Complicated acute type B aortic dissection: Midterm results of emergency endovascular stent–grafting

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Objective: This study assessed midterm results of emergency endovascular stent–grafting for patients with life-threatening complications of acute type B aortic dissection.

Methods: Between November 1996 and June 2004, 16 patients with complicated acute type B aortic dissections (mean age 57 years, range 16–88 years) underwent endovascular stent–grafting within 48 hours of presentation. Complications included contained rupture, hemothorax, refractory chest pain, and severe visceral or lower limb ischemia. Stent–graft types included custom-made first-generation endografts and second-generation commercial stent–grafts (Gore Excluder or TAG; W. L. Gore & Associates, Inc, Flagstaff, Ariz.). Follow-up was 100% complete, averaged 36 ± 36 months, and included postprocedural surveillance computed tomographic scans.

Results: Early mortality was 25% ± 11% (70% confidence limit), with no late deaths. No new neurologic complications occurred. According to the latest scan, 4 patients (25%) had complete thrombosis of the false lumen; the lumen was partially thrombosed in 6 patients (38%). Distal aortic diameter was increased in only 1 patient. Actuarial survival at 1 and 5 years was 73% ± 11%; freedom from treatment failure (including aortic rupture, device fault, reintervention, aortic death, or sudden, unexplained late death) was 67% ± 14% at 5 years.

Conclusion: With follow-up to 9 years, endovascular stent–grafting for patients with complicated acute type B aortic dissection conferred benefit. Consideration of emergency stent–grafting may improve the dismal outlook for these patients; future refinements in stent–graft design and technology and earlier diagnosis and intervention should be associated with improved results.

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The feasibility of endovascular stent–graft repair for descending thoracic aortic pathology was established more than 15 years ago.[1] Subsequently, stent–grafts have been used to treat aneurysmal disease, intramural hematoma with penetrating ulcer, traumatic aortic disruption, and acute or chronic aortic dissection.[2,3] What remain unknown are the long-term efficacy and durability of stent–grafts associated with these various disease entities. Because it is less invasive than a conventional open surgical procedure,[4] the endovascular approach for repair of descending thoracic aortic aneurysms is associated with less early postprocedural morbidity and mortality.[5] Although complications occur, they are usually manageable by endovascular means, with the necessity for surgical conversion being infrequent.[6] Complicated acute type B aortic dissection is perhaps the most promising application for endovascular stent–grafting,[6] because surgical repair has been accompanied by high morbidity and mortality.[7-14] Nonetheless,

Abbreviations and Acronyms
CTA = computed tomographic angiography
PIT = primary intimal tear

acute type B dissection remains a challenging endovascular target, in that it requires coverage of the primary intimal tear (PIT) in addition to correction associated end-organ malperfusion, whether the ischemic mechanism be static or dynamic.[15,16]

Although several reports of patients with aortic dissection treated with stent–grafts have been published, these mixed series frequently commingled type A and type B dissections, acute and chronic dissections, or complicated and uncomplicated cases; further, clinical follow-up has been short, hindering critical assessment of results. We report our midterm stent–graft results in patients with acute type B aortic dissection with life-threatening complications.

Materials and Methods

From October 1996 to June 2004, a total of 16 consecutive patients were treated by a multidisciplinary team (interventional radiology and cardiovascular surgery) at Stanford University. Emergency endovascular treatment was carried out with first-generation, custom-fabricated devices initially and then later with a Gore Excluder or TAG commercial stent–graft (W. L. Gore & Associates, Inc, Flagstaff, Ariz) under a physician-sponsored IDE investigational protocol approved by the Stanford University School of Medicine institutional review board. Informed consent was obtained from each patient or a responsible relative. Computed tomographic angiography (CTA) confirmed the diagnosis, assisted in surgical planning, and was used for follow-up. All patients were initially admitted to the cardiovascular surgical intensive care unit for stabilization, usually because of threatened rupture, persistent pain, rapid expansion, or critical visceral or lower limb ischemia.

