

Fenestrated and branched endovascular aneurysm repair outcomes for type II and III thoracoabdominal aortic aneurysms

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Objective: Thoracoabdominal aortic aneurysm (TAAA) repair remains a challenging clinical pathology. Endovascular technology, in particular the evolution of fenestrated and branched (F/B) endografts used in endovascular aneurysm repair (EVAR) has provided a less invasive method of treating these complex aneurysms. This study evaluated the technical and clinical outcomes of F/B-EVAR for extensive type II and III TAAA.

Methods: Data from 354 high-risk patients enrolled in a physician-sponsored investigational device exemption trial (2004-2013) undergoing F/B-EVAR for type II and III TAAA were evaluated. Technical success, perioperative clinical outcomes, and midterm outcomes (36 months) for branch patency, reintervention, aneurysm-related death, and all-cause mortality were analyzed. Data are presented as mean \pm standard deviation and were assessed using Kaplan-Meier, univariate, and multivariate analysis.

Results: F/B-EVARs incorporating 1305 fenestration/branches were implanted with 96% of target vessels successfully stented. Completion aortography showed 2.8% patients had a type I or III endoleak. Procedure duration (6.0 ± 1.7 vs 5.5 ± 1.6 hours; $P < .01$) and hospital stay (13.1 ± 10.1 vs 10.2 ± 7.4 days; $P < .01$) were longer for type II TAAA. Perioperative mortality was greater in type II repairs (7.0% vs 3.5%; $P < .001$). Permanent spinal cord ischemia occurred in 4% and renal failure requiring hemodialysis occurred in 2.8% of patients. Twenty-seven branches (7.6%) required reintervention for stenosis or occlusion; and celiac artery, superior mesenteric artery, and renal artery secondary patency at 36 months was 96% (95% confidence interval [CI], 0.93-0.99), 98% (95% CI, 0.97-1.0), and 98% (95% CI, 0.96-1.0), respectively. Eighty endoleak repairs were performed in 67 patients, including 55 branch-related endoleaks, 4 type Ia, 5 type Ib, and 15 type II endoleaks. At 36 months, freedom from aneurysm-related death was 91% (95% CI, 0.88-0.95), and freedom from all-cause mortality was 57% (95% CI, 0.50-0.63). The treatment of type II TAAA ($P < .01$), age ($P < .01$), and chronic obstructive pulmonary disease ($P < .05$) negatively affected survival.

Conclusions: F/B-EVAR is a robust treatment option for patients at increased risk for conventional repair of extensive TAAAs. Technical success and branch patency are excellent, but some patients will require reintervention for branch-related endoleak. Aneurysm extent portends a higher risk of perioperative and long-term morbidity and mortality. Additional efforts are needed to improve outcomes and understand the utility of this treatment option in the general TAAA population. (*J Vasc Surg* 2016;63:930-42.)

Thoracoabdominal aortic aneurysms (TAAAs) remain a challenging clinical pathology. Decades of experience have significantly improved outcomes for those patients fit enough for conventional surgery.¹⁻⁶ Endovascular aneurysm repair (EVAR) therapy offers a potential alternative for

patients who are physiologically high risk for conventional surgery.⁷ Initially, advanced EVAR was limited to short-necked abdominal aortic aneurysms,⁸ but further aortic coverage was attainable with the development of both the aortic endograft and bridging stent graft technology. Global experience with fenestrated endografts to treat juxtarenal abdominal aortic aneurysms (JRAAA) and type IV TAAA is rapidly growing, with excellent long-term results.^{9,10}

Early experience with the application of fenestrated and branched endografts (F/B-EVAR) to treat more extensive TAAA demonstrates acceptable outcomes.¹¹⁻¹³ Perioperative mortality, renal failure, and spinal cord ischemia (SCI) rates challenge the outcomes attained for conventional surgery in high-volume centers. Most series to date, however, remain relatively small and report outcomes from a heterogeneous group of aneurysm extents, including JRAAA and types I, II, III, and IV TAAA.¹⁴⁻¹⁹ Despite improvements in the technology, this approach is limited by the development of SCI, branch-related endoleaks, and branch occlusion.²⁰⁻²³

Longer-term outcomes are excellent but highlight the need for a better understanding of the effects of endograft

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treatment of extensive TAAA. The aim of this current study was to evaluate outcomes for patients with extensive TAAA (type II and III) treated with F/B-EVAR.

METHODS

Patients who underwent F/B-EVAR for type II and III TAAA under a physician-sponsored investigational device exemption protocol (2004 and 2013) were included in this series ([ClinicalTrials.gov](https://clinicaltrials.gov/ct2/show/study/NCT00583050) identifier NCT00583050). Patients undergoing F/B-EVAR for type IV and JRAAA were excluded and previously reported.¹⁰ This study was approved by the Cleveland Clinic Institutional Review Board, and all patients signed an informed consent agreeing to participate. Details of the device design and implantation methods have previously been described.¹¹⁻¹³ Design of the devices was determined by the treating physician and evolved over time based on device design alterations and surgeon experience.

Patient demographics, operative and outcomes data, and imaging end points were prospectively recorded in the Oracle Clinical database (Oracle Corp, Redwood Shores, Calif). All patients were considered high risk for conventional surgery.^{7,24} A standard procedure for assessing imaging outcomes events was used, and images were assessed by the treating vascular surgeon and a trained vascular imaging specialist.^{10,21,25} Clinical and imaging follow-up occurred in the 30-day postoperative period and annually thereafter, and more frequently as the clinical scenario required, as previously described.¹⁰ Technical success was defined as placement of the aortic graft with successful stenting of all target vessels. Other end points assessed included perioperative morbidity, acute and late mortality, branch vessel patency, endoleak development, and freedom from reintervention.

Data are summarized using mean \pm standard deviation for continuous variables. Statistical analyses were performed using the χ^2 test or the Fisher exact test for categorical variables. Continuous variables were compared using the Student *t*-test or the Wilcoxon rank sum test. Kaplan-Meier curves were used for time-to-event analyses for survival, freedom from reintervention, and branch vessel patency. Primary patency was defined as stenosis or occlusion of any target vessel or an intervention of the vessel to re-establish flow, or both. Multivariable analyses for all-cause and aneurysm-related mortality, need for secondary intervention, and permanent SCI were performed to determine independent factors related to each outcome. Demographic characteristics, medical history, and aneurysm type were all considered for inclusion into each of the models. Bootstrap sampling methods were used to determine which variables would be included in the final models. Candidate variables were retained at the $\alpha = .20$ level. Analyses were performed using SAS 9.2 software (SAS Institute Inc, Cary, NC).

