Torino). 2009;50:159-164.
16. Van Marrewijk CJ, Leurs LJ, Vallabhaneni SR, et al. Risk-adjusted outcome analysis of endovascular abdominal aortic aneurysm repair in a large population: how do stent-grafts compare? J Endovasc Ther. 2005;12:417-429.
17. Cieri E, De Rango P, Isernia G, et al. Effect of stent-graft model on aneurysm shrinkage in 1,450 endovascular aortic repairs. Eur J Vasc Endovasc Surg. 2013;46:192-200.
18. Van Zeggeren L, Bastos Goncalves F, van Herwaarden JA, et al. Incidence and treatment results of Endurant endografts occlusion. J Vasc Surg. 2013;57:1246-1254.
19. Kristmundsson T, Sonesson B, Resch T. A novel method to estimate iliac tortuosity in evaluating EVAR access. J Endovasc Ther. 2012;19:157-164.
20. Chaikof EL, Blankensteijn JD, Harris PL, et al. Reporting standards for endovascular aortic aneurysm repair. J Vasc Surg. 2002;35:1048-1060.

Improving Your Practice by Operating Within the IFU

Safely achieving desired results with EVAR.

BY TIMOTHY RESCH, MD, PHD

The development of endovascular aneurysm repair (EVAR) has fundamentally changed the way aortic aneurysm surgery is perceived and performed. Early on, even before any mid- or long-term data were available, the minimally invasive nature of EVAR was seen as a dramatic development allowing aneurysm repair to be offered to the elderly and those who are physically unfit for surgery.[1]

The early endografts were often somewhat crude in design, simply using interrupted metal stents sewn onto graft fabric. Delivery systems were often bulky and rigid, causing difficulties when passing the EVAR devices to their target positions. However, quite rapidly, major design improvements were made to increase and expand the applicability of this exciting new technology.[2] Dedicated stent grafts with improved fixation systems, radiopaque markers, and flexibility were combined with delivery systems with lower profiles, better trackability, and enhanced deployment features. This resulted in an increasing number of second- and third-generation devices, expanding the scope of EVAR and, perhaps also, the perception of what EVAR could offer.

In parallel to the evolution of infrarenal stent grafts, more complex stent graft designs were developed to allow treatment of aneurysm pathology that precluded infrarenal EVAR. In the late 1990s,[3] fenestrated stent grafts for the treatment of juxta- and suprarenal aneurysms were introduced, as well as branched stent grafts to treat even more complex thoracoabdominal aneurysms.[4] Branched iliac grafts for preservation of the internal iliac circulation in the setting of common iliac aneurysm were also introduced.[5]

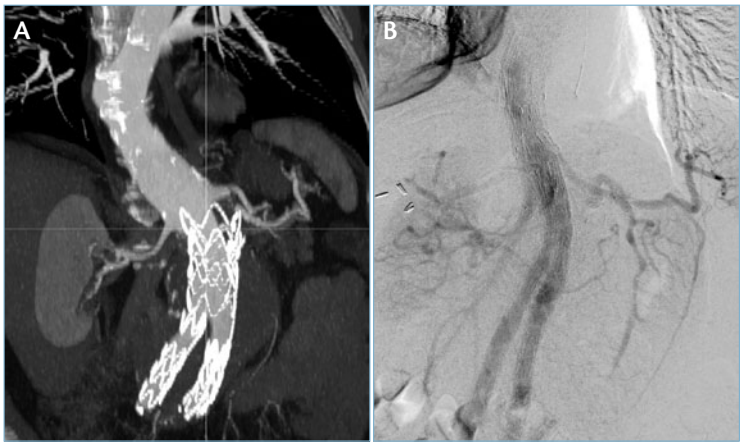

Figure 1. Failed infrarenal EVAR due to neck dilatation (A). Also note the aneurysm in the distal descending aorta. The aneurysm was repaired using a t-Branch device (Cook Medical, Bloomington, IN) (B).

SEALING IN A HEALTHY AORTA

After technical development had overcome the initial difficulties of device delivery and early technical failures, focus shifted to the durability of stent grafts. Early on, several factors were identified that directly affected the mid- and long-term outcomes of infrarenal endovascular repair. The presence of "suboptimal" sealing zones was clearly the main determinant of EVAR failures (Figure 1).[6-8] In the proximal infrarenal neck, the presence of nonparallel aortic walls, thrombus, and severe angulation predicted poor outcomes, and the same was true in the distal sealing zone in the common iliac arteries. Short and ectatic common iliac arteries during initial implantation predicted late failures with resulting endoleaks and aneurysm nonexclusion.[9]

The same phenomenon has also been observed when using more complex stent grafts. Even though the stent graft was now placed more proximally in the aorta, the presence of adverse features of the sealing zone, indicating a nonhealthy aorta, clearly predicted device failure.

