Supplement to

Sponsored by Cook Medical

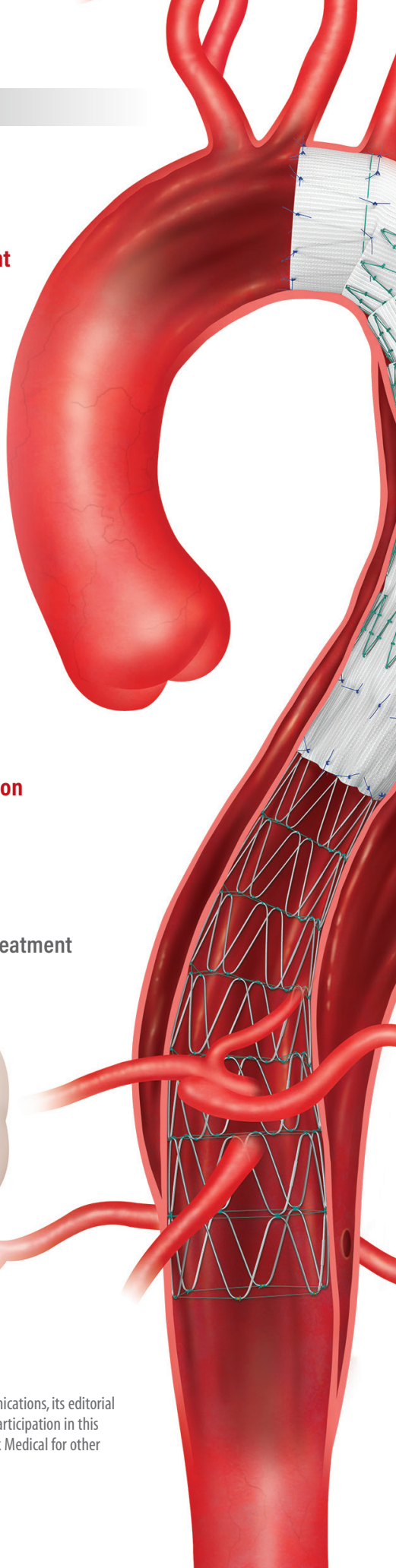
Endovascular TODAY

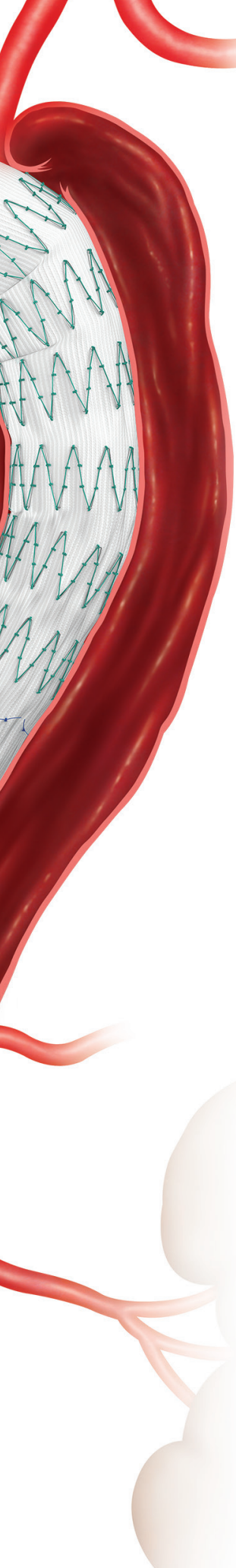
November 2019

DISEASE-SPECIFIC DEVICES

Experts address the challenges of TBAD and share their perspectives on the first FDA-approved endovascular system designed specifically for managing type B aortic dissection.

Contents





Raising the Bar for TBAD Treatment

BY MARK BREEDLOVE

Aortic dissection presents physicians with a complex series of challenges due to its unique pathology, variability in timing of presentation, and extent of disease progression and aortic/branch vessel involvement.

At Cook Medical, we acknowledge the clinical evidence showing that dissection presents differently than aneurysm in terms of symptoms and outcomes—it is more dynamic and its progression is less predictable. We believe that dissection requires its own solutions and approaches to management. As such, we have developed a disease-specific endovascular system, designed to help physicians manage the pathology throughout the course of disease progression and enable a durable endovascular repair of type B aortic dissection (TBAD).

Cook Medical is committed to the needs of patients and the physicians who care for them. In the spirit of collaboration, we asked a group of experienced physicians to present articles that highlight some of the most important and innovative developments in the treatment of aortic dissection. We begin with Athanasios Katsargyris, MD; Pablo Marques de Marino, MD; Balazs Botos, MD; and Eric Verhoeven, MD, PhD, who discuss principles for guiding TBAD treatment decisions and the aim of repair in acute, subacute, and chronic phases, as well as complicated and uncomplicated cases in their article, "Considerations for Short- and Long-Term Goals in TBAD Treatment."

Next, Joseph V. Lombardi, MD, and Qing Zhou, PhD, discuss, "Results from the STABLE Clinical Trials." In "TEVAR Alone Versus the STABLE Technique for Acute Complicated TBAD," Jonathan Sobocinski, MD, PhD; Dominique Fabre, MD; Richard Azzaoui, MD; and Stéphan Haulon, MD, PhD, review data from comparative analyses on these approaches.

We hear again from Joseph V. Lombardi, MD, this time on the "Impact of Proximal Seal Zone in Managing Type B Aortic Dissection." Darren Klass, MBChB, MD, MRCS, FCRC, FRCPC, then provides insight into advanced imaging methods for diagnosis and treatment in his article, "Imaging for TBAD: Essential and Optimal Techniques." In "Controversies in Dissection Repair: Addressing Paraplegia," Keagan Werner-Gibbings, MS, FRACS, and Bijan Modarai, PhD, FRCS, review this devastating complication and the roles of left subclavian artery revascularization, aortic coverage, and cerebrospinal fluid drainage. To round out these expert perspectives, Sukgu Han, MD, MS, and Fernando Fleischman, MD, present "Building a Multidisciplinary Aortic Center" in which they share key components to providing comprehensive care for patients with aortic dissections.

The intent of this *Endovascular Today* supplement is to engage and inform our physician readers and raise the conversation around thoracic endovascular aortic repair to a new level. We acknowledge the progressive nature of aortic disease, and we're working hard to find solutions that create long-term, durable repairs. Cook Medical will always strive to ensure that we show the necessary rigor and discipline to be the responsible partner that physicians expect. We hope this supplement provides a new perspective and even some take-home points that physicians can use in the fight against aortic disease. ■

Thank you,
Mark Breedlove
Vice President, Cook Medical
Global Vascular Division

Sponsored by Cook Medical

Considerations for Short- and Long-Term Goals in TBAD Treatment

The evidence to guide type B aortic dissection decisions and the aims of repair in acute, subacute, and chronic phases, as well as complicated and uncomplicated cases.

BY ATHANASIOS KATSARGYRIS, MD; PABLO MARQUES DE MARINO, MD; BALAZS BOTOS, MD; AND ERIC VERHOEVEN, MD, PHD

Over the last decade, type B aortic dissection (TBAD) has gained increasing interest among vascular surgeons, as well as other cardiovascular specialties. Additional scientific knowledge about TBAD was badly needed to address this often complex pathology. Data increasingly demonstrate that TBAD is neither an easy-to-treat nor a benign disease and may have devastating complications in both the acute and chronic phases.

Global registries have shown suboptimal long-term results for medically treated TBAD patients. Dilatation of the false lumen occurs in 20% to 40% of patients over 5 years. Survival rates range from 86% to 100% at 1 year and can be as low as 59% at 5 years. Freedom from aortic events ranges from 34% to 84%.[1] The classic definitions of complicated and uncomplicated TBAD have been challenged, and some authors suggest that they should both be considered potential vascular complications requiring repair by an effective and durable strategy.[2] Because the disease affects younger patients and many of the deaths during follow-up are aortic-related, the focus is on establishing a treatment that prevents aortic-related complications and mortality in the longer term.

IRAD (International Registry of Aortic Dissection) reported reduced mortality at 5 years in patients with acute TBAD treated by thoracic endovascular aortic repair (TEVAR) compared with those who were managed medically.[3] Two prospective randomized studies have compared best medical treatment (BMT) alone to BMT + TEVAR for TBAD. The ADSORB trial recruited patients with acute uncomplicated TBAD.[4] BMT + TEVAR showed positive aortic remodeling at 1 year compared to BMT alone. The trial, however, was underpowered for mortality at 1-year follow-up.

The INSTEAD trial compared BMT to BMT + TEVAR for patients in stable condition at least 2 weeks after symptom onset (subacute and early chronic phase).[2,5] Initial results failed to show a benefit for BMT + TEVAR regarding 2-year cumulative survival rates but showed favorable aortic remodeling in the BMT + TEVAR group. In the later INSTEAD-XL report that analyzed patients during the time interval 2 to 5 years after the index procedure, it was shown that the risk of all-cause mortality (11.1% vs 19.3%; $P = .13$), aorta-specific mortality (6.9% vs 19.3%; $P = .04$), and progression of dissection (27.0% vs 46.1%; $P = .04$) after 5 years was lower for BMT + TEVAR compared to BMT alone. The authors suggested that in patients with stable type B dissection and suitable anatomy, preemptive TEVAR should be considered in order to improve late outcomes.[2]

INTERVENTIONAL TREATMENT FOR TBAD

TBAD With Acute "Hard" Complications

Rupture, visceral ischemia, and limb ischemia are the feared "hard" complications in acute TBAD. They all require immediate action ("hyperacute" treatment) with damage control aiming for patient survival as a first step. In case of rupture, emergency TEVAR aiming to seal both proximally and distally is the treatment of choice. In case of malperfusion, proximal TEVAR to close the entry tear is the first step. The purpose is to re-expand the true lumen and correct the malperfusion. TEVAR alone may work but, at the same time, may not be enough. It is important to have all available tools on hand to "finish the job." An important asset is the use of a bare stent as a distal extension over the visceral arteries (Zenith Dissection stent, Cook Medical) to further help the opening of the true lumen by providing support to delaminated segments of the aorta at that level without the risk of covering the visceral arteries. The bare stent may also facilitate additional adjunctive procedures that may be needed in this situation to reestablish organ perfusion (eg, adjunctive visceral or iliac stenting, fenestration techniques that open the dissection flap, open revascularization techniques

for visceral arteries or lower limbs). Especially for patients with malperfusion due to dynamic obstruction, endovascular fenestration of the intimal flap can be considered to increase the outflow of the false lumen.[6]

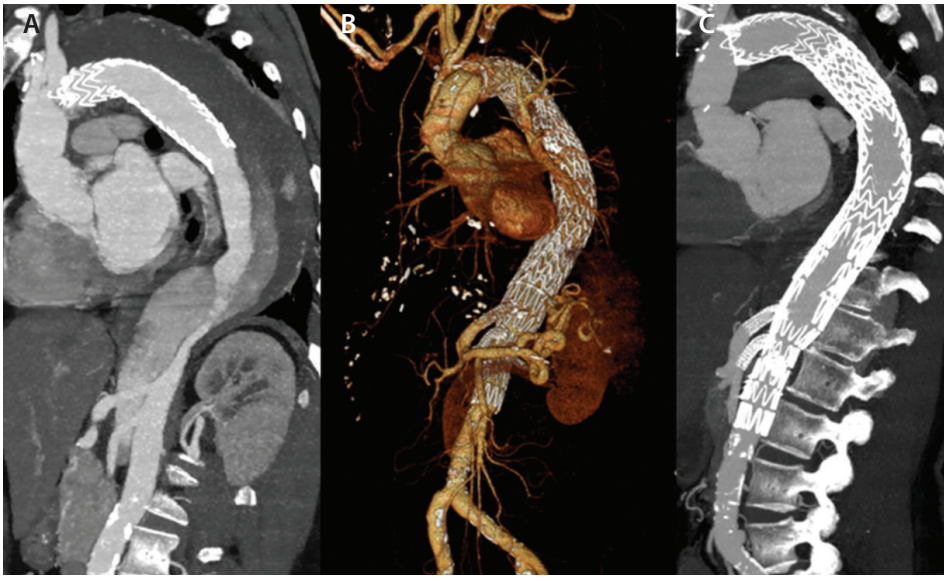


Figure 1. Chronic postdissection TAAA following TEVAR for acute TBAD (A). Treatment with four-fenestration FEVAR to achieve complete sealing (B, C).

TBAD With Subacute "Soft" Complications

Although opinions on the topic may vary, refractory pain, uncontrollable hypertension, increasing pleural effusion, rapid aneurysmal expansion, and progressive narrowing of the false lumen are all potential "soft" complications during the initial admission for acute TBAD in our practice. Patients with subacute soft complications are rightfully increasingly considered for TEVAR. Timing for TEVAR in this cohort of patients remains controversial.[7,8] In the acute phase, an increased risk of retrograde aortic dissection has been reported.[9] Those who recommend waiting at least 2 weeks for the dissection process to settle down justify their choice based on lower perioperative complications with acceptable aortic remodeling rates.[2] Those who recommend treating patients in the acute phase believe that the risks are acceptable and aortic remodeling will be maximized the sooner TEVAR is performed.[10] In our practice, patients with soft complications are most commonly treated in the subacute phase, usually after a first control CT at day 3 and a second at day 10.

Uncomplicated TBAD With High-Risk Anatomical Features

In our experience, patients without clinical complications but with anatomical features of the dissection associated with a higher risk for future complications can and perhaps should be considered for TEVAR. High-risk anatomical features have been widely studied and include aortic diameter > 4 cm with true and false lumens both patent, rapid expansion of the aortic diameter, primary entry tear diameter ≥ 10 mm, false lumen diameter ≥ 22 mm, large single entry tear in the inner curvature of the aortic arch, etc.[11] TEVAR can be considered in this subgroup of patients with the aim to induce positive aortic remodeling and reduce the risk for late complications (ie, aortic dilatation, aneurysm formation, rupture). Timing of TEVAR is again controversial, but a more conservative strategy toward the subacute phase seems logical in our opinion and is also more practical in terms of logistics (ie, time to plan the operation and materials).

In the aforementioned patient categories, TEVAR is considered the first choice above surgery. It is important to realize that TEVAR alone may not "do the job" as previously mentioned. Distal extension with a bare stent (provisional extension to induce complete attachment [PETTICOAT] technique) has been extensively studied in both the STABLE I and II trials in the United States.[12] They used the noncovered Zenith Dissection stent and showed a clear benefit with regard to true lumen perfusion. During follow-up, however, no significant reduction of distal aneurysmal degeneration could be demonstrated.

It is important to realize that TEVAR in acute TBAD is not without risks. Devastating complications such as stroke, spinal cord ischemia, and retrograde type A dissection have been reported. Arm ischemia after left subclavian artery coverage is also a serious complication. Stent graft–related complications like collapse, migration, and infection have also been reported during follow-up. The risk of TEVAR-related complications along with the fact that a significant number of TBAD patients will not develop an aneurysm during follow-up means that TEVAR in patients with soft indications sometimes may be an overtreatment, exposing the patient to operative risk without later benefit. A critical appraisal is therefore crucial for selection of suitable patients, despite accumulating data favoring TEVAR.

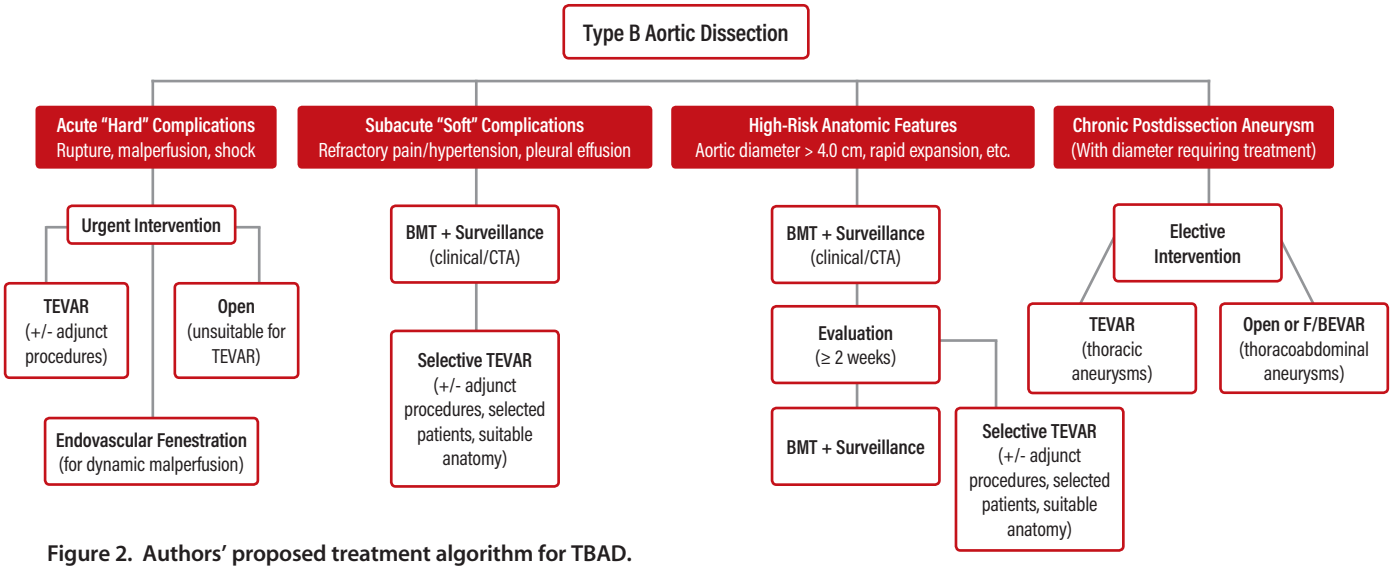


Figure 2. Authors' proposed treatment algorithm for TBAD.

