Reoperative surgery on the thoracoabdominal aorta

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ABSTRACT

Objective: Since the advent of endovascular repair for aortic aneurysms, many centers have justified the use of endovascular approaches in patients with previous open distal aortic repair by deeming these patients “high risk” because of their previous operation. We sought to determine whether patients who undergo reoperative repair for thoracoabdominal aortic aneurysm (TAAA) have worse outcomes than patients who undergo non-reoperative repair.

Methods: We reviewed our data on 3379 TAAA repairs from 1986 to 2016. We compared patients’ preoperative characteristics, surgical variables, and outcomes among reoperative (n = 726) and non-reoperative (n = 2653) cases. Furthermore, we examined reoperative indications to identify repairs performed because of repair failure (n = 93) and reoperations performed as an adjacent extension of repair (n = 633). A multivariable analysis was conducted to identify predictors of adverse events by using relevant preoperative and intraoperative factors.

Results: The operative mortality rate did not significantly differ between groups (8.1% for reoperative vs 7.3% for non-reoperative; $P = .5$); in addition, reoperative repair was not associated with an increased risk of adverse event. However, Kaplan-Meier survival analysis showed that over the first 10 years, the reoperative groups fared significantly worse than the non-reoperative group ($P < .001$) (survival estimates at 10 years: $23.9\% \pm 4.9\%$ for patients with repair failure, $28.4\% \pm 2.0\%$ for those with extension of repair, and $40.1\% \pm 1.1\%$ for non-reoperative repairs).

Conclusions: We were unable to detect noteworthy differences in early outcomes between reoperative and non-reoperative TAAA repair. However, mid-term results indicate worse survival for patients who undergo reoperative surgery. (J Thorac Cardiovasc Surg 2018;155:474-85)

Illustrations demonstrate a common form of reoperative thoracoabdominal aortic aneurysm repair.

Central Message

Reoperative TAAA repair yields respectable early outcomes that do not significantly differ from those of non-reoperative TAAA repair.

Perspective

Reoperative thoracoabdominal aortic surgery is typically necessitated by (1) complications of a previous open repair or (2) aortic disease that progresses into an aortic segment adjacent to an intact previous open repair. Although reoperative surgery may necessitate a modified approach, such repair produces respectable early outcomes that do not significantly differ from those of non-reoperative TAAA repair.

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Reoperative surgery on the thoracoabdominal aorta is not uncommon. In our recent report of 3309 thoracoabdominal aortic aneurysm (TAAA) repairs,¹ more than one quarter of

the repairs (n = 858; 25.9%) involved patients who had undergone a previous repair of the distal aorta (descending thoracic, thoracoabdominal, or abdominal aorta). Other aortic centers have reported 13% to 22% rates of previous distal aortic repair in patients undergoing TAAA repair, with the bulk of previous repairs involving the infrarenal abdominal aorta.²⁻⁶

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Read at The American Association for Thoracic Surgery Aortic Symposium, New York, New York, May 12-13, 2016.

Received for publication Aug 30, 2016; revisions received July 18, 2017; accepted for publication Aug 9, 2017; available ahead of print Sept 22, 2017.

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0022-5223/\$36.00

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<http://dx.doi.org/10.1016/j.jtcvs.2017.08.024>

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Abbreviations and Acronyms

AAA	= abdominal aortic aneurysm
CSF	= cerebrospinal fluid
DTA	= descending thoracic aneurysm
TAAA	= thoracoabdominal aortic aneurysm

Since the advent of endovascular repair for aortic aneurysms, many centers have justified the use of endovascular approaches in patients with previous open aortic repair by deeming these patients “high risk” because of their previous operation.⁷ Concerns about enhanced risk are supported by the well-understood risk of bleeding and other complications during re-entry of potentially hostile thoracic and abdominal cavities, as well as by evidence suggesting elevated rates of paraplegia after TAAA repair in patients with previous open abdominal aortic aneurysm (AAA) repair.⁸ Conversely, other reports^{9,10} suggest that the risk of postoperative paraplegia can be reduced by “staging” open distal repairs to permit the development of a collateral network of arteries that can improve spinal perfusion between repairs. Despite considerable interest in these issues, the literature contains few reports that specifically describe TAAA repair after previous open descending thoracic aneurysm (DTA), TAAA, or AAA repair or that describe the risks associated with “redo” TAAA repair^{4,5,11-15}; thus, we sought to better assess the risk of paraplegia and other complications after reoperative TAAA repair, both in comparison with non-reoperative repair and by indication for reoperation.

PATIENTS AND METHODS

Study Enrollment and Patient Characteristics

Baylor College of Medicine’s institutional review board approved our clinical research protocol in 2006. For patients who underwent surgery after protocol approval, data were collected prospectively, and informed consent was obtained whenever possible; waiver of consent was approved for patients who could not provide consent because of illness and whose family members were not available. For patients who underwent surgery before protocol approval, data were collected retrospectively from medical records, and consent was waived.

We examined our experience involving 3379 TAAA repairs to identify factors associated with reoperation and to determine whether risk is greater in reoperative TAAA surgery than in non-reoperative surgery. We theorized that reoperative TAAA surgery is typically necessitated by 1 of 2 factors: (1) complications signaling the failure of a previous open repair (Figure 1) or (2) aortic disease that progresses into an aortic segment adjacent to an intact previous open repair (Figure 2).

Of 3379 TAAA repairs performed between 1986 and 2016, 726 repairs (21.5%) were identified as reoperative and the other 2653 repairs (78.5%) were considered non-reoperative (Figure 3). Indications for reoperation were characterized as TAAA repair failure ($n = 93$; 2.8%) or progression of disease necessitating extension of repair to an adjacent aortic segment ($n = 633$; 18.7%). To better elucidate the characteristics of patients in

FIGURE 1. Drawing illustrates late repair failure of open TAAA surgery. In the residual native aortic tissue of the visceral patch, an aneurysm has formed.

need of reoperative surgery after open repair, we did not include in the reoperative group patients who underwent operations performed after entirely endovascular aortic repairs, second-stage operations for completion of elephant trunk repair (if no other distal aortic repair had been performed previously; $n = 140$), or operations in which the previous repair involved a noncontiguous portion of the distal aorta (such as an extent I TAAA repair after a previous infrarenal AAA repair; $n = 110$) (Table 1). These repairs remained in the overall series but were categorized as non-reoperative TAAA repairs.

