

Complications of endovascular repair of high-risk and emergent descending thoracic aortic aneurysms and dissections

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Purpose: The advent of endovascular prostheses to treat descending thoracic aortic lesions offers an alternative approach in patients who are poor candidates for surgery. The development of this approach includes complications that are common to the endovascular treatment of abdominal aortic aneurysms and some that are unique to thoracic endografting.

Methods: We conducted a retrospective review of 60 emergent and high-risk patients with thoracic aortic aneurysms (TAAs) and dissections treated with endovascular prostheses over 4 years under existing investigational protocols or on an emergent compassionate use basis.

Results: Fifty-nine of the 60 patients received treatment, with one access failure. Thirty-five patients received treatment of TAAs. Four of these procedures were performed emergently because of active hemorrhage. Twenty-four patients with aortic dissections (16 acute, 8 chronic) also received treatment. Eight of the patients with acute dissection had active hemorrhage at the time of treatment. Three devices were used: AneuRx (Medtronic; n = 31), Talent (Medtronic; n = 27), and Excluder (Gore; n = 1). Nineteen secondary endovascular procedures were performed in 14 patients. Most were secondary to endoleak (14 of 19), most commonly caused by modular separation of overlapping devices (n = 8). Other endoleaks included 4 proximal or distal type I leaks and 2 undefined endoleaks. The remaining secondary procedures were performed to treat recurrent dissection (n = 1), pseudoaneurysm enlargement (n = 3), and endovascular abdominal aortic aneurysm repair (n = 1). One patient underwent surgical repair of a retrograde ascending aortic dissection after endograft placement. Procedure-related mortality was 17% in the TAA group and 13% in the dissection group, including 2 acute retrograde dissections that resulted in death from cardiac tamponade. Overall mortality was 28% at 2-year follow-up.

Conclusion: Although significant morbidity and mortality remain, endovascular repair of descending TAAs and dissections in patients at high-risk patients can be accomplished with acceptable outcomes compared with traditional open repair. The major cause for repeat intervention in these patients was endoleak, most commonly caused by device separation. Improved understanding of these complications may result in a decrease in secondary procedures, morbidity, and mortality in these patients. The need for secondary interventions in a significant number of patients underscores the necessity for continued surveillance. (J Vasc Surg 2004;40:228-34.)

The successful development of endovascular prostheses for treatment of abdominal aortic aneurysms (AAA) has led to concomitant use of this technology for treatment of thoracic aortic disease. Because the morbidity and mortality associated with open surgical repair of thoracic lesions is high, the advantages of an endovascular approach to thoracic aortic aneurysms (TAAs) and dissections may exceed those of endovascular treatment of AAAs.¹⁻³ In addition, the advent of endovascular prostheses to treat descending thoracic aortic lesions offers an alternative approach in patients who are poor surgical candidates.

Several authors have reported early initial success with endografting of thoracic aortic lesions.⁴⁻⁹ Like endovascu-

lar treatment of AAAs, however, endoluminal treatment of TAAs and dissections is not without complications. This report describes a continuing single-center experience with endovascular management of thoracic aortic disease, including TAAs and dissections, with a variety of endovascular devices.

METHODS

The objective of the study was to analyze the single-center experience with endovascular treatment of thoracic aortic disease, including both emergent and elective TAAs, and acute and chronic thoracic dissection, with a combination of stent-graft systems. Patients received either AneuRx (Medtronic AVE), Talent (Medtronic AVE), and Excluder (W. L. Gore) thoracic devices over 4 years from October 1998 to January 2003. All patients were entered into a protocol approved by the institutional review board at our institution, and signed consent forms for the investigational devices and surveillance protocols used to generate the data reported. The inclusion and exclusion criteria for patients in the initial AneuRx investigational device exemption (IDE) have been detailed,⁵ and the later patients (Talent, Excluder) received treatment as part of an investigator IDE

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or, if they did not meet the entry criteria for the IDE, were reported as emergency use. An outline of indications in each subgroup is presented below. Prophylactic use of cerebrospinal fluid (CSF) drainage or other adjunctive therapy to prevent spinal cord ischemia was not routinely used. However, CSF drainage was used subsequent to development of lower extremity paresis.