Cases of acute type B aortic dissections were judged to be complicated if one or more of the following four clinical problems were present: (1) ongoing intractable chest pain, despite maximal medical therapy; (2) clinical or radiographic evidence of rupture or impending rupture; (3) symptomatic presentation with an aortic diameter greater than 5 cm or documented rapid aortic enlargement; and (4) symptomatic branch vessel occlusion with distal organ ischemia, as determined by clinical symptoms or laboratory testing.

Patients were excluded if arterial access was inadequate (small [<7 mm] iliac or femoral arteries) or if the PIT was located within 1 cm from the left common carotid artery origin. Patients with Marfan syndrome or other connective tissue disorders were excluded (contraindication to stent–grafting in our institutional review board protocols), but a 16-year-old patient with type IV (vascular type) Ehlers–Danlos syndrome who was initially seen with aortic rupture and paraplegia was treated on a compassionate use basis. Three patients were first seen with a propagating (>20 cm) genuine aortic dissection caused by traumatic aortic disruption at the level of the ligamentum, but patients with isolated traumatic tears and localized traumatic pseudoaneurysms were excluded. Spontaneous dissection was the etiology in the remaining 12 cases.

Preprocedural Imaging

All patients underwent thin-slice CTA with 3-dimensional reconstructions to determine the exact location of the PIT, the distance from the PIT to the left subclavian and carotid arteries, the lengths and diameters of the diseased aortic segments, the morphologic characteristics of the proximal and distal landing zones, the diameters of the true and false lumens, and the mechanism of end-organ malperfusion. The stent–graft was oversized by 10% to 15% relative to the proximal true lumen to make it just slightly larger than the distal aortic arch diameter. When possible, the stent–graft was deployed at least 20 to 40 mm beyond the PIT to ensure adequate wall contact and a tight circumferential seal.

Stent–graft Devices

First-generation stent–grafts were custom fabricated from modified Gianturco Z stents (Cook Medical Inc, Bloomington, Ind) that were wired together and then covered with a woven polyester graft (Boston Scientific Corporation, Natick, Mass). A 20F to 24F pusher-rod sheath delivery system (Cook) was used for deployment of the first-generation devices in the initial 8 patients. Second-generation, commercially manufactured Gore Excluder or TAG stent–grafts became available later and were used in the remaining 8 patients. These devices consist of a self-expanding nitinol exoskeleton lined with expanded polytetrafluoroethylene graft material, require the introducer sheath be inserted only as far as the abdominal aorta, are advanced over a coaxial wire, and are rapidly deployed by a drawstring mechanism that releases a constraining shroud.

Details of stent–graft deployment techniques have been described previously.[1,6] With the patient under general anesthesia in an operating room-equipped catheterization laboratory with a Siemens 2300 Imaging System (Siemens Medical Systems, Inc, Ultrasound Group, Issaquah, Wash), transesophageal echocardiography, angiography, and occasionally intravascular ultrasonography were used to define the aortic true and false lumens. After arterial access was gained through either the femoral or iliac artery, a hydrophilic guide wire was advanced into the aortic arch and exchanged for a stiff wire; the delivery system dilator–sheath was then advanced into the aortic true lumen, and the stent–graft was advanced to the target to cover the PIT completely. After optimal positioning of the device, the stent–graft was deployed. Ballooning of the stent–graft was performed only if a large type IA proximal endoleak was documented, and only at the proximal landing zone. When needed, a second stent–graft was placed. Angiography confirmed the absence of endoleaks and determined the perfusion status of previously ischemic arterial beds.

