Endovascular Repair of the Thoracic Aorta: Lessons Learned

Frank J. Criado, MD, Omran R. Abul-Khoudoud, MD, Gregory S. Domer, MD, Christine McKendrick, RN, Mark Zuzga, DO, Nancy S. Clark, MD, Kerry Monaghan, BS, and Marcos F. Barnatan, MD

Center for Vascular Intervention and Division of Vascular Surgery, Union Memorial Hospital/MedStar Health, Baltimore, Maryland

Background. **Available information on outcome and best strategies for thoracic endovascular repair is somewhat limited and unclear. We sought to gain a better understanding of these issues through a retrospective review of our 8-year clinical experience in the treatment of thoracic aortic aneurysms and dissections.**

Methods. **A retrospective chart review of 186 patients undergoing stent-graft repair of thoracic aortic lesions at our institution during the 92-month period ending on December 31, 2004 was performed. Patients were divided into two groups based on the indication for treatment; group A had thoracic aortic aneurysms (TAA) and group B had type B aortic dissections (TBAD). Both groups were analyzed for outcome variables including technical success, mortality, major morbidity, endoleak rate and type, secondary endovascular interventions, and long-term survival. Mean follow-up was 40 months (range, 1 to 92 months).**

Results. **Compared to group B, group A patients were older and had a higher incidence of peripheral vascular disease and chronic obstructive pulmonary disease. Sixty percent of all patients were American Society of Anesthesiologists class III and the remainder were class IV (38.3%) and V (1.7%). The procedure was completed in 180 patients (96.7%), with all 6 failures being access-related. The average procedure time was 149 minutes (range, 72 to 405). The 30-day mortality was 4.7% (9 patients), and serious morbidity was 19.9% (37 patients). Eight patients (4.3%) developed spinal cord ischemia, 4 immediately after the procedure and 4 delayed (1 to 3 days). Total hospital length of stay averaged 6.7 days. Secondary endovascular interventions were successful in 17 patients with angiographically confirmed endoleaks (type I and III). At an average follow-up of 40 months, freedom from all-cause mortality was 62.5% in group A and 58.1% in group B.**

Conclusions. **Stent-graft repair for TAA and TBAD can be achieved with high technical success and comparatively low rates of morbidity and mortality. Midterm survival appears to be favorable. Further refinements in device technology and procedural techniques are needed.**

(Ann Thorac Surg 2005;80:857–63)

Stent-graft intervention for treatment of thoracic aortic diseases is an exciting new frontier in endovascular therapy. Beyond the initial report by Volodos and colleagues [1], Dake and colleagues' [2, 3] and Fann and Miller's [4] pioneering works at Stanford did the most to stimulate the evolutionary developments and growing interest that have taken place over the past several years. The availability of commercial devices and improvements in procedural capabilities catapulted all such procedures to the forefront of the endovascular specialty. Thoracic aortic aneurysms (TAA) and type B aortic dissection (TBAD) constitute the two most common indications for repair. Data are beginning to accumulate on best interventional strategies, technical results, and early and midterm clinical outcome [5–12], but much remains unclear or unknown. We sought to gain a better understanding of these issues through a retrospective review of our 8-year clinical experience with endovascular treatment of TAA and TBAD.

Patients and Methods

Two hundred and fifteen patients with thoracic aortic pathologies were treated endovascularly in the 92-month period ending December 31, 2004. However, only 186 could be classified as having TAA or TBAD and they represent the study group for all subsequent analysis. Patients were asked to sign an investigational informed consent approved by the Institutional Review Board, and made fully aware of the investigational nature of the therapy. Regulatory pathways used to gain access to thoracic stent-graft devices included "emergent" and "compassionate" cases, a phase I clinical study of the

Accepted for publication March 16, 2005.

Presented at the Fortieth Annual Meeting of The Society of Thoracic Surgeons, San Antonio, TX, Jan 26–28, 2004.

Address reprint requests to Dr Criado, 3333 N. Calvert Street, Suite 570, Baltimore, MD 21218; e-mail: frank.criado@medstar.net.

Dr Criado discloses that he has a financial relationship with Medtronic Vascular.

Published by Elsevier Inc

0003-4975/05/$30.00
doi:10.1016/j.athoracsur.2005.03.110

Talent thoracic stent-graft (Medtronic Vascular, Santa Rosa, CA), a physician-sponsored investigational device exemption, and the VALOR Talent thoracic trial. For analysis purposes, patients were divided into two groups: group A (n = 111) included patients with TAA (both true and false) and penetrating aortic ulcers; group B (n = 75) included patients with TBAD (both acute and chronic). For demographic data, χ^2 analysis was used to compare categorical comorbidity data. Age comparisons were analyzed by the Student *t* test.

Indications for Intervention

Group A patients were considered for endovascular repair when one or more of the following were present: symptoms (pain), maximum diameter of the sac greater than 5.0 cm, or more than twice the diameter of an adjacent nonaneurysmal segment of the thoracic aorta, or documented sac growth of greater than 0.5 cm over a recent 6-month period.

