

Results of a New Surgical Paradigm: Endovascular Repair for Acute Complicated Type B Aortic Dissection

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Background. Conventional open repair of acute complicated type B aortic dissection is associated with significant morbidity and mortality. This study examined the results of thoracic endovascular aortic repair (TEVAR) in acute type B aortic dissection complicated with rupture or malperfusion syndrome.

Methods. From 2004 through 2007, 35 patients (22 men) with acute complicated type B aortic dissection were treated with TEVAR. Indications included rupture in 18 (51.4%) and malperfusion syndrome in 17 (48.6%; mesenteric or renal, 5; lower extremities, 3; both, 9). Three types of endograft devices were used (mean per patient, 1.9 devices). Intravascular ultrasound imaging was used in 15 patients (42.8%). In patients with malperfusion syndrome, distal adjunct procedures to expand the true lumen included infrarenal aortic stents in 4, mesenteric/renal stents in 4, and iliofemoral stents in 7. Follow-up

was 93.9% during a period of 18.3 months (range, 3 to 47 months).

Results. The mean age was 58.6 ± 13.4 years. Technical success (coverage of the primary tear site) was achieved in 34 patients (97.1%). Coverage of the left subclavian artery was required in 25 patients (71.4%). Thirty-day mortality was 2.8%. One-year survival was $93.4\% \pm 4.6\%$. Complications included permanent renal failure (2.8%), stroke (2.8%), spinal cord ischemia (transient [5.7%], permanent [(2.8%)]), and vascular access (14.2%). The mean intensive care unit and hospital stay were 4.7 ± 2.6 and 16.7 ± 12.0 days, respectively.

Conclusions. Endovascular repair of acute complicated type B aortic dissection is associated with low morbidity and mortality and has emerged as the surgical therapy of choice.

(Ann Thorac Surg 2008;86:87-94)

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The role of thoracic endovascular aortic repair (TEVAR) in the management of acute type B aortic dissection remains to be debated [1]. For patients who present with acute uncomplicated type B aortic dissection, in-hospital survival approaches 90% with medical therapy alone [2]. The role of TEVAR in this group of patients remains controversial.

Patients with acute type B aortic dissections who present with life-threatening complications, including rupture or malperfusion syndrome, remain a challenging group to manage. Historically, conventional open surgical therapy in this group of patients has been associated with significant morbidity and mortality, ranging from 30% to 50% [3-5]. Despite improvement in surgical technique, in-hospital mortality remains significant. In the

most recent International Registry of Acute Aortic Dissection (IRAD) review, in-hospital mortality in patients undergoing surgical repair of type B aortic dissection was 29.3%. For patients presenting with malperfusion and rupture, the in-hospital mortality were 27.8% and 62.5%, respectively [5].

An alternative surgical option for this group of patients remains desirable. Although first introduced for the treatment of thoracic aortic aneurysms, the application of TEVAR has been extended to other acute thoracic aortic syndromes, including aortic dissection. Multiple studies have examined the role of TEVAR in acute type B aortic dissection. However, the studies often examined a heterogeneous population of patients including both complicated and uncomplicated aortic dissection as well as variations on the timing of intervention (immediate vs

Accepted for publication April 1, 2008.

Presented at the Forty-fourth Annual Meeting of The Society of Thoracic Surgeons, Fort Lauderdale, FL, Jan 28-30, 2008.

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Dr Bavaria discloses that he has a financial relationship with W. L. Gore Inc, Cook Inc, and Medtronic Vascular; Dr Fairman with Medtronic Vascular and Cook Inc; and Dr Carpenter with W. L. Gore Inc, and Cook Inc.

Table 1. Preoperative Characteristics in the 35 Patients

Variables	No (%) or Mean ± SD
Age, years	58.6 ± 13.4
Sex	
Male	22 (62.8)
Female	13 (37.2)
Hypertension	31 (88.6)
Previous CVA	5 (14.3)
PVD	7 (20.0)
Renal failure ^a	8 (22.9)
COPD	14 (40.0)
Previous CV operation	5 (14.3)
AVR	1
AAA repair	3
TAAD repair	1

^a Serum creatinine > 2.0 mg/dL.