Of the 93 repair failures, few involved graft infection ($n = 5$), graft-related fistula ($n = 1$), graft rupture ($n = 2$), or expansion after aortoplasty ($n = 1$). Most repair failures manifested as pseudoaneurysm ($n = 44$), patch aneurysm ($n = 30$), or both ($n = 10$). In the extension-of-repair group ($n = 633$), the majority of the previous distal open aortic repairs were infrarenal AAA repairs (436/633; 68.9%) (Table 1), and most of these were performed outside our center (572/633; 90.4%). In addition, 10.1% ($n = 75$) of the 726 reoperative repairs were made in patients with more than 1 previous distal open aortic repair; this proportion was 25.8% in the repair-failure group (ie, 24 of 93 patients had more than 1 previous distal aortic repair).

Study Definitions and Follow-up

As reported in recent publications, all preoperative, operative, and outcomes data were collected with the use of standard definitions.^{1,16} Reoperative surgery was defined as that necessitated by repair failure or that involved extension of a contiguous repair; any procedure that did not meet this criterion was considered non-reoperative. We defined repair failure as any complication of previous open distal aortic repair that necessitated aortic reoperation: pseudoaneurysm, patch aneurysm,

FIGURE 2. Drawings illustrate a common scenario involving reoperative TAAA surgery. A, An extent III TAAA has developed above the intact previous open extent III TAAA repair. B, To treat the progression of aneurysmal disease, open repair was performed. The previous graft is incorporated into the distal anastomosis.

expansion after aortoplasty, graft infection, graft rupture, or graft-related fistula. We defined extension of repair as repair necessitated by the progression of disease into aortic segments that are adjacent to and contiguous with the previously repaired segment; in most cases, the previously placed graft was left in place.

Patients were considered symptomatic if they had symptoms indicative of aortic disease (eg, pain, dysphagia, hoarseness). To estimate the glomerular filtration rate, we used the Chronic Kidney Disease Epidemiology Collaboration equation.¹⁷ Repairs were divided into 3 eras based on the adoption and use of key surgical adjuncts: Era 1 (1986-1998; before the use of key adjuncts became standard practice; n = 1158), Era 2 (1999-2004; early adoption of key adjuncts; n = 1059), and Era 3 (2005-2016; contemporary use of key adjuncts; n = 1162).^{1,18} Operative death was defined as death that occurred within 30 days of surgery or during the index hospitalization (ie, before final discharge from our hospital or from any other hospital or long-term acute care facility to which the patient was transferred postoperatively). Adverse event, a composite endpoint, was defined as operative death or discharge with any of the following complications: renal failure necessitating dialysis, paraplegia, paraparesis, or stroke.¹⁹ Complications were considered persistent if they were present at the time of early death or hospital

discharge. Early survivors were those patients who survived to hospital discharge. Clinical follow-up information was obtained by clinic visit, telephone interview, or written correspondence. The Social Security Death Index was used to identify deaths among patients who were lost to follow-up.

Surgical Techniques

Our technique for open TAAA repair has been refined over time, follows a multimodal strategy that is largely based on the Crawford classification scheme for defining the extent of TAAA repair, and generally reserves the use of surgical adjuncts for extensive aortic repair (extents I and II). Our 30-year clinical practice is described in greater detail elsewhere, and for the last decade, our approach largely has been standardized.^{1,18} In brief, we routinely use moderate systemic heparinization (1.0 mg/kg), permit mild passive hypothermia (32°C-34°C), use cold renal perfusion (4°C) whenever the renal ostia are accessible, and, when appropriate, reattach at least 1 pair of intercostal or lumbar arteries. For extensive aortic repair, we typically use left heart bypass, selective visceral perfusion, and cerebrospinal fluid (CSF) drainage; in less-extensive repairs (extents III and IV), these adjuncts are not used commonly but may be selectively added, such as in cases of reoperation after previous abdominal aortic replacement or endovascular aneurysm repair. As needed, we treat the 4 visceral arteries (the celiac axis, superior mesenteric artery, and both renal arteries) with bypass grafts, endarterectomy, or stents.

Whereas our overall strategy is similar for reoperative and non-reoperative TAAA repair, we modify our approach for reoperations with an awareness that the need to lyse adhesions adds complexity to the procedure and unavoidably lengthens its overall duration. Further modifications for reoperative surgery include efforts to make an offset incision through a different intercostal space and to limit exposure whenever feasible so as to avoid bleeding complications and unintentional injury of nearby structures when adhesions are dissected. Sharp dissection is typically used to free adhesions that hamper exposure, but an electrocautery may be selectively used at low power settings. In addition, we tend to minimize lung retraction during anticoagulation so as to minimize the risk of bleeding. If separating the lung from the chest wall results in parenchymal injury, we often apply a sealant to the involved area of the lung before closing the chest. Because adhesions involving the spleen are common, we carefully inspect the spleen before closure. If a small capsular tear or other damage to the spleen is found, it may be possible to repair it by using cautery and Surgicel (Ethicon US, LLC, Cincinnati, Ohio); however, if the tear cannot be repaired, the spleen generally is removed. In our previous report on reoperative surgery,²⁰ we noted that the aggressive use of blood products appeared to minimize bleeding complications and that fresh-frozen plasma and platelets particularly are helpful in ameliorating perioperative bleeding. In addition, in reoperative surgery, we use CSF drainage more liberally; for example, we often use CSF drainage during extent III and IV repair in patients who have had previous DTA repair.

In the most complex form of reoperation, namely repairing a failure of a previous open repair, a minimalist approach is taken in that only the problematic portion of the existing graft is repaired. For example, in cases of previous extent II TAAA repair in which a visceral patch aneurysm has formed, only the segment containing the visceral arteries typically is repaired. Although surgery for repair failure related to graft infection was uncommon in this series, infection necessitates the use of additional techniques, such as replacing the entire graft, extensive debridement, using pedicled omentum to cover the replacement graft, delivering antibiotics via irrigation catheters, or using a graft soaked in antibiotics (ie, prepared tableside by soaking a polyester graft in rifampin [600 mg/vial]). The use of a homograft, rather than a standard synthetic graft, in the distal aorta is not recommended.

FIGURE 3. Schematic pathway shows the grouping of 3379 TAAA repairs by reoperative status and indication. Repair failure ($n = 93$) was defined as a complication of previous distal OAR; extension of repair ($n = 633$) was defined as a repair necessitated by the progression of disease into aortic segments that are adjacent to and contiguous with the previously repaired segment. The remaining 2653 repairs were defined as non-reoperative and included 110 extent I TAAA repairs that were noncontiguous with previous open infrarenal abdominal aortic repairs. TAAA, Thoracoabdominal aortic aneurysm; OAR, open aortic repair.