Indications for repair in group B patients included imperatives for intervention in 64 (85.3%): acute expansion of the false lumen (> 4.5 cm), organ or limb malperfusion, progressive or persistent pain. Eleven patients (14.7%) undergoing stent-graft procedures had uncomplicated acute aortic dissection.

Device Strategy and Procedural Techniques

Endograft sizing and design, and implant strategy, were based on evaluation of morphology by angiography, intravascular ultrasound (IVUS), and 3-mm slice contrast-enhanced spiral computed tomography (CT) with or without three-dimensional (3D) reconstructions. The procedures were performed in the operating room under fluoroscopic and angiographic guidance (40 degree left anterior oblique projection). Anesthesia used was general endotracheal in 124 (66.7%), spinal in 52 (27.9%), and local with intravenous sedation in 10 (5.4%) patients. The anesthesiologist was asked to maintain the mean blood pressure at 80 mm Hg or lower during deployment of the proximal-most endograft segment and ballooning. In TAA cases, the stent-graft device was placed to cover the length of the aneurysm and extend (if possible) a minimum of 20 mm proximally and distally. Endovascular strategy for TBAD patients, on the other hand, consisted of endograft placement from the origin (or just above) of the left subclavian artery (LSA) down to the proximal abdominal aorta, covering the full length of the descending thoracic aorta (DTA). An extra stiff (Lunderquist) guidewire was inserted retrograde into the proximal ascending aorta using a catheter exchange technique under fluoroscopy. Such stiff wire provided the necessary support for advancement and delivery of the endograft system. The device used in all instances was the Talent self-expanding thoracic stent graft (Medtronic Vascular, Santa Rosa, CA), with a 4 to 6 mm diameter oversize, and an uncovered proximal stent (bare spring) allowing for transostial placement with preservation of branch-artery patency (Fig 1).

Fig 1. Distal thoracic aortic aneurysm (left), repaired endovascularly with Talent stent-graft (middle, right).

Surveillance and Follow-Up

Stent-graft repair surveillance was performed using spiral thin section CT scans, with and without intravenous contrast and with 3D reconstruction, at intervals of 1 month, 6 months, 12 months, and then yearly from the date of the procedure. Patients with evidence of endoleak were further evaluated by angiography (and sometimes IVUS). Secondary interventions were performed when indicated (type I or III endoleak, or expanding sac). Clinical follow-up was also done at regular intervals and averaged 3.2 years. Actuarial survival and endoleak-free survival Kaplan-Meier curves were generated using the life-table method.

Results

Patients' demographics and comorbid risk factors are summarized in Table 1. Group A patients were older than patients in group B, and were more likely to have a history of peripheral vascular disease and chronic ob-

Table 1. Demographic and Clinical Risk Factors for Patients Undergoing Endovascular Repair of Thoracic Aortic Lesions

	Aneurysm	Dissection	*p* Value
Age (y)	72.4 ± 9.4	66.3 ± 15.8	0.005
Male (%)	54.5	52.5	0.82
Hypertension (%)	91.9	97.5	0.22
High cholesterol (%)	47.4	32.5	0.1
Myocardial infarction (%)	20.2	17.5	0.71
PTCA (%)	3.0	7.5	0.24
PVD (%)	27.3	5.0	0.003
Renal failure (%)	5.1	12.5	0.12
CABG (%)	12.1	7.5	0.42
CAD (%))	31.3	27.7	0.65
COPD (%)	29.3	10.0	0.02
Renal insufficiency (%)	10.1	2.5	0.13
Diabetes (%)	17.2	10.0	0.28

CABG = coronary artery bypss grafting; CAD = coronary artery disease; COPD = chronic obstructive pulmonary disease; PTCA = percutaneous transluminl coronary angioplasty; PVD = peripheral vascular disease.

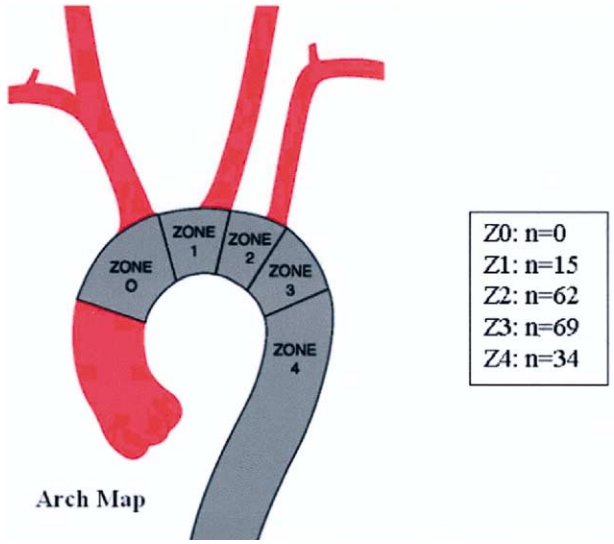

Fig 2. Arch map denoting zones of proximal endograft attachment. (Z0: n = 0; Z1: n = 15; Z2: n = 62; Z3: n = 69; Z4: n = 34.)

structive pulmonary disease (p value < 0.05). All patients had an American Society of Anesthesiologists class of III or higher (III: 60%; IV: 38.3%; V: 1.7%). Six patients in group A (5.4%) and 10 patients in group B (13.3%) had evidence of rupture at the time of the index procedure.

