Outcomes of open repairs of chronic distal aortic dissection anatomically amenable to endovascular repairs

Akiko Tanaka, MD, PhD, Harleen K. Sandhu, MD, MPH, Rana O. Afifi, MD, Charles C. Miller III, PhD, Amberly Ray, BS, Madiha Hassan, MD, Hazim J. Safi, MD, FACS, and Anthony L. Estrera, MD, FACS

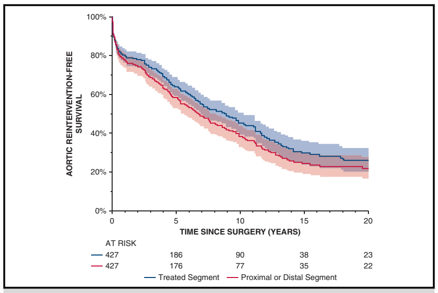
ABSTRACT

Objective: To review short-term outcomes and long-term survival and durability after open surgical repairs for chronic distal aortic dissections in patients whose anatomy was amenable to thoracic endovascular aortic repair (TEVAR).

Methods: Between February 1991 and August 2017, we repaired chronic distal dissections in 697 patients. Of those patients, we enrolled 427 with anatomy amenable to TEVAR, which included 314 descending thoracic aortic aneurysms (DTAAs) and 105 extent I thoracoabdominal aortic aneurysms (TAAAs). One hundred eighty-five patients (44%) had a history of type A dissection, and 33 (7.9%) had a previous DTAA/TAAA repair. Variables were assessed with logistic regression for 30-day mortality and Cox regression for long-term mortality. Time-to-event analysis was performed using Kaplan-Meier methods.

Results: Thirty-day mortality was 8.4% (n = 36). In all, 22 patients (5.2%) developed motor deficit (paraplegia/paraparesis), and 17 (4.0%) experienced stroke. Multivariable analysis identified low estimated glomerular filtration rate (eGFR; <60 mL/min/1.73 m^2), previous DTAA/TAAA repair, and chronic obstructive pulmonary disease (COPD) as associated with 30-day mortality. Patients without all 3 risk factors had a 30-day mortality rate of 2.6%. During a median follow-up of 6.5 years, 160 patients died. The survival rate was 81% at 1 year and 61% at 10 years. Cox regression analysis identified preoperative aortic rupture, eGFR <60 mL/min/1.73 m^2, previous DTAA/TAAA repair, COPD, and age >60 years as predictive of long-term mortality. Forty-five patients required subsequent aortic procedures, including 8 reinterventions to the treated segment. Freedom from any aortic procedures was 85% at 10 years, and aortic procedure-free survival was 45% at 10 years. Hereditary aortic disease was the sole predictor for any aortic interventions (hazard ratio, 3.2; $P = .004$).

Conclusions: Open surgical repair provided satisfactory low neurologic complication rates and durable repairs in chronic distal aortic dissection. Patients without low eGFR, redo, and COPD are the low-risk surgical candidates and may benefit from open surgical repair at centers with similar experience to ours. Patients with hereditary aortic disease warrant close surveillance. (J Thorac Cardiovasc Surg 2021;161:36-43)



Aortic reintervention-free survival after open repair in chronic distal aortic dissection.

Central Message

Open surgery provides satisfactory outcomes, with excellent durability in chronic distal dissection. It should be considered a mainstay of treatment, especially in patients without renal or lung disease and redo.

Perspective

Until the long-term durability of endovascular repair for chronic distal dissection is proven, open surgery may benefit low-risk surgical candidates at centers with similar experience to ours.

See Commentaries on pages 44 and 46.

Chronic distal aortic dissections have historically been treated by open surgical repairs, but a recent study among the Medicare population demonstrated a steady increase in the application of thoracic endovascular aortic repair

Scanning this QR code will take you to the article title page to access supplementary information.

From the Department of Cardiothoracic and Vascular Surgery, McGovern Medical School at The University of Texas Health Science Center at Houston and Memorial Hermann Hospital, Houston, Tex.

Read at the American Association for Thoracic Surgery Aortic Symposium 2018, New York, New York, April 26-27, 2018.

Received for publication Sept 4, 2018; revisions received Sept 12, 2019; accepted for publication Sept 15, 2019; available ahead of print Oct 1, 2019.

Address for reprints: Anthony L. Estrera, MD, FACS, Department of Cardiothoracic and Vascular Surgery, McGovern Medical School at UTHealth, 6400 Fannin St, Suite 2850, Houston, TX 77030 (E-mail: Anthony.L.Estrera@uth.tmc.edu).

0022-5223/$36.00

https://doi.org/10.1016/j.jtcvs.2019.09.083

Abbreviations and Acronyms

COPD	= chronic obstructive pulmonary disease
CSF	= cerebrospinal fluid
DTAA	= descending thoracic aortic aneurysm
eGFR	= estimated glomerular filtration rate
TAAA	= thoracoabdominal aortic aneurysm
TEVAR	= thoracic endovascular aortic repair

(TEVAR).[1] Comparisons between open surgical repairs and TEVAR for chronic distal aortic dissections have not been well investigated, and there is a paucity of data on either of these treatment methods.[2] There are currently fewer than 10 studies on both open surgical repair and TEVAR for chronic distal aortic dissections with more than 50 patients enrolled.[3-16] In recent years, an increasing number of studies on TEVAR for chronic distal aortic dissection have been published, and studies on open surgical repairs have been reporting "superior freedom from reintervention." However, follow-up data are scanty for both treatment methods; most TEVAR follow-up series have a 2- to 3-year follow-up, and most open repair series also have a limited follow-up of 2 to 4 years.[3-16] The purpose of the present study was to evaluate contemporary short-term outcomes, long-term survival, and durability after open surgical repair for chronic distal aortic dissections, focusing on patients who were anatomically amenable to endovascular aortic repair.

PATIENTS AND METHODS

The Committee for Protection of Human Subjects, the local Institutional Review Board, approved this study. Between February 1, 1991, and August 31, 2017, we repaired 2033 distal aortic (descending thoracic/thoracoabdominal) aneurysms, including 697 chronic dissections. In this article, we use the term "distal aortic dissection" instead of "type B dissection," because we included patients with both de novo Stanford type B aortic dissection and residual dissection in the descending thoracic aorta after Stanford type A aortic dissection repair. Of 697 open chronic distal aortic dissection repairs, our prospective database captured 427 patients who would have been anatomically amenable to TEVAR to the present study. This included descending thoracic aortic repair of any extents and extent I thoracoabdominal aortic repairs. Aortic diameter exceeding 40 mm at the proximal descending or at the level of visceral arteries were excluded. Patients who required circulatory arrest for proximal anastomosis were also excluded, because it implies proximal extension of the disease to the aortic arch, which would require nonstandard endovascular techniques for repair. Extent I thoracoabdominal aortic aneurysm (TAAA) is included in the study, because celiac artery coverage is now more commonly indicated for distal chronic dissections (Figure E1). Indications for surgical interventions included aortic diameter >50 mm, rapid growth of the aorta (>5 mm/year), and fistulization (bronchial or esophageal). Patient perioperative demographics and follow-up data were collected from record reviews by a trained nurse and physician evaluators and were entered in a dedicated department database. Analyses were retrospective. Patient follow-up was obtained by direct patient contact in clinic, telephone interview, or the National Death Index. The last date of follow-up was the last clinic visit, last imaging date, or last day of phone interview. Patients were asked about outside clinic visits, recent imaging, delayed complications, and secondary procedures. Data were managed under Health Insurance Portability and Accountability Act confidentiality guidelines in a database with encrypted patient identifiers.

