Impact of left ventricular ejection fraction on the outcomes of open repair of descending thoracic and thoracoabdominal aneurysms

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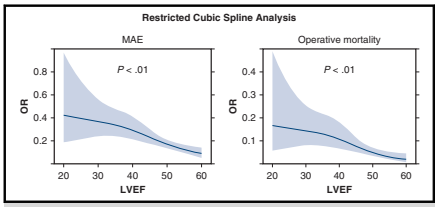
ABSTRACT

Objective: To discern the impact of depressed left ventricular ejection fraction (LVEF) on the outcomes of open descending thoracic aneurysm (DTA) and thoracoabdominal aneurysms (TAAA) repair.

Methods: Restricted cubic spline analysis was used to identify a threshold of LVEF, which corresponded to an increase in operative mortality and major adverse events (MAE: operative death, myocardial infarction, stroke, spinal cord injury, need for tracheostomy or dialysis). Logistic and Cox regression were performed to identify independent predictors of MAE, operative mortality, and survival.

Results: DTA/TAAA repair was performed in 833 patients between 1997 and 2018. Restricted cubic spline analysis showed that patients with LVEF <40% (n = 66) had an increased risk of MAE (odds ratio [OR], 2.17; 95% confidence interval [CI], 1.22-3.87; $P < .01$) and operative mortality (OR, 2.72; 95% CI, 1.21-6.12; $P = .02$) compared with the group with LVEF ≥40% (n = 767). The group with LVEF <40% had a worse preoperative profile (eg, coronary revascularization, 48.5% vs 17.3% [$P < .01$]; valvular disease, 82.8% vs 49.39% [$P < .01$]; renal insufficiency, 45.5% vs 26.1% [$P < .01$]; respiratory insufficiency, 36.4% vs 21.2% [$P = .01$]) and worse long-term survival (35.5% vs 44.7% at 10 years; $P = .01$). Nonetheless, on multivariate regression, depressed LVEF was not an independent predictor of operative mortality, MAE, or survival.

Conclusions: LVEF is not an independent predictor of adverse events in surgery for DTA. (J Thorac Cardiovasc Surg 2021;161:534-41)



Increased adverse events after DTA/TAAA repair in patients with LVEF <40%.

CENTRAL MESSAGE

Left ventricular ejection fraction is not an independent predictor of adverse events in surgery of the descending thoracoabdominal aorta.

PERSPECTIVE

Left ventricular ejection fraction is commonly used in the preoperative assessment of cardiac function before surgery of the descending thoracoabdominal aorta. Nonetheless, our analysis showed that it is not an independent predictor of adverse outcomes.

See Commentaries on pages 542 and 543.

Open repair of descending thoracic aneurysm (DTA) and thoracoabdominal aortic aneurysm (TAAA) is one of the most extensive procedures in cardiovascular surgery, as witnessed by the fact that even in highly specialized centers, estimated operative mortality is still between 5% and 8%.[1] Surgical outcomes largely depend on the insult to end-organs, whose preoperative status and functional reserve in turn modulate the degree of such iatrogenic injury.[2,3] More specifically, preoperative impairment of renal[4] and respiratory[5] functions have been shown to significantly affect the outcome of DTA/TAAA repair. In addition, a suboptimal left ventricular ejection fraction (LVEF; <50%) was associated with increased operative mortality in such operations.[6] It would be clinically useful to identify a nonarbitrary threshold of depressed LVEF that is related to worse outcomes not only in terms of

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Abbreviations and Acronyms

CBP = cardiopulmonary bypass
CI = confidence interval
DHCA = deep hypothermic circulatory arrest
DTA = descending thoracic aneurysm
FEV_1 = forced expiratory volume in 1 minute
HR = hazard ratio
LHB = left heart bypass
LVEF = left ventricular ejection fraction
MAE = major adverse events
NYHA = New York Heart Association
OR = odds ratio
SMD = standardized mean difference
TAAA = thoracoabdominal aortic aneurysm

operative mortality, but also in terms of major complications and postdischarge survival. Indeed, survival is significantly decreased in patients with reduced LVEF, especially those with LVEF <40%.[7] In this study, we aimed to determine this threshold.

METHODS

Patients, Definitions, and Endpoints

This study was approved by Weill Cornell Medicine's Institutional Review Board (protocol no. 1607017424), which waived the need for individual patient consent. A retrospective review of prospectively collected data from the Weill Cornell Medicine Department of Cardiothoracic Surgery's aortic surgery database was conducted to identify all consecutive patients who underwent repair of TAAA or DTA between May 1997 and December 2018. The database is constantly updated and maintained by a team of research personnel. Preoperative and perioperative variables are entered prospectively during the hospital stay. Postoperatively, clinical and radiologic follow-up is performed and recorded each year or in the event of clinical symptoms suggestive of aortic disease. In the event of missing/unreliable data, a direct interview with the patient, a relative, or the treating physician is performed if necessary after reviewing the patient's electronic chart.

The primary study endpoint was a composite of major adverse events (MAE) that included operative mortality (death during the same hospitalization of the index surgical procedure or within 30 days postoperatively), myocardial infarction, stroke, renal failure necessitating de novo dialysis, respiratory failure necessitating tracheostomy, and spinal cord injury (paraparesis and paralysis). Secondary endpoints were operative mortality and survival at 1, 5, and 10 years.

Surgical Technique

Details of our surgical procedure have been published previously.[8] In brief, a fifth, sixth, or seventh intercostal space thoracotomy or thoracoabdominal incision was made. Projected spinal, mesenteric, and renal ischemic times (based on the extent and complexity of the planned aortic reconstruction) dictated the use of systemic (ie, left heart bypass [LHB]) or splanchnic (ie, warm hematic mesenteric and cold crystalloid renal) perfusion adjuncts, according to evidence.[9,10] When perfusion adjuncts were deemed unnecessary, a "clamp and go" technique was pursued. Consequently, with exceptions dictated by the singularity of each case, our approach to organ protection was the following: DTAs with "clamp and go"; extent I TAAA with selective LHB (particularly when the etiology is acute/chronic dissection); extent II TAAA with customary LHB plus customary mesenteric and renal perfusion; extent III TAAA with customary mesenteric and renal perfusion with or without LHB (rarely); extent IV TAAA with selective mesenteric perfusion plus customary renal perfusion. Besides these criteria based on aneurysmal configuration, LHB was also used to make aortic clamping more tolerable in the event of compromised myocardial or valvular function. For DTAs and extent I and II TAAAs, the site of proximal clamping was preferentially distal to the left subclavian artery. The use of cardiopulmonary bypass (CPB) with deep hypothermic circulatory arrest (DHCA) was required when even clamping proximal to the left subclavian artery was not a "bail out" option.