RESULTS

Patient information. A total of 354 patients underwent type II (128; 36%) or type III (226; 64%) TAAA

Table I. Patient demographics

Variable	Total (N = 354)	Type II (n = 128)	Type III (n = 226)	P value
Mean age \pm SD, years	73.5 \pm 8.36	71.9 \pm 7.97	74.4 \pm 8.45	.003
Male	270 (76.3)	82 (64.1)	188 (83.2)	<.001
Smoking status				.765
Never	27 (8.1)	11 (9.2)	16 (7.5)	
Former	248 (74.5)	90 (75)	158 (74.2)	
Current	58 (17.4)	19 (15.8)	39 (18.3)	
Coronary artery disease	155 (43.8)	43 (33.6)	112 (49.6)	.004
Diabetes	52 (14.7)	14 (10.9)	38 (16.8)	.133
Renal disease				.459
Renal failure	9 (2.5)	2 (1.6)	7 (3.1)	
Renal insufficiency	66 (18.6)	21 (16.4)	45 (19.9)	
Aortic or mitral valve disease	25 (7.1)	14 (10.9)	11 (4.9)	.032
Arrhythmia or pacemaker use	88 (24.9)	34 (26.6)	54 (23.9)	.577
COPD	109 (30.8)	42 (32.8)	67 (29.6)	.535
Lung cancer	9 (2.5)	3 (2.3)	6 (2.7)	1.000
ASA classification				
1	0 (0.0)	0 (0.0)	0 (0.0)	
2	1 (0.3)	0 (0.0)	1 (0.4)	
3	168 (47.6)	55 (43.0)	113 (50.2)	
4	182 (51.6)	73 (57.0)	109 (48.4)	
5	2 (0.6)	0 (0.0)	2 (0.9)	

ASA, American Society of Anesthesiologists Physical Status Classification; COPD, chronic obstructive pulmonary disease; SD, standard deviation. Data are presented as number (%) unless otherwise indicated.

repair (Table I). Nonstaged prior aortic surgery was performed in 153 patients (43.2%), including 18 EVAR (5.1%), 25 thoracic endovascular aortic repair (TEVAR; 7.1%), 95 open AAA repair (26.8%), and 27 open TAA repair (7.6%).

F/B-EVAR repairs were planned to incorporate 1320 target vessels including 323 celiac arteries (CAs), 334 superior mesenteric arteries (SMAs), 331 left renal arteries (RAs), and 332 right RAs. Grafts incorporated 5 target vessels in 7 patients (1.9%) for multiple RAs, 4 target vessels in 261 patients (73.7%), 3 target vessels in 69 patients (19.4%), and 2 target vessels in 17 patients (4.8%). Grafts with reinforced fenestrations for visceral vessels were implanted in 274 patients (77.4%) and included 53.1% of type II repairs and 91.2% of type III repairs. The graft design in 80 patients (22.6%) incorporated a helical direction branch to the CA only ($n = 37$), SMA only ($n = 8$), or both CA and SMA ($n = 35$). All remaining visceral branches and all RA branches were constructed with reinforced fenestrations.

Sixty-six patients (18%) underwent related to iliofemoral access concurrent with F/B-EVAR. Twenty-one had a concomitant procedure, including 4 (1.1%) iliac artery conduits, 7 (2%) femoral artery aneurysm repairs, 4 (1.1%) conduits sewn in an end-to-side fashion to the femoral limb of a prior aortobifemoral bypass, and 5

femoral conduits sutured to the femoral artery after concomitant endarterectomy and patch angioplasty. One patient underwent recanalization and stenting of an iliac system with an endoconduit.

Iliofemoral reconstruction was performed in 45 patients (72%) at an antecedent surgery, comprising three bilateral femoral artery aneurysm repairs and 41 iliofemoral conduits. Anastomoses were performed to the common iliac artery in an end-to-side configuration ($n = 7$) or an end-to-end configuration ($n = 34$) with ligation of the distal common iliac artery. Six were performed in conjunction with a first-stage TEVAR. In one patient, an aortofemoral conduit based off of a prior aortic graft was placed in the setting of an occluded common iliac artery. All conduits were sewn distally to the common femoral artery in an end-to-side fashion. No conduit required thrombectomy before use. Efforts were made to place conduits while the custom graft was being constructed, although in some instances, conduits were placed just before F/B-EVAR to limit the amount of travel for patients. The mean time from staged iliofemoral surgery until F/B-EVAR placement was 3.1 ± 0.4 months (median, 2.7; range 2 days-16 months). Seven delays were >4 months' duration due to insurance-related delay ($n = 1$), new-onset medical problem ($n = 2$), and surgeon-related delay ($n = 4$).

F/B-EVAR was performed in 39 patients (11%) in an intentionally staged fashion, with placement of a TEVAR component at an antecedent surgery (34 for type II and 6 for type III). Procedures were performed at a mean of 3.9 ± 0.3 months before F/B-EVAR repair (median, 3.7 months; range, 1-9 months). Longer times until second repair were secondary to groin wound infection ($n = 1$), insurance delay ($n = 1$), patient refusal ($n = 3$), and surgeon-related delay ($n = 7$).

SCI symptoms developed in one patient after the first stage and resolved before F/B-EVAR. One patient ruptured after the second stage (3 months) and underwent urgent F/B-EVAR.

The overall analysis excluded five patients who underwent an antecedent conduit or a planned staged TEVAR and did not proceed to F/B-EVAR. Three patients died after iliac conduit placement. One patient underwent a redo aortofemoral bypass for an occluded graft limb in the setting of limb-threatening ischemia. The remaining two underwent standard conduits. All three patients were discharged from the hospital after their conduit surgery (hospital stays of 3-7 days) but died before F/B-EVAR. Two causes of death are unknown, and one patient died secondary to new-onset cardiac dysrhythmia. Two patients were discharged from the hospital after a first-stage TEVAR but died before F/B-EVAR. One patient died of complications of chemoradiation therapy for cancer diagnosed after the TEVAR, and the other died of a subdural hematoma after a fall.

In 14 patients (4%), an elephant trunk graft procedure was performed before F/B-EVAR to provide a suitable proximal landing zone. In addition, 26 (7.3%) underwent concomitant placement of a hypogastric branched

endograft for an iliac artery aneurysm, with four patients undergoing bilateral hypogastric branched graft placement.

Intraoperative information. One patient was converted to open repair due to device maldeployment. Mean procedure duration was 5.7 ± 1.7 hours (range, 1.6-12.0 hours). Procedure time (from skin incision to skin closure) was 6.0 ± 1.8 hours (range, 1.6-11.4 hours) for type II repairs and 5.5 ± 1.6 hours (range 2.1-12.0) for type III repairs ($P = .004$). Procedure duration varied significantly ($P = .022$) over time with a bimodal distribution. The mean procedure time in 2004 was 6.9 ± 1.7 hours and decreased annually to 4.8 ± 1.4 hours through 2008. In 2009, two additional surgeons increased operative time to 6.9 ± 2.2 hours, which reduced to 5.4 ± 1.6 hours over the ensuing years.

The mean volume of packed red cell transfusion was 542 ± 729 mL (660 ± 757 mL in type II repairs and 476 ± 706 mL in type III repairs; $P = .002$). Average contrast delivered was 143.5 ± 78.1 mL (157.6 ± 77.6 mL for type II repairs and 135.7 ± 77.5 mL for type III repairs; $P = .002$).

Technical success was 94.1%. Technical failure occurred in 21 patients (5.9%), with the inability to incorporate 13 CAs (4%) and eight RAs (1%). Four of the failed CAs were planned helical branches, and seven were reinforced fenestrations. Cannulation of the vessel in 12 of the CA cases was inhibited by a high-grade stenosis ($>95\%$) or occlusion at the CA. The CA in seven cases was angiographically occluded at the time of the F/B-EVAR, despite appearing patent on preoperative computed tomography imaging. In all of these cases, the fenestration or branch was covered with an aortic component to prevent endoleak. One CA failure was secondary to malalignment of the aortic component. This patient underwent placement of a celiac bridging stent at a later date.