Indications. Indications for treatment of TAAs included diameter greater than 5 cm; aneurysm diameter 4 to 5 cm, with an increase of more than 0.5 cm over 6 months; or saccular aneurysm or penetrating ulcer. Only patients with descending thoracic aortic dissection (Stanford type B) were considered candidates for treatment. Specific indications for treatment of thoracic dissection were acute dissection with intractable pain, uncontrollable hypertension, progression of dissection, or end-organ ischemia; chronic dissection with aneurysm dilatation of the proximal descending aorta; or chronic dissection with acute symptoms.

Thoracic devices. The AneuRx thoracic device, a self-expanding device, is constructed of polyethylene terephthalate fabric and nitinol stents, and is 23F in outer diameter. The thoracic devices are supplied in 6-cm and 12-cm long modular components, with stent-graft diameters of 32, 36, and 40 mm.⁵ The Talent thoracic devices are also constructed of self-expanding nitinol stents. They vary in length, width, and configuration, and can be customized with a maximum stent-graft diameter of 46 mm and maximum device size of 25F. Devices with exposed proximal or distal stents or "open-web" design are available.⁸ The Excluder also uses a self-expanding nitinol framework, which is combined with an expandable polytetrafluoroethylene graft.⁹ This was used in 1 patient on compassionate use basis. The devices used were 40 or 34 mm in diameter, 15-cm or 20-cm long, and 22F or 24F outer diameter. When multiple devices were used a complete overlap of at least 2 cm was used, with more overlap for severely angulated segments. Stent diameters were oversized at 10% to 20% compared with the diameter of the aorta at the fixation sites in aneurysms or 10% to 20% of the estimated adjacent proximal aorta in dissections.

Deployment. Both TAAs and dissections were approached similarly. Catheter access was obtained through unilateral or bilateral groin incisions after isolating the common femoral arteries. Generally a 0.025-inch guide wire was then passed through the entire length of the thoracoabdominal aorta, and intravascular ultrasound (IVUS) interrogation was performed with an 8.2-MHz catheter (Jomed) to define diameter and length of the proximal and distal landing zones. Proximal neck length was defined as the distance from the descending aorta just distal to the left subclavian artery to the origin of the aneurysm or dissection.

In dissections, the wire was passed with frequent interval IVUS imaging to ensure proper position of the wire in the true lumen of the aorta. Once proper position was confirmed, angiography was performed to define aortic branch vessel anatomy. Devices were then positioned with

confirmatory angiography before deployment, to enable precise positioning. In patients with short proximal necks a 5F pigtail catheter was percutaneously introduced from the right upper extremity, usually the radial artery, passed through the innominate artery, and positioned in the aortic arch, to aid in identification of the origin of the left carotid artery. Once deployed, endografts were assessed with a combination of angiography and IVUS to identify the presence of endoleak and evaluate the degree of device apposition.

Clinical data. Patient data including demographic information, preexisting comorbid conditions, clinical symptoms, procedure details, postoperative complications, secondary procedures, and mortality were prospectively collected as part of Food and Drug Administration–approved trials, as part of an investigator IDE, or treatment as emergency use. Patients were classified on the basis of the primary aortic disease, that is, TAA or dissection. Patients were screened preoperatively with computed tomography (CT), and followed up post-procedure with CT at 1 and 6 months, then yearly, to assess the success of aneurysm exclusion and to observe the morphologic characteristics of the aneurysms. Additional scans were obtained as needed to address specific device or anatomic considerations. The protocol consisted of helical or spiral mode CT with section reconstruction at 2 or 3 mm, collimation 5, and pitch 1:1.5. The CT angiograms were reconstructed in an interactive environment (Medical Media Systems). Maximum aortic diameters were obtained from centerline images.