Chronic Postdissection Aneurysms

In chronic TBAD, the indication for treatment is usually the postdissection aneurysm (PDA). The goal of treatment is to exclude the aneurysm to prevent future rupture. This can only be achieved by sealing both proximally and distally. With standard TEVAR, this should only be attempted in those exceptional cases when the PDA is confined to the thoracic descending aorta.[13-17] For more extensive thoracoabdominal PDA, a more complex fenestrated and branched endovascular aneurysm repair (F/BEVAR) may be required to exclude all entry and reentry tears, as well as to achieve complete sealing (Figure 1), the availability of which may be restricted/limited depending on the region. For completeness, we report that some authors have used TEVAR plus the Zenith Dissection bare stent to treat selected cases of more extensive PDA. However, the Zenith Dissection bare stent is intended for placement only in nonaneurysmal segments of dissected aorta.

F/BEVAR has been used in recent years to treat PDA of the thoracoabdominal aorta. Additional technical difficulties compared to standard atherosclerotic thoracoabdominal aortic aneurysm (TAAA) include the narrow true lumen, target vessels that originate from the false lumen, and finding/creating adequate proximal and distal sealing zones. Due to these technical difficulties, the experience with F/BEVAR in the treatment of PDA has been limited to a few referral centers.[18-21] The first reported experience with F/BEVAR in PDA was published by our group in 2012 and only included six patients.[22] In 2014, our combined experience with the University Hospital of Regensburg was published with a total of 31 patients.[23] The technical success in this series was 93.5% with a 30-day mortality of 9.6%, reflecting a steep learning curve. Mortality has now regressed below 5% in our personal series of more than 70 patients. The updated published combined experience of Nuremberg and Regensburg includes 71 patients.[24] Technical success was 95.8% with an in-hospital mortality of 5.6%. Cumulative survival rates at 12, 24, and 36 months were 84.7% ± 4.5%, 80.7% ± 5.1%, and 70.0% ± 6.7%, respectively. Mean aneurysm sac regression during follow-up was 9.2 ± 8.8 mm, with a false lumen thrombosis rate of 85.4% for patients with a follow-up longer than 12 months. No ruptures occurred during follow-up, showing that F/BEVAR can be a safe and effective treatment for extensive thoracoabdominal PDA.

Personal Treatment Algorithm for TBAD Patients

Where do we stand today in terms of decision-making for TBAD? According to the available evidence, urgent TEVAR should be the first-line intervention in patients with acute (hard) complicated TBAD. For patients with subacute (soft) complications and/or anatomic features that predispose them to future complications, TEVAR should probably be considered on an individual basis in the subacute phase. Finally, for chronic PDA, standard TEVAR has only a limited role in patients where distal sealing can be achieved in the thoracic aorta. For more extensive PDA, adequate sealing requires the use of F/BEVAR. This is summarized in a treatment algorithm proposed by the authors (Figure 2).

REMAINING QUESTIONS AND FUTURE PERSPECTIVES

Despite evident progress in the understanding and management of TBAD during the last decade, several questions remain unanswered. Further studies aiming to define subgroups of patients who are more likely to have

late aortic events and therefore justify early treatment with TEVAR are needed. More evidence is also needed with regard to the best timing for TEVAR, especially for patients who can wait without missing the best treatment window to maximize aortic remodeling. Late distal aneurysmal degeneration after both medical treatment and TEVAR for acute TBAD is a serious concern and has led to the evolution of several adjunctive endovascular techniques to counteract distal aortic dilatation.

The PETTICOAT technique with additional stenting over the visceral arteries using the Zenith Dissection stent has demonstrated benefits with regard to true lumen diameter but failed to show a clear advantage with regard to prevention of aneurysmal dilatation. In Europe, a new adjunctive technique is being evaluated by physicians (without industry involvement or support due to its off-label use), the stent-assisted balloon-induced intimal disruption and relamination in aortic dissection repair (STABILISE) concept, which includes the use of a stent graft to cover the proximal entry tear (TEVAR), followed by a noncovered stent over the visceral arteries (like PETTICOAT), and then additional ballooning with a larger balloon to disrupt the dissection flap with the aim of obliterating the false lumen and restoring single-lumen flow.[25-27] In theory, the technique seems to be a serious attempt to "cure" dissection patients and prevent late aneurysmal degeneration, but more studies are required before widespread use can be advocated. The European registry on the STABILISE concept was created by Melissano and colleagues[25] and aims to collect data from multiple European centers to monitor the technique in the long-term with the hope of providing some answers to these remaining questions. ■

1. Moulakakis KG, Mylonas SN, Dalainas I, et al. Management of complicated and uncomplicated acute type B dissection. A systematic review and meta-analysis. Ann Cardiothorac Surg. 2014;3:234-246.
2. Nienaber CA, Kische S, Rousseau H, et al. Endovascular repair of type B aortic dissection: long-term results of the randomized investigation of stent grafts in aortic dissection trial. Circ Cardiovasc Interv. 2013;6:407-416.
3. Fattori R, Montgomery D, Lovato L, et al. Survival after endovascular therapy in patients with type B aortic dissection: a report from the International Registry of Acute Aortic Dissection (IRAD). JACC Cardiovasc Interv. 2013;6:876-882.
4. Brunkwall J, Kasprzak P, Verhoeven E, et al. Endovascular repair of acute uncomplicated aortic type B dissection promotes aortic remodelling: 1 year results of the ADSORB trial. Eur J Vasc Endovasc Surg. 2014;48:285-291.
5. Nienaber CA, Rousseau H, Eggebrecht H, et al. Randomized comparison of strategies for type B aortic dissection: the investigation of stent grafts in aortic dissection (INSTEAD) trial. Circulation. 2009;120:2519-2528.
6. Riambau V, Böckler D, Brunkwall J, et al. Editor's choice 2013—Management of descending thoracic aorta diseases: clinical practice guidelines of the European Society for Vascular Surgery (ESVS). Eur J Vasc Endovasc Surg. 2017;53:4-52.
7. Song C, Lu Q, Zhou J, et al. The new indication of TEVAR for uncomplicated type B aortic dissection. Medicine. 2016;95:e3919.
8. Conrad MF, Crawford RS, Kwolek CJ, et al. Aortic remodeling after endovascular repair of acute complicated type B aortic dissection. J Vasc Surg. 2009;50:510-517.
9. Chen Y, Zhang S, Liu L, et al. Retrograde type A aortic dissection after thoracic endovascular aortic repair: a systematic review and meta-analysis. J Am Heart Assoc. 2017;6:e004649.
10. Qin YL, Wang F, Li TX, et al. Endovascular repair compared with medical management of patients with uncomplicated type B acute aortic dissection. J Am Coll Cardiol. 2016;67:2835-2842.
11. Acosta S, Blomstrand D, Gottsater A. Epidemiology and long-term prognostic factors in acute type B aortic dissection. Ann Vasc Surg. 2007;21:415-422.
12. Lombardi JV, Cambria RP, Nienaber CA, et al. Prospective multicenter clinical trial (STABLE) on the endovascular treatment of complicated type B aortic dissection using a composite device design. J Vasc Surg. 2012;55:629-640.e2.
13. Kang WC, Greenberg RK, Mastracci TM, et al. Endovascular repair of complicated chronic distal aortic dissections: intermediate outcomes and complications. J Thorac Cardiovasc Surg. 2011;142:1074-1083.
14. Mani K, Clough RE, Lyons OT, et al. Predictors of outcome after endovascular repair for chronic type B dissection. Eur J Vasc Endovasc Surg. 2012;43:386-391.
15. Sayer D, Bratby M, Brooks M, et al. Aortic morphology following endovascular repair of acute and chronic type B aortic dissection: implications for management. Eur J Vasc Endovasc Surg. 2008;36:522-529.
16. Manning BJ, Dias N, Ohrlander T, et al. Endovascular treatment for chronic type B dissection: limitations of short stent-grafts revealed at midterm follow-up. J Endovasc Ther. 2009;16:590-597.
17. Scali ST, Feezor RJ, Chang CK, et al. Efficacy of thoracic endovascular stent repair for chronic type B aortic dissection with aneurysmal degeneration. J Vasc Surg. 2013;58:10-17.e1.
18. Kitagawa A, Greenberg RK, Eagleton MJ, et al. Fenestrated and branched endovascular aortic repair for chronic type B aortic dissection with thoracoabdominal aneurysms. J Vasc Surg. 2013;58:625-634.
19. Spear R, Sobocinski J, Settembre N, et al. Early experience of endovascular repair of post-dissection aneurysms involving the thoraco-abdominal aorta and the arch. Eur J Vasc Endovasc Surg. 2016;51:488-497.
20. Spear R, Hertault A, Van Calster K, et al. Complex endovascular repair of postdissection arch and thoracoabdominal aneurysms. J Vasc Surg. 2018;67:685-693.
21. Law Y, Tsilimparis N, Rohlffs F, et al. Fenestrated or branched endovascular aortic repair for postdissection thoracoabdominal aortic aneurysm. J Vasc Surg. 2019;70:404-412.
22. Verhoeven EL, Paraskevas KI, Oikonomou K, et al. Fenestrated and branched stent-grafts to treat post-dissection chronic aortic aneurysms after initial treatment in the acute setting. J Endovasc Ther. 2012;19:343-349.
23. Oikonomou K, Kopp R, Katsargyris A, et al. Outcomes of fenestrated/branched endografting in post-dissection thoracoabdominal aortic aneurysms. Eur J Vasc Endovasc Surg. 2014;48:641-648.
24. Oikonomou K, Kasprzak P, Katsargyris A, et al. Mid-term results of fenestrated/branched stent grafting to treat post-dissection thoraco-abdominal aneurysms. Eur J Vasc Endovasc Surg. 2019;57:102-109.
25. Melissano G, Bertoglio L, Rinaldi E, et al. Satisfactory short-term outcomes of the STABILISE technique for type B aortic dissection. J Vasc Surg. 2018;68:966-975.
26. Hofferberth SC, Nixon IK, Boston RC, et al. Stent-assisted balloon-induced intimal disruption and relamination in aortic dissection repair: the STABILISE concept. J Thorac Cardiovasc Surg. 2014;147:1240-1245.
27. Faure EM, El Batti S, Abou Rjeili M, et al. Mid-term outcomes of stent assisted balloon induced intimal disruption and relamination in aortic dissection repair (STABILISE) in acute type B aortic dissection. Eur J Vasc Endovasc Surg. 2018;56:209-215.

Athanasios Katsargyris, MD
General Hospital Nuremberg
Paracelsus Medical University
Nuremberg, Germany
Disclosures: None.

Pablo Marques de Marino, MD
General Hospital Nuremberg
Paracelsus Medical University
Nuremberg, Germany
Disclosures: None.

Balazs Botos, MD
General Hospital Nuremberg
Paracelsus Medical University
Nuremberg, Germany
Disclosures: None.

Eric Verhoeven, MD, PhD
Chief, Department of Vascular and Endovascular Surgery
General Hospital Nuremberg
Paracelsus Medical University
Nuremberg, Germany
eric.verhoeven@klinikum-nuernberg.de
Disclosures: Speaker, proctor, consultant, and research grants from Cook Medical.

Results From the STABLE Clinical Trials

Recently published data from the STABLE I and STABLE II trials supporting a composite device approach to endovascular treatment of TBAD.

BY JOSEPH V. LOMBARDI, MD, AND QING ZHOU, PHD

The management of type B aortic dissection (TBAD) has been completely transformed in the last 2 decades, during which, thoracic endovascular aortic repair has become the treatment of choice for TBAD with complicated presentations of rupture and malperfusion.[1-3] Many challenges still exist for the management of this complex disease, such as persistent true lumen compression despite proximal coverage of the primary entry tear, aortic remodeling limited within the stent graft coverage, persistent false lumen perfusion, and the risk of aneurysmal growth and degeneration along the dissected aorta in the long term.

The Zenith Dissection endovascular stent system (Cook Medical) is a unique composite device system (proximal stent graft and distal bare-metal stent) that has been developed to provide disease-specific treatment for TBAD. The proximal stent graft is placed first to cover the primary entry tear, thus directing the flow into the true lumen. The bare-metal stent is then placed distally to the stent graft to provide expansile support of the true lumen along the dissected aorta, without blocking important branch vessels. This composite device system received FDA approval in December 2018 for the endovascular treatment of patients with TBAD.[4] This approval was based on results from the pivotal clinical study of the Zenith Dissection endovascular system (the STABLE II clinical study) and was also supported by supplementary data from the STABLE I clinical study.

THE STABLE STUDIES

STABLE I and STABLE II are two prospective, nonrandomized, multicenter studies conducted to evaluate the aforementioned composite device system. In the STABLE I feasibility study, 86 patients were enrolled between December 2007 and February 2012 at sites in the United States, Europe, and Australia. STABLE I (registered at clinicaltrials.gov as NCT02094300 for the portion of the study conducted within the United States and as NCT00526487 for the portion of the study conducted outside of the United States) included patients treated in the acute phase (≤ 14 days of symptom onset) or outside the acute phase (15–90 days), who presented with branch vessel obstruction/compromise, impending rupture, resistant hypertension, persistent pain/symptoms, or rapid aortic growth (or large transaortic diameter).

The STABLE II pivotal study (NCT01568320), on the other hand, focused on only acute, complicated TBAD that presented with aortic rupture or branch vessel malperfusion, and 73 patients were enrolled between August 2012 and January 2015 at sites in the United States and Japan. As for the study device, the current designs (the barbless Zenith TX2 Dissection endovascular graft with Pro-Form and the Zenith Dissection endovascular stent made of nitinol) were evaluated in the STABLE II pivotal study, while a predicate iteration (stent graft with barbs and stainless-steel dissection stent) was used in the earlier STABLE I study. For both studies, the total follow-up duration is 5 years.

RESULTS FROM STABLE I

Clinical and aortic remodeling results from the STABLE I study have been published since the first report in 2012,[5,6] and the final 5-year study results were recently published.[7] Two important findings from the STABLE I study are the low 30-day mortality and paraplegia rates. The 30-day all-cause mortality rate was 5.5% (3/55) for acute dissection patients and 3.2% (1/31) for nonacute patients. Likewise, the 30-day paraplegia rate was 1.8% (1/55) in the acute dissection patients and 0% in the nonacute patients. Only one additional patient treated for nonacute dissection experienced paraparesis within 30 days. Although the STABLE I study included patients with relatively wider entry criteria for presenting complications, most patients presented with multiple complications (median, three indications per patient) and extensive, DeBakey type IIIb dissection, thus representative of the complicated circumstances requiring intervention beyond medical management.

At 5 years, the Kaplan-Meier estimate of freedom from dissection-related mortality (including deaths of indeterminate relatedness to dissection repair) was 83.9% (standard error [SE], 5.9%) for acute patients and 90.1% (SE, 5.9%) for nonacute patients. Freedom from secondary intervention was 65.5% (SE, 7.5%) for acute

and 71.2% (SE, 9.0%) for nonacute patients. In terms of aortic remodeling, complete false lumen thrombosis in the thoracic aorta increased over time and was seen in 74.1% of acute and in 58.8% of nonacute patients at 5 years. In both acute and nonacute patients, there was an overall increase in true lumen diameter and a concomitant decrease in false lumen diameter in not only the thoracic aorta but also the abdominal aorta from preprocedure through 5 years. Most patients exhibited a stable or shrinking transaortic diameter in the thoracic aorta at 5 years (acute, 65.5%; nonacute, 81.3%), and the proportion was slightly lower in the abdominal aorta (acute, 48.3%; nonacute, 76.5%).

Overall, patients treated in acute and nonacute phases appeared to respond similarly to the endovascular treatment with the composite device design, which showed low 30-day mortality and paraplegia rates and favorable improvement in aortic remodeling through 5 years. The need for reintervention in approximately 30% of patients by 5 years underscores the importance of lifelong and close surveillance of this patient population.

RESULTS FROM STABLE II

The STABLE II study results through 1 year were recently published.[8] Device implantation was successful in all patients, with an average procedure time of 154.9 minutes (range, 54–519 minutes), average intensive care unit stay of 6.3 days (range, 0–30 days), and average hospital stay of 11.8 days (range, 1–47 days). Thirty-day mortality occurred in 5 of 73 (6.8%) patients, and 30-day major adverse events (MAEs) included myocardial infarction (1.4%), bowel ischemia (1.4%), renal insufficiency/renal failure requiring dialysis (6.8%), stroke (6.8%), paraplegia/paraparesis (5.5%), and prolonged ventilatory support (13.7%).