Reasons for RA stent deployment failures included maldeployment in 2 aortic grafts, high-grade ($>95\%$) stenosis in 2 RAs, tortuosity in 1 RA, and inability to cannulate 3 RAs through the fenestration. In all cases, the fenestration was left open and an attempt was made to incorporate the vessel at a later time: four were subsequently stented, and four underwent occlusion of the fenestration with loss of the RA.

Ten patients (2.8%) had a suspected type I or type III endoleak on completion angiography despite having stented all target vessels. Classification of the leak was based on the operating surgeon's interpretation, and reintervention was not immediately performed because the exact etiology of the leak could not be determined. Contrasted, cross-sectional imaging was available for eight patients during the postoperative period, revealing two with endoleak resolution, one endoleak arising from an iliac limb, and three branch-related endoleaks (two RA, one SMA). All branch- and limb-related endoleaks resolved on reintervention. Two patients did not receive contrasted computed tomography in follow-up due to renal function. One demonstrated endoleak resolution on duplex ultrasound

Table II. Major perioperative morbidity

Morbidity	No. (%)
Return to operating room	13 (3.7)
SCI	31 (8.8)
Permanent	14 (4)
Renal failure	18 (5.1)
Requiring hemodialysis	10 (2.8)
Increase in creatinine ^a	8 (2.3)
Myocardial infarction	10 (2.8)
Non-ST elevation	7 (2.0)
ST elevation	3 (0.8)
Cardiac dysrhythmia	24 (6.8)
Atrial fibrillation	16 (4.5)
Other	8 (2.3)
Respiratory failure	32 (9.0)
Branch vessel occlusion (without reintervention)	4 (1.1)
Hematologic	9 (2.5)
DVT/PE	3 (0.8)
Hemorrhage not requiring return to operating room	2 (0.6)
Wound complications	9 (2.5)
Dehiscence	2 (0.6)
Hematoma	7 (2.0)
Gastrointestinal disorder	13 (3.7)
Gastrointestinal bleeding	3 (0.8)
Ileus	6 (1.7)
Pancreatitis	1 (0.3)
Liver failure	1 (0.3)
Ischemic colitis	2 (0.6)
Postoperative stroke	8 (2.3)
Infectious	19 (5.4)
Urinary tract infection	10 (2.8)
Pneumonia	5 (1.4)
<i>Clostridium difficile</i> colitis	4 (1.1)

DVT/PE, Deep venous thrombosis/pulmonary embolism; SCI, spinal cord ischemia.

^aSerum creatinine increase of >30%.

imaging, and the second died in the perioperative period after developing SCI.

Perioperative outcomes. Overall length of stay was 11.3 ± 8.59 days (range, 2-78 days) and was longer in type II repairs (13.1 ± 10.1 days) vs type III repairs (10.2 ± 7.4 days; $P < .001$). Postoperative 30-day mortality occurred in 17 patients (4.8%). There was no factor that was associated with an increased risk of perioperative mortality. The causes of death included myocardial infarction ($n = 2$), hepatic failure ($n = 1$), stroke ($n = 2$), respiratory failure ($n = 2$), and multisystem organ failure ($n = 10$). The underlying etiology in the patients who developed multisystem organ failure was attributable to ischemic colitis ($n = 2$), pancreatitis ($n = 1$), myocardial infarction ($n = 1$), and pneumonia ($n = 5$). Three of the five patients that developed pneumonia had developed a dense paraplegia immediately postoperatively related to SCI.

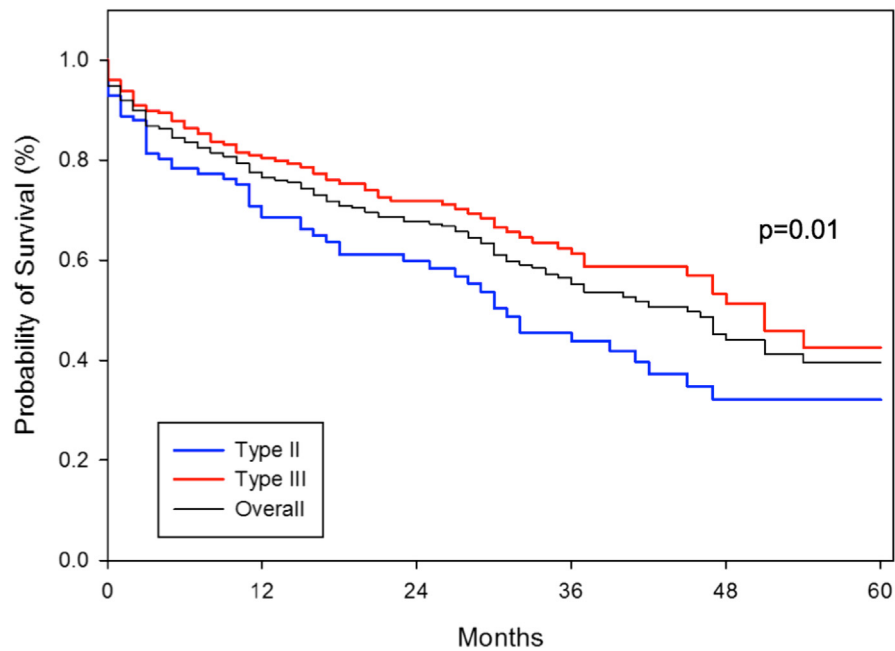
Thirty-day major morbidity occurred in 142 patients (40%; Table II). The only factor that was related to the development of an adverse event in the perioperative period was longer procedure duration (odds ratio, 1.215; $P = .004$). Thirteen patients (3.7%) underwent reoperation in the perioperative period due to access site hemorrhage

($n = 4$), external iliac artery/femoral artery occlusion ($n = 3$), lower extremity compartment syndrome ($n = 1$), renal hemorrhage ($n = 1$), retrograde aortic dissection ($n = 2$), kinking of the thoracic component of the stent graft ($n = 1$), and aortic rupture ($n = 1$). The patient with aortic rupture had an unstented RA fenestration, and a ruptured aneurysm occurred on postoperative day 2. The patient underwent occlusion of the reinforced fenestration. Two patients were noted to have retrograde aortic dissection on postoperative imaging and underwent proximal TEVAR extension. One patient underwent coil embolization of an RA branch for hemodynamic instability related to a subcapsular hematoma.

SCI occurred in 31 patients (8.8%) and was permanent in 14 (4%). SCI developed in 21 patients (16.4%) undergoing type II repair, but occurred in only 10 (4.4%) of the type III repairs ($P < .001$). SCI symptoms were permanent in 10 patients (7.8%) with type II TAAA repair and in four patients (1.8%) with type III TAAA repair ($P = .005$). SCI development was related to longer procedure duration (odds ratio, 1.67; $P = .001$). Acute kidney injury (defined as a creatinine rise >30% above baseline) occurred in 10 patients (2.8%), affecting seven (5.5%) of the type II repairs and three (1.3%) of the type III repairs ($P = .04$). An additional eight patients (2.3%) developed a transient increase (>30%) in their serum creatinine but did not require hemodialysis.

Midterm outcomes. Mean follow-up was 22 ± 19 months. Freedom from all-cause mortality was 57% (95% confidence interval [CI], 0.50-0.63) at 36 months, 46% (95% CI, 0.34-0.57) in type II TAAA repairs, and 62% (95% CI, 0.55-0.70) in type III repairs ($P = .01$; Fig 1). Factors associated with mortality include repair of a type II TAAA (hazard ratio [HR], 1.739; 95% CI, 1.226, 2.467; $P = .002$), age (HR, 1.031; 95% CI, 1.008-1.054; $P = .008$), cerebrovascular disease (HR, 1.620; 95% CI, 1.096-2.394; $P = .016$), chronic obstructive pulmonary disease (HR, 1.507; 95% CI, 1.055-2.153; $P = .024$), and higher American Society of Anesthesiologists Physical Status Classification ($P = .001$). Freedom from aneurysm-related death at 36 months was 91% (95% CI, 0.88-0.95; Fig 2) and did not differ between type II and type III repairs.