Motor deficits included both paraplegia and paraparesis derived from the spinal cord ischemia. Any paraplegia/paraparesis observed on awakening from anesthesia was reported as immediate; any that developed afterward (minutes, hours, or days) was reported as delayed. The estimated glomerular filtration rate (eGFR) was calculated by the Cockcroft-Gault method.

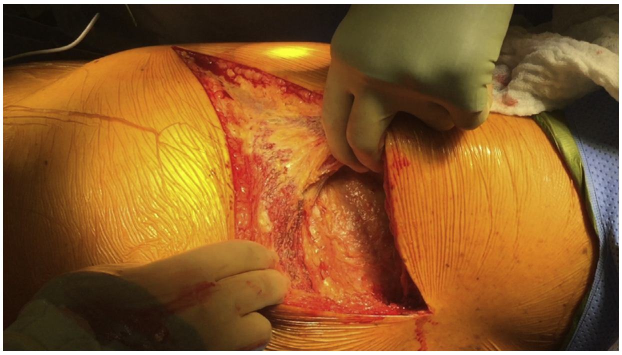
Surgical Technique

Our operative technique for open surgical repair of the descending and thoracoabdominal aortic pathologies have been reported previously.[17] Over this 27-year study period, we used the same adjunctive spinal cord protection techniques: distal aortic perfusion, cerebral spinal fluid drainage (CSF) drainage, and moderate hypothermia (Video 1). We used the technique for all elective cases; CSF drainage was not used for cases concerning for infectious etiology.

After induction of general anesthesia, the patient is intubated with a double-lumen endotracheal tube and placed in the right lateral decubitus position. A CSF catheter is placed to maintain the CSF pressure below 10 mm Hg throughout the perioperative period. Somatosensory potentials, motor-evoked potentials, and electroencephalography are monitored. Transcranial Doppler was also used in the early era, but this was switched to regional oxygen saturation in the later era. After exposure of the descending thoracic/thoracoabdominal aorta, sodium heparin is given, and the left heart bypass is established with left inferior pulmonary vein drainage and arterial return via the left femoral artery. Distal aortic perfusion begins as the cross-clamp is applied and mean arterial pressure is maintained to approximately 60 to 80 mm Hg. Core temperature is targeted at 33°C (permissive mild hypothermia). Graft replacement is performed under sequential clamping. Patent lower intercostal arteries (ie, T8 to T12) are reimplanted under neuromonitoring guidance while upper intercostal arteries are ligated.[18] The dissection flap extending beyond the distal anastomosis is fenestrated to allow blood flow in both the true and false lumens. For TAAA, the retroperitoneal space is entered, and the muscular portion of the diaphragm is cut to expose the aorta. The celiac axis and superior mesenteric artery are perfused with tepid blood, and the kidneys are cooled with the crystalloid solution. The celiac axis and superior mesenteric artery are reattached to the Dacron graft as an island patch or incorporated in the beveled distal aortic anastomosis. In cases of hereditary aortic disease or a widely separated celiac axis and superior mesenteric artery, a Carrel button patch is created for each branch and reconstructed individually to the side arm of the graft. After completion of the proximal and distal anastomosis, the patient is warmed. The cannulae are then removed, and protamine is administered.

Postoperative Management

CSF is drained to maintain a pressure <10 mm Hg, with a maximum drainage rate of 15 mL/h if the neurologic status is intact. The CSF drain is clamped on the second postoperative day and removed the next day. The blood pressure goal in the early era was a mean blood pressure >60 mm Hg and systolic blood pressure >130 mm Hg in the late era of the study.[19] If paraplegia or paraparesis is observed, we immediately initiate the modified COPS protocol (CSF drain status, oxygen delivery, patient status). At this point, we drain the CSF without a limit to achieve CSF pressure <5 mm Hg, transfuse blood to maintain hemoglobin at >10 g/dL, and add catecholamine to maintain the cardiac index at >2.5 L/min/m^2 and mean blood pressure >80 mm Hg (or systolic blood pressure >140 mm Hg). We may continue the modified COPS protocol until the seventh postoperative day. If the origin of the motor deficits—spinal cord or brain, such as monoplegia—was unclear, we obtain computed tomography scans



VIDEO 1. A representative surgical repair of chronic type B aortic dissection. In this video, in addition to our routine repair techniques, distal first technique and reversed elephant trunk technique are demonstrated. In addition, preparations for the intercostal artery reattachment is summarized: creating an additional side branch to the Dacron graft, preserving the intercostal arteries using a Fogarty balloons, and assessing for spinal cord ischemia with neuromonitoring. The patient is a 63-year-old male who had type B aortic dissection 9 years ago, which required femoral-to-femoral bypass for limb ischemia. He now presented with a 6-cm descending thoracic aortic aneurysm. We used a thoracoabdominal incision, cut the costal margin, and entered the sixth intercostal space to approach the aorta. After the exposing the aorta, we prepared the graft by creating an additional side branch to the body and invaginating the proximal end for a reversed elephant trunk. We usually cannulate the left femoral artery for the arterial return of the left heart bypass; however, due to the presence of a femoro–femoral bypass, we used the distal first technique in this case. We performed distal anastomosis in end-to-end fashion with a 3-0 polypropylene sutures after a fenestration of the distal dissection flap, to allow blood flow to both the true and false lumens. We then established left heart bypass to maintain distal aortic perfusion by draining blood from the left inferior pulmonary vein and perfusing through the side branch of the graft. The proximal descending aorta and the Dacron graft just proximal to the side arm were clamped. We then longitudinally opened the false lumen of the descending and excise the dissection flap. The patent intercostal arteries from Th8 to Th12 were preserved with 3 Fr Fogarty balloons. The proximal anastomosis was performed in end-to end fashion with 3-0 Prolene, and left heart bypass was weaned off. Although in this case we did not reconstruct intercostal artery, because motor-evoked and somatosensory-evoked potentials remained intact throughout the case, our recent study demonstrated that it is important to reattach Th8 to Th12 to prevent delayed paraplegia, we now reconstruct the intercostal arteries regardless of the intraoperative findings. The patient had an uneventful postoperative course and was discharged home on postoperative day 9. Video available at: https://www.jtcvs.org/article/S0022-5223(19)32094-X/fulltext.

and/or magnetic resonance imaging of the brain and/or spinal magnetic resonance imaging for diagnosis.