The extent of resection depended on aortic size, symptoms/complications, and sporadic versus familial pathology. Aneurysmal segments were always replaced, whereas dissected segments were replaced only if symptomatic or in the event of a connective tissue disorder. This approach aimed to preserve the maximum number of segmental arteries and so minimize spinal cord ischemia while eliminating problematic aortic segments on prognostic or symptomatic grounds. Rapid reinfusion of shed blood was performed using a Belmont warm rapid infusion system (Belmont Instrument Corp, Billerica, Mass). When partial bypass or the clamp and sew technique were used, core temperature was allowed to passively decline to 33°C before cross-clamping the aorta. Reimplantation of intercostal arteries was performed with the inlay-inclusion technique. Visceral and renal arteries were either reimplanted or bypassed as dictated by the patient's anatomy. Hemashield Dacron grafts (Macquet Corp, Oakland, NJ) were used. Preoperative cerebrospinal fluid drainage was inserted preoperatively in all hemodynamically stable patients and at the completion of surgery in unstable patients. Cerebrospinal fluid pressure was maintained at <12 cmH_2O during the surgery and for 72 hours postoperatively.

Statistical Analysis

Continuous variables were summarized as median and interquartile range and compared using the Mann–Whitney U test, and categorical variables were presented as frequency and percentage and compared using χ^2 test or Fisher's exact test, as appropriate. Restricted cubic spline analysis was used to identify a clinically meaningful threshold of LVEF that corresponded to a significant worsening of the investigated outcomes. This analysis considered the following factors: (1) the majority of patients in our cohort had an LVEF between 30% and 50%; (2) LVEF >50% lies within the normal range; (3) patients with LVEF <30% represented only a small minority (0.2%); and (4) it is only worthwhile to investigate commonly used LVEF used and have practical meaning in a clinical context.

Based on these considerations, we iteratively evaluated LVEF values of 30%, 40%, and 50% to identify the threshold with the most significant impact on the primary endpoint. Univariate and multivariate analyses for postoperative mortality, MAE, and overall mortality at the latest follow-up (follow-up mortality) were performed with logistic and Cox regression analyses, respectively, for short- and long-term outcomes. Inclusion criteria for the variables included in the multivariable regression model are provided in the Appendix E1. To corroborate the results of the primary analysis, propensity score matching was used as an alternative model; the relative methodologic approach is specified in the Appendix E1. Results were expressed for cubic spline analysis and logistic regression as odds ratio (OR) with 95% confidence interval (CI) and for Cox regression as hazard ratio (HR) with 95% CI. Kaplan–Meier curves were used to illustrate survival, and their estimates were compared using the log-rank test and expressed as mean ± standard error (SE). Estimates expressed as percentage or median were truncated after the first decimal. Measures of confidence (95% CI, SE), significance (P value), and effect size (standardized mean difference [SMD]) were truncated after the second decimal, except when the third decimal was essential to discern statistical

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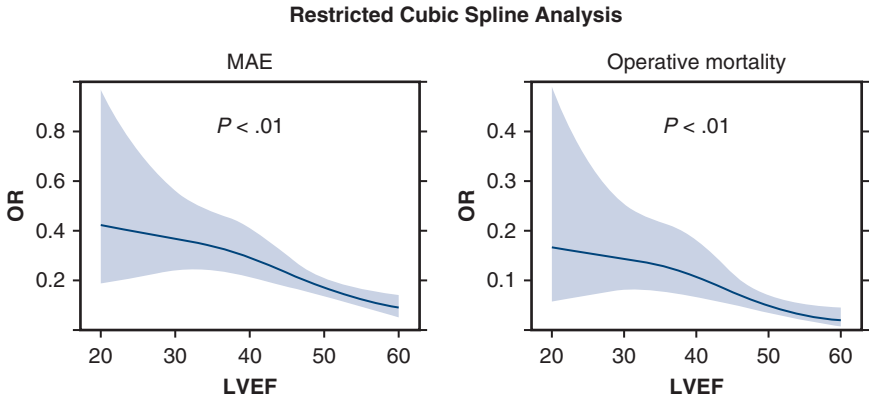

FIGURE 1. Left ventricular ejection fraction (*LVEF*) and adverse outcomes in surgery of the descending thoracoabdominal aorta. Restricted cubic spline analysis for the identification of knots, along the curve of LVEF values, corresponds to a significant deterioration in the operative mortality and major adverse events (*MAEs*; a combined endpoint of operative mortality, myocardial infarction, stroke, respiratory failure requiring tracheostomy, renal failure requiring de novo dialysis, paraparesis or paraplegia). When LVEF fell below 40%, there was a significant deterioration in operative mortality (odds ratio [*OR*], 2.72; 95% confidence interval [CI] 1.21-6.12; $P < .01$) and MAE (OR, 2.17; 95% CI, 1.22-3.87; $P < .01$).

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significance. For all statistical analyses, $P < .05$ was considered significant. Statistical analyses were performed using the R packages "tableone", "survminer", and "survival" in RStudio (R version 3.3.3; R Project for Statistical Computing, Vienna, Austria).

RESULTS

To avoid duplication, the results of our analysis are discursively summarized in this section, with an emphasis on differences that are statistically significant between the compared groups, and fully detailed in the related tables.

Preoperative Characteristics

During the study period, 833 patients underwent DTA/TAAA repair at our institution. Restricted cubic spline analysis showed that when LVEF fell below 40%, there was a significant increase in operative mortality (OR, 2.72; 95% CI, 1.21-6.12; $P = .02$) and MAE (OR, 2.17; 95% CI, 1.22-3.87; $P < .01$) (Figure 1). Therefore, the overall sample was dichotomized into groups of LVEF ≥40% (n = 767) versus LVEF <40% (n = 66). Patients with LVEF <40% were older (70.5 vs 68.0 years; $P = .02$), more frequently male (83.3% vs 57.2%; $P < .01$), and had a worse performance status (New York Heart Association class III/IV, 87.9% vs 16.6%; $P < .01$). They also had a greater cardiovascular burden (ie, coronary revascularization, 48.5% vs 17.3% [$P < .01$]; valvular disease, 82.8% vs 49.39% [$P < .01$]; peripheral arterial disease, 51.5% vs 23.3% [$P < .01$]), and higher prevalence of end-organ impairment (chronic renal insufficiency, 45.5% vs 26.1% [$P < .01$]; chronic respiratory insufficiency, 36.4% vs 21.2% [$P = .01$]) and cerebrovascular disease (30.3% vs 14.5%; $P < .01$) (Table 1).

