# Five-year results of the STABLE II study for the endovascular treatment of complicated, acute type B aortic dissection with a composite device design

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## ABSTRACT

**Objective:** To provide the 5-year outcomes of the use of a composite device (proximal covered stent graft + distal bare stent) for endovascular repair of patients with acute, type B aortic dissection complicated by aortic rupture and/or malperfusion.

**Methods:** Study of Thoracic Aortic Type B Dissection Using Endoluminal Repair (STABLE) II was a prospective, multicenter study of the Zenith Dissection Endovascular System (William Cook Europe). Patients were enrolled between August 2012 and January 2015 at sites in the United States and Japan. Five-year follow-up was completed by January 2020.

**Results:** In total, 73 patients (mean age: 60.7 ± 10.9 years; 65.8% male) with acute type B dissection complicated by malperfusion (72.6%), rupture (21.9%), or both (5.5%) were enrolled. Patients were treated with either a composite device (79.5%) or the proximal stent graft alone (no distal bare stent, 20.5%). Dissections were more extensive in patients who received the composite device (408.9 ± 121.3 mm) than in patients who did not receive a bare stent (315.9 ± 100.1 mm). The mean follow-up was 1209.4 ± 754.6 days. Freedom from all-cause mortality was 80.3% ± 4.7% at 1 year and 68.9% ± 7.3% at 5 years. Freedom from dissection-related mortality remained at 97.1% ± 2.1% from 1-year through 5-year follow-up. Within the stent-graft region, the rate of either complete thrombosis or elimination of the false lumen increased over time (82.1% of all patients at 5 years vs 55.7% at first postprocedure computed tomography), with a higher rate at 5 years in patients who received the composite device (90.5%) compared with patients without the bare stent (57.1%). Throughout the follow-up, overall true lumen diameter increased within the stent-graft region, and overall false lumen diameter decreased. At 5 years, 20.7% of patients experienced a decrease in maximum transaortic diameter within the stent-graft region, 17.2% experienced an increase, and 62.1% experienced no change. Distal to the treated segment (but within the dissected aorta), 23.1% of patients experience no change in transaortic diameter at 5 years; a bare stent was deployed in all these patients at the procedure. Five-year freedom from all secondary intervention was 70.7% ± 7.2%.

**Conclusions:** These 5-year outcomes indicate a low rate of dissection-related mortality for the Zenith Dissection Endovascular System in the treatment of patients with acute, complicated type B aortic dissection. Further, these data suggest a positive influence of composite device use on false lumen thrombosis. Continuous monitoring for distal aortic growth is necessary in all patients. (J Vasc Surg 2022;■:1-9.)

**Keywords:** Aortic dissection; Type B; Malperfusion; False lumen; Bare stent

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Author conflict of interest: J.V.L. is a consultant for Cook Medical and the principal investigator for the STABLE I & II studies. T.G.G. received institutional grant support from Medtronic and Boston Scientific, but no personal remuneration. He serves on the Medical Advisory Board for Abbott, for which he is paid nominally for his time, and on the Medical Advisory Board for Cytosorbants, for which he has received no remuneration. J.M.P. receives consultant fees from Terumo Aortic, Cook Medical, Medtronic, and W. L. Gore; he is on the speakers bureau of Terumo Aortic, Medtronic Inc, and Philips Volcano and the advisory board of Medtronic Inc, W. L. Gore, and Mellon Medical. B.W.S. is a cofounder of the Aortica Corporation. M.D.D. is a consultant for W. L. Gore & Associates and Cook Medical. S.H. is a consultant for Cook Medical. E.S. and P.B. are salaried employees of Cook Research Incorporated, a Cook Group Company.

Presented at the Vascular Annual Meeting, San Diego, Calif, August 19, 2021.

Additional material for this article may be found online at www.jvascsurg.org.

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The editors and reviewers of this article have no relevant financial relationships to disclose per the JVS policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

0741-5214



https://doi.org/10.1016/j.jvs.2022.06.092

The standard of care for the treatment of acute, complicated type B aortic dissection (TBAD) is thoracic endovascular aortic repair (TEVAR), and the techniques and devices used in TEVAR have rapidly evolved to further improve patient outcomes. One such technique, first described as the staged thoracoabdominal and branch vessel endoluminal repair technique,[1] involves the deployment of a distal bare stent to re-expand the collapsed true lumen in the distal aorta after the placement of the proximal stent graft for coverage of the primary entry tear. Early[2,3] to long-term outcomes[4] on the use of this technique support its safety and effectiveness, as well as the technique's positive influence on aortic remodeling,[5,6] compatibility ease with potential further intervention,[4,7] and expansive true lumen support without blocking critical vessels.[8]

The Zenith Dissection Endovascular System (William Cook Europe ApS) is indicated for the endovascular treatment of patients with TBAD. Early results from the Study of Thoracic Aortic Type B Dissection Using Endoluminal Repair (STABLE) I and II studies supported the safety (ie, 30-day freedom from adverse events) and effectiveness (ie, 30-day survival) of both the previous[2] and current[3] generations of the device system. Longer follow-up from the STABLE studies[2-4] and others[5,6,9,10] indicates favorable clinical outcomes and positive aortic remodeling after treatment with the device system, with the caveat that all patients should undergo continuous monitoring for distal aortic growth.[7,11] Herein, we report the STABLE II 5-year outcomes on the use of the Zenith Dissection Endovascular System for the treatment of acute, complicated TBAD.

## ARTICLE HIGHLIGHTS

- **Type of Research:** Multicenter, prospective, nonrandomized cohort study on clinical outcomes after treatment with the Zenith Dissection Endovascular System
- **Key Findings:** A total of 73 patients were treated for acute, complicated type B aortic dissection using the Zenith Dissection Endovascular System. Freedom from all-cause mortality was 68.9% ± 7.3% at 5 years, and freedom from dissection-related mortality remained 97.1% ± 2.1% from 1-year through 1-year follow-up.
- **Take Home Message:** Five-year outcomes indicate a low rate of dissection-related mortality for the Zenith Dissection Endovascular System in the treatment of patients with acute, complicated type B aortic dissection.

## METHODS

**The STABLE II study.** The STABLE II study was a prospective, nonrandomized study conducted under a U.S. Food and Drug Administration investigational device exemption to evaluate the safety and effectiveness of the Zenith Dissection Endovascular System for the treatment of patients with acute, complicated TBAD (NCT01568320). Full details of the study design and patient inclusion-exclusion criteria were published previously.[3] In brief, suitable patients included those with acute (within 14 days of onset) TBAD complicated by aortic rupture or malperfusion characterized by visceral branch vessel obstruction and/or compromise. Enrollment began in August 2012 and was completed by January 2015. The study included 22 investigational sites with centers in the United States (67 patients at 21 sites) and Japan (six patients at one site). Before study commencement, approval was obtained from local ethics committees or institution review boards, and the study was performed in accordance with the Declaration of Helsinki. All patients provided informed consent.