Follow-up Completeness

Follow-up was completed on February 8, 2019. The median duration of follow-up was 6.5 years (interquartile range, 2.2-11.9 years). Of 382 patients who were discharged alive, 19 were international patients, 8 of whom were lost to follow-up after discharge. Of the remaining domestic patients, 160 patients died during follow-up. Of those surviving domestic patients, 14 were lost to follow-up after discharge, all from the early 1990s. The 30-day follow-up rate was 100%; our rate, with at least 90 days of follow-up, was 94.2%. Approximately one-half of the patients had a follow-up computed tomography scan available after discharge.

TABLE 1. Preoperative and intraoperative patient demographic data (N = 427)

Characteristic	Value
Preoperative demographics	
Age, y, mean (IQR)	61 (52-70)
Age >60 y, n (%)	229 (53.6)
Female sex, n (%)	116 (27.2)
Hypertension, n (%)	385 (90.2)
Diabetes mellitus (N = 425), n (%)	48 (11.3)
COPD, n (%)	94 (22.0)
Peripheral vascular disease, n (%)	49 (11.5)
Coronary artery disease, n (%)	88 (20.6)
Cerebrovascular disease, n (%)	61 (14.3)
Chronic kidney disease staging (N = 423), n (%)	
Stage 1 (eGFR ≥90 mL/min/1.73 m^2)	186 (43.6)
Stage 2 (eGFR 60-89 mL/min/1.73 m^2)	114 (27.0)
Stage 3a (eGFR 45-59 mL/min/1.73 m^2)	60 (14.2)
Stage 3b (eGFR 30-44 mL/min/1.73 m^2)	31 (7.3)
Stage 4 (eGFR 15-30 mL/min/1.73 m^2)	17 (4.0)
Stage 5 (eGFR <15 mL/min/1.73 m^2)	15 (3.5)
Hereditary aortic disease, n (%)	81 (19.0)
Previous aortic procedures, n (%)	
Type A aortic dissection repair	185 (43.3)
Stage 1 elephant trunk	89 (20.8)
DTAA repair	20 (4.7)
AAA repair	27 (6.3)
TAAA repair	17 (4.0)
History of CABG/PCI, n (%)	9 (2.1)
Rupture, n (%)	20 (4.7)
Infection, n (%)	3 (0.7)
Intraoperative demographics	
Extent of aneurysm repair, n (%)	
DTAA-extent A	95 (22.2)
DTAA-extent B	8 (1.9)
DTAA-extent C	216 (50.6)
TAAA-extent I	108 (25.3)
Distal aortic perfusion with CSF drain, n (%)	367 (87.6)
Cells saved, unit (N = 420), median (IQR)	21 (3-34)
Packed red blood cells, unit (N = 426), median (IQR)	3 (2-6)
Fresh frozen plasma, unit (N = 425), median (IQR)	4 (0-8)
Platelets, units (N = 424), median (IQR)	6 (0-12)
Aortic clamp time, min (N = 426), median (IQR)	36 (30-45)
Bypass time, min (N = 416), median (IQR)	42 (32-54)
DHCA use, n (%)	18 (4.2)
Intercostal arteries, reattached, n (%)	145 (34.6)

Extent A indicates left subclavian artery to T6; extent B, T6 to T12; extent C, left subclavian artery to T12. *IQR*, Interquartile range; *COPD*, chronic obstructive pulmonary disease; *eGFR*, estimated glomerular filtration rate; *DTAA*, descending thoracic aortic aneurysm; *AAA*, abdominal aortic aneurysm; *TAAA*, thoracoabdominal aortic aneurysm; *CABG*, coronary artery bypass grafting; *PCI*, percutaneous coronary intervention; *CSF*, cerebrospinal fluid; *DHCA*, deep hypothermic circulatory arrest.

Statistical Methods

Continuous data are reported as mean ± standard deviation for normally distributed data or median plus interquartile range for skewed data.

TABLE 2. Postoperative outcomes (N = 427)

Outcome	Value, n (%)
Tracheostomy, n (%)	33 (7.7)
Prolonged ventilation (>48 h), n (%)	148 (34.7)
Newly required dialysis, n (%)	36 (8.4)
Temporary	21 (4.9)
Dialysis on discharge	15 (3.5)
Re-exploration for bleeding, n (%)	12 (2.8)
Stroke, n (%)	17 (4.0)
TIA, n (%)	2 (0.5)
Motor deficit (paraplegia/paraparesis), n (%)	22 (5.2)
Immediate	5 (1.2)
Delayed	17 (4.0)
Present at discharge	11 (2.6)
Length of stay, d, median (IQR)	12 (9-18)
30-d mortality, n (%)	36 (8.4)
Follow-up, y, median (IQR)	6.5 (2.2-11.9)
Actuarial survival, %, mean ± SD	
At 5 y (N = 188)	64.5 ± 2.5
At 10 y (N = 90)	45.2 ± 3.0
Aortic reintervention, n (%)	53 (13.9)*
Treated segment	8 (2.1)*
Proximal segment	11† (2.9)*
Distal segment	34† (7.1)*

TIA, Transient ischemic attack; *IQR*, interquartile range; *SD*, standard deviation. *Percentage was calculated using 382 as a denominator (patients who were discharged alive). †One patient had both proximal and distal aortic reinterventions.

Categorical variables are expressed as number plus percentage. Comparisons between continuous variables were performed using the Student t test for parametric data or the Wilcoxon rank-sum test for nonparametric data. Categorical variables were compared using a the χ^2 or Fisher exact test. For all analyses, 2-sided tests were used. Multivariable assessment of 30-day mortality risk factors (Table E1) was conducted using multiple logistic regression analysis. Multivariable risk factor assessments for long-term survival (Table E2) were conducted by multiple proportional hazards regression. Model selection for all multivariable analyses was conducted by forward stepwise model selection followed by purposeful selection. For the variable of renal function, an eGFR of 60 mL/min/1.73 m^2 was chosen as a cutoff, because this represents chronic kidney dysfunction stage 3 to 5. The age cutoff of 60 years was selected, because that median age of our study population was 61 years and it also provided a larger effect compared with a cutoff of 70 years. Dichotomized values for age and eGFR were used in the multivariable analysis to provide information easier to be interpreted. Model fit was assessed for logistic regression models using the Hosmer-Lemeshow method and Akaike information criterion minimization. For the Cox models, assumptions were tested using time-varying covariate tests in SAS PROC PHREG. Separate long-term probabilities of redo operation and progression of disease into previously uninvolved aorta were estimated using univariable Kaplan-Meier analysis, which is reported as freedom from reintervention and reintervention-free survival (ie, death as a competing risk). All data were analyzed using SAS version 9.4 (SAS Institute, Cary, NC).