A situation that affects EVAR, and the entire field of endovascular treatment, is that as causes of failures are identified, technical innovation moves forward at a rapid pace with renewed promise to overcome these failures. However, as the applicability of EVAR expands with improved devices, the tendency for off-label use seems to expand even more. The basic premise of suc-

cessful EVAR—safe sealing and anchoring in a healthy aortic segment—often continues to be ignored. Devices are designed and manufactured to deliver durable aortic repair for a given set of anatomical constraints. Abundant literature exists to show that these basic rules are often violated.

The recent report by Schanzer et al,[10] reviewing more than 10,000 patients undergoing EVAR and included in the M2S database (M2S, West Lebanon, NH), again highlighted these circumstances. Apart from the fact that almost 50% of patients treated had aneurysms < 50 mm, the authors clearly demonstrated that adverse anatomical features of the infrarenal sealing zone correlated to late aneurysm growth, thus indicating treatment failure.

Reports on long-term outcomes of fenestrated aortic repair show that moving the sealing zone more proximal in the aorta provides a durable outcome without significantly affecting the perioperative results compared to infrarenal repair.[11,12] In fact, increasing experience suggests that even if the fenestrated repair is made technically more complex (ie, adding more fenestrations), this can be achieved with less exposure to both contrast and radiation.[11]

The same holds true for the distal sealing zone. Landing a stent graft in an ectatic landing vessel greatly increases the risk of late problems. Simply embolizing the internal iliac artery and creating a landing zone in the external iliac artery causes claudication and other complications in a large number of patients. Using iliac branched grafts provides a standardized procedure to provide both seal and flow preservation without interfering with the abdominal aortic aneurysm repair. Data suggest that this is feasible in many patients and that long-term outcomes are very good, with patency rates of approximately 90% at 5 years.[13]

BUILDING FOR THE FUTURE

Designing the device of the future requires examining the failures of the past. Aortic disease is a chronic process and demands that vascular surgeons not only treat the segment that is failing right now but also analyze where it might fail next. This knowledge must then be incorporated into the repair so that doors are open for future therapy.

By utilizing knowledge of aortic disease, as well as applying modern EVAR with available devices, we can provide both a durable, stable repair for our patients and a bridge to future surgery. By moving within the instructions for use of current endovascular devices, as opposed to going off label once too often, these goals can be achieved. ■

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1. Hynes N, Sultan S. A prospective clinical, economic, and quality-of-life analysis comparing endovascular aneurysm repair (EVAR), open repair, and best medical treatment in high-risk patients with abdominal aortic aneurysms suitable for EVAR: the Irish patient trial. J Endovasc Ther. 2007;14:763-776.
2. Greenberg RK, Chuter TA, Sternbergh WC 3rd, Fearnot NE; Zenith Investigators. Zenith AAA endovascular graft: intermediate-term results of the US multicenter trial. J Vasc Surg. 2004;39:1209-1218.
3. Anderson JL, Adam DJ, Berce M, Hartley DE. Repair of thoracoabdominal aortic aneurysms with fenestrated and branched endovascular stent grafts. J Vasc Surg. 2005;42:600-607.
4. Chuter TA, Gordon RL, Reilly LM, et al. An endovascular system for thoracoabdominal aortic aneurysm repair. J Endovasc Ther. 2001;8:25-33.
5. Oderich GS, Greenberg RK. Endovascular iliac branch devices for iliac aneurysms. Perspect Vasc Surg Endovasc Ther. 2011;23:166-172.
6. Fulton JJ, Farber MA, Sanchez LA, et al. Effect of challenging neck anatomy on mid-term migration rates in AneuRx endografts. J Vasc Surg. 2006;44:932-937; discussion 937.
7. Abbruzzese TA, Kwolek CJ, Brewster DC, et al. Outcomes following endovascular abdominal aortic aneurysm repair (EVAR): an anatomic and device-specific analysis. J Vasc Surg. 2008;48:19-28.
8. Falkensammer J, Hakaim AG, Andrew Oldenburg W, et al. Natural history of the iliac arteries after endovascular abdominal aortic aneurysm repair and suitability of ectatic iliac arteries as a distal sealing zone. J Endovasc Ther. 2007;14:619-624.
9. McDonnell CO, Semmens JB, Allen YB, et al. Large iliac arteries: a high-risk group for endovascular aortic aneurysm repair. J Endovasc Ther. 2007;14:625-629.
10. Schanzer A, Greenberg RK, Hevelone N, et al. Predictors of abdominal aortic aneurysm sac enlargement after endovascular repair. Circulation. 2011;123:2848-2855.
11. Kristmundsson T, Sonesson B, Dias N, et al. Outcomes of fenestrated endovascular repair of juxtarenal aortic aneurysm. J Vasc Surg. 2014;59:115-120.
12. Mastracci TM, Greenberg RK, Eagleton MJ, Hernandez AV. Durability of branches in branched and fenestrated endografts. J Vasc Surg. 2013;57:926-933; discussion 933.
13. Parlani G, Verzini F, De Rango P, et al. Long-term results of iliac aneurysm repair with iliac branched endograft: a 5-year experience on 100 consecutive cases. Eur J Vasc Endovasc Surg. 2012;43:287-292.