RESULTS

Fifty-nine of 60 patients received treatment, with 1 access failure. Patient age ranged from 38 to 92 years (mean, 72 years). Lesions were American Society of Anesthesiologists grade II to V (mean, grade IV). The average maximum aortic diameter in patients with TAAs was 65 mm (range, 40–100 mm). Demographic comparisons between patients with TAAs and dissections are shown in Table I, and demographic comparisons between TAA and dissection subgroups are shown in Table II.

Thirty-five patients underwent treatment of TAAs. Specific primary indications for treatment are shown in Table III. Most TAAs were treated because of size or rapid expansion. Other elective indications included penetrating ulcers or saccular aneurysms. Four patients received emergent treatment because of active extravasation from the causes listed. Twenty-four patients received treatment of thoracic dissections. Specific treatment indications are shown in Table IV. In patients with chronic dissections, indications included increasing size of concomitant pseudoaneurysm and failure of medical therapy to control recurrent chest pain or hypertension. In patients with acute dissections, indications included enlarging pseudoaneurysm, failure of medical therapy to control pain or hypertension, and active extravasation with hemothorax or hemoptysis. One patient received treatment of a traumatic thoracic aortic dissection secondary to a motor vehicle accident.

Table I. Patient characteristics

	TAA (N = 35)		Dissection (N = 24)	
	Mean	Range	Mean	Range
Age (y)	73	38-92	69	43-86
ASA classification	IV	II-IV	IV	III-V
Maximum aortic diameter (cm)	6.5	4-10	5	2-10
Proximal neck length (cm)	5.0	0.5-10	3.0	0-10
	<i>n</i>	%	<i>n</i>	%
Emergent	4	11	9	38
Symptomatic	10	29	19	80

TAA, Thoracic aortic aneurysm; ASA, American Society of Anesthesiologists.

Table II. Patient characteristics

	TAA emergent (N = 4)		TAA elective (N = 31)		Dissection acute (N = 16)		Dissection chronic (N = 8)	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Age (y)	72	62-83	73	38-92	70	51-86	68	43-83
ASA classification	IV		IV	II-IV	IV	III-IV	IV	III-IV
Maximum aortic diameter (cm)	6.8	6-7	6.5	4-10	5	3-10	5	2-7
Proximal neck length (cm)	2.2	2-2.3	5.0	1-10	3	0-10	4	3-8

TAA, Thoracic aortic aneurysm; ASA, American Association of Anesthesiologists.

Table III. Primary treatment indications for thoracic aortic aneurysm

	Emergent (N = 4)	Elective (N = 31)
Size (>5 cm or rapidly expanding)		21
Penetrating ulcer		4
Saccular aneurysm		6
Extravasation		
Acute perforation	2	
Perioperative hemorrhage	1	
Bronchial-aortic fistula	1	

Table IV. Primary treatment indications for thoracic dissection

	Acute (N = 16)	Chronic (N = 8)
Pseudoaneurysm (>5 cm or rapidly expanding)	4	6
Failure of medical therapy	4	2
Extravasation		
Hemothorax	6	
Hemoptosis	1	
Traumatic aortic dissection	1	

Successful device deployment occurred in 58 of 59 patients (98.3%). Three thoracic devices were used: AneuRx (n = 31), Talent (n = 27), and Excluder (n = 1). Most procedures (66%) were performed with the patient under local anesthetic supplemented by intravenously administered hypnotic and analgesic agents as needed.¹⁰ The mean duration of the procedure was 197 minutes (range, 38-530 minutes). Median blood loss was 500 mL (range, 100-7500 mL; mean, 1066 mL). In most patients blood was captured in an autotransfusion canister and reinfused. Twelve patients (20%) required on average 0.5 units (range, 1-6 units) of additional blood transfusion. Patients were routinely admitted to the surgical intensive care unit postoperatively, with a median length of stay of 2 days (range, 1-33 days), followed by a median total length of stay of 7 days (range, 1-36 days).

