Outcomes of 3309 thoracoabdominal aortic aneurysm repairs

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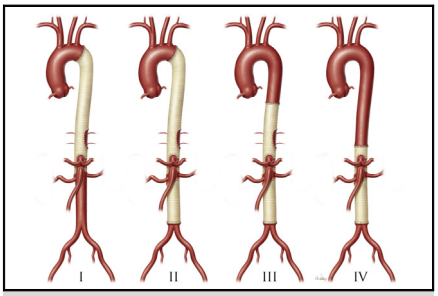
ABSTRACT

Objective: Since the pioneering era of E. Stanley Crawford, our multimodal strategy for thoracoabdominal aortic aneurysm repair has evolved. We describe our approximately 3-decade single-practice experience regarding 3309 thoracoabdominal aortic aneurysm repairs and identify predictors of early death and other adverse postoperative outcomes.

Methods: We analyzed retrospective (1986-2006) and prospective data (2006-2014) obtained from patients (2043 male; median age, 67 [59-73] years) who underwent 914 Crawford extent I, 1066 extent II, 660 extent III, and 669 extent IV thoracoabdominal aortic aneurysm repairs, of which 723 (21.8%) were urgent or emergency. Repairs were performed to treat degenerative aneurysm (64.2%) or aortic dissection (35.8%). The outcomes examined included operative death (ie, 30-day or in-hospital death) and permanent stroke, paraplegia, paraparesis, and renal failure necessitating dialysis, as well as adverse event, a composite of these outcomes.

Results: There were 249 operative deaths (7.5%). Permanent paraplegia and paraparesis occurred after 97 (2.9%) and 81 (2.4%) repairs, respectively. Of 189 patients (5.7%) with permanent renal failure, 107 died in the hospital. Permanent stroke was relatively uncommon (n = 74; 2.2%). The rate of the composite adverse event (n = 478; 14.4%) was highest after extent II repair (n = 203; 19.0%) and lowest after extent IV repair (n = 67; 10.2%; $P < .0001$). Estimated postoperative survival was 83.5% ± 0.7% at 1 year, 63.6% ± 0.9% at 5 years, 36.8% ± 1.0% at 10 years, and 18.3% ± 0.9% at 15 years.

Conclusions: Repairing thoracoabdominal aortic aneurysms poses substantial risks, particularly when the entire thoracoabdominal aorta (extent II) is replaced. Nonetheless, our data suggest that thoracoabdominal aortic aneurysm repair, when performed at an experienced center, can produce respectable outcomes. (J Thorac Cardiovasc Surg 2016;151:1323-38)



Outcomes of TAAA repair differ by Crawford extent.

Central Message

Open TAAA repair produces respectable outcomes, but there is clearly room for improvement. Outcome differs by repair extent.

Perspective

We present the results of 3309 open TAAA repairs to elucidate operative risk. These repairs require interrupting blood flow to vital organs, which incurs the risk of postoperative paraplegia, renal failure, and other complications. Our data suggest that open TAAA repair performed at an experienced center can produce respectable outcomes, but further improvement is needed.

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Thoracoabdominal aortic aneurysm (TAAA) repair was first performed in the 1950s[1-3] and soon became a signature repair under Michael E. DeBakey's tutelage at Baylor College of Medicine[4]—the premier aortic center of this pioneering era. Into the 1970s and beyond, E. Stanley Crawford refined TAAA repair by invoking many surgical concepts that remain in use today: endoaortic graft inclusion, expeditious repair, reattachment of intercostal and lumbar arteries, and patch reattachment of visceral vessels, to name a few.[5] Through the efforts of Drs DeBakey, Denton A. Cooley, and Crawford and their

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Abbreviations and Acronyms

eTAAR = endovascular thoracoabdominal aortic repair
HCA = hypothermic circulatory arrest
MOF = multisystem organ failure
RRR = relative risk ratio
TAAA = thoracoabdominal aortic aneurysm
TEVAR = thoracic endovascular aortic repair

colleagues, Baylor College of Medicine developed into a global, tertiary-care aortic center that continues to treat many patients with highly complex TAAA pathology who are in acute need. Even after his untimely death, Crawford continued to guide TAAA repair through the many surgeons he had mentored, the widespread use of his namesake classification system (the Crawford extents of TAAA repair[6]) (Figure 1), and information gleaned from his published experience of 1509 TAAA repairs.[7] As we assess postoperative outcomes during our approximately 3-decade single-practice experience regarding 3309 TAAA repairs, we honor the legendary men that laid the foundation of modern TAAA repair.

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 by dedicated staff, 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.

From October 1986 to December 2014, 3320 consecutive open TAAA repairs were performed on our service. We were unable to obtain consent from 11 patients with repairs performed after our protocol was approved; these patients were excluded from our analyses, and the remaining 3309 repairs form the basis of this report (Figure 2). Repairs included 914 Crawford extent I TAAA repairs, 1066 extent II repairs, 660 extent III repairs, and 669 extent IV repairs (Table 1). Of these, 142 repairs (4.3%) involved patients living outside the United States, in 28 different countries. Patients were characterized as symptomatic (n = 2117; 64.0%) if they had any symptom related to their aortic disease (eg, pain, hoarseness, dysphagia); in 729 (21.8%), repair was nonelective, with patients presenting in acute distress or being transferred to us for urgent or emergency care. Approximately two thirds of repairs were performed to treat degenerative aneurysms without dissection (64.2%); the other one third were performed to treat aortic dissection (35.8%), including the late aortic dilatation that is common in chronic dissection (30.8%). The distributions of several preoperative characteristics differed significantly by Crawford extent of repair (Table 1), including age, aortic dissection,

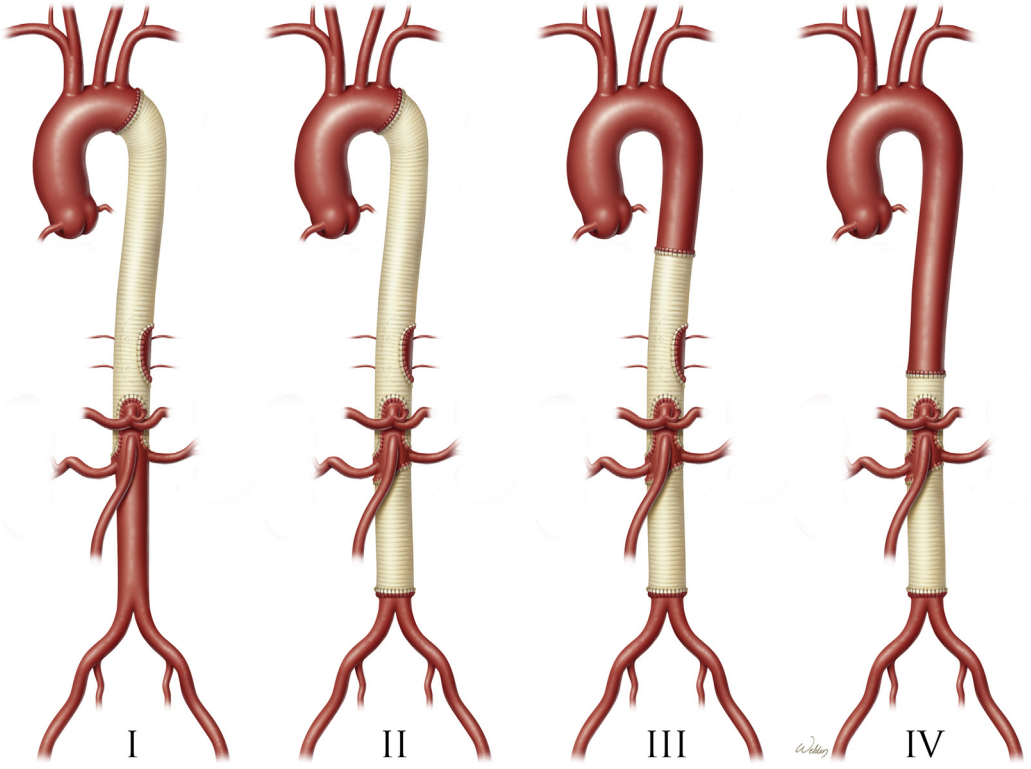

FIGURE 1. Drawing illustrating the Crawford extents of thoracoabdominal aortic aneurysm (TAAA) repair. Extent I repairs involve most or all of the descending thoracic aorta and the upper abdominal aorta. Extent II repairs involve the same segments as extent I repairs but also extend into the infrarenal abdominal aorta. Extent III repairs involve a combination of the distal half, or less, of the descending thoracic aorta (beginning below the sixth rib) and varying portions of the abdominal aorta. Extent IV repairs involve the abdominal aorta below the diaphragm. Used with permission of Baylor College of Medicine.

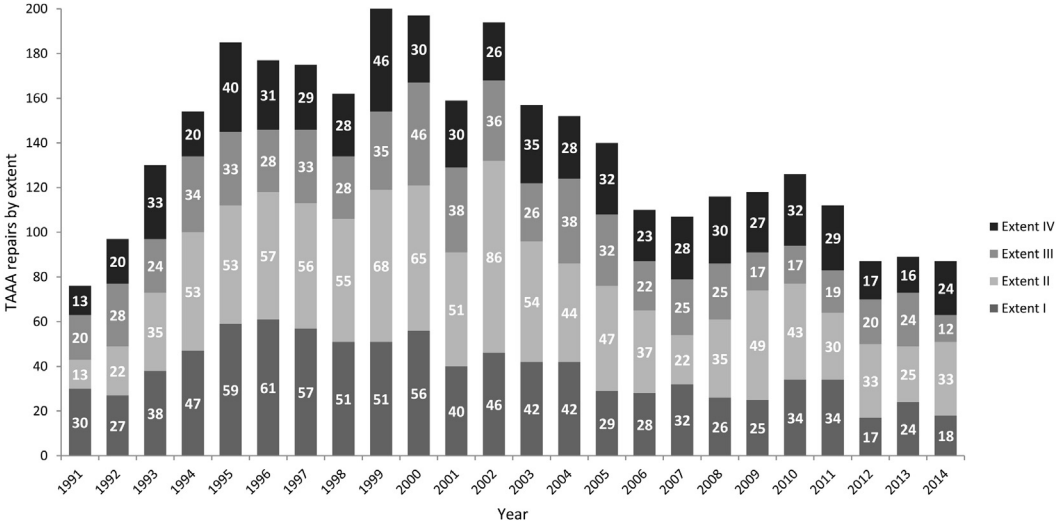

FIGURE 2. Graph depicting the number of TAAA repairs, stratified by Crawford extent, performed between January 1991 and December 2014. Two repairs, 1 performed in October 1986 and the other in April 1990, are not shown. *TAAA*, Thoracoabdominal aortic aneurysm.

coronary artery disease, cerebrovascular disease, serum creatinine, pulmonary disease, and peripheral vascular disease (all with $P < .001$).