Pathologies of Progressive Thoracic Aortic Disease

With the natural tendency of the aorta to dilate, there are inherent practical considerations to account for when planning a TEVAR procedure.

BY THOMAS L. FORBES, MD

In many series describing thoracic aortic disease, it is apparent that there are two main differences regarding patient presentation in this anatomic area compared to the abdominal aorta. First, patients are proportionately more inclined to present urgently with acute onset of back pain, as opposed to patients with abdominal aortic pathologies who are more likely to have asymptomatic lesions. Second, thoracic aortic pathologies are more disparate than those in the abdominal aorta and include thoracic aortic aneurysm (TAA), aortic dissection (AD), intramural hematoma (IMH), penetrating aortic ulcer (PAU), and blunt traumatic thoracic aortic injury (BTAI).

The term *acute aortic syndrome*[1,2] has been attributed to the urgent presentation of thoracic aortic pathologies including AD, IMH, and PAU. Once other etiologies such as acute myocardial infarction are excluded, investigations should lead to contrast-enhanced and nonenhanced CT scan of the chest, abdomen, and pelvis. Complete visualization is important to determine the extent of the acute thoracic aortic process and a potential access for endovascular therapies.[3] Once the specific pathologic variant is identified, the appropriate course of treatment can be determined, recognizing the shared pathophysiology of these clinical entities and the understanding that PAU and IMH represent focal manifestations of the classically more extensive AD.[4]

PATHOPHYSIOLOGY

Acquired and genetic conditions can increase susceptibility to these acute thoracic aortic conditions. Of course, the most common pre-existing condition is hypertension, which leads to intimal thickening, fibrosis, and calcification, in turn leading to degradation of the extracellular matrix and eventual disruption of the intima.[1] Genetic conditions can also cause intimal disruption and AD. These conditions include Marfan syndrome, Ehlers-Danlos syndrome, and bicuspid aortic valve, among others.[1]

Although these clinical entities (PAU, IMH, AD) can be viewed as variations of the same disease process, there are clinical features distinct to each. Patients with PAU and IMH tend to be older (the mean age was 74 years in one series[4]) than those with dissections, and most patients are hypertensive. Penetrating ulcers are associated with atherosclerotic disease of the thoracic aorta, whereas AD often occurs in aortas with lesser degrees of calcification. In addition, PAU and IMH tend to affect thoracic aortas of larger diameters than AD.[4] These pathologic distinctions likely reflect differences in the depth of penetration of the aortic wall between PAU, IMH, and AD.

NATURAL HISTORY

The thoracic aorta is a hostile environment for stent grafts, with violent hemodynamic stresses and forces. These forces can lead to eventual failure of thoracic endovascular aneurysm repair (TEVAR) and result in reinterventions. In addition, the thoracic aorta is a dynamic organ that is prone to progressive dilatation, even in the nondiseased state. In a review of more than 1,000 normal thoracic aortas, Hartley et al[5] observed a steady dilatation of approximately 1 cm between teenagers and those in their 80s. This finding was irrespective of sex, race, or the presence of hypertension, pulmonary disease, or diabetes.

With a progressively dilating thoracic aorta subjected to strong hemodynamic forces, and with variable pathologies, it is apparent that thoracic aortic disease is progressive, and when choosing any therapy, either open surgery or TEVAR, one needs to take this into account. This is true for the more diffuse pathologies such as dissections and thoracoabdominal aneurysms, but also for more focal lesions including isolated TAAs, IMHs, penetrating thoracic aortic ulcers, and the most focal of them all, BTAIs.

TAAs

Even the most localized TAAs that are easily treated (at least initially) with TEVAR can require major reinter-