Procedure-related mortality was 17% (n = 6) in the TAA group and 13% (n = 3) in the dissection group. Subgroup analysis of the TAA group revealed that 6 patients who underwent elective treatment sustained all of the perioperative mortality (19% vs 0%), whereas all 4 patients who underwent emergent procedures initially survived (Table V). Most of the procedure-related mortality in the TAA group was a result of perioperative myocardial infarction (MI; n = 3); 1 patient each died of intraoperative cardiac arrest, massive embolization, and multisystem organ failure.

Subgroup analysis of patients with dissections revealed that 2 patients in the acute group and 1 patient in the chronic group died, resulting in an identical perioperative mortality of 13% (Table V). One patient died of rupture due to endoleak. Two others died of retrograde dissection

Table V. Mortality

	<i>TAA emergent</i>	<i>TAA elective</i>	<i>Dissection acute</i>	<i>Dissection chronic</i>
Perioperative (<30 d) (%)	0	19	13	13
Overall (%)	50	35	19	13

TAA, Thoracic aortic aneurysm.

of the ascending aorta leading to cardiac tamponade. This complication was observed in 1 additional patient in the dissection group (discussed below), who ultimately underwent surgical conversion.

At average follow-up of 2 years, combined overall mortality for both groups was 28% (37% for TAA, 17% for dissection). With regard to TAAs, overall mortality for the emergent and elective groups was 35% and 50%, respectively. Of the 4 patients in the emergent group 2 later died, of MI and metastatic breast cancer, respectively. Five additional patients in the elective TAA group died at a mean of 9 months after treatment, for an overall mortality rate of 35%. Cause of death in three of these patients was cardiac secondary to coronary bypass, hepatic failure secondary to acute hepatitis A, and mesenteric thrombosis, respectively; 2 patients died of unknown causes. There was 1 late death in the acute dissection group, contributing to an overall mortality of 19% in that group. There were no late deaths in the chronic dissection group, in which the overall mortality rate remained at 13%.

Successful treatment of dissection was indicated by relief of pain, and sealing of acute perforations with control of hemorrhage and thrombosis of the false lumen. All patients who underwent treatment of dissection underwent thrombosis of the false lumen within the 1-month follow-up. One patient had recurrent proximal dissection within 2 months, and underwent placement of a proximal cuff. Proximal dilatation occurred in 3 patients who underwent treatment of dissections resulting in expanding proximal pseudoaneurysms. Because of concern about the potential for endoleak formation and rupture, additional proximal devices were deployed in each.

Two patients (3%) underwent surgical conversion. One patient who was being treated for a traumatic thoracic aortic dissection underwent immediate surgical conversion because of failure of the device to deploy correctly and exacerbation of the dissection with concern for rupture. One patient underwent surgical conversion with ascending arch repair after initial successful deployment of thoracic endografts to treat thoracic dissection, which resulted in subsequent development of a retrograde ascending aortic dissection. Both patients survived and were eventually discharged. Cumulative postoperative complications are listed in Table VI. The most common complication was arrhythmia ($n = 6$), followed by pleural effusion ($n = 4$), acute renal insufficiency, defined as a transient elevation in creatinine concentration to greater than 1.5 ($n = 4$), and urinary tract infection ($n = 4$). Three patients had acute ascending thoracic dissections. Paraplegia occurred in 1 patient (2%)

successfully treated for TAA but in whom lower extremity paralysis subsequently developed approximately 18 hours post-procedure. The rate of all major and minor complications was 59%, and the rate of major complications was 25%.

Nineteen secondary procedures were performed in 14 patients (24%). Indications for secondary intervention are listed in Table VII. The average time from initial procedure to secondary intervention was 12 months. Most of these procedures (14 of 19) were performed to treat endoleak, most commonly caused by the modular separation of overlapping devices ($n = 8$). Three patients underwent secondary procedures because of proximal pseudoaneurysm enlargement. One patient was treated for recurrent dissection. One patient underwent subsequent endovascular repair of AAA.