Follow-up and Definitions

Before discharge, at 3, 6, and 12 months, and then annually thereafter, we attempted to obtain follow-up CTA scans for all patients. Aortic diameter was assessed just below the distal end of the stent–graft. Clinical status was ascertained by contacting the patients or their primary physicians. Causes of death were determined by reviewing death certificates and, when available, autopsy reports. Technical success was defined as exclusion of the false lumen with reperfusion of previously ischemic, mesenteric, renal, or lower extremity arterial beds. Treatment failure was defined as a composite

TABLE 1. Demographic patient data

Patient	Sex	Age (y)	Comorbidities	Clinical Indicators
1	Male	43	HPB	Refractory HPB, chest pain
2	Male	53	HPB	Refractory HPB, leg ischemia
3	Male	46	HPB	Chest pain, mesenteric ischemia, claudication
4	Male	37	HPB	Refractory HPB, renal ischemia
5	Female	40	HPB	Mesenteric ischemia
6	Male	16	HPB/delirium	Refractory chest pain, pleural effusion
7	Male	54	Ehlers–Danlos	Refractory HPB, chest pain
8	Female	40	HPB	Mesenteric ischemia
9	Female	74	HBP/renal fibrodysplasia	Refractory HPB, chest pain
10	Male	65	HPB	Renal ischemia
11	Female	58	HPB	Refractory HPB, chest pain
12	Male	83	Trauma	Extensive pleural effusion
13	Male	87	Trauma	Large pleural effusion
14	Male	62	Trauma	Leg ischemia
15	Male	74	HPB	Large pleural effusion
16	Female	75	HPB	Refractory HPB, chest pain

HBP, Arterial hypertension.

TABLE 2. Stent–graft and aortic (combined true and false lumen) diameters at primary intimal tear level

Patient	Stent–graft type	Stent–graft units and size (mm)	Acute aortic diameter (mm)	Follow-up (>3 mo) aortic diameter (mm)
1	Custom made	2, 28 × 75	45	48
2	Custom made	38 × 50	50	53
3	Custom made	36 × 45	45	50
4	Custom made	32 × 50	23	45
5	Custom made	30 × 77,5	38	Died
6	Custom made	36 × 50	40	Died
7	Custom made	35 × 70	35	Surgery
8	Custom made	26 × 75	36	39
9	Excluder	31 × 100	41	50
10	Excluder	31 × 100	42	40
11	Excluder	26 × 100	26	Died
12	Excluder	40 × 150, 40 × 100	44	45
13	Excluder	34 × 150	45	32
14	Excluder	31 × 150	32	Died
15	Excluder	37 × 200	48	47
16	TAG	34 × 125	38	40

Excluder and TAG are registered trademarks of W. L. Gore & Associates, Inc, Flagstaff, Ariz.

end point including perioperative death, primary or secondary endoleaks, late device failure, the necessity for reintervention, late aortic-related death, and all sudden, unexplained late deaths, as previously advocated by our group. Continuous variables are expressed as mean ± SD and median values; important rates and fractions are expressed with 70% confidence limits. The Kaplan–Meier method generated actuarial survival estimates, which are reported SE, which approximates the 70% confidence limits. All statistical analyses were performed with SPSS software (SPSS, Inc, Chicago, Ill).

Results

Eleven of the 16 patients were male, and 5 were female. Average age was 57 ± 19 years (range 16–88 years), and hypertension was present in 15 of 16 patients. Demographic data are summarized in Table 1. Mean follow-up interval was 36 ± 36 months, ranged from 3 to 93 months, and was 100% complete. Eight patients remained at risk at 3 years, and 6 remained at risk at 5 years. A total of 48 patient-years of follow-up was available for analysis.

Early Outcome

Aortic access was achieved in all patients, all devices were deployed in the intended position, and the main PIT was covered in each patient. In 2 patients, a second stent–graft was placed proximally to eliminate a type I endoleak. No immediate open surgical conversions were necessary. Stent–graft characteristics and aortic dimensions are listed in Table 2. Early mortality was 25% ± 11%; all 4 patients died within 6 days of stent–grafting. Two patients almost certainly died of aortic rupture (sudden death consistent with exsanguination without autopsy). Two patients died of fulminant multiorgan failure, 1 each after extensive bowel resection and amputation of a gangrenous leg.

