Thoracic endovascular aortic repair: A single center's 15-year experience

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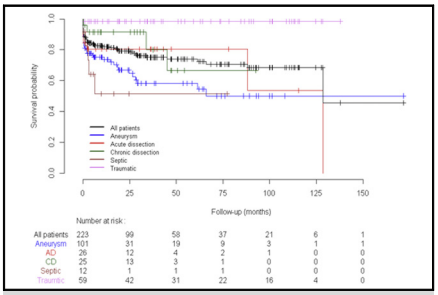
ABSTRACT

Objective: Specific complications of thoracic endovascular aortic repair (TEVAR) exist and long-term data are lacking. The purpose of this study was to evaluate our long-term TEVAR results.

Methods: This is a single-center retrospective study of 223 patients undergoing TEVAR from 1998 to 2013. Indication was aneurysm (45%), traumatic (26%), dissection (23%), and septic (6%).

Results: Patients' mean age was 62.7 ± 17.9 years, 84% of them had an American Society of Anesthesiologists score ≥3, and 42% had an aortic rupture. TEVAR was performed in zone 0 (n = 17), 1 (n = 17), or 2 (n = 59) in 42% of patients. Technical success rate was 96.4%. Overall 30-day mortality was 11.7% (elective aneurysm, 11.6%; emergent aneurysm, 34.3%; acute type B dissection, 14.8%; chronic dissection, 4.2%; septic, 8.3%; and traumatic, 1.7%). Major adverse events included stroke in 4.5%, spinal cord ischemia in 1.8%, and retrograde aortic dissection in 2.7%. Mean follow-up was 43.4 ± 38 months. Estimated aortic complications-free survivals at 12, 36, 60, and 120 months were (% ± standard error) 73% ± 3%, 64% ± 4%, 62% ± 4% and 57% ± 5%, respectively. Multivariate analysis showed that patients treated for a chronic aortic dissection had a significant risk of late reintervention ($P = .001$)

Conclusions: Because of its simplicity and low morbimortality rate, TEVAR has become the first-line approach for thoracic aortic diseases. Mortality outcomes are related to aortic pathology, emergent status, and proximal landing zone. To improve long-term results, rigorous patient selection and follow-up, development of referral centers, and technologic evolution of materials have to be reached.



Product-limit survival estimates curves.

Central Message

We present a single-institution's experience with early and late outcomes of TEVAR across 15 years.

Perspective

Mortality outcomes after thoracic endovascular aortic repair are related to the aortic pathology, elective or emergent status, and proximal landing zone. To improve long-term results, rigorous patient selection and follow-up, development of referral centers, and technologic evolution of materials have to be reached.

Since its first description in 1994,[1] thoracic endovascular aortic repair (TEVAR) has evolved from homemade experimental devices to being the first-line therapy for most thoracic aortic pathologies using commercially available thoracic stent-grafts for anatomically suitable patients.[2,3]

Experience with abdominal aortas showed that the early mortality advantage associated with endovascular repair of abdominal aneurysms was affected by specific reported complications, such as late aortic rupture and endoleaks.[4] Similar long-term durability issues concerning TEVAR have been raised. Furthermore, encouraging results of TEVAR have prompted many authors to use stent-grafts in more challenging anatomical regions using a hybrid approach,[5-8] increasing the risk of TEVAR failure.

Most studies describe early outcomes from TEVAR with relatively short-term follow-up.[9-17] We present the early and late outcomes of our single-institution experience with TEVAR.

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Abbreviations and Acronyms

ASA = American Society of Anesthesiologists
CT = celiac trunk
LSA = left subclavian artery
MSOF = multisystem organ failure
RAD = retrograde aortic dissection
SAV = supra-aortic vessels
SCI = spinal cord ischemia
SMA = superior mesenteric artery
TEVAR = thoracic endovascular aortic repair

MATERIALS AND METHODS

Patients

From November 1998 to January 2013, 235 patients were treated in our institution for various thoracic aortic pathologies. Among these patients, 223 had favorable preoperative anatomic conditions to perform TEVAR. They constituted the basis of our study. A retrospective analysis of this series was performed using a prospectively maintained database. Patients were divided into 4 groups (aneurysm, dissection, traumatic, and septic).

All patients underwent a preoperative computed tomography scan to assess the feasibility of TEVAR. As a prerequisite for successful stent-graft placement, a proximal landing zone of healthy and nondissected aorta (or graft) of at least 20 mm and a diameter <40 mm were deemed necessary. Our criteria for oversizing have changed with time. At the beginning of our experience, the goal of TEVAR was to achieve lesion exclusion and we used a 20% oversizing for all the different diseases. Furthermore, at the beginning of our experience, the range of stent-graft diameter available was limited when compared with the broad range of stent-graft diameters currently available. However, complications related to excessive oversizing such as stent-graft collapse and RAD have prompted us to change our surgical strategy. Oversizing is currently moderate (maximum, 15%) and depends on the disease: 10% to 15% for traumatic transection and degenerative aneurysm, 10% for chronic aortic dissection, and 5% for acute aortic dissection.

According to the classification proposed by Fillinger and colleagues[18] patients were divided into 5 groups based to the proximal landing zone (Figure E1).

Study protocols were in accordance with the Declaration of Helsinki and were approved by the Montpellier Institutional Review Board.

Procedure

In case of pathologies involving the aortic arch, successful stent-graft placement required supra-aortic vessel (SAV) debranching before stent-graft deployment to achieve a suitable proximal landing zone. Technical details of SAV debranching have been described in previous publications.[7,8,19] In case of staged procedure, 1 week was observed between debranching and TEVAR. Prophylactic use of cerebrospinal fluid drainage to prevent spinal cord ischemia (SCI) was not used.

In cases where overstenting of the origin of the left subclavian artery (LSA) was necessary, revascularization was systematic in elective cases and in emergent patients without unstable hemodynamic status at the time of treatment.

In a few cases, overstenting of the celiac aorta was deemed necessary. Visceral debranching was performed via a prosthetic bypass from the aorta or iliac artery to the celiac trunk (CT), superior mesenteric artery (SMA), and/or renal arteries.

After achieving suitable landing zones, endograft deployment was performed. Technical details on endograft deployment have been described in previous publications.[7,8,19] After deployment, stent-graft modeling with a low-pressure balloon was performed, except in dissection cases.

Outcome criteria were defined according to the Reporting Standards for Thoracic Endovascular Aortic Repair.[18] Technical success required successful introduction and successful deployment of the device in the absence of surgical conversion to open repair, death ≤24 hours, type I or III endoleaks as evidenced by procedural angiography, or graft obstruction. Follow-up included clinical examination and computed tomography scans during hospital stay, at 1 month, 6 months, and yearly thereafter.

Statistics

Primary research concerned all patients and pathology outcomes. Pathology-specific mortality analyses were presented as secondary objectives without adjusted *P* value for multiple comparisons.

Descriptive data were summarized as mean ± standard deviation or median with interquartile range according to the normality of the distribution, assessed with the Shapiro-Wilk test and compared with Mann-Whitney *U* or *t* test. Categorical data were expressed as number and percentages and compared with a χ^2 analysis.