Study Definitions and Follow-up

Operative mortality was defined as death within 30 days of surgery or before final discharge from hospitalization (including acute care at other facilities).[8] Adverse event, a composite end point, was defined as operative death or permanent stroke, paraplegia, paraparesis, or renal failure necessitating dialysis.[9] Complications were considered permanent if they were present at the time of hospital discharge or early death. Survivors were defined as having a life-altering complication if they were discharged with stroke, paraplegia, paraparesis, or renal failure necessitating dialysis. Preoperative, operative, and outcomes data were collected according to standardized definitions reported in our recent publications.[10,11] Patients with lower-extremity neurologic deficits not due to stroke were considered to have spinal cord deficits, namely, paraplegia or paraparesis. Visceral artery procedure was defined as endarterectomy, stenting, or bypass of visceral arteries. Postoperative complications were captured regardless of whether they were immediate or delayed, including delayed complications related to multisystem organ failure (MOF). We categorized MOF as primary MOF when several organ systems failed simultaneously because of the physiologic insult of the operation or as secondary MOF when organ systems failed progressively after a specific inciting complication, such as stroke or pneumonia. Postoperative repair failure was defined as failure directly involving the index TAAA graft, namely, pseudoaneurysm, patch aneurysm, fistula, or graft infection, and did not include subsequent repair necessitated by progression of aortic disease adjacent to the repair.[12]

Follow-up data were obtained by clinic visit, telephone, or written correspondence and were available for all but 34 patients (1.0%), who were lost at hospital discharge and remained without 30-day follow-up. Medical records and Social Security Death Index data were reviewed for late events. Recent follow-up data (from September 2013 or later) were available for 1101 (92.1%) of 1196 late survivors, including 1048 (96.3%) of surviving US patients. Fifty-five international patients (1.7%) lacked recent follow-up data (20 were lost at hospital discharge <30 days postoperatively) and were assumed to be alive; 40 US patients (1.2%) lacked recent follow-up data (14 were lost at hospital discharge <30 days postoperatively) and were indicated to be alive by the Social Security Death Index. The median follow-up time was 6.03 [2.9-11.1] years (range, 30 days to 23.1 years) for surviving patients with at least 30-day follow-up after repair (n = 1162) and 6.2 [3.1-11.5] years (range, 33 days to 23.1 years) for US patients with recent follow-up (n = 1048).

Surgical Techniques

Our technique for open repair of TAAA has evolved over the last 3 decades into a multimodal approach that is based on the Crawford extents of repair and other selective factors (eg, prior distal aortic repair, poor cardiac function, other health factors). By 1998, all of the varied techniques (Table 2) that form our current operative approach (described in detail previously[13-15] and illustrated in a companion article[16]) had been introduced to our clinical practice, but their use had not yet been standardized, and several techniques were in their infancy (Era 1, n = 1158). As we shifted into an investigational period (1999-2004; Era 2, n = 1059), we retrospectively reviewed our clinical data regarding the use of left heart bypass,[17] conducted randomized clinical trials on the use of cerebrospinal fluid drainage[18] and cold renal perfusion,[19] and described our experience with using balloon-expandable stents to manage visceral artery lesions.[20] In 2005, we published our multimodal approach to TAAA repair,[21] which largely standardized our approach to repair in the contemporary era (2005-2014; Era 3, n = 1092); however, we continued to evaluate innovative approaches, and in 2009, we published our second randomized clinical trial evaluating different approaches to cold renal perfusion.[22]

In brief, for all extents of repair, we routinely use moderate systemic heparinization (1.0 mg/kg) and mild permissive hypothermia (32°C-34°C, nasopharyngeal); when possible, we reattach 1 or more pairs of intercostal and lumbar arteries (especially between T8 and L1) and intermittently perfuse the renal arteries with a cold (4°C) solution. Operative adjuncts that are generally reserved for more extensive TAAA repairs (ie, Crawford extents I and II) include cerebrospinal fluid drainage, left heart bypass, and selective perfusion of the celiac and superior mesenteric arteries. As needed, visceral arteries are managed with endarterectomy, balloon-expandable stents, or bypass grafts. To treat dissection extending into the visceral arteries, the septum is fenestrated, excised, sutured closed, or obliterated with a small balloon-expandable stent. When the visceral arteries are especially fragile or significantly displaced, 1 or more branch-grafts are used.[15] We rarely use deep hypothermic circulatory arrest (HCA), usually only because safe proximal

TABLE 1. Preoperative characteristics

Variable	All n = 3309	Extent I n = 914	Extent II n = 1066	Extent III n = 660	Extent IV n = 669	*P* value
Age (y)	67 [59-73]	67 [57-73]	66 [56-72]	69 [61-74]	70 [64-75]	<.001
Age ≤50 y	439 (13.3)	131 (14.3)	192 (18.0)	69 (10.5)	47 (7.0)	<.001
Age >79 y	193 (5.8)	38 (4.2)	38 (3.6)	56 (8.5)	61 (9.1)	<.001
Male	2043 (61.7)	513 (56.1)	691 (64.8)	378 (57.3)	461 (68.9)	<.001
Genetically triggered disorder	523 (15.8)	153 (16.7)	227 (21.3)	87 (13.2)	56 (8.4)	<.001
Connective tissue disorder	330 (10.0)	67 (7.3)	177 (16.6)	56 (8.5)	30 (4.5)	<.001
Marfan syndrome	288 (8.7)	53 (5.8)	154 (14.5)	52 (7.9)	29 (4.3)	<.001
Aortic aneurysm without dissection	2124 (64.2)	484 (53.0)	538 (50.5)	519 (78.6)	583 (87.1)	<.001
Aortic dissection	1185 (35.8)	430 (47.1)	528 (49.5)	141 (21.4)	86 (12.9)	<.001
Acute dissection	110 (3.3)	47 (5.1)	41 (3.9)	13 (2.0)	9 (1.4)	<.001
Subacute dissection	55 (1.7)	31 (3.4)	18 (1.7)	4 (0.6)	2 (0.3)	<.001
Acute or subacute dissection	165 (5.0)	78 (8.5)	59 (5.5)	17 (2.6)	11 (1.6)	<.001
Chronic dissection	1020 (30.8)	352 (38.5)	469 (44.0)	124 (18.8)	75 (11.2)	<.001
DeBakey type I	427 (12.9)	150 (16.4)	216 (20.3)	32 (4.9)	29 (4.3)	<.001
DeBakey type IIIa	121 (3.7)	59 (6.5)	33 (3.1)	17 (2.6)	12 (1.8)	<.001
DeBakey type IIIb	607 (18.3)	219 (24.0)	271 (25.4)	78 (11.8)	39 (5.8)	<.001
Localized dissection	33 (1.0)	4 (0.4)	9 (0.8)	14 (2.1)	6 (0.9)	.009
Maximum distal aortic diameter, cm	6.2 [5.5-7.2] (n = 2936)	6.2 [5.5-7.2] (n = 801)	6.3 [5.7-7.2] (n = 958)	6.2 [5.6-7.2] (n = 586)	6.1 [5.4-7.2] (n = 591)	.02
Hypertension	2805 (84.8)	765 (83.7)	917 (86.0)	555 (84.1)	568 (84.9)	.5
Hyperlipidemia	967 (29.2)	229 (25.1)	263 (24.7)	215 (32.6)	260 (38.9)	<.001
Diabetes	261 (7.9)	63 (6.9)	67 (6.3)	56 (8.5)	75 (11.2)	.001
Coronary artery disease	1232 (37.2)	259 (28.3)	351 (32.9)	291 (44.1)	331 (49.5)	<.001
Previous CABG	496 (15.0)	98 (10.7)	136 (12.8)	121 (18.3)	141 (21.1)	<.001
Prior myocardial infarction	652 (19.7)	131 (14.3)	181 (17.0)	162 (24.6)	178 (26.6)	<.001
History of atrial arrhythmia	402 (12.2)	125 (13.7)	119 (11.2)	81 (12.3)	77 (11.5)	.4
History of LV hypertrophy	1236 (37.4)	330 (36.1)	410 (38.5)	258 (39.1)	238 (35.6)	.4
Cerebrovascular disease	575 (17.4)	117 (12.8)	183 (17.2)	144 (21.8)	131 (19.6)	<.001
Prior stroke	334 (10.1)	67 (7.3)	113 (10.6)	82 (12.4)	72 (10.8)	.007
Prior transient ischemic attack	158 (4.8)	40 (4.4)	49 (4.6)	38 (5.8)	31 (4.6)	.6
Chronic renal insufficiency*	99 (3.0)	15 (1.6)	28 (2.6)	30 (4.6)	26 (3.9)	.004
Chronic renal failure (dialysis)	60 (1.8)	11 (1.2)	17 (1.6)	18 (2.7)	14 (2.1)	.1
Preoperative serum creatinine level, mg/dL	1.1 [0.9-1.4] (n = 3037)	1.05 [0.9-1.3] (n = 822)	1.1 [0.9-1.4] (n = 980)	1.2 [0.9-1.5] (n = 614)	1.16 [0.9-1.5] (n = 621)	<.001
Atrophic kidney	325 (9.8)	43 (4.7)	108 (10.1)	82 (12.4)	92 (13.8)	<.001
Polycystic kidney disease	211 (6.4)	52 (5.7)	67 (6.3)	45 (6.8)	47 (7.0)	.7
Previous nephrectomy (whole)	74 (2.2)	15 (1.6)	26 (2.4)	19 (2.9)	14 (2.1)	.4
Pulmonary disease	1298 (39.2)	314 (34.4)	363 (34.1)	322 (48.8)	299 (44.7)	<.001
COPD	1214 (36.7)	288 (31.5)	334 (31.3)	303 (45.9)	289 (43.2)	<.001
Past or current tobacco use	2616 (79.1)	669 (73.2)	835 (78.3)	546 (82.7)	566 (84.6)	<.001
Asymptomatic	1192 (36.0)	311 (34.0)	400 (37.5)	239 (36.2)	242 (36.2)	.5
Symptomatic	2117 (64.0)	603 (66.0)	666 (62.5)	421 (63.8)	427 (63.8)	.5
Acute symptoms	549 (16.6)	169 (18.5)	161 (15.1)	109 (16.5)	110 (16.4)	.3
Chronic symptoms	1664 (50.3)	465 (50.9)	538 (50.5)	328 (49.7)	333 (49.8)	1.0
Peripheral vascular disease	855 (25.8)	148 (16.2)	252 (23.6)	191 (28.9)	264 (39.5)	<.001
Rupture	170 (5.1)	48 (5.3)	32 (3.0)	43 (6.5)	47 (7.0)	.001
Prior distal aortic repair†	858 (25.9)	143 (15.6)	197 (18.5)	275 (41.7)	243 (36.3)	<.001
Failure of prior aortic repair‡	136 (4.1)	9 (1.0)	23 (2.2)	47 (7.1)	57 (8.5)	<.001