AAA = abdominal aortic aneurysm; AVR = aortic valve replacement; COPD = chronic obstructive pulmonary disease; CV = cardiovascular; CVA = cerebrovascular accident; PVD = peripheral vascular disease; TAAD = type A aortic dissection.

delayed). Technical success has been consistently high, ranging from 86% to 100% [6, 7]. The 30-day mortality has also been encouraging, with reports ranging from 3.2 to 20% [7-12].

Our results suggest that TEVAR is an effective surgical alternative and support a new surgical paradigm for the treatment of acute complicated type B aortic dissection.

Table 2. Operative Strategy

Variable	No (%) or Mean (range)
Stent graft devices	
Gore TAG	31 (88.6)
Cook Zenith ELSE	3 (8.6)
Medtronic Talent	1 (2.8)
Indications for TEVAR	
Rupture	18 (51.4)
Malperfusion	17 (48.6)
Visceral (celiac, mesenteric, or renal)	5 (29.5)
Iliofemoral (lower extremity)	3 (17.6)
Both	9 (52.9)
Access	
Femoral artery (groin)	31 (88.6)
Iliac artery (retroperitoneal)	2 (5.7)
Axillary (infraclavicular)	2 (5.7)
IVUS	15 (42.9)
Stent graft devices deployed	1.9 (1-3)
Coverage of left subclavian artery	25 (71.4)
Distal adjunct procedures (BMS)	
Infrarenal aorta	4
Renal/celiac artery	4
Iliofemoral artery	7

BMS = bare metal stents; IVUS = intravascular ultrasound; TEVAR = thoracic endovascular aortic repair.

We report our experience with emergency TEVAR in this difficult subset of patients who present with life-threatening complications, including malperfusion or rupture.

Material and Methods

Patients

From February 2004 through October 2007, 35 patients with acute complicated type B aortic dissection were treated with endovascular repair. The mean age was 58.6 ± 13.4 years. There were 22 men (62.8%) and 13 women (37.2%). Five patients had previous cardiovascular operations. Preoperative patient characteristics are listed in Table 1.

Acute type B aortic dissection was defined as any nontraumatic dissection that involved the descending thoracic aorta with an entry tear distal to the origin of the left subclavian artery and presentation within 14 days of symptoms onset [13, 14]. Diagnosis was based on history and physical examination and confirmed by imaging, including echocardiography, computed tomography (CTA) or magnetic resonance angiography (MRA). The Institutional Review Board of the University of Pennsylvania approved the study and waived the need for patient consent.

Indications for TEVAR

All 35 patients who underwent TEVAR presented with life-threatening complications (Table 2), consisting of rupture in 18 (51.4%) and malperfusion syndrome in 17 (48.6%). Of the 17 patients who presented with malperfusion, end-organ ischemia involved the visceral segment (renal, celiac or mesenteric artery) in 5 patients (29.5%), the iliofemoral vasculature in 3 (17.6%), or both the visceral segment and iliofemoral vasculature in 9 (52.9%).

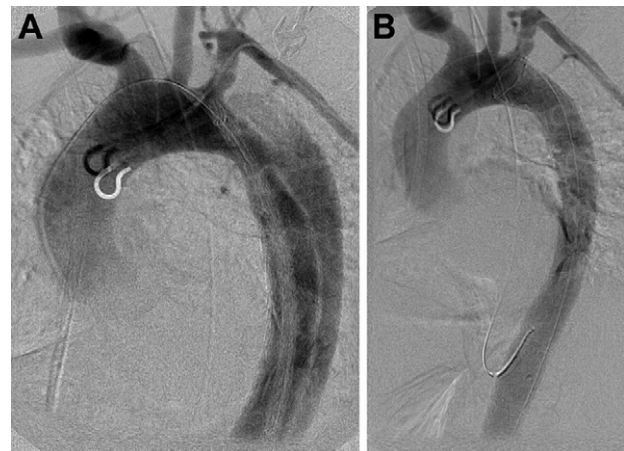


Fig 1. (A) A predeployment aortogram demonstrates acute type B aortic dissection. (B) A postdeployment aortogram demonstrates expansion of the true lumen and obliteration of the false lumen.