Early outcome data were analyzed using logistic regression after calibration using the Hosmer-Lemeshow goodness-of-fit test. Late outcome is a time-related data point and was analyzed using a Cox regression model. Every variable associated with a *P* value below .20 in the univariate analysis was entered into multivariate models. A stepwise procedure was used to obtain the final multivariate model.

Survival status was assessed by the Kaplan-Meier method and compared between groups with the log-rank test.

Statistical analysis was performed using R Software version 3.1.0 (R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

We performed 223 thoracic aortic stent-grafting procedures from November 1998 to January 2013. The number of patients treated in each calendar year is reported in Figure E2. A total of 280 endografts were deployed (Table E1). There was no evidence for propensity for certain types of grafts for certain pathologies or zones. Patient demographic data are described in Table E2.

Distribution of pathologies treated included aneurysm group (n = 101; 45%), traumatic transections (n = 59; 26%), acute and chronic dissections (n = 51; 23%), and septic pathologies (n = 12; 5%). Procedure was emergent in 121 patients (54%), including 93 aortic ruptures (42%); 102 (46%) procedures were elective.

In the aneurysm group, there were 87 degenerative aneurysms, 9 anastomotic pseudoaneurysms, and 5 penetrating ulcers. Thirty-two procedures were emergent, including 24 aortic ruptures.

In the dissection group, there were 25 chronic dissections (including 1 aortic rupture) and 26 type B acute dissections with 8 aortic ruptures, 5 malperfusions, 7 patients with best medical treatment having rapid aortic expansion, and 6 uncomplicated patients included in the Acute Dissection: Stent Graft or Best Medical Therapy (ADSORB) study.[20]

In the traumatic group, diagnosis of aortic disruption was achieved at initial admission for 50 patients. Thirty-three (56%) had TEVAR procedure during the first 24 hours. The delay between the time of aortic disruption and endovascular treatment was <5 days for 82% of patients with a mean interval of 4.1 ± 8.8 days. For 6 patients, treatment was delayed due to septic state or major cerebral lesions.

TABLE 1. Distribution of proximal landing zone

Zone	Total (n = 223)	Aneurysm group (n = 101)	Dissection group (n = 51)	Traumatic group (n = 59)	Septic group (n = 12)
Zone 0	17 (8)	10 (9.9)	6 (11.8)	0 (0)	1 (8.3)
Emergent	6 (2.7)	3 (3.0)	2 (3.9)	0 (0)	1 (8.3)
Elective	11 (4.9)	7 (6.9)	4 (7.8)	0 (0)	0 (0)
Zone 1	17 (8)	10 (9.9)	3 (5.9)	1 (1.7)	3 (25)
Emergent	6 (2.7)	1 (1.0)	1 (2.0)	1 (1.7)	3 (25)
Elective	11 (4.9)	9 (8.9)	2 (3.9)	0 (0)	0 (0)
Zone 2	59 (26)	24 (23.8)	17 (33.3)	15 (25.4)	3 (25)
Emergent	29 (13)	6 (5.9)	10 (19.6)	10 (16.9)	3 (25)
Elective	30 (13.4)	18 (17.8)	7 (13.7)	5 (8.5)	0 (0)
Zone 3	81 (36)	21 (20.8)	18 (35.3)	41 (69.5)	1 (8.3)
Emergent	63 (28.2)	13 (12.9)	12 (23.5)	37 (62.7)	1 (8.3)
Elective	18 (8.1)	8 (7.9)	6 (11.8)	4 (6.8)	0 (0)
Zone 4	33 (15)	26 (25.7)	4 (7.8)	0 (0)	3 (25)
Emergent	11 (4.9)	7 (6.9)	1 (2.0)	0 (0)	3 (25)
Elective	22 (9.9)	19 (18.8)	3 (5.9)	0 (0)	0 (0)
Zone 5	16 (7)	10 (9.9)	3 (5.9)	2 (3.4)	1 (8.3)
Emergent	6 (2.7)	2 (2.0)	1 (2.0)	2 (3.4)	1 (8.3)
Elective	10 (4.5)	8 (7.9)	2 (3.9)	0 (0)	0 (0)

Values are presented as n (%).

Nine patients had their aortic disruption discovered later after initial traumatism. These patients underwent an elective procedure.

In the septic group, there were 4 septic pseudoaneurysms, 3 aortobronchials fistulas, and 2 aortoesophageal fistulas. All these procedures were emergent with 11 aortic ruptures.

Distribution of proximal landing zones is shown in Table 1 and distal landing zones in Table E3.

For Zone 0, SAV debranching and endograft deployment was performed as a single-stage procedure for 13 patients. Five patients had only brachiocephalic trunk and left common carotid artery revascularizations.

For Zone 1, 16 patients had revascularizations of the left common carotid artery and of the LSA. Only 1 patient underwent a staged procedure.

For Zone 2, 20 patients had an LSA covering without revascularization. Revascularization was performed as a staged procedure in 5 cases.

Eight patients (3.6%) required coverage of the visceral trunks: 2 patients had CT coverage without prior revascularization and 6 patients underwent a single stage procedure with 4 revascularizations of both CT and SMA (2 patients also underwent renal bypass), 1 revascularization of the CT, and 1 revascularization of the SMA.

The mean number of stent-grafts per case was 1.25 ± 0.25 (range, 1-4). Mean stent-graft diameter was 34.8 ± 5.7 mm and mean aortic length covering was 168.0 ± 79.2 mm (range, 55-500 mm). Access locations included common femoral artery (211 patients; 94.6%), common iliac artery (8 cases; 3.6%), and abdominal aorta (4 cases; 1.8%). There were no intraoperative surgical conversions.

Intraoperative Complications

Intraoperative mortality was 1.3%. Three patients treated for a ruptured aortic aneurysm in zone 3 had a fatal cardiac evolution during TEVAR. Nonfatal complications are reported in Table E4. Multivariate analysis (Table 2) showed that the statistically significant factor of stent-graft migration in the cohort was the use of the TAG or C-Tag device (Gore Medical, Flagstaff, Ariz) ($P = .02$). There was trend toward a higher migration rate in patients who underwent TEVAR in zone 0, 1, or 2 ($P = .06$).

Early Outcome

Stroke, SCI, and death were reported for elective and nonelective TEVAR with respect to pathology and proximal landing zone (Table 3). Overall 30-day mortality was 11.7% (12.6% during the first half of the study, compared with a 10.8% 30-day mortality during the second half; $P = .83$). Multivariate analysis showed that the statistically significant factor of stroke was patients treated for an acute type B aortic dissection ($P = .03$) (Table 2).

Technical success was 96.9% (216 patients). Technical failures were related to 3 intraoperative deaths (cardiac related) and 4 deaths during the first postoperative day (1 ruptured degenerative aneurysm in zone 3, 1 elective degenerative aneurysm in zone 3, 1 type B acute aortic dissection in zone 2, and 1 aortobronchial fistulae).