In addition to endoleaks caused by device separation, 4 type I leaks also occurred (3 distal type I in the TAA group, 1 proximal type I in the dissection group). These patients were treated expeditiously to prevent potential rupture. Two other unidentified leaks occurred in the TAA group. Although the origin of these leaks could not be identified, because there was evidence of aneurysm sac perfusion these were also treated without delay. No type II endoleaks were identified in either group. No endoleaks were identified in the chronic dissection subgroup. In addition, no difference in endoleak with regard to device type was identified.

Four patients in this study had short proximal neck lengths of 1 cm or less distal to the left subclavian artery. To achieve acceptable proximal fixation (>2 cm) in these patients endografts were deployed immediately distal to the left carotid artery, resulting in exclusion of the ostium of the left subclavian artery from the aortic arch. None of these patients underwent prophylactic left subclavian bypass. There were no complications resulting from occlusion of the ostium.

DISCUSSION

Open repair of thoracic aortic lesions entails an appreciable mortality rate even among patients who are thought to be reasonable candidates for repair. A recent study has reported mortality rates for elective open TAA repair of 8%. Patients operated on emergently do much worse. The same study reports mortality rates for these patients at 57%.¹¹ Najibi et al¹² showed that elective endovascular repair of TAA can be accomplished with similar mortality, shorter intensive care unit stay, and less blood loss compared with historical controls.

This study represents a single-center experience with treating emergent and high-risk patients with TAA and

Table VI. Cumulative postoperative complications

<i>Complication</i>	<i>TAA emergent</i>	<i>TAA elective</i>	<i>Dissection acute</i>	<i>Dissection chronic</i>	<i>Overall</i>
Cardiac					
Arrhythmia	1	3	2		6
CHF		1	2		3
Q-wave MI		3			3
Non-Q-wave MI		1			2
Cardiac arrest			1	1	2
Tamponade			1	1	2
Vascular					
Ascending thoracic dissection			1	2	3
Pulmonary					
Pleural effusion	1	1	2		4
Intubation >24 hr	1	1			2
ARDS		1	1		2
Pneumonia					1
Pulmonary edema		1			1
Renal					
Acute renal insufficiency		2	1	1	4
Acute renal failure	1	1			2
Stroke		1	1		2
Paraplegia		1			1
Urinary tract infection		1	2	1	4

TAA, Thoracic aortic aneurysm; CHF, congestive heart failure; MI, myocardial infarction; ARDS, acute respiratory distress syndrome.

Table VII. Indications for secondary procedure

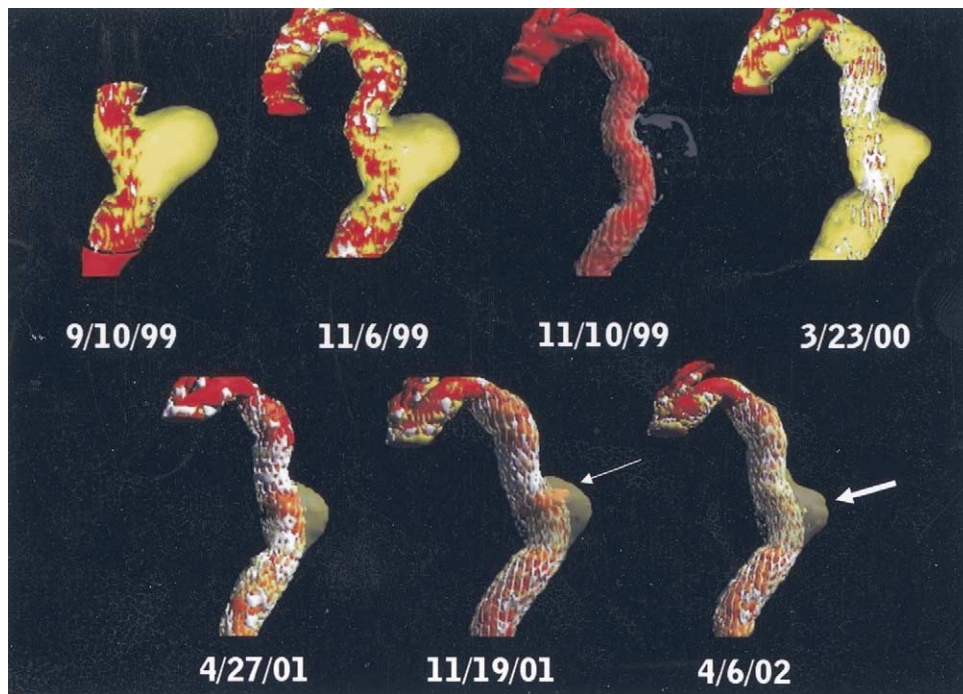
<i>Indication</i>	<i>TAA emergent</i>	<i>TAA elective</i>	<i>Dissection acute</i>	<i>Dissection chronic</i>	<i>Overall (N = 19)</i>
Endoleak					
Device separation	0	5	3	0	8
Proximal leak	0	0	1	0	1
Distal leak	0	3	0	0	3
Undefined leak	0	2	0	0	2
Enlargement					
Proximal	0	0	2	1	3
Distal	0	0	0	0	0
Recurrent dissection	NA	NA	1	0	1
AAA	0	0	0	1	1