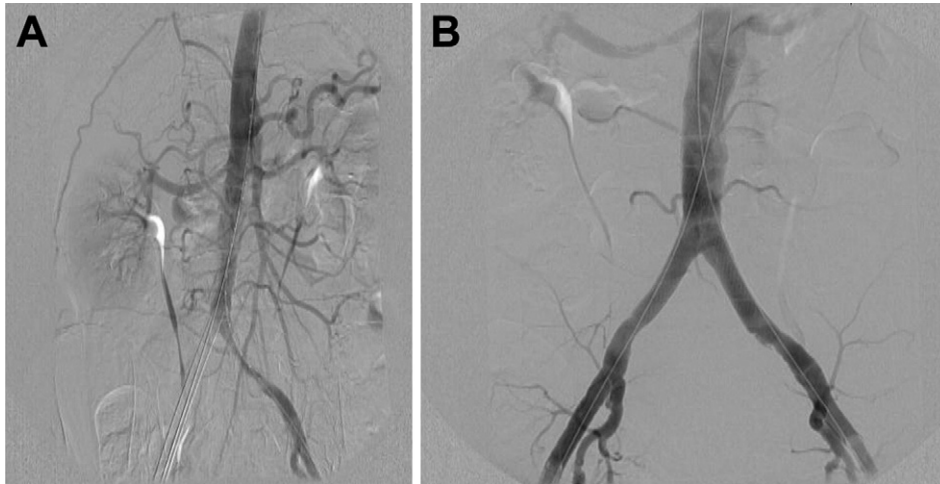


Fig 2. Intraoperative infrarenal aortogram shows (A) malperfusion of the right lower extremity and (B) expansion of the true lumen after deployment of infrarenal aortic and bilateral iliac bare metal stents.

Operative Strategy: The Algorithmic Approach

All patients underwent TEVAR within 14 days of initial presentation. General anesthesia was initiated, and the surgical procedure was performed in the operating room (hybrid endosuite) equipped with the universal floor-mounted angiographic C-arm system (Siemens, Axiom Artis FA, Malvern, PA).

The operating team consisted of cardiovascular surgeons, anesthesiologists, and neurologists. When available, neuromonitoring with continuous electroencephalogram (EEG) and somatosensory evoked potential (SSEP) was used to evaluate and detect neurologic events, including stroke or spinal cord ischemia, throughout the operation according to standard protocol for thoracic aortic procedures [15-17].

Endovascular treatment of acute complicated type B aortic dissection requires an algorithmic approach. The fundamental principles of endovascular treatment of aortic dissection have major conceptual differences from aneurysmal pathology. The goals of therapy are to (1) cover the primary tear site, (2) expand the true lumen with obliteration of the false lumen, (3) and restore adequate flow and perfusion in the distal aorta.

Wire Access in the True Lumen

In contrast to aneurysmal pathology, wire access in the true lumen may be difficult to achieve in aortic dissections. If true lumen access is uncertain, confirmation must be obtained with the aid of intravascular ultrasound (IVUS) imaging before stent graft deployment. The choice of peripheral access site may be determined preoperatively with the aid of CTA or MRA. The brachial or axillary artery may be needed for access to the true lumen if significant compromise to the true lumen is present in the distal aorta or iliofemoral vasculature.

Coverage of Primary Tear Site

The primary tear site must be identified and covered by the stent graft device to achieve technical success. Coverage of the primary tear site initiates the expansion of the compressed true lumen, resulting in thrombosis and

obliteration of the false lumen. Because most tear sites are at the level of the left subclavian artery, coverage of its origin may be required (Fig 1).

Malperfusion Syndrome Versus Rupture

For acute type B aortic dissection complicated by malperfusion syndrome, coverage of the primary tear site alone with a proximal stent graft device may be adequate to expand the true lumen, obliterate the false lumen, and correct the malperfusion syndrome (because there is no site of rupture). After deployment, reestablishment of distal perfusion is assessed. If distal malperfusion persists, as evidenced by mesenteric ischemia or renal failure, extension with additional stent graft device in the distal thoracic aorta should be performed.