There were important differences in the pathology-specific mortality. Aneurysm group mortality (18.8%) and traumatic group mortality (1.7%) were significantly different ($P < .01$). Comparison with dissection group mortality (9.8%) or septic group mortality did not disclose any

TABLE 2. Multivariate logistic regression analysis for predictors of early and long-term outcomes data

Variable	Hazard ratio	95% Confidence interval	*P* value
Device migration			
Tag or C-Tag*	23.78	1.43-396.46	.03
Proximal landing zone 0, 1, or 2	2.73	0.95-7.89	.06
Death			
Dialysis	10.13	1.27-80.63	.03
Chronic obstructive pulmonary disease	7.18	2.45-21.08	<.01
Emergent	4.44	1.53-12.90	<.01
Stroke			
Acute dissection	25.36	1.52-422.68	.03
Retrograde aortic dissection			
Proximal landing zone 0 or 1	16.35	3.20-83.44	<.01
Early reintervention			
Proximal landing zone 0 or 1	7.10	2.42-20.79	<.01
Late reintervention			
Chronic dissection	8.96	1.67-47.99	.01
Endoleak			
Proximal landing zone 5	0.09	0.01-1.10	.07
Major endoleak (Type I or III)			
Zenith TX2† device	12.25	0.97-154.79	.05

*Gore Medical, Flagstaff, Ariz. †Cook, Bloomington, Ind.

significant differences. For the aneurysm group, the mortality rate was significantly higher for pathologies requiring emergent (34.4%) interventions compared with elective procedures (11.6%) ($P = .006$). Postoperative causes of deaths are detailed in Table E5. With a 5.4% rate, multisystem organ failure (MSOF) was the principal cause of perioperative death. Statistical analysis could not demonstrate any significant risk factor for MSOF, but dialysis ($P = .03$), chronic obstructive pulmonary disease ($P < .01$), and emergent procedure ($P < .01$) were demonstrated as predictive factors for perioperative death (Table 2).

The median length of stay was 9 days (range, 1-108 days). Overall, 72% of patients underwent TEVAR without any complication or secondary intervention (Tables E6 and E7). Multivariate analysis showed that TEVAR procedure performed in zone 0 or 1 was the only predictive factor of early reintervention ($P < .001$) (Table 2).

Late Outcome

The median follow-up period was 29.9 months (range, 1.25-171.8). At 12, 36, and 60 months, follow-up was completed for 74%, 56%, and 43% of patients, respectively. Kaplan-Meier estimates of survival at 12, 36, 60, and 120 months were, respectively (% ± standard error), 82.0% ± 2.7%, 75.1% ± 3.3%, 73.8% ± 3.5%, and 68.3% ± 4.5% (see Figure 1, *A* and *B*, for confidence limits). Causes of death are listed in Table E8. At 1, 3, and 5 years, estimated survival rates were, respectively, 73.6% ± 4.6%; 58.2% ± 6.1%, and 58.2% ± 6.1% for the aneurysm group, 85.8% ± 5.0%, 81.3% ± 6.5%, and 73.9% ± 9.2% for the dissection group. No late death was observed in the traumatic group and long-term mortality remains at 1.7%. The septic group was too small to draw any firm conclusion. Kaplan-Meier estimates curves of

TABLE 3. Perioperative outcomes

Outcome	Stroke	Spinal cord ischemia	Mortality
All patients (n = 223)	10 (4.5)	6 (2.7)*	26 (11.7)
Emergent (n = 121)	4 (3.3)	1 (0.8)	17 (14.0)
Elective (n = 102)	6 (5.9)	5 (4.9)	9 (8.9)
Zone 0 (n = 17)	3 (17.6)	1 (5.9)	3 (17.6)
Zone 1 (n = 17)	2 (11.8)	0 (0)	2 (11.8)
Zone 2 (n = 59)	3 (5.1)	2 (3.4)	8 (13.6)
Zone 3 (n = 81)	2 (2.5)	1 (1.2)	8 (9.9)
Zone 4 (n = 33)	0 (0)	1 (3.0)	3 (9.1)
Zone 5 (n = 16)	0 (0)	1 (6.2)	2 (12.5)
Aneurysm group (n = 101)	4 (4.0)	4 (4.0)*	19 (18.8)
Emergent (n = 32)	0 (0)	1 (3.1)	11 (34.4)
Elective (n = 69)	4 (5.8)	3 (4.3)	8 (11.6)
Zone 0 (n = 10)	0 (0)	0 (0)	2 (20.0)
Zone 1 (n = 10)	1 (10.0)	0 (0)	2 (20.0)
Zone 2 (n = 24)	3 (12.5)	2 (8.3)	5 (20.8)
Zone 3 (n = 21)	0 (0)	0 (0)	6 (28.6)
Zone 4 (n = 26)	0 (0)	1 (3.8)	3 (11.5)
Zone 5 (n = 10)	0 (0)	1 (10)	1 (10)
Dissection group (n = 51)	5 (9.8)	2 (3.9)*	5 (9.8)
Acute dissection (n = 26)	3 (11.5)	0 (0)	4 (15.4)
Chronic dissection (n = 25)	2 (8.0)	2 (8.0)	1 (4.0)
Zone 0 (n = 6)	3 (50.0)	1 (16.7)	1 (16.7)
Zone 1 (n = 3)	0 (0)	0 (0)	0 (0)
Zone 2 (n = 17)	0 (0)	0 (0)	2 (11.8)
Zone 3 (n = 18)	2 (11.1)	1 (5.6)	1 (5.6)
Zone 4 (n = 4)	0 (0)	0 (0)	0 (0)
Zone 5 (n = 3)	0 (0)	0 (0)	1 (33.3)
Traumatic group (n = 59)	1 (1.7)	0 (0)	1 (1.7)
Emergent (n = 50)	1 (2.0)	0 (0)	1 (2.0)
Elective (n = 9)	0 (0)	0 (0)	0 (0)
Zone 0 (n = 0)	–	–	–
Zone 1 (n = 1)	1 (100)	0 (0)	0 (0)
Zone 2 (n = 15)	0 (0)	0 (0)	0 (0)
Zone 3 (n = 41)	0 (0)	0 (0)	1 (4.9)
Zone 4 (n = 0)	–	–	–
Zone 5 (n = 2)	0 (0)	0 (0)	0 (0)
Septic group (n = 12)	0 (0)	0 (0)	1 (8.3)
Emergent (n = 12)	0 (0)	0 (0)	1 (8.3)
Elective (n = 0)	–	–	–
Zone 0 (n = 1)	0 (0)	0 (0)	0 (0)
Zone 1 (n = 3)	0 (0)	0 (0)	0 (0)
Zone 2 (n = 3)	0 (0)	0 (0)	1 (33.3)
Zone 3 (n = 1)	0 (0)	0 (0)	0 (0)
Zone 4 (n = 3)	0 (0)	0 (0)	0 (0)
Zone 5 (n = 1)	0 (0)	0 (0)	0 (0)

Values are presented as n (%). *Spinal cord ischemia referred to permanent paraplegia (which was observed in 4 out of 6 patients: 3 aneurysms and 1 dissection) and transient paraplegia (observed in 1 aneurysm group patient and 1 dissection group patient).