Operative Conduct

The compared groups had a similar extent of aortic replacement, systemic and splanchnic ischemic times, circulatory support, and end-organ protection adjuncts. The only exception was DHCA, which was more frequently used in patients with LVEF <40% (15.2% vs 7.6%; $P = .05$) (Table 2).

Unadjusted Outcomes

Patients with a LVEF <40% had higher rates of operative mortality (12.1% vs 4.8%; $P = .03$), MI (4.7% vs 0.3%; $P < .01$), and MAE (27.3% vs 14.7%; $P = .01$) (Table 3). They also had a lower mean 1-year overall survival (OS) (66.3 ± 0.06% vs 85.1 ± 0.01%), 5-year OS (53.5 ± 0.07% vs 67.1 ± 0.02%), and 10-year OS (35.5 ± 0.08% vs 44.7 ± 0.03%) (P for trend = .01) (Figure 2).

Primary Model: Multivariable Regression

The variance inflation factor was <2 for all the included variables, and thus multicollinearity among them was ruled out. MAE was predicted by respiratory insufficiency (0.48; 95% CI, 0.30-0.76; $P < .01$) and renal insufficiency (2.44; 95% CI, 1.57-3.81; $P < .01$), but not by LVEF (0.42; 95% CI, 0.06-2.91; $P = .55$). Operative mortality was predicted by renal insufficiency (5.14; 95% CI, 2.45-10.79; $P < .01$), but not by LVEF (0.28; 95% CI, 0.02-4.14; $P = .58$). Follow-up mortality was increased by respiratory insufficiency (0.71; 95% CI, 0.54-0.93; $P = .01$) and renal insufficiency (1.75; 95% CI, 1.34-2.27; $P < .01$), but not by LVEF (0.55; 95% CI, 0.17-1.80; $P = .23$). The influences of procedural variables, demographics, and performance status on MAE, operative mortality, and follow-up mortality are detailed in Table 4.

Supplementary Model: Propensity Score Matching

Sixty-five patients with LVEF <40% were matched to 128 patients with LVEF ≥40%. All the endpoints were similar between the matched groups, corroborating the

TABLE 1. Preoperative variables in patients with LVEF ≥40% vs <40% undergoing descending thoracic or thoracoabdominal aneurysm repair

Variable	LVEF ≥40% (N = 767)	LVEF <40% (N = 66)	*P* value
Demographics and presentation			
Age, y, median (IQR)	68.00 (57.0-75.0)	70.50 (61.2-78.0)	.02
Male sex, n (%)	439 (57.2)	55 (83.3)	<.01
Urgency or emergency, n (%)	382 (49.8)	36 (54.5)	.54
Performance and metabolic status, n (%)			
NYHA class III/IV	126 (16.6)	58 (87.9)	<.01
Diabetes mellitus	67 (8.7)	16 (24.2)	<.01
Hypertension	733 (95.6)	66 (100.0)	.08
Cardiovascular disease, n (%)			
Coronary revascularization*	133 (17.3)	32 (48.5)	<.01
Valvular disease	377 (49.3)	53 (82.8)	<.01
Peripheral arterial disease	178 (23.2)	34 (51.5)	<.01
Neurologic status, n (%)			
Cerebrovascular accident	111 (14.5)	20 (30.3)	<.01
Paraparesis/paraplegia	10 (1.3)	1 (1.5)	.60
End-organ impairment, n (%)			
Renal (creatinine >1.5 mL or dialysis)	200 (26.1)	30 (45.5)	<.01
Hemodialysis	18 (2.3)	5 (7.6)	.03
Respiratory (FEV_1 ≤50%)	158 (21.2)	24 (36.4)	<.01
Liver disease	3 (0.4)	1 (2.5)	.28
Family and social history, n (%)			
History of smoking	571 (74.4)	61 (92,4)	<.01
Marfan syndrome	85 (11.0)	5 (7.6)	.53

LVEF, Left ventricular ejection fraction; *IQR*, interquartile range; *NYHA*, New York Heart Association; *FEV_1*, forced expiratory volume in 1 second. *Coronary revascularization includes percutaneous and open surgical coronary revascularization.

results of the primary model. Details of this supplementary model are provided in the Appendix E1.

DISCUSSION

Depressed LVEF is associated with increased operative mortality and decreased survival after cardiac[11] and major vascular[12] surgery. More specifically for open surgery of the descending thoracoabdominal aorta, Safi's group investigated the impact of LVEF <50% in their cohort, which was similar in size to ours (respectively 854 and 833 patients).[6] Their operative mortality was doubled by LVEF <50% (25.5% vs 13.8%; $P < .005$), which multivariate regression confirmed to be an independent risk factor for mortality (OR, 1.85; 95% CI, 1.09-3.15; $P = .03$).

We aimed to identify an LVEF threshold that demarcated a significant worsening in the outcomes of DTA/TAAA repair, avoiding reliance on a cutoff value based on a choice that was either arbitrary or extrapolated from nonpertinent evidence (eg, medical heart failure literature). This purpose was achieved with restricted cubic spline analysis, which identified a threshold of 40% as the value of LVEF demarcating a significant increase in adverse outcomes in our population. Interestingly, this result echoes the results of a meta-analysis based on individual data from 41,972 patients affected by symptoms of heart failure with or without reduced LVEF, which found an increased risk of death when LVEF fell below 40%.[7] It is intriguing to note that such an LVEF threshold is a broad predictor of mortality across disparate populations, irrespective of whether they consist of cardiologic patients on medical therapy or of non–risk-adjusted surgical patients undergoing major aortic reconstruction. Nonetheless, LVEF was not an independent predictor of adverse outcomes as respiratory and renal impairment were in the multivariate regression model. Rather, in our cohort, poor ventricular function was represented a marker of an unfavorable preoperative profile owing to its association with impairment of other end organs. This interpretation was corroborated by our supplementary analysis, which showed similar outcomes between patients with LVEF ≥40% versus <40% in the propensity score–matched cohort. We preoperatively correct both valvular diseases following published guidelines[13] and coronary disease according to our protocol as detailed previously[14] and recapitulated here as follows. All patients who underwent elective surgery underwent routine coronary angiography, except patients age <40 years with connective tissue disease and normal ventricular function (in which case, the angiogram was triggered by positive nuclear stress imaging).