Values are n (%) or median [interquartile range]. *CABG*, Coronary artery bypass graft; *LV*, left ventricular; *COPD*, chronic obstructive pulmonary disease. *Serum creatinine level ≥3.0 mg/dL or dialysis. †Prior open or endovascular repair. ‡Failure of prior distal open or endovascular repair necessitating the in-window repair.

aortic clamping is precluded by frank rupture, enormous aneurysm, or other complicating factors. During extent II, III, and IV repairs, the distal anastomosis usually is constructed at the level of the aortic bifurcation (or, occasionally, to each iliac or femoral artery separately).

Data Presentation

Data were analyzed with IBM SPSS Statistics 22 (International Business Machines Corp, Armonk, NY) and Stata IC 13 (StataCorp 2013. Stata Statistical Software: Release 13. StataCorp LP, College Station,

TABLE 2. Operative details

Variable	All n = 3309	Extent I n = 914	Extent II n = 1066	Extent III n = 660	Extent IV n = 669	*P* value
Urgency of operation						
Elective	2586 (78.2)	700 (76.6)	866 (81.2)	504 (76.4)	516 (77.1)	.03
Urgent	428 (12.9)	131 (14.3)	127 (11.9)	86 (13.0)	84 (12.6)	.4
Emergency	295 (8.9)	83 (9.1)	73 (6.8)	70 (10.6)	69 (10.3)	.02
Urgent or emergency	723 (21.8)	214 (23.4)	200 (18.8)	156 (23.6)	153 (22.9)	.03
Aortic repair details						
Redo thoracotomy	563 (17.0)	84 (9.2)	125 (11.7)	192 (29.1)	162 (24.4)	<.001
Extraction of endograft*	48 (1.5)	9 (1.0)	13 (1.2)	9 (1.4)	17 (2.5)	.06
Extraction of TEVAR	29 (0.9)	8 (0.9)	12 (1.1)	6 (0.9)	3 (0.4)	.5
Extraction of EVAR	24 (0.7)	1 (0.1)	3 (0.3)	5 (0.8)	15 (2.2)	<.001
Reverse elephant trunk	80 (2.4)	32 (3.5)	48 (4.5)	0	0	<.001
Elephant trunk completion repair	133 (4.0)	60 (6.6)	73 (6.8)	0	0	<.001
Clamping proximal to LSCA	522 (15.8)	272 (29.8)	245 (23.0)	2 (0.3)	3 (0.4)	<.001
ICA/LA reattachment	1675 (50.6)	498 (54.5)	941 (88.3)	221 (33.5)	15 (2.2)	<.001
Iliac/femoral bypass graft	270 (8.2)	2 (0.2)	95 (8.9)	59 (8.9)	114 (17.0)	<.001
Total estimated blood loss, mL	2500 [1500-5000]	2500 [1500-4500]	3400 [2000-6500]	2000 [1000-4000]	2000 [1000-3900]	<.001
Splenectomy	401 (12.1)	96 (10.5)	115 (10.8)	95 (14.4)	95 (14.2)	.02
Cholecystectomy	72 (2.2)	8 (0.9)	25 (2.3)	22 (3.3)	17 (2.5)	.007
Clamp and ischemic times						
Aortic clamp time, min	48 [36-62]	41 [32-52]	63 [53-75]	43 [34-53]	41 [31-51]	<.001
Intercostal, total, min	40 [30-52]	40 [31-50]	52 [41-64]	35 [27-44]	31 [24-40]	<.001
Intercostal, unprotected, min	31 [24-39]	26 [20-32]	35 [27-42]	34 [26-42]	31 [24-40]	<.001
Left renal, total, min	45 [33-59]	40 [31-50]	60 [50-73]	39 [30-50]	35 [27-47]	<.001
Left renal, unprotected, min	35 [27-46]	27 [20-33]	42 [35-52]	38 [29-48]	35 [26-47]	<.001
Right renal, total, min	42 [32-56]	40 [31-50]	57 [48-69]	37 [29-45]	32 [25-42]	<.001
Right renal, unprotected, min	33 [26-42]	27 [20-33]	40 [33-47]	35 [29-44]	32 [25-42]	<.001
Celiac axis, total, min	42 [22-55]	40 [31-50]	57 [48-69]	37 [29-46]	32 [25-41]	<.001
Celiac axis, unprotected, min	33 [26-42]	27 [20-33]	39 [32-46]	36 [29-45]	32 [25-41]	<.001
SMA, total, min	42 [32-55]	40 [31-50]	57 [48-68]	37 [29-46]	32 [25-41]	<.001
SMA, unprotected, min	33 [26-42]	27 [20-33]	39 [32-47]	36 [29-44]	32 [25-41]	<.001
Left leg, total, min	46 [35-59]	40 [31-50]	62 [52-73]	41 [32-50]	39 [31-48]	<.001
Left leg, unprotected, min	37 [28-47]	27 [20-33]	44 [36-52]	39 [31-49]	39 [31-48]	<.001
Right leg, total, min	46 [35-59]	40 [31-50]	61 [52-72]	41 [32-50]	40 [31-49]	<.001
Right leg, unprotected, min	37 [28-47]	27 [20-33]	44 [36-52]	39 [31-49]	39 [31-48]	<.001
Management of visceral/renal arteries						
Bypass graft	825 (24.9)	38 (4.2)	334 (31.3)	214 (32.4)	239 (35.7)	<.001
To celiac axis	199 (6.0)	20 (2.2)	67 (6.3)	66 (10.0)	46 (6.9)	<.001
To SMA	183 (5.5)	2 (0.2)	62 (5.8)	65 (9.8)	54 (8.1)	<.001
To right renal artery	299 (9.0)	0	138 (12.9)	87 (13.2)	74 (11.1)	<.001
To left renal artery	686 (20.7)	19 (2.1)	271 (25.4)	184 (27.9)	212 (31.7)	<.001
With 4-branch prefabricated graft	166 (5.0)	1 (0.1)	63 (5.9)	60 (9.1)	42 (6.3)	<.001
Endarterectomy	857 (25.9)	51 (5.6)	275 (25.8)	250 (37.9)	281 (42.0)	<.001
Stenting	264 (8.0)	15 (1.6)	86 (8.1)	73 (11.1)	90 (13.5)	<.001
Endarterectomy or stenting	940 (28.4)	58 (6.3)	303 (28.4)	270 (40.9)	309 (46.2)	<.001
Endarterectomy, stenting, or bypass	1360 (41.1)	88 (9.6)	510 (47.8)	357 (54.1)	405 (60.5)	<.001
Adjuncts						
Cerebrospinal fluid drainage†	1484 (44.8)	471 (51.5)	647 (60.7)	260 (39.4)	106 (15.8)	<.001
Left heart bypass	1480 (44.7)	557 (60.9)	874 (82.0)	43 (6.5)	6 (0.9)	<.001
LHB time, min	25 ± 9 (n = 1462)	26 ± 9 (n = 553)	24 ± 9 (n = 863)	22 ± 10 (n = 40)	27 ± 23 (n = 6)	.001
HCA	48 (1.5)	28 (3.1)	18 (1.7)	2 (0.3)	0	<.001
Cold renal perfusion	1934 (58.4)	158 (17.3)	701 (65.8)	511 (77.4)	564 (84.3)	<.001
Selective visceral perfusion	776 (23.5)	60 (6.6)	675 (63.3)	36 (5.5)	5 (0.7)	<.001

Values are n (%), mean ± standard deviation, or median [interquartile range]. *TEVAR*, Thoracic endovascular aortic repair; *EVAR*, endovascular aortic repair; *LSCA*, left subclavian artery; *ICA/LA*, intercostal/lumbar artery; *SMA*, superior mesenteric artery; *LHB*, left heart bypass; *HCA*, hypothermic circulatory arrest. *Full or partial extraction of endograft. Four extraction cases overlap by type of extraction, with simultaneous extraction of prior TEVAR and prior EVAR. †Intraoperative insertion of cerebrospinal fluid drainage catheter. Does not include postoperative use of cerebrospinal fluid drainage as a rescue measure.

Tex). Continuous variables are presented as mean ± standard deviation or median [interquartile range], as appropriate. Categorical variables are presented as number and percentage. Univariate comparisons were made with the Pearson chi-square test, Fisher exact test, or Kruskal–Wallis equality-of-populations rank test, as appropriate. All clinically relevant preoperative or intraoperative factors whose univariate association with outcomes had a P value of $< .05$ were entered into multivariable logistic regression models to identify independent predictors; postoperative complications were not entered into models.