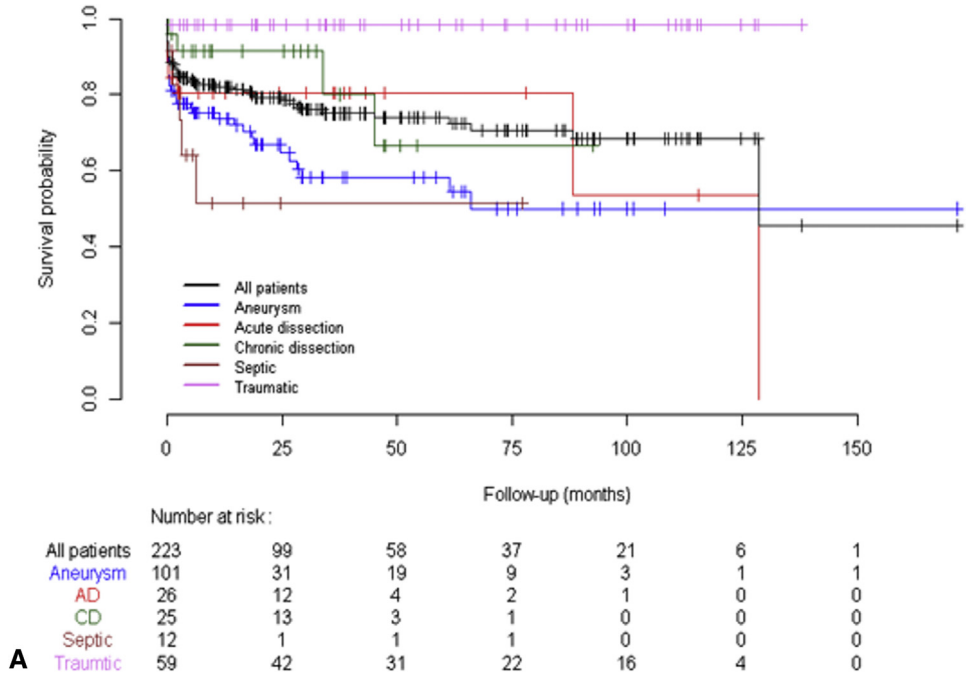

FIGURE 1. A, Product-limit survival estimates curves. B, Confidence limits of product-limit survival estimates curves. *AD*, Acute dissection; *CD*, chronic dissection.

survival are drawn in Figure 1. Estimated log-rank test showed statistically significant difference ($P < .0001$) between estimated survivals.

During follow-up, multivariate analysis did not disclose any predisposing factor of endoleak after TEVAR. There was a trend toward a lower endoleak rate in patients who underwent TEVAR in zone 5 ($P = .07$). Endoleak follow-up is reported in Table 4. Furthermore, occurrence of endoleak did not have any significant influence on survival ($P = .58$) (Figure E3, *A* and *B*).

During follow-up, 12.2% of the patients required reintervention (Tables 5 and 6). Multivariate analysis showed that patients treated for a chronic aortic dissection had a significant higher risk of late reintervention ($P = .001$) (Table 2). Kaplan-Meier estimates curves of survival-free from aortic complications (reinterventions and endoleaks) at 12, 36, 60, and 120 months were, respectively, 73% ± 3%, 64% ± 4%, 62% ± 4%, and 57% ± 5% (Figure E4, *A* and *B*).

DISCUSSION

In our experience, reported postoperative causes of death show that TEVAR remains a major procedure. Nevertheless, only 3 patients (1.3%) had a procedure-related death (2 neurologic and 1 retrograde aortic dissection [RAD]). One could argue that other postoperative cause of deaths (MSOF or cardiac, respiratory, or bowel ischemia) are mostly related to preexisting comorbidities that predisposed patients to organ failure. Mortality is clearly influenced by patient selection, with 84% of patients presenting American Society of Anesthesiologists (ASA) score ≥3 and 54% of emergent procedures.

This study demonstrated that outcomes after TEVAR were pathology specific. Statistical analysis found that early mortality rate for patients with aneursyms (18.8%) was higher than patients with traumatic aortic transection (1.7%) ($P < .01$). Comparison with the dissection group or septic group could not demonstrate any significant difference between the groups, but the aneurysm group early mortality seems to be higher than other groups' mortality (respectively, 9.8% and 8.3%). This difference is particularly obvious for traumatic ruptures. Clearly, with only 1 patient treated in both zone 0 and 1, procedures for traumatic aortic disruption are much less invasive than procedures for the aneurysm, dissection, or septic groups because aortic arch debranching for zones 0 and 1 were performed in 19.8%, 17.6%, and 33.3%, respectively. Nevertheless, log-rank tests show a significant higher survival for this group made up of patients with multiple acute comorbidities. For this specific pathology, TEVAR has changed short- and long-term prognosis, avoiding cardiopulmonary bypass for patients with cerebral or abdominal trauma. Perioperative mortality in the aneurysm group (18.8%) seems to be higher than previously reported mortality rates.[9-17] Composition of our aneurysm group may partially explain our results. With 83% of patients having an ASA score ≥3, including 24% of aortic rupture, our group has an important operative risk.[21] For example, 11 of 32 emergent patients died, representing 57.9% of the aneurysm