Primary study end points included rates of 30-day survival and 30-day freedom from major adverse events (defined as myocardial infarction, chronic renal insufficiency/chronic renal failure requiring dialysis, bowel ischemia, stroke, paraplegia or paraparesis, and prolonged [>72 hours] ventilatory support). Primary study end points were met, as published previously.[3] Clinical and/or imaging follow-up occurred on the following schedule: within 7 days after the procedure, at 30 days, at 6 months, at 1 year, and annually through 5 years. First postprocedure computed tomography (CT) imaging was performed before hospital discharge or at 1-month follow-up in patients with impaired renal function at the time of discharge. An independent data safety monitoring board oversaw the clinical trial. All patient deaths were adjudicated by an independent clinical events committee (CEC) to assess death relatedness to a preexisting or unrelated condition and to the procedure, technique, and/or device. Dissection-related mortality was determined by the CEC as death related to dissection repair. An independent core laboratory analyzed all imaging datasets and completed measurements according to definitions in the imaging charter. Device migration was defined as antegrade or retrograde movement of more than 10 mm relative to anatomic landmarks identified on the first postprocedure CT imaging.

**Study device.** The Zenith Dissection Endovascular System is a composite device system comprising a proximal covered stent-graft component (Zenith TX2 Dissection Endovascular Graft with Pro-Form, "stent-graft") and a distal bare metal stent component (Zenith Dissection Endovascular Stent, "dissection stent"). Full design details of these components have been published previously.[3] Devices were deployed using standard endovascular techniques, with recommended deployment of the proximal stent graft for coverage of the primary tear before the deployment of the distal

**Table I.** Summary of mortality and notable adverse events

| Event | Percentage of patients (n) | | |
|---|---|---|---|
| | 0-30 days (N = 73) | 31-365 days (N = 67) | >365 days (N = 56) |
| Death | 6.8 (5) | 13.4 (9) | 10.7 (6) |
| Rupture | 1.4 (1) | 1.5 (1) | 1.8 (1) |
| Conversion to open surgical repair | 0 (0) | 0 (0) | 1.8 (1) |
| Myocardial infarction | 1.4 (1) | 0 (0) | 3.6 (2) |
| Renal failure requiring dialysis | 6.8 (5) | 1.5 (1) | 3.6 (2) |
| Bowel ischemia | 1.4 (1) | 3.0 (2) | 1.8 (1) |
| Stroke | 6.8 (5) | 1.5 (1) | 3.6 (2) |
| Paraplegia | 2.7 (2) | 0 (0) | 0 (0) |
| Paraparesis | 4.1 (3) | 0 (0) | 0 (0) |
| Prolonged (>72 hours) ventilatory support | 13.7 (10) | 0 (0) | 3.6 (2) |

dissection stent. In most patients, the dissection stent was placed to support the distal, delaminated segments of the aorta after coverage of the primary tear with the stent graft. All patients were treated with a stent graft; however, use of the dissection stent was at the discretion of the implanting physician.

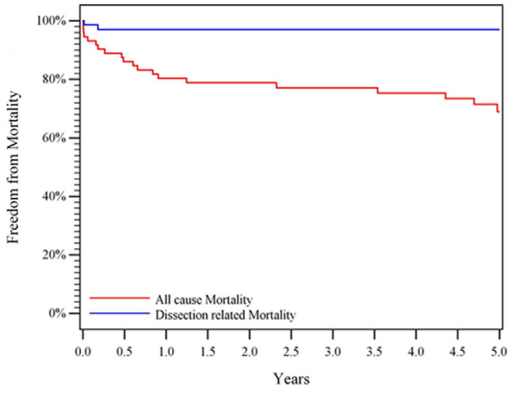
**Data analysis.** All study data were managed by a centralized data coordinating center, Cook Research Incorporated. Statistical analyses were performed using SAS for Windows (version 9.4; SAS Institute). Unless noted otherwise, continuous variables were reported as mean ± standard deviation. Categorical variables were reported as percentages and frequencies. Differences in preprocedure dissection extent were assessed by Student's *t*-test. Freedom from all-cause mortality, dissection-related mortality, and secondary intervention were estimated using the Kaplan-Meier analysis. A *P* value of less than .05 was considered statistically significant. False lumen entry flow was described in accordance with the reporting standards by Lombardi et al.[12]

## RESULTS

Full details of patient demographics are described in the prior publication of the STABLE II 1-year results.[3] In brief, the study included 73 patients, 65.8% (43 of 73) of whom were male, with a mean age of 60.7 ± 10.9 years. All patients presented with acute, complicated dissections; 53 patients presented with dissection complicated by malperfusion, 16 by aortic rupture, and 4 by both malperfusion and rupture. Details of the types and number of devices were described previously,[3] wherein 58 patients received at least one stent graft and at least one dissection stent during the index procedure. The remaining 15 patients received only stent graft(s) (ie, no dissection stent was deployed). Reasons provided for not using the dissection stent included adequate coverage with stent graft(s) alone (nine patients), no false lumen flow through the re-entry tears (five patients), and investigator judgment (one patient). A significant difference in dissection extent ($t_{51}$ = 2.50, $P$ = .016) was observed between patients who received a dissection stent (408.9 ± 121.3 mm, 95% confidence interval: 370.1-447.7 mm) compared with patients who did not receive a dissection stent (315.9 ± 100.1 mm, 95% confidence interval: 255.4-376.4 mm). Because of this significant difference in initial dissection extent, additional subanalyses of patient outcomes by devices received were not performed (in addition, the study was not designed or powered to assess these potential outcome differences). All follow-up visits were completed by January 2020; mean follow-up was 1209.4 ± 754.6 days.

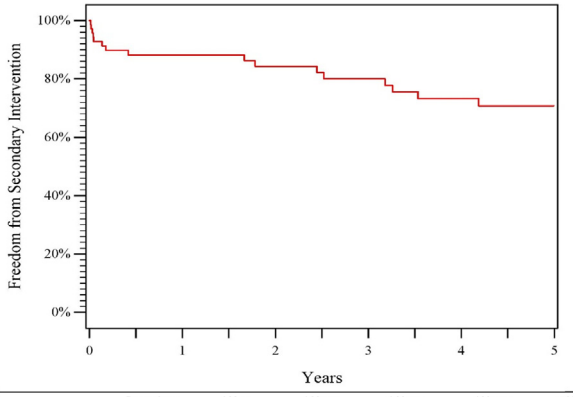
**Morbidity and mortality.** Most major adverse events occurred within the first 30 days after the procedure, as shown in Table I, and events occurring before 1-year follow-up were described in prior publication.[3] All instances of bowel ischemia and renal failure requiring dialysis after 1 year were adjudicated as unrelated to the procedure or device. Throughout the study, aortic rupture occurred in three patients with dissection stent(s) placed at the procedure and resulted in death in two of these patients. The two ruptures that resulted in death occurred within 1 year, as described previously;[3] the cause of one rupture could not be determined and the other was related to a pre-existing, untreated type A dissection (as evident from preprocedural CT imaging). On postprocedure day 1813, the third patient underwent open surgical repair for an abdominal infrarenal aortic rupture. Throughout the study duration, one patient underwent conversion to open repair with subsequent explantation of the study device on postprocedure day 650 due to stent-graft infection.

