Complications after endovascular repair of acute symptomatic and chronic expanding Stanford type B aortic dissections

Dittmar Böckler, MD,^a Hardy Schumacher, MD, PhD,^a Marika Ganten, MD,^b Hendrik von Tengg-Kobligk, MD,^c Matthias Schwarzbach, MD, PhD,^a Christian Fink, MD,^c Hans-Ulrich Kauczor, MD, PhD,^c Hubert Bardenheuer, MD, PhD,^d and Jens-Rainer Allenberg, MD, PhD^a

Drs Böckler, Allenberg, and Schumacher
(left to right)

Supplemental material is available online.

Objective: To outline the complications after endovascular repair in patients with acute symptomatic and chronic expanding Stanford type B aortic dissections.

Methods: Between 1997 and 2004, of 125 patients with acute and chronic aortic type B dissections, 88 were treated conservatively. Thirty-seven patients (29 male, mean age 58 years, range 30-82 years) underwent endovascular repair (30%) using 44 stent grafts of 3 different designs: Excluder (W. L. Gore & Associates, Inc, Flagstaff, Ariz), Talent (Medtronic Vascular, Santa Rosa, Calif), and Endofit (Endomed, Inc, Phoenix, Ariz). Indications for treatment were acute symptomatic type B dissection in 15 patients, chronic expanding aortic dissection greater than 55 mm in 14, rupture in 3, and simultaneous type A repair in 5 patients. Twenty-two operations were performed on an emergency basis. Patient characteristics, procedural variables, outcome, and complications were prospectively recorded. All patients underwent follow-up by computed tomography before discharge, at 6 and 12 months, and annually thereafter (mean follow-up: 24 months).

Results: Correct deployment was achieved in 97% of cases. There were no instances of primary conversion, paraplegia, or stroke. Complete false lumen thrombosis was observed in 11 patients (44%). Perioperative complication rate was 22%. Thirty-day mortality rate in acute and chronic dissections was 19% and 0%, respectively. Freedom from aortic reintervention was 81%, 73%, and 68%, freedom from late rupture was 97%, 90%, and 80%, and overall success rate was 76%, 65%, and 57% at 1, 2, and 5 years, respectively. Results for patients with chronic dissections are significantly ($P = .038$) better than results in those with acute dissections.

Conclusions: Despite the minimally invasive approach, the complication and mortality rates for endovascular therapy of aortic dissections are still high. Frank reporting of these sequelae is of great importance to clarify the recent limitations of the method.

From the Departments of Vascular and Endovascular Surgery,^a Radiodiagnostics,^b and Anaesthesiology,^d Ruprecht-Karls University Heidelberg, and Department of Radiology,^c German Cancer Research Center (DKFZ), Heidelberg, Germany.

Hardy Schumacher reports receiving a consulting fee and payment for a workshop from WL Gore & Associates.

Received for publication April 24, 2005; revisions received Jan 14, 2006; accepted for publication Feb 21, 2006.

Address for reprints: Dittmar Böckler, MD, Abteilung für Gefäßchirurgie, Chirurgische Universitätsklinik Heidelberg, Im Neuenheimer Feld 110, 69120 Heidelberg Germany (E-mail: dittmar.boeckler@med.uni-heidelberg.de).

J Thorac Cardiovasc Surg 2006;132:361-8
0022-5223/\$32.00

Copyright © 2006 by The American Association for Thoracic Surgery

doi:10.1016/j.jtcvs.2006.02.056

Aortic dissection is most often a catastrophic event which, if untreated, can be potentially life threatening. In a population-based longitudinal study, Meszaros and associates¹ report mortality rates of acute untreated aortic dissection of 22.7%, 50%, and 68% within 6 hours, 24 hours, and the first week, respectively. The International Registry of Acute Aortic Dissection (IRAD) study, with an overall mortality of 27.4%, confirmed that aortic dissection is a lethal disease.² Open surgical repair also is associated with high morbidity and mortality.³

The advent of endovascular prostheses to treat descending thoracic aortic lesions offers an alternative approach in patients with dissections and severe comorbidities who are poor candidates for open surgery.^{4,5}

Abbreviations and Acronyms

ABF	= aortobronchial fistula
CEAD	= chronic expansive aortic dissection
CT	= computed tomography
IRAD	= International Registry of Acute Aortic Dissection
TLC	= true lumen collapse

However, endoluminal treatment is not without complications.⁶⁻⁸ Therefore, it is vital to report considerable complication rates and the midterm outcome after endografting. Hence, this report describes a continuing single-center experience with endoluminal stent-graft repair in acute complicated and chronic expanding aortic type B dissections, focusing on limitations and complications of endovascular management.

Patients and Methods

Between March 1997 and December 2004 (an 8-year period), 125 consecutive patients presented with acute and chronic Stanford type B dissections in our department. In all cases, diagnosis was confirmed with computed tomography (CT) or magnetic resonance imaging. Thereby, retrograde involvement of the ascending aorta and arch were excluded. Eighty-eight (70%) were treated conservatively and were followed-up routinely by CT scan on an annual basis. Thirty-seven patients with aortic dissection underwent trans-luminal endovascular stent-graft placement.