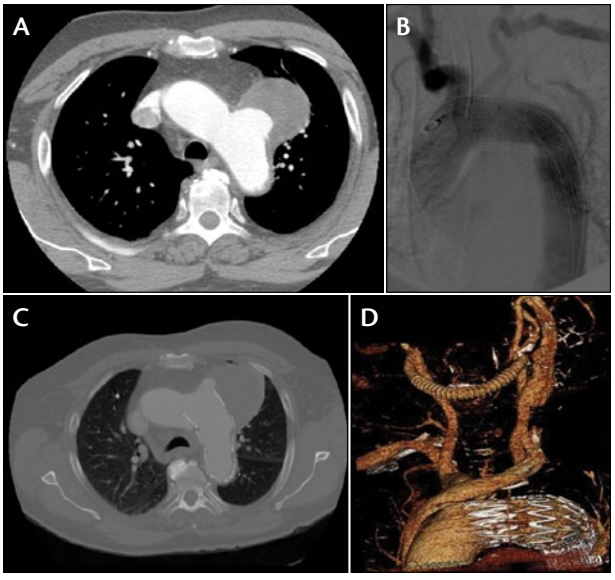

Figure 1. Focal aneurysm of the proximal descending thoracic aorta (A). The initial zone 2 TEVAR was successful, with left subclavian-to-left common carotid artery transposition (B). Two years later, the patient developed a proximal type IA endoleak (C). A carotid-carotid bypass and zone 1 TEVAR were performed (D).

ventions due to the inherent dilatory characteristics of the aorta[5] and the progressive nature of thoracic aortic disease. Figure 1 illustrates a case of a proximal descending TAA that was successfully treated with a zone 2 TEVAR and left subclavian artery–to–left common carotid artery transposition. Two years later, the patient developed a type IA endoleak due to progressive dilatation of the aortic arch and the proximal seal zone. This prompted extension of the TEVAR with a zone 1 proximal landing site and a carotid-carotid bypass.

Any surgeon with even moderate TEVAR experience will recognize this scenario as not being terribly unusual. The published literature is full of reports describing reinterventions following initially successful TEVAR. Our group has published midterm outcomes following TEVAR for all thoracic aortic pathologies.[6] The majority (60%) of these cases were for TAAs. At a median length follow-up of 21 months, 6% of patients developed an endoleak, and 4% of patients, overall, required a TEVAR reintervention.

In a more recent and larger series, the Northwestern group reported their experience with reinterventions following elective TEVAR for TAAs in 83 patients.[7] Overall, 10% of patients required aortic reinterventions at a mean of 32 months following initial TEVAR, indicating the dynamic and progressive nature of thoracic aortic disease. More than half of the reinterventions were for

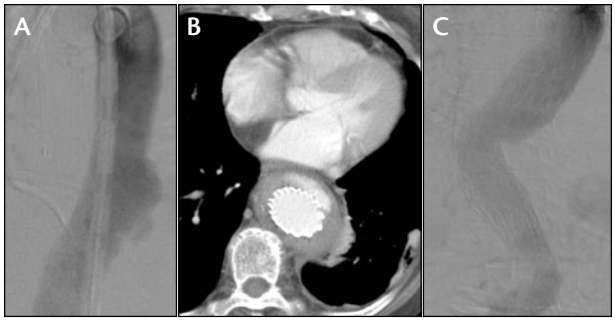

Figure 2. TEVAR for a penetrating aortic ulcer (A). A distal type IB endoleak following initially successful TEVAR for PAU (B). A distal extension was placed for a distal type IB endoleak (C).

type I endoleaks because of progressive aortic dilatation. There was also a trend, although not statistically significant ($P = .052$), for more extensive aneurysms (fusiform) to require secondary interventions compared to those that were more focal (saccular). This information supports the philosophy that all aneurysms of the thoracic aorta represent a panaortic pathology and that no matter how localized the initial endovascular repair, a proportion of these will progress to the extent of requiring reintervention. These issues require attention when planning the initial repair.

IMHs AND PAUs

These lesions are generally more localized than TAAs, but there are numerous examples of the progressive nature of these lesions resulting in further degeneration of the thoracic aorta after initially successful TEVAR. Figure 2 illustrates a case of a PAU with surrounding hematoma that was initially treated with TEVAR with good results. The patient subsequently developed a distal type IB endoleak because of progressive dilatation of the distal descending thoracic aorta. This necessitated a distal extension.

Although AD is widely seen as progressive, less has been known of the progressive nature of more localized pathologies such as IMH and PAU. However, this has recently changed with information provided by the multicenter International Registry of Acute Aortic Dissection (IRAD) that included patients with IMH and PAU. Of the 2,830 patients included in IRAD, 178 had IMH.[8] In-hospital mortality did not differ between IMH of the descending thoracic aorta compared to type B AD (4.4% vs 11.1%; $P = .06$), and mortality at 1 year did not differ either. Very importantly, however, IMH of the descending thoracic aorta was shown to be progressive in nature and resulted in aortic dilatation in more than one-third (39%) of patients.[9] This needs to be considered when planning treatment and the extent of any endovascular

repair, in order to prevent such situations as illustrated in the previous case (Figure 2).