Patients who present with lower extremities malperfusion often will not be successfully treated with endovascular therapy in the thoracic aorta alone and may require adjunct infrarenal strategies. In patients with persistent infrarenal and lower extremity malperfusion, bare metal stents in the infrarenal aorta and iliofemoral arteries may be required to expand the true lumen and alleviate the malperfusion syndrome. Because the goal is expansion of the true lumen, covered stent graft devices are not necessarily required in the distal aorta (Fig 2).

For patients who present with rupture, coverage of the rupture site in addition to the primary tear site must be achieved. Because of the extent of the dissection, often the coverage of the entire thoracic aorta from left subclavian artery to the celiac artery is required.

Data Collection and Follow-Up

Hospital and outpatient clinical charts were retrospectively reviewed for patient characteristics, preoperative comorbidities, indications, intraoperative events, and postoperative course. Follow-up data were obtained by clinic visits, retrospective chart review, and the Thoracic Aortic Surgery clinical database at the University of Pennsylvania.

Statistical Analysis

Statistical analysis was performed using SPSS Base 12.0 software (SPSS Inc, Chicago, IL). Continuous variables were expressed as the mean \pm standard deviation (SD). Survival was analyzed with the Kaplan-Meier, and 1-year survival was expressed as a percentage \pm SD.

Results

Three different stent graft devices were used. The TAG thoracic endoprosthesis (W. L. Gore Inc, Flagstaff, AZ) was used in 31 patients (88.6%). The Talent thoracic endoprosthesis (Medtronic Vascular, Santa Clara, CA) was used in 1 (2.8%), and the Zenith ELSE (Cook Inc, Bloomington, IN) was used in 3 (8.6%). The mean number of devices used per patient was 1.9 devices (range, 1 to 3; Table 2).

Access for the TEVAR device included the common femoral artery through a groin incision in 31 patients (88.6%), the iliac artery through a retroperitoneal incision in 2 (5.7%), or the axillary artery through an infraclavicular incision in 2 (5.7%). Access for the diagnostic angiogram (a pigtail catheter through percutaneous technique) included the contralateral femoral artery in 24 (68.6%) or brachial artery in 11 (31.4%). Intraoperative IVUS was used to confirm wire access in the true lumen in 15 (42.9%) patients (Table 2).

Technical success as defined by coverage of the primary tear site was achieved in 34 patients (97.1%). No patients were converted to open repair. Adjunct procedures were performed in 12 patients (34.3%) and included distal extension with bare metal stents in the infrarenal aorta in 4, renal artery in 3, celiac artery in 1, and iliofemoral vasculature in 7. Three patients with significant distal malperfusion required both infrarenal and bilateral iliac stents. In 1 patient, planned coverage of the left subclavian artery resulted in left arm ischemia requiring a left carotid-subclavian artery bypass on postoperative day 6.

Morbidity and Mortality

Overall, 2 patients in the series died (Table 3). The 30-day mortality was 2.8% ($n = 1$), and the 1-year survival was 93.4% \pm 4.6%. The first death occurred in a patient who presented with an acute type B dissection complicated by a contained rupture. Failure to cover the primary tear and the site of rupture was not recognized, and a free rupture was the cause of death on postoperative day 3. The second patient, who died on postoperative day 63, had presented with evidence of visceral malperfusion of the celiac and superior mesenteric artery (SMA). The cause of death was not aortic-related. After TEVAR, the patient had multiple readmissions owing to occult malignancy, most likely metastatic pancreatic cancer.

The mean lengths of stay were 4.7 \pm 2.6 days in the intensive care unit and 16.7 \pm 12.0 days in the hospital. Complications related to vascular access occurred in 5 patients (14.2%). Repair included femoral thrombectomy in 1, axillary artery stent in 1 common iliac artery stents in

Table 3. Hospital Morbidity and Mortality

Variable	No (%) or Mean \pm SD
Morbidity	
Cerebrovascular accident	1 (2.8)
Spinal cord ischemia	
Transient (full recovery)	2 (5.7)
Permanent	1 (2.8)
Renal failure ^a	1 (2.8)
Vascular access	5 (14.2)
ICU stay, days	4.7 \pm 2.6
Hospital stay, days	16.7 \pm 12.0
Mortality	
30-day/in-hospital	1 (2.8)
1-year survival, %	93.4 \pm 4.6

^a Permanent hemodialysis.

CVA = cerebral vascular accident; ICU = intensive care unit.