Kaplan-Meier freedom from all-cause mortality was 80.3% ± 4.7% at 1 year and 68.9% ± 7.3% at 5 years, as shown in Fig 1. A total of 20 deaths were reported during the study duration, with details of time to death, cause, and relatedness provided in Supplementary Table I




| | Procedure | 1 Year | 2 Years | 3 Years | 4 Years | 5 Years |
|---|---|---|---|---|---|---|
| All-cause mortality | 100% ± 0% | 80.3% ± 4.7% | 78.8% ± 5.0% | 77.1% ± 5.4% | 75.3% ± 5.7% | 68.9% ± 7.3% |
| Cumulative events | 0 | 14 | 15 | 16 | 17 | 20 |
| Number at risk | 73 | 56 | 49 | 44 | 41 | 24 |
| Dissection-related mortality | 100% ± 0% | 97.1% ± 2.1% | 97.1% ± 2.1% | 97.1% ± 2.1% | 97.1% ± 2.1% | 97.1% ± 2.1% |
| Cumulative events | 0 | 2 | 2 | 2 | 2 | 2 |
| Number at risk | 73 | 56 | 49 | 44 | 41 | 24 |

**Fig 1.** Kaplan-Meier analysis of 5-year freedom from all-cause or dissection-related mortality.




| | Procedure | 1 Year | 2 Years | 3 Years | 4 Years | 5 Years |
|---|---|---|---|---|---|---|
| Secondary intervention | 100% ± 0% | 88.2% ± 4.1% | 84.3% ± 5.1% | 80.0% ± 5.9% | 73.2% ± 6.7% | 70.7% ±7.2% |
| Cumulative events | 0 | 8 | 10 | 12 | 15 | 16 |
| Number at risk | 73 | 50 | 42 | 36 | 30 | 15 |

**Fig 2.** Kaplan-Meier analysis of 5-year freedom from secondary intervention.

(online only). Fourteen deaths occurred within 1 year; six deaths occurred during the remaining follow-up period. Of the six deaths that occurred after 1 year, five deaths were adjudicated as not procedure- or dissection-related by the independent CEC, and the relatedness of one death was unable to be determined. Kaplan-Meier freedom from dissection-related mortality was 97.1% ± 2.1% at 1-year through 5-year follow-up. Two dissection-related deaths occurred within the first year, as described previously.[3] No other dissection-related deaths were reported throughout the remainder of the study.

**Secondary interventions.** During follow-up, 16 patients required a total of 22 secondary intervention procedures; six of these procedures occurred within the first 30 days (with details of the reason and type of secondary intervention in Supplementary Table II, online only). Most secondary interventions were percutaneous and included additional stent graft, dissection stent, or bifurcated abdominal graft placement (7); visceral or iliac vessel stent placement (5); coil embolization (3); balloon angioplasty (2); and thrombectomy of right femoropopliteal artery (1). Surgical interventions included surgical bypass procedures (2), conversion to open repair (1, described previously), and other procedures (11, as described in Supplementary Table II, online only). Throughout the study, the most common reasons for secondary intervention were re-entry flow, particularly from secondary tears (five instances), type I proximal entry flow (four instances), or type I distal entry flow (two instances). Kaplan-Meier freedom from secondary intervention was 88.2% ± 4.1% at 1 year and 70.7% ± 7.2% at 5 years, as shown in Fig 2.

**False lumen status and entry flow.** False lumen thrombosis status was described by region of device deployment, and initial dissection location at preprocedure and device location at postprocedure CT by the aortic zone (defined by Fillinger et al[13]) are shown in Fig 3. In the stent-graft region, complete thrombosis of the false lumen or no apparent false lumen was observed in 82.1% (23 of 28) of all patients at 5 years, an increase from 55.7% (34 of 61) of all patients at first postprocedure CT (illustrated graphically in Fig 3 and detailed further in Supplementary Table III, online only). Seventeen of the 23 patients with complete thrombosis or no apparent false lumen at 5 years were free of secondary intervention throughout the study (the other six patients had reinterventions performed; patients 1, 7, 10, 14, and 15 in Supplementary Table II, online only). Partial thrombosis in the stent-graft region decreased from 42.6% (26 of 61) at first postprocedure CT to 17.9% (5 of 28) at 5 years. Thrombosis status in the stent-graft region varied between patients who received the dissection stent and those who did not. At 5 years, the complete thrombosis or no apparent false lumen rate in the stent-graft region was higher in patients who received the dissection stent (90.5%, 19 of 21) than in patients who did not receive the dissection stent (57.1%, 4 of 7). The partial thrombosis rate was higher (42.9%, 3 of 7) in patients who did not receive the dissection stent compared with patients who did receive the dissection stent (9.5%, 2 of 21).

Within the dissection stent region, the complete thrombosis or no apparent false lumen rate was 38.1% (8 of 21) for eligible patients (ie, patients with dissection stent[s]) at 5 years, an increase from 6.3% (3 of 48) at first postprocedure CT. The rate of false lumen patency decreased from 10.4% (5 of 48) at first postprocedure CT to none (0%, 0 of 21) at 5 years. The 5-year partial thrombosis rate distal to the treated segment was similar between patients with or without the dissection stent, with