BTAIs

BTAIs are the most localized lesion in the thoracic aorta and most commonly affect patients with normal, nondiseased aortas. However, there is mounting evidence that even these very localized injuries represent a progressive lesion that results in dilatation of the thoracic aorta. In a report from our center involving patients with BTAIs who were treated with TEVAR, we reviewed postoperative CT scans to observe any dilatation at different levels of the thoracic aorta.[10] Although all levels of the thoracic aorta showed some progressive dilatation, as would be expected from natural history data,[5] the segment just distal to the left subclavian artery expanded at a slightly greater rate (0.83 mm per year; P =.025).[10]

Whether this accelerated expansion of the thoracic aorta is a temporary response after BTAI or continues during the longer-term has yet to be determined. How much is due to the BTAI or simply due to the expansile forces of the endograft is also unknown. Recent histologic studies in pigs have illustrated the deleterious effects of endograft oversizing with reduced numbers of muscle and elastic fibers.[11] Longer-term follow-up of these generally young BTAI patients will be essential to determine whether the natural tendency of the normal thoracic aorta to dilate is synergistic with that of the injured aorta and the aorta's histologic response to the presence of an endograft.

CONCLUSION

Although the thoracic aorta is subjected to different pathologies of varying anatomic extents and etiologies, they all have the potential to augment the natural tendency of the aorta to dilate. The natural tendency of the aorta, treated or untreated, is to dilate. This has important ramifications when planning an initial TEVAR procedure in which the length of aortic coverage needs to be carefully determined to minimize the risk of secondary aortic interventions, but not at the expense of neurologic or spinal cord complications. The dynamic nature of the thoracic aorta and the progressive nature of thoracic aortic diseases will continue to challenge vascular specialists involved their treatment. ■

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1. Ince H, Nienaber CA. [Management of acute aortic syndromes]. Rev Esp Cardiol. 2007;60:526-541.
2. Nordon IM, Hinchliffe RJ, Loftus IM, et al. Management of acute aortic syndrome and chronic aortic dissection. Cardiovasc Intervent Radiol. 2011;34:890-902.
3. Fleischmann D, Mitchell RS, Miller DC. Acute aortic syndromes: new insights from electrocardiographically gated computed tomography. Semin Thorac Cardiovasc Surg. 2008;20:340-347.
4. Coady MA, Rizzo JA, Elefteriades JA. Pathologic variants of thoracic aortic dissections. Penetrating atherosclerotic ulcers and intramural hematomas. Cardiol Clin. 1999;17:637-657.
5. Hartley MC, Langan EM 3rd, Cull DL, et al. Evaluation of the diameter of the proximal descending thoracic aorta with age: implications for thoracic aortic stent grafting. Ann Vasc Surg. 2009;23:639-644.
6. Chu MW, Forbes TL, Kirk Lawlor D, et al. Endovascular repair of thoracic aortic disease: early and midterm experience. Vasc Endovasc Surg. 2007;41:186-191.
7. Lee CJ, Rodriguez HE, Kibbe MR, et al. Secondary interventions after elective thoracic endovascular aortic repair for degenerative aneurysms. J Vasc Surg. 2013;57:1269-1274.
8. Harris KM, Braverman AC, Eagle KA, et al. Acute aortic intramural hematoma: an analysis from the International Registry of Acute Aortic Dissection. Circulation. 2012;126(11 suppl 1):S91-96.
9. Tolenaar JL, Harris KM, Upchurch GR Jr, et al. The differences and similarities between intramural hematoma of the descending aorta and acute type B dissection. J Vasc Surg. 2013;58:1498-1504.
10. Forbes TL, Harris JR, Lawlor DK, Derose G. Aortic dilatation after endovascular repair of blunt traumatic thoracic aortic injuries. J Vasc Surg. 2010;52:45-48.
11. Sincos IR, da Silva ES, Belczak SQ, et al. Histologic analysis of stent graft oversizing in the thoracic aorta. J Vasc Surg. 2013;58:1644-1651.e4.

Chasing the Progressive Aortic Dissection

A case report illustrating the extreme complexity of aortopathy and our current knowledge in addressing this potentially devastating presentation.

BY BENJAMIN W. STARNES, MD

On July 5, 2007, Derek, a 42-year-old engineer, was traveling in Iceland with his wife when he experienced the acute onset of tearing back pain, like someone was stabbing him with a knife. It began in his upper middle back and traveled down to his lower back and then into his abdomen. He was taken by the Icelandic Coast Guard by helicopter to Landspitali Reykjavik University Hospital and was diagnosed with a Stanford type B aortic dissection, with an entry tear just beyond the left subclavian artery. He was managed medically with anti-impulse therapy for 18 days and returned to his home in Seattle on July 23, 2007.

Derek has a history of Marfan syndrome and had undergone aortic root replacement for an ascending aortic aneurysm in March 1999 at age 34. Despite this history, it was recommended that he undergo thoracic endovascular aneurysm repair (TEVAR) of his dissection 4 months later. He underwent this procedure with placement of an endoprosthesis, but 10 days later, his stent graft collapsed, and Derek became paraplegic. He underwent urgent placement of a giant Palmaz stent (Cordis Corporation, Bridgewater, NJ) but, unfortunately, never regained use of his lower extremities.