TABLE 3. Multivariable analysis of preoperative predictors for 30-day mortality

Variable	OR (95% CI)	*P* value
Low eGFR	4.1 (2.1-8.2)	<.001
History of DTAA/TAAA repair	4.1 (1.7-10.2)	.002
COPD	2.4 (1.2-4.8)	.016

Low eGFR indicates eGFR <60 mL/min/1.73 m^2. *OR*, Odds ratio; *CI*, confidence interval; *eGFR*, estimated glomerular filtration rate; *DTAA*, descending thoracic aortic aneurysm; *TAAA*, thoracoabdominal aortic aneurysm; *COPD*, chronic obstructive pulmonary disease.

TABLE 4. Cox regression analysis of preoperative predictors for long-term mortality

Variable	HR (95% CI)	*P* value
History of D/TAAA repair	3.3 (1.7-6.6)	<.001
Age >60 y	2.3 (1.5-3.2)	<.001
Preoperative aortic rupture	2.2 (1.1-4.2)	.019
eGFR <60 mL/min/1.73 m^2	1.9 (1.4-2.7)	<.001
COPD	1.6 (1.1-2.3)	.007

HR, Hazard ratio; *CI*, confidence interval; *DTAA*, descending thoracic aortic aneurysm; *TAAA*, thoracoabdominal aortic aneurysm; *eGFR*, estimated glomerular filtration rate; *COPD*, chronic obstructive pulmonary disease.

RESULTS

Short-Term Outcomes

Preoperative and intraoperative data are summarized in Table 1. Of 427 patients with chronic type B dissection, 185 (43%) had a history of type A aortic dissection, and nearly one-half of these patients (n = 89) had undergone previous transverse aortic arch replacement. Thirty-seven (8.7%) had undergone previous descending thoracic aortic aneurysm (DTAA) or TAAA repair. Postoperative outcomes are summarized in Table 2. Thirty-day mortality was 8.4% (n = 36). Twenty-two patients (5.2%; 5 with immediate onset and 17 with delayed onset) developed motor deficit (paraplegia/paraparesis), but 11 patients had resolution with the COPS protocol, leaving 11 (2.6%) symptomatic patients at the time of discharge. Although we switched from transcranial Doppler to regional cerebral oxygenation monitoring and the target pressure target from the mean systolic blood pressure, the rates of motor deficit and 30-day mortality were similar in the early (1991-2000) and late (2001-2017) eras (motor deficit: 4.5% vs 5.4%, $P = .720$; mortality: 7.2% vs 8.9%, $P = .860$). Multivariable analysis identified low eGFR (<60 mL/min/1.73 m^2), history of DTAA/TAAA repair, chronic obstructive pulmonary disease (COPD) as preoperative predictors of 30-day mortality (Table 3). The 30-day mortality in patients with eGFR ≥60 mL/min/1.73 m^2, without COPD, and the rate of nonredo (ie, low-risk patient) was 2.6%.

Long-Term Survival

The survival rate was 67.9% at 5 years, 53.1% at 10 years, and 41.5% at 15 years. Cox regression analysis identified preoperative aortic rupture, low eGFR

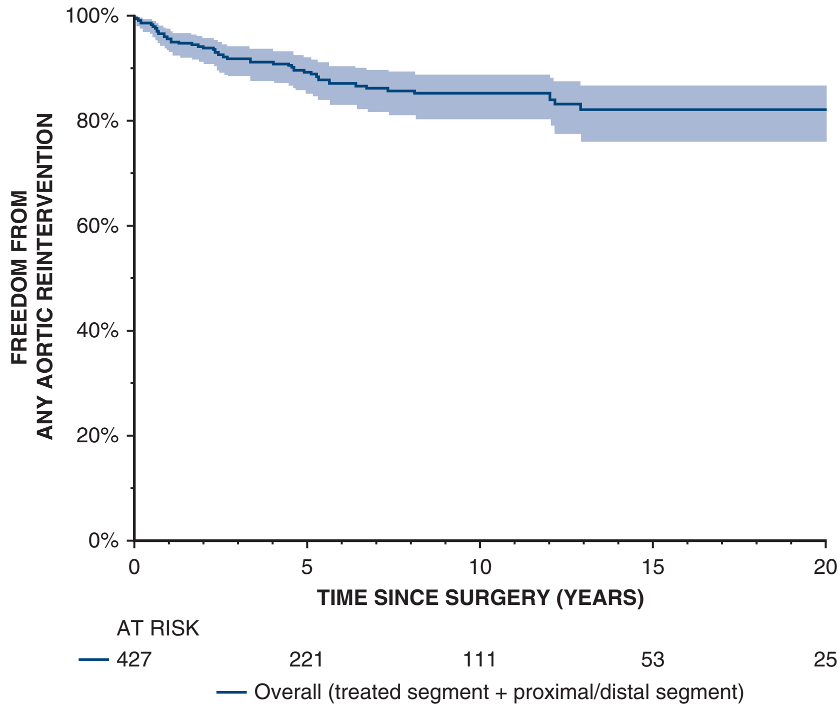


FIGURE 1. Kaplan-Meier curve demonstrating freedom from any aortic reintervention: 88% at 5 years and 85% at 10 years.

(<60 mL/min/1.73 m^2), history of DTAA/TAAA repair, COPD, and age >60 years as preoperative predictors for long-term mortality (Table 4, Figures E2-E6).

Long-Term Durability

Of 382 patients who were discharged alive, 53 (13.9%) required subsequent aortic procedures, including 8 (2.1%) with reinterventions to the treated segment (2 graft infections, 2 intercostal artery patch aneurysms, 4 pseudoaneurysms), and 45 (11.8%) with aortic repairs, due to progression of aneurysm (11 proximal and 34 distal). Freedom from any aortic procedures was 88% at 5 years and 85% at 10 years (Figure 1). Freedom from reintervention to the treated segment was 98% at 5 years and 97% at 10 years, and aortic reintervention–free survival was 45% at 10 years. Aortic reintervention–free survival by reintervention type, treated segment (ie, treatment failure) versus proximal/distal segment (ie, progression of aortic disease) is shown in Figure 2. Hereditary aortic disease was the sole significant predictor for subsequent aortic interventions (HR, 3.2; $P = .004$).

DISCUSSION

Controversy in Treating Chronic Dissection With Open Repair Versus TEVAR

With increased experience and favorable results in acute distal aortic dissection, TEVAR has gained popularity for chronic distal aortic dissection.[1] An expert multidisciplinary consensus panel also proposed the application of TEVAR to the chronic distal dissection as the first-line therapy if TEVAR is not contraindicated; however, this statement was based on a relatively small series.[2] Thus, in the present study, we focused on outcomes after open surgery for chronic distal dissections with anatomy amenable to TEVAR to provide supporting data for the comparison of these 2 treatment methods. We did not exclude hereditary aortic disease from the cohort, because this patient population is a potential target population for TEVAR in chronic distal dissection.[20] In most expert centers that perform TEVAR for chronic distal dissection repair with pavement of stent graft to the celiac axis, we have included not only DTAA, but also extent I TAAA.