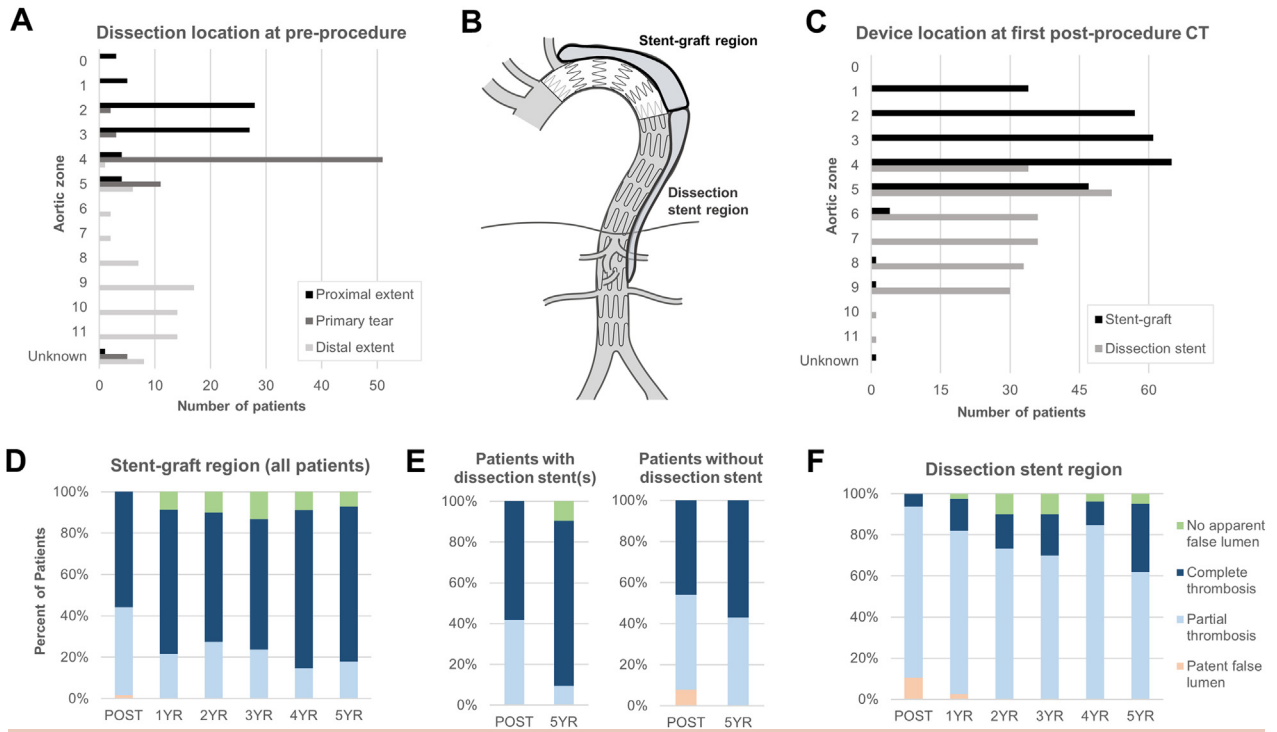



**Fig 3.** Dissection location, device location, and false lumen status by the device component region. Dissection extent varied among patients at the preprocedure **(A)**, and regions adjacent to the device were defined per patient (as shown by an example in **B**). Device location was assessed at first postprocedure computed tomography (*CT*) **(C)**. In the stent-graft region **(D)**, an increase in complete thrombosis was observed for most patients at 5 years; however, a higher percentage of patients with the dissection stent (**E**, left) experienced complete thrombosis in this region compared with patients who did not receive the dissection stent (**E**, right). For patients who received the dissection stent, partial thrombosis was common at 5 years in the dissection stent region **(F)**.

52.6% (10 of 19) partial thrombosis rate observed in those with the dissection stent and 42.9% (3 of 7) partial thrombosis rate observed in those without the dissection stent. At 5 years, complete thrombosis or no apparent false lumen distal to the treated segment was observed in 42.1% (8 of 19) of patients who received the dissection stent; no complete thrombosis was observed in this region in patients without the dissection stent although 42.9% (3 of 7) of patients had no apparent false lumen. A patent false lumen distal to the treated segment was observed in one patient with the dissection stent and in one patient without a dissection stent at 5 years.

At or after 1 year, sources of flow into the false lumen via the primary entry tear in the descending thoracic aorta included type I proximal flow in patients with either an inadequate proximal landing zone (three patients) or both an inadequate proximal landing zone and device undersizing (two patients). There were no instances of type I distal, type II, type III, or type IV flow into the false lumen at the primary entry tear in either the descending thoracic or abdominal aorta at or after 1-year follow-up. Flow directly into the false lumen via collateral or secondary tears was the major source of false lumen perfusion throughout the 5-year study duration.

**True lumen, false lumen, and maximum transaortic diameter.** Within the stent-graft region, average true lumen diameter increased from first postprocedure CT through 5-year follow-up, and average false lumen diameter conversely decreased, with change in diameters over time shown in Fig 4. At 4 years, 17.2% (5 of 29) of patients experienced an increase in diameter (>5 mm relative to first postprocedure CT), 20.7% (6 of 29) experienced a decrease, and 62.1% (18 of 29) experienced no change, as described per follow-up visit in Table II. In patients who received the dissection stent, the average true lumen diameter increased within the dissection stent region, yet average false lumen diameter remained relatively stable (≤5 mm change). At 5 years, 61.9% (13 of 21) of patients experienced an increase in maximum transaortic diameter in this region; nine of these thirteen patients had partial thrombosis of the false lumen. Distal to the treated segment, the average true lumen diameter was stable from first postprocedure CT through 5-year follow-up, whereas the average false lumen diameter increased (>5 mm). At 5 years, 76.9% (10 of 13) of all patients experienced an increase in maximum transaortic diameter distal to the treated segment; eight of these ten patients had either partial

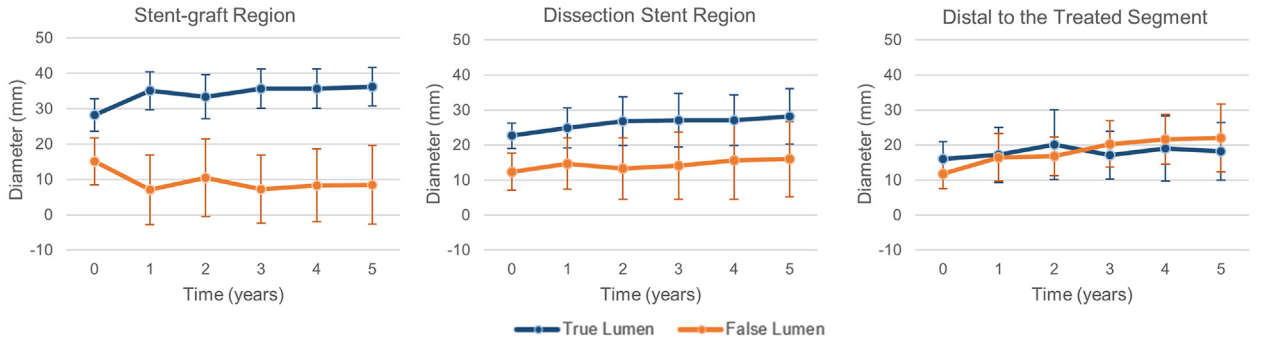



**Fig 4.** Lumen diameters at maximum transaortic diameter by region of device deployment. True and false lumen diameters at the maximum transaortic diameter in the stent-graft region (left), dissection stent region (middle), or distal to the treated segment (right). Mean ± standard deviation is shown.

**Table II.** Changes in maximum transaortic diameter

| | Percentage of patients (n/N) | | | | |
|---|---|---|---|---|---|
| | 1 year | 2 years | 3 years | 4 years | 5 years |
| Stent-graft region | | | | | |
| All patients | | | | | |
| Increase | 14.9 (7/47) | 23.8 (10/42) | 22.5 (9/40) | 27.8 (10/36) | 17.2 (5/29) |
| Decrease | 25.5 (12/47) | 19.0 (8/42) | 17.5 (7/40) | 19.4 (7/36) | 20.7 (6/29) |
| No change | 59.6 (28/47) | 57.1 (24/42) | 60.0 (24/40) | 52.8 (19/36) | 62.1 (18/29) |
| Patients with dissection stent(s) | | | | | |
| Increase | 16.2 (6/37) | 25.0 (8/32) | 25.8 (8/31) | 28.6 (8/28) | 19.0 (4/21) |
| Decrease | 27.0 (10/37) | 21.9 (7/32) | 12.9 (4/31) | 17.9 (5/28) | 19.0 (4/21) |
| No change | 56.8 (21/37) | 53.1 (17/32) | 61.3 (19/31) | 53.6 (15/28) | 61.9 (13/21) |
| Patients without dissection stent | | | | | |
| Increase | 10.0 (1/10) | 20.0 (2/10) | 11.1 (1/9) | 25.0 (2/8) | 12.5 (1/8) |
| Decrease | 20.0 (2/10) | 10.0 (1/10) | 33.3 (3/9) | 25.0 (2/8) | 25.0 (2/8) |
| No change | 70.0 (7/10) | 70.0 (7/10) | 55.6 (5/9) | 50.0 (4/8) | 62.5 (5/8) |
| Dissection stent region | | | | | |
| Patients with dissection stent(s) | | | | | |
| Increase | 38.5 (15/39) | 45.2 (14/31) | 58.1 (18/31) | 59.3 (16/27) | 61.9 (13/21) |
| Decrease | 5.1 (2/39) | 6.5 (2/31) | 6.5 (2/31) | 3.7 (1/27) | 4.8 (1/21) |
| No change | 56.4 (22/39) | 48.4 (15/31) | 35.5 (11/31) | 37.0 (10/27) | 33.3 (7/21) |
| Patients without dissection stent | NA | | | | |