TAA, Thoracic aortic aneurysm; AAA, abdominal aortic aneurysm; NA, not available.

dissection. Although direct comparisons with open repair are not possible in this study, it is worth noting that even among a patient population deemed at high-risk or ineligible for open repair because of preexisting comorbid conditions, combined perioperative mortality was 15%. Overall mortality was 28% with a mean follow-up of 2 years. These results are similar to previous reports of procedure-related mortality in patients undergoing endovascular treatment of TAA and dissections.^{4,6-8}

Although the patient population in this study was at high risk, the combined minor and major morbidity rate (60%) was similar to results of electively performed open repair. In addition, the cumulative rate of pulmonary events (17%), renal insufficiency (10%), and paraplegia (2%) compare favorably with previous results from series of elective open repair.

With regard to cardiac complications, we previously reported an incidence of (20%) for endovascular treatment of TAAs and dissections.¹³ This rate is similar to that reported for open TAA repair, and is congruent with previous reports in which the cardiac event rate for AAA was similar for open and endovascular approaches.^{14,15} These similarities suggest that cardiac complications may be a function of the anesthetic or other surgical factors, such as the inflammatory response, rather than the operative procedure itself. Because minor cardiac complications are included, primarily transient postoperative arrhythmias and non-Q-wave MI, the rate of cardiac complications in this study is higher than that previously reported for endovascular treatment of thoracic lesions.⁷⁻⁹ When cardiac events are limited to Q-wave MI, the cardiac complication rate decreases to 5%, which is in line with previous reports.



Composite sequential computed tomography angiogram reconstructions demonstrate follow-up of patient treated for descending thoracic aneurysm. Regression occurred until 11/01, when a new endoleak was identified (*small arrow*) originating from device separation. Additional thoracic endografts were deployed. Six-month follow-up (4/02) demonstrated continued regression with absence of endoleak.

Although not sufficiently powered to provide statistical comparison between subgroups, it is interesting to note that patients with TAA who required emergent treatment had the lowest procedure related mortality (0%), although all had active bleeding at presentation. In addition, procedure-related mortality in patients with dissections was identical (13%) between the acute and chronic groups, even though the primary indication for treatment in half of the acute group was active bleeding and none of the patients in the chronic group had active bleeding. These results suggest that, even among patients with the highest risk, endovascular repair can be undertaken with acceptable mortality. Given the high mortality in patients who undergo emergent open repair, these patients would seem to benefit most from endovascular treatment.

Most secondary interventions in this study were performed to treat endoleak, which was most commonly the result of modular device separation. Device separation was the leading cause of secondary intervention in both the TAA and dissection groups. Of interest, device separation was not a problem in the chronic dissection subgroup. The precise mechanism for development of this complication is unknown, but is likely due in part to thoracic aortic remodeling. As the thoracic aorta changes shape in response to endoluminal treatment or progression of disease, the fixed-length devices can separate. An example is shown in the Figure. This patient underwent treatment of a TAA, with subsequent regression of the aneurysm. Aneurysm regres-

sion and increased angulation ultimately led to device separation, with subsequent endoleak. Additional devices were used, and the patient continues to undergo regular surveillance.