Patients

Patient evaluation, selection, and endovascular procedures were performed by vascular surgeons. All patients with suitable aortic morphologic characteristics were offered a stent graft as our preferred alternative to open repair. Clinical characteristics, imaging, and procedural data were documented prospectively. Fifty-nine percent ($n = 22$) of all operations were emergency interventions.

For this study, the records of 29 men and 8 women (mean age 57 years, range 30-82 years) were reviewed. Patient characteristics, indications, stent-graft data, and results of treatment are summarized in Table E1. Fifteen patients had visceral, renal, or leg ischemia with true lumen collapse (TLC), and 3 had rupture of the false lumen (1 presenting with aortobronchial fistula [ABF] 2 years after initial uncomplicated dissection). Fourteen patients had chronic expansive aortic dissection (CEAD) greater than 55 mm of maximum diameter. All patients treated on an emergency basis had some degree of chest pain as the first presenting symptom. Five patients were treated by endografting after type A dissection repair: 2 immediate repairs in cooperation with cardiac surgeons (2 antegrade deployments necessitated by bleeding at the distal anastomosis of the arch repair) and 3 staged procedures necessitated by TLC with malperfusion syndrome (1 visceral, 1 renal ischemia) and due to chronic aneurysmal expansion and consecutive infrarenal aortic rupture via transfemoral access.

Pre-procedural Imaging

All patients underwent spiral contrast-enhanced CT angiography with 3-mm slices and 3-dimensional reconstruction (Leonardo Workstation; Siemens AG, Erlangen, Germany; and Vitrea 2 workstation; Vital Images, Inc, Plymouth, Minn). Entry site, intended landing zone and diameter, distribution of abdominal vessels, length of dissection, and involvement of iliac arteries to evaluate the suitability for vascular access were determined. Measuring of the aortic arch diameter and the proximal anchoring zone was performed in the so-called "centerline" at the work station. Owing to the fragility of the aortic wall, oversizing was performed very gently (~10%) and only self-expanding endografts were implanted.

Device Details and Stent-Graft Placement

Six patients received more than 1 stent graft system simultaneously. Five patients received 2 devices and 1 patient received 3 devices. In total, 44 stent graft systems were implanted: 34 Excluder thoracic endoprostheses (TAG; W. L. Gore & Associates, Inc, Flagstaff, Ariz), 9 Talent endoluminal stent grafts (Medtronic Vascular, Santa Rosa, Calif), and 1 Endofit thoracic endoprosthesis (Endomed, Inc, Phoenix, Ariz) with lengths from 100 to 220 mm and diameters between 28 and 40 mm. The median stent graft-covered aortic segment was 169 mm (range 100-220 mm). Two surgeons equally qualified in endovascular experience performed all endovascular procedures with the patients under general anesthesia in an operating theater equipped with fluoroscopic and angiographic capabilities (series 9800; OEC Medical Systems, Inc, Salt Lake City, Utah) and a carbon-fiber operating table. Each patient received single-shot antibiotic therapy and 3000 units of heparin intravenously. With the exception of 1 patient (No. 30: antegrade approach during aortic arch repair), vascular access was obtained in 29 patients via the common femoral artery and in 7 patients by implanting a temporary 10-mm Dacron conduit to the common iliac artery because of small vessel size. The nondissected iliac/femoral artery, if iliac vessels were affected, was preferred for access. A 0.035-inch guide wire (Terumo, Frankfurt, Germany) was advanced and exchanged for a 7F calibrated angiography catheter. Digital subtraction angiography was performed with apnea followed by manual injection of 20 mL of nonionic contrast medium (Iopamidol [Solutrast 300], Byk Gulden, Konstanz, Germany). The aortic arch was rotated to about 45° in the left anterior oblique projection to adjust the parallax error for exact visualization of the proximal landing zones (minimum 15 mm). A sheath (up to 26F) was inserted over the guide wire and exchanged with a 0.035-inch, 260-cm Amplatz Super Stiff guide wire (Boston Scientific, Rattingen, Germany) and passed to the aortic arch. Temporary adenosine-induced cardiac arrest with an average dose of 60 mg was used for precise endograft placement in 26 patients. Additionally, all patients were provided with external pacemakers. Ballooning was not performed. Transesophageal echocardiography was not used routinely during deployment, and there was no standby heart-lung machine. Completion angiography was performed to assess accurate placement and exclusion of the entry site of the dissection. Stent grafts were only deployed at the entry sites to depressurize the false lumen and to induce thrombosis. Stent grafting of longer aortic segments was avoided because of fear of paraplegia. To achieve a sufficient proximal anchoring zone in

patients with CEAD and without any healthy landing zone, we performed one transposition of the left subclavian artery alone and polytetrafluoroethylene crossover bypass of both left common carotid and subclavian arteries in 2 patients before elective endografting.

Follow-up

Follow-up status between 1997 and 2004 is 100% complete and was performed in our department. The follow-up protocol included postoperative contrast-enhanced CT scanning and plain chest radiography before hospital discharge, 6 months and 12 months after endografting, and annually thereafter. The mean follow-up is 24 months (range 0–56 months). Cause of death was obtained by medical reports or by contacting the primary care physician.