Early Morbidity

Four patients required concomitant true lumen stenting of aortic branches because of static occlusion. This treatment included renal, mesenteric, or iliac artery stenting, as detailed in Table 3. Early false-lumen thrombosis was documented in 2 cases, and the false lumen thrombosed subsequently in 2 additional cases. No new stroke or paraplegia occurred, but preprocedural paraplegia in 1 patient did not improve. One patient had a temporary paraparesis, which resolved within 20 days. After rehabilitation, at the time of follow-up, this patient could walk with a cane. Four patients (25%) required surgical arterial repair, including iliofemoral bypass in 1 case. Pulmonary complications occurred in 2 patients. Renal insufficiency (defined as serum creatinine >3.5 mg/dL) occurred in 8 patients (50%), often after severe preprocedural renal ischemia; 3 of these patients required temporary hemodialysis.

Late Outcome

Follow-up was 100% complete, and it was encouraging that there were no late deaths. Actuarial survival was 73% ± 11% at 1, 3, and 5 years (Figure 1); furthermore, no stent migration, dislocation, or erosion was documented. One patient with a retrograde A dissection in which the false lumen in

TABLE 3. Summary of malperfused arteries and late status of aortic false lumen

Patient	Malperfused branch arteries	Stenting of branch arteries	Thrombosis of false lumen*
1	0	0	Complete
2	Left renal and right common iliac	Wall stent, left renal and right common iliac	Complete
3	Supramesenteric	Wall stent, supramesenteric	Partial
4	Right renal	0	Patent
5	Both common iliac and supramesenteric	Wall stent, supramesenteric and left and right common iliac	died
6	Left renal and right femoral	0	died
7	0	0	Partial
8	Celiac mesenteric, left renal, and right iliac	Palmaz stent, right iliac	Patent
9	0	0	Partial
10	Left femoral	0	Complete
11	0	Died	died
12	0	0	Complete
13	0	0	Partial
14	Supramesenteric, both common iliac, and both renal	Infrarenal thrombectomy	died
15	0	0	Partial
16	0	0	Partial

*According to last computed tomographic scan.

the ascending aorta had thrombosed required repeated thoracic aortic stent–grafting 3 months later because of a proximal type I endoleak with enlargement of the false lumen. Balloon dilatation of the extension stent–graft tore the distal dissection flap, which created a large communication between the true and false lumens. After this patient underwent open surgical graft replacement of his distal arch and proximal descending thoracic aorta, he recovered uneventfully.

Endoleaks

According to the most recent CTA scan, 1 patient had a persistent type I endoleak (Figure 2); absence of any demonstrable expansion of the false lumen permitted continued expectant observation for this patient. An increase in aortic diameter (Figure 3) was documented in 1 case (patient 9). Actuarial freedom from treatment failure (broadly defined as aortic rupture, migration or failure of the mechanical device, any necessity for reintervention, late aortic-related death, or any sudden, unexplained late death) was 67% ± 14% at 1, 3, and 5 years (Figure 4).

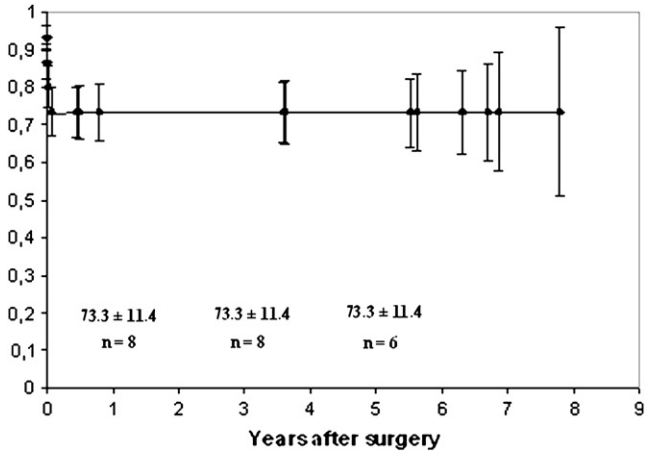

Figure 1. Kaplan–Meier actuarial patient survival estimates. Five-year survival was 76% ± 9%. Nine patients remained alive and at risk 5 years after the procedure.