Variables selected for multivariable modeling of operative death and adverse event were age, degenerative aneurysm without dissection, chronic aortic dissection, coronary artery disease, cerebrovascular disease, chronic renal insufficiency, pulmonary disease, rupture, urgent or emergency repair, aortic clamp time, visceral artery procedure, HCA, connective tissue disorder, and peripheral vascular disease; additionally, the variables symptomatic and extent I, II, or IV TAAA repair were entered into the operative death model, and the variables acute symptoms and extent II TAAA repair were entered into the adverse event model. Variables selected for multivariable modeling of permanent renal failure were age, genetically triggered disorder, chronic aortic dissection, cerebrovascular disease, chronic renal insufficiency, serum creatinine, chronic obstructive pulmonary disease, acute symptoms, peripheral vascular disease, rupture, emergency repair, extent I repair, extent II repair, aortic clamping proximal to the left subclavian artery, aortic cross-clamp time, maximum unprotected ischemic time of left or right renal artery, visceral artery bypass, visceral artery procedure, iliac or femoral bypass, HCA, cold renal perfusion, and selective visceral perfusion. Variables selected for multivariate modeling of permanent paraplegia and permanent paraplegia or paraparesis after extent II TAAA repair were age, genetically triggered disorder, chronic aortic dissection, coronary artery disease, serum creatinine, past or current tobacco use, chronic symptoms, intercostal or lumbar artery reattachment, and visceral artery endarterectomy or stent. Variables selected for multivariate modeling of permanent paraplegia and permanent paraplegia or paraparesis after extent III TAAA repair were cerebrovascular disease, serum creatinine, past or current tobacco use, rupture, emergency repair, aortic cross-clamp time, visceral artery procedure, and selective visceral perfusion.

Stepwise models with a removal criterion of $P \geq .1$ were estimated, after which multinomial logistic regression models were generated to present parameter estimates as relative risk ratios (RRRs). Model fit was assessed with likelihood ratio chi-square tests, goodness-of-fit tests, and pseudo R^2. During modeling, 272 missing values (0.8%) for preoperative serum creatinine level were replaced with mean values[23,24] based on matched categories of age, gender, and extent of repair, or with the postoperative creatinine value (if it was lower than the mean preoperative value). Survival over time and freedom from late events were estimated by the Kaplan–Meier or Cox regression methods.

RESULTS

Early Outcomes

There were 249 operative deaths (7.5%; Table 3), which included 37 of 193 repairs performed in octogenarians (19.2% mortality), 37 of 170 repairs involving rupture (21.8%), 14 of 439 repairs in patients aged 50 years or less (3.2%), 9 of 288 repairs in patients with Marfan syndrome (3.1%), and 58 of 1020 repairs of chronic dissection (5.7%). In 2586 elective repairs, operative death occurred after 161 repairs (6.2%, Table 4); in 723 urgent or emergency repairs, operative death occurred after 88 repairs (12.2%; $P < .001$). The rate of operative death differed among the 4 groups ($P = .002$), being higher in extents II and III (9.5% and 8.8%) than in extents I and IV (5.9% and 5.4%, respectively) (Table 3). The most common cause of operative death (Table 5) was MOF, which resulted in 173 deaths (67.5%) and was classified as primary MOF in 55 cases (31.8%) and as secondary MOF due to inciting complications in 118 cases (68.2%). In addition, there were 5 early repair failures (3 fistulas and 2 graft infections) that resulted in operative death.

Permanent paraplegia and paraparesis occurred after 97 (2.9%) and 81 (2.4%) repairs, respectively; only 5 of 439 repairs (1.1%) in patients aged ≤50 years resulted in permanent paraplegia. Of 189 patients (5.7%) with permanent renal failure, the majority (n = 107; 56.6%) did not survive to hospital discharge. Permanent stroke was relatively uncommon (n = 74; 2.2%). Unsurprisingly, adverse event rates were highest after extent II repair (n = 203; 19.0%) and lowest after extent IV repair (n = 67; 10.0%; $P < .001$ across all extents). Of the 3060 survivors, 229 (6.9%) were discharged with a life-altering complication; of these, renal failure was the most common (n = 82 [2.5%]), followed by paraparesis (n = 73 [2.2%]), paraplegia (n = 52 [1.6%]), and stroke (n = 35 [1.1%]).

Within our cohort, we found that the subset of patients with rupture (n = 170) included significantly more octogenarians (n = 25 [14.7%]) than the subset of patients without rupture (n = 168 [5.4%]; $P < .001$), and the patients with rupture more often had prior repair failure (n = 14 [8.2%] vs n = 122 [3.9%], $P = .015$). In octogenarians (n = 193), operative death (n = 37 [19.2%]) varied significantly by extent of repair ($P < .001$), which our prior publication also noted[25]; the rate of operative death was greatest in extent II repair (n = 14/38 [36.8%]) and lowest in extent IV repair (n = 6/61 [9.8%]). In octogenarians, the need for repair is often unexpected—more than one third of octogenarians underwent urgent or emergency repair (n = 69 [35.8%]), compared with only one fifth of nonoctogenarians (n = 654 [21.0%], $P < .001$).

In regard to the need for urgent or emergency repair, median aortic diameter was approximately 1 cm larger in patients who required such repair (6.9 [5.9-8.0] cm) than in those who underwent elective repair (6.1 [5.5-7.0] cm; $P < .001$). We compared preoperative and other characteristics between patients undergoing repair for aortic dissection (n = 1185) versus degenerative aneurysm without dissection (n = 2124) (Table 6); patients in the dissection cohort were younger, more often had connective tissue disorders, and, in general, were healthier. Of note, the median diameter at the time of repair was similar between groups (6.2 [5.5-7.0] cm vs 6.3 [5.6-7.2] cm), but extent II repair was significantly more frequent in patients with dissection (44.6%) than in patients with aneurysm without dissection (25.3%).

TABLE 3. Early outcomes

Variable	All n = 3309	Extent I n = 914	Extent II n = 1066	Extent III n = 660	Extent IV n = 669	*P* value
Adverse event*	478 (14.4)	98 (10.7)	203 (19.0)	110 (16.7)	67 (10.0)	<.001
Operative death	249 (7.5)	54 (5.9)	101 (9.5)	58 (8.8)	36 (5.4)	.002
In-hospital	237 (7.2)	50 (5.5)	97 (9.1)	56 (8.5)	34 (5.1)	.001
30-d†	159 (4.9)	34 (3.8)	68 (6.5)	34 (5.2)	23 (3.4)	.01
	(n = 3275)	(n = 899)	(n = 1052)	(n = 656)	(n = 668)	
During operation	11 (0.3)	6 (0.7)	3 (0.3)	2 (0.3)	0	.2
Cerebral complication (composite)	267 (8.1)	74 (8.1)	124 (11.6)	40 (6.1)	29 (4.3)	<.001
Encephalopathy	147 (4.4)	37 (4.0)	68 (6.4)	27 (4.1)	15 (2.2)	.001
Intracranial hemorrhage	29 (0.9)	6 (0.7)	15 (1.4)	6 (0.9)	2 (0.3)	.09
Seizure	32 (1.0)	7 (0.8)	15 (1.4)	8 (1.2)	2 (0.3)	.1
Stroke	98 (3.0)	33 (3.6)	46 (4.3)	9 (1.4)	10 (1.5)	<.001
Permanent‡	74 (2.2)	22 (2.4)	36 (3.4)	7 (1.1)	9 (1.3)	.004
Temporary	24 (0.7)	11 (1.2)	10 (0.9)	2 (0.3)	1 (0.1)	.04
Hemorrhagic	19 (0.6)	6 (0.7)	11 (1.0)	0	2 (0.3)	.03
Ischemic or embolic	82 (2.5)	27 (3.0)	38 (3.6)	9 (1.4)	8 (1.2)	.003
Transient ischemic attack	8 (0.2)	2 (0.2)	3 (0.3)	1 (0.2)	2 (0.3)	.9
Spinal cord deficit (composite)	317 (9.6)	71 (7.8)	147 (13.9)	71 (10.8)	27 (4.0)	<.001
Permanent‡ SCD	178 (5.4)	31 (3.4)	85 (8.0)	46 (7.0)	16 (2.4)	<.001
Permanent‡ paraplegia	97 (2.9)	12 (1.3)	50 (4.7)	29 (4.4)	6 (0.9)	<.001
Immediate	50 (1.5)	6 (0.7)	26 (2.4)	14 (2.1)	4 (0.6)	.001
Delayed	47 (1.4)	6 (0.7)	24 (2.3)	15 (2.3)	2 (0.3)	.001
Permanent‡ paraparesis	81 (2.4)	19 (2.1)	35 (3.3)	17 (2.6)	10 (1.5)	.1
Immediate	29 (0.9)	3 (0.3)	18 (1.7)	4 (0.6)	4 (0.6)	.006
Delayed	52 (1.6)	16 (1.8)	17 (1.6)	13 (2.0)	6 (0.9)	.006
Temporary SCD	146 (4.4)	41 (4.5)	65 (6.1)	28 (4.2)	12 (1.8)	<.001
Temporary paraparesis	94 (2.8)	26 (2.8)	46 (4.3)	15 (2.3)	7 (1.0)	.001
Temporary paraplegia	52 (1.6)	15 (1.6)	19 (1.8)	13 (2.0)	5 (0.7)	.3
Neurogenic bladder	22 (0.7)	2 (0.2)	12 (1.1)	6 (0.9)	2 (0.3)	.04
Acute renal dysfunction	406 (12.3)	73 (8.0)	170 (15.9)	81 (12.3)	82 (12.3)	<.001
Renal failure necessitating dialysis	250 (7.6)	45 (4.9)	102 (9.6)	52 (7.9)	51 (7.6)	.002
Permanent‡	189 (5.7)	29 (3.2)	78 (7.3)	43 (6.5)	39 (5.8)	.001
Temporary	61 (1.8)	16 (1.8)	24 (2.3)	9 (1.4)	12 (1.8)	.6
Cardiac complication (composite)	860 (26.0)	221 (24.2)	334 (31.3)	158 (23.9)	147 (22.0)	<.001
Atrial arrhythmia	541 (16.3)	147 (16.1)	215 (20.2)	93 (14.1)	86 (12.9)	<.001
Myocardial infarction	41 (1.2)	12 (1.3)	13 (1.2)	10 (1.5)	6 (0.9)	.8
Cardiac tamponade	10 (0.3)	5 (0.5)	5 (0.5)	0	0	.08
Drainage of pericardial effusion	20 (0.6)	7 (0.8)	9 (0.8)	1 (0.2)	3 (0.4)	.3
Pulmonary complication (composite)	1185 (35.8)	347 (38.0)	455 (42.7)	223 (33.8)	160 (23.9)	<.001
Respiratory failure	830 (25.1)	225 (24.6)	330 (31.0)	159 (24.1)	116 (17.3)	<.001
Necessitating tracheostomy	281 (8.5)	74 (8.1)	131 (12.3)	46 (7.0)	30 (4.5)	<.001
ARDS	140 (4.2)	41 (4.5)	63 (5.9)	20 (3.0)	16 (2.4)	.001
Atelectasis necessitating bronchoscopy	333 (10.1)	109 (11.9)	135 (12.7)	52 (7.9)	37 (5.5)	<.001
Chylothorax	33 (1.0)	11 (1.2)	8 (0.8)	8 (1.2)	6 (0.9)	.7
Drainage of pleural effusion	233 (7.0)	81 (8.9)	83 (7.8)	49 (7.4)	20 (3.0)	<.001
Pneumonia	225 (6.8)	62 (6.8)	88 (8.3)	41 (6.2)	34 (5.1)	.07
Evacuation of pneumothorax	68 (2.1)	19 (2.1)	25 (2.3)	15 (2.3)	9 (1.3)	.5
Pulmonary embolism	28 (0.8)	9 (1.0)	8 (0.8)	5 (0.8)	6 (0.9)	.9
Reintubation	479 (14.5)	130 (14.2)	190 (17.8)	99 (15.0)	60 (9.0)	<.001
Left vocal cord paralysis	502 (15.2)	239 (26.1)	248 (23.3)	9 (1.4)	6 (0.9)	<.001
Bleeding requiring reoperation	110 (3.3)	33 (3.6)	40 (3.8)	20 (3.0)	17 (2.5)	.5
Gastrointestinal complication (composite)	126 (3.8)	20 (2.2)	45 (4.2)	30 (4.5)	31 (4.6)	.03
Gastrointestinal ischemia	31 (0.9)	2 (0.2)	11 (1.0)	9 (1.4)	9 (1.3)	.05
Pancreatitis	9 (0.3)	1 (0.1)	4 (0.4)	2 (0.3)	2 (0.3)	.7
Gastrointestinal bleeding	63 (1.9)	8 (0.9)	23 (2.2)	19 (2.9)	13 (1.9)	.03
Gastrointestinal obstruction	37 (1.1)	10 (1.1)	10 (0.9)	4 (0.6)	13 (1.9)	.1