On May 22, 2008, Derek travelled to Cleveland to meet with Dr. Roy Greenberg at the Cleveland Clinic regarding enlargement and progression of his descending thoracic aorta, which had grown aneurysmally to > 6 cm, with persistent false lumen perfusion. Dr. Greenberg recommended endovascular repair instead of open repair because of the underlying severe aortic valvular insufficiency and the patient's inability to tolerate an aortic cross-clamp. I met Derek in July of that year and, after consultation with Dr. Greenberg, extended the repair from his existing stent graft to his diaphragm with a Zenith TX2 device* (Cook Medical, Bloomington, IN).[1,2] Amazingly, Derek's aorta was completely remodeled with false lumen thrombosis and an eventual normal appearance of the aorta 6 years later (Figure 1).

This case illustrates the extreme complexity of aortic dissection. Whether it be dissection in the setting of a connective tissue disorder, as in this case, or a result of illicit drug use, there is still much that we do not know about this aortopathy. With much of the current enthu-

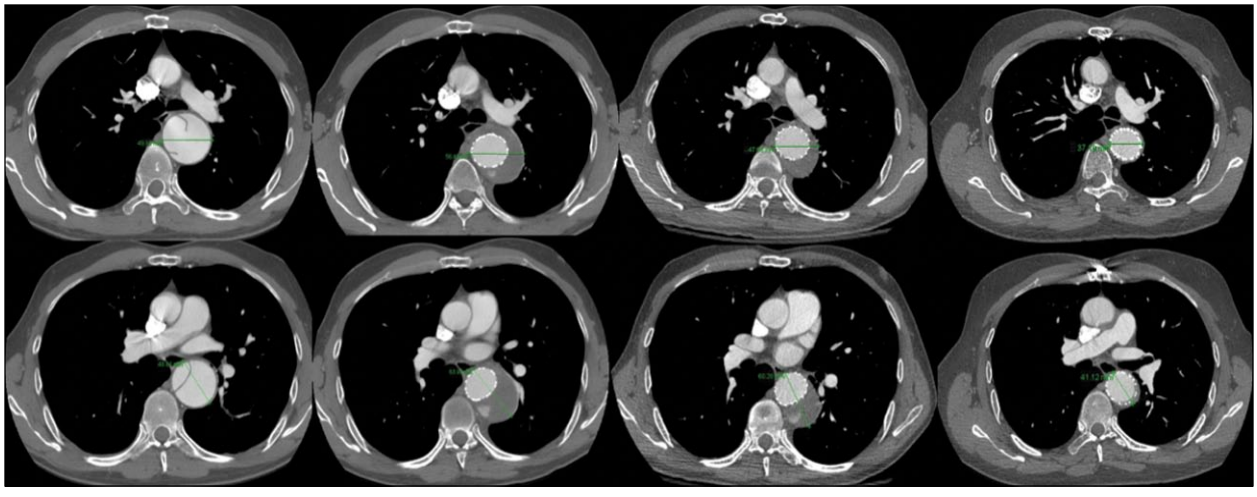

Figure 1. Six-year follow-up with representative axial CT images taken at the level of the carina (top panels) and the top of T-7 (lower panels) on October, 11, 2007; February 27, 2008; July 26, 2009; and December 4, 2013; respectively.

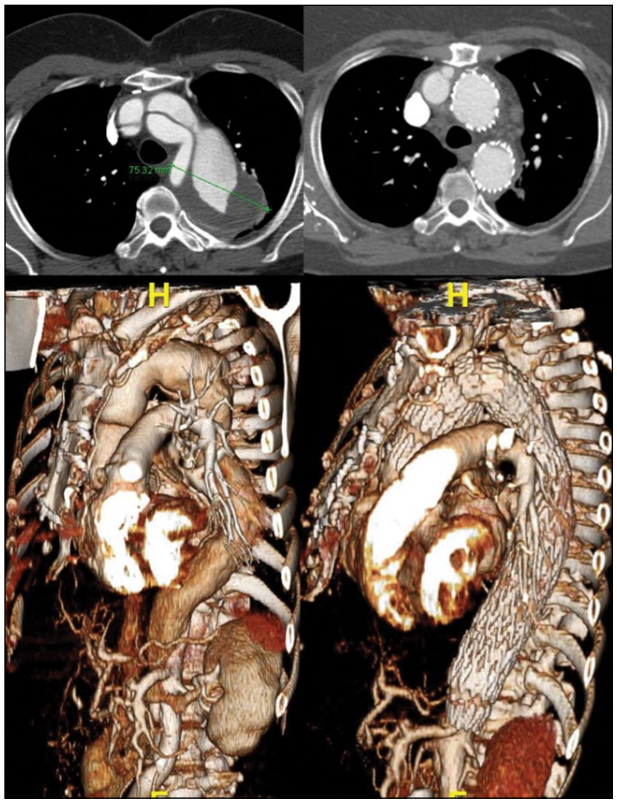

Figure 2. Hybrid repair of a chronic aortic dissection utilizing aortic root replacement, arch debranching, and successful TEVAR, with dramatic aortic remodeling during a 4-year period.