Freedom from unplanned secondary intervention was 54% (95% CI, 0.47-0.61) at 36 months (Fig 3) and was greater in type III repairs (58% [95% CI, 0.50-0.67] vs 45% [95% CI, 0.33-0.56]; $P = .001$). The only factor associated with the need for reintervention was a history of prior, unstaged TEVAR (HR, 2.381; 95% CI, 1.268-4.473; $P = .007$). Secondary procedures were required in 129 patients (36.3%), including 13 (3.7%) perioperative reoperations, as outlined above, 27 (7.6%) reinterventions to maintain branch vessel patency, and 67 (18.9%) for an endoleak. An additional 24 reinterventions occurred at a mean follow up of 21.3 ± 3.7 months (range, 2-66 months). Seven secondary procedures were performed for component separation without endoleak development; five involved a branch component, and two involved the



	1 month	12 months	24 months	36 months	48 months	60 months
Number at Risk	329	212	147	82	38	18
Number of Events	18	71	94	113	125	129
Cumulative Subjects Censored	5	69	111	157	189	205
Kaplan-Meier Estimate	0.95	0.78	0.68	0.57	0.45	0.40
95% Confidence Interval	(0.93, 0.97)	(0.73, 0.82)	(0.62, 0.73)	(0.50, 0.63)	(0.37, 0.53)	(0.31, 0.48)

Type II	1 month	12 months	24 months	36 months	48 months	60 months
Number at Risk	114	63	44	27	11	9
Number of Events	9	32	41	50	56	56
Cumulative Subjects Censored	4	32	42	50	60	62
Kaplan-Meier Estimate	0.93	0.71	0.60	0.46	0.32	0.32
95% Confidence Interval	(0.88, 0.97)	(0.62, 0.79)	(0.50, 0.70)	(0.34, 0.57)	(0.20, 0.44)	(0.20, 0.44)

Type III	1 month	12 months	24 months	36 months	48 months	60 months
Number at Risk	215	149	103	55	27	9
Number of Events	9	39	53	63	69	73
Cumulative Subjects Censored	1	37	69	107	129	143
Kaplan-Meier Estimate	0.96	0.81	0.72	0.62	0.53	0.43
95% Confidence Interval	(0.93, 0.99)	(0.76, 0.86)	(0.65, 0.78)	(0.55, 0.70)	(0.44, 0.63)	(0.30, 0.55)

Fig 1. Kaplan-Meier analysis of survival after fenestrated/branched endovascular aneurysm repair (F/B-EVAR) for extensive thoracoabdominal aortic aneurysms (TAAAs). Overall survival is represented by the *black line*, and overall survival is lower for those undergoing repair for a type II TAAA (*blue line*) compared with type III TAAA (*red line*).

main body. Four reinterventions were for femoral pseudoaneurysm at the access site. Three reinterventions were performed for chronic lower extremity ischemia; of which, two involved stenting of a dissected external iliac artery, and the third involved a femoral endarterectomy with patch angioplasty. One patient required endovascular repair for expansion of an iliac artery aneurysm. Nine patients underwent an aortic-related reintervention at a mean follow up of 23.8 ± 4.3 months. An ascending aortic aneurysm was repaired in one patient, and five patients underwent TEVAR for proximal aortic expansion, one in an emergent fashion. Two of the patients with proximal degeneration had a new aortic dissection. All of these patients had

initially undergone repair of a type III TAAA. Three patients underwent subsequent repair of an AAA, two for ruptures. All of these patients had prior AAA repair with tube grafts involving an anastomosis to the aortic bifurcation with aneurysmal degeneration at this site.

Primary CA, SMA, and left RA and right RA patency at 36 months was 96%, 95%, 94%, and 92%, respectively (Fig 4). During follow up, 27 patients (7.6%) underwent a reintervention on 29 branches for target vessel occlusion or stenosis, resulting in 36-month CA, SMA and RA secondary patency rates of 96% (95% CI, 0.93-0.99), 98% (95% CI, 0.97-1.0), and 98% (95% CI, 0.96-1.0), respectively (Fig 4).

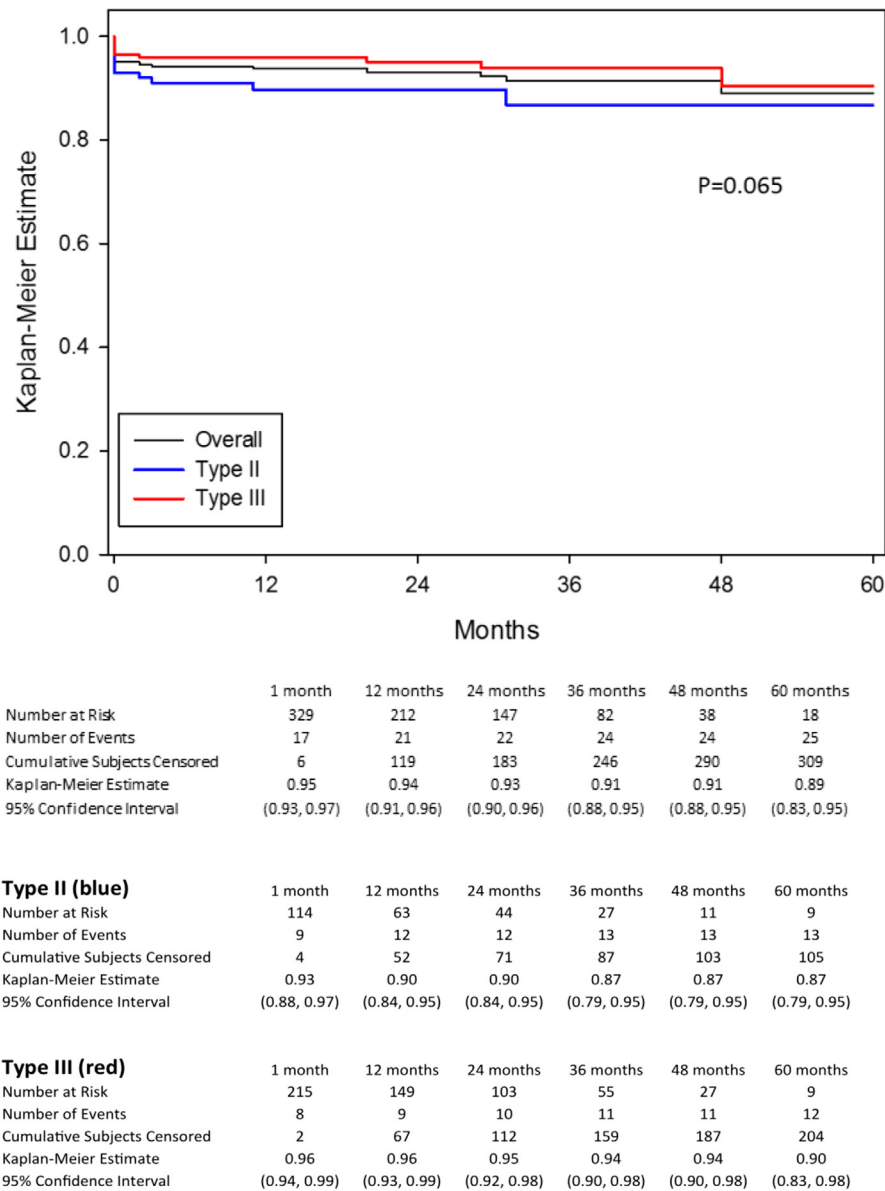


Fig 2. Kaplan-Meier analysis of freedom from aneurysm-related mortality (*black line*). The outcomes were unaffected by the extent of aneurysm treated (type II [*blue line*] vs type III [*red line*]).