(Continued)

TABLE 3. Continued

Variable	All n = 3309	Extent I n = 914	Extent II n = 1066	Extent III n = 660	Extent IV n = 669	*P* value
Sepsis	148 (4.5)	34 (3.7)	60 (5.6)	29 (4.4)	25 (3.7)	.1
Wound infection	108 (3.3)	31 (3.4)	51 (4.8)	17 (2.6)	9 (1.3)	.004
Discharge to long-term acute care	274 (8.3)	61 (6.7)	113 (10.6)	60 (9.1)	40 (6.0)	.001
Early survival with life-altering complication§	229 (6.9)	44 (4.8)	102 (9.6)	52 (7.9)	31 (4.6)	<.001
Survival with renal failure (dialysis)	82 (2.5)	12 (1.3)	36 (3.4)	17 (2.6)	17 (2.5)	.03
Survival with permanent stroke	35 (1.1)	9 (1.0)	18 (1.7)	4 (0.6)	4 (0.6)	.08
Survival with permanent paraplegia	52 (1.6)	7 (0.8)	25 (2.3)	17 (2.6)	3 (0.4)	.001
Survival with permanent paraparesis	73 (2.2)	17 (1.9)	31 (2.9)	16 (2.4)	9 (1.3)	.1
Early survivors‖: length of intensive care unit stay, d	4 [3-6]	4 [3-7]	4 [3-8]	4 [3-6]	3 [2-5]	<.001
Early survivors‖: length of hospital stay, d	12 [9-17]	12 [9-17]	13 [10-20]	11 [9-15]	10 [8-13]	<.001

Values are n (%) or median [interquartile range]. *ARDS*, Acute respiratory distress syndrome. *Adverse event is defined as operative death or permanent (present at hospital discharge) stroke, paraplegia, paraparesis, or renal failure necessitating dialysis. †Patients without 30-day follow-up (n = 34) are removed. ‡Present at the time of hospital discharge or early death. §Discharged with stroke, paraplegia, paraparesis, or renal failure necessitating dialysis (n = 3060). ‖Early survivors (n = 3060).

Independent predictors of adverse event, permanent renal failure, permanent paraplegia or paraparesis, and operative death are shown in Tables 7, 8, and 9. Regarding adverse event, permanent renal failure, and operative death, greater age (by each year), rupture, the need for a visceral artery procedure, and HCA were associated with greater risk. In the operative death model, extent IV repair was associated with a 50% reduction in risk (RRR = 0.5, P = .001). Extent II repair was associated with increased risk in the adverse event and permanent renal failure models (RRR = 1.49, P = .001; RRR = 1.39, P = .05, respectively). In regard to modeling for paraplegia, we developed separate analyses for extents II and III, which were associated with higher rates of paraplegia than extents I and IV (4.7% and 4.4% vs 1.3% and 0.9%, respectively); notably, independent predictors (Table 8) were different between the 2 paraplegia models. For patients who underwent extent II repairs, coronary artery disease and chronic symptoms increased the risk of paraplegia; however, having a genetically triggered disorder or the intraoperative reattachment of intercostal or lumbar arteries decreased risk. For patients who underwent extent III repairs, cerebrovascular disease, emergency repair, and selective perfusion of the visceral arteries were associated with increased risk of paraplegia—no characteristics were associated with decreased risk.

To assess the effects of era on surgical outcomes, we stratified the data and applied the overall operative mortality multivariable model to all repairs by surgical era (Table 9); only age at repair was significantly predictive of operative mortality in all 3 eras. In Era 1, chronic renal insufficiency was highly predictive of operative death, but it was less predictive in Eras 2 and 3. In Era 2, rupture was highly predictive of operative death, but it was less so in Era 3. In Era 3, preoperative pulmonary disease played a considerably larger role than it did in Eras 1 and 2. We then used the overall operative mortality model to determine the predicted probability of operative death for Eras 1, 2, and 3. The predicted operative mortality rates by era were 5.4% (Era 1), 4.9% (Era 2), and 6.4% (Era 3). Unfortunately, the observed/expected frequency ratios were greater than 1 for each era (Era 1 = 1.3; Era 2 = 1.4; Era 3 = 1.4), indicating that the model underpredicted operative mortality.

TABLE 4. Results of consecutive elective cases (n = 2586)

Variable	All n = 2586	Extent I n = 700	Extent II n = 866	Extent III n = 504	Extent IV n = 516	*P* value
Adverse event	329 (12.7)	63 (9.0)	154 (17.8)	73 (14.5)	39 (7.6)	<.001
Operative mortality	161 (6.2)	32 (4.6)	72 (8.3)	41 (8.1)	16 (3.1)	<.001
Permanent paraplegia*	66 (2.6)	8 (1.1)	37 (4.3)	18 (3.6)	3 (0.6)	<.001
Permanent paraparesis*	57 (2.2)	14 (2.0)	25 (2.9)	10 (2.0)	8 (1.6)	.4
Permanent renal failure necessitating dialysis*	132 (5.1)	17 (2.4)	64 (7.4)	28 (5.6)	23 (4.5)	<.001
Permanent stroke*	60 (2.3)	17 (2.4)	31 (3.6)	5 (1.0)	7 (1.4)	.007
Survival with life-altering complication†	168 (6.5)	31 (4.4)	82 (9.5)	32 (6.3)	23 (4.5)	<.001

Values are n (%). Outcomes of interest (paraplegia, paraparesis, renal failure necessitating dialysis, and stroke) are permanent complications present at discharge or present in those patients with early death. *Excludes 5 patients who died during the operation. †Discharge with permanent paraplegia, paraparesis, renal failure, or stroke in 2425 early survivors of elective repair.

TABLE 5. Causes of early death (n = 249 of 3309 patients)

Cause	No. (%)
Multisystem organ failure	173 (67.5)
Primary (n = 55)	
Secondary (n = 118)	
Cardiac failure	28 (11.2)
Respiratory failure	13 (5.2)
Stroke	10 (4.0)
Unknown	9 (3.6)
Exsanguination	5 (2.0)
Gastrointestinal bleed	4 (1.6)
Pulmonary embolism	4 (1.6)
Neoplasm	1 (0.4)
Intracranial hemorrhage	1 (0.4)
Suicide	1 (0.4)

Late Outcomes

There were 1864 late deaths. Estimated survival was 83.5% ± 0.7% at 1 year, 63.6% ± 0.9% at 5 years, 36.8% ± 1.0% at 10 years, and 18.3% ± 0.9% at 15 years (Figure 3, *A*). Over time, there were 88 late repair failures; these included failures with overlapping causes, such as graft infection (n = 18), pseudoaneurysm formation (n = 43), and patch aneurysm (n = 33). Freedom from repair failure was 97.9% ± 0.3% at 5 years, 95.3% ± 0.6% at 10 years, and 94.1% ± 0.8% at 15 years (Figure 3, *B*). Of these 88 patients, 44 had aortic reintervention; 29 of these 88 patients (33.0%) remain alive. Patients with chronic dissection had significantly higher survival rates than patients without aortic dissection (Figure 3, *C*), even after correction for age (using the mean age, 64.6 years) (Figure 3, *D*).