Endoluminal access was achieved by femoral cutdown in 162 patients (87.1%), an iliac artery access conduit in 23 (12.4%), and through the infrarenal bifurcated graft at the time of simultaneous open abdominal aortic aneurysm repair and endovascular TAA repair in 1 patient (0.5%). A diagnostic pigtail catheter was used in all cases; transbrachial in 134 (72%, 130 through the left brachial artery, 3 through the right, and 1 bilateral) and transfemoral in 52.

Six procedures (2.6%) were aborted because of access failure; all but one in female patients. Among the 180 patients actually receiving an implant, proximal endograft attachment was in the DTA more than 2 cm distal to the origin of the LSA in 34, within 2.0 cm of the LSA (parasubclavian) in 69, and proximal to the LSA in 77 (Fig 2). The endograft was placed across the origin of the LSA (occluding the vessel) in 62 (34.4%). Eighteen patients (9.7%) had preliminary adjunctive cervical operations designed to transpose and/or bypass the LSA and/or the left common carotid artery to enable more proximal stent-graft attachment within the aortic arch. Distal stent-graft attachment was at the level of T12 or lower in 123 (68.3%). Among these, the celiac artery origin was crossed with the uncovered stent in 9, and intentionally occluded (covered) by the endograft in 3. Nine patients with history of prior infrarenal aortic aneurysm repair had adjunctive placement of a cerebrospinal fluid drainage (CSFD) catheter in an effort to minimize or prevent risks of spinal cord ischemia (SCI). The average procedure time was 149 minutes (range, 72 to 405).

The 30-day mortality was 4.7% (n = 9). Four of the 9 patients who died had presented with ruptured TAA or TBAD. The mortality for intact (nonruptured) lesions was a low 2.9% (5/170), but it was 37% (6/16) among those who had ruptured prior to the index procedure.

Within 30 days of the procedure, nonfatal serious adverse events (SAE) occurred in 37 patients (19.9%). Eight patients (4.3 %) developed SCI: it was immediately apparent postoperatively in 4, but delayed in onset (1 to 3 days) in the other 4. Predisposing or precipitating conditions for this complication included prior abdominal aortic aneurysm (AAA) repair in 2, extensive aortic coverage (LSA to celiac artery) in 2, shaggy aorta in 1, hypotension in the setting of rupture in 1, and unclear in 2. The ischemic syndrome consisted of paraplegia in 3 patients (1 improved dramatically after CSFD), paraparesis in 2, and monoparesis in 3. Four of the five patients with monoparesis or paraparesis had complete resolution of their symptoms and one had dramatic improvement (4 received CSFD after the onset of symptoms). Three other patients (1.6%) sustained carotid (n = 2) or vertebro-basilar (n = 1) ischemic strokes that were felt to represent embolic phenomena, likely induced by catheter and guidewire manipulations in the aortic arch region. One patient recovered fully and the other 2 had significant improvement.

The remaining nonfatal SAEs were in order of decreasing frequency: respiratory complications prolonging hospitalization (n = 9), access vessel thrombosis, bleeding or rupture (n = 8), "post-implant syndrome" with severe back pain, fever, and leukocytosis lasting several days (n = 6), acute renal failure (n = 2), and lymph leakage from a cervical incision after a preliminary subclavian-to-carotid transposition (n = 1). The average hospital length of stay was 6.7 days (range, 1 to 45 days; median of 2 days).

Length of follow-up ranged from 1 to 92 months, and averaged 40 months. Eleven additional deaths have occurred: TAA-related in 2, TBAD-related in 1, unrelated in 5, and unknown in 3. The 2 aneurysm-related deaths (at 2 and 4 months) occurred in the setting of extensive thoracoabdominal aortic aneurysm (TAAA) with distal type 1 endoleak and failed secondary intervention (cuff extensions); in both cases, the aneurysm extended to or beyond the origin of the celiac artery. One patient with known proximal endoleak after repair of chronic TBAD died presumably from rupture after a carotid-carotid bypass in preparation for planned proximal extension into the arch.