2, and femoral-femoral artery bypass in 1. No wound infections or seroma developed during follow-up.

Of the 17 patients with malperfusion, 9 presented with either isolated acute renal failure or concomitant with mesenteric or lower extremity malperfusion. The preoperative serum creatinine level exceeded 2.0 mg/dL in 8 of the 9 patients. Six patients required transient hemodialysis during the hospitalization, but only 1 patient required hemodialysis on discharge from the hospital. She had presented with 12 hours of oliguria and a serum creatinine level of 4.8 mg/dL. Despite TEVAR and an adjunct right renal bare metal stent, her renal function did not recover.

Twelve patients presented with either isolated mesenteric malperfusion or concomitant with renal or lower extremities malperfusion. Evidence of mesenteric malperfusion was demonstrated by abdominal pain, distention, or ileus. One patient required an adjunct celiac artery stent. Two patients required exploratory laparotomy after TEVAR for continuing symptoms of abdominal pain and ileus. On exploration, neither patient had evidence of ischemic bowel, and no bowel resection was performed. For patients with lower extremities malperfusion requiring adjunct distal procedures, 4 infrarenal aortic bare metal stents and 7 iliofemoral stents were deployed. Lower extremity fasciotomies were required in 2 patients, but no amputation was required.

Neurologic complications occurred in 4 patients and included stroke in 1 (2.8%) and spinal cord ischemia in 3 (8.5%). For the patient who sustained a postoperative stroke, the neurologic deficit was permanent and included right sided weakness in both upper and lower extremities. A CT scan of the head demonstrated a basal ganglia infarct. Spinal cord ischemia occurred in 3 patients postoperatively and was managed according to our protocol [15, 18]. Spinal cord ischemia was transient in 2 patients. Full neurologic recovery was achieved with hypertensive therapy and volume expansion alone in the first patient, and a postoperative lumbar drain was required in the second patient. Permanent paraplegia

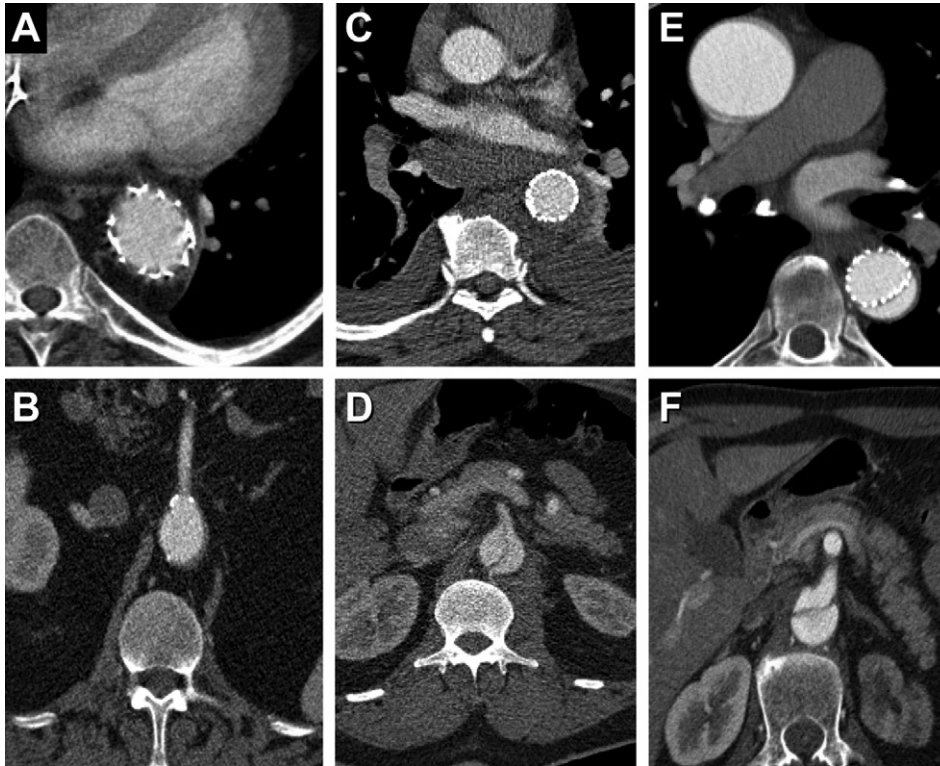


Fig 3. Computed tomography angiography on most recent follow-up shows (A-B) complete thrombosis of the false lumen in both thoracic and abdominal aorta, (C-D) complete thrombosis of the false lumen in the thoracic aorta with patent false lumen in the abdominal aorta, and (E-F) patent false lumen in both thoracic and abdominal aorta.

occurred in the third patient despite aggressive spinal cord rescue protocol with a postoperative lumbar drain.