Statistical Analysis and Data Presentation

Statistical analyses were performed with SAS, version 9.4 (SAS Institute, Inc, Cary, NC), IBM SPSS Statistics 22 (IBM Corp, Armonk, NY), and R version 3.4.0 from The R Project for Statistical Computing (Vienna, Austria). Analyses were first performed to compare all reoperative patients to the non-reoperative patients. The reoperative patients also were analyzed separately according to their indication for repair (repair failure vs extension of repair). Continuous variables are presented as mean $\pm$ SD or median (interquartile range), as appropriate. Categorical variables are presented as number and percentage. Bivariate comparisons

were conducted with the Pearson χ^2 test, Fisher exact test, or nonparametric Wilcoxon rank sum test, as appropriate.

To identify independent predictors of adverse event in the overall group of 3379 repairs, we built multivariable logistic regression models by entering clinically relevant preoperative and intraoperative factors that showed a univariate association with a P value of $\leq .1$ into the model and then applied a backward selection method with a removal P value of .05; reoperative status did not meet criteria for model entry and was forced into the model (these variables are listed in Table E1). We also performed an adjusted analysis of operative death and persistent stroke, renal failure

TABLE 1. Details regarding previous aortic repairs for patients stratified by reoperative status and indication

Variable	All ($n = 3379$)	Reoperative status		P value	Reoperative indication		P value
		Reoperative ($n = 726$)	Non-reoperative ($n = 2653$)*		Repair failure ($n = 93$)	Extension of repair ($n = 633$)	
Previous aortic repair†	1475 (43.7)	726 (100)	749 (28.2)	<.001	93 (100)	633 (100)	–
Proximal OAR	772 (22.8)	146 (20.1)	626 (23.6)	.05	34 (36.6)	112 (17.7)	<.001
Distal OAR	836 (24.7)	726 (100)	110 (4.1)*	<.001	93 (100)	633 (100)	–
DTA	176 (5.2)	176 (24.2)	0	–	19 (20.4)	157 (24.8)	.4
Extent I TAAA	65 (1.9)	65 (9.0)	0	–	16 (17.2)	49 (7.7)	.006
Extent II TAAA	35 (1.1)	35 (4.8)	0	–	35 (37.6)	0	<.001
Extent III TAAA	16 (0.5)	16 (2.2)	0	–	14 (15.1)	2 (0.3)	<.001
Extent IV TAAA	33 (1.0)	33 (4.5)	0	–	18 (19.4)	15 (2.4)	<.001
Direct repair‡	6 (0.2)	6 (0.8)	0	–	0	6 (0.9)	1
AAA	579 (17.1)	469 (64.6)	110 (4.1)*	–	20 (21.5)	449 (70.9)	<.001
Infrarenal AAA	563 (16.7)	453 (62.4)	110 (4.1)*	–	17 (18.3)	436 (68.9)	<.001
≥ 2 distal OAR	75 (2.2)	75 (10.3)	0	<.001	24 (25.8)	51 (8.1)	<.001
Distal EAR	75 (2.2)	13 (1.8)	62 (2.3)	.4	3 (3.2)	10 (1.6)	.3

Data are presented as no. (%). OAR, Open aortic repair; DTA, descending thoracic aorta; TAAA, thoracoabdominal aortic aneurysm; AAA, abdominal aortic aneurysm; EAR, endovascular aortic repair. *This group included 110 patients who underwent extent I TAAA repair after noncontiguous previous open infrarenal abdominal aortic repair and who did not meet criteria for reoperative repair. †Previous open or endovascular aortic repair of any section of the aorta. ‡Direct repair involves no graft replacement, is a patch repair, or is a TAAA repair of unknown extent.

TABLE 2. Preoperative characteristics of patients stratified by reoperative status and indication