Nineteen TAA patients (17%) were found (by CT) to have an endoleak within 30 days. Only 1 of these was apparent on completion angiography at the time of implantation. Diagnostic angiography confirmed the presence of a type 1 (attachment site) or type 3 (modular disconnection) endoleak in 17 of 19 patients uncovered by CT. It was negative in the other 2. There were no confirmed type 2 (branch flow) endoleaks. Secondary intervention was undertaken in all 17 cases with angiographic confirmation. Eleven patients underwent placement of extension cuffs (proximal n = 5, distal n = 6) between 1 and 6 months after the initial stent-graft procedure. Five patients had relining of their previous repair with a new endoluminal stent-graft system

that was deployed to seal a type 3 endoleak. Direct transcatheter (transbrachial) coil embolization of the endoleak nidus was performed in 1. Extension cuff placement and relining resulted in resolution of the endoleak in 13 of 15 (84%) patients, but failed in 2 whose thoracoabdominal aneurysm extended down to or beyond the celiac artery. They both died from aneurysm rupture (see above). Coil embolization of the endoleak nidus resulted in complete thrombosis in the one patient it was attempted. Finally, the 2 patients with negative angiogram (but positive evidence of endoleak on CT) have stable (nonenlarging) aneurysms; the endoleak persists in 1, but has resolved on the other.

The TBAD patients have been scrutinized in much the same manner as those with TAA after stent-graft repair. Among the 75 treated patients, 64 (85.3%) presented with an imperative for intervention (malperfusion, continuing pain, acute expansion of the false lumen, etc), but 11 (14.7%) had none. The dissection was acute (< 2 weeks from onset) in 56 (74.7%), and 10 patients (13.3%) had ruptured by the time the endovascular procedure was undertaken. Preoperatively, the false lumen was patent in 67 (89.3%) and thrombosed in 8 (10.7%). Endovascular repair resulted in thrombosis of the false lumen in the thoracic aorta (by CT at 1 to 3 months) in 70 of 75 (93%) patients. Continued patency of the false lumen in the abdominal segment, on the other hand, remained unchanged in the majority of cases where the dissection extended to the infrarenal segment or iliac arteries. Two patients underwent secondary intervention to extend endograft coverage of the true lumen to encompass the full length of the DTA. Both of these patients had been treated during the initial phase of our TBAD experience, before adopting the policy of endograft coverage of the entire DTA. In both cases, continued patency and pressurization of the false lumen had resulted in continued aneurysmal dilatation in the thoracic aorta.

Survival was analyzed by the life-table methodology using available long-term follow-up data (TAA, n = 99; dissection, n = 41). Kaplan-Meier curves were compared using a log-rank test. At the end of the study period, after an average follow-up of 40 months, 62.5% of group A and 58.1% of group B patients were still alive ($p = 0.54$) (Fig 3). The endoleak-free survival was 43% and 44.9% in groups A and B, respectively ($p = 0.9$) (Fig 4).

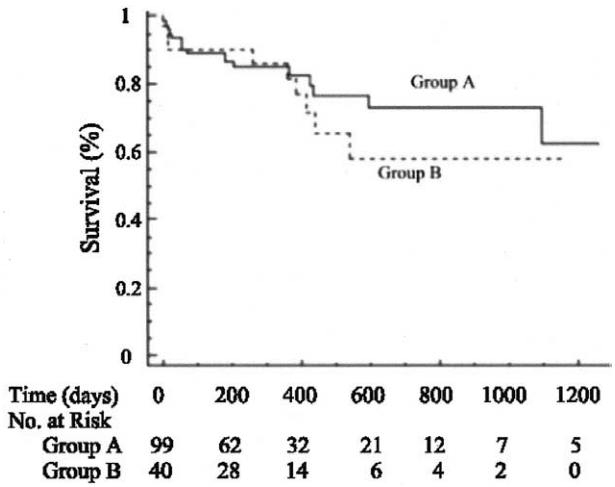

Fig 3. Kaplan-Meier survival curve of the two groups.

Comment

It is clear that endovascular treatment represents an exciting new treatment option for many patients with life-threatening thoracic aortic lesions, for whom standard surgical reconstruction carries significant or prohibitive risks. Technical approaches and overall strategies have evolved considerably in the recent past as a result of rapidly mounting experience. Numerous reports in the literature have described use of these techniques for a variety of conditions including degenerative aneurysms, traumatic injuries, acute and chronic type B dissections, aortoesophageal and aortobronchial fistulas, mycotic aneurysms, and penetrating aortic ulcers [12–23]. The advantages over standard open surgery are quite obvious: the avoidance of a thoracotomy incision, aortic cross-clamping, and cardiopulmonary bypass (among others) lead to enhanced safety and significantly lower rates of operative morbidity and mortality. Thoracic stent-graft technology, on the other hand, has not kept pace with these developments. Most devices in use today are "close cousins" or simple derivatives of endografts created for treatment of AAA. Their performance is less than optimal because they have not been designed to address the unique anatomic, hemodynamic, and pathologic features that characterize the aortic arch and DTA. Additionally, the bulky nature of most such devices with their large (> 22F) delivery systems represent significant limitations (and increased risk of arterial injury) for patients with small and/or diseased access femoral-iliac arteries. Such shortcomings can be traced back, at least in part, to the failure of leading investigators and endovascular industry as a whole to recognize the potential of stent-graft intervention in the treatment of thoracic aortic diseases. Fortunately, this situation is rapidly changing at this time. Significant technologic developments and new ideas are all but sure to emerge in the very near future.