DISCUSSION

Patients with TAAA face an extremely difficult clinical situation.[26] They risk life-threatening rupture without TAAA repair; however, repair itself carries substantial risk, including risk of death or, in operative survivors, life-altering complications such as permanent stroke, paraplegia, and renal failure necessitating dialysis. We and others[7,13,27-29] long ago established that the postoperative risks vary by the extent of TAAA repair. Rates of early mortality range from 5% to 8% for extent I, 8% to 13% for extent II, 8% to 21% for extent III, and 2% to 6% for extent IV at experienced centers[27,30-32]; likewise, our results vary significantly by extent of repair (I = 5.9%, II = 9.5%, III = 8.8%, IV = 5.4%; *P* = .002). As expected, we found that

TABLE 6. Comparison of preoperative and other factors in aortic dissection (n = 1185) versus aneurysm without dissection (n = 2124)

Variable	All n = 3309	Aortic dissection n = 1185	Aneurysm without dissection n = 2124	*P* value
Age (y)	67 [59-73]	58 [48-68]	70 [65-75]	<.001
Age ≤50 y	439 (13.3)	354 (29.9)	85 (4.0)	<.001
Age >79 y	193 (5.8)	24 (2.0)	169 (8.0)	<.001
Maximum distal aortic diameter, cm	6.2 [5.5-7.2] (n = 2936)	6.2 [5.5-7.0] (n = 1077)	6.3 [5.6-7.2] (n = 1859)	.1
Genetically triggered disorder	523 (15.8)	427 (36.0)	96 (4.5)	<.001
Connective tissue disorder	330 (10.0)	303 (25.6)	27 (1.3)	<.001
Hypertension	2805 (84.8)	992 (83.7)	1813 (85.4)	.2
Hyperlipidemia	967 (29.2)	261 (22.0)	706 (33.2)	<.001
Diabetes	261 (7.9)	72 (6.1)	189 (8.9)	.004
Coronary artery disease	1232 (37.2)	304 (25.7)	928 (43.7)	<.001
Prior myocardial infarction	652 (19.7)	156 (13.2)	496 (23.4)	<.001
Pulmonary disease	1298 (39.2)	293 (24.7)	1005 (47.3)	<.001
Cerebrovascular disease	575 (17.4)	163 (13.8)	412 (19.4)	<.001
Prior stroke	334 (10.1)	110 (9.3)	224 (10.5)	.3
Peripheral vascular disease	885 (25.8)	184 (15.5)	671 (31.6)	<.001
Past or current tobacco use	2616 (79.1)	751 (63.4)	1865 (87.8)	<.001
Chronic renal insufficiency	99 (3.0)	28 (2.4)	71 (3.3)	.1
Chronic renal failure (dialysis)	60 (1.8)	18 (1.5)	42 (2.0)	.4
Symptomatic	2117 (64.0)	783 (66.1)	1334 (62.8)	.06
Acute symptoms	549 (16.6)	252 (21.3)	297 (14.0)	<.001
Chronic symptoms	1664 (50.3)	567 (47.8)	1097 (51.6)	.04
Rupture	170 (5.1)	42 (3.5)	128 (6.0)	.002
Urgent or emergency repair	723 (21.8)	304 (25.7)	419 (19.7)	<.001
Extent II repair	1066 (32.3)	528 (44.6)	538 (25.3)	<.001
Redo thoracotomy	563 (17.0)	190 (16.0)	373 (17.6)	.3

Values are n (%) or median [interquartile range].

TABLE 7. Relative risk ratios for preoperative and operative predictors of adverse events (n = 481) and permanent renal failure (n = 190) among all patients

Variable	Outcome	
	RRR (CI)	*P* value
Adverse event model		
HCA	4.16 (2.09-8.28)	<.001
Rupture	2.42 (1.61-3.62)	<.001
Chronic renal insufficiency	2.32 (1.45-3.71)	<.001
Extent II TAAA repair	1.49 (1.18-1.88)	.001
Urgent or emergency repair	1.42 (1.10-1.84)	.007
Endarterectomy, stenting, or bypass of visceral arteries	1.36 (1.11-1.68)	.004
Cerebrovascular disease	1.24 (0.97-1.60)	.09
Age, y	1.05 (1.03-1.06)	<.001
Aortic clamp time, min*	1.02 (1.01-1.02)	<.001
Connective tissue disorder	0.60 (0.34-1.05)	.07
Permanent renal failure model		
HCA	3.16 (1.15-8.68)	.03
Rupture	2.66 (1.64-4.32)	<.001
Endarterectomy, stent, or bypass of visceral arteries	1.53 (1.08-2.17)	.02
Extent II TAAA repair	1.39 (0.99-1.95)	.054
Preoperative serum creatinine level, mg/dL†	1.38 (1.22-1.55)	<.001
Age, y	1.06 (1.05-1.08)	<.001
Maximum left or right renal artery unprotected ischemic time, min*,‡	1.02 (1.01-1.03)	<.001
Aortic clamp time, min*	1.01 (1.00-1.01)	.007

RRR, Relative risk ratio; *CI*, confidence interval; *HCA*, hypothermic circulatory arrest; *TAAA*, thoracoabdominal aortic aneurysm. *Variable is not thought to make a substantial clinical contribution, because the RRR approaches 1. †Missing preoperative serum creatinine values were substituted with mean values that were matched by age, gender, and extent of TAAA repair. However, if the peak postoperative value was lower than the matched mean value, then this value was used. ‡The greater value of either renal artery. Unprotected ischemic time is the total ischemic time minus left heart bypass time. For continuous data, RRR is per unit.

extent II repairs confer the greatest overall risk of mortality and morbidity.[33]

However, it is clear that risk stems from more than just the amount of aorta replaced during repair and that patient-specific variables also play a substantial role in poor postoperative outcomes. Going further, the process by which extensive aneurysms (extents I and II) form often is different from what occurs in the development of less-extensive aneurysms (extents III and IV). Of note, we found that patients with dissection generally had fewer comorbidities than patients with degenerative aneurysm without dissection (Table 6), with the caveat that some of the health conditions we studied (eg, hyperlipidemia, coronary artery disease, cerebrovascular disease) are typically associated with the aging process itself. Clearly, there are select groups of patients who should expect good to excellent outcomes after even the most extensive TAAA repair (extent II repair); typically, this includes patients aged 50 years or less, patients with connective tissue disorders,[34,35] and to a lesser extent, patients with chronic aortic dissection.[36] Likewise, there are select groups of patients for whom extent II TAAA repair might exceed a reasonable level of risk, such as octogenarians.[25,37,38]

Age remains a powerful predictor of poor postoperative outcomes, and greater age is an independent predictor of adverse event, operative mortality, and permanent renal failure. Likewise, rupture is an independent predictor of adverse events, operative mortality, and permanent renal failure; rupture predicts the patient's risk of permanent renal failure more strongly than it predicts operative mortality or adverse events, more than doubling the risk (RRR = 2.66).

In regard to permanent spinal cord deficits, approximately half of all cases of permanent paraplegia occurred in a delayed fashion and developed after spinal cord function was initially found to be unimpaired; this observation did not differ among extents of repair. We observed that permanent paraplegia primarily affected patients who underwent extent II or III repair, but we noted that these 2 groups of patients had dissimilar preoperative characteristics. Subsequently, we developed separate paraplegia models for the extent II and III cohorts—partly because of this dissimilarity and partly because of speculation that in extent II repair, paraplegia results from the interruption and loss of spinal-perfusing arteries, whereas in extent III repair, embolism may play a larger role.

Although both paraplegia models underwent the same preliminary analysis, the final models did not overlap. Among other factors, we found reattachment of intercostal or lumbar arteries in extent II repair to be an independent predictor of substantially lower risk of permanent paraplegia (RRR = 0.46, $P = .03$). In contrast, paraplegia after extent III repair was independently predicted by cerebrovascular disease (RRR = 3.08, $P = .006$), emergency repair (RRR = 3.44, $P = .01$), and the use of selective visceral perfusion (RRR = 8.11, $P < .001$). Cerebrovascular disease could be indicative of generalized atherosclerotic disease, which tends to be more common in the slightly older extent III cohort. When we expanded our analysis to include both permanent paraplegia and paraparesis, the extent II model was largely unchanged, but in the extent III model, the need for a visceral artery procedure (eg, endarterectomy, stent, or bypass; RRR = 2.51, $P = .009$) replaced cerebrovascular disease in the model—the need for such procedures can be interpreted as a marker for atherosclerotic disease, which tends to increase embolic debris. Tanaka and colleagues[39] recently identified embolism as a major cause of spinal cord deficit after distal aortic repair. In a small study, Zangrillo and coauthors[40] found postoperative paraplegia concentrated in patients with extent II and III repairs; the authors speculated that in patients with spinal cord

TABLE 8. Relative risk ratio for preoperative and operative predictors of permanent paraplegia or permanent paraparesis after extent II (n = 1066) or extent III (n = 660) repair

Variable	Outcome	
	RRR (CI)	*P* value
Permanent paraplegia after extent II TAAA repair (n = 50)		
Chronic symptoms of aortic disease	3.00 (1.56-5.75)	.001
Past or current tobacco use	2.75 (0.83-9.15)	.10
Coronary artery disease	1.92 (1.07-3.43)	.03
Reattachment of intercostal or lumbar arteries	0.46 (0.23-0.93)	.03
Genetically triggered disorder	0.23 (0.05-0.98)	.047
Permanent paraplegia or paraparesis after extent II TAAA repair (n = 85)		
Chronic symptoms of aortic disease	1.83 (1.15-2.91)	.01
Coronary artery disease	1.78 (1.13-2.81)	.01
Reattachment of intercostal or lumbar arteries	0.55 (0.31-0.99)	.045
Genetically triggered disorder	0.37 (0.17-0.82)	.02
Permanent paraplegia after extent III TAAA repair (n = 29)		
Selective perfusion of visceral arteries	8.11 (3.06-21.45)	<.001
Emergent repair	3.44 (1.28-9.22)	.01
Cerebrovascular disease	3.08 (1.39-6.82)	.006
Permanent paraplegia or paraparesis after extent III TAAA repair (n = 46)		
Selective perfusion of visceral arteries	5.61 (2.40-13.12)	<.001
Endarterectomy, stent, or bypass of visceral arteries	2.51 (1.26-4.98)	.009
Emergency repair	2.23 (0.97-5.12)	.06

RRR, Relative risk ratio; *CI*, confidence intervals; *TAAA*, thoracoabdominal aortic aneurysm.

circulation already damaged by arterial plaque or mural thrombi, aortic clamping may exacerbate spinal ischemia, which was evidenced by elevated levels of intrathecal lactate. Of note, in both of our paraplegia models, age was not predictive of permanent paraplegia, and, somewhat surprisingly, neither was rupture.