Variable	All (n = 3379)	Reoperative status		P value	Reoperative indication		P value
		Reoperative (n = 726)	Non-reoperative (n = 2653)*		Repair failure (n = 93)	Extension of repair (n = 633)	
Age, y	64 [54-71]	69 [61-74]	67 [59-73]	.002	65 [53-73]	69 [62-74]	.8
Age ≤ 50 y	451 (13.3)	88 (12.1)	363 (13.7)	.3	19 (20.4)	69 (10.9)	.009
Male sex	2092 (61.9)	538 (74.1)	1554 (58.6)	<.001	57 (61.3)	481 (76.0)	.003
Connective tissue disorder	326 (9.6)	86 (11.8)	240 (9.0)	.02	24 (25.8)	62 (9.8)	<.001
Marfan syndrome	296 (8.8)	81 (11.2)	215 (8.1)	.01	21 (22.6)	60 (9.5)	<.001
Aortic aneurysm without dissection	2157 (63.8)	496 (68.3)	1661 (62.6)	.005	51 (54.8)	445 (70.3)	.003
Aortic dissection							
Acute or subacute aortic dissection	170 (5.0)	20 (2.8)	150 (5.7)	.002	4 (4.3)	16 (2.5)	.3
Chronic aortic dissection	1052 (31.1)	210 (28.9)	842 (31.7)	.1	38 (40.9)	172 (27.2)	.007
DeBakey type I aortic dissection	443 (13.1)	69 (9.5)	374 (14.1)	.001	15 (16.1)	54 (8.5)	.02
DeBakey type III aortic dissection	743 (22.0)	154 (21.2)	589 (22.2)	.6	25 (26.9)	129 (20.4)	.2
Maximum distal aortic diameter, cm	6.1 [5.5-7.0] (n = 2990)	6.3 [5.7-7.2] (n = 665)	6.2 [5.5-7.2] (n = 2325)	.1	7.0 [6.0-8.0] (n = 84)	6.3 [5.6-7.2] (n = 581)	.95
Diameter >7.5 cm	578 (17.1)	130 (17.9)	448 (16.9)	.5	28 (30.1)	102 (16.1)	.001
Hypertension	2874 (85.1)	635 (87.5)	2239 (84.4)	.04	81 (87.1)	554 (87.5)	.9
Hyperlipidemia	1009 (29.9)	239 (32.9)	770 (29.0)	.04	30 (32.3)	209 (33.0)	.9
Diabetes	276 (8.2)	57 (7.9)	219 (8.3)	.7	7 (7.5)	50 (7.9)	.9
Coronary artery disease	1134 (33.6)	311 (42.8)	823 (31.0)	<.001	37 (39.8)	274 (43.3)	.5
Previous CABG	508 (15.0)	153 (21.1)	355 (13.4)	<.001	11 (11.8)	142 (22.4)	.02
Previous MI	662 (19.6)	185 (25.5)	477 (18.0)	<.001	25 (26.9)	160 (25.3)	.7
Cerebrovascular disease	589 (17.4)	176 (24.2)	413 (15.6)	<.001	15 (16.1)	161 (25.4)	.05
History of stroke	346 (10.2)	105 (14.5)	241 (9.1)	<.001	8 (8.6)	97 (15.3)	.09
Peripheral vascular disease	862 (25.5)	200 (27.5)	662 (25.0)	.2	19 (20.4)	181 (28.6)	.1
Chronic renal insufficiency†	102 (3.0)	25 (3.4)	77 (2.9)	.5	4 (4.3)	21 (3.3)	.6
History of renal failure or dialysis	65 (1.9)	19 (2.6)	46 (1.7)	.1	3 (3.2)	16 (2.5)	.7
eGFR	68 [52-87] (n = 3082)	61 [45-79] (n = 671)	65 [49-83] (n = 2411)	.008	67 [47-89] (n = 86)	60 [44-78] (n = 585)	.3
eGFR quartiles				<.001			.01
eGFR Q1 [4-40]	506 (15.0)	143 (19.7)	363 (13.7)	—	16 (17.2)	127 (20.1)	—
eGFR Q2 [41-76]	1606 (47.5)	342 (47.1)	1264 (47.6)	—	39 (41.9)	303 (47.9)	—
eGFR Q3 [77-112]	879 (26.0)	167 (23.0)	712 (26.8)	—	24 (25.8)	143 (22.6)	—
eGFR Q4 [113-148]	91 (2.7)	19 (2.6)	72 (2.7)	—	7 (7.5)	12 (1.9)	—
History of COPD, emphysema, or bronchitis	1229 (36.4)	310 (42.7)	919 (34.6)	<.001	31 (33.3)	279 (44.1)	.05
Past or current tobacco use	2667 (78.9)	611 (84.2)	2056 (77.5)	<.001	70 (75.3)	541 (85.5)	.01
Asymptomatic	1210 (35.8)	268 (36.9)	942 (35.5)	.5	30 (32.3)	238 (37.6)	.3
Symptomatic	2169 (64.2)	458 (63.1)	1711 (64.5)	.5	63 (67.7)	395 (62.4)	.3
Acute symptoms	562 (16.6)	113 (15.6)	449 (16.9)	.4	22 (23.7)	91 (14.4)	.02
Chronic symptoms	1705 (50.5)	357 (49.2)	1348 (50.8)	.4	42 (45.2)	315 (49.8)	.4
Rupture	173 (5.1)	33 (4.5)	140 (5.3)	.4	10 (10.8)	23 (3.6)	.002

Data are presented as no. (%) or median [IQR]. CABG, Coronary artery bypass grafting; MI, myocardial infarction; eGFR, estimated glomerular filtration rate; Q, quartile; COPD, chronic obstructive pulmonary disease. *This group included 110 patients who underwent extent I TAAA repair after noncontiguous previous open infrarenal abdominal aortic repair and who did not meet criteria for reoperative repair. †Serum creatinine level ≥3.0 mg/dL or dialysis.

TABLE 3. Operative details of repair stratified by reoperative status and indication

Variable	All (n = 3379)	Reoperative status		P value	Reoperative indication		P value
		Reoperative (n = 726)	Non-reoperative (n = 2653)*		Repair failure (n = 93)	Extension of repair (n = 633)	
Era of repair				.07			.06
Era 1 (1986-1998)	1158 (34.3)	244 (33.6)	914 (34.5)	–	29 (31.2)	215 (34.0)	–
Era 2 (1999-2004)	1059 (31.3)	252 (34.7)	807 (30.4)	–	42 (45.2)	210 (33.2)	–
Era 3 (2005-2016)	1162 (34.4)	230 (31.7)	932 (35.1)	–	22 (23.7)	208 (32.9)	–
Urgency of operation				.2			.005
Elective	2632 (77.9)	579 (79.8)	2053 (77.4)	–	64 (68.8)	515 (81.4)	–
Urgent or emergent	747 (22.1)	147 (20.2)	600 (22.6)	–	29 (31.2)	118 (18.6)	–
Extent I	933 (27.6)	27 (3.7)	906 (34.2)	<.001	1 (1.1)	26 (4.1)	.1
Extent II	1089 (32.2)	191 (26.3)	898 (33.8)	<.001	10 (10.8)	181 (28.6)	<.001
Extent III	678 (20.1)	277 (38.2)	401 (15.1)	<.001	40 (43.0)	237 (37.4)	.3
Extent IV	679 (20.1)	231 (31.8)	448 (16.9)	<.001	42 (45.2)	189 (29.9)	.003
Reverse elephant trunk	83 (2.5)	5 (0.7)	78 (2.9)	.001	0 (0)	5 (0.8)	.4
Elephant trunk completion repair	143 (4.2)	3 (0.4)	140 (5.3)	<.001	0 (0)	3 (0.5)	.5
Extraction of endograft	57 (1.7)	8 (1.1)	49 (1.8)	.2	3 (3.2)	5 (0.8)	.03
Clamping proximal to left subclavian artery	536 (15.9)	37 (5.1)	499 (18.8)	<.001	1 (1.1)	36 (5.7)	.06
Intercostal/lumbar artery reattachment	1624 (48.1)	271 (37.3)	1353 (51.0)	<.001	20 (21.5)	251 (39.7)	.001
Splenectomy	405 (12.0)	151 (20.8)	254 (9.6)	<.001	47 (50.5)	104 (16.4)	<.001
Aortic clamp time, min	50 [38-66]	50 [39-67]	50 [37-66]	.6	50 [39-70]	50 [39-66]	.8
Management of visceral/renal arteries							
Bypass graft	859 (25.4)	308 (42.4)	551 (20.8)	<.001	48 (51.6)	260 (41.1)	.06
Endarterectomy	877 (26.0)	247 (34.0)	630 (23.7)	<.001	21 (22.6)	226 (35.7)	.01
Stenting	272 (8.0)	84 (11.6)	188 (7.1)	<.001	9 (9.7)	75 (11.8)	.5
Endarterectomy, stenting, or bypass	1400 (41.4)	429 (59.1)	971 (36.6)	<.001	56 (60.2)	373 (58.9)	.8
Adjuncts							
Cerebrospinal fluid drainage	1574 (46.6)	314 (43.3)	1260 (47.5)	.04	40 (43.0)	274 (43.3)	.96
Left heart bypass	1521 (45.0)	163 (22.5)	1358 (51.2)	<.001	2 (2.2)	161 (25.4)	<.001
Left heart bypass time, min	24 [19-29] (n = 1503)	21 [17-27] (n = 159)	24 [19-30] (n = 1344)	<.001	20 [16-24] (n = 2)	21 [17-27] (n = 157)	.5
Hypothermic circulatory arrest	52 (1.5)	5 (0.7)	47 (1.8)	.03	3 (3.2)	2 (0.3)	.002
Cold renal perfusion	1988 (58.8)	540 (74.3)	1448 (54.6)	<.001	67 (72.0)	473 (74.7)	.6
Selective perfusion of visceral arteries	802 (23.7)	108 (14.9)	694 (26.2)	<.001	1 (1.1)	107 (16.9)	<.001