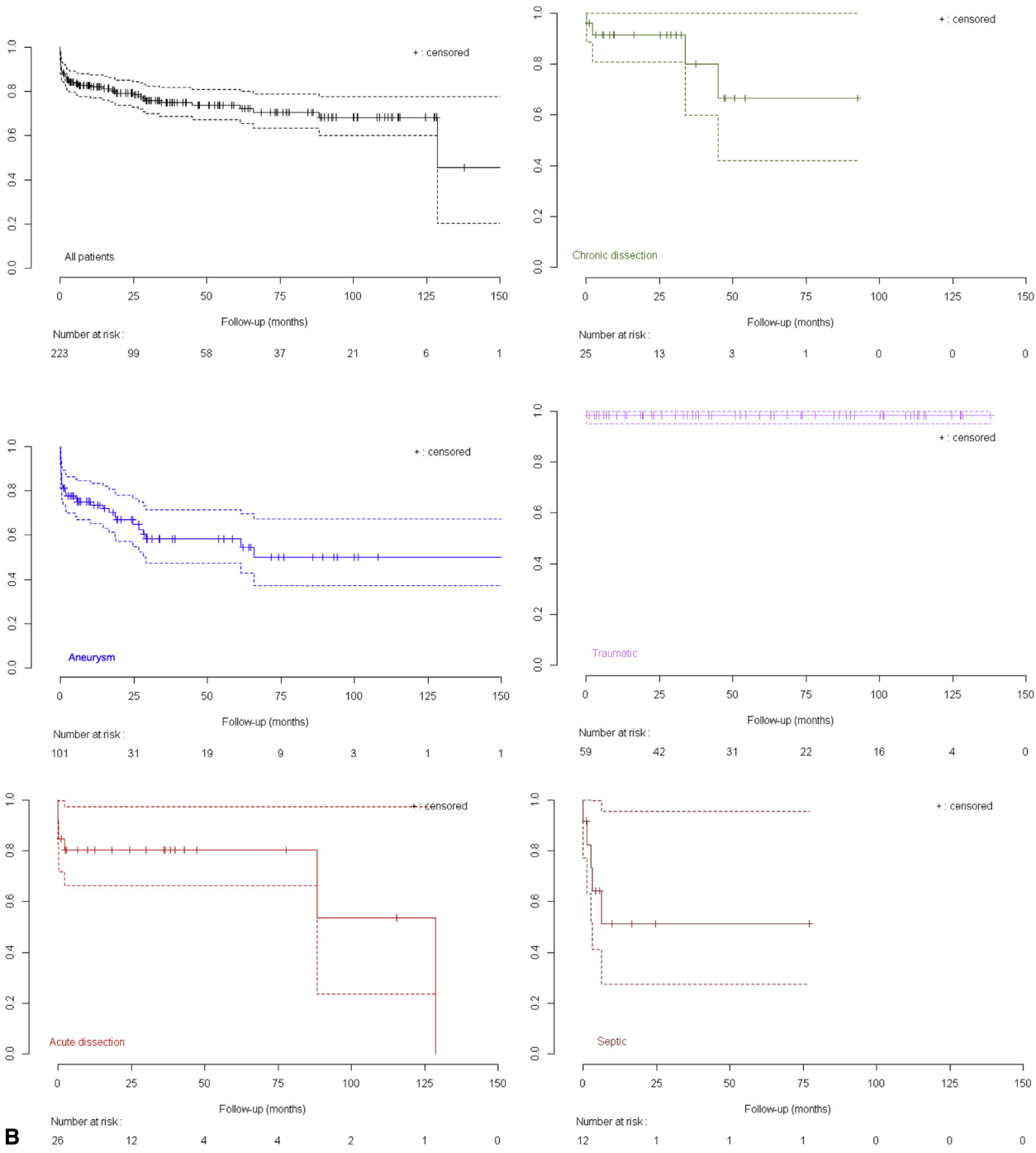

FIGURE 1. (*continued*).

mortality. In comparison, Leurs and colleagues[13] reported a 10.4% mortality rate for 58% of patients with an ASA score $\geq$3.

Even in cases where there was no statistical difference, perioperative mortality rate for all patients seems to be also higher for aortic arch disease treated with debranching techniques (11.8%-17.6%) than for patients treated in zone 3 or 4 (9.1% and 9.9%). This difference had been expected as the difference during traditional repair. The main reason is the anatomic challenge represented by SAV: During debranching or stent-graft deployment procedures, many issues may appear such as stroke, upper limb ischemia, or RAD.[22] Statistical analysis could not demonstrate any significant difference between different zone mortality, but it is probably due to the small number of patients. Complexity of procedures involving aortic arch is also demonstrated by

TABLE 4. Endoleak follow-up

	All patients (n = 197)	Aneurysm group (n = 82)	Dissection group (n = 46)	Traumatic group (n = 58)	Septic group (n = 11)
Patients presenting with any type of endoleak	36 (18.3)*	20 (25.6)*	14 (30.4)	0 (0)	2 (18.2)
Type I	21 (10.7)	9 (11.0)	10 (21.7)	0 (0)	2 (18.2)
Type Ia	15 (7.6)	6 (7.3)	7 (15.2)	0 (0)	2 (18.2)
Type Ib	6 (3.0)	3 (3.7)	3 (6.5)	0 (0)	0 (0)
Type II	11 (5.6)	7 (8.5)	4 (8.7)	0 (0)	0 (0)
Type III	3 (1.5)	3 (3.7)	0 (0)	0 (0)	0 (0)
Type V (endotension)	4 (2.0)	4 (4.9)	0 (0)	0 (0)	0 (0)

*Three patients presented 2 types of endoleak: 1 patient presented with 2 type-III endoleaks, 1 patient presented with a type Ib endoleak and an indeterminate endoleak, and 1 patient presented with a type II and a type Ia endoleak.

a higher migration rate in patients who underwent TEVAR in zone 0, 1, or 2, although it fell short of statistical significance ($P = .06$). However, statistical analysis showed that the C-Tag device was statistically associated with a higher migration rate ($P = .02$). The C-Tag release system, without bare-metal stent and proximal hooks, is probably an explanation of migration risk during TEVAR.

Neurologic morbidity in our study is quite similar to previously reported outcomes. SCI occurred during the perioperative period for 2.7% of patients. Literature showed similar results for 2.0% to 9.3% of the patients. Strokes occurred in 4.5% of patients. The literature shows similar results for 3% to 4.8% of patients.[9-17] Study of our cohort found that acute aortic dissection was significantly associated with a higher proportion of strokes ($P = .02$). It is probably because major fragility of the aortic wall in this indication can facilitate the extension of the dissection to SAV during wire manipulations, vessel clamping, or endograft deployment.

RAD is a serious complication after TEVAR. It has been reported in 1% to 2% of cases.[22-24] It is likely that RAD is more frequent after hybrid arch procedures[22-24] compared with single TEVAR. This was confirmed by multivariate analysis, which showed the significant predictor of RAD was TEVAR procedure performed in zone 0 or 1 ($P < .001$). During our TEVAR experience, we reported a total of 8 RADs (3.6%). This rate is only 2.7% during perioperative period. Patterson and colleagues[25] also reported recently a RAD risk during long-term follow-up with an increase incidence from 3.2% to 4.9% for acute type B aortic dissection and from 1.5% to 2.3% for chronic dissections. We recently modified our approach in zone 0 to avoid RAD: We used rapid pacing not only during stent-graft deployment but also during aortic crossclamping and declamping.

Logically, statistical analysis found that zones 0 and 1 were associated with a higher rate ($P < .001$) of early reintervention. Complexity of these procedures, including SAV debranching, is an additional source of adverse events (eg, RAD, bypass stenosis, or thrombosis) that could lead to reintervention. Long-term follow-up shows that a main reason of failure after TEVAR is the occurrence of major endoleak. The percentage of type I or III endoleak reported is very variable in publications.[9-17] Desai and colleagues[9] reported a major endoleak rate of 12.7%, including 67% requiring reintervention. However, in our series, the major endoleak rate was 10.3% (24 for 23 patients) and only 7.6% (15 patients) required a reintervention to treat the endoleak. Reintervention was not necessary in 56.4% of endoleaks (28.2% of spontaneous thrombosis and 28.2% are still being followed).

In the present study, the endoleak rate was higher in the dissection group (30.4%) than in the aneurysm group

TABLE 5. Reported indications for late reintervention

Indication for reintervention	All patients (n = 197)	Aneurysm group (n = 82)	Dissection group (n = 46)	Traumatic group (n = 58)	Septic group (n = 11)
Total patients	24 (12.2)	9 (11.0)*	10 (21.7)†	4 (6.9)	1 (9.1)
Aneurysmal expansion of aorta	1 (0.5)	0 (0)	1 (2.2)	0 (0)	0 (0)
Continued perfusion of distal false lumen	5 (2.5)	0 (0)	5 (10.9)	0 (0)	0 (0)
Endograft collapse	1 (0.5)	0 (0)	0 (0)	1 (1.7)	0 (0)
Type Ia endoleak	8 (4.1)	3 (3.7)	5 (10.9)	0 (0)	0 (0)
Type Ib endoleak	2 (1.0)	2 (3.8)	0 (0)	0 (0)	0 (0)
Type III endoleak	1 (0.5)	1 (1.2)	0 (0)	0 (0)	0 (0)
Other endoleak	2 (1.0)	2 (3.8)	0 (0)	0 (0)	0 (0)
Endograft infection	3 (1.5)	1 (1.2)	1 (2.2)	0 (0)	1 (9.1)
Peripheral	4 (2.0)	1 (1.2)	0 (0)	3 (5.2)	0 (0)

Values are presented as n (%). *One patient with a type Ib endoleak initially treated by additional stent-graft presented an indeterminate additional endoleak treated by open surgery. †Two patients underwent reoperation twice: One patient treated by open surgery for an abdominal aneurysm evolution of chronic dissection presented a type 1a endoleak treated by additional stent-graft. Another patient with an aneurysmal expansion of distal aorta due to false lumen perfusion had 2 additional procedures: 1 distal stent-graft deployment and 1 bronchial resection for an aortobronchial fistulae during follow-up.