*NA*, Not applicable.
Increase or decrease was indicated by >5 mm difference in diameter relative to first postprocedure computed tomography (CT) measurement (either before discharge or at 1 month after the procedure).

thrombosis of the false lumen (six patients) or false lumen patency (two patients). Three patients (23.1%, 3 of 13) did not experience transaortic growth distal to the treated segment at 5 years; all three of these patients received the dissection stent at the procedure.

**Device integrity, separation, and migration.** All events related to device integrity (ie, kink, fracture, compression, and infolding) occurred within the first year of stent implantation, as described previously.[3] Four patients experienced component separation throughout the study: three patients experienced separation between the stent graft and the dissection stent, and one patient experienced separation between two stent grafts. All instances of component separation occurred with concurrent aortic elongation. Device migration occurred in 10 patients, including in four patients by 1 year, three patients by 2 years, and three patients by 3 years. In all cases, the stent-graft component migrated caudally. Migration occurred in patients with inadequate landing zones (4), both inadequate landing zones and graft undersizing (5), or in the presence of further aortic

dilatation (1). One patient with both device migration and component separation had a secondary intervention performed (coil embolization, stent placement) for an expanding false lumen on postprocedure day 131. Otherwise, none of the other patients with migration required a secondary intervention.

**Progression of dissection.** Site-reported proximal dissection events were described in three patients after 1 year, including one patient with retrograde progression of dissection and two patients with a new tear or new type A dissection (see Supplementary Table II, online only, for related secondary interventions performed; patients 14-16). Importantly, none of these three proximal dissection events were considered retrograde progression of type B dissection to type A dissection. Two patients had evidence of pre-existing disease proximal to the left subclavian artery before study enrollment, as shown by either initial stent-graft landing in dissected aorta (ie, inadequate device landing zone) or notable enlargement of the ascending aorta (per preprocedural imaging). The third patient had a type B dissection that was no longer apparent before developing a new type A dissection. No instances of distal extension of dissection were reported after 1 year.

## DISCUSSION

Open surgical repair for acute, complicated type B dissection carries a high risk for in-hospital mortality (19%),[14] whereas 30-day mortality after TEVAR for complicated, acute type B dissection in clinical trials ranges from approximately 5% to 8%.[15-17] In STABLE II, 30-day all-cause mortality was favorable at 6.8%, as published previously.[3] In this report of 5-year outcomes, freedom from all-cause mortality was 80.3% ± 4.7% at 1 year and 68.9% ±7.3% at 5 years. One-year and five-year freedom from all-cause mortality in STABLE II were similar to those observed after the treatment of acute, complicated type B dissection with the other endovascular devices.[17,18] Current practice favors the treatment of acute, complicated type B dissection with endovascular repair compared with open surgical repair or medical management,[19-21] and other studies[22-24] support TEVAR as a safe and effective treatment option for this indication.

In STABLE II, freedom from dissection-related mortality was 97.1% ± 2.1% at 1-year through 5-year follow-up, with only two dissection-related deaths occurring within the first year, as published previously.[3] Most deaths were related to pre-existing disease and unrelated to the treated dissection. In STABLE I, 5-year freedom from dissection-related mortality was 83.9%± 5 .9% for acute, complicated type B dissection patients,[4] with a higher incidence of dissection-related deaths within the first year of treatment, including two deaths after stroke and three deaths after aortic rupture. These reported freedoms from dissection-related mortality in STABLE I and II compare favorably with those reported for the Valiant device, with 90.0% at 1 year and 82.5% at 5 years,[17] and the Conformable GORE TAG device, with 90% from 1-year through 5-year follow-up.[18]

In treatment of aortic dissection, the composite TEVAR technique with distal stent placement has been shown to support complete false lumen thrombosis and positive aortic remodeling in the thoracic aorta.[1,8,9,25,26] In this study, the patients treated with a dissection stent had significantly longer dissection extents than patients who did not receive a dissection stent. Through 5-year follow-up in STABLE II, true and false lumen diameters expanded and regressed, respectively, within the stent-graft region. At 5 years, use of the dissection stent was associated with an increased rate of the complete thrombosis or no apparent false lumen within the stent-graft region (90.5%) compared with the rate in those without the dissection stent (57.1%). In the dissection stent region, lumen diameters remained stable, with the average true lumen diameter greater than the false lumen diameter throughout all 5 years of follow-up. Although further analysis into the role of the dissection stent in STABLE II is not possible because only a small number of patients did not receive the dissection stent, and the potential confound of differences in preprocedure dissection extent, these data support the use of the dissection stent to assist with complete false lumen thrombosis and to help induce positive aortic remodeling.[9,27,28]

Previous research suggests that composite device usage may be associated with favorable aortic remodeling with benefits extending through the abdominal aorta and/or distal to the treated aortic segment.[6,10,28,29] In the previous STABLE I, use of the composite device was associated with positive aortic remodeling distal to the covered segment of the aorta,[2,4,7] and, in another comparison using the STABLE I cohort, bare stent usage reduced false lumen volume in the abdominal aorta postoperatively and through 1-year follow-up (although not statistically significant from standard TEVAR).[9] However, aortic expansion in the visceral and abdominal aorta appears to be a common consequence of TEVAR treatment, particularly for acute TBAD,[7,9] as the majority of TEVAR patients experience aneurysmal degeneration or aortic expansion within 5 years after the procedure.[11,30,31] In STABLE II, transaortic growth in the dissection stent region was observed in 61.9% of patients who received the dissection stent, and this observed aortic growth was associated with partial thrombosis of the false lumen for 9 of the 13 patients with transaortic growth at 5 years. Distal to the treated aorta, 76.9% of all study patients experienced an increase in transaortic diameter at 5-year follow-up, where again, aortic growth was associated with partial thrombosis of the false lumen or false lumen patency for most of these patients.