Patient Follow-Up

Follow up was 93.9% (31 of 33 patients) during a 13-month period (range, 3 to 47 months). Three patients required aortic interventions during follow-up. One patient underwent an elective reimplantation valve-sparing aortic root replacement (David V with hemiarch reconstruction) for a sinus of Valsalva aneurysm at 6 months after TEVAR. The second patient required coil embolization of the left subclavian artery for persistent type II endoleak at 4 months after TEVAR. The third patient required emergency repair for an acute type A aortic dissection at 1 month after TEVAR.

Postoperative surveillance imaging with either CTA or MRA was obtained in 27 of 31 patients. The status of the false lumen in the most recent CTA was evaluated in the entire thoracoabdominal aorta. Complete thrombosis of the false lumen in the descending thoracic aorta was achieved in 20 of 27 patients (74.1%). Of these, 11 had persistent patent false lumen in the abdominal aorta, and the remaining 7 exhibited a persistent patent false lumen in both the descending thoracic and abdominal aorta (Fig 3).

Comment

The management of acute complicated type B aortic dissection remains a clinical challenge. Our experience supports that TEVAR is an effective surgical alternative to

conventional open repair. In our series, the rates for renal failure (2.8%), cerebrovascular accident (2.8%), permanent spinal cord ischemia (2.8%), vascular access complications (14.2%), and 30-day mortality (2.8%) compare favorably with conventional open repair. The recent IRAD database demonstrated that conventional open repair for acute type B aortic dissection in the current era is still associated with a significant risk of cerebrovascular accident (9.0%), paraplegia (4.5%), visceral ischemia/infarction (6.8%), and acute renal failure (18.3%), all of which were correlated with postoperative death. The overall in-hospital mortality was 29.3%, and for patients whose procedures were within 48 hours, the in-hospital mortality was 39.2% [5]. The dramatic difference in morbidity and mortality has led to a new surgical paradigm at our institution, and TEVAR has emerged as the surgical therapy of choice for the management of acute complicated type B aortic dissection.

Endovascular therapy for acute aortic dissection is technically demanding. Some have argued that the current technology is not ideal and that devices designed specifically for dissection are needed [19, 20]. Nonetheless, we emphasize that the complexity involved with endovascular therapy for aortic dissection requires an algorithmic approach that must begin at the primary tear site. The level of complexity is further demonstrated by the requirement of distal adjunct procedures in 12 of the 35 patients (34.2%) in our series.

Wire access in the true lumen cannot be over emphasized, because deployment of thoracic stent graft devices in the false lumen will have catastrophic consequences.

When confirmation of wire access in the true lumen is needed, IVUS has proven to be a valuable tool.

The fundamental principles of endovascular treatment of aortic dissection have major conceptual differences from aneurysmal pathology. The primary goal is coverage of the primary tear site, thus expanding the true lumen and initiating thrombosis and obliteration of the false lumen. Often, the tear site is close to the left subclavian artery and coverage is necessary. Despite successful thoracic stent graft therapy, persistent patency of the false lumen may occur due to complex reentry points in the distal thoracoabdominal aorta. Particularly in cases with malperfusion syndrome (since the goal of therapy is to restore distal perfusion and correct end-organ ischemia), persistent false lumen patency may continue to compromise the true lumen, thereby resulting in continued end organ ischemia.