At 1 year, the Kaplan-Meier estimate of freedom from all-cause mortality was 80.3% (SE, 4.7%), with nine deaths occurring from 31 to 365 days and only one of them related to dissection repair. Within 1 year, 12.3% (9/73) of patients underwent secondary interventions, and none of the patients required conversion to open surgery. In terms of aortic remodeling, among patients with 12-month follow-up CT imaging, complete or partial thrombosis of the false lumen was seen in 100% of patients within the stent graft region and in 97.4% of patients within the dissection stent region. Growth (> 5 mm) of the maximum transaortic diameter was seen in 14.9% of patients in the stent graft region and in 38.5% of patients within the dissection stent region.

According to the endpoint analysis per protocol among 67 patients who met the clarified study criteria, both the primary safety end-point (30-day freedom from MAEs: 71.6%; 95% confidence interval [CI], 59%–82%) and the primary effectiveness endpoint (30-day survival rate: 95.5%; 95% CI, 87%–99%) met the performance goals derived from the published Society for Vascular Surgery data set.

SUMMARY

Results from the STABLE II pivotal study for acute, complicated TBAD provided a reasonable assurance of safety and effectiveness in support of device approval in the United States having met the primary endpoints (30-day survival and MAEs) and demonstrated outcomes consistent with expectations for endovascular treatment of TBAD, including adverse event rates beyond 30 days, reinterventions, and results from follow-up imaging assessments. Results from the STABLE I feasibility study provided additional evidence (in combination with other data sources) to support a broader indication inclusive of acute and chronic TBAD. ■

1. Dake MD, Kato N, Mitchell RS, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. N Engl J Med. 1999;340:1546-1552.
2. Trimarchi S, Nienaber CA, Rampoldi V, et al. Role and results of surgery in acute type B aortic dissection: insights from the International Registry of Acute Aortic Dissection (IRAD). Circulation. 2006;114(1 suppl):I357- I364.
3. Fattori R, Cao P, De Rango P, et al. Interdisciplinary expert consensus document on management of type B aortic dissection. J Am Coll Cardiol. 2013;61:1661-1678.
4. Zenith Dissection Endovascular System—P180001. U.S. Food and Drug Adminstration web site. https://www.fda.gov/medical-devices/recently-approved-devices/zenith-dissection-endovascular-system-p180001. Accessed September 26, 2019.
5. Lombardi JV, Cambria RP, Nienaber CA, et al. Prospective multicenter clinical trial (STABLE) on the endovascular treatment of complicated type B aortic dissection using a composite device design. J Vasc Surg. 2012;55:629-640.e2.
6. Lombardi JV, Cambria RP, Nienaber CA, et al. Aortic remodeling after endovascular treatment of complicated type B aortic dissection with the use of a composite device design. J Vasc Surg. 2014;59:1544-1554.
7. Lombardi JV, Cambria RP, Nienaber CA, et al. Five-year results from the Study of Thoracic Aortic Type B Dissection Using Endoluminal Repair (STABLE I) study of endovascular treatment of complicated type B aortic dissection using a composite device design. J Vasc Surg. 2019;70:1072-1081.e2.
8. Lombardi JV, Gleason TG, Panneton JM, et al. STABLE II clinical trial on endovascular treatment of acute, complicated type B aortic dissection with a composite device design [published online August 30, 2019]. J Vasc Surg.

Joseph V. Lombardi, MD
Head, Division of Vascular and Endovascular Surgery
Professor of Surgery
Cooper Medical School of Rowan University
Director, Cooper Aortic Center
Cooper University Health Care
Camden, New Jersey
lombardi-joseph@cooperhealth.edu
Disclosures: Speaker, consultant, and principal investigator for Cook Medical.

Qing Zhou, PhD
Team Lead, Scientific Publications
Cook Research Incorporated
West Lafayette, Indiana
Disclosures: Full-time employee of Cook Research Incorporated.

Sponsored by Cook Medical

TEVAR Alone Versus the STABLE Technique for Acute Complicated TBAD

A review of the history of the STABLE technique and data from two post hoc comparative analyses comparing these approaches.

BY JONATHAN SOBOCINSKI, MD, PHD; DOMINIQUE FABRE, MD; RICHARD AZZAOUI, MD; AND STÉPHAN HAULON, MD, PHD

Thoracic endovascular aortic repair (TEVAR) is the first-line therapy for acute complicated type B aortic dissection (TBAD).[1] The first goal is to treat complications such as malperfusion and/or exclude aortic rupture to save the patient's life. The principal function of TEVAR is to exclude/cover the main proximal entry tear, thereby redirecting aortic flow exclusively toward the true lumen, and ultimately decreasing the pressure within the false lumen. In a subset of patients with specific anatomic features, early TEVAR might reduce the risk of aneurysmal degeneration during follow-up by promoting early aortic remodeling. Various treatment options have been proposed to reduce early mortality and reduce late aneurysmal degeneration. In this article, we focus on potential benefits of extending TEVAR with a bare-metal stent implanted within the true lumen beyond the thoracoabdominal aortic junction, which is known as the *PETTICOAT* or *STABLE* technique.

HISTORY OF THE STABLE TECHNIQUE

Before the advent of TEVAR via stent graft placement, endovascular options were restricted to visceral artery stenting and/or intimal flap fenestration for TBAD.[2] Since the first publications in 1999,[3,4] TEVAR has evolved to be the best invasive treatment strategy. In our practice, TEVAR for acute TBAD is restricted to complicated cases, defined as aortic rupture (frank or periaortic effusion) and/or organ malperfusion syndrome. However, some authors have suggested consideration of other criteria as well, such as large aortic diameters at onset, refractory pain, and/or persistent hypertension, to define complicated dissections.[5]

In 2005, Mossop et al published their initial experience combining thoracic endografting with a self-expandable bare stent placed distally,[6] which was called the *PETTICOAT* strategy. This technique has two main goals: (1) to increase the expansion of the true lumen and thus reduce malperfusion, and (2) to promote aortic remodeling. Several authors have reported their experience with this strategy,[7,8] and Cook Medical has developed an aortic bare-metal stent specifically for dissection treatment. Cook's Zenith Dissection endovascular system, comprising a stent graft component and the distal bare stent component specifically for dissection treatment, was evaluated in the STABLE I and II studies.[9,10] The STABLE I study assessed an earlier iteration of the device combination (Zenith TX2 stent grafts and stainless steel bare stents) in patients who were treated at up to 90 days from dissection symptom onset and presented with a wide range of indications. The STABLE II pivotal study, conducted later, evaluated the current device system (barbless stent grafts and nitinol bare stents) in patients who presented with only acute, complicated TBAD. Both studies assessed outcomes up to 5 years, but neither compared the results of this combined strategy to endografting alone. We thus conducted two secondary analyses comparing the results from the STABLE cohorts to the results from high-volume European aortic centers, where endografting alone was performed to treat acute complicated TBAD.[11,12]

AORTIC REMODELING

Aortic remodeling is a combination of true lumen expansion and false lumen thrombosis and shrinkage. It is well described in the literature that TEVAR promotes aortic remodeling at the level of the endograft but has little or no influence on the aorta beyond the diaphragm. There has been scarce literature on the impact of bare stent placement at the level of the thoracoabdominal aorta on aortic remodeling.[8,13]

In this context, we conducted a post hoc comparative analysis of two groups of patients surviving 1 year after

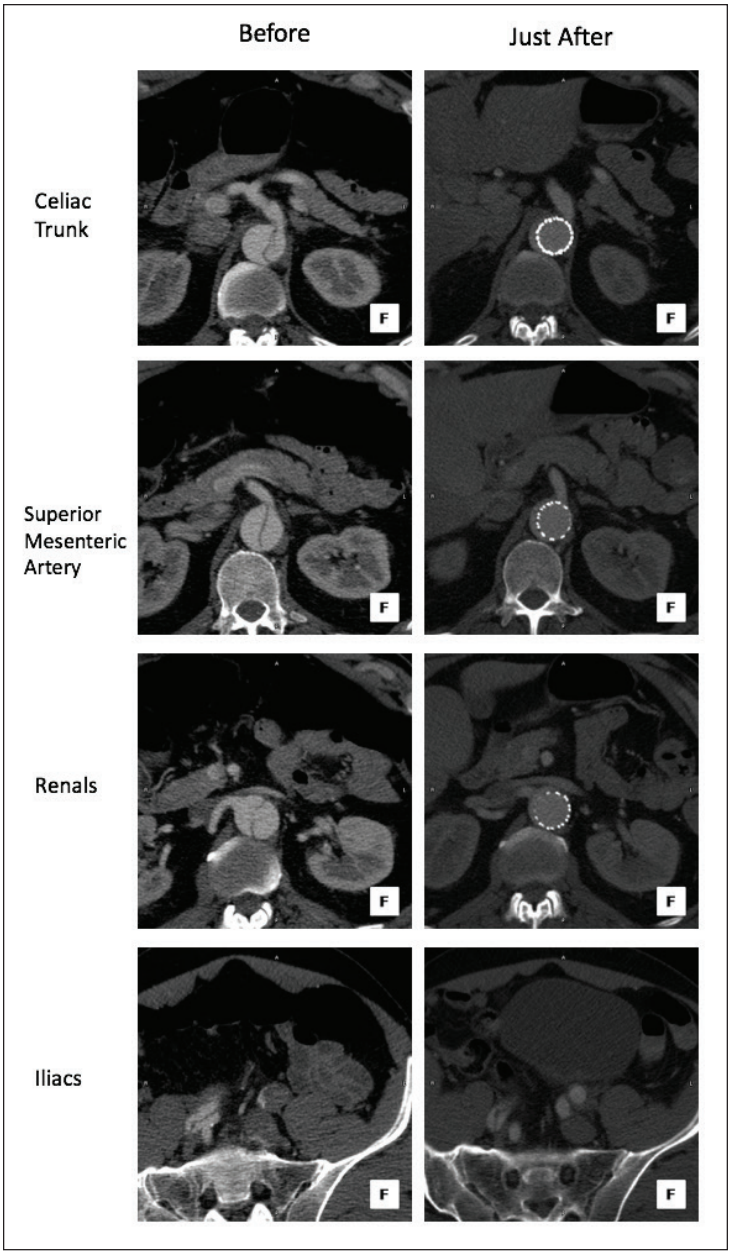


Figure 1. Images of a patient treated for acute complicated TBAD with the STABLE technique. In the left column, slices of the preoperative CTA show collapse of the true lumen along the aorta beyond the thoracoabdominal junction. In the right column, slices of the immediate postoperative CTA exhibit a satisfying opening of the true lumen scaffolding by the bare-metal stent toward the abdominal aorta.

endovascular treatment of an acute complicated type B dissection, with available CT at baseline and at 12 months: one group was treated with TEVAR alone (n = 45) at three high-volume institutions in Lille, France; Caen, France; and Malmö, Sweden; the other group was treated with the PETTICOAT strategy (n = 39) in the STABLE I study.[11] A thorough morphological analysis of the aorta, including changes in aortic volumes, was conducted through 1 year, and details of the initial and secondary procedures were collected.

During the initial procedure, the length of aorta covered by the endograft within the descending thoracic aorta was comparable in both groups (184.0 ± 48.7 mm in TEVAR alone vs 166.6 ± 47.2 mm in STABLE; $P = .11$). In terms of clinical outcomes at 1 year, the reintervention rates were similar between the two groups (11.1% in TEVAR alone vs 12.8% in STABLE). In terms of aortic remodeling results, while both groups showed significant remodeling in the thoracic aorta (true lumen increase and false lumen decrease in aortic volume), we observed some differences in the abdominal aorta. Only the STABLE group exhibited a statistically significant increase in true lumen volume at the level of the abdominal aorta, most prominently seen on the postoperative as compared to the preprocedure CT scans ($P < .001$), as well as from postprocedure to 1 year ($P = .035$), while the changes within the TEVAR alone group were not statistically significant. When compared between the two groups, the overall change in the true lumen volume from preprocedure to 1 year was greater in the STABLE group (16 cm^3) than in the TEVAR group (10 cm^3) but was not statistically significant ($P = .10$).

From these results, we hypothesized that this early benefit of true lumen expansion in the abdominal aorta, in relation to the implantation of a bare self-expandable stent in the true lumen, could have an impact on the outcomes of patients presenting with malperfusion at onset (Figure 1). We thus conducted the study described thereafter.

MALPERFUSION

We performed a second post hoc comparative analysis focusing on short-term outcomes of two patient groups treated for acute TBAD with malperfusion (imaging findings and/or clinical signs) diagnosed at onset.[12] The first group (n = 41; from Lille, France and Malmö, Sweden) was treated with TEVAR alone, whereas the second group (n = 84; from both the STABLE I and STABLE II studies) was treated with the composite device design.

At presentation, comparable organ system involvement in malperfusion was depicted, and both groups showed

similar lengths of dissection and similar locations of the proximal and distal aspects of the dissection. The STABLE patients presented with a higher American Society of Anesthesiologists class, greater prevalence of renal insufficiency, and worse preoperative hypertension and renal function status (according to Society for Vascular Surgery scores) compared with the TEVAR patients. Both groups received a median of one stent graft component (range, 1–2 for TEVAR alone vs 1–3 for STABLE; $P = .66$). Additional selective stenting of visceral and renal branches was required in 46% of TEVAR patients and 30% of STABLE patients after endograft deployment ($P = .08$).

The 30-day mortality rate in the STABLE group was half of that in the TEVAR group, but this difference was not statistically significant (8.3% [7/84] vs 17.1% [7/41]; $P = .22$). Malperfusion-related mortality, defined as deaths caused by bowel/mesenteric ischemia or multiple organ failure, was statistically lower in the STABLE group (2.3% [2/84] vs 12.2% [5/41]; $P = .038$). The 30-day rates of morbidity such as renal failure requiring dialysis, bowel ischemia, and neurologic events were similar between the groups, as were the 30-day rates of secondary interventions (7.3% for TEVAR alone and 7.1% for STABLE group). Similar to findings from our earlier aortic volume study, the amount of true lumen diameter increase was statistically significantly greater in the STABLE group than in the TEVAR group in the abdominal aorta ($P < .001$) but not in the thoracic aorta ($P = .835$).

CONCLUSION

Our volume analysis comparing TEVAR and STABLE showed no statistically significant difference in terms of overall aortic remodeling at 1 year, but the STABLE cohort showed a significant increase of true lumen volume in the abdominal aorta postoperatively. This more prominent true lumen expansion in the distal aorta was also observed at postprocedure in the composite device group in our second study focusing on acute type B dissections in the setting of malperfusion and may have contributed to alleviation of branch vessel malperfusion. In this study, TEVAR + bare-metal stenting showed a twofold reduction in all-cause early mortality, albeit statistically insignificant, and statistically significantly lower 30-day malperfusion-related mortality in patients with acute TBAD with malperfusion compared to TEVAR alone.

Our results suggest that aortic bare-metal stenting in addition to endografting of the proximal descending thoracic aorta should be proposed for patients with malperfusion at onset to improve early survival. Larger cohorts and prospective randomization of patients to both treatment options would be required to confirm these results. ■

1. Fattori R, Cao P, De Rango P, et al. Interdisciplinary expert consensus document on management of type B aortic dissection. J Am Coll Cardiol. 2013;61:1661-1678.
2. Elefteriades JA, Hammond GL, Gusberg RJ, et al. Fenestration revisited. A safe and effective procedure for descending aortic dissection. Arch Surg. 1990;125:786-790.
3. Nienaber CA, Fattori R, Lund G, et al. Nonsurgical reconstruction of thoracic aortic dissection by stent-graft placement. N Engl J Med. 1999;340:1539-1545.
4. Dake MD, Kato N, Mitchell RS, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. N Engl J Med. 1999;340:1546-1552.
5. Tolenaar JL, Froehlich W, Jonker FH, et al. Predicting in-hospital mortality in acute type B aortic dissection: evidence from International Registry of Acute Aortic Dissection. Circulation. 2014;130(11 suppl 1):S45-S50.
6. Mossop PJ, McLachlan CS, Amukotuwa SA, Nixon IK. Staged endovascular treatment for complicated type B aortic dissection. Nat Clin Pract Cardiovasc Med. 2005;2:316-321.
7. Hofferberth SC, Foley PT, Newcomb AE, et al. Combined proximal endografting with distal bare-metal stenting for management of aortic dissection. Ann Thorac Surg. 2012;93:95-102.
8. Melissano G, Bertoglio L, Rinaldi E, et al. Volume changes in aortic true and false lumen after the "PETTICOAT" procedure for type B aortic dissection. J Vasc Surg. 2012;55:641-651.
9. Lombardi JV, Cambria RP, Nienaber CA, et al; STABLE investigators. Prospective multicenter clinical trial (STABLE) on the endovascular treatment of complicated type B aortic dissection using a composite device design. J Vasc Surg. 2012;55:629-640.e2.
10. Lombardi JV, Cambria RP, Nienaber CA, et al; STABLE I Investigators. Five-year results from the Study of Thoracic Aortic Type B Dissection Using Endoluminal Repair (STABLE I) study of endovascular treatment of complicated type B aortic dissection using a composite device design. J Vasc Surg. 2019;70:1072-1081.
11. Sobocinski J, Lombardi JV, Dias NV, et al. Volume analysis of true and false lumens in acute complicated type B aortic dissections after thoracic endovascular aortic repair with stent grafts alone or with a composite device design. J Vasc Surg. 2016;63:1216-1224.
12. Sobocinski J, Dias NV, Hongku K, et al. Thoracic endovascular aortic repair with stent grafts alone or with a composite device design in patients with acute type B aortic dissection in the setting of malperfusion [published online July 4, 2019]. J Vasc Surg.
13. Lombardi JV, Cambria RP, Nienaber CA, et al; STABLE investigators. Aortic remodeling after endovascular treatment of complicated type B aortic dissection with the use of a composite device design. J Vasc Surg. 2014;59:1544-1554.