Unlike abdominal aortic repair, where conventional surgical reconstruction is widely available and produces good results with relatively low mortality rates, open repair of the aortic arch and thoracic aorta is accompanied by very high (if not prohibitive) mortality and morbidity. Published mortality rates vary between 3% and nearly 30% for elective cases, and even higher in emergent situations [12, 18, 23–26]. Our own results, with an overall 30-day mortality of 4.7% and a low 2.9% rate in intact-nonruptured cases, are impressive examples of the advantages of the endovascular approach. The same cannot be said about stent-graft intervention for ruptured TAA and TBAD, where the overall mortality was 37%.

In addition to its role in aneurysmal disease and related conditions, stent-graft repair is emerging as an attractive treatment choice for complicated acute type B aortic dissection, where open surgical repair has pro-

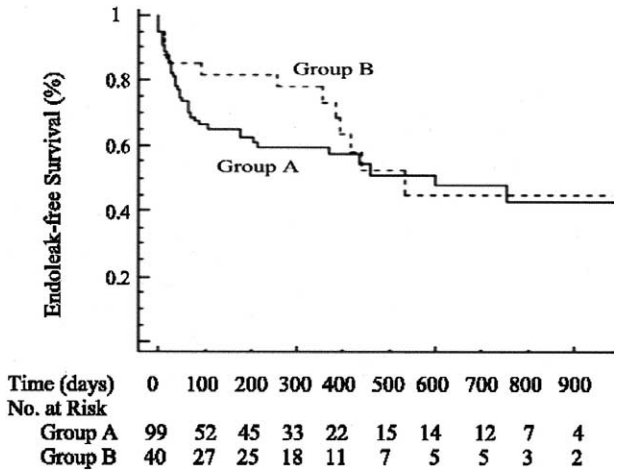

Fig 4. Kaplan-Meier curve depicting endoleak-free survival.

duced dismal results [12, 14, 26–28]. The principle of "simple" coverage of the entry tear, thereby redirecting flow exclusively down the true lumen and decompressing the false lumen, is sound and appealing. However, we have found that persistent flow in the false lumen and the need for secondary intervention can be minimized through more extensive endograft coverage during the initial endovascular procedure [27, 29]. The result has been the adoption of the new policy of covering essentially the whole length of the DTA with the endograft during endovascular repair of acute TBAD. While this may theoretically increase the risk of SCI and paraplegia, we have not had a single such occurrence. Stent-graft repair is particularly sound and effective for treatment of malperfusion, which is largely related to compression of the true lumen by the pressurized false channel. On the other hand, intervention for uncomplicated TBAD (14.7% in the current series) is controversial and cannot be based on available evidence. Randomized clinical trials will hopefully resolve this important issue in the near future. In the meantime, stent-graft repair should be used sparingly in the absence of clinical imperatives, although it is tempting to think that it should "logically" represent an improvement over the rather unfavorable natural history of untreated aortic dissection.

The incidence of SCI was 4.3% in this series, consistent with other published results [5, 6, 8, 30, 31]. Risk factors for this dreaded complication include concomitant or previous AAA repair, extensive endograft coverage of the aorta, and hypotension. Cerebrospinal fluid drainage has been shown to prevent SCI during open repair of TAAA (level I evidence) [32], which has prompted several investigators to do the same during endovascular repair [33, 34]. We adhere to this policy as well, and maintain CSF drainage intraoperatively and for 2 to 3 days postoperatively on all patients at risk, especially those who have had prior infrarenal replacement. A CSF catheter is also promptly inserted on any patient who develops SCI after repair.

Cerebral ischemia and strokes have also been reported as a complication of thoracic endovascular repair [25]. Three patients (1.6%) suffered such events in our series. They were likely caused by embolic phenomena from endovascular manipulation in the aortic arch. Advancement of the delivery system and actual graft deployment maneuvers may be implicated as well. It cannot be overemphasized that optimal interventional skills and gentle execution are paramount requirements for the safe conduct of these procedures. Further device refinements will also be helpful in this regard.

Not unexpectedly, endoleaks occur with a relatively high incidence. The rate in this series (17% of TAA patients) is consistent with other publications [17, 25]. It is noteworthy that all endoleaks were found to be type 1 (attachment site) or type 3 (modular disconnections). There were no instances of confirmed type 2 leaks. The latter, of course, are the most frequent type after stent-graft repair of AAA. Insufficient endograft coverage of the diseased aortic segment could be implicated in several of the cases, necessitating second intervention for placement of an extension cuff. More aggressive pursuit of good-quality completion angiography, including more frequent use of general endotracheal anesthesia that allows for respiratory cessation for better imaging, and more generous endograft coverage of the diseased aorta have emerged as important lessons learned from this experience. Type 3 leaks, on the other hand, may be related to insufficient overlap length between modular components. Relining the repair with a new endograft placed within it has worked well in such a scenario. Prevention of this problem can be achieved through the use of longer, single-segment endografts, and more generous (5.0 cm+) overlap of modular components when necessary.