To help prevent this complication we now preferentially use longer devices (≥ 100 mm), when available. In addition, we have modified our approach from a 20-mm overlap to as much as half the length of a given device (50-65 mm). A better approach might be use of a unibody device of sufficient length to treat the thoracic lesion with a single device. No such device is yet available.

The observation of these morphologic changes, in addition to development of endoleaks over time, underscores the need for continued surveillance. As is the case with endovascular treatment of AAA, postoperative surveillance is mandatory to recognize adverse morphologic changes, and device separation and type I leaks. More recently patients have been followed up with an endograft surveillance website available through the Lifeline Registry of the Society for Vascular Surgery/American Association for Vascular Surgery (<http://www.neri.org/html/research/clinical/lifeline.asp>).

Proximal open-wire configuration of thoracic endografts placed in the thoracic arch have been previously implicated as contributing to retrograde ascending aortic dissections in patients with TAA or dissection.¹⁶⁻¹⁸ All patients in this study with this complication were treated with this device configuration. Although reported in pa-

tients with TAA and dissection, all 3 patients in this study had dissections. Although similar in anatomic location, it is our belief that dissections represent a unique disease process that may respond differently to the forces exerted by certain open-wire stent configurations. For this reason and on the basis of our clinical experience, we selectively deploy open-wire stents in the aortic arch in patients with dissection, and are evaluating other custom-built covered stent device configurations as part of the investigator IDE.

Paraplegia occurred in 1 patient. This patient underwent seemingly successful endoluminal exclusion of the TAA without evidence of complications, but subsequently had delayed-onset paraplegia despite identification and deliberate avoidance of excluding any significant collateral vessels. Immediately at onset of symptoms a lumbar drain was placed for CSF decompression, and the hemodynamic status was optimized. Although the patient improved, he continues to have weakness in the lower extremities bilaterally.⁵

Our approach to avoiding this complication includes thorough delineation of large intercostal arteries, especially in the lower thoracic (T9-T12) distribution. Identification of these potential collateral vessels is accomplished with the aid of IVUS imaging in addition to preoperative CT. We do not cover large intercostal branches, if possible, although this is occasionally unavoidable. We do not use prophylactic CSF drainage. However CSF drainage is expeditiously used in any patient who demonstrates symptoms of spinal ischemia post-procedure.

Four patients required exclusion of the left subclavian artery to obtain adequate proximal neck length. We did not perform prophylactic left subclavian bypass in these patients. Our experience and that of others suggests that prophylactic subclavian bypass is not routinely necessary to prevent complications related to exclusion of the left subclavian artery.^{19,20} It should be noted, however, that some patients, for example, those with a dominant left vertebral artery, may require bypass to prevent complications. Since this study we have performed carotid-subclavian bypass for this very indication.

CONCLUSIONS

Although significant morbidity and mortality remain, endovascular repair of descending thoracic aneurysms and dissections in patients at high risk can be accomplished with acceptable outcomes compared with traditional open repair. Despite the favorability of these findings, complications unique to endovascular repair of descending thoracic aortic lesions (eg, device separation) did occur. The major cause for repeat intervention in these patients was endoleak, most commonly caused by device separation. These complications in part reflect the results of an evolving technology that involves a requisite learning curve and developmental issues. Improved understanding and implementation of this emerging technology in light of these complications may result in fewer secondary procedures in these patients, and may lead to reduced morbidity and mortality. The observed occurrence of endoleaks and con-

tinued proximal expansion underscore the need for continued surveillance.