TABLE 2. Intraoperative variables in patients with LVEF ≥40% vs <40% undergoing DTA or TAAA repair

Variable	LVEF ≥40% (N = 767)	LVEF <40% (N = 66)	*P* value
Era of surgery >2007, n (%)	391 (51.0)	22 (33.3)	<.01
Extent of aortic replacement			
TAAA/DTA, n (%)	531 (69.2)/236 (30.8)	47 (71.2)/19 (28.8)	.74
Crawford classification for TAAA, n (%)			.35
• Extent I	281 (52.9)	19 (40.4)	
• Extent II	103 (19.4)	12 (25.5)	
• Extent III	108 (20.3)	13 (27.7)	
• Extent IV	39 (7.3)	3 (6.4)	
Systemic and splanchnic ischemic time			
Aortic cross-clamp time, min, median (IQR)	33.0 (24.0-45.0)	34.5 (21.7-42.7)	.78
Mesenteric/renal ischemic time, min, median (IQR)	25.0 (17.0-31.0)	26.0 (19.2-31.0)	.57
Circulatory support, n (%)			
Clamp and sew/circulatory support	491 (64.2)/274 (35.8)	39 (59.1)/27 (40.9)	.49
Deep hypothermic circulatory arrest	58 (7.6)	10 (15.2)	.05
Left heart bypass	218 (30.7)	17 (30.7)	.95
End-organ protection adjuncts, n (%)			
Splanchnic perfusion*	155 (20.3)	19 (28.8)	.14
Cerebrospinal fluid drainage	653 (85.5)	52 (78.8)	.15

LVEF, Left ventricular ejection fraction; *TAAA*, thoracoabdominal aortic aneurysm; *DTA*, descending thoracic aneurysm; *IQR*, interquartile range. *Splanchnic perfusion: warm hematic perfusion to mesenteric vessel plus cold crystalloid perfusion to the kidney.

Patients with severe left main or triple-vessel coronary disease were treated with coronary artery bypass grafting, whereas patients with significant 1- or 2-vessel disease underwent percutaneous intervention with bare metal stents. DTA/TAAA surgery was performed during the same admission following percutaneous intervention, at a mean time of 3 months after coronary artery bypass grafting.[14] Therefore, our results should be extrapolated with caution to practices with different perioperative policies that do not call for preemptive treatment of all potential valvular and coronary causes of depressed ventricular function. Even though experienced groups perform LHB[3] and DHC[15] routinely with good results, we prefer to avoid the unwanted effects of indiscriminate use of extracorporeal circulation to end-organs[16] and coagulation system.[17]

TABLE 3. Adverse outcomes after DTA or TAAA repair in patients with LVEF ≥40% vs <40%

Variable	LVEF ≥40% (N = 767)	LVEF <40% (N = 66)	*P* value
Operative adverse events, n (%)			
Mortality	37 (4.8)	8 (12.1)	.03
Myocardial infarction	2 (0.3)	3 (4.7)	<.01
Tracheostomy	51 (7.6)	7 (11.9)	.36
De novo dialysis	37 (5.0)	5 (7.9)	.37
Stroke	13 (1.7)	2 (3.1)	.34
Paraparesis/paraplegia	18 (2.4)	2 (3.1)	1
Major adverse events*	113 (14.7)	18 (27.3)	.01
Kaplan-Meier survival estimate			.01
1-y, %, mean ± SE	85.1 ± 0.01	66.3 ± 0.06	<.01
5-y, %, mean ± SE	67.1 ± 0.02	53.5 ± 0.07	
10-y, %, mean ± SE	44.7 ± 0.03	35.5 ± 0.08	

LVEF, Left ventricular ejection fraction; *SE*, standard error. *Major adverse events: combined endpoint of operative mortality, myocardial infarction, tracheostomy, de novo dialysis, stroke, paraparesis/paraplegia.

Previous reports from our institution and other corroborate the evidence showing that respiratory and renal impairment have negative effects on the outcomes of DTA/TAAA repair, along with the realization that options to alleviate such effects are limited. In Svensson and colleagues' pioneering experience of 1414 TAAAs, operative survival was decreased in patients with chronic pulmonary disease (98% vs 83%; $P < .01$).[18] Based also on our institutional experience of a lack of a procedural measure able to neutralize the fact that a FEV_1 ≤50% increased the rate of MAE by 6-fold (OR, 6.99; 95% CI, 1.663-9.411; $P < .01$), we suggested considering an endovascular alternative in patients with chronic pulmonary disease and suitable DTA.[5] In addition, we reported on the inability of circulatory adjuncts to counteract the negative impact of chronic kidney insufficiency on mortality after DTA/TAAA repair (OR, 4.91; 95% CI, 2.01-11.97; $P < .01$).[4] Other initially promising measures, such as intraoperative stenting of the renal arteries, have been proven unsuccessful in alleviating the impact of renal impairment on the outcomes of surgery of the thoracoabdominal aorta.[19]

Our cohort mainly represents patients who were best treated with open surgery as opposed to endovascular repair, but this statement requires better contextualization given the time span (2 decades) and heterogeneity (DTA

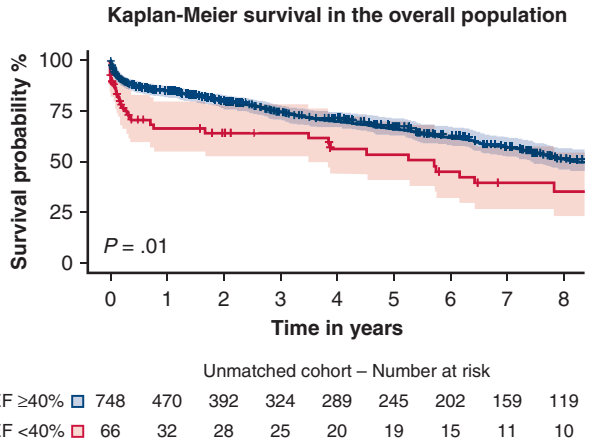

FIGURE 2. Relationship between left ventricular ejection fraction (*LVEF*) and adverse outcomes in surgery of the descending thoracoabdominal aorta. Kaplan-Meier survival after open descending thoracic and thoracoabdominal aortic aneurysm repair is shown. In the overall population, patients with LVEF <40% had lower survival compared with patients with LVEF ≥40%. The error bars represent the 95% confidence interval. The *P* value for survival was specifically calculated at 1 year postoperatively, because the greatest discrepancy in survival between the compared group was at 1 year in the unmatched population.