Called the PETTICOAT (Provisional Extension to Induce Complete Attachment) concept [21], the principle refers to an algorithmic evaluation and treatment of the thoracoabdominal aorta in type B aortic dissection. After coverage of the primary tear site, the status of the true lumen is assessed. If persistent malperfusion is present, an additional distal device is deployed. This evaluation and treatment algorithm is repeated with each adjunct device until distal malperfusion is corrected. In patients with persistent visceral malperfusion despite coverage of the primary tear site, TEVAR in the distal thoracic aorta with adjunct celiac, SMA, or renal bare metal stents should be considered. Subsequently, persistent lower extremity malperfusion may require infrarenal aortic and iliofemoral adjunct procedures including bare metal stents.

For dissections complicated by rupture, coverage of the primary tear site is equally essential; however, the site of rupture must also be addressed with TEVAR. Because of the extent of the dissection and the potential for perfusion of the false lumen through distal complex reentry sites, often the coverage of the entire thoracic aorta from left subclavian artery to the celiac artery is required. Failure to recognize this principle most likely contributed to the one early death in our series. This patient had presented with an acute type B aortic dissection complicated by a contained rupture. With coverage of the left subclavian artery, the aortic dissection was treated with TEVAR to the middle descending thoracic aorta at the level of the pulmonary veins. On postoperative day 3, the patient became hemodynamically unstable and died of a free rupture into the left hemithorax.

Other series examining the role of TEVAR in acute complicated type B aortic dissection have also been encouraging, with similar results. Nienaber and coworkers [11] reported 11 patients undergoing TEVAR for acute type B aortic dissections complicated by contained ruptures. There was no morbidity or stent graft-related complications, and no operative deaths. Chen and coworkers [9] reported 62 patients undergoing TEVAR for acute type B aortic dissection. In the 23 patients who underwent emergency TEVAR (at presentation or within

2 weeks), the technical success was 100% and the 30-mortality was 4.4% (1 or 23) [9].

Eggebrecht [7] recently performed a meta-analysis of 39 series involving TEVAR for all type B aortic dissections. For patients with acute dissection (no distinction between complicated vs uncomplicated), the 30-day mortality was 9.8%. In a recent IRAD review of 384 patients with acute type B aortic dissection, the 30-day mortality was 6.5% in patients undergoing percutaneous intervention, including stents and fenestration, compared with 32.1% for conventional surgery [22].

Although our and others' experiences and others have demonstrated favorable mortality rates for emergency TEVAR in acute complicated type B aortic dissection, serious complications have not been insignificant. Therefore, we caution and have reserved this therapy for patients with definitive evidence of life-threatening complications such as rupture or malperfusion. For patients presenting with pain or difficult to control hypertension, our approach has continued to involve aggressive medical management with antiimpulse therapy. As in aneurysmal pathology, spinal cord ischemia is a devastating complication that remains a risk. In our series, the incidence of spinal cord ischemia (permanent and transient) was 8.5%.

Vascular access in our series was 14.2%, similar to other published reports [7]. Neuhauser and coworkers [23] recently reported 28 patients undergoing TEVAR for acute symptomatic type B aortic dissection. Indications included rupture, impending rupture, visceral or peripheral ischemia (or both), uncontrollable hypertension, and therapy-resistant pain. Secondary intervention was required in 5 patients on follow-up. Conversion to open procedures for retrograde type A dissection was required in 4 patients. Endovascular repair of a type III endoleak was required in 1 patient. Procedure-related mortality after secondary complications was 20%. In our series, 3 patients required additional aortic interventions during the follow-up period. One required emergency open repair 1 month postoperatively for a retrograde type A aortic dissection. The second patient required coil embolization of the left subclavian artery for persistent type II endoleak. In the third patient, an elective valve sparing aortic root replacement was performed for a sinus of Valsalva aneurysm.

The long-term implication regarding the status of the distal false lumen in aortic remodeling after TEVAR remains unanswered. In our series, coverage of the primary tear site was achieved in 97.1% of patients. Although thrombosis of the false lumen in the thoracic aorta was achieved in 20 of 27 patients (74.1%), 11 of these 20 patients continued to demonstrate persistent patent false lumen in the abdominal aorta. Furthermore, 7 of 27 patients demonstrated persistent patent false lumen in both the thoracic and abdominal aorta. Partial thrombosis of the false lumen has been demonstrated to be a significant independent predictor of postdischarge mortality in acute type B aortic dissection [24]. Others studies have suggested the continued increase in the diameter of the patent false lumen leaves patients vulnerable to

future complications, including rupture and aneurysmal degeneration [6, 25].