Jonathan Sobocinski, MD, PhD
Aortic Centre
University Hospital of Lille
Lille, France
Disclosures: Proctor and consultant for Cook Medical.

Dominique Fabre, MD
Aortic Centre
Hôpital Marie Lannelongue
Université Paris Sud
Le Plessis Robinson, France
Disclosures: None.

Richard Azzaoui, MD
Aortic Centre
University Hospital of Lille
Lille, France
Disclosures: None.

Stéphan Haulon, MD, PhD
Aortic Centre
Hôpital Marie Lannelongue
Université Paris Sud
Le Plessis Robinson, France
haulon@hotmail.com
Disclosures: Speaker, proctor, and consultant for Cook Medical.

Impact of Proximal Seal Zone in Managing Type B Aortic Dissection

A look at how zone 2 involvement and proximal seal affect TEVAR outcomes for TBAD.

BY JOSEPH V. LOMBARDI, MD

Zone 2 involvement (ie, disease extending to the aortic segment between the distal margin of left common carotid artery [LCCA] and distal margin of the left subclavian artery [LSA]) in type B aortic dissection (TBAD) can be a dilemma for surgeons who perform thoracic endovascular aortic repair (TEVAR). Although landing in healthy aorta is always the goal, many surgeons proceed with placing the stent graft in a suboptimal landing zone with disease involvement (frank dissection or, more commonly, intramural hematoma [IMH]) or inadequate sealing length.

In the STABLE studies, the protocols required that TBAD did not extend proximal to the LSA and a ≥ 20-mm proximal landing zone length between the LCCA and the most proximal extent of dissection. Although these criteria were met according to best site assessments, centralized core laboratory analysis of the three-dimensional reconstructed CT imaging indicated that many patients exhibited more extensive dissections. The reason for this inconsistency is multifactorial. When patients present in the acute setting and need emergent treatment, it can be difficult to obtain a full evaluation of the entire proximal landing zone as thoroughly as the core laboratory. Also, no current standards allow for consistent categorization of full disease involvement in the proximal seal zone, which may also have contributed to inconsistency in imaging assessment among surgeons and sites. Needless to say, this phenomenon likely occurs with a similar or greater frequency in the "real world," outside the confines of a clinical trial.

The effect on suboptimal proximal seal zone on outcomes after endovascular TBAD repair has not been well studied in the literature. The literature that does exist suggests that landing in an unhealthy aorta increases the risk of retrograde dissection. In a study by Kuo et al involving 71 patients who underwent TEVAR for complicated TBAD, a majority (63%) had a proximal seal zone entirely in IMH or dissected aorta.[1] During a mean follow-up of 14 months, two confirmed cases and one suspected case of retrograde dissection occurred exclusively in the patients with circumferential IMH in the landing zone. In the STABLE I and II studies, it was also found that most patients who experienced retrograde dissection or proximal type I entry flow had inadequate proximal landing zone by core laboratory analysis.[2,3]

Recently, the effects of the achieved proximal seal length on outcomes from the STABLE studies have been studied.[4] This analysis included 110 patients from the STABLE I and STABLE II studies who were treated for acute TBAD and who had available core laboratory measurements for the achieved seal length, calculated as the difference between available seal length (from the LCCA to the proximal extent of dissection) on preprocedure CT and uncovered length (from the LCCA to the first 360° visualization of the stent) on postoperative CT. Based on the achieved seal length, these 110 patients were divided into four groups: ≥ 20 mm (n = 19), 10 to < 20 mm (n = 25), 0 to < 10 mm (n = 36), and < 0 mm (n = 30). The low proportion of patients who achieved 20 mm of proximal seal was due to not only inadequate seal zone to begin with in some patients but also inadequate use of available sealing zone during device deployment in additional patients. For example, in patients who required stent graft delivery adjacent to the LCCA, the investigators tended to be more cautious for fear of covering the carotid orifice.

We examined a composite outcome of device events (proximal type I entry flow, device migration, transaortic growth > 5 mm, or retrograde dissection) and observed an inverse relationship between this outcome and achieved seal length. The cumulative rate of this outcome at a mean follow-up of 39.6 ± 20.4 months was lowest in patients with proximal seal length of ≥ 20 mm (15.8%), and this rate increased as the seal length decreased: 32.0% for seal length of ≥ 10 to < 20 mm, 52.8% for ≥ 0 to < 10 mm, and 60.0% for < 0 mm (P < .01, Cochran-Armitage trend test).

These results highlight the importance, albeit with many challenges, of landing an endograft in healthy and

stable aorta in patients requiring urgent management for complicated TBAD. Emergent TEVAR with inadequate seal length should lead to heightened surveillance algorithms for retrograde extension and early aneurysm formation. Utilization of intraoperative transesophageal echocardiography and predischarge CTA can be useful in establishing baseline anatomical characteristics and early diagnosis of aortic-related morbidity. ■

1. Kuo EC, Veranyan N, Johnson CE, et al. Impact of proximal seal zone length and intramural hematoma on clinical outcomes and aortic remodeling after thoracic endovascular aortic repair for aortic dissections. J Vasc Surg. 2019;69:987-995.
2. Lombardi JV, Cambria RP, Nienaber CA, et al. Prospective multicenter clinical trial (STABLE) on the endovascular treatment of complicated type B aortic dissection using a composite device design. J Vasc Surg. 2012;55:629-640.e2.
3. Lombardi JV, Gleason TG, Panneton JM, et al. STABLE II clinical trial on endovascular treatment of acute, complicated type B aortic dissection with a composite device design [published online August 30, 2019]. J Vasc Surg.
4. Lombardi JV, Famularo M, Seale MM, et al. RS08. Inadequate proximal seal length predicts complications after endovascular repair of acute type B aortic dissection. J Vasc Surg. 2018;67:e240.

Joseph V. Lombardi, MD
Head, Division of Vascular and Endovascular Surgery
Professor of Surgery
Cooper Medical School of Rowan University
Director, Cooper Aortic Center
Cooper University Health Care
Camden, New Jersey
lombardi-joseph@cooperhealth.edu
Disclosures: Speaker, consultant, and principal investigator for Cook Medical.

Sponsored by Cook Medical

Imaging for TBAD: Essential and Optimal Techniques

An overview of advanced imaging methods for the diagnosis and treatment of type B aortic dissection.

BY DARREN KLASS, MBChB, MD, MRCS, FCRC, FRCPC

Imaging of aortic dissection can be performed both periprocedurally with computed tomography (CT) and intraprocedurally with transesophageal echocardiography (TEE) and subtraction angiography. Although there are limitations to each modality, overall, these methods are capable of demonstrating the pathology, allowing for a diagnosis to be made, a procedure to be performed, and follow-up to be completed with relative ease.

Aortic dissection is an extremely complex disease, often with multiple fenestrations along the length of the intimomedial flap; many of these are large and therefore pose potential pitfalls regarding passage of wires and stent grafts inadvertently into the false lumen, with potentially catastrophic consequences. In addition to the complexities encountered intraprocedurally, assessment of the morphology of the dissection and aorta at the time of diagnosis is essential to ensure the correct management strategy is undertaken. If a decision is made to proceed with endovascular repair of the dissection with stent grafts, accurate measurement of the landing zone is required to confirm appropriate stent graft sizing to minimize the risk of a retrograde dissection due to device oversizing. This seemingly simple task can be relatively inaccurate if the appropriate imaging protocols are not followed and setup of the CT scanner is not optimized.

The aim of this article is to highlight the advanced imaging techniques available that aid in diagnosis, treatment planning, procedural execution, problem solving, and follow-up.

COMPUTED TOMOGRAPHY

CT angiography is a fast, reliable, and reproducible method of assessment of acute aortic syndromes. The technology has advanced rapidly, and the speed at which images are acquired has significantly improved. Many CT scanners can acquire images using two separate imaging sources (dual energy), which in turn allow a patient to move much faster through the scanner, obtaining whole body scans with submillimeter resolution in a matter of milliseconds.[1,2]

The combination of rapid patient movement through the CT scanner and the dual energy acquisition have allowed imaging of the aorta to be acquired between two heart beats, which then images the ascending aorta with little or no motion. The coronary cusps, ascending aorta, and transverse arch are free from artifact, so diagnosing a subtle type A dissection becomes much easier for the clinician and can be made with confidence (Figure 1A). If this imaging protocol is utilized, it negates the need for a confirmatory TEE in many cases. The lack of motion of the aorta during image acquisition improves the resolution and allows for accurate measurement of the aorta, as the wall is easy to identify (Figure 1B). In cases of acute and subacute dissection, where oversizing can lead to a retrograde dissection with stent graft deployment, utilizing this technique allows measuring and planning with much more confidence and accuracy than standard image acquisition.

This technique is termed *ultrahigh-pitch CT*, where the patient is moved through the scanner much faster than a conventional scan. Ultrahigh-pitch CT scanning is possible on modern scanners due to the speed at which images are acquired, the computational power of the processors, as well as the ability to utilize electrocardiography (ECG) gating.

ECG gating allows the scanner to initiate image acquisition at particular stages of the cardiac cycle. The patient is monitored via ECG leads and the software identifies the QRS complex and triggers between the complexes to decrease motion as much as possible. This method of image acquisition has a temporal resolution of approximately 75 ms, which allows for a full high-resolution cardiac CT scan in 250 ms.

This not only allows for motion reduction in the ascending aorta but also a much more sensitive and

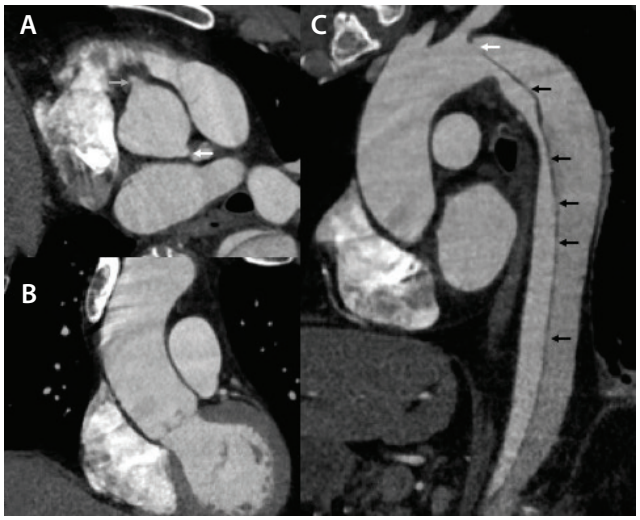


Figure 1. CT images using an ultrahigh-pitch protocol demonstrating well-visualized coronary origins (arrows) with no aortic motion (A); motionless ascending aorta, aiding in excluding a dissection and improving measurement accuracy (B); and the entry tear (white arrow) and multiple tiny fenestrations along the entire length of the intimomedial flap (black arrows) (C).

specific image where submillimeter fenestrations can be identified (Figure 1C), which may influence decisions regarding stent graft length.

In addition, modern CT scanners allow for time-resolved imaging, where a small volume of tissue can be interrogated in real time, with the scanner moving the patient backward and forward over a short distance (15–25 cm) to assess for dynamic changes in contrast flow. Contrast can be imaged throughout the cardiac cycle in the arterial and delayed phase, allowing an angiographic rendering with significantly better soft tissue assessment and longer scan times, as well as relatively low doses of contrast (typically 50 mL) and radiation.

MAGNETIC RESONANCE IMAGING

Historically, magnetic resonance imaging (MRI) has not been widely utilized in aortic imaging due to the lack of robust protocols and adequate expertise for interpretation. MR angiography has evolved with the design of parallel imaging protocols, which allow simultaneous imaging of multiple slices of tissue, decreasing scan times by up to eight orders of magnitude.[3] A main benefit of MRI of the aorta is that it carries no dose penalty and therefore is the ideal screening and follow-up tool for patients requiring follow-up of aortic pathology for life (eg, those with connective tissue disorders) or for screening patients with risk factors for aortic pathology. Many of the examinations for screening and follow-up can be performed via MRI without the use of gadolinium contrast agents, as evaluation of only interval change in the size of the aorta is often required, and the soft tissue resolution and signal-to-noise ratio are more than adequate for answering these simple questions and are superior to noncontrast CT for basic follow-up. If there is any progression in the aortic disease, including an increase in size, the patient can then be imaged with contrast-enhanced CT for procedure planning.

Although MRI plays little or no role in the diagnosis of acute aortic syndromes because it is time consuming and requires specialist radiology training to develop protocols and interpret images, it does play an important role in problem solving for complex patients. The use of time-resolved MRI allows for the dynamic assessment of blood flow after the administration of gadolinium. This assessment often can be performed with half the dose of gadolinium required for conventional imaging and provides detailed information to the clinician on flow dynamics in the aorta.[4] As opposed to four-dimensional MR flow assessment, time-resolved MRI cannot quantify flow but does provide information on the direction of flow and enhancement. The temporal resolution of the imaging protocol can be adjusted where the scan volume is sampled more or less frequently depending on the question asked. This can be particularly helpful after stenting for dissection, where the direction and speed of flow into the false lumen can be of particular importance for patients in whom there is the question of antegrade flow in a false lumen after stenting (Figure 2) and earlier intervention would be preferred.

A second potential use of time-resolved MRI is for perfusion assessment of end organs, such as the kidneys, in patients with resistant hypertension after an aortic dissection. The study can be performed and the kidneys imaged throughout the arterial, corticomedullary, and excretory phases to assess for comparative enhancement of the kidneys and the presence of a delayed nephrogram, which would suggest altered perfusion to that kidney.

Delayed imaging of the aorta at 2 to 5 minutes following contrast administration allows for high-resolution imaging with a spatial resolution of 1 mm. Any slow, delayed filling of the false lumen, which may be missed on CT imaging traditionally at 90 seconds, can be evaluated using a fast breath-hold sequence or a high-resolution sequence. Delayed vascular imaging is referred to as *steady-state imaging* as the contrast reaches equilibrium in the arterial and venous systems.

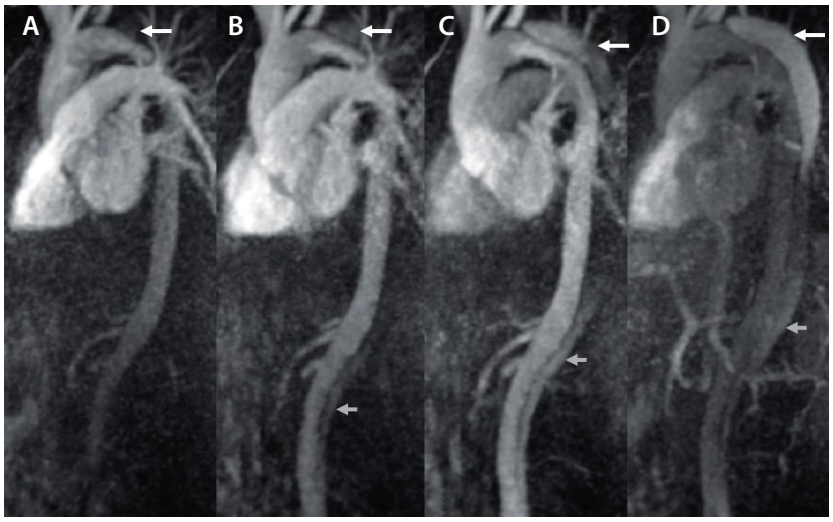


Figure 2. Time-resolved MR angiography demonstrating very early antegrade filling of the false lumen (arrow) (A), with further antegrade filling (B), and both antegrade and retrograde filling with altered signal in each due to the flow differential (C, D).