and TAAA) of the series. Regarding the latter, a distinction should be made between endovascular repair of DTAs (ie, thoracic endovascular aneurysm repair [TEVAR]) and of TAAAs (ie, endovascular thoracoabdominal aneurysm repair [eTAAAR]). After initial approval from the Food and Drug Administration in 2005, the use of TEVAR showed a steadily increase and surpassed open repair for aneurysm in 2006 and for dissection in 2010,[20] according to a pool of 155,187 patients from the National Inpatient Sample. A cohort of 11,669 patients from the same database indicated that the 2 treatments had exactly equal risk-adjusted operative mortality nationwide (both 2.3%); whereas TEVAR was associated with less procedure-related complications (OR, 0.39; $P < .01$) and length of stay (by 1.3 days), open repair had substantially lower hospital cost (by $6713).[21] Survival was worse with TEVAR compared with open repair in a propensity-matched sample of 15,305 Medicare patients in both the short term (1 year: 82% vs 87%; $P < .01$) and medium term (5 year: 62% vs 72%; $P < .01$).[22] Conversely, the use of eTAAAR has struggled to get past a prolonged experimental phase, leaving open repair of aneurysms spanning across the diaphragm as still the standard of care. Logistically, one of the main hurdles has been overcoming the need for custom-made devices that require several weeks for delivery.[23] Clinically, evidence from a small number of centers demonstrate that eTAAAR has similar to higher rates of mesenteric ischemia, side branch occlusion, aortic reoperation, and especially paraplegia (up to 50% for extent II TAAAs) compared with open repair.[24,25] Even though selected cohorts of eTAAAR recipients had lower operative mortality and length of hospital stay in recent studies,[26,27] this finding will need to stand the test of broad commercialization if developing modular devices will ever be able to offer a practical alternative to treat all-comers without stringent patient selection and manufacturer-friendly criteria. Although the hybrid approach aimed to exploit the advantages of open and endovascular techniques, it proved to entail the downsides of both techniques, with formidable rates of operative mortality (8.3%-34.2%), paraplegia (3.3%-11.8%), mesenteric ischemia (8.3%-17.1%), and renal failure (14%-28.9%).[28-30]

Over the 21-year time span of our series, although, commendably, an ever-increasing number of centers focused on developing TEVAR, regrettably, an ever-decreasing number of centers retained and optimized the

TABLE 4. Multivariable regression to identify independent predictors of major adverse events, operative and follow-up mortality after DTA or TAAA repair

Predictor	Major adverse events*	Operative mortality	Follow-up mortality
Procedure variables			
Era of surgery (>2007 vs ≤2007)	1.19 (0.77-1.82); $P = .44$	1.48 (0.77-2.87); $P = .24$	0.66 (0.48-0.90); $P = .01$
Site (DTA vs TAAA)	0.58 (0.36-0.94); $P = .03$	0.90 (0.44-1.84); $P = .78$	1.08 (0.83-1.41); $P = .56$
Demographics and performance status			
Age (continuous variable)	1.17 (0.87-1.58); $P = .31$	1.06 (0.65-1.72); $P = .82$	1.76 (1.45-2.14); $P < .01$
Sex (male vs female)	1.44 (0.94-2.22); $P = .10$	1.52 (0.77-2.99); $P = .22$	1.30 (1.01-1.68); $P = .04$
NYHA class (I/II vs III/IV)	1.76 (1.02-3.04); $P = .04$	2.09 (0.90-4.86); $P = .09$	1.32 (0.98-1.77); $P = .06$
End-organ function			
Respiratory (FEV_1 ≥50% vs <50%)	0.48 (0.30-0.76); $P < .01$	0.73 (0.35-1.52); $P = .40$	0.71 (0.54-0.93); $P = .01$
Cardiac (LVEF as continuous variable)†	0.42 (0.06-2.91); $P = .55$	0.28 (0.02-4.14); $P = .58$	0.55 (0.17-1.80); $P = .23$
Renal (creatinine ≥1.5 mg/dL or dialysis)	2.44 (1.57-3.81); $P < .01$	5.14 (2.45-10.79); $P < .01$	1.75 (1.34-2.27); $P < .01$

Values are expressed as odds ratio (95% confidence interval [CI]), with *P* values for major adverse events and operative mortality, and as hazard ratio (95% CI) with *P* values for follow-up mortality. *DTA*, Descending thoracic aneurysm; *TAAA*, thoracoabdominal aortic aneurysm; *NYHA*, New York Heart Association; FEV_1, forced expiratory volume in 1 second; *LVEF*, left ventricular ejection fraction. *Major adverse events: combined endpoint of operative mortality, stroke, myocardial infarction, tracheostomy, de novo dialysis, and paraplegia/paraparesis. †LVEF was assessed as a continuous variable using restricted cubic spline analysis.

capability to offer open surgery of the descending thoracic aorta.[31] Even though we routinely offer TEVAR, our unit is primarily known as a tertiary referral center for open repair of TAAAs and of DTAs that are unsuitable for TEVAR because of anatomic barriers (eg, lack of landing zones), indications (eg, chronic dissection), infected state, etiology (eg, connective tissue disease), complications of endovascular repair, and so on. This explains the discrepancy in the DTA-to-TAAA ratio between the general population (~4:1)[32] and our open series (<1:2; ratio, 0.44; n = 255/578) as follows. First, there is a referral bias; nontertiary centers refer most of the TAAAs (which are complex cases irrespective of an endovascular vs an open approach), but only those DTAs that are unsuitable for TEVAR. Second, there is a treatment bias; we offer endovascular and open approaches for DTAs, but virtually all-comers with TAAAs are treated with open surgery for the aforementioned reasons. There is no evidence to support a change in this policy because of the higher-risk profile of certain patient subsets. Indeed, the choice between endovascular and open repair should rely mainly on aortic-related criteria (as noted regarding the unsuitability of patients for TEVAR) rather than on preoperative risk profile. This concept has been robustly corroborated by level 1A evidence regarding endovascular repair of abdominal aneurysm (endovascular aneurysm repair [EVAR]), which benefits from a much more solid body of literature than TEVAR. In patients suitable for either open or endovascular repair, after the index operation, mortality was (1) lower for EVAR during the first 6 months, (2) equivalent in the 2 treatments between 6 months and 8 years, and (3) lower for open repair after 8 years.[33] In high-risk patients unfit for open surgery, endovascular repair was associated with substantial operative mortality (9%) with no survival benefit, along with a higher reintervention rate and higher costs compared with best medial therapy.[34]