TABLE 6. Late types of reintervention

Mode of reintervention	All patients (n = 197)	Aneurysm group (n = 82)	Dissection group (n = 46)	Traumatic group (n = 58)	Septic group (n = 11)
Total patients	24 (12.2)	9 (11.0)*	10 (21.7)†	4 (6.9)	1 (9.1)
Proximal/distal extension of stent-graft/additional stent-graft	14 (7.1)	5 (6.1)	9 (19.6)	0 (0)	0 (0)
Supra-aortic bypass	6 (3.0)	1 (1.2)‡	3 (6.5)‡	2 (3.4)	0 (0)
Open thoracic aortic surgery	6 (3.0)	4 (4.9)	0 (0)	1 (1.7)	1 (9.1)
Open abdominal aortic surgery	2 (1.0)	0 (0)	2 (4.3)	0 (0)	0 (0)
Peripheral stent	2 (1.0)	1 (1.2)	0 (0)	1 (1.7)	0 (0)

Values are presented as n (%). *One patient with a type Ib endoleak initially treated by additional stent-graft presented an indeterminate additional endoleak treated by open surgery. †Two patients underwent reoperation twice: One patient treated by open surgery for an abdominal aneurysm evolution of chronic dissection presented a type 1a endoleak treated by additional stent-graft. Another patient with an aneurysmal expansion of distal aorta due to false lumen perfusion had 2 additional procedures: 1 distal stent-graft deployment and 1 bronchial resection for an aortobronchial fistulae during follow-up. ‡Debranching procedure for additional stent-graft.

(25.6%) or the septic group (18.2%), without statistical significance. In the traumatic group, during follow-up, no endoleak was observed. We did not observed device issues. Causes of reintervention were related to degeneration of the landing zones over time. This is corroborated by the not-uncommon risk of distal or proximal aortic reintervention after open repair for degenerative aneurysm or chronic dissection, whereas aortic reintervention after open repair is clearly uncommon after traumatic transection or acute type B dissection. One explanation is that aneurysm or dissection pathologies are chronic and involved a long aortic segment; it is sometimes necessary to pile up stent-grafts. On the other hand, traumatic lesions appear most of the time on healthy aortas and concern a very isolated portion. But the traumatic group had specific complications related to these healthy aortas: Small aortic diameter and aortic arch major angulation may induce endograft collapse.[26-28]

Type I endoleak appears to be more frequent in chronic dissection (9 out of 25 patients; 36%) than in acute dissection (1 out of 26 patients; 4%) ($P < .05$). The explanation is that many acute dissections (19 out of 26; 73%) were not treated for aneurysmal expansion (but rather, rupture or malperfusion). For these patients, type I endoleak is exceptional: Aortic diameter remains stable and provides a long neck for endograft fixation. Furthermore, without aortic enlargement, the aortic wall remains superimposed on the stent-graft, without possibility for blood flow to perfuse the aneurysm.

For aneurysms, surprisingly, endoleaks appear to be more frequent in the elective group (15 out of 69 patients; 22%) than emergent aneurysms (5 out of 32 patients; 16%). Nevertheless, this discrepancy was not statistically significant ($P = .47$). Multivariate analysis could not demonstrate any significant risk factors in subpopulations.

Reintervention has been an ongoing issue with TEVAR. In the present study, secondary procedure rate was 13.2% during follow-up, but this rate was statistically different between groups. The dissection group presented a reintervention rate of 23.9%, which was higher than other groups. Analysis found that chronic aortic dissection was associated with a higher risk of late reintervention ($P = .01$).

Several limitations of this study should be considered. Results must be interpreted within the context of it being a retrospective study design without a control group to compare against. A direct consequence is the impossibility to conclude any superiority or noninferiority between our cohort of patients treated by stent-graft and patients treated by conventional surgery. Furthermore, the single-institution nature of this study limited the cohort sample size to 223 patients. Unfortunately, the great diversity of pathologies, modalities of care, and landing zones revealed an important heterogeneity of our cohort, which is composed of relatively small groups. This limitation leads to underpowering of the study and outcome results, leading to the possibility of mistakes during analysis.

CONCLUSIONS

Long-term outcomes show an important heterogeneity of reported results with specific long-term complications. TEVAR performed for aortic traumatic rupture has totally changed the prognosis of these patients. Results for patients treated for a thoracic aortic aneurysm are acceptable despite important perioperative mortality, probably due to our rate of emergent procedures. Aortic dissection remains a challenging pathology that should be considered as a chronic pathology with an important reintervention rate. Septic aortic diseases remain extremely rare, with an important mortality rate and the place of TEVAR is still under evaluation.[29,30] We now consider TEVAR the first choice in the treatment of descending thoracic aorta aneurysms, aortic disruptions, and thoracic aortic dissections.

Conventional surgical repair of aortic arch remains substantial and we also consider hybrid TEVAR as the first choice in high-risk patients or after previous aortic surgery despite early and late specific adverse events.

Improvement of endovascular devices may increase flexibility and conformability of stent-grafts to decrease morbidity and mortality rates.[31] Better patient selection through multidisciplinary discussions may also improve TEVAR outcomes, selecting patients for open repair, endovascular repair, or hybrid techniques (such as debranching or frozen elephant trunk). Frozen elephant trunk has the

potential to further minimize morbidity by avoiding the second-step procedure of conventional surgical repair and the theoretical advantage over hybrid repair of preventing the occurrence of type Ia endoleak and RAD. The present optimized selection criteria for use of frozen elephant trunk in elective cases has been associated with a remarkable decrease in mortality compared with initial experiences.[32] Total endovascular repair of the aortic arch remains an experimental technique but should be assessed in the future. Several comparative studies are mandatory to determine indications of each technique.

Conflict of Interest Statement

Authors have nothing to disclose with regard to commercial support.