Regardless of choice of therapy, the long-term prognosis and survival for patients with aortic dissection continues to be sobering. A recent IRAD review demonstrated no difference in 3-year survival in patients with acute type B aortic dissections managed medically (77.6%), surgically (82.8%), or with endovascular therapy (76.2%) [2]. The data emphasize the importance of follow-up regardless of the mode of therapy.

In conclusion, our study demonstrates that endovascular repair for acute complicated type B dissection can be performed with a high rate of technical success and low perioperative morbidity and mortality. We believe that TEVAR offers an effective therapeutic alternative for patients with a historically lethal pathology. At our institution, thoracic endovascular aortic repair has emerged as the therapy of choice, representing a new surgical paradigm for acute type B aortic dissection complicated by rupture or malperfusion. The most significant clinical application of TEVAR may well be for patients who present with acute aortic syndromes, including acute type B aortic dissections.

We would like to acknowledge Brenton Moore and Patrick Moeller for their database management.

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DISCUSSION

DR GRAYSON H. WHEATLEY III (Phoenix, AZ): Thank you for an excellent presentation, Dr Szeto, and for sending a copy of

the well-written manuscript to me in advance of this meeting. This is a timely paper and comes at a time that we, along with

vascular surgeons and our interventional colleagues, are trying to better understand the evolving indications of endovascular technologies for the treatment of aortic diseases. It represents the quality and cutting-edge work we have come to expect from the group at the University of Pennsylvania. The results presented today are impressive and represent a definitive step forward in improving patient outcomes for this life-threatening problem. There are several take-away points.

First, the proximal entry tear and aortic rupture site, when present, can be distinct and separate, and addressing both defects is essential. Second, endovascular stent grafts can successfully be utilized in the acutely dissected aorta, which, with its thin septum, fragile aortic wall, and smaller aortic diameter, is a completely different physiologic situation than the aneurysmal aorta.

My first question is, 1 patient developed a stroke and 3 patients developed paraplegia postoperatively, and since the patients in this study, in theory, have a different atherosclerotic burden and potentially a less well-developed spinal cord collateral arcade than aneurysmal patients because of the acute nature of the dissection process, could you comment on whether neuromonitoring is equally or more important in these patients as compared with patients with aneurysmal disease?

My second question is, in the 1 patient that was left with permanent paraplegia, did neuromonitoring identify any evidence of preprocedure spinal cord malperfusion, which might explain this patient's adverse outcome, or do you think it was related to coverage of the left subclavian artery in association with a significant extent of the descending thoracic aorta?

My final question is, 12 patients presented with mesenteric malperfusion and only one patient required an additional intervention with a celiac artery stent, which means that mesenteric perfusion improved significantly after sealing the proximal entry

tear alone. We can sometimes cover the celiac artery for the treatment of aneurysmal disease when there is adequate collateralization from the superior mesenteric artery. How did you decide to stent the celiac artery in this particular patient and what criteria should we use to stent branch vessels?

I would like to thank the program committee for the opportunity to discuss this important paper which I believe will fundamentally change the way we think of and approach complicated acute type B aortic dissections. Thank you.

DR SZETO: Grayson, thank you for those kind comments. We do believe neuromonitoring is important. Often, however, these patients present in extremis and it is not available. If the clinical scenario permits it, we believe neuromonitoring is important, because I think our data demonstrates that spinal cord ischemia is a real issue, even in dissection patients.

The second question is in reference to that one patient with permanent spinal cord ischemia. Interestingly enough, she had a focal dissection fairly low in the distal thoracic aorta. She was a patient that we did not feel was at a high risk for spinal cord ischemia. The repair required one endovascular stent graft device. But in retrospect, what she had was extensive intramural hematoma along the entire thoracic aorta, and that might have been the contributing factor of why she had such an adverse event.

In terms of stenting the celiac artery for malperfusion, it is usually an angiographic determination for us. As you mentioned, in the majority of time, proximal correction of the tear site will resolve mesenteric ischemia. But for this one patient, angiographic evaluation demonstrated no flow in the celiac artery. Our group believes and stresses the importance of an algorithmic evaluation of the entire aorta after endovascular repair.

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