Definitions

By definition, acute type B dissection is present when diagnosis is made within 2 weeks after the initial onset of symptoms. Primary success was defined as insertion and accurate placement of the stent graft in the intended landing zone, successful immediate entry occlusion, and lack of immediate perioperative complications without any additional interventions. End points and criteria for late outcome were false lumen thrombosis of the thoracic aorta, lack of thoracic aorta expansion, and freedom from rupture. Endoleak is defined as radiologic evidence of blood flow outside the stent graft according to published guidelines, except persistent retrograde false lumen perfusion.⁹ Reintervention is defined as any need for surgical or endovascular intervention on the treated aortic segment during follow-up. Treatment failure was defined as proximal type I endoleak, perioperative death, aortic reintervention, dissection-related death, and all sudden unexplained late deaths during follow-up.

Statistical Analysis

Comparison of acute and chronic dissections was done by the Wilcoxon signed-rank test. Kaplan-Meier life-table survival analysis was performed with XLSTAT V7.5.2 software (Addinsoft, New York, NY).

Results

Early Outcome

Procedural success. The overall primary technical success rate was 97%: Stent grafts were deployed successfully in all but 1 patient in whom device tracking was difficult. Endovascular repair was successfully performed in a second intervention 4 days later. The left subclavian artery was covered intentionally in 13 patients for tight proximal fixation and secure device attachment. One patient received an immediate subclavian-carotid bypass after inadvertent partial covering of the left carotid artery resulting from inaccurate placement. No primary conversion or additional simultaneous intervention was needed. The perioperative complication rate was 22% (cardiac 2, pulmonary 3, retrograde dissection 1, acute renal failure 1, subclavian steal syndrome 1). Paraplegia or stroke was not observed. One patient had subclavian steal syndrome and underwent by-

TABLE 1. Causes of early and late deaths in 37 patients

	Early deaths	Late deaths
Multiorgan failure, mesenteric ischemia	3	
Multiorgan failure, hepatic failure		1
Disseminated bleeding disorder	1	
Infection, sepsis	1	
Respiratory failure, aspergillosis	1	
Brain edema, central respiratory failure	1	
Aortobronchial fistula, hemoptysis		1
Cardiac failure		2
Infrarenal aortic rupture		1

pass grafting 2 days later. The perioperative additional reintervention rate was 13.5% (acute 18%, chronic 7%). Two patients required early aortic reintervention because of retrograde dissection and infrarenal rupture on day 5 (5.4%). Overall perioperative mortality (30 day) was 19%, all in acute cases. Multiorgan failure ($n = 4$) as the leading cause of death was observed predominantly in patients with continuing visceral ischemia. Five of 6 patients with TLC and mesenteric ischemia showed a drop of lactate and liver enzymes 12 hours after endovascular entry occlusion, and a return to normal on average 4.5 days after the intervention. A 50-year-old man with TLC and a 2-day history of abdominal pain, primarily treated successfully with immediate re-expansion of the true lumen on completion angiography, developed colon ischemia and multiorgan failure and died 2 days after subtotal colectomy. Two patients with complicated type A dissection died perioperatively; 1 died of respiratory failure and 1 of a bleeding disorder (Table 1). Thirty-day mortality was 10% for type B dissections and 57% for type A dissections.

Late Outcome

Survival. The overall survival at 1, 2, and 5 years after endografting was 81%, 64%, and 64%, respectively (Figure 1). There is significant difference ($P = .038$) in survivals between patients with acute and chronic type B dissections (Table 2, Figure 2). Overall mortality totals 27%. Causes of early and late deaths are listed in Table 1.

Endoleaks and material fatigue. On postoperative CT scanning, one type II endoleak and one late proximal type I endoleak were diagnosed. This patient, initially treated for CEAD, primarily refused secondary intervention and finally was treated by a proximal extension graft. The proximal endoleak persisted because of a diameter mismatch of the endograft and the aortic arch. The patient died of infrarenal false lumen rupture while she was scheduled for secondary conversion (No. 17). Plain chest radiographs showed no wire form fractures in any patients.

Reintervention. Freedom from aortic reintervention averaged 81%, 73%, and 68% at 1, 2, and 5 years: in acute

Figure 1. Overall survival rate. *CI*, Confidence interval.

cases, 82%, 73%, and 73%; in chronic cases, 80%, 74%, and 60% ($P = .412$) (Figure 3). During follow-up, no conversion (stent-graft explantation) was performed. One secondary proximal type I endoleak was diagnosed. In this patient (No. 17), initially treated for CEAD, a second proximal extension graft was inserted. The proximal endoleak persisted because of a disproportion of the different diameters of the endograft and aortic arch. She died of infrarenal false lumen rupture while she was scheduled for secondary conversion. A 78-year old man (No. 23) with ABF was treated by transfemoral coiling embolization of the false lumen to avoid conversion in a high-risk patient. He died 2 weeks later of another massive hemorrhage. One female patient with Marfan syndrome had chronic visceral aortic expansion and was treated with a hybrid procedure (case discussed later). Two more patients received aorto-biiliac bypass grafting 3 years after stent grafting.

Aortic rupture. Patient 23 had a rupture from the false lumen with ABF at 18 months. Repetitive hemoptysis after endograft placement was unsuccessfully treated earlier with coil embolization of the false lumen (Figure E1). Patient 17, as reported, had infrarenal rupture just before elective conversion. Actual freedom from rupture was 97%, 90%, and 80% at 1, 2 and 3 years, respectively.