Short-Term Outcomes

Open surgical repair of acute aortic dissection is known to carry high mortality, owing to the fragility of the aortic tissue. TEVAR is the treatment of choice for complicated acute type B aortic dissection. Deployment of a stent graft at the entry tear re-expands the true lumen, induces the false lumen thrombosis, and allows reestablishment of the flow to the ischemic end-organ with malperfusion.

This positive aortic remodeling process does not always apply to chronic aortic dissection, however. The intimal flap in the chronic phase is rigid and does not allow easy expansion of the true lumen. Not all the aortic branches take off from the true lumen, and thrombosis of the false

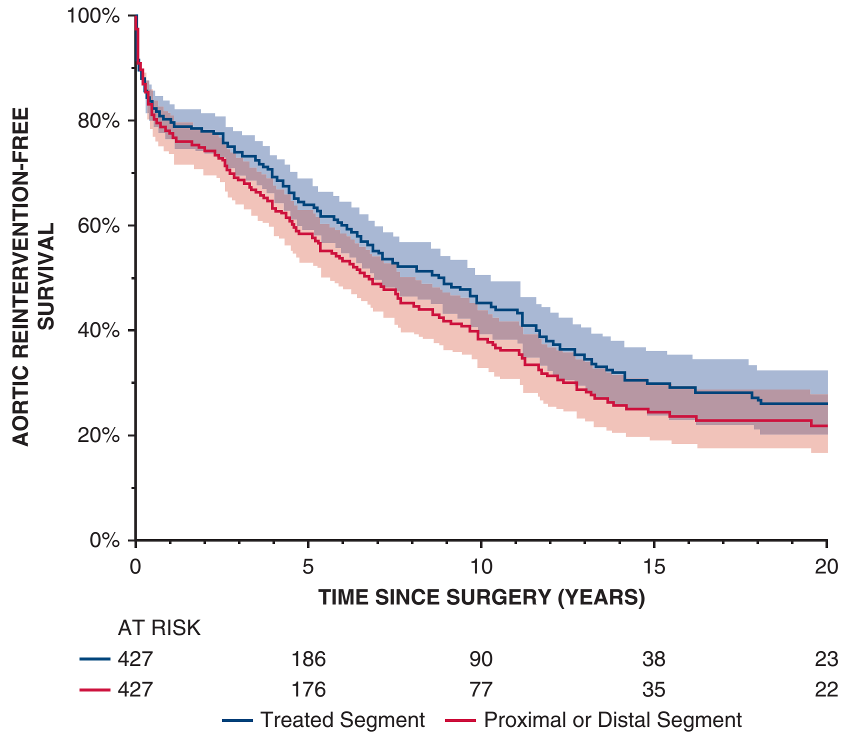


FIGURE 2. Kaplan-Meier curve demonstrating aortic reintervention-free survival after open repair in chronic distal aortic dissection by reintervention type. Deaths and aortic reinterventions are censored. The treated segment reintervention–free survival was 63% at 5 years and 44% at 10 years. The proximal/distal segment reintervention–free survival was 58% at 5 years and 38% at 10 years.

channel may cause end-organ ischemia. Moreover, a simple "entry" closure does not result in false lumen depressuring or thrombosis, because there is usually more than 1 communication between the true and false lumens (ie, multiple reentry tears). Recent reports have demonstrated that nearly 70% of patients who underwent TEVAR for chronic distal aortic dissection failed to regress the aortic diameter.[21,22] Patients with large aortic diameter and fewer visceral branches off the true lumen were negative predictors of false lumen thrombosis in chronic distal dissections after TEVAR.[23,24] In the chronic phase, the aortic wall thickens and strengthens and becomes amenable to holding sutures. Our results are comparable to those reported in other relatively large series of open surgical repairs; 30-day mortality was 9.8%, and the rate of permanent motor deficit was 2.6%. Nonetheless, previous series demonstrated superior 30-day mortality with TEVAR compared with open surgical repair (TEVAR, 0%-5%; open, 1%-10%[3-16]), because TEVAR is less invasive. We found low eGFR, redo, and COPD were the independent predictors of mortality after open repairs for DTAA and extent I TAAA with chronic distal dissection. In contrast, patients without these 3 factors are good candidates for open repair, with a mortality of only 2.6%. Motor deficits and stroke rates after repair are equivalent in the 2 treatment methods (permanent motor deficits: TEVAR, 0%-6%; open, 0%-6%; stroke: TEVAR, 0%-8%; open, 1%-6%).[3-16]

Long-Term Survival

Our survival rate was 67.9% at 5 years. Previous studies of open repair for chronic distal aortic dissection reported rates ranging from 55% to 83%,[8,10-13] equivalent to those for TEVAR (57%-84%[3-7]). Studies with younger populations in both open repair and TEVAR had higher survival rates, as expected. The advantage of lower mortality with TEVAR may be lost in the long-term. However, it is unknown if patient selection affected the outcomes as older age, low eGFR, COPD, redo, and rupture identified as independent predictors of long-term in the current study is often seen in the TEVAR population. A registry to compare the 2 treatment arms may be warranted to reach a conclusion.

Long-Term Durability

We found excellent durability of open surgical repair for chronic distal aortic dissection. The treated segment reintervention rate was 3% at 10 years, and the rate of any aortic intervention was 15% at 10 years. These findings are supported by other studies demonstrating superior long-term durability with open surgery compared with

TEVAR.[3-16] The reintervention rate for treated segments is 0% to 8% in open surgery and 5% to 16% in TEVAR, and the reintervention rate for any aortic segment is 0% to 13% in open surgery and 5% to 35% in TEVAR. Our study has the longest follow-up after open repair for chronic distal aortic dissection reported in the English literature. Our median follow-up was 6.5 years, compared with the 1.7 to 5.0 years in other large open repair series.[10-16] Our data also show a median time to the next aortic procedure of 2.4 years (interquartile range, 0.8-5.1 years). In other words, our results truly demonstrate the reintervention rate, whereas other reports may have underestimated the reintervention rate, owing to a limited follow-up period. This underestimation may also apply to TEVAR, for which major studies have reported a median follow-up ranging from 2.0 to 4.1 years (predominately 2 to 3 years).[3-9] Nonetheless, there is little doubt about the superior long-term durability of open surgical repair compared with TEVAR in patients with chronic dissection.

Of note, the incidence of hereditary aortic disease, the predictor of reintervention in our study, was <10% in all these TEVAR series,[3-9] whereas that of most open surgery series was >10% (range, 8% to 56%).[10-16] Thus, further study is required before encouraging the use of TEVAR in patients with hereditary aortic disease.

Limitations

This study has several limitations. First, it is a single-center, retrospective study, in which the patient selection may have been biased. Second, owing to the limited number of patients who underwent TEVAR at our institution during the study period, we focused our analysis on patients who underwent open repair instead of performing a direct comparison. Third, this is an institutional experience from a team dedicated to aortic surgery, and thus our results might not be reflective of the real world.