Data are presented as n (%) or median [IQR]. *This group included 110 patients who underwent extent I TAAA repair after non-contiguous previous open infrarenal abdominal aortic repair and who did not meet criteria for reoperative repair.

necessitating dialysis, and paraparesis or paraplegia among the extent II repairs to compare relative risk by reoperative status. Postoperative survival was first estimated by the Kaplan-Meier method; the log-rank test was used to differentiate between groups. We then built a Cox proportional hazards model to adjust survival for clinically relevant covariates: patient age at repair, coronary artery disease, cerebrovascular disease, and past or current tobacco use. For this model, we used a backward selection method and a removal *P* value of .05. The assumption of proportional hazards was examined based on weighted Schoenfeld residuals.

RESULTS

Preoperative Characteristics

Preoperative characteristics are presented in Table 2. There were several notable differences between reoperative

and non-reoperative repair patients. There were also substantial differences between patients who underwent surgery for repair failure versus progressively worsening aortic disease. The extension-of-repair cohort was older than both the repair-failure group and the non-reoperative group. This cohort also had greater preoperative prevalence of several comorbidities, including a history of coronary artery disease, cerebrovascular disease, peripheral vascular disease, and chronic obstructive pulmonary disease or bronchitis; moreover, these patients were more likely to be male, to have an aneurysm unrelated to dissection, and to be current or former smokers.

The repair-failure group was younger than both the extension-of-repair and the non-reoperative groups, and it

TABLE 4. Early outcomes for patients stratified by reoperative status and indication

Variable	All (n = 3379)	Reoperative status		P value	Reoperative indication		P value
		Reoperative (n = 726)	Non-reoperative (n = 2653)*		Repair failure (n = 93)	Extension of repair (n = 633)	
Adverse event†	487 (14.4)	96 (13.2)	391 (14.7)	.3	17 (18.3)	79 (12.5)	.1
Operative death	253 (7.5)	59 (8.1)	194 (7.3)	.5	10 (10.8)	49 (7.7)	.3
In-hospital	240 (7.1)	56 (7.7)	184 (6.9)	.5	9 (9.7)	47 (7.4)	.4
30-d	163 (4.8)	34 (4.7)	129 (4.9)	.8	6 (6.5)	28 (4.4)	.4
Stroke	106 (3.1)	21 (2.9)	85 (3.2)	.7	4 (4.3)	17 (2.7)	.4
Persistent stroke‡	79 (2.3)	15 (2.1)	64 (2.4)	.6	3 (3.2)	12 (1.9)	.4
Spinal cord deficit	325 (9.6)	58 (8.0)	267 (10.1)	.09	5 (5.4)	53 (8.4)	.3
Persistent paraplegia‡	101 (3.0)	23 (3.2)	78 (2.9)	.7	2 (2.2)	21 (3.3)	.5
Persistent paraparesis‡	82 (2.4)	12 (1.7)	70 (2.6)	.1	1 (1.1)	11 (1.7)	.6
Acute renal dysfunction	412 (12.2)	87 (12.0)	325 (12.3)	.8	17 (18.3)	70 (11.1)	.045
Renal failure necessitating dialysis	254 (7.5)	53 (7.3)	201 (7.6)	.8	12 (12.9)	41 (6.5)	.03
Persistent renal failure	192 (5.7)	41 (5.6)	151 (5.7)	.96	9 (9.7)	32 (5.1)	.07
Cardiac complication	879 (26.0)	178 (24.5)	701 (26.4)	.3	21 (22.6)	157 (24.8)	.6
Atrial arrhythmia	553 (16.4)	105 (14.5)	448 (16.9)	.1	8 (8.6)	97 (15.3)	.09
Pulmonary complication	1208 (35.8)	248 (34.2)	960 (36.2)	.3	27 (29.0)	221 (34.9)	.3
Respiratory failure	840 (24.9)	180 (24.8)	660 (24.9)	.98	23 (24.7)	158 (25.0)	.96
Tracheostomy	288 (8.5)	58 (8.0)	230 (8.7)	.6	9 (9.7)	49 (7.7)	.5
Left vocal cord paralysis	520 (15.4)	56 (7.7)	464 (17.5)	<.001	4 (4.3)	52 (8.2)	.2
Bleeding requiring reoperation	112 (3.3)	25 (3.4)	87 (3.3)	.8	1 (1.1)	24 (3.8)	.2
Early survivors	(n = 3126)	(n = 667)	(n = 2459)		(n = 83)	(n = 584)	
Discharge with life-altering complication§	234 (7.5)	37 (5.5)	197 (8.0)	.03	7 (8.4)	30 (5.1)	.2
Length of ICU stay, d	4 [3-8] (n = 3029)	4 [2.25-6]	4 [3-7] (n = 2381)	.07	4 [3-6] (n = 82)	4 [2-6] (n = 566)	.3
Length of hospital stay, d	12 [9-17] (n = 3118)	11 [9-15]	12 [9-17] (n = 2453)	.001	10 [9-14] (n = 82)	11 [9-15] (n = 583)	.8

Data are presented as n (%) or median [IQR]. ICU, Intensive care unit. *This group included 110 patients who underwent extent I TAAA repair after noncontiguous previous open infrarenal abdominal aortic repair and who did not meet criteria for reoperative repair. †Adverse event is defined as operative death or discharge with renal failure necessitating dialysis, paraplegia, paraparesis, or stroke. ‡Persistent is defined as present at the time of operative death or discharge. §Discharged with renal failure necessitating dialysis, paraplegia, paraparesis, or stroke.

had the greatest rates of connective tissue disorders and chronic aortic dissection; additionally, more patients in this group had large aneurysms. Although there was little difference among the groups regarding the presence of overall aortic symptoms, patients with repair failure had a high rate of aortic rupture—nearly 3 times greater than in the extension-of-repair group and more than twice as great as in the non-reoperative group.