Although cerebrovascular disease was present in approximately one fifth of all patients (n = 575, 17.4%), permanent stroke was a relatively infrequent postoperative complication in our series (overall rate of 2.2%; the rate was lowest after extent III repairs [1.1%] and highest after extent II repairs [3.4%]). An analysis of National Surgical Quality Improvement Program data[41] regarding 450 open surgical repairs of nonruptured TAAA found a similarly low overall rate of stroke (2.2%). In single-center reports of TAAA repair, the incidence of stroke ranges from 1.9% to 8.9%.[27,42,43] Several centers favor the routine use of HCA in TAAA repair, but such use typically does not include extent IV repairs, and a few of these centers further restrict standard HCA use to extent II TAAA repair. In centers that routinely use HCA, the contemporary incidence of stroke ranges from 3.7% to 6.3%.[44,45]

Pulmonary complication, a broad category composed of several variables (shown in Table 3), remained the most common complication,[10,11,27,37,46] occurring after 35.8% of all TAAA repairs and 42.7% of extent II repairs. Pulmonary complications were expected because of the single-lung ventilation that extensive TAAA repair necessitates. Similar to our previously published results,[10,11] cardiac complications occurred after 26.0% of TAAA repairs; again, in extent II repairs, the rate was higher (31.3%). Many of these complications were cases of atrial arrhythmia (which occurred after 20.2% of extent II repairs), which is a poorly understood complication of TAAA repair. Paralysis of the left vocal cord is another common complication[10,11]; although the rate was 15.4% overall, it was higher (26.1%) after extent I repair. In addition to causing hoarseness, left vocal cord paralysis can compromise pulmonary function; recurrent laryngeal nerve injury can result from localized

TABLE 9. Relative risk ratio stratified by surgical era for preoperative and operative predictors of operative mortality (n = 249) after 3309 thoracoabdominal aortic aneurysm repairs

Variable	Operative death (nonstratified model)		Era 1 (≤1998)		Era 2 (1999-2004)		Era 3 (2005-2014)	
	RRR (CI)	*P* value	RRR (CI)	*P* value	RRR (CI)	*P* value	RRR (CI)	*P* value
HCA	2.69 (1.12-6.46)	.03	5.56×10^{-6}*	.9	2.50 (0.46-13.62)	.3	3.90 (1.27-11.94)	.02
Rupture	2.38 (1.47-3.83)	<.001	1.67 (0.71-3.94)	.2	3.69 (1.48-9.18)	.005	2.26 (1.02-4.98)	.04
Chronic renal insufficiency	1.98 (1.09-3.56)	.02	2.91 (1.03-8.24)	.045	1.16 (0.33-4.10)	.8	2.32 (0.91-5.91)	.08
Symptomatic at time of repair	1.62 (1.16-2.25)	.004	1.94 (1.08-3.50)	.03	1.49 (0.86-2.58)	.2	1.36 (0.73-2.51)	.3
Endarterectomy, stent, or bypass of visceral arteries	1.46 (1.10-1.94)	.009	1.55 (0.94-2.57)	.09	1.18 (0.71-1.95)	.5	1.55 (0.93-2.58)	.1
Urgent or emergency repair	1.35 (0.96-1.91)	.09	1.49 (0.82-2.69)	.2	0.83 (0.42-1.63)	.6	1.90 (1.27-3.41)	.02
Cerebrovascular disease	1.34 (0.97-1.85)	.08	1.34 (0.73-2.43)	.9	1.27 (0.71-2.27)	.8	1.42 (0.83-2.40)	.2
Pulmonary disease	1.29 (0.98-1.69)	.07	0.92 (0.57-1.49)	.7	1.23 (0.74-2.01)	.4	1.96 (1.23-3.12)	.005
Age, y	1.05 (1.04-1.07)	<.001	1.05 (1.02-1.08)	<.001	1.04 (1.02-1.07)	.001	1.07 (1.04-1.09)	<.001
Aortic clamp time, min†	1.01 (1.01-1.02)	<.001	1.02 (1.01-1.03)	.001	1.01 (1.00-1.01)	.06	1.02 (1.01-1.03)	<.001
Extent IV TAAA repair	0.52 (0.35-0.76)	.001	0.55 (0.27-1.10)	.09	0.70 (0.35-1.40)	.3	0.37 (0.19-0.69)	.002

RRR, Relative risk ratio; *CI*, confidence interval; *HCA*, hypothermic circulatory arrest; *TAAA*, thoracoabdominal aortic aneurysm. *Value is too small to be meaningful in Era 1.
†Variable is not thought to make a substantial clinical contribution, because the RRR approaches 1. For continuous data, RRR is per unit.

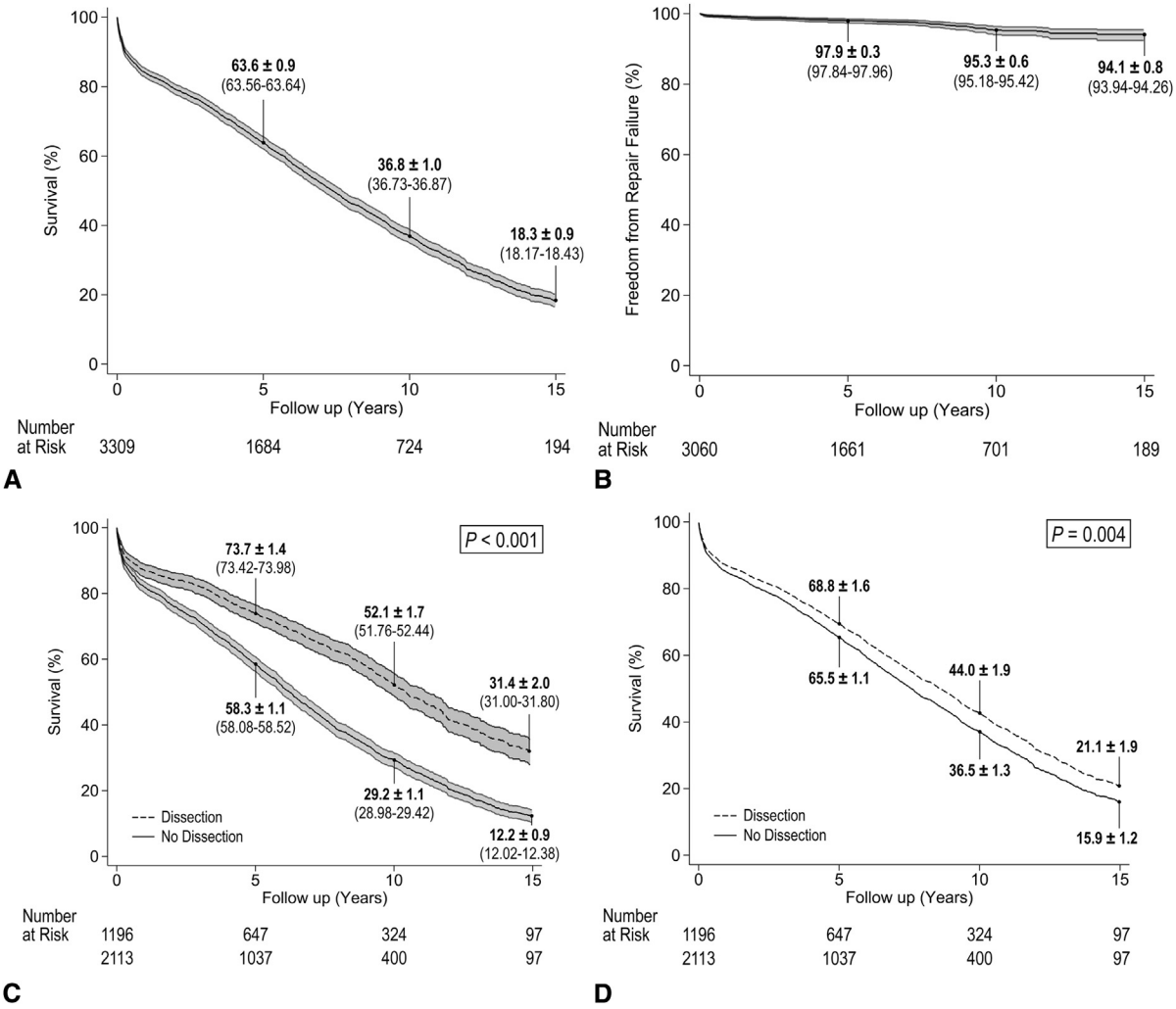

FIGURE 3. Kaplan–Meier curves of estimated survival (A) after 3309 TAAA repairs and (B) freedom from repair failure in 3060 early survivors. Survival was stratified by aortic dissection versus degenerative aneurysm without dissection (C). When we corrected for age (mean age, 64.6 years) by using a Cox regression model in analyzing survival after repair of aortic dissection versus degenerative aneurysm without dissection, (D) a survival advantage for patients with aortic dissection remained statistically significant ($P = .004$). Confidence intervals (95%) are shown in parentheses. *TAAA*, Thoracoabdominal aortic aneurysm.

trauma during exposure, aortic clamping, or suturing, especially when working proximal to the left subclavian artery in patients with chronic dissection.

Although our data show that open TAAA repair produces respectable outcomes when performed at an experienced center, there is clearly room for improvement. Surgical TAAA repair continues to pose substantial risks in the current era, particularly when the entire thoracoabdominal aorta (extent II) is replaced. It is certainly possible that incorporating additional interventions aimed at reducing rates of major complications would be of benefit to our practice, and we have actively explored doing so. For example, we briefly attempted to use motor-evoked potential monitoring techniques to reduce the incidence of spinal cord deficits,[43] but because of logistical problems, we had difficulty obtaining consistently accurate, useful information, and we ultimately abandoned the effort. We will continue to explore the use of existing and investigational adjuncts in an effort to improve outcomes.