Endoleak repair was performed in 67 patients (18.9%) to treat 80 endoleaks. Four patients (1.1%) underwent additional TEVAR for the development of a type Ia endoleak, and five patients (1.4%) underwent treatment for a type Ib endoleak. The proximal endoleaks developed secondary to migration of the main body graft due to progression of proximal aortic dilation. The distal endoleaks involved the common iliac artery ($n = 1$), external iliac artery ($n = 1$), and the hypogastric artery as part of a hypogastric branched endograft ($n = 3$). Type II endoleaks underwent embolization in 15 patients (4.2%) due to an expanding aneurysm. Separation of the F/B component and another aortic component developed in one patient,

resulting in a type III endoleak. In total, 55 (4.2% of branches) branch-related endoleaks were treated involving 9 CAs (2.8%), 15 SMAs (4.5%), 15 left RAs (4.5%), and 15 right RAs (4.5%) in 42 patients (11.9%). Stent fracture occurred in eight patients (2.3%), resulting in a branch-related endoleak.

An analysis of composite outcomes is presented in Fig 5 and represents freedom from first endoleak occurrence, migration, growth, secondary intervention, permanent SCI, or branch vessel occlusion. Factors that were associated with these events included treatment of a type II TAAA ($P = .002$), prior unplanned TEVAR ($P = .012$), and longer procedural duration ($P = .036$).

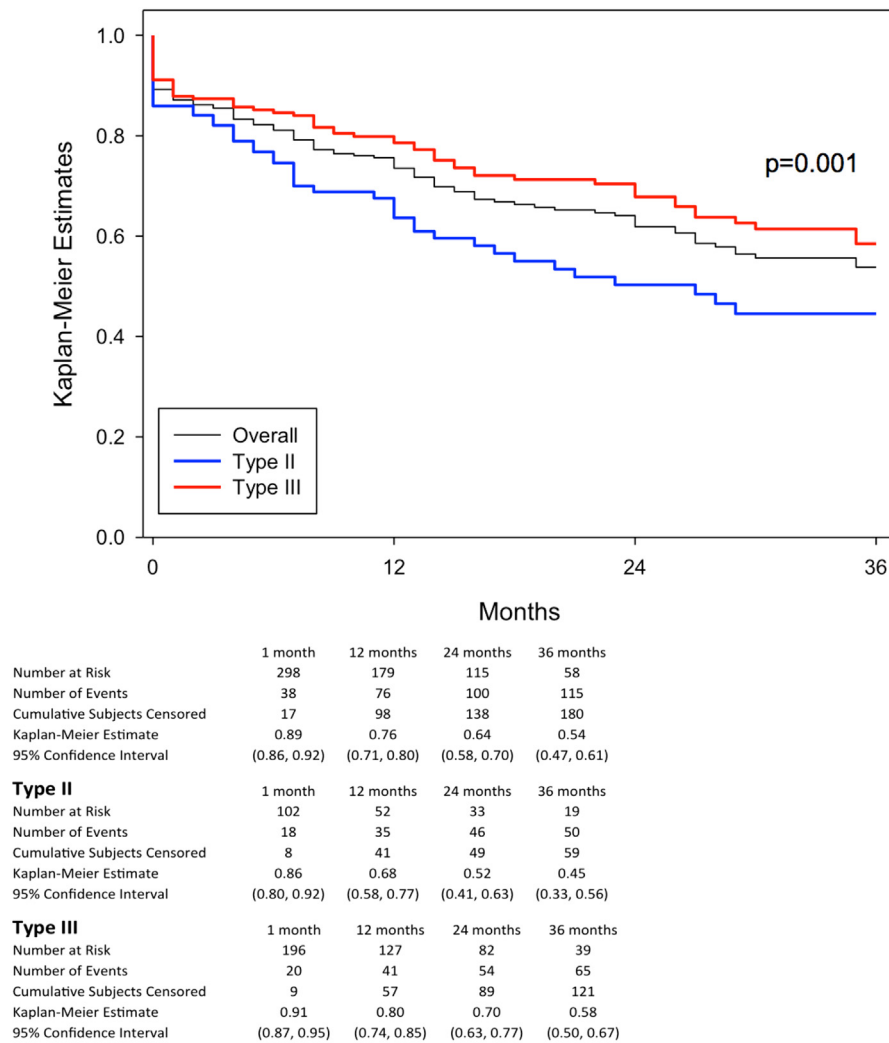


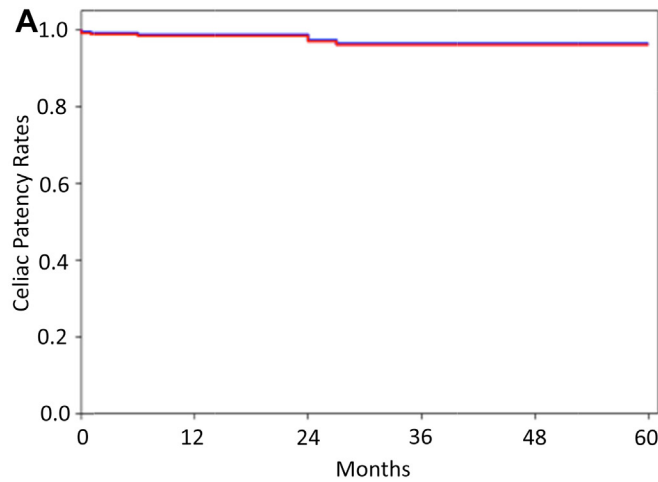
Fig 3. Kaplan-Meier analysis of freedom from reintervention (*black line*). Reintervention rates were higher in those patients treated for more extensive type II aneurysms (*blue line*) compared with those undergoing type III thoracoabdominal aortic aneurysm (TAAA) repair (*red line*).