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Key Words: TEVAR, endovascular, aorta, stent-graft, aortic arch

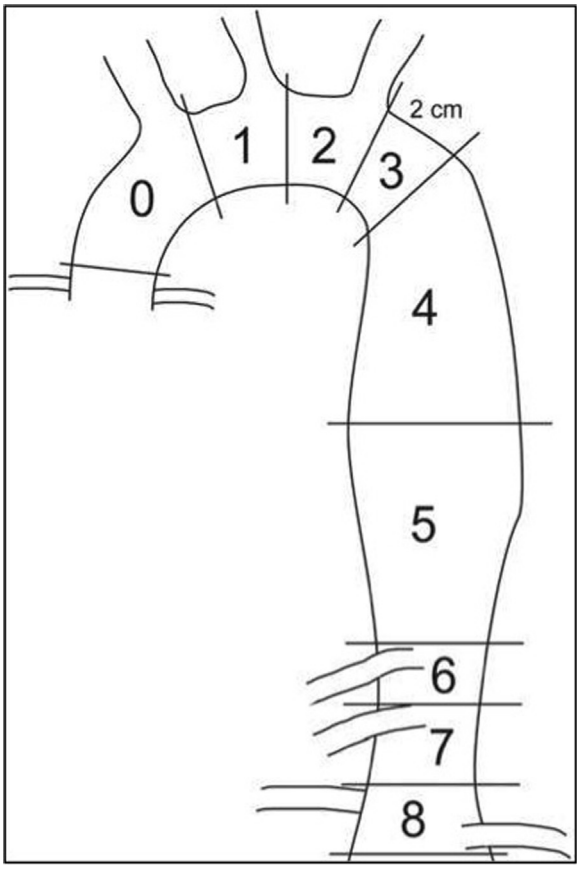


FIGURE E1. Landing zones described by Fillinger and the Society for Vascular Surgery.[18]

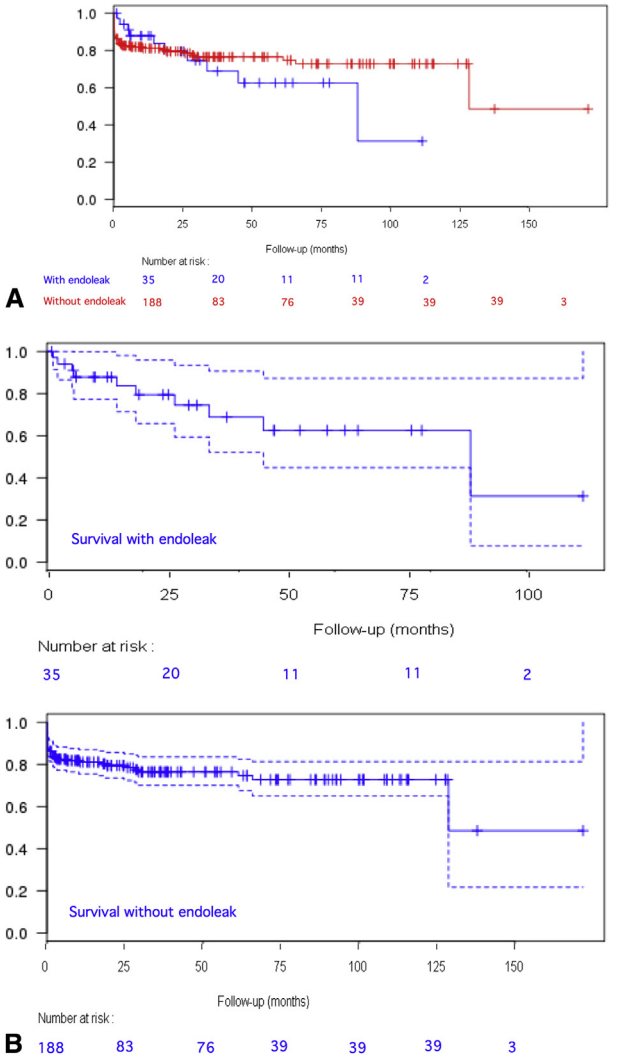


FIGURE E3. A, Product-limit survival with and without endoleak estimate curves. B, Confidence limits of product-limit survival with and without endoleak estimate curves.

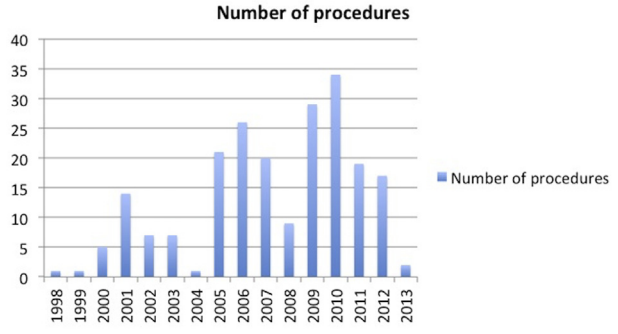
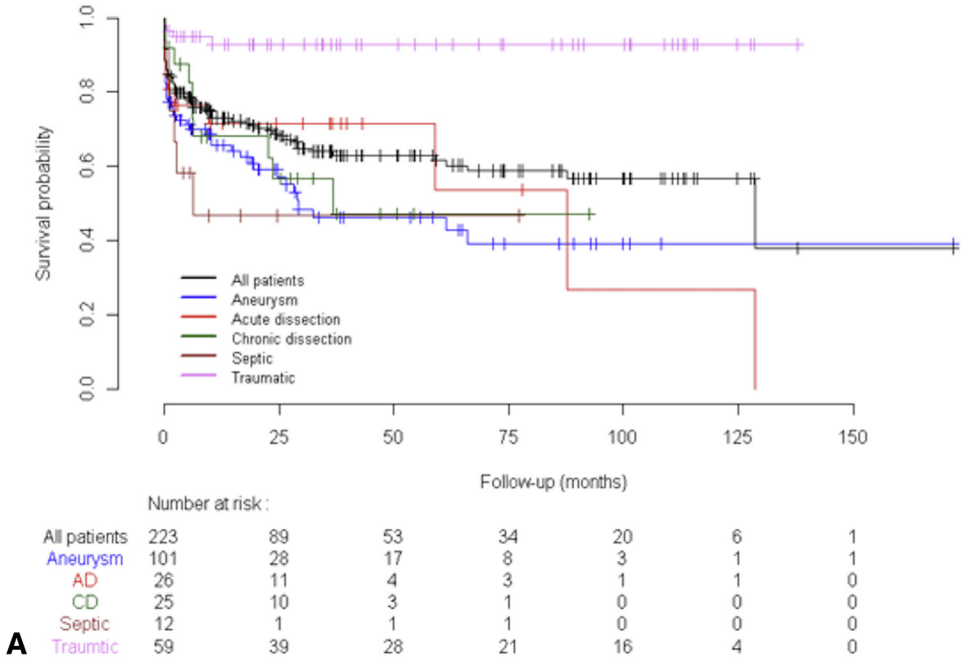


FIGURE E2. Number of procedures by year.



A

Number at risk :

All patients	223	89	53	34	20	6	1
Aneurysm	101	28	17	8	3	1	1
AD	26	11	4	3	1	1	0
CD	25	10	3	1	0	0	0
Septic	12	1	1	1	0	0	0
Traumtic	59	39	28	21	16	4	0

FIGURE E4. A, Product-limit survival free from aortic complications estimate curves. B, Confidence limits of product-limit survival free from aortic complications estimate curves. *AD*, Acute dissection; *CD*, chronic dissection.