Discussion

Patients with life-threatening complications of acute type B aortic dissection remain a high-risk subset.[13,17,18] Open surgical mortality risk is high compared to emergency endovascular stent–grafting, as we first reported in 1999.[6] Even with endovascular treatment,[19,20] mortality risk can still be high (25% in this small series), with some deaths due to irreversible infarction of distal end organs. Our stent–graft strategy is

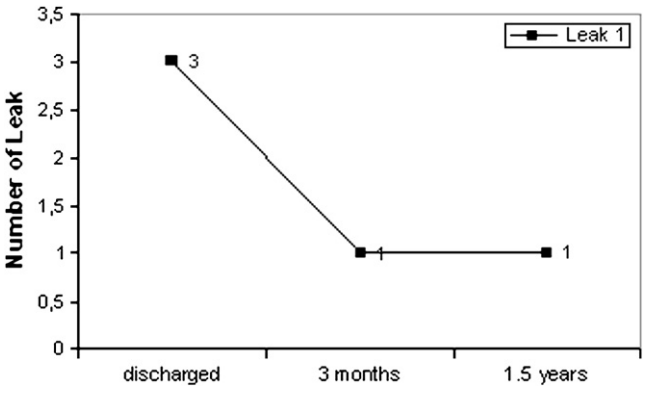

Figure 2. Evolution with time of type I and type II endoleaks. Most endoleaks spontaneously resolved or were manageable by endovascular means, but 1 patient required surgical graft replacement 3 months later for a proximal type 1 endoleak.

		Aortic diameter			Total
		Smaller	Unchanged	Larger	
False lumen status (last CTA)	Complete thrombosis	1	3	0	4
	Partial thrombosis	2	3	1	6
	Patent	0	6	0	6
Total		3	12	1	16

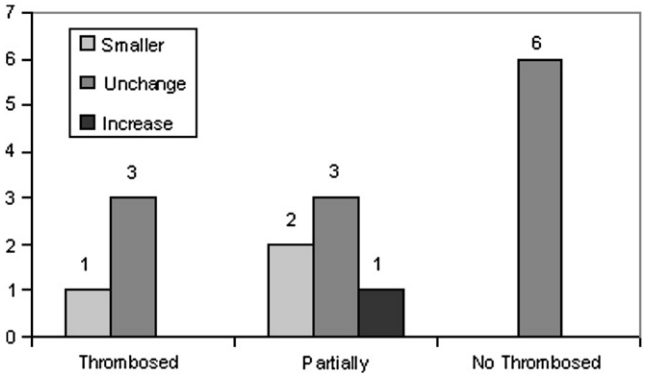

Figure 3. Comparison between thrombosed false lumen and change in aortic diameter. There was no relationship between thrombosed false lumen and increasing aortic diameter.

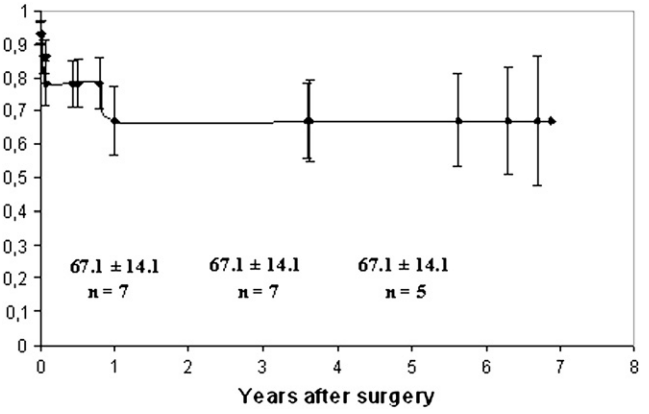

Figure 4. Actuarial freedom from treatment failure (including aortic rupture, device mechanical fault, reintervention, late aortic-related death, or sudden, unexplained late death).