DISCUSSION

Extensive TAAAs remain a challenging clinical pathology. The current study evaluated outcomes of F/B-EVAR for type II and III TAAA during a 9-year period. This represents the progression from our first patient treated to our more contemporary understanding of the application of this technology. Although analyzed in total, the outcomes here represent an evolution in device development, patient selection, procedural performance, and three separate physician learning curves. Overall perioperative mortality was 4.8% and was higher in those undergoing type II repair. These outcomes are similar to series evaluating F/B-EVAR for all extent TAAA, including type IV and JRAAA, with reported perioperative mortality rates of 3% to 9%.^{14,17,19,26,27} Contemporary series from high-volume centers report perioperative mortality rates after

open surgery of 5% to 10%, making F/B-EVAR competitive.^{1,3,5,6,28}

Despite the low mortality rate, perioperative morbidity is high at 40%. This appears to be related to exacerbation of patients' comorbidities weighted by the development of myocardial infarction, cardiac dysrhythmia, and pulmonary dysfunction. Other morbidities, however, are directly related to the procedure, including reoperation, renal failure, and SCI. These results, however, mirror those reported after conventional surgery.^{5,6,28,29} An analysis of the American College of Surgeons National Surgical Quality Improvement Program outcomes demonstrates similar perioperative morbidity, with the highest attributable to pulmonary dysfunction at nearly 40%.³⁰ In fact, outcomes from the WINDOWS (Medical and Economical Evaluation of Endovascular Therapy of Complex Aortic Aneurysms

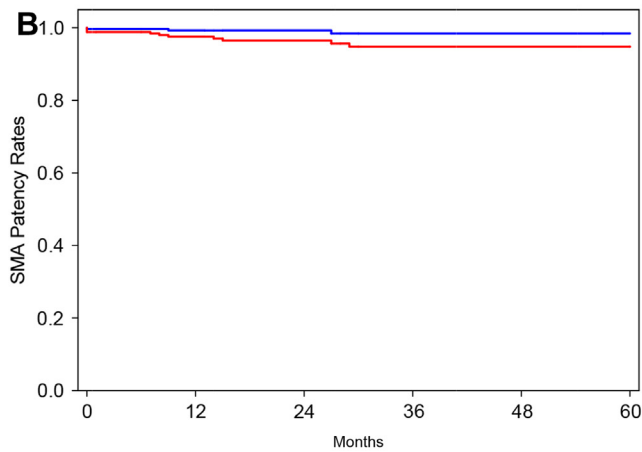


Primary patency (red)

	1 month	12 months	24 months	36 months	48 months	60 months
Number at Risk	315	201	138	72	34	18
Number of Events	3	5	5	8	8	8
Cumulative Subjects Censored	18	130	193	256	294	310
Kaplan-Meier Estimate	0.99	0.98	0.98	0.96	0.96	0.96
95% Confidence Interval	(0.98, 1.00)	(0.97, 1.00)	(0.97, 1.00)	(0.93, 0.99)	(0.93, 0.99)	(0.93, 0.99)

Secondary patency (blue)

	1 month	12 months	24 months	36 months	48 months	60 months
Number at Risk	316	201	138	72	34	18
Number of Events	2	4	4	7	7	7
Cumulative Subjects Censored	18	131	194	257	295	311
Kaplan-Meier Estimate	0.99	0.99	0.99	0.96	0.96	0.96
95% Confidence Interval	(0.99, 1.00)	(0.97, 1.00)	(0.97, 1.00)	(0.93, 0.99)	(0.93, 0.99)	(0.93, 0.99)



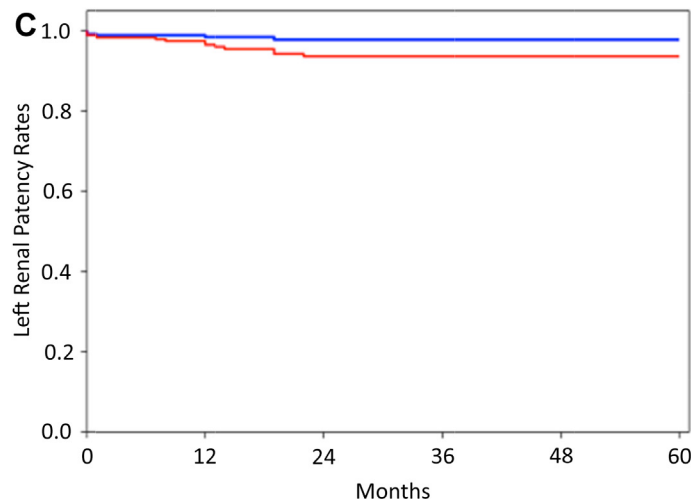
Primary patency (red)

	1 month	12 months	24 months	36 months	48 months	60 months
Number at Risk	327	207	142	80	38	18
Number of Events	4	7	9	11	11	11
Cumulative Subjects Censored	21	138	201	261	303	323
Kaplan-Meier Estimate	0.99	0.98	0.97	0.95	0.95	0.95
95% Confidence Interval	(0.98, 1.00)	(0.96, 0.99)	(0.94, 0.99)	(0.92, 0.98)	(0.92, 0.98)	(0.92, 0.98)

Secondary patency (blue)

	1 month	12 months	24 months	36 months	48 months	60 months
Number at Risk	329	211	146	80	38	18
Number of Events	1	2	2	3	3	3
Cumulative Subjects Censored	22	139	204	269	311	331
Kaplan-Meier Estimate	1.00	0.99	0.99	0.98	0.98	0.98
95% Confidence Interval	(0.99, 1.00)	(0.98, 1.00)	(0.98, 1.00)	(0.97, 1.00)	(0.97, 1.00)	(0.97, 1.00)

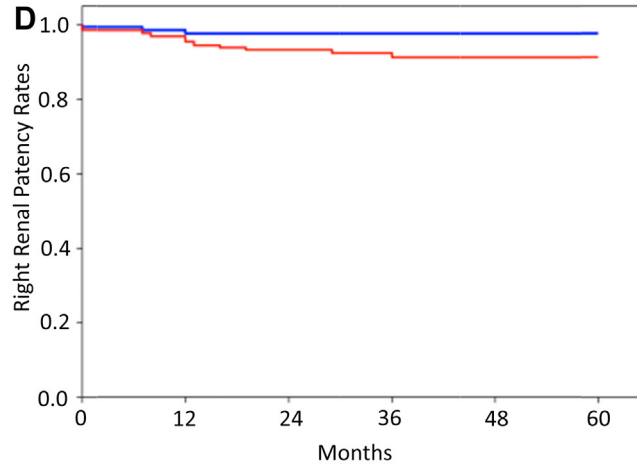
Fig 4. Primary (*blue line*) and secondary (*red line*) branch vessel patency rates are shown for (A) celiac artery (CA), (B) superior mesenteric artery (SMA), (C) left renal artery (RA), and (D) right RA.

**Primary patency (red)**

	1 month	12 months	24 months	36 months	48 months	60 months
Number at Risk	325	209	142	80	36	16
Number of Events	4	8	15	15	15	15
Cumulative Subjects Censored	23	135	195	257	301	321
Kaplan-Meier Estimate	0.99	0.97	0.94	0.94	0.94	0.94
95% Confidence Interval	(0.98, 1.00)	(0.96, 0.99)	(0.90, 0.97)	(0.90, 0.97)	(0.90, 0.97)	(0.90, 0.97)

Secondary patency (blue)

	1 month	12 months	24 months	36 months	48 months	60 months
Number at Risk	326	210	144	81	37	17
Number of Events	3	4	6	6	6	6
Cumulative Subjects Censored	23	138	202	265	309	329
Kaplan-Meier Estimate	0.99	0.99	0.98	0.98	0.98	0.98
95% Confidence Interval	(0.98, 1.00)	(0.98, 1.00)	(0.96, 1.00)	(0.96, 1.00)	(0.96, 1.00)	(0.96, 1.00)

**Primary patency (red)**

	1 month	12 months	24 months	36 months	48 months	60 months
Number at Risk	324	207	140	79	36	16
Number of Events	5	9	16	17	18	18
Cumulative Subjects Censored	23	136	196	256	298	318
Kaplan-Meier Estimate	0.99	0.97	0.93	0.92	0.91	0.91
95% Confidence Interval	(0.97, 1.00)	(0.95, 0.99)	(0.90, 0.97)	(0.89, 0.96)	(0.87, 0.96)	(0.87, 0.96)

Secondary patency (blue)

	1 month	12 months	24 months	36 months	48 months	60 months
Number at Risk	327	210	144	80	36	16
Number of Events	2	4	6	6	6	6
Cumulative Subjects Censored	23	138	202	266	310	330
Kaplan-Meier Estimate	0.99	0.99	0.98	0.98	0.98	0.98
95% Confidence Interval	(0.99, 1.00)	(0.97, 1.00)	(0.96, 1.00)	(0.96, 1.00)	(0.96, 1.00)	(0.96, 1.00)

Fig 4. Continued.