Intuitively, these concepts can also be applied to TEVAR in the absence of specific evidence, with the expectation of an ever-decreasing margin of gain for the endovascular approach with the complexity of eTAAAR. Nonetheless, even though a high-risk preoperative profile is not a default indication for endovascular repair, there are specific independent predictors of adverse events that can be mitigated by TEVAR. This is the case for respiratory impairment, as discussed above, but not for depressed LVEF that was not a significant risk factor in our analysis. In our series, the percentage of patients with LVEF <40% was higher before 2007 (51.0% vs 33.3%; $P < .01$). As we have maintained the above-delineated policy regarding surgical indication over the entire study period, this finding likely reflects preoperative cardiac optimization due to improved medical therapy.

Finally, the decision to operate on patients with depressed LVEF ought to have a holistic basis that considers MAE and OS. Indeed, the yearly risk of death in a carrier of thoracic aneurysm ≥6 cm is 11.8%,[35] which translates in a 5-year survival of 41%. The latter can be compared with the 5-year survival of our patients who underwent open DTA/TAAA repair, which was 44.6% versus 67.1% with depressed versus preserved LVEF. Therefore, open repair provides an approximate 5-year survival benefit of 3.6% in patients with depressed LVEF, at the price of a quality of life likely affected by the sequelae of 35% MAE in this group. Conversely, there is a higher survival benefit of 26.1% with surgery in patients with preserved LVEF, whose quality of life is also less affected by a lower MAE of 14.8%.

We acknowledge the limitations inherent to the retrospective nature of our analysis. Caution is warranted in extrapolating the results of a sample from a high-volume aortic center to the broader population of patients undergoing repair of the descending thoracic aorta. The possibility of type II error should be entertained in our failure to identify LVEF <40% as an independent predictor of adverse outcomes, because of our small sample size. Despite its ubiquitous use, LVEF is limited in its capability to assess global ventricular function (eg, confined to the ejection phase, not indexed for modulators, such as afterload).

In conclusion, our data show that LVEF is not an independent predictor of adverse outcomes in surgery of the DTA.

Conflict of Interest Statement

The authors have nothing to disclose with regard to commercial support.

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Key Words: thoracoabdominal aneurysm, aortic aneurysm, descending thoracic aneurysm, left ventricular ejection fraction, aortic surgery

APPENDIX E1. METHODS

Primary Model: Multivariable Regression

Multivariable logistic regression included variables with $P < .2$ on univariate analysis, in addition to clinically relevant variables according to the following categories: (1) procedure variables: era of surgery (>2007 vs ≤2007), surgical site (DTA vs TAAA); (2) demographics and performance status: age (continuous variable), sex (male vs female), NYHA class (I-II vs III-IV); and (3) end-organ function: renal (creatinine ≥1.5 mg/dL or dialysis[4]), respiratory (FEV_1 ≥50% vs <50%[5]), cardiac (LVEF threshold as determined by restricted cubic spline analysis) (Table E1). Variance inflation factor analysis was conducted to exclude multicollinearity between variables in the regression model.

Alternative Model: Propensity Score Matching

Propensity score matching was used to corroborate the results of the primary analysis. Using the threshold of LVEF identified by the restricted cubic spline analysis, patients with depressed LVEF were propensity score matched to patients with preserved LVEF. The matching protocol included a 1:2 ratio, allowed a caliper size of 0.2 SD, and used the nearest-neighbor method without replacement. The matching was nonparsimonious and inclusive as tolerated of variables, which were preferentially considered and retained for their impact on the endpoints according to relevant literature and clinical judgment. The adequacy of variable matching was assessed using Cohen's *d*, with the effect size expressed as SMD.

RESULTS

Sixty-five patients with LVEF <40% were matched to 128 patients with LVEF ≥40%. The variables retained for the propensity score matching had a SMD <0.1, and were categorized as follows: demographics and presentation (eg, age, sex, nonelective status), neurologic status (eg, preoperative stroke), and end-organ and systemic vasculature morbidity (eg, respiratory and renal impairment as defined for primary model of logistic regression, peripheral arterial disease) (Table E2). The adequacy of matching is also graphically appreciable by distribution histograms for propensity score density (Figure E1), mirror histograms for distributional distance (Figure E2), a Love plot for covariate balance (Figure E3), and a line plot for absolute standard difference in means (Figure E4). A detailed breakdown of the preoperative profile, operative conduct, and endpoint analysis for the matched cohort is reported in Table E2, with a graphical depiction of survival shown in Figure E5.

In summary, all endpoints were similar between the matched groups, and thus the supplementary analysis corroborated the results of the primary model. As in the unmatched sample, the greatest discrepancy in survival was at 1 year postoperatively, this was specifically analyzed in the matched sample: 1-year survival was similar in patients with LVEF ≥40% and those with LVEF <40% (81.2 ± 0.04% vs 67.3 ± 0.06%; $P = .054$).

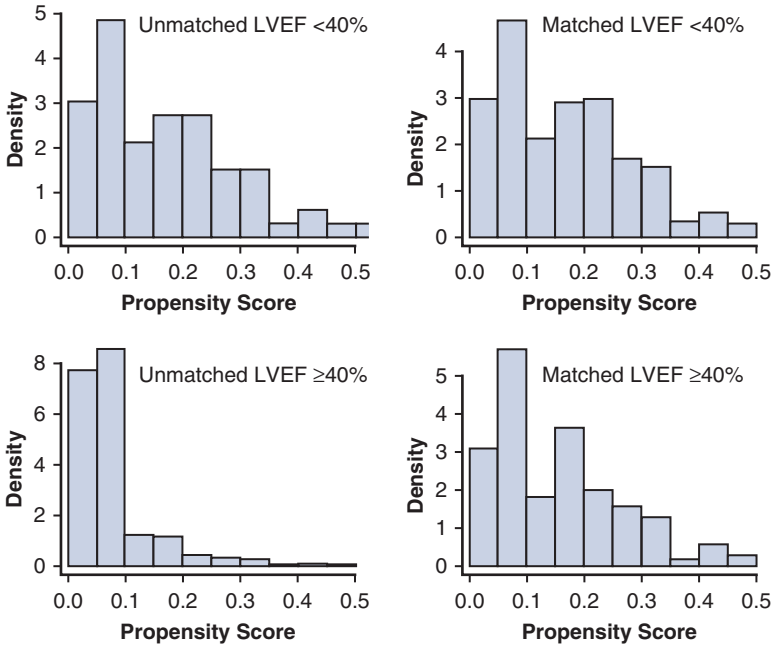

FIGURE E1. Density of propensity score distribution. The histograms show graphical assessment of the density of propensity score distribution in patients with left ventricular ejection fraction (*LVEF*) ≥40% vs those with LVEF <40% undergoing open descending thoracic and thoracoabdominal repair.