Management of aortic arch branches ranks among the most significant unresolved and somewhat controversial issues [11, 13, 35]. In 34.4% of the cases in this series, the LSA origin was covered-occluded by the endograft device. By and large, we have found this maneuver to be very well-tolerated. Approximately 35% of the patients develop arm claudication, which tends to be self-limited and nonincapacitating. One patient died from bilateral cerebellar infarctions; he had preexistent occlusion or absence of the contralateral vertebral artery. At present, in all elective situations where coverage of the LSA is contemplated, patients are referred for magnetic resonance angiography studies where patency of the vertebral arteries and intactness of the circle of Willis are assessed. Preliminary performance of LSA transposition or bypass is reserved for patients with absent contralateral vertebral artery flow, or those with a history of coronary artery bypass graft using the left internal mammary artery. In all others, we prefer to maintain patency-continuity of the LSA, allowing placement of a transbrachial-LSA catheter that has been found to be so helpful for safe and precise placement of the stent-graft device in the aortic arch (Fig 5). Transcatheter coil embolization of the LSA (performed at the end of the procedure) can address issues related to possible backflow endoleak when proximity to or involvement by the TAA make such occurrence likely.

Issues related to endograft coverage of aortic branches at the caudad end of the endovascular repair are worth discussing as well. Most investigators (ourselves included)

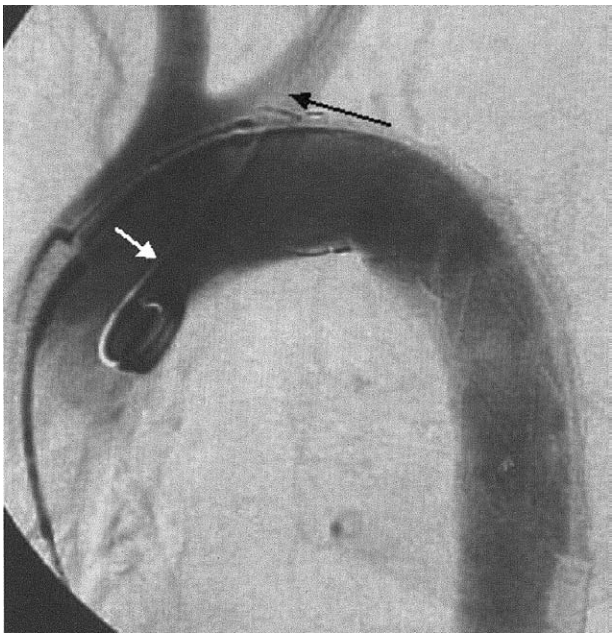

*Fig 5. Transbrachial left subclavian artery catheter (*white arrow*) used as the critical landmark (junction of left subclavian artery and aortic arch:* black arrow*) to guide device placement.*

would be reluctant or unwilling to cover-occlude the celiac artery, especially without prior angiographic demonstration of collateral filling from branches of the superior mesenteric artery. Albeit anecdotally, several instances of catastrophic ischemic complications have been reported. While we managed to achieve inconsequential intentional coverage of the celiac artery in three patients with extensive TAA, this maneuver should obviously be avoided as much as possible.

In conclusion, current advances with stent-graft thoracic intervention must be viewed as exciting new developments that offer hope to many patients with TAA, TBAD, and other serious conditions affecting the thoracic aorta. Techniques and technologies continue to evolve, and present-day concepts and results should be viewed within the context of work-in-progress. Most significantly, early and midterm results demonstrate a significant advantage over historic surgical outcomes. It is likely that these techniques will soon become first-line therapy for most cases with suitable anatomy. However, since little if any information is available on long-term results in terms of durability of the repair and device integrity, these procedures should probably be performed only within the confines of well-controlled clinical trials at present.