False lumen thrombosis. Among 26 survivors, partial false lumen thrombosis of the stented aortic segment was

TABLE 2. Overall survivals of patients with acute, chronic, and overall type B dissection

	Survival (%)		
	1 year	2 years	5 years
Acute	62	62	62
Chronic	100	90	72
Overall	81	64	64

Figure 2. Rates of freedom from procedure-related death in patients with acute and chronic type B dissection.

induced in 7 patients and retrograde perfusion persisted by distal re-entries. Thrombosis of the false lumen of the entire descending thoracic aorta was observed only in 11 patients (44%). A still patent false lumen was observed in 3 patients.

Chronic abdominal aortic expansion. Overall expansion rate was 32% (12/37) and 48% among survivors. Only 12 patients (acute, chronic) showed no enlarged aortic diameter during follow-up. Expansion by more than 30 mm but less than 50 mm was seen in 7 patients with continuing surveillance by CT scan. Five patients (3 chronic, 2 acute) developed aortic expansion of more than 50 mm over a 2-year period, and except for 1, all underwent open abdominal aortic bifurcated bypass grafting. One female patient (No. 19) with Marfan syndrome had progressive dilatation of the abdominal aorta after valve replacement and thoracic

Figure 3. Rates of freedom from secondary interventions.

Figure 4. A, Three-dimensional reconstruction of postoperative CT scanning shows patent open retrograde bypass reconstruction of the celiac trunk, superior mesenteric artery, and left renal artery, a separate retrograde revascularization of the right renal artery (8-mm Dacron graft), and complete endovascular repair of the thoracoabdominal aneurysmal expanded aorta with 4 TAG devices (28-100 and 31-150 mm in reversed thrombone technique). B, Retrograde false lumen reperfusion is still documented, originating from the left common iliac artery.

endografting of the distal arch. In a staged hybrid procedure, she was treated by extra-anatomic mesenteric and renal revascularization and endografting of the thoracoabdominal aorta (Figure 4).

Total treatment failure. Actual freedom from all postoperative events (treatment failure including death) was 76%, 65%, and 57% at 1, 2, and 5 years, respectively. Patients with chronic dissections do significantly ($P = .034$) better than those with acute dissections (64%, 50%, and 45% vs 93%, 87%, and 73%) (Figure 5).

Discussion

Aortic treatment paradigms have evolved with the development of endovascular stent-graft repair of thoracic aortic aneurysms, which started at Stanford in 1992.¹⁰ The same authors published the only available study describing midterm results (4.5 years) of endovascular stent-graft repair of a thoracic aneurysm.¹¹ Although there are no long-term data to support definitive evidence, endovascular treatment is also proposed as an alternative therapy in patients with type B dissections. Whether stent grafting provides lasting protection from aortic rupture (especially false lumen rupture),

Figure 5. Rates of freedom from any adverse event (treatment failure).

from chronic aortic expansion, or from end organ ischemia still has to be determined. Therefore, we analyzed our midterm results for endovascular therapy on patients with type B aortic dissections.

The specific findings of this study highlight the problems and open questions of endovascular treatment in aortic dissections: The 30-day mortality rate of acute type B dissections after endograft implantation is still high (19%); in type A dissections it is as high as 57%. These disparate mortality rates likely pertain to patient selection bias and the acuity or pretreatment physiologic status of the patients. The high percentage of emergency interventions in this study (59%) and no diagnosis or delayed diagnosis of visceral ischemia in the prehospital time period may be a cause for patients dying of multiorgan failure despite technically successful entry closure. Many authors have published results on transluminal placement of endovascular stent grafts for the treatment of descending thoracic aortic dissections (Table 3).¹²⁻¹⁴ Dake and associates⁴ reported a series of 19 acute symptomatic dissections with 16% early mortality. This is in line with the 19% mortality in our own series. Early mortality in a third study from Palma and coworkers¹⁵ published an early mortality of 6% in 58 patients, and of those, 35 had acute dissection but were asymptomatic. A comparative study of both therapy modalities, “endovascular versus conservative,” for uncomplicated courses of asymptomatic patients is currently running. The excellent results achieved in 82 patients by Rehders and Nienhaber¹⁶ are difficult to interpret because of a lack of differentiation between acute and chronic dissections, ruptures, and ischemia. Frequently, no difference between the results of asymptomatic and symptomatic patients has been made. Numerous retrospective studies with univariate and multivariate data analysis have been carried out to

TABLE 3. Selected literature review of endovascular therapy in patients with Stanford type A/B dissections

First author	Patients (n)	Type of dissection	Technical success (%)	Thirty-day mortality rate	Major complications	Mean follow-up (mo)
Dake (1999) ⁴	19	A 4 B 15	100	3/19	4/19	4-28
Nienhaber (1999) ⁵	12	A 0 B 12	100	0/12	—	12
Rehders (1999) ¹⁶	94	—	100	0/94	5/94	12
Ince (2000) ²⁶	120	—	98	2/120	—	12
Lonn (2002) ²⁷	41	—	87.8	—	—	1-19
Orendt (2003) ¹⁹	14	B 14	100	0/14	9/14	22
Own series (2004)	37	A 7 (with descending tear, symptomatic) B 30	92	4/73/30	13/37	22

define those initial factors of the acute phase that determine the clinical course and the long-term prognosis.^{5,17} Mesenteric ischemia has a major impact on outcome. Two of our patients died of multiorgan failure because of visceral ischemia and unresolved malperfusion persisting postoperatively. One 48-year-old male patient (No. 27), who was stent grafted with clinically asymptomatic abdomen but occlusion of the celiac trunk and superior mesenteric artery, developed severe colon ischemia and died despite early entry closure within 48 hours. Malperfusion remains a major challenge. If mesenteric ischemia is suspected, an aggressive posture toward laparotomy or laparoscopy is appropriate, even if endovascular revascularization has been achieved. Following laboratory values for potential markers of mesenteric infarction can be crucial. Four of our patients died because of delayed recognition.