CONCLUSIONS

Open surgical repair provided satisfactory low neurologic complication rates and durable repairs in chronic distal aortic dissection. Patients without low eGFR, redo, and COPD are the low-risk surgical candidates and may benefit from open surgical repair at centers with similar experience to ours. Patients with hereditary aortic disease warrant close surveillance.

Conflict of Interest Statement

Drs Estrera and Sandhu serve as consultants for W. L. Gore. All other authors have nothing to disclose with regard to commercial support.

We thank Troy Brown for editing, Chris Akers for illustrations, and Samuel Leonard for assistance with patient follow-up.

References

1. Jones DW, Goodney PP, Nolan BW, Brooke BS, Fillinger MF, Powell RJ, et al. National trends in utilization, mortality, and survival after repair of type B aortic dissection in the medicare population. *J Vasc Surg*. 2014;60:11-9.e1.
2. Fattori R, Cao P, De Rango P, Czerny M, Evangelista A, Nienaber C, et al. Interdisciplinary expert consensus document on management of type B aortic dissection. *J Am Coll Cardiol*. 2013;61:1661-78.
3. Xu SD, Huang FJ, Yang JF, Li ZZ, Yang S, Du JH, et al. Early and midterm results of thoracic endovascular aortic repair of chronic type B aortic dissection. *J Thorac Cardiovasc Surg*. 2010;139:1548-53.
4. Mani K, Clough RE, Lyons OT, Bell RE, Carrell TW, Zayed HA, et al. Predictors of outcome after endovascular repair for chronic type B dissection. *Eur J Vasc Endovasc Surg*. 2012;43:386-91.
5. Lee M, Lee DY, Kim MD, Lee MS, Won JY, Park SI, et al. Outcomes of endovascular management for complicated chronic type B aortic dissection: effect of the extent of stent graft coverage and anatomic properties of aortic dissection. *J Vasc Interv Radiol*. 2013;24:1451-60.
6. Scali ST, Feezor RJ, Chang CK, Stone DH, Hess PJ, Martin TD, et al. Efficacy of thoracic endovascular stent repair for chronic type B aortic dissection with aneurysmal degeneration. *J Vasc Surg*. 2013;58:10-7.e1.
7. Patterson B, Holt P, Nienaber C, Cambria R, Fairman R, Thompson M. Aortic pathology determines midterm outcome after endovascular repair of the thoracic aorta: report from the medtronic thoracic endovascular registry (MOTHER) database. *Circulation*. 2013;127:24-32.
8. van Bogerijen GH, Patel HJ, Williams DM, Yang B, Dasika NL, Eliason JL, et al. Propensity-adjusted analysis of open and endovascular thoracic aortic repair for chronic type B dissection: a twenty-year evaluation. *Ann Thorac Surg*. 2015;99:1260-6.
9. Kim TH, Song SW, Lee KH, Baek MY, Yoo KJ, Cho BK. The effect of false lumen procedures during thoracic endovascular aortic repair in patients with chronic DeBakey type IIIB dissections. *J Vasc Surg*. 2018;68:976-84.
10. Zoli S, Etz CD, Roder F, Mueller CS, Brenner RM, Bodian CA, et al. Long-term survival after open repair of chronic distal aortic dissection. *Ann Thorac Surg*. 2010;89:1458-66.
11. Pujara AC, Roselli EE, Hernandez AV, Vargas Abello LM, Burke JM, Svensson LG, et al. Open repair of chronic distal aortic dissection in the endovascular era: implications for disease management. *J Thorac Cardiovasc Surg*. 2012;144:866-73.
12. Coselli JS, Green SY, Zarda S, Nalty CC, Price MD, Hughes MS, et al. Outcomes of open distal aortic aneurysm repair in patients with chronic DeBakey type I dissection. *J Thorac Cardiovasc Surg*. 2014;148:2986-93.e1-2.
13. Conway AM, Sadek M, Lugo J, Pillai JB, Pellet Y, Panagopoulos G, et al. Outcomes of open surgical repair for chronic type B aortic dissections. *J Vasc Surg*. 2014;59:1217-23.
14. Hata M, Orime Y, Wakui S, Nakamura T, Akiyama K, Shiono M. Outcomes of open surgical repair for type B dissecting aortic aneurysm with alternative methods in the endovascular stent era. *Semin Thorac Cardiovasc Surg*. 2015;27:106-12.
15. Fujikawa T, Yamamoto S, Sekine Y, Oshima S, Kasai R, Mochida Y, et al. Operative results and clinical features of chronic stanford type B aortic dissection: examination of 234 patients over 6 years. *Eur J Vasc Endovasc Surg*. 2015;50:738-43.
16. Kouchoukos NT, Kulik A, Castner CF. Open thoracoabdominal aortic repair for chronic type B dissection. *J Thorac Cardiovasc Surg*. 2015;149(2 Suppl):S125-9.
17. Estrera AL, Sandhu HK, Charlton-Ouw KM, Afifi RO, Azizzadeh A, Miller CC III, et al. A quarter century of organ protection in open thoracoabdominal repair. *Ann Surg*. 2015;262:660-8.
18. Estrera AL, Sheinbaum R, Miller CC III, Harrison R, Safi HJ. Neuromonitor-guided repair of thoracoabdominal aortic aneurysms. *J Thorac Cardiovasc Surg*. 2010;140(6 Suppl):S131-5; discussion S142-6.
19. Sandhu HK, Evans JD, Tanaka A, Atay S, Afifi RO, Charlton-Ouw KM, et al. Fluctuations in spinal cord perfusion pressure: a harbinger of delayed paraplegia after thoracoabdominal aortic repair. *Semin Thorac Cardiovasc Surg*. 2017;29:451-9.

20. Shalhub S, Eagle KA, Asch FM, LeMaire SA, Milewicz DM, GenTAC Investigators for the Genetically Triggered Thoracic Aortic Aneurysms and Cardiovascular Conditions (GenTAC) Consortium. Endovascular thoracic aortic repair in confirmed or supected genetically triggered thoracic aortic dissection. *J Vasc Surg*. 2018;68:364-71.
21. Sultan I, Siki MA, Bavaria JE, Dibble TR, Savino DC, Kilic A, et al. Predicting distal aortic remodeling after endovascular repair for chronic DeBakey III aortic dissection. *Ann Thorac Surg*. 2018;105:1691-6.
22. Conway AM, Qato K, Mondry LR, Stoffels GJ, Giangola G, Carroccio A. Outcomes of thoracic endovascular aortic repair for chronic aortic dissections. *J Vasc Surg*. 2018;67:1345-52.
23. Mizoguchi T, Zempo N, Kaneda Y. Early and mid-term outcomes following TEVAR for chronic type B aortic dissection. *Ann Vasc Dis*. 2017;10:345-50.
24. Huang CY, Hsu HL, Chen PL, Chen IM, Hsu CP, Shih CC. The impact of distal stent graft-induced new entry on aortic remodeling of chronic type B dissection. *Ann Thorac Surg*. 2018;105:785-93.