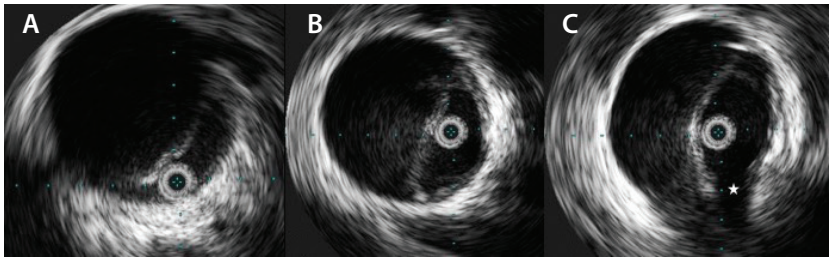


Figure 3. IVUS demonstrating the mobility of the intimomedial flap in systole with compression of the true lumen (A), diastole with expansion of the true lumen (B), and near-complete delamination of the intimomedial flap and extension into the left renal artery ostium (star) with a dynamic obstruction (C).

INTRAVASCULAR ULTRASOUND

Intravascular ultrasound (IVUS) provides high-resolution, real-time imaging of the vascular system and has become an increasingly recommended standard of care when treating aortic dissection. A number of vendors produce IVUS transducers with either radial 360° array transducers (Visions PV .035, Philips) or side facing transducers.

Each configuration has its unique aspects. The IVUS catheter, for instance, is very suitable for image-guided intervention and can be oriented and directed with the side-facing transducer, as the transducer provides a limited visual field in the direction of the transducer only. The operator can therefore turn the IVUS catheter in the direction of the intended intervention. If a 360° view is required, the operator is able to visualize the entire aorta on a single image; however, it is more difficult to direct the catheter.

The radial 360° array transducer allows for a circumferential view of the aorta (Figure 3). When the exact orientation of vessels is known, such as the visceral and renal vessels, the catheter can be appropriately oriented. The 360° array transducer is well suited to aortic imaging, as the entire aorta and dissection can be evaluated in each image in real time. The movement of the intimomedial flap can be evaluated (Figure 3A and 3B), particularly in dynamic obstruction of branch vessels (Figure 3C). The morphology of the flap can be assessed, and most importantly, the path of the wire can and should be assessed throughout to ensure that when stenting is performed, the stent is placed in the true lumen. Without IVUS guidance, it is possible to place the wire in the true lumen distally and for the wire to traverse multiple fenestrations into the false lumen and back into the true lumen without the knowledge of the operator, with potentially catastrophic consequences. The use of IVUS allows accurate navigation of the wire through the true lumen of the access vessel to the ascending aorta for stenting.

A further benefit of IVUS is the ability to measure the diameter of the aorta and lumina prior to stenting. This is of particular importance if the initial diagnostic images were not obtained using ultrahigh-pitch CT and motion may have precluded accurate measurement of the landing zone. The transducer used for aortic imaging is placed over a 0.035-inch wire and has a field of view of 5 cm in diameter. This catheter (Visions PV .035) requires a 9-F sheath for access and is a single-use item.

TRANSESOPHAGEAL ECHOCARDIOGRAPHY

TEE has been used extensively in diagnosing acute aortic pathology and can be used intraoperatively to aid in guidance of the wire in the true lumen. However, this is limited to the thoracic aorta and therefore the abdominal path of the wire cannot be assessed. This modality, with the increased use of IVUS, has become an adjunct imaging tool for the endovascular operator and also can be used to assess for flow in the false and true lumina following stenting, as some IVUS systems do not

have Doppler capability. TEE also has a role in the workup of acute aortic syndromes when the CT is equivocal, particularly in the diagnosis of type A dissection or when a type A dissection is suspected in patients with renal failure and iodinated contrast agents should be avoided.

CONCLUSION

Imaging has rapidly advanced and the ability to obtain high-resolution, accurate imaging of the entire aorta with little motion is not only possible but can be done without the need for ß blockade. It is necessary for any physician treating aortic dissection to become familiar with advanced imaging options and incorporate them into the workup, treatment, and follow-up protocols for patients with aortic dissection. ■

1. Hiratzka LF, Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with Thoracic Aortic Disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. Circulation. 2010;121:e266-e369.
2. Ueda T, Chin A, Petrovitch I, Fleischmann D. A pictorial review of acute aortic syndrome: discriminating and overlapping features as revealed by ECG-gated multidetector-row CT angiography. Insights Imaging. 2012;3:561-571.
3. Vessie EL, Liu DM, Forster B, et al. A practical guide to magnetic resonance vascular imaging: techniques and applications. Ann Vasc Surg. 2014;28:1052-1061.
4. Thakor AS, Chung J, Patel P, et al. Use of blood pool agents with steady-state MRI to assess the vascular system. J Magn Reson Imaging. 2017;45:1559-1572.

Darren Klass, MBChB, MD, MRCS, FCRC, FRCPC
Department of Radiology
Division of Interventional Radiology
University of British Columbia
Vancouver, British Columbia, Canada
darren.klass@vch.ca
Disclosures: Proctor, speaker, and consultant for Cook Medical.

Sponsored by Cook Medical

Controversies in Dissection Repair: Addressing Paraplegia

A review of this devastating complication including incidence rates after TEVAR for TBAD and the roles of left subclavian artery revascularization, aortic coverage, and cerebrospinal fluid drainage.

BY KEAGAN WERNER-GIBBINGS, MS, FRACS, AND BIJAN MODARAI, PHD, FRCS

Paraplegia is a devastating and unpredictable clinical syndrome that remains an important consideration in the management of type B aortic dissection (TBAD). Although spinal cord ischemia (SCI) can manifest as a de novo sequela of TBAD at presentation, it is encountered more frequently as a complication of both endovascular and open TBAD repair. Systematic reviews have suggested SCI rates of up to 4% for undifferentiated patients undergoing endovascular repair of TBAD[1,2]; however, significantly higher rates have been reported, especially in case series dealing with acute presentations.[3]

A complex interplay of factors impacts the likelihood of SCI complicating endovascular repair of TBAD. The blood supply of the spinal cord arises via a variety of different vascular territories, including the intercostal, lumbar, left subclavian, and internal iliac arteries.[4] Disruption of the blood flow from any of these territories reduces perfusion to spinal cord neural tissue, increasing the risk of SCI. As such, the extent of aorta covered during thoracic endovascular aortic repair (TEVAR), patency of the left subclavian artery (LSA) and internal iliac arteries, and perioperative blood pressure are just some of the factors that affect the periprocedural risk of SCI. Patients who develop paraplegia after TEVAR have a poor long-term functional outlook and significantly reduced life expectancy.[5]

COMPARATIVE PARAPLEGIA RATES AFTER TEVAR FOR TBAD AND ANEURYSMAL DISEASE

Published literature to date suggests that SCI rates after TEVAR for TBAD are lower than for TEVAR carried out to repair thoracic aneurysms, with rates of 4% reported for the former and up to 10% for the latter.[6,7] The EUROSTAR registry reported outcomes from 606 patients who underwent TEVAR, of whom 291 were treated for aneurysmal disease and 215 for TBAD.[8] Fourteen patients in this cohort experienced SCI postoperatively: 11 (3.8%) in the aneurysm group and three (1.4%) in the dissection group. The reasons for these reported disparities are likely multifactorial. The lower burden of mural atheroma and thrombus in TBAD presents a lower risk for atheroembolism after manipulation in the aorta. Another factor that may contribute to lower SCI rates in TBAD is that some false lumen perfusion often persists after TEVAR, either maintaining perfusion through the intercostal arteries or allowing time for collateralization. In contrast, the aneurysmal aorta is promptly sealed after TEVAR with rapid sac thrombosis and cessation of flow through the intercostal arteries before requisite collaterals have developed.

RISK OF PARAPLEGIA AFTER TREATMENT OF ACUTE AND CHRONIC TBAD

Treatment of TBAD in the acute phase, defined as within 2 weeks of presentation, is another significant risk factor for the development of SCI compared with TEVAR carried out for chronic TBAD. Case series reporting on the endovascular treatment of acute, complicated TBAD have demonstrated SCI rates as high as 15%.[9,10] These results are similar in registry data for thoracic devices, with rates of SCI in the treatment of acute complicated TBAD reaching 6% to 8%.[11,12] Multicenter studies have repeatedly demonstrated double the rate of parapalegia[13] and SCI[14] in acute versus chronic TBAD treatment. A meta-analysis from 2013 reported SCI risks of 1.5% associated with endovascular treatment of chronic TBAD and 4.2% after treatment of acute cases.[2] TEVAR in the acute phase is frequently carried out for complicated TBAD in a patient who is more likely to exhibit episodes of hypotension with consequent reduction in spinal cord perfusion and increased susceptibility to SCI. Another important factor dictating higher SCI rates may be the fact that patients who require TEVAR necessitating coverage of the LSA are less likely to have prophylactic LSA revascularization in the acute scenario.[9,15] Finally, delaying TEVAR to the chronic phase may allow intercostal collateralization and protect against SCI after aortic coverage.

LSA REVASCULARIZATION AFTER TEVAR FOR TBAD

A large proportion of TBADs originate at, or just distal to, the LSA; consequently, establishing an acceptable proximal seal zone in healthy aorta for TEVAR necessitates coverage of the LSA in these cases.[16] Given the contribution of this vessel to the blood supply of the spinal cord via the anterior spinal artery, it would seem logical to assume that LSA coverage without routine revascularization may influence spinal cord outcomes. Although multiple studies have aimed to address LSA management, conclusive evidence for a benefit associated with routine LSA revascularization prior to coverage remains elusive. A Cochrane review published in 2016 was unable to provide any guidance on this matter due to the lack of good-quality evidence.[17] Two meta-analyses have described the outcomes of LSA management in TBAD repair.[18,19] Both of these studies demonstrated trends toward higher paraplegia rates with coverage of the LSA. Revascularization of the LSA was associated with lower SCI rates, but these were nonsignificant trends and the studies collated were of relatively poor quality.

Studies incorporating TEVAR for both TBAD and aneurysmal pathology similarly present inconclusive outcomes related to LSA revascularization and effect on paraplegia rates. A 2018 meta-analysis reported lower SCI rates (4.7%) when the LSA was covered and revascularized, compared with when it was covered without revascularization (6.7%).[20] Likewise, two meta-analyses published in 2009 noted increased risks of SCI in patients who had LSA coverage without revascularization.[21,22] More recent collated evidence, however, suggests no difference in paraplegia rates after revascularization of the LSA.[23,24]

The Society for Vascular Surgery (SVS) and European Society for Vascular Surgery practice guidelines suggest revascularization of the LSA in all elective cases and expectant revascularization in the acute scenario, but both guidelines acknowledge a lack of quality evidence to support these recommendations.[25,26] Given the likelihood of reintervention after the index TEVAR for TBAD, however, it would seem prudent to adopt an aggressive LSA revascularization approach in stable patients to protect against future risk of paraplegia with interventions that will necessitate further aortic coverage. It is the authors' belief that total endovascular solutions, such as an off-the-shelf LSA branch, that facilitate routine LSA revascularization would be an invaluable adjunct for TBAD treatment.

LENGTH OF AORTIC COVERAGE

Extensive endografting of the thoracic aorta is associated with higher SCI rates, particularly with concomitant LSA coverage without revascularization.[8,27] Limiting the extent of aortic coverage at the index TEVAR for TBAD can, however, increase the likelihood of persistent false lumen flow and the need for secondary interventions.[28] Although some authors suggest a critical length of coverage of 150 mm, beyond which the risk of paraplegia significantly increases, it is more likely that this risk exists on a continuum, increasing as aortic coverage increases.[19] This point is noteworthy in light of current trends toward coverage of the entire thoracic aorta to the level of the celiac artery to promote false lumen thrombosis, improve aortic remodeling, and reduce long-term aneurysmal degeneration. This strategy, however, may come at the cost of higher paraplegia rates.

The Zenith Dissection endovascular system (Cook Medical) for the treatment of aortic dissection is a modular system consisting of a proximal component, the Zenith TX2 Dissection thoracic endovascular graft, and a distal component, the Zenith Dissection endovascular bare-metal stent. The stent graft covers the proximal entry tear, depressurizes the false lumen and redirects flow into the true lumen. The distal bare stent, extending below the visceral/renal arteries, expands the distal true lumen and stabilizes the intimal flap. This is known as the *PETTICOAT technique*. The STABLE I study, a multicenter experience with this technique, reported false lumen thrombosis in 59% of patients and was associated with an SCI rate of 2.5%.[29] STABLE II was a prospective study examining the PETTICOAT technique in acute, complicated TBAD and reported SCI in four (5.5%) patients.[30] Both studies reported SCI rates consistent with or slightly lower than those reported in other trials (6%–8%) and the SVS data set (9.4%).[30]

In our recently collated multicenter European experience of 121 patients treated for acute and chronic TBAD with the Zenith Dissection endovascular system (unpublished data), five (4.1%) patients developed paraplegia. All cases of paraplegia were in patients treated acutely. There were no instances of paraplegia in the 34 patients in whom the PETTICOAT technique was used. In this cohort, the length of covered stent graft used was shorter than in the remaining 87 patients who did not have concomitant use of the Zenith Dissection endovascular bare-metal stent.

THE ROLE OF CEREBROSPINAL FLUID DRAINAGE

Perioperative cerebrospinal fluid (CSF) drainage has been shown to reduce paraplegia after open thoracoabdominal aortic surgery.[31] Its role during TEVAR is less clear. A recent meta-analysis demonstrated a modest benefit for prophylactic CSF drainage for endovascular treatment of thoracic and thoracoabdominal pathology associated with a reduction in SCI rate from 2.5% to 1.5%.[32] It was

noted that patients in whom spinal drains are placed as a rescue measure in an attempt to reverse paraplegia have significantly worse outcomes than those in whom prophylactic drainage is instituted. Up to 20% of rescue cases were discharged with residual neurological impairment.

CSF drainage carries a small but significant risk of complications including epidural hematoma, intracranial hemorrhage, infection, and catheter fracture/retention, hence it should be considered in select cases where TEVAR is deemed to be associated with a high risk of paraplegia. These include cases treated in the acute phase in which coverage of the entire thoracic aorta is planned and the collateral supply to the spinal cord is also impaired, for example because one of the internal iliac arteries is occluded. An additional role for CSF drainage in the future may be to facilitate continuous sampling of spinal fluid for measuring biomarkers that herald the onset of SCI, but this concept remains a subject of research at present and is not in clinical use.[33]

Near-infrared spectroscopy, a technique that measures blood flow to the paraspinous musculature, can also herald SCI and may be a useful adjunct that dictates the duration of CSF drainage in cases that are high risk for paraplegia.[34]

CONCLUSION

Although the risk of paraplegia after TEVAR for TBAD is lower than that associated with repair of degenerative aneurysms, the incidence of this devastating complication remains significant. Given the need for extensive aortic coverage in the majority of patients, ensuring flow through the collateral spinal circulation and judicious use of adjuncts, such as CSF drainage, are important in this cohort. ■