Growth in both the dissection stent region (for applicable patients) and distal to the treated segment was gradual over 5-year follow-up, but this growth trend suggests that close monitoring for disease progression is warranted throughout a patient's lifetime.[11]

Throughout the study, no proximal dissection events were considered retrograde progression of type B to type A dissection, similar to a report of less than 1% incidence of retrograde dissection after use of the Zenith Dissection Endovascular System from other authors.[32] Similarly, no instances of distal extension of dissection were reported after 1 year.

Throughout follow-up, the use of the bare dissection stent did not hinder the performance of secondary interventions—in STABLE II, 15 of the 16 patients who required secondary intervention had a dissection stent deployed at the procedure. A recent report by Kong et al[33] suggested that use of the distal bare metal stent in the treatment of acute, complicated type B dissection may indeed be related to a lower incidence of secondary intervention compared with standard TEVAR performed without a bare stent. A meta-analysis by Qiu et al[34] on supports that the distal bare stent uses may promote lower rates of reintervention. Deployment of the bare dissection stent may assist subsequent reinterventions by stabilizing the delaminated sections of the aorta, thus creating an aortic environment more amenable for further repair. Furthermore, careful considerations must be made not only to the distal aspects of disease but also to the proximal landing zone during procedural planning. An inadequate proximal landing zone has been identified as a significant factor influencing TEVAR outcomes for TBAD.[35] In STABLE II, type I entry flow into the false lumen via the primary entry tear was only observed in the setting of the inadequate proximal landing zone and/or inadequate device oversizing. Furthermore, device migration and component separation only occurred in the presence of an inadequate landing zone, graft undersizing, or aortic elongation.

Limitations of this study include the small number of patients with assessable imaging in late follow-up and the inability to assess the role of the dissection stent on aortic remodeling (as the study was not initially powered to do so). Strengths of the study include the addition of long-term evidence that TEVAR for the treatment of acute, complicated type B dissection is safe and effective through 5 years. Additional reports on long-term outcomes are nevertheless necessitated because of the limited number of research reports available, particularly randomized controlled trials,[36] with substantial long-term follow-up and the heterologous presentation of TBAD (ie, complicated vs uncomplicated, acute vs nonacute).

## CONCLUSIONS

In STABLE II, dissection-related mortality was 97.1% ± 2.1% at 1-year through 5-year follow-up, with only two deaths related to dissection throughout the study duration. Long-term positive remodeling of the thoracic aorta was observed and appeared to be enhanced by the deployment of the dissection stent; dissection stent use was associated with early true lumen re-expansion and did not inhibit the ability to perform successful secondary interventions. Observed distal aortic growth suggests a need for continuous monitoring, which may be associated more with general TBAD disease progression rather than a device-specific outcome.

The authors thank all patients who participated in this study, as well as all the study investigators and research personnel. The authors also thank Brandon Coates, PhD, and Meridith Doyle, PhD, salaried employees of Cook Research Incorporated, for respective data analysis and medical writing support.

## AUTHOR CONTRIBUTIONS

Conception and design: JL, PM, PB
Analysis and interpretation: JL, TG, JP, BS, MD, SH, PM, ES, PB
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Statistical analysis: ES
Obtained funding: Not applicable
Overall responsibility: JL

## REFERENCES

1. Mossop PJ, McLachlan CS, Amukotuwa SA, Nixon IK. Staged endovascular treatment for complicated type B aortic dissection. Nat Clin Pract Cardiovasc Med 2005;2:316-21. quiz: 322.
2. Lombardi JV, Cambria RP, Nienaber CA, Chiesa R, Teebken O, Lee A, 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;55:629-40.e2.
3. Lombardi JV, Gleason TG, Panneton JM, Starnes BW, Dake MD, Haulon S, et al. STABLE II clinical trial on endovascular treatment of acute, complicated type B aortic dissection with a composite device design. J Vasc Surg 2020;71:1077-87.e2.
4. Lombardi JV, Cambria RP, Nienaber CA, Chiesa R, Mossop P, Haulon S, 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;70:1072-81.e2.
5. Matsuoka T, Hashizume K, Honda M, Harada D, Ohno M, Ikebata K, et al. The provisional extension to induce complete attachment technique is associated with abdominal aortic remodeling and reduces aorta-related adverse events after aortic dissection. J Vasc Surg 2021;74:45-52.e1.
6. Sultan I, Dufendach K, Kilic A, Bianco V, Trivedi D, Althouse AD, et al. Bare metal stent use in type B aortic dissection may offer positive remodeling for the distal aorta. Ann Thorac Surg 2018;106:1364-70.