REFERENCES

1. Svensson LG, Crawford ES, Hass KR, Coselli JS, Safi HJ. Experience with 1509 patients undergoing thoracoabdominal aortic operations. *J Vasc Surg* 1993;17:357-70.
2. Safi HJ, Miller CC III, Subramaniam MH, Campbell MP, Iliopoulos DC, O'Donnell JJ, et al. Thoracic and thoracoabdominal aneurysm repair using cardiopulmonary bypass, profound hypothermia, and circulatory arrest via left side of the chest incision. *J Vasc Surg* 1998;28:591-8.
3. Webb TH, Williams GM. Thoracoabdominal aneurysm repair. *Cardiovasc Surg* 1999;7:573-85.
4. Dake MD, Miller DC, Mitchell RS, Semba CP, Moore KA, Sakai T. The "first" generation" of endovascular stent-grafts for patients with aneurysms of the descending thoracic aorta. *J Thorac Cardiovasc Surg* 1998;116:689-704.
5. White R, Donayre CE, Walot I, Lippmann M, Woody J, Lee J, et al. Endovascular exclusion of descending thoracic aortic aneurysm and chronic dissections: initial clinical results with the AneuRx device. *J Vasc Surg* 2001;33:927-34.
6. Greenberg R, Resch T, Nyman U, Lindh M, Brunkwall J, Brunkwall P, et al. Endovascular repair of descending thoracic aortic aneurysms: an early experience with intermediate-term follow-up. *J Vasc Surg* 2000;31:147-56.
7. Cambria RP, Brewster DC, Lauterbach SR, Kaufman JL, Geller S, Fan C, et al. Evolving experience with thoracic aortic stent graft repair. *J Vasc Surg* 2002;35:1129-36.
8. 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.
9. Thompson CS, Gaxotte VD, Rodriguez JA, Ramaiah VG, Vranic M, Ravi R, et al. Endoluminal stent grafting of the thoracic aorta: initial experience with the Gore Excluder. *J Vasc Surg* 2002;35:1163-70.
10. Lippmann M, White R, Lee J, Kopchok G, Walot I, Wu Y. Endovascular repair of descending thoracic aortic aneurysms using fast track MAC anesthesia. *Anesth Analg* 2002;93:SCA1-112.
11. Clouse WD, Hallet JW, Schaff HV, Gayari MM, Ilstrup DM, Melton LJ. Improved prognosis of thoracic aortic aneurysms. *JAMA* 1998;280:1926-9.
12. Najibi S, Terramani TT, Weiss VJ, MacDonald J, Lin PH, Redd DC. Endoluminal versus open treatment of descending thoracic aortic aneurysms. *J Vasc Surg* 2002;36:732-7.
13. Bui H, Haukoos J, Donayre CE, White RA, de Virgilio C. Predictors of cardiac morbidity and mortality in patients undergoing endovascular repair of the thoracic aorta. *Ann Vasc Surg* 2004;18:22-5.
14. de Virgilio C, Bui H, Donayre C, Ephraim L, Lewis RJ, Elbassir M, et al. Endovascular vs. open abdominal aortic aneurysm repair: a comparison of cardiac morbidity and mortality. *Arch Surg* 1999;134:947-51.
15. Bui H, de Virgilio C. Preoperative cardiac evaluation and interventions before aortic surgery: are they justified? *Perspect Vasc Surg* 2000;12:25-54.
16. White RA, Donayre CE, Walot I, Hansen C, Aziz I, Kim B, et al. Retrograde ascending aortic dissection complicating endoluminal graft repair of thoracic aortic dissection. Submitted for publication.
17. Bethuyn N, Bove T, van den Brande P, Goldstein JP. Acute retrograde aortic dissection during endovascular repair of a thoracic aortic aneurysm. *Ann Thorac Surg* 2003;75:1967-9.
18. Totaro P, Miraldi F, Fanelli F, Mazzesi G. Emergency surgery for retrograde extension of type B dissection after endovascular stent graft repair. *Eur J Cardiothorac Surg* 2001;20:1057-8.
19. Gorich J, Asquan Y, Seifarth H, Kramer S, Kapfer X, Orend KH, 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.
20. Scharrer-Palmer R, Kotsis T, Kapfer X, Gorich J, Orend KH, Sunder-Plassmann L. Complications after endovascular treatment of thoracic aortic aneurysm. *J Endovasc Ther* 2003;10:711-8.

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