1. Thrumurthy SG, Karthikesalingam A, Patterson BO, et al. A systematic review of mid-term outcomes of thoracic endovascular repair (TEVAR) of chronic type B aortic dissection. Eur J Vasc Endovasc Surg. 2011;42:632-647.
2. Fattori R, Cao P, De Rango P, et al. Interdisciplinary expert consensus document on management of type B aortic dissection. J Am Coll Cardio. 2013;61:1661-1678.
3. Ehrlich MP, Dumfarth J, Schoder M, et al. Midterm results after endovascular treatment of acute, complicated type B aortic dissection. Ann Thorac Surg. 2010;90:1444-1448.
4. Czerny M, Eggebrecht H, Sodeck G, et al. Mechanisms of symptomatic spinal cord ischemia after TEVAR: insights from the European Registry of Endovascular Aortic Repair Complications (EuREC). J Endovasc Ther. 2012;19(1):37-43.
5. DeSart K, Scali ST, Feezor RJ, et al. Fate of patients with spinal cord ischemia complicating thoracic endovascular aortic repair. J Vasc Surg. 2013;58:635-642.e2.
6. Khoynezhad A, Donayre CE, Bui H, et al. Risk factors of neurologic deficit after thoracic aortic endografting. Ann Thorac Surg. 2007;83:S882-S889; discussion S890-S892.
7. Patterson B, Holt P, Nienaber C, et al. Aortic pathology determines midterm outcome after endovascular repair of the thoracic aorta: report from the Medtronic Thoracic Endovascular Registry (MOTHER) database. Circulation. 2013;127:24-32.
8. Buth J, Harris PL, Hobo R, et al. Neurologic complications associated with endovascular repair of thoracic aortic pathology: incidence and risk factors. A study from the European Collaborators on Stent/Graft Techniques for Aortic Aneurysm Repair (EUROSTAR) registry. J Vasc Surg. 2007;46:1103-1111.
9. Sobocinski J, Dias NV, Berger L, et al. Endograft repair of complicated acute type B aortic dissections. Eur J Vasc Endovasc Surg. 2013;45:468-474.
10. Scali ST, Wang SK, Feezor RJ, et al. Preoperative prediction of spinal cord ischemia after thoracic endovascular aortic repair. J Vasc Surg. 2014;60:1481-1490.e1.
11. Bavaria JE, Brinkman WT, Hughes GC, et al. Outcomes of thoracic endovascular aortic repair in acute type B aortic dissection: results from the Valiant United States investigational device exemption study. Ann Thorac Surg. 2015;100:802-809.
12. Cambria RP, Conrad MF, Matsumoto AH, et al. Multicenter clinical trial of the conformable stent graft for the treatment of acute, complicated type B dissection. J Vasc Surg. 2015;62:271-278.
13. Eggebrecht H, Nienaber CA, Neuhäuser M, et al. Endovascular stent-graft placement in aortic dissection: a meta-analysis. Eur Heart J. 2005;27:489-498.
14. Wang GJ, Cambria RP, Lombardi JV, et al. Thirty-day outcomes from the Society for Vascular Surgery Vascular Quality Initiative thoracic endovascular aortic repair for type B dissection project. J Vasc Surg. 2019;69:680-691.
15. White RA, Miller DC, Criado FJ, et al; for the Multidisciplinary Society for Vascular Surgery Outcomes Committee. Report on the results of thoracic endovascular aortic repair for acute, complicated, type B aortic dissection at 30 days and 1 year from a multidisciplinary subcommittee of the Society for Vascular Surgery Outcomes Committee. J Vasc Surg. 2011;53:1082-1090.
16. Jia X, Guo W, Li T, et al. The results of stent graft versus medication therapy for chronic type B dissection. J Vasc Surg. 2013;57:406-414.
17. Hajibandeh S, Hajibandeh S, Antoniou SA, et al. Revascularisation of the left subclavian artery for thoracic endovascular aortic repair. Cochrane Database of Syst Rev. 2016;4:CD011738.
18. Sobocinski J, Patterson BO, Karthikesalingam A, Thompson MM. The effect of left subclavian artery coverage in thoracic endovascular aortic repair. Ann Thorac Surg. 2016;101:810-817.
19. Rehman SM, Vecht JA, Perera R, et al. How to manage the left subclavian artery during endovascular stenting for thoracic aortic dissection? An assessment of the evidence. Ann Vasc Surg. 2010;24:956-965.
20. Huang Q, Chen XM, Yang H, et al. Effect of left subclavian artery revascularisation in thoracic endovascular aortic repair: a systematic review and meta-analysis. Eur J Vasc Endovasc Surg. 2018;56:644-651.
21. Cooper DG, Walsh SR, Sadat U, et al. Neurological complications after left subclavian artery coverage during thoracic endovascular aortic repair: a systematic review and meta-analysis. J Vasc Surg. 2009;49:1594-1601.
22. Rizvi AZ, Murad MH, Fairman RM, et al. The effect of left subclavian artery coverage on morbidity and mortality in patients undergoing endovascular thoracic aortic interventions: a systematic review and meta-analysis. J Vasc Surg. 2009;50:1159-1169.
23. Maldonado TS, Dexter D, Rockman CB, et al. Left subclavian artery coverage during thoracic endovascular aortic aneurysm repair does not mandate revascularization. J Vasc Surg. 2013;57:116-124.
24. Hajibandeh S, Hajibandeh S, Antoniou SA, et al. Meta-analysis of left subclavian artery coverage with and without revascularization in thoracic endovascular aortic repair. J Endovasc Ther. 2016;23:634-641.
25. Riambau V, Böckler D, Brunkwall J, et al. Editor's choice--management of descending thoracic aorta diseases: clinical practice guidelines of the European Society for Vascular Surgery (ESVS). Eur J Vasc Endovasc Surg. 2017;53:4-52.
26. Matsumura JS, Lee WA, Mitchell RS, et al. The Society for Vascular Surgery Practice Guidelines: management of the left subclavian artery with thoracic endovascular aortic repair. J Vasc Surg. 2009;50:1155-1158.
27. Feezor RJ, Martin TD, Hess Jr PJ, et al. Extent of aortic coverage and incidence of spinal cord ischemia after thoracic endovascular aneurysm repair. Ann Thorac Surg. 2008;86:1809-1814; discussion 1814.
28. Eggebrecht H, Herold U, Kuhnt O, et al. Endovascular stent-graft treatment of aortic dissection: determinants of post-interventional outcome. Eur Heart J. 2005;26:489-497.
29. Lombardi JV, Cambria RP, Nienaber CA, et al; for the STABLE I Investigators. Five-year results from the Study of Thoracic Aortic Type B Dissection Using Endoluminal Repair (STABLE I) study of endovascular treatment of complicated type B aortic dissection using a composite device design. J Vasc Surg. 2019;70:1072-1081.
30. Lombardi JV, Gleason TG, Panneton JM, et al. RS17. STABLE II clinical trial on endovascular treatment of acute, complicated type B aortic dissection with a composite device design. J Vasc Surg. 2016;63(supp):149S.
31. Coselli JS, LeMaire SA, Köksoy C, et al. Cerebrospinal fluid drainage reduces paraplegia after thoracoabdominal aortic aneurysm repair: results of a randomized clinical trial. J Vasc Surg. 2002;35:631-639.
32. Suarez-Pierre A, Zhou X, Gonzalez JE, et al. Association of preoperative spinal drain placement with spinal cord ischemia among patients undergoing thoracic and thoracoabdominal endovascular aortic repair. J Vasc Surg. 2019;70:393-403.
33. Choong AMTL, Wee IJY, Almond M, et al. A systematic review of the use of biochemical markers in the assessment of spinal cord ischemia in thoracoabdominal aortic aneurysm repair. Vasc Endovasc Surg. 2019;53:230-241.
34. Boezeman RP, van Dongen EP, Morshuis WJ, et al. Spinal near-infrared spectroscopy measurements during and after thoracoabdominal aortic aneurysm repair: a pilot study. Ann Thorac Surg. 2015;99:1267-1274.

Keagan Werner-Gibbings, MS, FRACS
Senior Fellow
Department of Vascular Surgery
St Thomas' Hospital
King's Health Partners
London, United Kingdom
Disclosures: None.

Bijan Modarai, PhD, FRCS
Professor of Vascular Surgery
Academic Department of Vascular Surgery
School of Cardiovascular Medicine and Sciences
BHF Centre of Research Excellence
St Thomas' Campus
King's College London and King's Health Partners
London, United Kingdom
bijan.modarai@kcl.ac.uk
Disclosures: Proctor, speaker, and consultant for Cook Medical; advisory board for Cydar Medical.

Sponsored by Cook Medical

Building a Multidisciplinary Aortic Center

Five key components to providing comprehensive care for patients with aortic dissections.

BY SUKGU HAN, MD, MS, AND FERNANDO FLEISCHMAN, MD

Aortic dissections make up a significant portion of aortic emergencies that can be rapidly fatal. Time-dependent mortality and morbidity associated with aortic dissections highlight the importance of prompt, accurate evaluation and treatment for successful patient outcomes. Recent advances in endovascular technology have enabled a wide array of less invasive therapeutic options for aortic dissections. These rapidly evolving endovascular options may result in further expansion of indications for intervention. Recent approval of the Zenith Dissection stent system (Cook Medical) is an example of pathology-specific devices with potential to significantly impact the natural history of aortic dissections. Consequently, optimal care of patients with aortic dissections requires multidisciplinary expertise in both open and endovascular repairs at centers with established resources and infrastructure to offer rapid treatment.[1-3]

To improve care for these patients, dedicated aortic centers have begun to emerge. The ability to offer complex open ascending and arch reconstructions for type A aortic dissections, as well as expertise in endovascular therapy in arch, descending, and thoracoabdominal aortic segments for type B aortic dissections, is essential for such centers to be truly comprehensive. At most centers, this skill set resides across cardiac and vascular surgery specialties. Furthermore, synergistic partnership of cardiac and vascular surgeons who are dedicated to aortic pathologies is only the first element of forming a successful aortic center. There are five additional key components that we have found to be helpful in building a true cross-disciplinary aortic program at the Keck Medical Center. They include (1) development of dedicated aortic expertise, (2) establishment of a dedicated "aortic hotline" and rapid transport system, (3) raising regional awareness through outreach efforts, (4) development of multidisciplinary aortic case conferences, and (5) development of a multidisciplinary follow-up clinic and surveillance program. In addition to describing these five components, we aim to share our experience in overcoming common roadblocks to a successful, truly collaborative partnership.

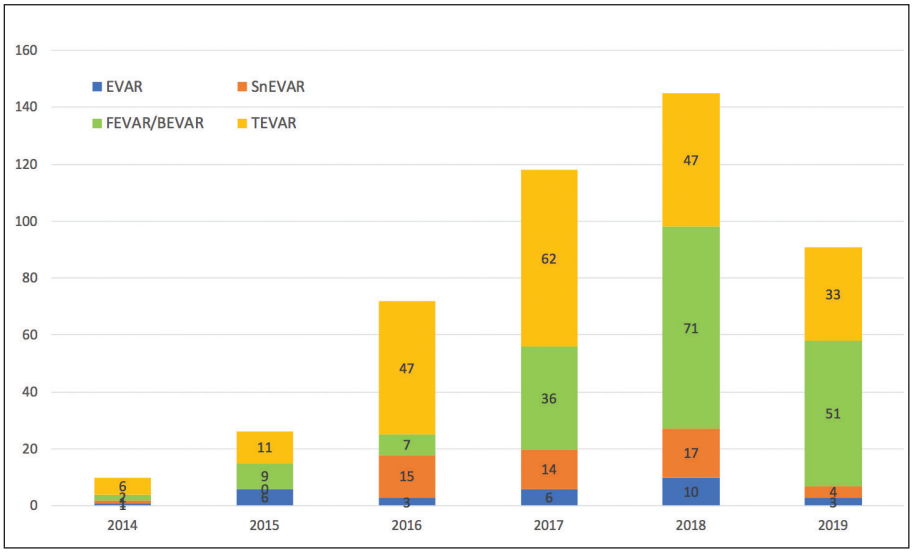


Figure 1. Growth in endovascular aortic experience in a single dedicated complex endovascular aortic surgeon by year, achieved through a shared concentrated experience model (year 2019 includes 6-month period). BEVAR, branched endovascular aneurysm repair*; FEVAR, fenestrated endovascular aneurysm repair; SnEVAR, snorkel endovascular aneurysm repair.

* These treatment options may not be available in all regions.

1. DEVELOPMENT OF DEDICATED AORTIC EXPERTISE

Implementing a "Shared Concentrated Experience" Model

Overcoming the learning curve to reach expertise in both complex open and

endovascular aneurysm repairs (EVARs) takes time and experience. When our aortic center was starting in 2012, we were performing simple thoracic endovascular aortic repairs (TEVARs), limited hybrid arch reconstructions, and infrarenal EVARs. Particularly, endovascular branch incorporations were seldom performed. Recognizing the need to rapidly develop expertise in these areas, the vascular surgery division assigned a single surgeon with a strong focus in complex endovascular aortic procedures to be the dedicated specialist. All partners within the division funneled cases to this specialist, thereby concentrating our collective experience into this surgeon, who then became the champion for cutting-edge technology and advanced endovascular techniques. Every complex EVAR was performed with the referring surgeon and the dedicated specialist surgeon as cosurgeons. This strategy of concentrating experience to support the initial development of expertise led to a rapid accumulation of case volume along with the skill set, which was then shared with the rest of the group (Figure 1).

An exponential increase in case volume of type A aortic dissections* has allowed our open arch experience to grow and mature. Expertise in arch reconstruction involves multiple skill sets obtained through repetition. These cases may not be part of a typical cardiac surgery training experience and therefore are often beyond the scope of most cardiac surgeons' expertise. The Society of Thoracic Surgeons reported that only 20% of centers perform more than five type A repairs annually.[4] Better outcomes associated with high-volume centers have been demonstrated in TEVAR, with 40 or more cases per year resulting in superior patient outcomes.[5] By comparison, we perform approximately 80 type A repairs per year, and our graduates finish their training with 25 cases. Refinement of technical skill as well as improved knowledge of multiple perfusion models and a dissection-specific critical care protocol resulted in improved patient outcomes. Developing expertise in treating patients with type A aortic dissection has expanded our willingness to offer open repair to more patients with challenging anatomy and/or a high-risk comorbidity profile. Cases that were once sent away to specialized centers are now referred to our center for repair.

Dedicated Training

The dedicated specialist surgeon then spent 3 months of sabbatical at another high-volume center to further refine technical skills and the workflow of complex endovascular aortic procedures. This translated to an immediate improvement in technical efficiency and operating room workflow during complex EVAR, as evidenced by the increased number of target vessels, while decreasing total fluoroscopy time, and contrast used (Figure 2).

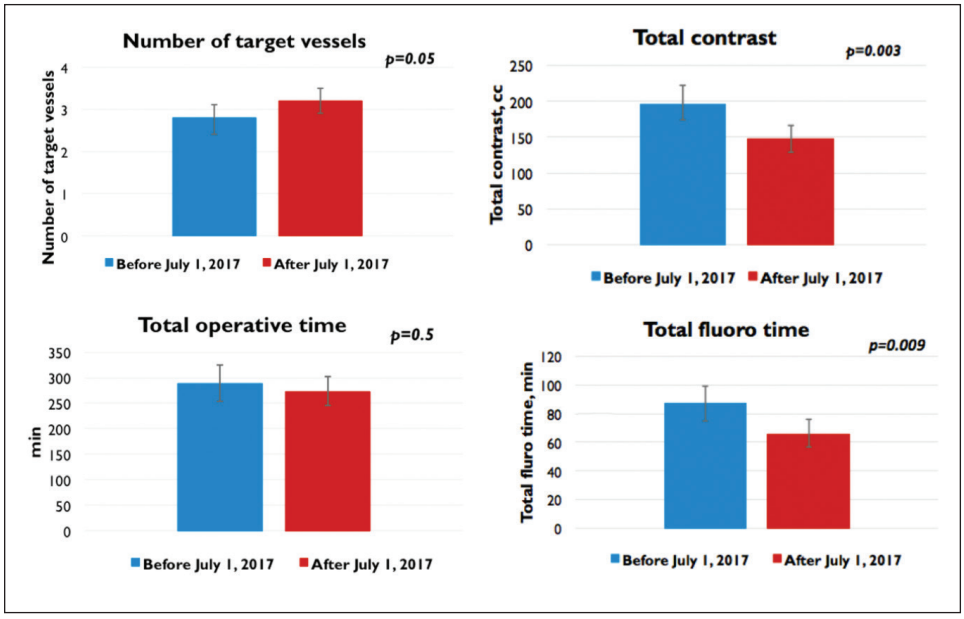


Figure 2. Impact of dedicated additional training in complex EVAR. We were able to increase the number of target vessels during complex EVAR, while reducing the amount of contrast and the fluoroscopy time.

*Cook Zenith Dissection system is not indicated for use in type A dissections.

2. DEDICATED AORTIC HOTLINE AND RAPID TRANSPORT SYSTEM

We aimed to develop a system that could overcome the delay caused by the multitude of logistical steps involved in a typical transfer process of aortic dissection patients. A rapid transport system dedicated to aortic emergencies is our solution to this issue. The transport team consists of an aortic hotline operator, nurse practitioner, administrative assistants, and on-call vascular and cardiac surgeons. A call schedule specific to aortic emergencies is shared among vascular and cardiac surgeons. All calls

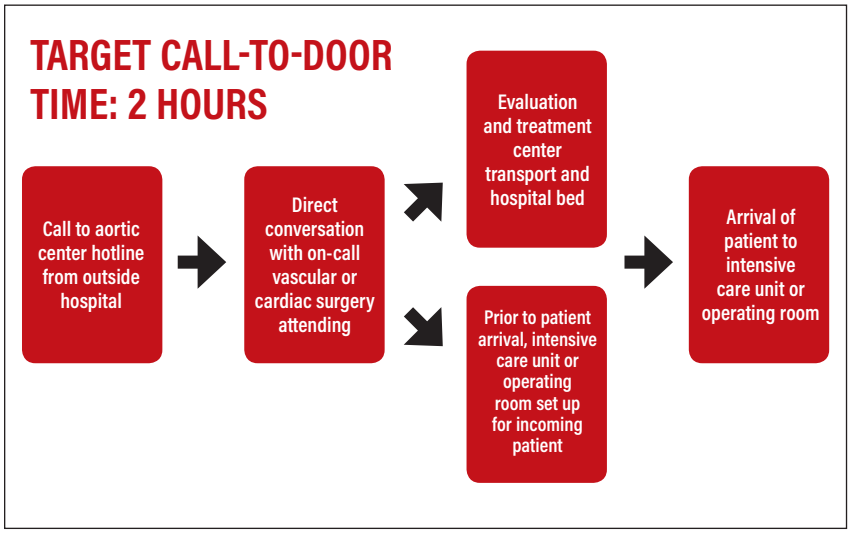


Figure 3. Workflow of our aortic hotline and rapid transport system.