based on the notion that early reversal of the most severe acute malperfusion complications will allow the patient to be resuscitated and clinically stabilized,,[21-23] after which more definitive treatment, possibly including open surgical repair, can be accomplished with lower cumulative risk. Our therapeutic goals for patients with acute dissections are to reperfuse the lower body and prevent rupture, not necessarily to eliminate all flow into the false lumen.

It is difficult to know with certainty the results of endovascular stent–graft repair in patients with complicated acute type B aortic dissections for a host of reasons. Most reports contain both complicated and uncomplicated cases, as well as mixed cases of acute and chronic dissections. Furthermore, most physicians in Europe and South America consider stent–grafting too risky until at least 2 weeks have passed since presentation; such patients with subacute dissections constitute a naturally selected subset. To define complicated acute type B dissection,[6,24,25] we considered the threat of continued aortic expansion or rupture and the extent and severity of distal end-organ ischemia. Indicators of high risk included ongoing chest or abdominal pain despite optimal medical therapy, rapid dilation of the aortic false lumen, increasing pleural effusion accompanied by pain, radiographic signs of rupture or impending rupture, evolution of clinical symptoms, and laboratory markers associated with gut ischemia accompanied by signs of compromised visceral perfusion on CTA scan. It is generally accepted that these criteria define a high-risk subgroup of acute type B aortic dissection that requires emergency treatment. Risk of rupture is usually obvious from progressive radiologic signs, clinical instability, and inability to control the pain. Intestinal ischemia, however, may progress more insidiously, with results of abdominal examination potentially remaining unremarkable until visceral infarction has become established. At this stage, gut salvage may not be possible, even after successful abdominal aortic reperfusion. Despite early stent–graft deployment, restoration of adequate branch artery true-lumen perfusion with bare stents, and prompt surgical bowel resection when necessary, death was not preventable in cases of irreversible infarction of two or more vascular beds. At an earlier stage, when reperfusion can still salvage important end organs, patients with malperfusion and evolving visceral ischemia could be identified. A third scenario exists where there is severe malperfusion, usually involving the kidneys, without clinical symptoms. Loss of adequate perfusion to one kidney, despite satisfactory urine output from the contralateral functioning kidney, can eventually result in loss of a functioning kidney.[17]

In general, three treatment strategies are available. Direct open surgical aortic graft replacement through a left thoracotomy can redirect blood flow into the true lumen and remove the aortic segment with the most trauma. This, however, has historically been associated with high operative risk,[13] is invasive, and requires cardiopulmonary bypass and profound hypothermia for an open proximal anastomosis in the arch.[10,11] Further, immediate adequacy of distal reperfusion is not easily assessable,[18] which may necessitate additional downstream catheter interventional treatment. Endovascular stent–graft repair similarly redirects blood flow into the true lumen, and immediate assessment of the adequacy of distal perfusion is obtained with aortography. Nonreperfused organs cut off by static branch obstruction can then be quickly reperfused with catheter-based dissection flap fenestration or branch true-lumen stenting.[6,24] Finally, balloon fenestration of the dissection flap and stenting from the aortic true or false lumen into the true lumen of the ischemic branch

vessel is a third alternative, one that was used by our group before the availability of thoracic aortic stent–grafts. All these techniques may be effective in the short term, and each is associated with different degrees of invasiveness, effectiveness, and attendant morbidity. Open surgical aortic repair remains the treatment of choice for patients with major arch involvement, those in whom the PIT is too proximal to allow stent–grafting, and those with Marfan syndrome or other connective tissue disorders.