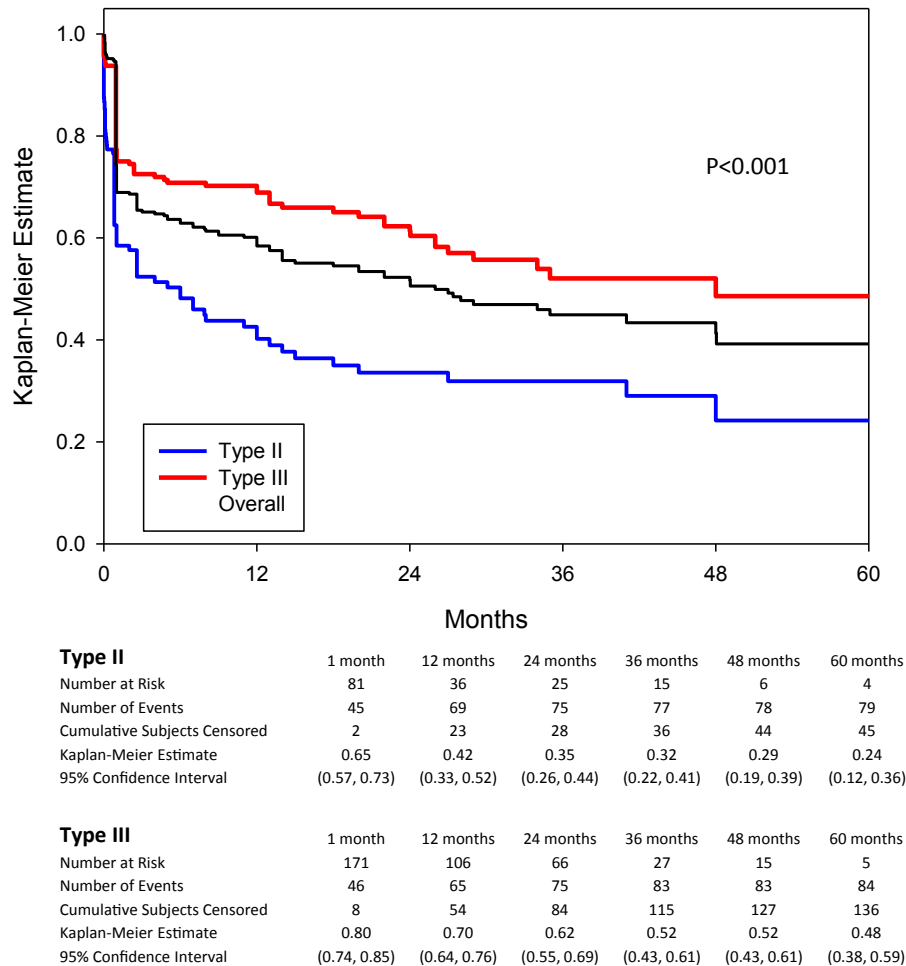


Fig 5. Composite end point analysis demonstrates freedom from first occurrence of endoleak, migration, aneurysm growth, secondary intervention, permanent spinal cord ischemia (SCI), or occlusion for the entire group (*black line*), type II thoracoabdominal aortic aneurysm (TAAA; *blue line*), and type III TAAA (*red line*).

[Para- & Supra- Renal Abdominal Aortic Aneurysms, Type 4 THORACO-Abdominal Aneurysms] by Fenestrated & Branched Stent-grafts) trial demonstrated that the number of fenestrations and prior aortic surgery were predictive of perioperative mortality and severe morbidity.¹⁹

Further analysis of the American College of Surgeons National Surgical Quality Improvement Program database, however, suggests that F/B-EVAR for complex aortic aneurysms may provide lower perioperative morbidity rates than those observed with open surgery.¹⁵ Longer procedure times were directly related to the development of adverse events. Procedural duration appeared to be related to physician learning curve, with longer times occurring at the early stages of surgeons' experiences. In addition, the development of these perioperative morbidities likely translated into longer hospital stays (11 days in this series) than would be expected after endovascular intervention, as has previously been observed.^{14,18}

Renal failure and SCI remain the most concerning post-operative morbidities associated with complex F/B-EVAR. Renal failure occurred at a low rate of 2.8% in this series and was greater (5.5%) in those patients undergoing type II repair. This again mirrors other reports for open and endovascular complex aneurysm repair.³¹ A long-term assessment of renal function in this patient population is currently underway.

SCI symptoms developed in 8% of patients overall, but the symptoms resolved in nearly half of the patients before discharge from the hospital. Greenberg et al,¹² in an earlier assessment of our outcomes of F/B-EVAR for TAAA, demonstrated that SCI rates were comparable with those observed with open repair.¹² Reported SCI rates were 19% and 5% for type II and III, respectively, for F/B-EVAR, and 22% and 10%, respectively, for open repairs. Rates in this series appear to be improved over those reported in 2008, but this trend did not reach statistical significance. Improvements

reflect aggressive efforts to minimize the risk of SCI through collateral vessel preservation, and potentially, by the use of staging.^{22,32} Similarly, SCI rates after open repair for these complex aneurysms also decreased during this time.³³

More recent evaluations of staged repair suggest that use of this strategy may reduce the incidence and severity of SCI.^{32,34} In this larger cohort, staging played a relatively small role. Only 11% of the patients had partial aneurysm coverage at a staged procedure, and an additional 10% had an antecedent iliac conduit. Although there was a trend toward lower mortality in those that were staged, this did not reach statistical significance ($P = .076$). O'Callaghan reported a smaller cohort from within this current series and demonstrated that for type II TAAA, staged repair may contribute to lower rates of SCI, less severe clinical presentation, and higher rates of recovery.³² That effect is diluted in this larger cohort by the relatively few patients who had aneurysm repair staging and the larger number of type III TAAs that inherently have lower SCI rates. Others have shown similar benefits from staging procedures both with regards to spinal cord protection and reduction in perioperative morbidities.^{34,35}

Staging, however, is not without risk. Although we made efforts to perform the first-staged procedure before the availability of the custom endograft, other factors contributed to patient delay. Some form of staged procedure was performed in 83 patients, including 48 iliofemoral reconstructions and 41 first-staged TEVAR, with six patients undergoing both concomitantly. Five (6%) of these patients died before F/B-EVAR. The cause of death in two patients was unknown and could potentially have been related to aneurysm rupture. At the time of their deaths, however, the custom aortic graft would not have been available. If this is combined with the patient that ruptured and was treated with F/B-EVAR, the risk of rupture was 3.6% for the staged approach. Staging, however, is associated with reduced SCI and potentially reduced perioperative mortality. This must be balanced with the risk of death or rupture from the staging procedure. Further analysis of this approach is necessary to determine its benefit.