7. Lombardi JV, Cambria RP, Nienaber CA, Chiesa R, Mossop P, Haulon S, 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;59:1544-54.
8. Bertoglio L, Rinaldi E, Melissano G, Chiesa R. The PETTICOAT concept for endovascular treatment of type B aortic dissection. J Cardiovasc Surg (Torino) 2019;60:91-9.
9. Sobocinski J, Lombardi JV, Dias NV, Berger L, Zhou Q, Jia F, 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;63:1216-24.
10. Hashizume K, Honda M, Mori M, Yagami T, Takaki H, Matsuoka T, et al. Full PETTICOAT in acute type B aortic dissection with patent false lumen may offer positive remodeling for the distal aorta. Gen Thorac Cardiovasc Surg 2021;69:926-33.
11. Mascia D, Rinaldi E, Salvati S, Melloni A, Kahlberg A, Bertoglio L, et al. Thoracic endovascular aortic repair with additional distal bare stents in type B aortic dissection does not prevent long-term aneurysmal degeneration. J Endovasc Ther 2021;28:425-33.
12. Lombardi JV, Hughes GC, Appoo JJ, Bavaria JE, Beck AW, Cambria RP, et al. Society for Vascular Surgery (SVS) and Society of Thoracic Surgeons (STS) reporting standards for type B aortic dissections. J Vasc Surg 2020;71:723-47.
13. Fillinger MF, Greenberg RK, McKinsey JF, Chaikof EL; Society for Vascular Surgery Ad Hoc Committee on TEVAR Reporting Standards. Reporting standards for thoracic endovascular aortic repair (TEVAR). J Vasc Surg 2010;52:1022-33. 1033.e15.
14. Moulakakis KG, Mylonas SN, Dalainas I, Kakisis J, Kotsis T, Liapis CD. Management of complicated and uncomplicated acute type B dissection. A systematic review and meta-analysis. Ann Cardiothorac Surg 2014;3:234-46.
15. Cambria RP, Conrad MF, Matsumoto AH, Fillinger M, Pochettino A, Carvalho S, et al. Multicenter clinical trial of the conformable stent graft for the treatment of acute, complicated type B dissection. J Vasc Surg 2015;62:271-8.
16. Bavaria JE, Brinkman WT, Hughes GC, Khoynezhad A, Szeto WY, Azizzadeh A, 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;100:802-8; discussion: 808-9.
17. Bavaria JE, Brinkman WT, Hughes GC, Shah AS, Charlton-Ouw KM, Azizzadeh A, et al. Five-year outcomes of endovascular repair of complicated acute type B aortic dissections. J Thorac Cardiovasc Surg 2022;163:539-48.e2.
18. W. L. Gore & Associates, Inc, Medical Products Division. GORE® TAG® thoracic endoprothesis annual clinical update—September 2018 2018, https://www.goremedical.com/ap/resource/as0089-en4. Accessed August 8, 2022.
19. Hogendoorn W, Hunink MG, Schlosser FJ, Moll FL, Sumpio BE, Muhs BE. Endovascular vs. open repair of complicated acute type B aortic dissections. J Endovasc Ther 2014;21:503-14.
20. Jones DW, Goodney PP, Nolan BW, Brooke BS, Fillinger MF, Powell RJ, et al. National trends in utilization, mortality, and survival after repair of type B aortic dissection in the Medicare population. J Vasc Surg 2014;60:11-9. 19.e1.
21. Weissler EH, Osazuwa-Peters OL, Greiner MA, Hughes GC, Long CA, Vemulapalli S, et al. National trends in repair for type B aortic dissection. Clin Cardiol 2021;44:1058-68.
22. Hanna JM, Andersen ND, Ganapathi AM, McCann RL, Hughes GC. Five-year results for endovascular repair of acute complicated type B aortic dissection. J Vasc Surg 2014;59:96-106.
23. Steuer J, Eriksson MO, Nyman R, Bjorck M, Wanhainen A. Early and long-term outcome after thoracic endovascular aortic repair (TEVAR) for acute complicated type B aortic dissection. Eur J Vasc Endovasc Surg 2011;41:318-23.
24. Wilkinson DA, Patel HJ, Williams DM, Dasika NL, Deeb GM. Early open and endovascular thoracic aortic repair for complicated type B aortic dissection. Ann Thorac Surg 2013;96:23-30; discussion: 230.
25. Melissano G, Bertoglio L, Rinaldi E, Civilini E, Tshomba Y, Kahlberg A, et al. Volume changes in aortic true and false lumen after the "PETTICOAT" procedure for type B aortic dissection. J Vasc Surg 2012;55:641-51.
26. Alsac JM, Girault A, El Batti S, Abou Rjeili M, Alomran F, Achouh P, et al. Experience of the Zenith Dissection Endovascular System in the emergency setting of malperfusion in acute type B dissections. J Vasc Surg 2014;59:645-50.
27. Lin Y, Dong S, Luo J, Bei W, Liu Q, Pang X, et al. Satisfactory long-term outcomes of thoracic endovascular aortic repair with a bare stent for acute complicated type B aortic dissections. J Endovasc Ther 2021;28:275-82.
28. Sobocinski J, Dias NV, Hongku K, Lombardi JV, Zhou Q, Saunders AT, 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. J Vasc Surg 2020;71:400-7.e2.
29. Nienaber CA, Kische S, Zeller T, Rehders TC, Schneider H, Lorenzen B, et al. Provisional extension to induce complete attachment after stent-graft placement in type B aortic dissection: the PETTICOAT concept. J Endovasc Ther 2006;13:738-46.
30. Fattori R, Montgomery D, Lovato L, Kische S, Di Eusanio M, Ince H, 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;6:876-82.
31. 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;66:924-30.
32. Patel JJ, Kasprzak P, Pfister K, Tsilimparis N, Kölbel T, Wahlgren C, et al. Early outcomes associated with use of the Zenith TX2 Dissection Endovascular Graft for the treatment of Stanford type B aortic dissection. J Vasc Surg 2021;74:547-55.
33. Kong X, Peng L, Wu F, Bi J, Pan H, Dai X. Distal bare metal stent implantation during thoracic endovascular aortic repair is beneficial to treat complicated type B aortic dissection: a systematic review and meta-analysis. Vascular 2021;29:499-508.
34. Qiu P, Zha B, Zhang X, Ye K, Qin J, Yang X, et al. A meta-analysis of combined proximal stent grafting with or without adjunctive distal bare stent for the management of aortic dissection. J Vasc Surg 2020;72:1109-20.e6.
35. Lombardi JV, Famularo M, Kratzberg J, Roeder BA. Effect of proximal fixation length on complications after endovascular repair of type B aortic dissection. J Vasc Surg 2021;73:1189-96.e3.
36. Rong D, Ge Y, Liu J, Liu X, Guo W. Combined proximal descending aortic endografting plus distal bare metal stenting (PETTICOAT technique) versus conventional proximal descending aortic stent graft repair for complicated type B aortic dissections. Cochrane Database Syst Rev 2019;2019:CD013149.

Submitted Apr 22, 2022; accepted Jun 30, 2022.

*Additional material for this article may be found online at www.jvascsurg.org.*

**Supplementary Table I (online only).** Summary of patient deaths

| Days after procedure | Cause of death | Relatedness (procedure, dissection) |
|---|---|---|
| 1 | Ischemic bowel | Not related: related to a pre-existing condition |
| 1 | Aortic dissection with resultant respiratory failure and cardiac arrest | Not related: related to presenting aortic dissection |
| 3 | Multiple organ failure | Not related: related to celiac artery and SMA occlusions before dissection stent placement |
| 5 | Brain dead due to stroke | Procedure related (dissection related) |
| 21 | Aortic rupture | Unable to be adjudicated |
| 57 | Type A aortic dissection with rupture | Not related: related to pre-existing type A dissection before device deployment |
| 66 | Unknown | Procedure related (dissection related): postoperatively the patient was ventilated and had a stroke; however, the terminal event is not clear |
| 96 | Unknown, found dead at home | Unable to be adjudicated |
| 170[a] | Angiosarcoma, cancer | Not related: related to other condition |
| 177 | Ischemic heart disease | Not related: related to pre-existing condition |
| 220 | Multiple organ failure | Not related: patient did not meet inclusion criteria |
| 240 | Respiratory failure | Not related: related to pneumonia with pre-existing lung cancer and COPD |
| 306 | Unknown | Unable to be adjudicated |
| 330 | Atherosclerotic cardiovascular disease | Unable to be adjudicated |
| 454 | Acute blood loss anemia and acute respiratory tract hemorrhage | Not related: related to respiratory tract hemorrhage |
| 848 | Coagulopathy | Not related: related to a pre-existing AAA condition |
| 1291 | Lung cancer | Not related: related to a pre-existing condition |
| 1589 | Cardiac arrest, coronary heart disease, and congestive heart failure | Not related: related to a pre-existing condition |
| 1714 | Metastatic cancer | Not related: related to pancreatic cancer |
| 1816 | Heart failure | Unable to be adjudicated |

*AAA*, Abdominal aortic aneurysm; *COPD*, chronic obstructive pulmonary disease; *SMA*, superior mesenteric artery.
[a]Did not receive a dissection stent at the procedure.