Long-term data suggest that up to 40% of asymptomatic patients will die of aorta-related causes or require a direct aortic reintervention over a 7-year period.¹⁸ Thirty percent to 40% of all symptomatic patients have complications over the further clinical course.¹ Consequently, achievement of complete false lumen thrombosis and prevention of aortic growth and expansion is the main goal in the treatment of dissections. We observed complete or false lumen thrombosis only in 44% of patients and severe visceral aortic expansion over 50 mm in 5 patients. In the series reported by Kato and associates,¹⁷ only 38.5% of chronic dissections showed complete obliteration during 27 months of follow-up.

Patients after type A repair and residual type B dissections are also potential candidates for “hybrid” distal stent grafting. The 30-day mortality of this approach in our series of 57% is closely related to complications after type A repair, to a sick and small cohort of 5 patients, and needs skeptical judgment. The strategy of immediate hybrid repair of the ascending and descending aorta including technical considerations of antegrade versus retrograde stent-graft deployments needs definitive future evaluation.

Spinal cord ischemia after stent-graft repair of dissections varies between 3% and 10% in the literature,¹⁹ 0% in this study.¹⁹ Potential factors for the lower incidence of spinal cord ischemia during endovascular repair are adequate collateral blood supply, no aortic crossclamping, short extension of stent graft-covered aorta, and deployment positioning far from the suggested origin of the artery of Adamkiewicz.

Sufficient proximal anchoring of 2 cm is necessary to avoid a proximal type I endoleak. To achieve this, coverage of the left subclavian or combined conventional transposition of the aortic arch branches may be necessary (Figure E2). We covered the left subclavian artery in 13 patients. One patient had clinically moderate dizziness and was observed without secondary intervention. Two patients had a proximal type I endoleak. Causes were incorrect endograft sizing and inflexible devices for the distal arch, which did not properly seal the proximal anchoring zone. Proximal tight fixation in the distal arch is mandatory and mostly makes covering the left subclavian artery necessary.

The importance of the chronic expansion and the factors of influence have been the subject matter of numerous studies.¹⁷ An initial diameter of more than 4 cm and a persistent entry into the false lumen have been identified as determinants for CEAD. Surgical intervention rate and associated mortality is reported to be high (30%-40%).²⁰ Overall expansion rate was 32% (12/37), and 48% among survivors in this study underlines this significant sequelae.

Asymptomatic patients with an open false lumen, an initially large aortic diameter greater than 40 mm, and ongoing “open entry” face unfavorable prognostic factors.²¹ The 1-year and 5-year survivals of asymptomatic patients under conservative medication amount to 94% and 86%, respectively. A comparison of these patients, however, to others who have undergone surgical therapy with a significantly higher risk is not permissible.

In a multicenter study comprising 465 patients, Hagan and associates² report a 3 times higher postoperative

mortality rate (31.4%) as compared with the conservative patient group (10.7%). Lansmann and colleagues,²² however, report a mortality of 0% with a fairly high associated complication rate of 47% in 34 patients with symptomatic type B dissection who had undergone surgery in the acute phase. In 1999, Nienhaber and colleagues⁵ published a small study that was the first prospective study to compare transluminal and open surgery, constituting that stent-graft repair is a viable therapeutic option.

Limitations

Further unanswered questions are the spontaneous course of an asymptomatic radiologic “TLC” and its potential influencing factors, which determine the fate of the true lumen. Experimental studies carried out by Chung and associates^{23,24} could prove the effectiveness of overstenting the intimal rupture site (“entry”) for treatment of TLC. However, as long-term results are still lacking, it remains to be seen whether this circumstance can be classified as a criterion of success or aim of therapy. After 2 years, false lumen thrombosis rate was only 44%. Persisting perfusion of the false channel correlates with late complications CEAD and death due to rupture.^{23,24} The meaning of patent false lumen in the further clinical course remains an open question. The term “true lumen collapse” (TLC) is difficult to interpret, as dissections are dynamic events with membrane movements, which can clearly be demonstrated by cine magnetic resonance imaging. The development of a specific endograft design for dissection therapy will become necessary. There is no ideal stent graft for dissections yet. The characteristics needed are gentle and flexible design, with no radial force and no bare springs. An important issue is the rigidity of the systems, which can cause penetration of the thin membrane. Of all the commercially available thoracic devices (Talent, Excluder, Endofit, and Zenith [Cook Incorporated, Bloomington, Ind]), we prefer the Excluder endoprosthesis, because of its access size and good flexibility. Finally, proof of the concept that endografts prevent long-term complications requires a prospective randomized trial. Our own therapy concept provides endovascular treatment in the symptomatic acute stage (any kind of ischemia, symptomatic TLC) or during the chronic course with aneurysmal expansion of more than 55 mm.²² Therefore, only 23% of 110 patients with Stanford type B dissection were selected for endovascular surgery.