siasm about treating all patients presenting with aortic dissection using endovascular methods, this case example should cause some consternation. What have we learned about TEVAR and aortic dissection in the endovascular era? Whom should we treat? Whom should we not treat? Do the successes outnumber the failures? Is this simply a chronic disease with an eventual death sentence? Although many of these questions remain ill-defined and unanswered, we must understand that aortic dissection is progressive, and to successfully manage these patients, we must have the tenacity to continue to chase the disease until the aorta is stable.

WHAT WE KNOW NOW

The natural history of aortic dissection has been better defined as of late. The International Registry of Acute Aortic Dissection has provided data on outcomes relating to acute dissection of both the ascending and descending aorta. Acute dissection of the ascending aorta has a poor prognosis when treated medically, and roughly 60% of patients will die in the short-term. The outcome of acute dissection of the descending aorta when managed medically is much better, with an in-hospital mortality of 13%,[1] but one-third of these patients will eventually require surgical or endovascular intervention in their lifetime.

There are multiple case reports of the successful endovascular management of either acute or chronic aortic dissection with aortic stent grafts (apropos our first case example). Hybrid approaches have also been shown to be highly successful in certain situations involving the aortic arch either in combination with or without aortic root replacement (Figure 2).

BUT WHAT HAPPENS WHEN ENDOVASCULAR THERAPY DOESN'T WORK?

Do we have the devices and tools we need to adequately treat this disease in its chronic state, or should we focus our efforts on early management of the disease for all patients? Why do some patients do well with medical management and some patients don't? Figure 3 shows a patient with a Stanford type B chronic aortic dissection with rapid aneurysm enlargement who

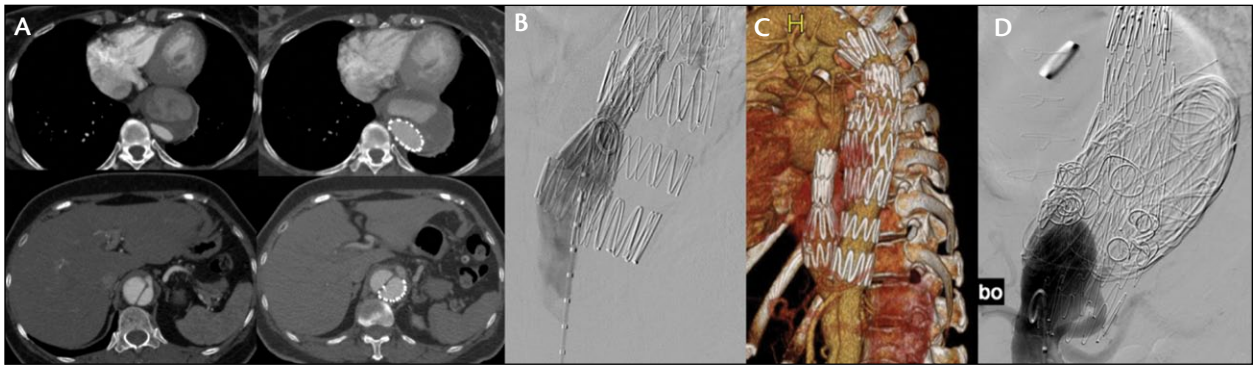

Figure 3. Imaging depicting the expanding aneurysm in association with chronic aortic dissection and subsequent TEVAR with persistent flow in the false aneurysm (A, B). Adjunctive measures were undertaken, resulting in successful false lumen embolization (C). A final coil embolization procedure successfully obliterated the false lumen and arrested expansion of the aneurysm 3 years later (D).

underwent TEVAR in July 2010. On follow-up CT angiography, the patient was noted to have a persistently patent false lumen and sac enlargement. This was confounded by the fact that she was chronically anticoagulated for a mechanical heart valve. She underwent multiple reinterventions, with subsequent complete false lumen thrombosis and complete obliteration of the aneurysm sac (Figure 3).