In our overall group of 3379 repairs, previous repair of any section of the aorta was fairly common (43.6%). Regarding previous distal open aortic repair, procedures in the repair-failure group were fairly well distributed across the distal aorta, extent II repair being the most common previous procedure. In contrast, in the extension-of-repair group, 68.9% of the previous repairs were for infrarenal AAA; as expected, there were no previous extent II repairs.

Operative Details

Operative details are presented in Table 3. Unsurprisingly, reoperative patients with repair failure more frequently underwent urgent or emergent repair than the extension-of-repair and non-reoperative patients. In addition, those with repair failure typically underwent less extensive (ie, extent III or IV) TAAA repair. Of note, reoperative patients infrequently underwent extent I repair. The most common reoperative procedure was extent III repair (277/726; 38.1%), which was less than one half as frequent in the non-reoperative group (401/2653; 15.1%). Regarding the management of visceral and renal arteries, more than one half of reoperative repairs involved endarterectomy, stenting, or bypass procedures, as compared with roughly one third of non-reoperative repairs. The rate of visceral or renal artery endarterectomy was greatest in the extension-of-repair cohort. CSF drainage

TABLE 5. Multivariable analysis of predictors of adverse events after 3379 TAAA repairs*

Variables	Odds ratio (CI)	P value
Reoperation		.03†
Extension of repair vs non-reoperative repair	0.70 (0.53-0.92)	–
Repair failure vs non-reoperative repair	1.14 (0.64-2.04)	–
Extension of repair vs repair failure	0.62 (0.33-1.14)	–
Rupture	2.16 (1.46-3.20)	<.001
Extent II TAAA repair	2.01 (1.63-2.48)	<.001
Chronic renal insufficiency	2.00 (1.25-3.22)	.004
Splenectomy	1.52 (1.14-2.02)	.004
Endarterectomy, stenting, or bypass	1.47 (1.20-1.81)	<.001
Urgent or emergent repair	1.34 (1.03-1.73)	.03
Previous myocardial infarction	1.29 (1.01-1.63)	.04
Symptomatic	1.27 (1.01-1.60)	.04
Increasing patient age at repair, per year	1.04 (1.03-1.05)	<.001

CI, Confidence interval; TAAA, thoracoabdominal aortic aneurysm. *Adverse event is defined as operative death or discharge with renal failure necessitating dialysis, paraplegia, paraparesis, or stroke. †Reflects a 3-way comparison of extension of repair, repair failure, and non-reoperative repair.

and cold renal perfusion were used liberally in reoperative repairs, but left heart bypass was used infrequently in the reoperative patients—probably because they had less extensive repairs. Notably, in the repair-failure cohort, more than half of the reoperations necessitated a splenectomy; this was probably related to dense adhesions contributing to a splenic tear that was too large to be repaired.

Early Outcomes

Interestingly, compared with the non-reoperative group, neither the reoperative group as a whole nor the repair-failure or extension-of-repair subgroup had a significantly higher rate of operative death. In addition, there were no substantial intergroup differences in the rates of persistent (ie, present at discharge) renal failure, paraplegia, paraparesis, or stroke, or the composite endpoint—adverse event (Table 4). Of the postoperative outcome variables examined, there were few substantial differences among the groups. In addition, multivariable analysis of adverse event rates (Table 5) for the full cohort did not associate reoperative repair with greater risk than primary repair; in fact, patients in the extension-of-repair group were less likely to have an adverse event than patients in the non-reoperative group. The strongest predictors (odds ratio ≥ 2) of adverse event were rupture, extent II TAAA repair, and chronic renal insufficiency.

Regarding extent II repair, we compared 181 such procedures in the extension-of-repair group to 898 in the non-reoperative group (Table 6). Although there were differences in several preoperative and operative variables, there were no important differences between rates of key outcomes. Likewise, multivariable analysis of 1089 extent II repairs did not identify reoperation as predictive of key postoperative complications (Table 7).

Midterm Outcomes

For the 3126 patients who survived to hospital discharge, the median clinical follow-up time was 5.6 years [interquartile range: 2.5-9.7 years]. Although we identified few differences between the reoperative and non-reoperative groups regarding early outcomes, Kaplan-Meier survival analysis showed that, over time, the reoperative groups fared significantly worse than the non-reoperative group ($P < .001$, log-rank test; Figure 4, A); 10-year survival was estimated to be $23.9\% \pm 4.9\%$ for patients with repair failure and $28.4\% \pm 2.0\%$ for those with extension of repair, whereas it was $40.1\% \pm 1.1\%$ for those with non-reoperative repair. In addition, after adjustment were made for age, coronary artery disease, cerebrovascular disease, and past or current tobacco use in a Cox regression survival analysis, the patients with non-reoperative repairs continued to fare better over time ($P < .001$; Figure 4, B; Table 8).

DISCUSSION

Despite the added complexity of reoperative TAAA surgery, our experience suggests that it is not associated with a greater risk of paraplegia, operative death, or other complications (Table 5); this apparently holds true even for the most extensive reoperative TAAA repairs, extent II repairs (Table 6). The reason could be related to the use of a more cautious surgical approach during reoperations so as to minimize further operative trauma. Like us, Kawaharada and colleagues¹³ reported no differences in the rates of early mortality, paraplegia, paraparesis, or renal failure in patients with TAAA who had undergone previous DTA repair and those who had not undergone previous repair. Our earlier report²⁰ concerning TAAA repair after a broader category of reoperations generally supports our current results; there, we speculated that the factors that enabled patients to withstand the stress of the initial aortic repair also conferred a late benefit on survivors who needed additional repair.