Conclusion

In summary, the concept of endoluminal entry occlusion in acute or symptomatic type B dissections is promising and represents at least an alternative method of choice in symptomatic type B dissections. However, long-term fol-

low-up data are required and caution is needed using endovascular therapy, especially in asymptomatic patients. Of paramount importance is the reporting of complications. According to our results, we continue to be restricted with thoracic endografting in dissections. We do not treat asymptomatic patients with uncomplicated courses and CEAD until the aortic diameter reaches more than 55 mm.²⁵ The concept of endovascular treatment of type B dissections should be subjected to randomized evaluation to settle all the answers.

References

1. Meszaros I, Morocz J, Szilavi J, Schmidt J, Tomoci L, Nagy L, et al. Epidemiology and clinicopathology of aortic dissection. *Chest*. 2000; 117:1271-8.
2. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA*. 2000;283: 897-903.
3. 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.
4. Dake MD, Kato N, Mitchell RS, Semba CP, Razavi MK, Shimono T, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. *N Engl J Med*. 1999;340:1546-52.
5. Nienhaber CA, Fattori R, Lund G, Dieckmann C, Wolf W, von Kodolitsch Y, et al. Non-surgical reconstruction of thoracic aortic dissection by stent-graft placement. *N Engl J Med*. 1999;340:1539-45.
6. Hansen CJ, Bui H, Donayre CE, Abiz I, Kim B, Kopchok G, et al. Complications after endovascular repair of high-risk and emergent descending thoracic aortic aneurysms and dissections. *J Vasc Surg*. 2004;40:228-34.
7. Pamlor RS, Kotsis T, Görich J, Kapfer X, Orend KH, Sunder-Plassmann L. Complications after endovascular repair of type B aortic dissection. *J Endovasc Ther*. 2002;9:822-8.
8. Cambria R, Brewster D, Gertler J, Moncure A, Gusberg R, Tilson M. Vascular complications associated with spontaneous aortic dissections. *J Vasc Surg*. 1988;7:199-209.
9. Chaikof EL, Blankensteijn JD, Harris PL, White GH, Zarins CK, Bernhard VM, et al. Reporting standards for endovascular aortic aneurysm repair. *J Vasc Surg*. 2002;35:1048-60.
10. Dake MD, Miller DC, Semba CP, Mitchell RS, Walker PJ, Liddell RP. Transluminal placement of endovascular stent-grafts for the treatment of descending thoracic aortic aneurysms. *N Engl J Med*. 1994;331: 1729-34.
11. Demers P, Miller DC, Mitchell RS, Kee ST, Sze D, Razavi MK, et al. Midterm results of endovascular repair of descending thoracic aortic aneurysms with the first-generation stent grafts. *J Thorac Cardiovasc Surg*. 2004;127:664-73.
12. Beregi J-P, Haulon S, Otal P, Thony F, Bartoli JM, Crochet D, et al. Endovascular treatment of acute complications associated with aortic dissection: Midterm results from a multicenter study. *J Endovasc Ther*. 2003;10:486-93.
13. Bell RE, Taylor PR, Aukett M, Sabharwal T, Reidy JF. Results of urgent and emergency thoracic procedures treated by endoluminal repair. *Eur J Vasc Endovasc Surg*. 2003;25:527-31.
14. Matravers P, Morgan R, Bell A. The use of stent grafts for the treatment of aneurysms and dissections in the thoracic aorta: a single centre experience. *Eur J Vasc Endovasc Surg*. 2003;26:587-95.
15. Palma JH, de Souza JAM, Rodrigues Alves CM, Carvalho AC, Buf-folo E. Self-expandable aortic stent-grafts for treatment of descending aortic dissections. *Ann Thorac Surg*. 2002;73:1138-42.
16. Rehders TC, Nienhaber CA. Complications of stent graft placement in the thoracic aorta. In: Branchereau A, Jacobs M, editors. Complica-