Key Words: open repair, chronic distal aortic dissection, endovascular

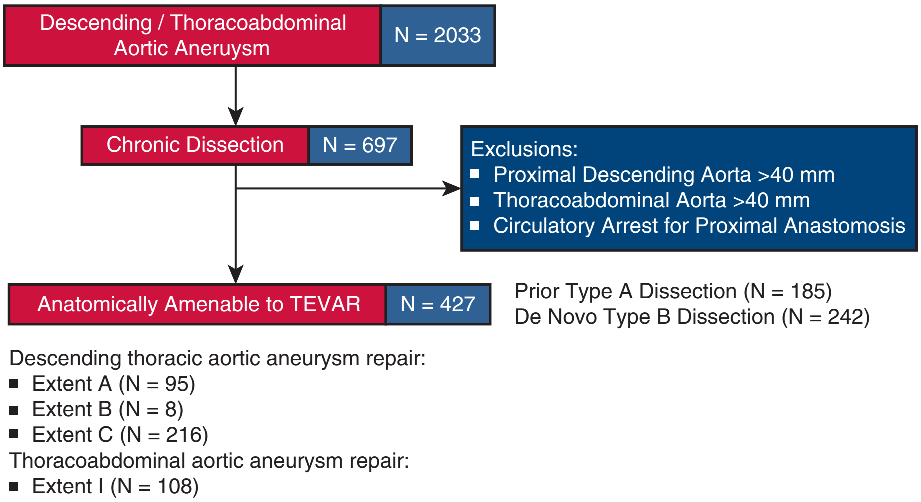


FIGURE E1. Flow chart of patient selection for the study.

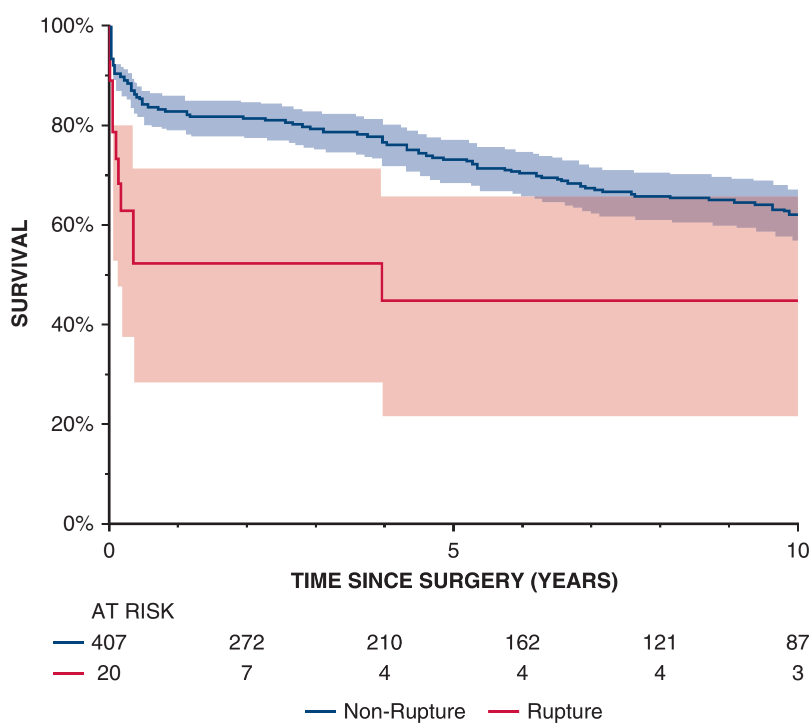


FIGURE E2. Kaplan-Meier curve demonstrating survival after open surgical repair of chronic distal aortic dissection in patients with and without aortic rupture.

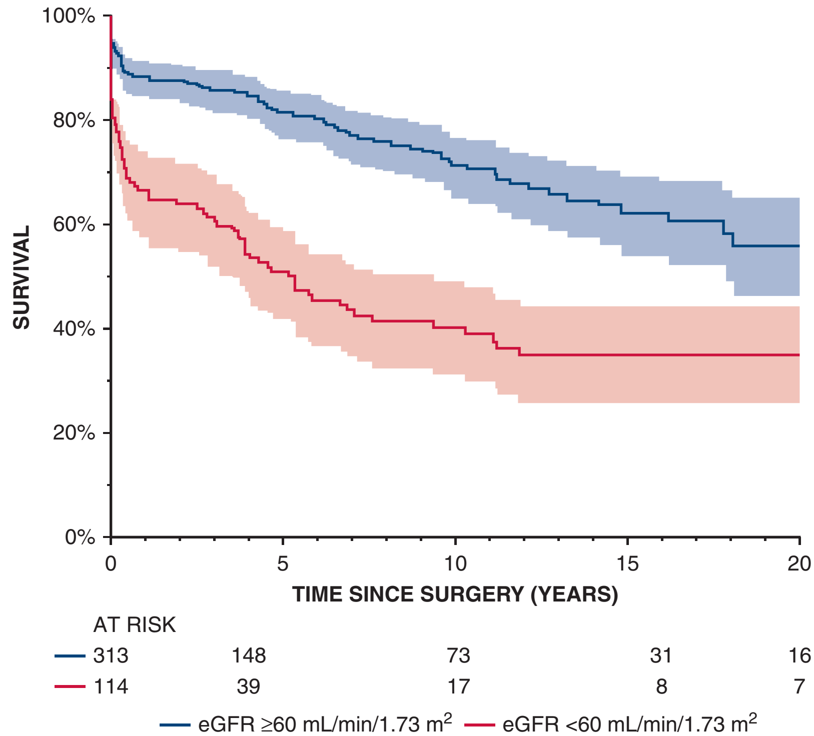


FIGURE E3. Kaplan-Meier curve demonstrating survival after open surgical repair of chronic distal aortic dissection in patients with estimated glomerular filtration rate (*eGFR*) <60 mL/min/1.73 m^2 and eGFR ≥60 mL/min/1.73 m^2.