References

1. Volodos NL, Karpovich IP, Troyan VI, et al. Clinical experience of the use of self-fixing synthetic prostheses for remote endoprosthetics of the thoracic and the abdominal aorta and iliac arteries through the femoral artery and as intraoperative endoprosthesis for aorta reconstruction. Vasa 1991; 33(suppl l):93–5.
2. Dake MD, Miller DC, Semba RS, et al. Transluminal placement of endovascular sten-grafts for the treatment of descending thoracic aortic aneurysms. N Engl J Med 1994;331:1729–34.
3. Dake MD, Miller DC, Mitchell RS, et al. The first generation of endovascular stent-grafts for patients with descending thoracic aortic aneurysms. J Thorac Cardiovasc Surg 1998; 116:689–704.
4. Fann JI, Miller DC. Endovascular treatment of descending thoracic aortic aneurysms and dissections. Surg Clin North Am 1999;79:551–74.
5. Greenberg R, Resch T, Nyman U, et al. Endovascular repair of descending thoracic aortic aneurysms: an early experience with intermediate-term follow-up. J Vasc Surg 2000;31:147–56.
6. White RA, Donayre CE, Walot I, et al. Endovascular exclusion of descending thoracic aortic aneurysms and chronic dissections: initial clinical results with the AneuRx device. J Vasc Surg 2001;33:927–34.
7. Rodrigues Alves CM, da Fonseca JH, de Souza JA, et al. Endovascular treatment of thoracic disease: patient selection and a proposal of a risk score. Ann Thorac Surg 2002;73: 1143–8.
8. Bortone AS, Schena S, Mannatrizio G, et al. Endovascular stent-graft treatment for diseases of the descending thoracic aorta. Eur J Cardiothorac Surg 2001;20:514–19.
9. Taylor PR, Gaines PA, McGuiness CL, et al. Thoracic aortic stent-grafts-early experience from two centers using commercially available devices. Eur J Vasc Endovasc Surg 2001; 22:70–6.
10. Won JY, Lee DY, Shim WH, et al. Elective endovascular treatment of descending thoracic aortic aneurysms and chronic dissections with stent-grafts. J Vasc Interv Radiol 2001;12:575–82.
11. Criado FJ, Clark NS, Barnatan MF. Stent-graft repair in the aortic arch and descending thoracic aorta: a 4-year experience. J Vasc Surg 2002;36:1121–8.
12. Nienaber CA, Fattori R, Lund G, et al. Nonsurgical reconstruction of thoracic aortic dissection by stent-graft placement. N Engl J Med 1999;340:1539–45.
13. Criado FJ, Barnatan MF, Rizk Y, et al. Technical strategies to expand stent-graft applicability in the aortic arch and proximal descending thoracic aorta. J Endovasc Ther 2002; 9(suppl II):II32–38.
14. Beregi JP, Haulon S, Otal P, et al. Endovascular treatment of acute complications associated with aortic dissection: midterm results from a multicenter study. J Endovasc Ther 2003;10:486–93.
15. Chabbert V, Otal P, Bouchard L, et al. Midterm outcomes of thoracic aortic stent-grafts: complications and imaging techniques. J Endovasc Ther 2003;10:494–504.
16. Bell RE, Taylor PR, Aukett M, et al. Successful endoluminal repair of an infected thoracic pseudoaneurysm caused by methicillin-resistant Staphylococcus aureus. J Endovasc Ther 2003;10:29–32.
17. Fattori R, Napoli G, Lovato, L, et al. Descending thoracic aortic diseases: stent-graft repair. Radiology 2003;229:176–83.
18. Bell RE, Taylor PR, Sabharwal T, et al. Mid-term results for second-generation thoracic stent grafts. Br J Surg 2003;90: 811–17.
19. Marty-Ane CH, Berthet JP, Branchereau P, et al. Endovascular repair for acute traumatic rupture of the thoracic aorta. Ann Thorac Surg 2003;75:1803–7.
20. Eggebrecht H, Baumgart D, Schmermund A, et al. Endovascular stent-graft repair for penetrating atherosclerotic ulcer of the descending aorta. Am J Cardiol 2003;91:1150–3.
21. Daenen G, Maleux G, Daenens K, et al. Thoracic aorta endoprosthesis: the final countdown for open surgery after traumatic aortic rupture? Ann Vasc Surg 2003;17:185–91.
22. Coselli JS, Plestis KA, La Francesca S, et al. Results of contemporary surgical treatment of descending thoracic aortic aneurysms: experience in 198 patients. Ann Vasc Surg 1996;10:131–7.
23. Heijmen RH, Deblier IG, Moll FL, et al. Endovascular stent-grafting for descending thoracic aortic aneurysms. Eur J Cardiothorac Surg 2002;21:5–9.

24. Borst HG, Jurmann M, Buhner B, et al. Risk of replacement of descending aorta with a standardized left heart bypass technique. J Thorac Cardiovasc Surg 1994;107:126–33.
25. Scharrer-Pamler R, Kotsis T, Kapfer X, et al. Complications after endovascular treatment of thoracic aortic aneurysms. J Endovasc Ther 2003:10:711–8.
26. Crawford ES, Svensson LG, Coselli JS, et al. Aortic dissection and dissecting aortic aneurysms. Ann Surg 1988;208:254–73.
27. Cambria RP, Brewster DC, Gertler J, et al. Vascular complications associated with spontaneous aortic dissection. J Vasc Surg 1988;7:199–209.
28. Elefteriades JA, Hartleroad J, Gusberg RJ, et al. Long-term experience with descending aortic dissection: the complication-specific approach. Ann Thorac Surg 1992;53:11–21.
29. Bernard Y, Zimmermann H, Chocron S, et al. False lumen patency as a predictor of late outcome in aortic dissection. Am J Cardiol 2001;87:1378–82.
30. Gravereaux EC, Faries PL, Burks JA, et al. Risk of spinal cord ischemia after endograft repair of thoracic aneurysms. J Vasc Surg 2001;34:997–1003.
31. Kasirajan K, Dolmatch B, Ouriel F, et al. Delayed onset of ascending paralysis after thoracic aortic stent graft deployment. J Vasc Surg 2000;31:196–99.
32. Coselli JS, LeMaire SA, Koksoy C, et al. Cerebrospinal fluid drainage reduces paraplegia after thoracoabdominal aneurysm repair: results of a randomized controlled trial. J Vasc Surg 2002;35:631–9.
33. Tiesenhausen K, Amann W, Koch G, et al. Cerebrospinal fluid drainage to reverse paraplegia after endovascular thoracic aortic aneurysm repair. J Endovasc Ther 2000;7:132–5.
34. Ortiz-Gomez JR, Gonzalez-Solis FJ, Fernandez-Alonso L, et al. Reversal of acute paraplegia with cerebrospinal fluid drainage after endovascular thoracic aortic aneurysm repair. Anesthesiology 2001;95:1288–9.
35. Gorich J, Asquan Y, Siefarth H, et al. Initial experience with intentional stent-graft coverage of the subclavian artery during endovascular thoracic aortic repairs. J Endovasc Ther 2002;9(suppl II):II39–43.