- tions in vascular and endovascular surgery. Armonk [NY]: Futura Publishing Co; 2001. p. 85-192.
17. Kato M, Bai H, Sato K, Kawamoto S, Kaneko M, Ueda T, et al. Determining surgical indications for acute type B dissection based on enlargement of aortic diameter during the chronic phase. *Circulation*. 1995;92 (Suppl):II107-12.
 18. DeBakey M, McCollum C, Crawford E, Morris G, Howell J, Noon G, et al. Dissection and dissecting aneurysms of the aorta: twenty year follow-up of five hundred twenty-seven patients treated surgically. *Surgery*. 1982;92:1118-34.
 19. Orendt KH, Scharrer-Pamler R, Kapfer X, Kotsis T, Gorich J, Sunder-Plassmann L. Endovascular treatment in diseases of the descending thoracic aorta: 6-year results of a single center. *J Vasc Surg*. 2003;37:91-9.
 20. Juvonen T, Ergin MA, Galla JD, Lansman SL, McCullough JN, Nguyen K, et al. Risk factors for rupture of chronic type B-dissections. *J Thorac Cardiovasc Surg*. 1999;117:776-86.
 21. Kozai Y, Watanabe S, Yonezawa M, Itani Y, Inoue T, Takasu J, et al. Long-term prognosis of acute aortic dissection with medical treatment: a survey of 263 unoperated patients. *Jpn Circ J*. 2001;65:359-63.
 22. Lansman SG, Hagl C, Fink D, Galla JD, Spielvogel D, Ergin MA, et al. Acute type B dissection: surgical therapy. *Ann Thorac Surg*. 2002;74:S1833-5.
 23. Chung JW, Elkins C, Sakai T, Kato N, Vestring T, Semba CP, et al. True-lumen collapse in aortic dissection: part I. Evaluation of causative factors in phantoms with pulsatile flow. *Radiology*. 2000;214:87-98.
 24. Chung JW, Elkins C, Sakai T, Kato N, Vestring T, Semba CP, et al. True-lumen collapse in aortic dissection: part II. Evaluation of treatment methods in phantoms with pulsatile flow. *Radiology*. 2000;214:99-106.
 25. Böckler D, Schumacher H, Ockert S, Schwarzbach M, Klemm K, Hansmann J, et al. Acute and chronic expanding type B dissection: what is the impact of endografts on indications for treatment? *Gefäßchirurgie*. 2003;8:192-9.
 26. Ince H, Rehders TC, Nienaber CA. Thoracic aneurysms and type B dissections should be treated by stent-graft—for the motion. In: Greenhalgh RM, editor. The evidence for vascular and endovascular reconstruction. Philadelphia: W.B. Saunders, Elsevier Science Limited; 2002. p. 119-25.
 27. Lonn L, Delle M, Lepore V. Endograft therapy of the thoracic aorta in aortic dissections. *Cardiovasc Intervent Radiol*. 2002;25(Suppl):164-71.

Online—www.aats.org

Now you can get **The Journal of Thoracic and Cardiovascular Surgery** online. The Journal online brings you faster delivery time, easy searching of current and back issues, links to PubMed, AATS, WTSA, and other important sites, and more. Visit the Journal online today.

Receive tables of contents by e-mail

To receive the tables of contents by e-mail, sign up through our Web site at <http://journals.elsevierhealth.com/periodicals/ymtc>

Choose *E-mail Notification*

Simply type your e-mail address in the box and click the *Subscribe* button.

You will receive an e-mail to confirm that you have been added to the mailing list.

Note that TOC e-mails will be sent out when a new issue is posted to the Web site.

Figure E1. Postembolization chest radiography in a patient who had hemoptysis due to ABF 17 months after successful stent-graft placement for aortic dissection. Coils were placed into the false lumen above and below the detected and localized fistula tract.

Figure E2. Postoperative 3-dimensional reconstruction (Vitrea, Vital Images) after transposition and crossover bypass grafting of supra-aortic vessels and staged distal arch endografting to create a suitable neck for proximal stentgraft fixation. A, Anterior view. B, Posterior view (patent No. 34).

TABLE E1. Patient characteristics of 37 patients treated with endovascular stent graft placement for acute complicated or chronic expanding aortic type B dissection (numbered consecutively to procedure date out of total 112 patients with thoracic endografting)

No./consecutive patient	Age (y)/sex	Acute (emergency) or chronic (elective)/indication	Endograft model/size/No. of grafts	AICA:±/covered left subclavian/simultaneous conventional procedures	Complications	Outcome
1/16	36/M	Acute/visceral ischemia/previous ascending + aortic valve replacement, Marfan syndrome	TAG/31-130/1	+/yes/no	Subclavian steal syndrome > bypass	Alive at 56 mo
2/18	38/M	Acute/TLC, renal ischemia, previous A dissection	TAG/34-100/1	+/yes/iliac-renal vein bypass	Visceral ischemia, MOF	Died at 1 mo of MOF
3/22	71/M	Acute/TLC, AAA 56 mm	TAG/28-100/1	+/no/no	Acute coronary syndrome, PTCA and stenting on day 3	Alive at 49 mo
4/23	35/M	Chronic/TLC, chronic renal and visceral ischemia (weight loss 18 kg/3 mo)	TAG/26-100/1	+/no/no		Alive at 48 mo
5/28	61/M	Chronic/prevention of rupture—CEAD (60 mm), previous ascending + arch replacement	TAG/34-125/1	—/no/aorto-biiliac bypass		Alive at 47 mo
6/32	62/M	Chronic/CEAD—prevention of rupture/previous ascending + arch replacement	TAG/40-200/1	+/yes/no		Alive at 43 mo
7/33	58/M	Chronic/CEAD, prevention of rupture	TAG/34-200/1	+/no/no		Alive at 43 mo
8/35	40/M	Acute/ischemia left kidney, left leg	TAG/28-150/1		Uncleared retrograde A, dissection	Died at 1mo of MOF
9/39	79/F	Acute/TLC and visceral ischemia	TAG/34-200/1	+/yes/no	Respiratory insufficiency, reintubation	Died at 1.5 mo of cardiopulmonary failure
10/40	44/F	FL—contained rupture	TAG/34-200/1	+/yes/no		Alive at 41 mo
11/42	78/M	Acute/TLC, ischemia left kidney, right leg	TAG/34-200/1	+/yes/no		Alive at 41 mo
11/42	78/M	Acute/rCEAD	TAG/28-150, 34-150, 40-150 rev. thrombone/3	+/no/no	Endoleak type II, pleural effusion	Alive at 37 mo
12/43	58/M	Acute/TLC, visceral ischemia	TAG/34-200, 34-100 thrombone/2	+/no/no		Alive at 32 mo
13/46	41/M	Acute/Ehlers-Danlos	TAG/37-200/1	—/no/no		Alive at 39 mo
14/47	62/M	Acute/visceral ischemia	TAG/34-200/1	—/no/tube graft		Alive at 39 mo
15/49	62/F	Chronic/CEAD (65 mm), prevention of rupture	TAG/34-200, 34-150 thrombone/2	+/no/no	Proximal endoleak type I, progressive FL, expansion, second proximal endograft	Died at 21 mo of infrarenal rupture and persisting endoleak, while waiting for conversion