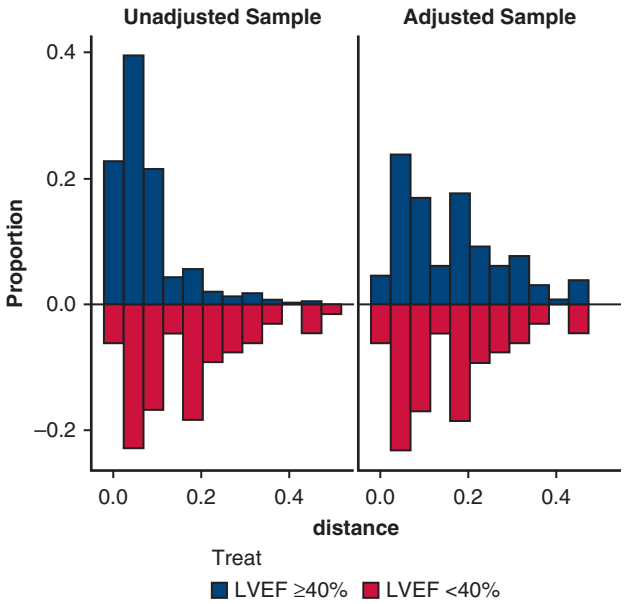

FIGURE E2. Distributional balance of propensity score distribution. Mirror histograms show the distributional balance of propensity score distribution in patients with left ventricular ejection fraction (*LVEF*) ≥40% vs those with LVEF <40% undergoing open descending thoracic and thoracoabdominal repair.

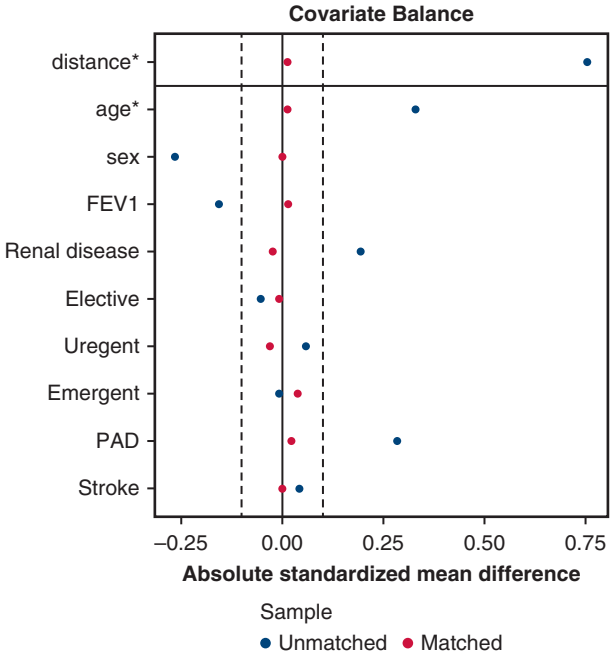

FIGURE E3. Love plot for covariate balance in the propensity score matching of patients with left ventricular ejection fraction (LVEF) ≥40% vs those with LVEF <40% undergoing open descending thoracic and thoracoabdominal repair. *FEV_1*, Forced expiratory capacity in 1 second; *PAD*, peripheral arterial disease.

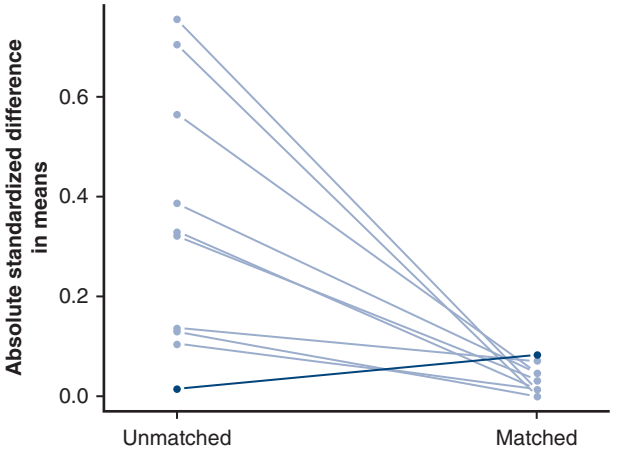

FIGURE E4. Line plot depicting the absolute standardized difference in means before and after propensity score matching in patients with left ventricular ejection fraction (LVEF) ≥40% vs those with LVEF <40% undergoing open descending thoracic and thoracoabdominal repair.

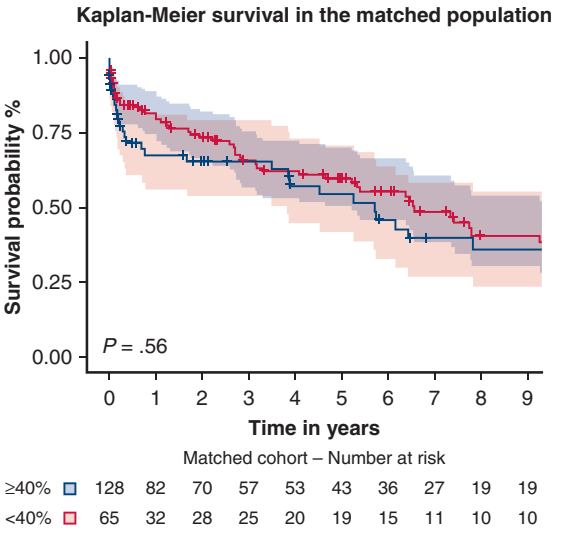

FIGURE E5. Effect of left ventricular ejection fraction (*LVEF*) on survival after surgery of the descending thoracoabdominal aorta in the matched population. Shown is Kaplan-Meier survival after open descending thoracic and thoracoabdominal aortic aneurysm repair. In the propensity-score matched population, survival was similar in patients with LVEF ≥40% vs those with LVEF <40%. Error bars represent the 95% confidence interval.