A NEW PERSPECTIVE

For nearly 50 years, we have classified aortic dissection based upon the anatomic location of the dissection in the aorta, with the implications affecting treatment choices, either surgical or medical. The widespread adoption of endovascular therapy to manage this disease has challenged the established classification systems to adequately account for the features that remain critical to making therapeutic decisions. In an attempt to modernize the classification of aortic dissection with relevance to endovascular therapies, the Working Group on Aortic Disease of the DEFINE Project recently proposed DISSECT, a new mnemonic-based approach to the categorization of aortic dissection (see the DISSECT Classification of Aortic Dissection sidebar).[2]

In the coming years, it will be crucial for us to appropriately classify patients so that we may compare apples to apples and oranges to oranges. We all know that a patient with a D:Ch, I:D, S:70 mm, SE:AI, C:C, T:P (previously known as simply a complicated Stanford type B or Debakey type 3) is entirely different from a D:Sa, I:D, S:30 mm, SE:D, C:UC, T:CT. In other words, not all type B dissections are the same, and our therapies should be directed at the natural history of the disease. I believe this new classification system is a step in the right direction toward classifying and managing patients presenting with a wide variety of anatomical and clinical manifestations.

LIFE GOES ON

I had coffee with Derek the other day at a local Starbucks. He is wheelchair bound, his legs taken from him in the prime of his life. Not a single day goes by that Derek doesn't think about his own aortic calamity. We owe our patients much better treatment methods, devices, and technology for aortic dissection in this new century.

We owe Derek. ■

**The Zenith TX2 is FDA approved with indications for use in the endovascular treatment of patients with aneurysms or ulcers of the descending thoracic aorta having vascular morphology suitable for endovascular repair.*

It is CE Mark approved with indications for use in the treatment of patients with atherosclerotic aneurysms, symptomatic acute or chronic dissections, contained ruptures, growing aneurysms and/or resulting in distal ischemia, in the descending thoracic aorta having vascular morphology suitable for endovascular repair.

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1. Suzuki T, Mehta RH, Ince H, et al. Clinical profiles and outcomes of acute type B aortic dissection in the current era: lessons from the International Registry of Aortic Dissection (IRAD). Circulation. 2003;108(suppl 1):II312-317.
2. Dake MD, Thompson M, van Sambeek M, et al. DISSECT: a mnemonic based approach to the categorization of aortic dissection. Eur J Vasc Endovasc Surg. 2013;46:175–190.

"DISSECT" CLASSIFICATION OF AORTIC DISSECTION

Duration (D) defined as time from onset of symptoms:
- Ac = acute: < 2 weeks from initial onset of symptoms
- Sa = subacute: 2 weeks to 3 months after symptom onset
- Ch = chronic: > 3 months from initial onset of symptoms

Intimal (I) tear (primary) location within the aorta:
- A = ascending aorta
- Ar = aortic arch
- D = descending aorta
- Ab = abdominal aorta
- Un = unknown

Size (S) of the aorta based on maximum transaortic diameter by centerline analysis at any level within the dissected segment of aorta

Segmental extent (SE) of aortic involvement from proximal to distal boundary:
- A = ascending aorta exclusively
- Ar = aortic arch exclusively
- D = descending exclusively
- Ab = abdomen exclusively
- AAr = ascending to arch
- AD = ascending to descending
- AAb = ascending to abdomen
- AI = ascending to iliac
- ArD = arch to descending
- ArAb = arch to abdomen
- ArI = arch to iliac
- DAb = descending to abdomen
- DI = descending to iliac

Clinical complications (C) related to dissection:
- C = complicated
  - Aortic valve involvement
  - Cardiac tamponade
  - Rupture
  - Branch vessel malperfusion: symptomatic branch vessel involvement defined as anatomic and clinical manifestations of branch vessel compromise (eg, static and/or dynamic branch involvement with accompanying stroke, paraplegia, coronary, mesenteric, visceral, renal, and/or extremity symptoms)
  - Progression of aortic involvement with proximal or distal extent of dissection
  - Other: uncontrollable hypertension, uncontrollable clinical symptoms, or rapid false lumen dilation and/or overall transaortic enlargement of > 10 mm within the first 2 weeks of initial diagnosis
- UC = uncomplicated (absence of complications listed above)

Thrombosis (T) of aortic false lumen:
- P = patent aortic false lumen: evidence of flow or contrast opacification within the false lumen throughout the length of dissected aorta
- CT = complete thrombosis of the aortic false lumen: no evidence of flow or contrast opacification within the following segments of the dissected aortic false lumen
  - A = ascending aorta
  - Ar = aortic arch
  - D = descending
  - Ab = abdomen
- PT = partial thrombosis of the aortic false lumen: longitudinal thrombosis of a portion of the aortic false lumen or circumferential thrombus that partially fills the false lumen constitute partial or incomplete thrombosis within the following segments of the dissected aorta:
  - A = ascending aorta
  - Ar = aortic arch
  - D = descending
  - Ab = abdomen

Data adapted from Dake MD, et al. Eur J Vasc Endovasc Surg. 2013;46:175–190.[2]

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