TABLE E1. Continued

No./consecutive patient	Age (y)/sex	Acute (emergency) or chronic (elective)/indication	Endograft model/size/No. of grafts	AICA:±/covered left subclavian/simultaneous conventional procedures	Complications	Outcome
16/51	68/M	Chronic/CEAD (mm)	TAG/37-150/1	+ /no/no		Alive at 39 mo
17/52	32/F	Acute/Marfan	TAG/31-150/1	+ /no /no		Alive at 36 mo. Distal aortic expansion, treated with hybrid procedure
18/60	80/M	Acute	TAG/37-150/1	+ /yes/no	Aspergillosis, long-term ventilation	Died at 0.6 mo of respiratory failure
19/65	46/M	Acute/visceral ischemia and right leg ischemia	TAG/34-150/1	+ /yes/no	Pneumonia, delirium	Alive at 26 mo
20/73	62/M	Chronic/CEAD—prevention of rupture (80 mm)	Endofit/38-220-24/1	+ /yes/subclavian—carotid bypass	Inadvertent partial covering of the left carotid ostium	Alive at 20 mo
21/74	78/M	Chronic/CEAD (55 mm) and ABF	Talent/40-115, 42-115-40/2	+ /yes/no	Persisting ABF, secondary endografting, selective coiling of the FL	Died at 18 mo of massive hemoptysis
22/79	67/M	Acute/Marfan	Talent/36-130/2	— /no/renal stenting		Alive at 18 mo
23/80	63/M	Acute/penetrating aortic ulcer	Talent/36-130 thrombone/2	+ /yes/no		Alive at 17 mo
24/83	82/F	Acute/TLC, renal and leg ischemia	Talent/36-130/1	— /yes/no	Persisting visceral ischemia	Died on day 1 of MOF
25/85	48/M	Acute/visceral ischemia	Talent/34-130/1	+ /no/no	Colon ischemia	Died on day 3 of MOF
26/91	60/M	Chronic/CEAD (55 mm)/renal cell carcinoma	TAG 34-150/1	+ /no/no		Alive at 9 mo
27/93	60/M	Chronic/CEAD (50 mm)	TAG/34-200/1	+ /no/no		Alive at 9 mo
28/96	61/M	Chronic/CEAD (55 mm)	TAG/34-150/1	— /no/no		Alive at 8 mo
29/97	65/M	Acute/CEAD, contained rupture FL, hemothorax	TAG/34-200/1	— /yes/no	Acute renal failure, hemodialysis, comatose tracheal respiration	Alive at 8 mo
30/100	48/F	Chronic/CEAD—prevention of rupture (60 mm)	Talent/26-130/1	— /no/no		Alive at 6 mo
31/102	50/M	Chronic/CEAD (mm), FL rupture before, conventional repair; primary suturing	TAG/40-200/1	+ /yes/no		Alive at 5 mo
32/105	50/M	Chronic/CEAD (48 mm) and large left subclavian artery aneurysm	TAG/37-150/1	+ /yes/yes	Thoracotomy, major bleeding	Alive at 3 mo

TABLE E1. Continued

No./consecutive patient	Age (y)/sex	Acute (emergency) or chronic (elective)/indication	Endograft model/size/No. of grafts	AICA:±/covered left subclavian/simultaneous conventional procedures	Complications	Outcome
33/108	78/F	Chronic/CEAD (62 mm)	TAG/34-200/1	+/yes/no	Postoperative asystole, successful reanimation, hypovolemia	Alive at 2 mo
34/112	61/M	Acute/persistent chest pain, diameter at entry site: 52 mm	TAG/34-200/1	+/yes/no		Alive at 0.5 mo
35/105	50/M	Chronic/CEAD (48 mm) and large left subclavian artery aneurysm	TAG/37-150/1	+/yes/yes	Thoracotomy major bleeding	Alive at 3 month
36/108	78/F	Chronic/CEAD (62 mm)	TAG/34-200/1	+/yes/no	Postoperative asystole, successful reanimation, hypovolemia	Alive at 2 mo
37/112	61/M	Acute/resistant chest pain, diameter at entry site: 52 mm	TAG/34-200/1	+/yes/no		Alive at 0.5 mo

CEAD, Chronic expanding aortic dissection; rCEAD, ruptured; TLC, true lumen collapse; MOF, multiorgan failure; FL, false lumen; ABF, aortobronchial fistula.