DISCUSSION

DR PETER C. PAIROLERO (Rochester, MN): This presentation clearly is a wake-up call for all thoracic surgeons, and it represents a form of new technology that Dr Guyton discussed so eloquently in his presidential address two days ago. If thoracic surgeons do not learn this technique, we will not be doing thoracic aortic surgery in the future. This technology is clearly in the domain of the interventionists, and they are right before us now.

DR JOSEPH S. COSELLI (Houston, TX): I congratulate you on a wonderful presentation and your reporting upon both candid and sobering results. I must agree entirely that this is a wake-up call to all cardiac surgeons to embrace technology, future opportunities for new surgical interventions, and to continue to lead in stretching the envelope.

When we compare the endovascular treatment of aortic pathology against standard open surgical therapy, I believe it is helpful to remember that the latter is, as yet, the gold standard. Clearly, open surgical technique cannot compare with the shorter recovery time, reduced ICU time, abbreviated hospitalization, and possibly, with time, preferred economics. However, when I evaluated 469 descending thoracic aortic replacements, our 30-day mortality was 4%, the combined paraplegia and paraplegia rate was 2.6%, renal failure was 2.3%, and postoperative bleeding requiring return to the operating room was 2.6%. We, of course, encountered no endoleaks and none of our patients died of later rupture of their aneurysms.

I would like to pose the following questions. What would the authors' recommendation be with regards to mycotic aneurysms employing current endovascular technology? What are the limits of what we are currently able to offer from an endovascular standpoint? Who would the authors not treat? In other words, who would they absolutely select for operative intervention at this point in time?

There are at least eight reports in the literature with retrograde extension into the ascending aorta and aortic root of endovascular therapy, in effect converting a DeBakey type III dissection into a DeBakey type I; ie, Stanford type A dissection. The authors refer to placing the uncovered portion of the stent graft into the anatomic transverse arch. Do they have any other recommendations to prevent this dreaded complication (retrograde extension), which requires urgent open surgical intervention? What other modifications do the authors offer regarding safety in this respect? Finally, what recommendations do the authors make regarding the training of thoracic residents in this important, if not critical, emerging technology?

I do believe that stent grafting will find a role in the treatment of thoracic aortic aneurysms; it is pioneering work such as this, which will help us define both its role and its limitations. Thank you.

DR CRIADO: I appreciate Dr Coselli's remarks and certainly recognize his pioneering monumental work in this area. I agree that surgery continues to be the gold standard, but I would add that this is going to change rapidly in the near future, especially for treatment of lesion from the mid arch down.

He asked about mycotic thoracic aortic aneurysms. I would be very hesitant to place an endograft for that indication. On the other hand, my experience and view are that most cardiothoracic surgeons are unwilling to operate on such cases anyway. So sometimes we are forced to intervene, albeit reluctantly, but I would certainly remain concerned about the potential for development of a serious endograft infection.

In terms of the retrograde dissection issues, there are 19 reported cases of either retrograde dissection or perforation and ruptures, and the majority have been associated with the placement of an endograft with a proximal bare spring. Interestingly and intriguingly, we have not had a single such occurrence. It is my view that the insistence of securing proximal attachment within the mid or distal arch may have contributed to prevention of these complications. Ultimately, further technological refinements and new endograft designs will likely address all such issues in the near future.

The training issues are enormously important and difficult to sort out. I would like to emphasize the point that thoracic endovascular procedures can be extraordinarily difficult to perform, even for seasoned interventional specialists. The thoracic aorta is not the place to start one's endovascular experience, same as repair of arch aneurysms is not where surgical trainees would begin. It would be very difficult to achieve minimum acceptable competency to perform these procedures without a previous overall endovascular background and training. Another potential pathway would be an association or collaboration with an interventional specialist.

I really appreciate the opportunity to present this information. Thank you very much.