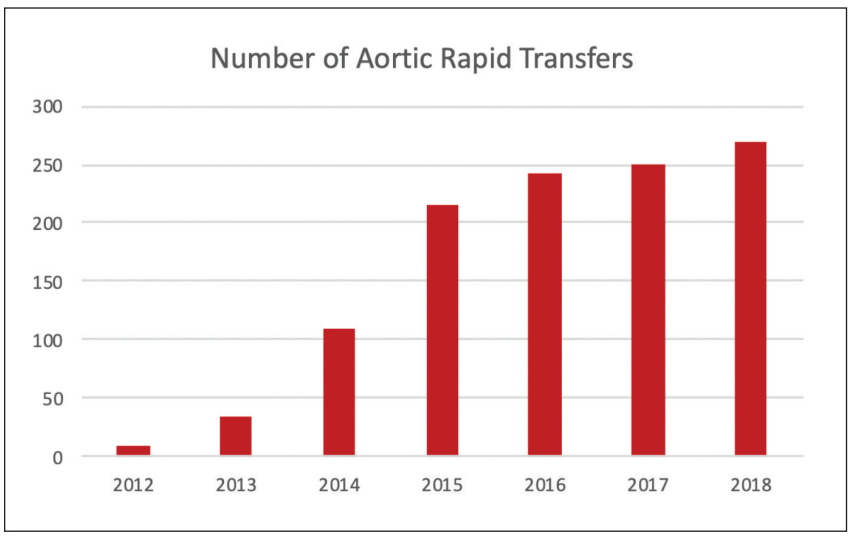


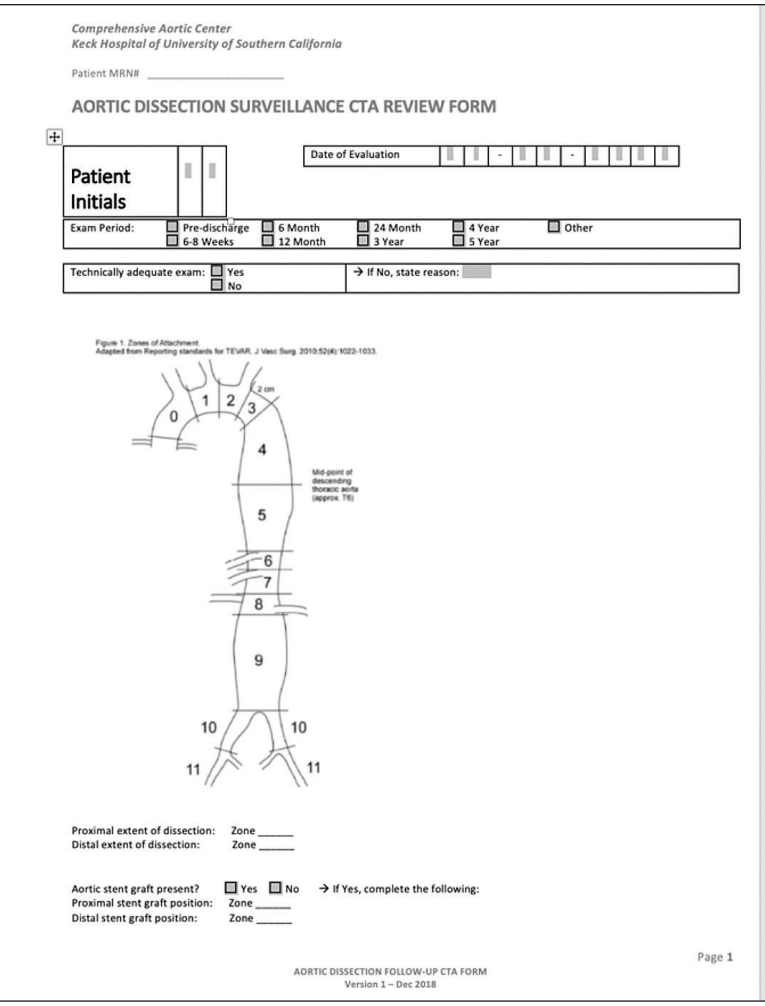
Figure 4. Growth of our aortic emergency case volume by year.

received by the aortic center hotline are immediately evaluated by the on-call cardiac or vascular surgeon. Following the surgeon's acceptance for transfer, the administrative assistant arranges land or air transport. Mode of transport is primarily determined on the basis of the distance from the referring center to ours and also by acuity. While the patient is en route, the accepting surgeon and administrative assistant activate the intensive care unit team as well as the operating room team, which includes the on-call cardiac anesthesiologist and, depending on the incoming emergency, on-call perfusionist or radiology technologists. On arrival, all patients are directly admitted to the intensive care unit, where the patient is assessed and the need for operation is rapidly determined. This system is designed with the goal of transferring any patient with an acute aortic emergency from a sending facility to the Keck Hospital intensive care unit within a 2-hour time frame (Figure 3).

This approach differs from a typical transfer process in three ways. First, the call bypasses the on-call house officer, who may be busy with inpatient issues or may be operating when these aortic emergency transfer requests are received. Having direct access to the on-call attending who can make immediate decisions facilitates expeditious treatment of these patients. Second, a dedicated aortic call schedule for the attending surgeons that is entirely separate from the general call schedule improves the attending response time by reducing the chance of the on-call attending being inundated with other emergencies. Third, we have contracted with ambulance and helicopter services, which enables immediate arrangement of transportation. The onus of arranging for transportation is lifted from the referring hospitals. Implementation of this system combined with outreach efforts, as outlined in the following section, have resulted in significant growth in the number of aortic emergency cases we have received (Figure 4).

3. RAISING REGIONAL AWARENESS THROUGH OUTREACH

The goal of our outreach was improving awareness of the importance of rapid and accurate diagnosis of aortic dissections in the emergency department setting, as well as the availability of the previously mentioned aortic hotline and rapid transport system. As part of our outreach, we focused on small- to medium-size emergency departments, as we thought these centers were most in need. These centers often do not have readily available access to cardiac or vascular surgeons who routinely handle aortic emergencies. We also found that more centers had vascular on-call services but lacked cardiac on-call services.



Comprehensive Aortic Center
Keck Hospital of University of Southern California

Patient MRN# _____

AORTIC DISSECTION SURVEILLANCE CTA REVIEW FORM

Patient Initials

Date of Evaluation

Exam Period: ☐ Pre-discharge ☐ 6-8 Weeks ☐ 6 Month ☐ 12 Month ☐ 24 Month ☐ 3 Year ☐ 4 Year ☐ 5 Year ☐ Other

Technically adequate exam: ☐ Yes ☐ No → If No, state reason:

Figure 1. Zones of Attachment.
Adapted from Reporting standards for TEVAR. J Vasc Surg. 2010;52(4):1022-1033.

Proximal extent of dissection: Zone _____
Distal extent of dissection: Zone _____

Aortic stent graft present? ☐ Yes ☐ No → If Yes, complete the following:
Proximal stent graft position: Zone _____
Distal stent graft position: Zone _____

AORTIC DISSECTION FOLLOW-UP CTA FORM
Version 1 – Dec 2018

Page 1

Comprehensive Aortic Center
Keck Hospital of University of Southern California

Patient MRN# _____

AORTIC DISSECTION SURVEILLANCE CTA REVIEW FORM

**** All diameter measurements should be performed orthogonal to the flow lumen true centerline reconstruction*

ASCENDING AORTA

Maximum transaortic centerline diameter (at Sinus of Valsalva):		mm
Total volume:		mm^3

AORTIC ARCH

Maximum transaortic centerline diameter:		mm
Total volume:		mm^3

DESCENDING THORACIC AORTA

Maximum transaortic centerline diameter:		mm
Total volume:		mm^3

ABDOMINAL AORTA

Maximum transaortic centerline diameter:		mm
Total volume:		mm^3
: mm		

ILIACS

Maximum right common iliac diameter:		mm
Maximum left common iliac diameter:		mm

Reviewer Name and Signature

___/___/_____
Date (mm/dd/yyyy)

AORTIC DISSECTION FOLLOW-UP CTA FORM
Version 1 – Dec 2018

Page 2

Figure 5. Standardized form for our aortic dissection imaging surveillance program.

Differentiating between ascending, arch, or descending aortic pathologies was often difficult, resulting in misdiagnosis and mistreatment. Therefore, we found that emergency department physicians were very eager to incorporate our services into their algorithm. In our experience, center or regional politics seldom prevented engagement.

4. DEVELOPMENT OF MULTIDISCIPLINARY AORTIC CENTER CONFERENCES

Members of aortic center conferences include vascular surgeons, cardiac surgeons, cardiovascular radiologists, dedicated nurse practitioners, and aortic research fellows. The conferences are centered around case discussions, where management options and surgical plans are formulated. This conference has also been a useful venue for discussing new technology and reviewing the latest literature affecting the treatment algorithm of aortic pathologies.

5. DEVELOPMENT OF A MULTIDISCIPLINARY FOLLOW-UP CLINIC AND SURVEILLANCE PROGRAM

Beyond the acute phase of aortic dissections, patients should be followed over their lifetime with serial surveillance imaging. Regardless of the treatment they underwent during the acute phase (medical, open surgical, or endovascular repair), a systematic surveillance program serves as an integral part of an aortic center to optimize the long-term outcomes. Growing evidence suggests that a significant portion of patients develop abdominal aneurysmal degeneration in the long term after TEVAR for aortic dissections.[6] Detection of early signs of failure or negative remodeling should trigger an individualized treatment plan for each patient to prevent catastrophic aortic events. A multidisciplinary follow-up clinic where patients can be seen by vascular and cardiac surgeons, as well

as antihypertension specialists, offers convenient, comprehensive care. In our experience, this patient-centered arrangement also improves patient satisfaction and compliance with the surveillance program.

Additionally, we have implemented a standardized aortic image surveillance program whereby the dedicated cardiovascular radiologists perform centerline and volume measurements in different aortic segments (Figure 5). These measurements provide a consistent tool to detect aortic dilatation.

COMMON ROADBLOCKS

Challenges to forming a truly collaborative partnership lie more often in the physicians rather than the patients. Concern for not receiving due credit (financial or otherwise) during a joint case, disagreement regarding treatment plan, as well as the desire to keep the skill set siloed are some of the common roadblocks and are rarely in the patients' interests. Sentiments such as, "This is my patient," "I should get the credit," "They don't have the skill set," "I am the real surgeon," and "Why should I teach them?" are counterproductive to the optimal care of patients with aortic dissections.

Overcoming these roadblocks requires recognition of overlapping pathologies that can be treated using different approaches, the value of nonoverlapping skill sets offered by different disciplines, and finally the willingness to share and learn from each other with the common goal of achieving the best possible patient outcome. At the outset of our aortic center, we formed an agreement that every TEVAR will be performed with both cardiac and vascular surgeons scrubbed. The primary attending in charge of the patient undergoing TEVAR is the primary surgeon billing for the case, while the counterpart bills for the adjunctive procedures such as branch stenting, and as an assistant for the index TEVAR. With this agreement and the robust growth of our total volume, financial concerns regarding these cases disappeared. Additionally, this approach more than doubled the TEVAR experience for our trainees in cardiac and vascular programs. Going through challenging cases and managing complications together, we saw mutual respect and a collaborative spirit grow and solidify.

CONCLUSION

Aortic dissections can be challenging to manage because of the complex pathology that patients can present, as well as the wide array of treatment options. Management of aortic dissections continues to evolve with rapid technical and technological refinement. As such, optimal care of aortic dissection patients involves a multidisciplinary approach with expertise in medical, open surgical, and endovascular treatments. A successful aortic center begins with a synergistic partnership between multidisciplinary specialists dedicated to aortic care, with vascular and cardiac surgeons as "co-captains" of the team. With this foundation, the five components discussed in this article to ensure the success of an aortic center can be implemented to deliver optimal care to aortic dissection patients. With growth of the aortic center and collective experience, mutual respect will solidify and help create a truly collaborative partnership. ■

1. Vilacosta I, San Román JA. Acute aortic syndrome. Heart. 2001;85:365-368.
2. Tsai TT, Nienaber CA, Eagle KA. Acute aortic syndromes. Circulation. 2005;112:3802-3813.
3. Manzur M, Han SM, Dunn J, et al. Management of patients with acute aortic syndrome through a regional rapid transport system. J Vasc Surg. 2017;65:21-29.
4. D'Agostino RS, Jacobs JP, Badhwar V, et al. The Society of Thoracic Surgeons Adult Cardiac Surgery Database: 2018 update on outcomes and quality. Annals Thorac Surg. 2018;105:15-23.
5. Gazoni LM, Speir AM, Kron IL, et al. Elective thoracic aortic aneurysm surgery: better outcomes from high-volume centers. J Am Coll Surg. 2010;210:855-859.
6. Famularo M, Meyermann K, Lombardi JV. Aneurysmal degeneration of type B aortic dissections after thoracic endovascular aortic repair: a systematic review. J Vasc Surg. 2017;66:924-930.

Sukgu Han, MD, MS
Co-director to the Comprehensive Aortic Center
Keck Medical Center
University of Southern California
Division of Vascular Surgery and Endovascular Therapy
University of Southern California
Los Angeles, California
sukgu.han@med.usc.edu
Disclosures: Proctor, speaker, and consultant for Cook Medical; consultant for WL Gore and Associates.

Fernando Fleischman, MD
Co-director to the Comprehensive Aortic Center
Keck Medical Center
University of Southern California
Division of Cardiothoracic Surgery
University of Southern California
fernando.fleischman@med.usc.edu
Disclosures: Proctor, speaker, and consultant for Cook Medical; consultant and principal investigator for WL Gore and Associates; consultant and principal investigator for Terumo/Bolton.

Zenith® Dissection Endovascular System (Zenith® TX2® Dissection Endovascular Graft with Pro-Form® and Zenith® Dissection Endovascular Stent)

CAUTION: U.S. federal law restricts this device to sale by or on the order of a physician (or a properly licensed practitioner).

INTENDED USE: The Zenith Dissection Endovascular System (Zenith TX2 Dissection Endovascular Graft with Pro-Form and Zenith Dissection Endovascular Stent) is indicated for the endovascular treatment of patients with Type B aortic dissection. The Zenith TX2 Dissection Endovascular Graft with Pro-Form is intended to seal the entry tears and to exclude aneurysms associated with chronic dissections. The Zenith Dissection Endovascular Stent is intended to be used as a distal component to provide support to delaminated segments of non-aneurysmal aorta with dissection distal to a Zenith TX2 Dissection Endovascular Graft with Pro-Form. The system is indicated for use in patients having vascular anatomy suitable for endovascular repair, (**Fig. 6 in the complete INSTRUCTIONS FOR USE**) including: • Adequate iliac/femoral access compatible with the required introduction systems • For the Zenith TX2 Dissection Endovascular Graft with Pro-Form: – Non-dissected/aneurysmal aortic segments (fixation sites) distal to the left common carotid artery and proximal to the entry tear with a length of at least 20 mm, – Non-dissected/aneurysmal aortic segments (fixation sites) distal to the left common carotid artery and proximal to the entry tear with a diameter (measured outer-wall-to-outer-wall) of no greater than 38 mm and no less than 20 mm, and • For the Zenith Dissection Endovascular Stent: – Diameter at non-aneurysmal intended implant site (measured outer-wall-to-outer-wall) of no greater than 38 mm (true lumen) and no less than 20 mm (total aortic diameter).

CONTRAINDICATIONS: The Zenith TX2 Dissection Endovascular Graft with Pro-Form and the Zenith Dissection Endovascular Stent are contraindicated in: • Patients with known sensitivities or allergies to stainless steel, polyester, polypropylene, nitinol or gold. • Patients with a systemic infection who may be at increased risk of endovascular graft/stent infection.

WARNINGS AND PRECAUTIONS:

General: Read all instructions carefully. Failure to properly follow the instructions, warnings, and precautions may lead to serious consequences or injury to the patient. • DO NOT place the device in a dissected proximal landing zone. Placement of the device has resulted in proximal post-treatment dissection events (retrograde progression of pre-existing or new Type A dissection) when the dissection extends proximal to the LSA or the proximal landing zone is dissected. • Always have a qualified surgery team available during implantation or reintervention procedures in the event that conversion to open surgical repair is necessary. • The Zenith TX2 Dissection Endovascular Graft with Pro-Form and the Zenith Dissection Endovascular Stent should only be used by physicians and teams trained in vascular interventional techniques (catheter-based and surgical) and in the use of this device. Specific training expectations are described in **Section 10.1, Physician Training, in the complete INSTRUCTIONS FOR USE**. • Additional/adjunctive endovascular and/or surgical interventions may be required to treat Type B dissections, including conversion to standard open surgical repair following initial endovascular repair should patients experience continued flow in the false lumen of the dissection which may lead to rupture. Further intervention should be considered for patients exhibiting compromise of organ vessel flow, or inadequate seal/fixation length proximal to the dissection.