In contrast, Lombardi and colleagues⁴ found that although previous AAA or TAAA repair was not associated with greater mortality in TAAA repair patients, previous AAA repair was associated with greater morbidity (arrhythmia, renal failure, pulmonary complications, and paraplegia). Likewise, Schlosser and colleagues⁵ found

TABLE 6. Key preoperative characteristics, operative details, and outcomes after 1089 extent II TAAA repairs

Variables	All (n = 1089)	Non-reoperative (n = 898)*	Extension of repair (n = 181)	P value†	Repair failure (n = 10)
Preoperative characteristics					
Age, y	66 [56-72]	65 [54-71]	68 [61-74]	.009	70 [66-73]
Connective tissue disorder	176 (16.2)	153 (17.0)	22 (12.2)	.1	1 (10)
Acute or subacute dissection	60 (5.5)	51 (5.7)	8 (4.4)	.6	1 (10)
Chronic dissection	484 (44.4)	421 (46.9)	60 (33.1)	.001	3 (30)
DeBakey type I aortic dissection	223 (20.5)	205 (22.8)	17 (9.4)	<.001	1 (10)
DeBakey type III aortic dissection	311 (28.6)	259 (28.8)	49 (27.1)	.7	3 (30)
Chronic renal insufficiency	28 (2.6)	23 (2.6)	5 (2.8)	.8	0
Previous myocardial infarction	183 (16.8)	134 (14.9)	45 (24.9)	.002	4 (40)
Symptomatic	681 (62.5)	562 (62.6)	110 (60.8)	.7	9 (90)
Diameter > 7.5 cm	173 (15.9)	134 (18.3)	34 (18.8)	.9	5 (50)
Rupture	32 (2.9)	30 (3.3)	2 (1.1)	.1	0
Operative details					
Urgent or emergent repair	203 (18.6)	167 (18.6)	30 (16.6)	.6	6 (60)
Intercostal/lumbar artery reattachment	940 (86.3)	777 (86.5)	156 (86.2)	.9	7 (70)
Splenectomy	115 (10.6)	84 (9.4)	28 (15.5)	.02	3 (30)
Management of visceral/renal arteries					
Bypass graft	348 (32.0)	269 (30.0)	75 (41.4)	.003	4 (40)
Endarterectomy	280 (25.7)	219 (24.4)	60 (33.1)	.02	1 (10)
Stenting	90 (8.3)	67 (7.5)	23 (12.7)	.03	0
Endarterectomy, stenting, or bypass	524 (48.1)	410 (45.7)	110 (60.8)	<.001	4 (40)
Adjuncts					
Cerebrospinal fluid drainage	678 (62.3)	568 (63.3)	107 (59.1)	.3	3 (30)
Left heart bypass	898 (82.5)	761 (84.7)	135 (74.6)	.002	2 (20)
Hypothermic circulatory arrest	18 (1.7)	15 (1.7)	2 (1.1)	.8	1 (10)
Cold renal perfusion	723 (66.4)	597 (66.5)	118 (65.2)	.7	8 (80)
Selective perfusion of visceral arteries	695 (63.8)	598 (66.6)	96 (53.0)	.001	1 (10)
Outcomes					
Adverse event‡	204 (18.7)	164 (18.3)	35 (19.3)	.8	5 (50)
Operative death	101 (9.3)	78 (8.7)	22 (12.2)	.2	1 (10)
Persistent stroke§	37 (3.4)	28 (3.1)	8 (4.4)	.4	1 (10)
Persistent paraplegia§	51 (4.7)	38 (4.2)	11 (6.1)	.3	2 (20)
Persistent paraparesis§	35 (3.2)	30 (3.3)	5 (2.8)	.8	0
Persistent renal failure§	79 (7.3)	66 (7.3)	12 (6.6)	.9	1 (10)
Bleeding necessitating return to OR	40 (3.7)	31 (3.5)	9 (5.0)	.4	0

Data are presented as no. (%) or median [IQR]. OR, Operating room. *This group included 110 patients who underwent extent I TAAA repair after noncontiguous previous open infrarenal abdominal aortic repair and who did not meet criteria for reoperative repair. †P value for the comparative analysis between the non-reoperative and extension-of-repair groups; the repair-failure group was not included in this analysis because of the small size (n = 10) of this group. ‡Adverse event is defined as operative death or discharge with renal failure necessitating dialysis, paraplegia, paraparesis, or stroke. §Persistent is defined as present at the time of operative death or discharge.

that the risk of postoperative renal failure after TAAA repair was greater in patients with previous AAA repair than in patients without AAA repair, but there was no difference in early mortality.

Our mid-term survival results revealed definitively that all patients tend to fare poorly by the 10-year time point; this could be related to their relatively advanced age at repair, which is often in the mid-to-late 60s. Notably, the reoperative groups had significantly worse mid-term survival than the non-reoperative group. Thus, although we detected few significant differences in early outcomes

between groups, the long-term implications of reoperative repair warrant further study. Regarding long-term survival, a Dutch series of 500 TAAA repairs revealed a 10-year survival rate of 34%, which is comparable with our findings in the non-reoperative group; of interest, this survival rate was substantially worse than that of a Dutch population matched by calendar year, age, and sex (85%).²¹

Notably, when we categorized reoperative repairs according to whether they were performed to treat failure of a previous open repair or progression of aortic disease into a contiguous section of the aorta, we found several

TABLE 7. Multivariable analysis of early outcomes after extent II thoracoabdominal aortic aneurysm repair (n = 1089)

Variables	Odds ratio (CI)	P value*
Operative death		.7
Extension of repair vs non-reoperative	1.22 (0.72-2.08)	–
Repair failure vs non-reoperative	0.75 (0.09-6.13)	–
Extension of repair vs repair failure	1.63 (0.18-13.99)	–
Persistent stroke†		.6
Extension of repair vs non-reoperative	1.28 (0.57-2.88)	–
Repair failure vs non-reoperative	2.84 (0.34-24.02)	–
Extension of repair vs repair failure	0.45 (0.05-4.13)	–
Persistent renal failure requiring dialysis†		.6
Extension of repair vs non-reoperative	0.72 (0.37-1.39)	–
Repair failure vs non-reoperative	1.18 (0.14-9.77)	–
Extension of repair vs repair failure	0.61 (0.07-5.34)	–
Persistent paraparesis or paraplegia†		.7
Extension of repair vs non-reoperative	1.07 (0.60-1.94)	–
Repair failure vs non-reoperative	2.01 (0.39-10.45)	–
Extension of repair vs repair failure	0.54 (0.10-2.95)	–

CI, Confidence interval. *Reflects a 3-way comparison of extension of repair, repair failure, and non-reoperative repair. †Persistent is defined as present at the time of operative death or hospital discharge.

differences in preoperative characteristics. For example, in the repair-failure reoperative group (in which the failures comprised mostly pseudoaneurysms and patch aneurysms), we found younger patients, with greater rates of aortic dissection, connective tissue disorders, and rupture. In addition, nearly one third of patients with repair failure presented for reoperation with large aneurysms (>7.5 cm) that were well beyond the guideline-based threshold for repair.²² This suggests that despite the well-established late risk of reoperation in young patients, especially those

with connective tissue disorders such as Marfan syndrome,¹⁴ adherence to postoperative surveillance protocols must be improved.