Study Limitations

Limitations of this study include its being a retrospective study from a single center. Although we collected prospective data during rounds for all patients who underwent TAAA repair since 2006, data for those who underwent operation before 2006 were collected solely by retrospective review of medical records. Thus, because of the inherent inadequacies of relying on past medical records, it is likely that before 2006 we did not fully capture preoperative characteristics or postoperative complications; this may have affected our predictive modeling efforts, including efforts to determine whether the surgical era

(1, 2, or 3) at the time of repair affected adverse outcomes. Other authors have similarly reported the inability to detect sustained improvement in complication rates between surgical eras.[46] Also, because of the large number of late deaths, we were unable to devote sufficient resources to determine causes for each death, so we may have undercaptured repair failure, and we were unable to determine the number of late deaths that resulted from aortic rupture. Although the use of HCA was found to be predictive of operative death, adverse event, and permanent renal failure, there is considerable selection bias regarding our use of HCA,[47] which we reserve for highly complex aortic repairs in which the proximal aorta is unclampable; thus, these findings are not generalizable to centers that use HCA as a standard technique. Likewise, in extent III repair, the selective use of visceral artery perfusion (n = 36, 5.5%) is biased toward atypically complex preoperative characteristics, so we suspect that its predictiveness of paraplegia or paraparesis in extent III repair is due to confounding factors and cannot be generalized to other aortic centers.

Although the dawn of endovascular TAAA (eTAAA) repair is clearly upon us, it is not yet clear whether this approach will benefit all patients or only select groups of them. In the United States, eTAAA remains an experimental approach, so its use here has been limited to a handful of centers. It is not clear what will happen when its use is broadened—real-world experience suggests that risk often increases when carefully crafted, industry-based criteria for use are loosened. At present, many eTAAA devices must be customized for the patient, which requires several weeks' notice, but off-the-shelf modular and fenestrated devices are in development. We know that compared with open TAAA repair, eTAAA repair is associated with lower operative mortality and length of hospitalization, whereas rates of mesenteric ischemia, paraplegia, branch-vessel occlusion, and late reintervention are often higher. Most pertinent, the reported rates of spinal cord deficit after extent II eTAAA have ranged as high as 30% to 50%.[48,49]

Comparison of open and eTAAA repair is complicated by the fact that Crawford extents of repair do not apply perfectly to eTAAA repairs. For example, an extent III TAAA open repair involves replacing the distal half of the descending thoracic aorta and a substantial portion of the abdominal aorta. In eTAAA for the same type of aneurysm, an even larger segment of the aorta needs to be covered (by approximately 4-6 cm, to secure proximal and distal landing zones), typically without revascularization of the intercostal and lumbar arteries; this can result in "upstaging" of the extent of repair, in that an aneurysm that would be treated by open extent III repair would need an extent II eTAAA repair because the area of coverage is extended across extent boundaries. Although efforts are being made to compensate for the resulting loss of perfusion by promoting spinal cord preconditioning or by creating intentional endoleaks,[50] this problem may prove to be a "real-world" limitation of eTAAA repair. In addition, one must consider that in certain subsets of younger patients, extent II TAAA repair can be performed at low risk of adverse outcomes. We know that in elderly patients, in whom extent III and IV TAAA repairs are common, the need for repair is often unexpected—more than one third of these patients' repairs are performed urgently or emergently, often in response to acute symptoms or rupture. These patients may benefit from off-the-shelf eTAAA devices, with the caveat that these patients commonly have severe atherosclerosis, and embolic debris may be discharged during repair, potentially causing stroke or paraplegia. Thus, the preferential adoption of eTAAA should be tempered by caution, and a balance should be sought with open repair. In the future, a larger challenge may be maintaining sufficient skill among aortic surgeons to perform open TAAA repair safely and effectively when it is needed.

CONCLUSIONS

We have presented our single-practice experience with TAAA repair, which spans approximately 3 decades. Although we have refined our operative technique over time in an effort to prevent early death and life-altering complications, continued improvement is needed. A better understanding of preoperative risk factors may enhance efforts to optimize patient health before repair, but the low-hanging fruit of reducing rates of urgent and emergent repair remain. For example, improving patient adherence to surveillance imaging protocols may reduce risk of TAAA rupture. More than one third of repairs were performed in response to the progressive aortic dilatation that is common in chronic distal aortic dissection; in general, these patients tend to do better than expected, and given this information, they may better prepare for the likelihood of needing additional aortic repair in the future and seek elective repair.

Conflict of Interest Statement

J.S.C. receives royalties for the 4-branched Coselli thoracoabdominal aortic graft and spoke at a sponsored meeting held by Vascutek Terumo; consults and participates in clinical research for Medtronic, Inc, Vascutek Terumo, Edwards Lifesciences, and WL Gore & Associates; and participates in clinical research for GlaxoSmithKline and Cook, Inc. S.A.L. serves as an investigator in clinical research for Vascutek Terumo; serves as a co-investigator in clinical research for Medtronic, Inc, WL Gore & Associates, Cook, Inc, and GlaxoSmithKline; has served as a consultant with Medtronic PS Medical, Inc; and has served on Advisory Boards for Baxter Health Care. O.P.

consults with Medtronic, Inc, and has received travel fees from WL Gore & Associates and Cook, Inc. T.K.R. serves as an investigator in clinical research for Edwards Lifesciences. All other authors have nothing to disclose with regard to commercial support.

You can watch a Webcast of this AATS meeting presentation by going to: http://webcast.aats.org/2015/Video/Monday/04-27-15_4E_0730_Coselli.mp4.

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

Discussion

Dr N. Kouchoukos *(St Louis, Mo)*. Dr Coselli and colleagues have presented their truly monumental experience with open thoracoabdominal aneurysm repair, more than 3000 procedures in an approximately 30-year span. This is the largest series of surgically treated patients ever reported, and it is unlikely that an experience of this magnitude will ever be duplicated. Those of us who perform or have observed thoracoabdominal aneurysm repairs recognize that the intense effort, both physical and intellectual, and the teamwork and man-hours required to achieve the excellent outcomes as he has reported are truly extraordinary.

Dr Coselli has based his experience on the pioneering work of his distinguished predecessors at Baylor College of Medicine, Drs Michael DeBakey, Denton Cooley, and Stanley Crawford, who were among the first to perform these procedures and who established a center for the treatment of aortic disease that is world renowned and remains at the forefront. I am certain that Stanley Crawford, wherever he is, is smiling and extremely proud of what Dr Coselli has accomplished to advance the treatment of thoracoabdominal aortic disease.

The article, as you saw, contains an enormous amount of extremely important and rigorously analyzed data that could not be adequately presented in the time allotted. I will limit my remarks and questions to a few specific areas.

Dr Coselli, did you observe a reduction in mortality and in the prevalence of major complications over time as you modified your technique? This was not directly addressed in the article because the variable of year of operation did not appear in the multivariate analysis.

Dr Coselli. Dr Kouchoukos, your efforts have been at the forefront of TAAA repair for many years. We did a rigorous analysis by various eras. We looked at all of the major morbidities and mortality, and I will have to say that even with controlling for increased risks of patient comorbidity over time, the actual results did not significantly change from our earliest era to our most contemporary era. Of note, we did identify brief time periods in which outcomes improved, such as several periods when we had no cases of paraplegia for more than a year.

Dr Kouchoukos. You observed in your study and others, including our group, have observed that the Crawford Extent II and III procedures are associated with the highest early mortality and prevalence of major complications, most important, permanent paralysis. Do you believe that the risks of spinal cord ischemic injury could be reduced by the addition of other interventions, such as monitoring of evoked potentials, deeper hypothermia, pharmacologic agents, and avoidance of sequential clamping to reduce the risk of embolization, or have we reached an irreducible minimum risk for this complication?

Dr Coselli. We report solely our own experience, and thus, can only speculate on what altering that experience might derive. I would say this, that in the earlier years, and particularly with Stanley Crawford, our greatest concerns were in the Extent II category and cases of aortic dissection. But with cerebrospinal fluid drainage and left heart bypass, having chronic dissection has become beneficial from a risk standpoint in Extent II repairs. Not so with the Extent IIIs and Extent IVs, which over the years have evolved into a group of patients with more atherosclerosis and less dissection, older age, more comorbidities, particularly pulmonary and renal preoperatively, and a heavy layer of superimposed atherosclerosis. This group has actually become more challenging.

We have operated in approximately 50 patients with deep HCA. In our own experience with those cases

being patients in whom it wasn't the primary strategy but one we thought necessary because cross-clamping was not possible secondary to anatomy and other complicating factors. It is very likely that in our cohort of patients with deep HCA, the results are similar to the overall group, if you adjust for the inherent risks present in this highly select cohort of patients with many complicating factors.

Dr Kouchoukos. Do you think we are at the irreducible minimum? Are we ever going to further reduce the risk of paraplegia?

Dr Coselli. Although there remains potential to further reduce the risk of postoperative paraplegia in the future, I think we are a long way off from bringing it to zero.

Dr Kouchoukos. Finally and perhaps most important, how do you see the role of open thoracoabdominal aneurysm repair in the next 15 to 30 years? Will endovascular repair supplant open repair as the preferred method of management or will open repair still have a role?

Dr Coselli. I think open repair is going to be around for some time. There remains a concern with some of the engineering and technologic issues present in purely endovascular repair of the thoracoabdominal aneurysm, especially for extent II repairs. Because of this, I think this supplantation is still in the future. However, a lot of work is being done by a number of institutions and endovascular companies to do just that. On the open side of TAAA repair, we have developed a new indication for operation, and that is the failed endovascular repair; somewhat unpredictably, we have seen rapid expansion of the aneurysm and rupture of patients with endovascular stent-grafts in place, and they have to come back and be rescued in an open fashion.

Dr L. Svensson *(Cleveland, Ohio)*. I congratulate you, Joe, on an incredible series, and thus you have shown that you have taken over the mantle of both Dr DeBakey and Dr Crawford, and, as Dr Kouchoukos said, I don't think this will ever be equaled again. By my back of the envelope calculation, the time you spent on this series is some 15,000 hours.

The challenging group is obviously the type IIs, and that's where we all see the complications. I was wondering, apart from the role of stenting in the type Is and type IVs, what do you think we should be doing for the IIs? Maybe cooling the patients more, being more aggressive in prescreening them for the intercostal or lumbar arteries?

Dr Coselli. Basically, I agree entirely with everything you have said. There is some potential for intercostal and anterior spinal artery identification with emerging technology and software that may provide substantial advantages regarding this identification in a few years. Although for now, purely eTAAA repair is clearly directed at the low-hanging fruit, such as the extent IV repairs and probably the extent I repairs. However, we are going to have to be very careful in choosing a method of repair for the extent II category, particularly for younger patients and those with connective tissue disorders or chronic dissection, who generally do well with open repair.