Patient Selection, Treatment and Follow-Up: Access vessel diameter (measured inner-wall to inner-wall) and morphology (tortuosity, occlusive disease, and/or calcification) should be compatible with vascular access techniques and introduction systems of the profile of a 20 French (7.7 mm OD) or 22 French (8.5 mm OD) vascular introducer sheath as is used for the Zenith Dissection Endovascular Graft, compared to 16 French (6.0 mm OD) for the Zenith Dissection Endovascular Stent. Vessels that are significantly calcified, occlusive, tortuous or thrombus-lined may preclude femoral introduction of the endovascular graft and/or may increase the risk of embolization. • **The Zenith TX2 Dissection Endovascular Graft with Pro-Form:** Key anatomic elements that may affect successful exclusion of the dissection entry tear include severe angulation (radius of curvature < 35 mm and localized angulation > 45 degrees); short proximal fixation site (< 20 mm of non-dissected aorta); necks > 38 mm or < 20 mm; an inverted funnel shape at the proximal fixation site (greater than 10% increase in diameter over 20 mm of fixation site length); and circumferential thrombus and/or calcification at the arterial fixation sites. Irregular calcification and/or plaque may compromise the attachment and sealing at the fixation site. Necks exhibiting these key anatomic elements may be more conducive to graft migration and or loss of seal. • **The Zenith Dissection Endovascular Stent:** Key anatomic elements that may affect successful treatment of dissection include severe angulation (radius of curvature < 35 mm and localized angulation > 45 degrees) and aortic true lumen diameters > 38 mm or total aortic (true lumen plus false lumen) diameter < 20 mm. • The safety and effectiveness of the Zenith TX2 Dissection Endovascular Graft with Pro-Form and the Zenith Dissection Endovascular Stent have not been evaluated in the following patient populations: – chronic Type B dissections – acute, uncomplicated Type B dissection – allergy to stainless steel, nitinol, polyester, polypropylene, or gold – bowel necrosis – ASA class V – diagnosed or suspected genetic connective tissue disease (e.g., Marfans or Ehlers-Danlos Syndrome) – females who are pregnant, breastfeeding, or planning to become pregnant within 60 months – patients less than 18 years of age – systemic infection (e.g., sepsis) – previous placement of thoracic endovascular graft – prior open repair involving descending thoracic aorta (including suprarenal aorta and/or arch) – surgical or endovascular AAA repair within 30 days before or after dissection repair – bleeding diathesis, uncorrectable coagulopathy, or refuses blood transfusion – hemorrhagic stroke within 30 days (or 14 days for embolic stroke) – untreatable reaction to contrast, which cannot be adequately premedicated – inability to preserve the native left common carotid artery and celiac artery origins – if occlusion of the left subclavian artery ostium is required to obtain adequate neck length for fixation and sealing, transposition or bypass of the left subclavian artery may be warranted. • The long-term performance of the endovascular graft and stent has not yet been established. All patients should be advised that endovascular treatment requires life-long, regular follow-up to assess their health and the performance of their endovascular graft and/or stent. Patients with specific clinical findings (e.g., persisting flow in the false lumen, enlarging aneurysms, or changes in the structure or position of the endovascular graft and or stent) should receive enhanced follow-up. Specific follow-up guidelines are described in **Section 12, IMAGING GUIDELINES AND POSTOPERATIVE FOLLOW-UP, in the complete INSTRUCTIONS FOR USE.** • The graft and stent are not recommended in patients unable to undergo, or who will not be compliant with, the necessary preoperative and postoperative imaging and implantation studies described in **Section 12, IMAGING GUIDELINES AND POSTOPERATIVE FOLLOW-UP, in the complete INSTRUCTIONS FOR USE.** • The graft and stent are not recommended for patients whose weight or size would compromise or prevent the necessary imaging requirements. • Graft implantation may increase the risk of paraplegia where graft exclusion covers the origins of dominant spinal cord or intercostal arteries. • Highly patent intercostal aortic branches or large collateral vessels are likely to result in retrograde flow after thoracic graft implantation. Patients with uncorrectable coagulopathy may also have an increased risk of Type II endoleak or bleeding complications.

Implant Procedure: The following apply to both the Zenith TX2 Dissection Endovascular Graft with Pro-Form and the Zenith Dissection Endovascular Stent: Strict adherence to the sizing guidelines provided in **Sections 10.4 and 10.5 in the complete INSTRUCTIONS FOR USE** is strongly recommended in order to mitigate the risk for events that could result from selecting inappropriate device sizes. Undersizing has resulted in migration, endoleak/entry-flow and false lumen growth. • **Table 1 in the complete INSTRUCTIONS FOR USE** incorporates appropriate graft oversizing. Sizing outside of the recommendations provided in **Table 1 in the complete INSTRUCTIONS FOR USE**, including that which could result from a difference in location of graft deployment relative to the location used for graft sizing, has resulted in false lumen expansion, endoleak/entry-flow, and migration. Fracture, device infolding, thrombosis, or compression may also result. • Systemic anticoagulation should be used during the implantation procedure based on hospital and physician preferred protocol. If heparin is contraindicated, an alternative anticoagulant should be used. • Minimize handling of the constrained endoprosthesis during preparation and insertion to decrease the risk of endoprosthesis contamination and infection. • To activate the hydrophilic coating on the outside of the sheath, the surface must be wiped with sterile gauze pads soaked in saline solution. Always keep the sheath hydrated for optimal performance. • Maintain wire guide position during introduction system insertion. • Do not bend or kink the introduction system. Doing so may cause damage to the introduction system and the graft/stent. • Always use fluoroscopy for guidance, delivery, and observation of the graft/stent within the vasculature. • The use of the graft/stent requires administration of intravascular contrast. Patients with pre-existing renal insufficiency may have an increased risk of renal failure postoperatively. Care should be taken to limit the amount of contrast media used during the procedure. • To avoid twisting the endovascular graft and/or stent, never rotate the introduction system during the procedure. Allow the device to conform naturally to the curves and tortuosity of the aorta. • As the sheath is withdrawn, anatomy and graft/stent position may change. Constantly monitor graft position and perform angiography to check position as necessary. • Incorrect deployment or migration of the graft and/or stent may require surgical intervention. • Do not continue advancing the wire guide or any portion of the introduction system if resistance is felt. Stop and assess the cause of resistance; vessel, catheter, or graft damage may occur. Exercise particular care in areas of stenosis, intravascular thrombosis, or calcified or tortuous vessels. • Use caution during manipulation of catheters, wires and sheaths within a dissection. Significant disturbances may dislodge fragments of thrombus, which can cause distal or cerebral embolization. • Avoid damaging the graft and/or stent or disturbing graft/stent positioning after placement in the event reinstrumentation (secondary intervention) of the graft/stent is necessary. • Do not attempt to re-sheath the graft or stent after partial or complete deployment. • To avoid entangling any catheters left in situ, rotate the introduction system during withdrawal. • Any sources for false lumen perfusion left untreated during the implantation procedure should be carefully followed after implantation. **The following apply to the Zenith TX2 Dissection Endovascular Graft with Pro-Form:** • Landing the proximal end of the device in dissected tissue could increase the risk of damage to the septum and could lead to new septal tears, aortic rupture, retrograde dissection, or other complications. • Inaccurate placement, incomplete sealing, inadequate oversizing, or lack of complete circumferential wall contact along the entire length of the Zenith TX2 Dissection Endovascular Graft with Pro-Form within the vessel may result in increased risk of endoleak, migration, or inadvertent occlusion of the left subclavian, left common carotid, and/or celiac arteries. • Consider the potential effects of hypovolemia on aortic diameters when selecting the device size. • If placing multiple grafts, ensure a minimum of 2 stent overlap. • Unless medically indicated, do not deploy the Zenith TX2 Dissection Endovascular Graft with Pro-Form in a location that will occlude arteries necessary to supply blood flow to organs or extremities. Do not cover significant arch or mesenteric arteries (exception may be the left subclavian artery) with the endoprosthesis. Vessel occlusion may occur. If a left subclavian artery is to be covered with the device, the clinician should be aware of the possibility of compromise to cerebral and upper limb circulation. • Repositioning the stent graft distally after partial deployment of the covered proximal stent may result in damage to the stent graft and/or vessel injury. • Molding balloon use is optional, and if used, it should not be inflated in the aorta outside of the graft. Additionally, complete deflation of the balloon should be confirmed prior to repositioning. For added hemostasis, the Captor Hemostatic Valve can be loosened or tightened to accommodate the insertion and subsequent withdrawal of a molding balloon. **The following apply to the Zenith Dissection Endovascular Stent:** • Use of the Zenith Dissection Endovascular Stent in an aneurysmal segment of a chronic dissection is not recommended. • As the sheath is withdrawn, do not advance the introduction system. Doing so can cause the stent to become inverted. • Overlapping of bare stent(s) or overlap with the Zenith TX2 Dissection Endovascular Graft with Pro-Form Straight Component or Tapered Component is left to the discretion of the implanting physician. Factors affecting whether or not to overlap, such as locations of reentries or expanded false lumen, should be judged by individual patient anatomy. When overlapping the bare stent within the stent graft component, no more than one-half of a partially overlapped bare stent body should be nonoverlapped, so as to prevent flaring of the bare stent. • If the distal end of the stent will be deployed in a funnel-shaped or angulated section of the aorta, or if the distal end of the stent appears conical in shape upon deployment, it is recommended to extend the treated segment distally with an additional stent, or choose a longer stent so it ends in a straight part of the aorta. Similarly, if the distal end of the stent will be deployed at the level of the diaphragm, or in a segment adjacent to the origin of the Celiac Trunk, Superior Mesenteric Artery and/or Renal Arteries, it is also recommended to extend the treated segment distally with an additional stent or choose a longer stent. • Use of a molding balloon inside a section of aorta treated with the Zenith Dissection Endovascular Stent is not recommended. • Avoid twisting or rotating the gray positioner against the introducer sheath assembly. Doing so may cause the loaded stent to become entangled and to deploy in a twisted state, or not to release from the introduction system. • Exercise caution when manipulating a wire guide through an in-situ Zenith Dissection Endovascular Stent; the wire guide may become entangled with the stent.

MRI Information: Nonclinical testing has demonstrated that the Zenith TX2 Dissection Endovascular Graft with Pro-Form overlapped with the nitinol Zenith Dissection Endovascular Stent is MR Conditional according to ASTM F2503. A patient with these devices can be scanned safely in a 1.5 T or 3.0 T MR system using the specific testing parameters described in **Section 12.4, MRI Information, in the complete INSTRUCTIONS FOR USE**.

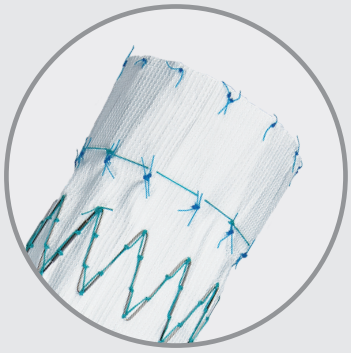
POTENTIAL ADVERSE EVENTS: Adverse events that may occur and/or require intervention include, but are not limited to: • Amputation • Anesthetic complications and subsequent problems (e.g., aspiration) • Aortic enlargement • Aortic rupture and death • Aortic damage, including perforation, dissection, bleeding, and rupture • Arterial or venous thrombosis and/or pseudoaneurysm • Bleeding, hematoma, or coagulopathy • Bowel complications (e.g., ileus, transient ischemia, infarction, necrosis) • Cardiac complications and subsequent problems (e.g., arrhythmia, tamponade, myocardial infarction, congestive heart failure, hypotension, hypertension) • Claudication (e.g., buttock, lower limb) • Death • Dissection extension (i.e., either proximal or distal extension) • Edema • Embolization (micro and macro) with transient or permanent ischemia or infarction • Endoleak • Endoprosthesis: improper component placement; incomplete component deployment; poor conformability of the graft to the vessel wall; component migration and/or separation; suture break; occlusion; infection; stent fracture; graft material wear; dilatation; erosion; puncture and perigraft flow • Fever and localized inflammation • Fistula (e.g., aortobronchial, aortoesophageal, arteriovenous) • Genitourinary complications and subsequent problems (e.g., ischemia, erosion, fistula, urinary incontinence, hematuria, infection) • Hepatic failure • Impotence • Infection of the dissection, device or access site, including abscess formation, transient fever and pain • Local or systemic neurologic complications and subsequent problems (e.g., stroke, transient ischemic attack, paraplegia, paraparesis, spinal cord shock, paralysis) • Lymphatic complications and subsequent problems (e.g., lymph fistula, lymphocele) • Occlusion of device or native vessel • Persisting flow in the false lumen • Pulmonary/respiratory complications and subsequent problems (e.g., pneumonia, respiratory failure, prolonged intubation) • Renal complications and subsequent problems (e.g., artery occlusion, contrast toxicity, insufficiency, failure) • Surgical conversion to open repair • Unintentional dissection septum rupture • Vascular access site complications, including infection, pain, hematoma, pseudoaneurysm, arteriovenous fistula • Vascular spasm or vascular trauma (e.g., ilio-femoral vessel dissection, bleeding, rupture, death) • Wound complications and subsequent problems (e.g., dehiscence, infection)

See instructions for use for full product information.

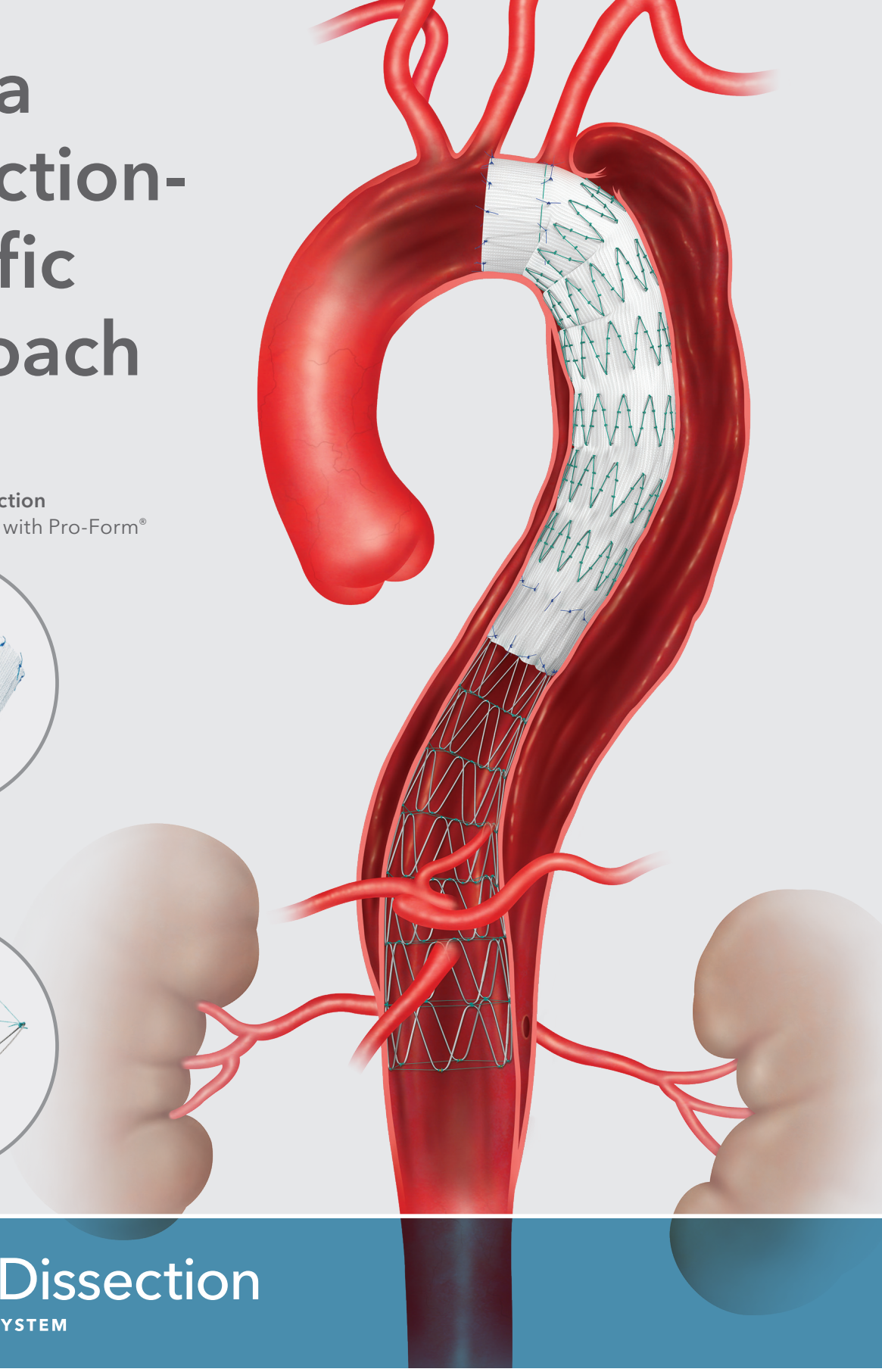
AB_I-DISSECT-SYSTEM-441_REV0

Take a dissection-specific approach

Zenith® TX2® Dissection
Endovascular Graft with Pro-Form®



Zenith® Dissection
Endovascular Stent



Zenith® Dissection
ENDOVASCULAR SYSTEM

COOK® MEDICAL