Limitations of thoracic aortic stent–grafting clearly exist. Multiple reentry fenestrations are usually present in the distal descending thoracic and abdominal aorta, allowing continued perfusion of the false lumen. Although flow through these communications may prevent complete thrombosis of the aortic false lumen, these fenestrations are usually smaller than the PIT and neither cause aortic true-lumen obstruction nor lead to acute rupture of the false lumen. Follow-up studies from Mei University in Japan have shown that late serious aortic complications occur after stent–grafting for aortic dissection, for example, delayed intimal tears caused by the stent–graft or migration of the stent–graft away from the aortic wall along the lesser curve of the distal arch.[25,26] Careful, continuous follow-up is essential indefinitely to ensure stability of the stent–graft, persistent patency of important aortic tributaries, and the aortic true- and false-lumen dimensions. The 25% incidence of complete false-lumen thrombosis observed in our experience was higher than that reported after surgical repair of patients with acute type A dissections, but a comparison of the likelihood of false-lumen thrombosis or enlargement is handicapped by the small sample size in this report. Again, our therapeutic goal for patients with acute type B dissections is not so much to eliminate all blood flow in the false lumen as to reperfuse the lower body quickly and prevent thoracic aortic rupture. A persistent proximal type I endoleak, which does not necessarily preclude early success because reperfusion of severely ischemic abdominal end organs is one of the key therapeutic goals, may result in late failure from progressive false-lumen expansion. Type II endoleaks, which may be confused with natural flap reentry fenestrations, however, do not connote failure and usually can be followed up observationally unless they result in progressive false-lumen aneurysmal dilation. In this experience, aortic diameter in the absence of an endoleak remained stable at midterm follow-up in most cases. Additional follow-up of a larger number of patients, however, is clearly mandatory to elucidate the long-term results of stent–grafting in patients with complicated acute type B dissections.

Although stent–graft repair is an attractive alternative because it is less invasive, angiography requires the use of iodinated contrast, which can exacerbate preexisting ischemic renal injury. Technologic limitations also exist. Half of the deaths in this series were due to aortic rupture and represent treatment failure. The acutely dissected aorta and flap are exceptionally fragile and thin, calling for special, disease-specific endograft designs that currently are lacking. The ideal stent–graft for acute dissection should be able to be fixed securely in the distal arch without hooks, must be soft and flexible to allow maneuvering within the arch without creation of a retrograde type A dissection, and must conform readily to the sharp curvature of the distal arch to avoid proximal endoleaks. The ideal stent–graft for acute dissections should also have sufficient radial strength to expand the true lumen, thus promoting false-lumen thrombosis without further intimal flap injury. Advances in stent–graft technology and newer disease-specific stent–grafts should translate into better results in the future. Finally, avoiding coverage of the distal half of the descending thoracic aorta is important in minimizing the likelihood of paraplegia.

In summary, emergency endovascular thoracic aortic stent–graft repair for patients with complicated acute type B dissections saves lives and is effective to 5 years. Promising observations in this report with extended follow-up include the absence of late deaths and the reasonable stability of the stent–graft. Stent–grafting rapidly reverses life-threatening malperfusion in cases of true-lumen collapse and probably minimizes the risk of rupture as well. Because of the high-risk nature of this patient population, it is unrealistic to expect that the mortality risk will approach zero; if the life-threatening consequences of acute distal malperfusion can be reversed early enough and the patient stabilized, more patients will survive the early high risk phase. Then the patient can be followed up clinically and the dissected aorta monitored carefully with serial scans. If false-lumen enlargement occurs, delayed open surgical repair can be performed in elective circumstances with a reasonably low risk. Finally, the long-term (5–10 years) effectiveness and durability of thoracic stent–grafting for acute type B aortic dissection will remain unknown until the results of larger studies with long-term follow-up are available.

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