**Supplementary Table II (online only).** Details of secondary interventions

| Patient No. | Days after the procedure | Reason for intervention | Type of intervention |
|---|---|---|---|
| 1 | 2 | Abdominal discomfort and rapid expansion of the abdominal false lumen with probable pseudoaneurysm | Coil embolization |
| | 15 | Rapidly expanding AAA and possible pseudoaneurysm | Repair of the abdominal aorta and bilateral iliac arteries with removal of the previous stent-graft system |
| 2 | 5 | Right common iliac artery true lumen compression | Placement of the iliac stent |
| 3 | 6 | Right retained hemothorax | Video-assisted thoracoscopic surgery evacuation of the hematoma, decortication of the right lung, and flexible bronchoscopy |
| 4 | 12 | Bleeding from the right groin and right femoral pseudoaneurysm | Groin exploration with bovine patch repair of the right femoral artery |
| 5 | 17 | Left arm pain with diminished sensation | Left carotid to subclavian bypass, left brachial artery embolectomy |
| 6[a] | 50 | Back pain, obstruction/compromise of branch vessels, type I proximal and distal entry flow, and sealing reentry tear | Placement of the dissection stent and covered stent grafts for proximal and distal extension; ascending aorta to innominate and LCC artery bypass |
| 7 | 65 | Secondary entry tear just distal to the covered stent | Placement of two covered stent grafts |
| 8 | 131 | Device/component separation attributed to expanding false lumen | Coil embolization and placement of the renal stent |
| | 1190 | Lower extremity claudication, obstruction/compromise of branch vessels | Placement of the bifurcated AAA graft and iliac extension stent |
| 9 | 153 | Type I proximal entry flow and secondary entry tear | Ascending aorta and total arch replacement; innominate, LCC artery, and LSA reconstruction |
| 10 | 390 | Type I proximal and distal entry flow | Placement of three covered stent grafts for proximal and distal extension; balloon angioplasty |
| | 608 | Type I proximal entry flow, sinus of Valsalva aneurysm with aortic valve insufficiency secondary to bicuspid aortic valve | Composite aortic root replacement and total arch replacement |
| 11 | 530 | Worsening chronic abdominal pain and chest pain, abdominal tenderness, as well as a secondary tear at the distal end of the stent graft | Placement of the covered stent graft for distal extension and balloon angioplasty |
| | 655 | Abdominal pain, secondary tears in the distal infrarenal aorta and at the celiac artery, collateral flow from the lumbar arteries | Placement of the covered stent graft for distal extension, right and left iliac leg grafts; placement of stents in right renal, celiac, and SMA; coil embolization |
| | 711 | Bloody stools and abdominal pain, incomplete collapse of the proximal SMA stent previously deployed | Placement of the stent in SMA |
| | 920 | Worsening abdominal pain with radiation to the back and right thigh, paresthesia and numbness in the right leg when walking, and thrombus in the right CFA extending into the profunda and SFA | Embolectomy and thrombectomy of the right femoropopliteal artery |
| 12 | 650 | Fatigue, lethargy, failure to thrive; infected stent graft (indicated by positive blood cultures and imaging) | Graft explanted and conversion to open repair |
| 13 | 893 | New penetrating ulcer and aneurysmal degeneration distal to the stent grafts, but within the dissection stent | Placement of the covered stent graft |
| 14 | 1161 | New tear in the ascending thoracic aorta, intermittent chest pressure at rest and when walking quickly | Aortic valve and ascending aorta replacement |
| 15 | 1290 | New separate type A dissection | Ascending aortic arch replacement |
| 16 | 1528 | Acute type A dissection with involvement of aortic arch and aneurysmal ascending aorta | Open surgical repair of type A aortic arch dissection |

*AAA*, Abdominal aortic aneurysm; *CFA*, common femoral artery; *LCC*, left common carotid; *LSA*, left subclavian artery; *SFA*, superficial femoral artery; *SMA*, superior mesenteric artery.
[a]Did not receive a dissection stent at the procedure.

**Supplementary Table III (online only).** False lumen status by device region

| | Percentage of patients (n/N) | | | | | |
|---|---|---|---|---|---|---|
| | First postprocedure CT[a] | 1 year | 2 years | 3 years | 4 years | 5 years |
| Stent-graft region | | | | | | |
| All patients | | | | | | |
| Patent | 1.6 (1/61) | 0 (0/46) | 0 (0/40) | 0 (0/38) | 0 (0/34) | 0 (0/28) |
| Partially thrombosed | 42.6 (26/61) | 21.7 (10/46) | 27.5 (11/40) | 23.7 (9/38) | 14.7 (5/34) | 17.9 (5/28) |
| Completely thrombosed | 55.7 (34/61) | 69.6 (32/46) | 62.5 (25/40) | 63.2 (24/38) | 76.5 (26/34) | 75.0 (21/28) |
| No apparent false lumen | 0 (0/61) | 8.7 (4/46) | 10.0 (4/40) | 13.2 (5/38) | 8.8 (3/34) | 7.1 (2/28) |
| Patients with dissection stent(s) | | | | | | |
| Patent | 0 (0/48) | 0 (0/37) | 0 (0/31) | 0 (0/30) | 0 (0/27) | 0 (0/21) |
| Partially thrombosed | 41.7 (20/48) | 13.5 (5/37) | 19.4 (6/31) | 20.0 (6/30) | 11.1 (3/27) | 9.5 (2/21) |
| Completely thrombosed | 58.3 (28/48) | 78.4 (29/37) | 71.0 (22/31) | 66.7 (20/30) | 77.8 (21/27) | 81.0 (17/21) |
| No apparent false lumen | 0 (0/48) | 8.1 (3/37) | 9.7 (3/31) | 13.3 (4/30) | 11.1 (3/27) | 9.5 (2/21) |
| Patients without dissection stent | | | | | | |
| Patent | 7.7 (1/13) | 0 (0/9) | 0 (0/9) | 0 (0/8) | 0 (0/7) | 0 (0/7) |
| Partially thrombosed | 46.2 (6/13) | 55.6 (5/9) | 55.6 (5/9) | 37.5 (3/8) | 28.6 (2/7) | 42.9 (3/7) |
| Completely thrombosed | 46.2 (6/13) | 33.3 (3/9) | 33.3 (3/9) | 50.0 (4/8) | 71.4 (5/7) | 57.1 (4/7) |
| No apparent false lumen | 0 (0/13) | 11.1 (1/9) | 11.1 (1/9) | 12.5 (1/8) | 0 (0/7) | 0 (0/7) |
| Dissection stent region | | | | | | |
| Patients with dissection stent(s) | | | | | | |
| Patent | 10.4 (5/48) | 2.6 (1/39) | 0 (0/30) | 0 (0/30) | 0 (0/26) | 0 (0/21) |
| Partially thrombosed | 83.3 (40/48) | 79.5 (31/39) | 73.3 (22/30) | 70.0 (21/30) | 84.6 (22/26) | 61.9 (13/21) |
| Completely thrombosed | 6.3 (3/48) | 15.4 (6/39) | 16.7 (5/30) | 20.0 (6/30) | 11.5 (3/26) | 33.3 (7/21) |
| No apparent false lumen | 0 (0/48) | 2.6 (1/39) | 10.0 (3/30) | 10.0 (3/30) | 3.8 (1/26) | 4.8 (1/21) |
| Patients without dissection stent | NA | | | | | |

*CT*, Computed tomography; *NA*, not applicable.
[a]First postprocedure CT imaging performed before patient discharge or at 1-month follow-up.