Technical success in this series was excellent and independent of the extent of repair. Technical failures in the CA are related to attempts to incorporate vessels with high-grade stenosis, many of which were ultimately occluded at the time of the index procedure. Exclusion of these vessels from repair in preoperative planning would have increased technical success to 96%. Fifteen of the 21 technical failures were secondary to target vessel disease. These diseased vessels should be approached with caution when these procedures are performed. Long-term branch patency in all vessels was excellent, with few patients requiring a reintervention for target vessel occlusion or stenosis. Patency rates appeared to be independent of branch design, with those

constructed from a reinforced fenestration functioning equally as well as those from a directional, helical branch. These outcomes are similar to those reported by others,³⁶ with perhaps a slight patency advantage for reinforced fenestrations compared with directional branches when targeting the RAs.²⁰

We previously demonstrated low rates of reintervention of visceral and renal branches secondary to the development of endoleaks,²¹ but with more extensive aneurysm treatment, the use of reinforced fenestrations may be associated with a higher rate of branch-related endoleaks. This may be the trade-off with the currently available technologies—endoleak vs patency. Outcomes, including branch patency and branch-related endoleak development, may be improved by better understanding the mechanisms and pathophysiology of their development after F/B-EVAR. It is likely that either morbidity could be overcome with purpose-built bridging stents that allow for improved patency rates or improved seals with the aortic component.

The need for reintervention, however, did not appear to adversely affect longer-term outcomes, because 3-year freedom from aneurysm-related mortality remained high at 91%. Overall survival at 3 years, however, was low at 54%, and adversely affected by the extent of repair, presence of pulmonary disease, and American Society of Anesthesiologists Physical Status Classification. Future analysis will attempt to identify more specific factors that can predict long-term survival in this high-risk patient population with the hope of more clearly selecting patients who will benefit long-term from this type of repair. Given clinical outcomes that appear to provide clinical equipoise with conventional open extensive TAAA repair, further analysis of its utility and cost in the low-risk patient is warranted.

CONCLUSIONS

F/B-EVAR provides a competitive treatment option for patients with TAAA. Perioperative mortality rates are low, but perioperative morbidity is high, reflective of the patient population's underlying comorbidities. Longer-term branch patency is excellent, with few patients developing branch vessel occlusion or stenosis. This may be reflective of branch design, in particular, for the RAs. Branch design based off of reinforced fenestrations, however, comes at the cost of the need for increased reinterventions for branch-related endoleaks. Graft and bridging stent design improvements may help to mitigate this in the future. Despite these shortcomings, this technology offers a less invasive treatment option to those patients with complex aortic aneurysm disease, with outcomes that may challenge those of open repair. Further assessment of these outcomes in patients considered standard risk for open repair is necessary.

This work represents the commitment of the late Dr Roy Greenberg to the development and implementation of F/B-EVAR. It reflects his dedication to educating peers and students with regard to this constantly evolving technology. We owe him a great deal of gratitude for his time, interest, and willingness to share his knowledge.

AUTHOR CONTRIBUTIONS

Conception and design: ME, MF, TM, YK
Analysis and interpretation: ME, MF, KW, TM
Data collection: ME, MF, KW, TM, YK
Writing the article: ME, MF, KW
Critical revision of the article: ME, MF, KW, TM, YK
Final approval of the article: ME, MF, KW, YK
Statistical analysis: ME, MF, KW, YK
Obtained funding: ME, TM
Overall responsibility: ME

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DISCUSSION

Dr Gustavo Oderich (*Rochester, Minn*). Matt, congratulations to you and your group for outstanding results. Thank you for bringing to our attention another important contribution from the Cleveland Clinic. I believe this is by far the largest endovascular experience with type I to III thoracoabdominal aortic aneurysms. And although your numbers are commendable and very large for an endovascular experience, they pale in comparison to the numbers already accumulated in some centers with open thoracoabdominal repair, and yet you already achieved a mortality that is half of what has been reported for the best open aortic centers: 4% vs 8% on average.

So my main question is: Why not extend the indication of endovascular repair to the lowest-risk patients? In addition, I have a second question pertaining to reinterventions. I suspect there are changes in design of these stents that over time you learned and may have affected reinterventions. Finally, how many of the reinterventions are percutaneous procedures done under local anesthesia or as an outpatient?

Dr Matthew J. Eagleton. Gustavo, I absolutely agree. I think we have proven that this can be a durable operation, that it is a safe procedure, and we ought to open up our enrollment criteria to patients that are not high risk.

When our investigational device exemption was originally written by Roy Greenberg, this one was written back in 1998, it was a crazy idea to treat thoracoabdominal aortic aneurysms with fenestrated and branched endografts, and so our enrollment criteria had been excluded to patients that were considered high risk. And nearly every patient in this series was previously evaluated by a surgeon who could perform those procedures in an open fashion and deemed the patients high risk.

There is a high rate of reintervention, particularly for endoleaks, in this series. And if we look at it over time, we have more interventions early in our experience than late in our experience, and we have more interventions based on the type of branches that we used. I think we have lower rates of reintervention for a directional branch than we do for a reinforced fenestration. Our patency rates for reinforced fenestrations though are

extraordinarily high, 98% for most target vessels. So I suspect what we are going to find, in particular for the renal arteries, that there is a trade-off between patency rates and endoleak development rates—at least for these current designs. The majority of our reinterventions were percutaneous.

Dr William Quinones-Baldrich (*Los Angeles, Calif*). I want to congratulate you on the excellent results in a very large experience. My question has to do with the choice between fenestrated and branched endograft. The fenestrated device that you showed looks very short for even a type III thoracoabdominal aneurysm, and yet, 77% of the patients had a fenestrated device. How do you choose between fenestrated and branched endografts? I am concerned in patients that have aneurysmal dilation in the visceral segment that a fenestrated device will allow too much movement of the stent and therefore lead to late complications.

Dr Eagleton. It has changed over time. Initially we used a lot of fenestrations, then went to branches, and now we are more selective. My preference would be to use a true directional branch for the celiac and the superior mesenteric artery, provided I can limit the amount of aorta I cover. Our goal in this, especially given the spinal cord ischemic rates with more extensive coverage, is to provide the least amount of coverage as possible but provide a durable repair. In some instances that requires use of a branch based off of reinforced fenestrations, and sometimes it allows for a directional branch.

Dr Mark Conrad (*Boston, Mass*). I have a question about spinal cord ischemia as well. Last year you presented a cohort of patients where the spinal cord ischemic rate was 40%, and it improved to 11% in patients treated in two stages, now it is 8%. What have you done differently in this group to improve the incidence of spinal cord ischemia?

Dr Eagleton. The ones we presented last year were strictly a cohort of type II thoracoabdominal aneurysm repair. For our type II thoracoabdominal aneurysm repair in this group, the entire series from 2004 was 15%. The 11% rate was in those patients that underwent a staged procedure.

INVITED COMMENTARY

Benjamin W. Starnes, MD, Seattle, Wash

Reported herein is the largest single-center and nearly decade-long experience with fenestrated and branched endografts for the management of extensive thoracoabdominal aortic aneurysms. The great Roy Greenberg began this experience at the Cleveland Clinic in 2004 under the umbrella of an investigational device exemption clinical trial, and it has been masterfully extended under the expert guidance of Matt Eagleton. We can

learn a lot of good lessons from this important report inclusive of 354 patients.

WHAT'S NEW?

We gain more information on the durability of reinforced fenestrations. Those of us who knew Roy knew that he was a big



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