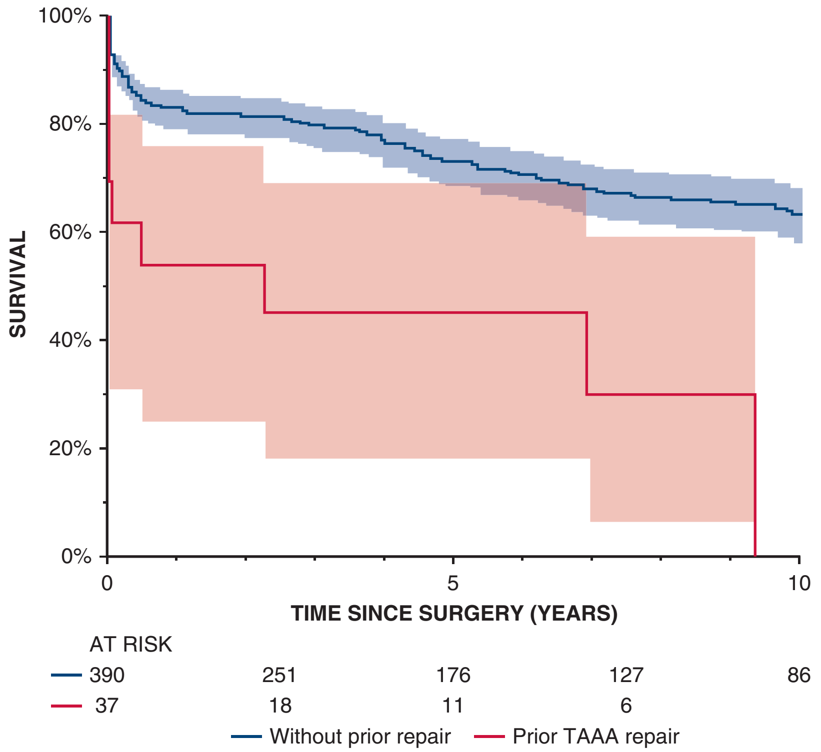


FIGURE E4. Kaplan-Meier curve demonstrating survival after open surgical repair of chronic distal aortic dissection in patients with and without prior thoracoabdominal aortic aneurysm. *TAAA*, Thoracoabdominal aortic aneurysm.

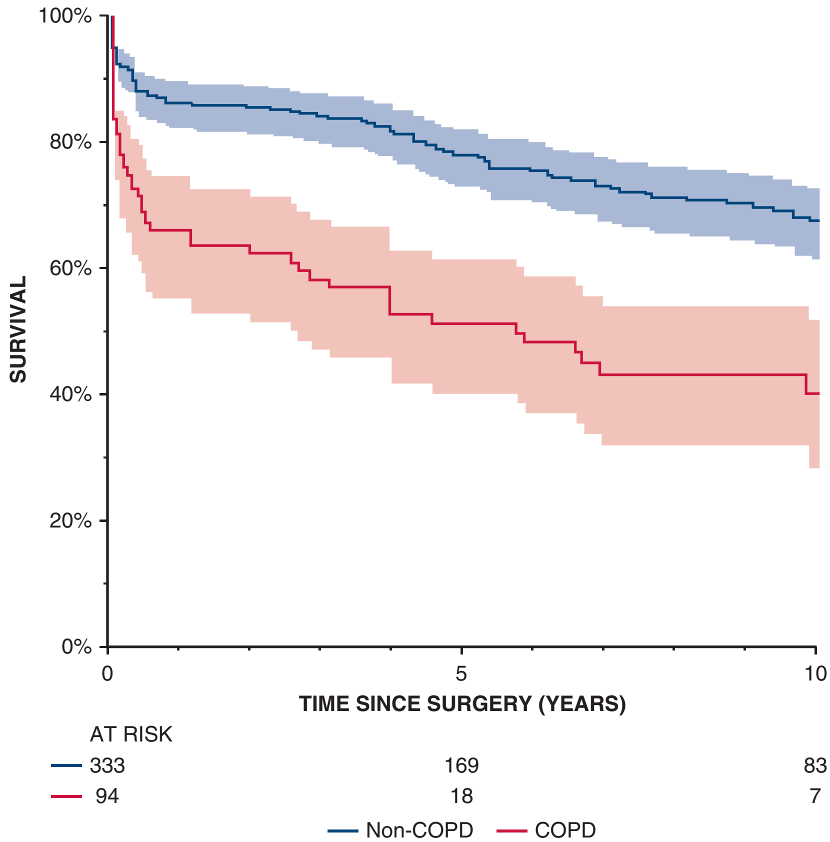


FIGURE E5. Kaplan-Meier curve demonstrating survival after open surgical repair of chronic distal aortic dissection in patients with and without chronic obstructive pulmonary disease. *COPD*, Chronic obstructive pulmonary disease.

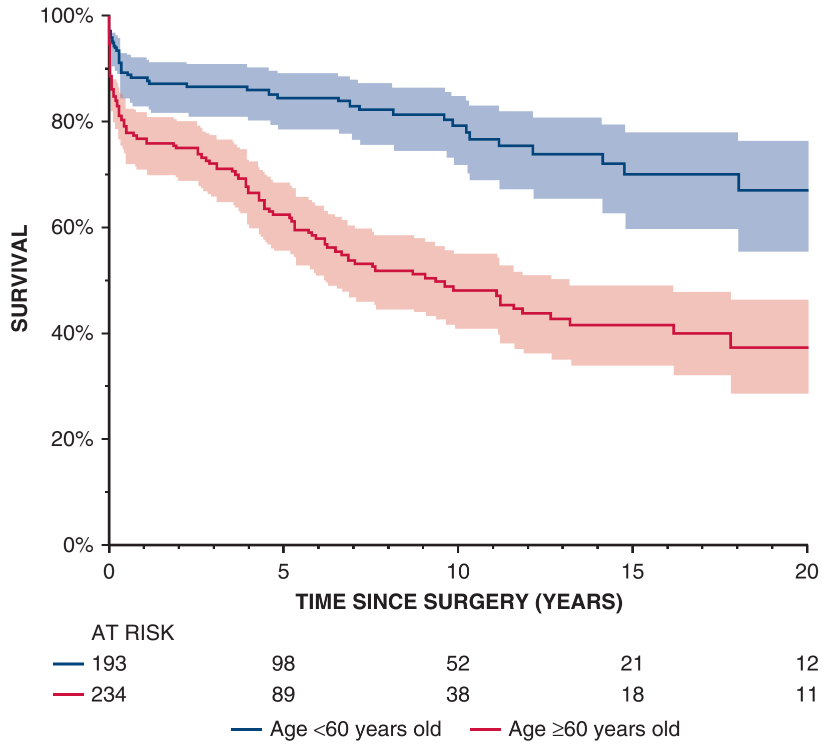


FIGURE E6. Kaplan-Meier curve demonstrating survival after open surgical repair of chronic distal aortic dissection in patients age ≥60 and <60 years.

TABLE E1. Variables considered in logistic regression analysis for 30-day mortality

Age >60 y
Rupture
Redo
Previous thoracoabdominal aortic aneurysm repair
Prior abdominal aortic aneurysm repair
Estimated glomerular filtration rate <60 mL/min/1.73 m^2
Chronic obstructive pulmonary disease

TABLE E2. Variables considered in cox regression analysis for long-term survival

Age >60 y
Rupture
Coronary artery disease
Cerebrovascular disease
Peripheral vascular disease
Diabetes mellitus
Estimated glomerular filtration rate <60 mL/min/1.73 m^2
Hyperlipidemia
COPD
History of type A aortic dissection
Prior thoracoabdominal aortic aneurysm repair
Prior descending thoracic aortic aneurysm repair
Prior abdominal aortic aneurysm repair
Prior ascending aortic repair
Prior aortic arch repair
Redo
Prior aortic surgery
Female sex
Hereditary aortic disease

COPD, Chronic obstructive pulmonary disease.