TABLE E1. Univariate regression to identify risk factors for major adverse events and operative and follow-up mortality after DTA or TAAA repair

Predictors	Major adverse events*	Operative mortality	Follow-up mortality
Procedure variables			
Era of surgery (>2007 vs ≤2007)	0.88 (0.48-1.61); $P = .68$	0.80 (0.55-1.17); $P = .25$	0.48 (0.36-0.65); $P < .01$
Site (DTA vs TAAA)	0.91 (0.47-1.77); $P = .79$	0.56 (0.36-0.88); $P = .01$	1.01 (0.78-1.31); $P = .91$
Demographics and performance status			
Age (continuous variable)	1.02 (1.00-1.05); $P = .03$	1.02 (1.01-1.04); $P < .01$	1.04 (1.03-1.05); $P < .01$
Sex (male vs female)	1.17 (0.64-2.15); $P = .59$	1.37 (0.94-2.00); $P = .09$	1.34 (1.07-1.69); $P = .01$
NYHA class (I/II vs III/IV)	4.03 (2.19-7.41); $P < .01$	3.05 (2.05-4.54); $P < .01$	2.18 (1.71-2.76); $P < .01$
End-organ function			
Respiratory (FEV_1 ≥50% vs <50%)	0.23 (0.09-0.57); $P = .01$	0.26 (0.17-0.39); $P < .01$	0.40 (0.31-0.52); $P < .01$
Cardiac (LVEF ≥40% vs <40%)	2.17 (1.22-3.87); $P < .01$	2.72 (1.21-6.12); $P = .02$	1.61 (1.12-2.31); $P = .01$
Renal (creatinine ≥1.5 mg/dL or dialysis)	6.55 (3.41-12.57); $P < .01$	3.46 (2.35-5.08); $P < .01$	2.40 (1.90-3.03); $P < .01$

DTA, Descending thoracic aneurysm; *TAAA*, thoracoabdominal aortic aneurysm; *NYHA*, New York Heart Association; *FEV_1*, forced expiratory volume in 1 second; *LVEF*, left ventricular ejection fraction. *Major adverse events: combined endpoint of operative mortality, stroke, myocardial infarction, tracheostomy, de novo dialysis, and paraplegia/paraparesis.

ADULT

TABLE E2. Preoperative profile, surgical conduct, operative adverse events, and survival of patients with LVEF ≥40% vs those with LVEF <40% undergoing DTA or TAAA repair

Variable	LVEF ≥40% (N = 128)	LVEF <40% (N = 65)	*P* value	SMD
Preoperative profile				
Demographics and presentation				
Age, y, median (IQR)	71.5 (62.0-77.0)	71.0 (61.0-78.0)	.90	0.02
Female sex, n (%)	22 (17.2)	11 (16.9)	1.00	>0.01
Urgency or emergency, n (%)	68 (53.1)	35 (53.9)	.85	0.08
Performance and metabolic status, n (%)				
NYHA class III/IV	32 (25.2)	57 (87.7)	<.01	1.62
Diabetes mellitus	15 (11.7)	16 (24.6)	.03	0.33
Hypertension	123 (96.1)	65 (100.0)	.25	0.28
Cardiovascular disease, n (%)				
Coronary revascularization	34 (26.6)	31 (47.7)	<.01	0.44
Peripheral arterial disease	61 (47.7)	33 (50.8)	.79	0.06
Neurologic status, n (%)				
Stroke	13 (10.2)	7 (10.8)	1.00	0.02
Paraparesis/paraplegia	2 (1.6)	1 (1.5)	.22	0.25
End-organ impairment, n (%)				
Respiratory (FEV_1 ≤50%)	46 (35.9)	23 (35.3)	1.00	0.01
Renal (creatinine >1.5 mL or dialysis)	59 (46.1)	29 (44.6)	.96	0.03
Family and social history, n (%)				
Aneurysm or dissection in the family	3 (2.4)	5 (7.7)	.16	0.26
Marfan syndrome	8 (6.2)	5 (7.7)	.94	0.05
Past or present smoking	107 (83.5)	60 (92.3)	.21	0.28
Operative conduct				
Date and site of procedure				
Year, median (IQR)	2006 (2002-2011)	2004 (2000-2011)	.13	0.18
Site: DTA vs TAAA, n (%)	39-89 (30.4-69.5)	18-47 (27.6-72.3)	.81	0.06
Circulatory support, n (%)				
DHCA	6 (4.7)	10 (15.4)	.02	0.36
Left heart bypass	29 (23.8)	17 (30.9)	.41	0.16
Systemic and splanchnic ischemic time				
Aortic cross-clamp time, min, median (IQR)	35.0 (25.0-43.2)	35.0 (21.0-43.0)	.83	0.05
Splanchnic ischemic time, min, median (IQR)	27.0 (18.0-34.5)	26.0 (19.00-31.0)	.50	0.09
End-organ adjuncts, n (%)				
Splanchnic perfusion	33 (25.7)	19 (29.2)	.19	0.25
Cerebrospinal fluid drainage	107 (83.6)	51 (78.5)	.49	0.13
Endpoint analysis				
Primary endpoint, n (%)				
Major adverse events	21 (16.4)	17 (26.2)	.15	0.24
Operative adverse events, n (%)				
Operative mortality	10 (7.8)	7 (10.8)	.67	0.10
Myocardial infarction	0 (0.0)	3 (4.8)	.06	0.31
Tracheostomy	7 (6.7)	7 (12.1)	.38	0.18
De novo dialysis	8 (6.7)	5 (8.1)	.97	0.05
Stroke	2 (1.6)	2 (3.1)	.87	0.10
Paraplegia/paraparesis	6 (4.8)	2 (3.1)	.86	0.08
Kaplan-Meier estimate of survival			.56	—
1 y, %, mean ± SE	81.2 ± 0.04	67.3 ± 0.06	.054	—
5 y, %, mean ± SE	59.5 ± 0.05	54.4 ± 0.07		
10 y, %, mean ± SE	36.2 ± 0.06	36.1 ± 0.08		

LVEF, Left ventricular ejection fraction; *SMD*, standardized mean difference; *IQR*, interquartile range; *NYHA*, New York Heart Association; *FEV_1*, forced expiratory volume in 1 second; *DTA*, descending thoracic aneurysm; *TAAA*, thoracoabdominal aneurysm; *DHCA*, deep hypothermic circulatory arrest; *SE*, standard error.