In contrast, the preoperative characteristics of our extension-of-repair group were different than we originally envisioned. We surmised that this group might have high rates of chronic aortic dissection, but in fact it had a lower rate of such dissection than did either the failure group or the non-reoperative group. In addition, in the extension-of-repair group, we found greater rates of comorbidities commonly associated with the atherosclerotic process: older age, coronary artery disease, cerebrovascular disease, and peripheral vascular disease, as well as a history of smoking. Although in our previously published series²⁰ we found reoperative patients to be older, with elevated rates of coronary artery, cerebrovascular, and other disease, more than 40% of those patients had chronic aortic dissection, whereas only 27% of the patients in our current study had chronic aortic dissection.

In addition, nearly 70% of the current series' extension-of-repair group had previous infrarenal AAA repair. This indicates that some persons treated for infrarenal abdominal aortic disease remain at risk for thoracic and thoracoabdominal aortic disease.¹⁵ Because the majority of these previous abdominal aortic repairs were done outside our facility, it is difficult to determine how many such patients go on to need TAAA repair. Also, we do not know how the shift in practice to endovascular AAA repair will affect the outcomes of reoperative TAAA repair. For extension-of-repair patients, in our experience, it appears that surveillance protocols might be effective: The rate of rupture was low, there were relatively few large aneurysms, and 38% of patients were asymptomatic.

FIGURE 4. A, Kaplan-Meier curves of estimated survival after 3379 thoracoabdominal aortic aneurysm repairs stratified by reoperative status (non-reoperative repair, extension of repair, and repair failure), indicating a survival advantage for those patients who underwent non-reoperative repair ($P < .001$). **B,** When we adjusted for age, coronary artery disease, cerebrovascular disease, and past or current tobacco use by using a Cox regression model to analyze survival after TAAA repair stratified by reoperative status, the non-reoperative repair patients' survival advantage remained ($P < .001$). Survival curves were formulated from all patients' data and include 253 operative deaths. Confidence intervals are shown by the shaded bands. The corresponding hazard ratios, 95% confidence intervals, and P values for panel B are shown in Table 8.

TABLE 8. Cox regression analysis of death after 3379 TAAA repairs, adjusted for age (see Figure 4, B, for graphical interpretation)

Variables	Hazard ratio	95% CI	P value
Reoperation			<.001
Repair failure vs non-reoperative repair	1.75	(1.36-2.25)	–
Extension of repair vs non-reoperative repair	1.23	(1.09-1.37)	–
Increasing patient age at repair, per year	1.05	(1.04-1.05)	<.001
Coronary artery disease	1.18	(1.07-1.30)	<.001
Cerebrovascular disease	1.36	(1.21-1.53)	<.001
Past or current tobacco use	1.13	(0.99-1.28)	.07

CI, Confidence interval.

Our findings represent the experience of a single practice led by the senior author (J.S.C.), which may differ substantially from other surgeons and centers. Limitations of our study include the fact that data were collected both retrospectively and prospectively from patients treated over a nearly 3-decade period, which we have previously speculated limits our ability to examine differences among surgical eras.^{1,18} Moreover, because our cohort was limited to patients who presented to our tertiary care center, the incidence of distal aortic open repair failure could not be determined definitively from our data. In addition, although we believe it is useful to categorize reoperative repair on the basis of repair failure or the type of previous repair, it is possible that the 110 non-reoperative procedures with previous open AAA repair (in which the previous AAA repair was not contiguous with the current TAAA repair and thus was categorized as non-reoperative repair) could in time cross over into the extension-of-repair cohort, which would alter the results presented here. Moreover, the small size of the repair-failure group (n = 93) limited the power of statistical analysis in comparing that group's outcomes to those of the other groups.

In conclusion, although reoperative TAAA surgery is more complicated than primary repair, the early postoperative results do not appear to be substantially different. However, multivariate analysis of early adverse event rates found less risk in the extension-of-repair group than in the non-reoperative repair group. Finally, mid-term results indicate worse survival for patients who undergo reoperative surgery.

Conflicts of Interest Statement

Dr Coselli participates in clinical research trials conducted by Glaxo Smith Kline, Edwards Lifesciences, and Bolton Medical; consults for, receives royalties and a

departmental educational grant from, and participates in clinical trials for Vascutek Terumo; and consults and participates in clinical trials for Medtronic, Inc, and WL Gore & Associates. Dr LeMaire participates in clinical trials for Medtronic, Inc, WL Gore & Associates, and Vascutek Terumo, and consults for Vascutek Terumo. All other authors have nothing to disclose with regard to commercial support.

The authors thank several members of the Surgical Research Core of the Michael E. DeBakey Department of Surgery at Baylor College of Medicine, including Laurie Fondren for managing patient enrollment, and Scott A. Weldon, MA, CMI, for creating illustrations and figures. We also thank the Section of Scientific Publications at the Texas Heart Institute for providing editorial support.

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Key Words: aneurysm (aorta), aortic dissection, aortic operation, reoperation, thoracoabdominal, outcomes

TABLE E1. Variables entered into multivariable logistic regression models (Tables 5 and 7)

Preoperative variables	Intraoperative variables
Reoperative status	Urgent or emergent repair
Male sex	Extraction of endograft
Age, y	Reverse elephant trunk
Symptomatic	Elephant trunk completion repair
Peripheral vascular disease	Clamping proximal to left subclavian artery
Connective tissue disorder	Intercostal/lumbar artery reattachment
Marfan syndrome	Splenectomy
Hypertension	Endarterectomy, stenting, or bypass
Hyperlipidemia	Extent II TAAA repair
Diabetes	
Previous CABG	
Previous MI	
Coronary artery disease	
Cerebrovascular disease	
History of renal failure or dialysis	
Chronic renal insufficiency	
Pulmonary history	
History of stroke	
Current tobacco use	
Rupture	

Body mass index, initial serum creatinine level, estimated glomerular filtration rate, maximum distal diameter, and diameter >7.5 cm had $P < .1$ in the univariate comparisons but were excluded because values were missing for approximately 10% of patients. TAAA, Thoracoabdominal aortic aneurysm; CABG, coronary artery bypass grafting; MI, myocardial infarction.