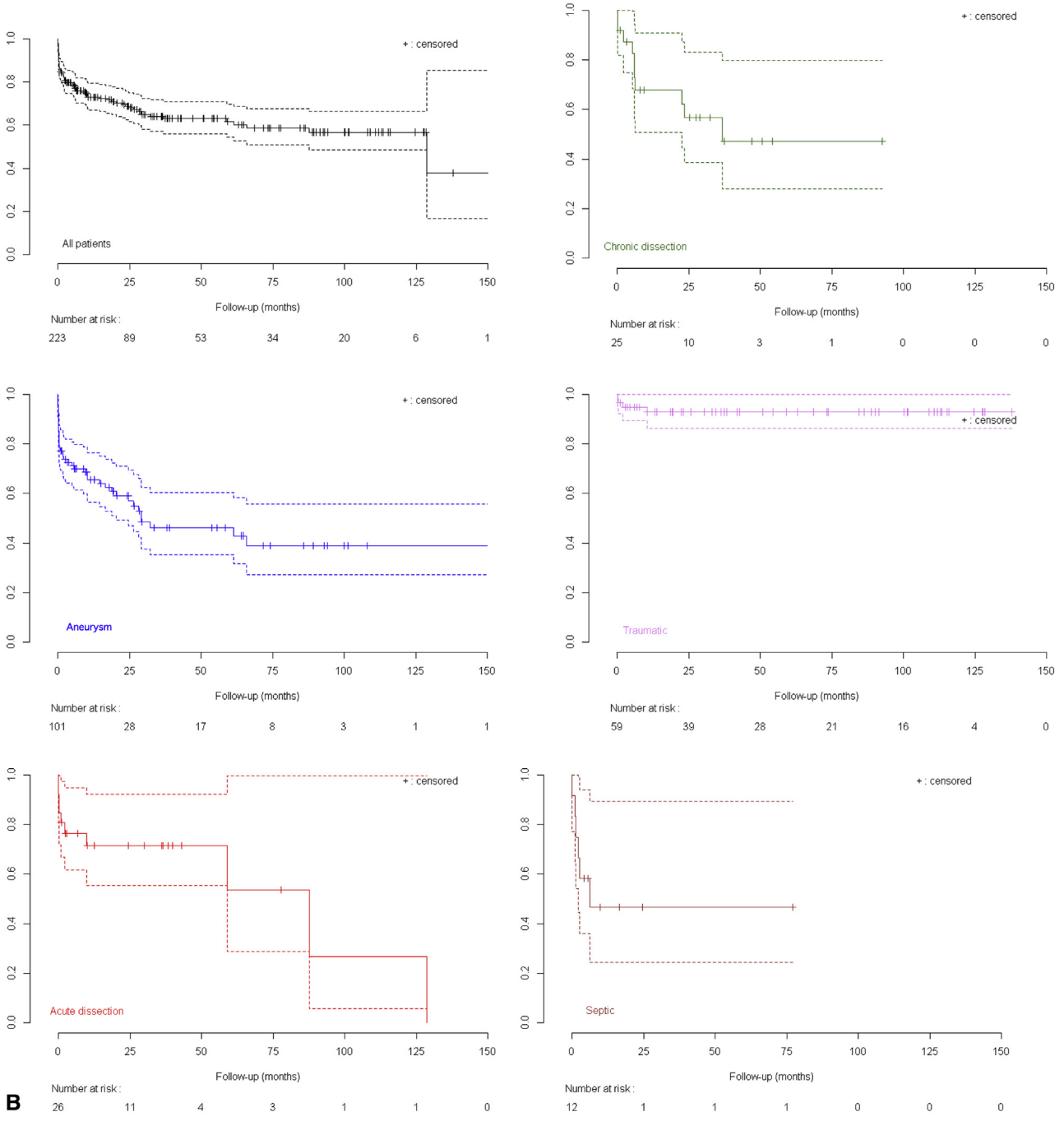


FIGURE E4. (*continued*).

TABLE E1. Stent-grafts used

Endograft	Total	Aneurysm group	Dissection group	Traumatic group	Septic group
Total	280 (100)	139 (50)	68 (24)	61 (22)	12 (4)
	[32, 23, 67, 98, 40, 20]	[18, 13, 30, 33, 32, 13]	[13, 6, 19, 21, 5, 4]	[0, 1, 15, 43, 0, 2]	[1, 3, 3, 1, 3, 1]
TAG*	127 (45)	73 (26)	27 (10)	22 (8)	5 (2)
	[9, 7, 32, 46, 21, 12]	[8, 5, 16, 18, 18, 8]	[1, 2, 10, 12, 1, 1]	[0, 0, 5, 15, 0, 2]	[0, 0, 1, 1, 2, 1]
Valiant-Captivia†	45 (16)	15 (5)	17 (6)	10 (4)	3 (1)
	[9, 4, 11, 17, 3, 1]	[3, 1, 2, 6, 3, 0]	[6, 1, 5, 4, 0, 1]	[0, 1, 2, 7, 0, 0]	[0, 1, 2, 0, 0, 0]
C-TAG*	37 (13)	22 (8)	10 (4)	4 (1)	1 (0)
	[5, 9, 7, 11, 4, 1]	[2, 6, 4, 6, 3, 1]	[3, 3, 3, 1, 0, 0]	[0, 0, 0, 4, 0, 0]	[0, 0, 0, 0, 1, 0]
Valiant†	35 (13)	13 (5)	8 (3)	11 (4)	3 (1)
	[9, 3, 10, 12, 1, 0]	[5, 1, 5, 1, 1, 0]	[3, 0, 1, 4, 0, 0]	[0, 0, 4, 7, 0, 0]	[1, 2, 0, 0, 0, 0]
Talent†	19 (7)	5 (2)	2 (1)	12 (4)	0 (0)
	[0, 0, 5, 8, 6, 0]	[0, 0, 1, 0, 4, 0]	[0, 0, 0, 0, 2, 0]	[0, 0, 4, 8, 0, 0]	[0, 0, 0, 0, 0, 0]
Zenith TX2‡	11 (4)	8 (3)	3 (1)	0 (0)	0 (0)
	[0, 0, 2, 2, 5, 2]	[0, 0, 2, 2, 3, 1]	[0, 0, 0, 0, 2, 1]	[0, 0, 0, 0, 0, 0]	[0, 0, 0, 0, 0, 0]
Zenith TX1‡	6 (2)	3 (1)	1 (0)	2 (1)	0 (0)
	[0, 0, 0, 2, 0, 4]	[0, 0, 0, 0, 0, 3]	[0, 0, 0, 0, 0, 1]	[0, 0, 0, 2, 0, 0]	[0, 0, 0, 0, 0, 0]

Values are presented as n (%), [Z0, Z1, Z2, Z3, Z4, Z5]. *Gore Medical, Flagstaff, Ariz. †Medtronic, Santa Rosa, Calif. ‡Cook, Bloomington, Ind.

TABLE E2. Patient demographic characteristics

Characteristic	Total (n = 223)	Aneurysm group (n = 101)	Dissection group (n = 51)	Traumatic group (n = 59)	Septic group (n = 12)	*P* value
Men	179 (80)	82 (81)	42 (82)	47 (80)	5 (62)	<.001
Mean age (y)	62.7 ± 17.9	71.0 ± 12.5	66.3 ± 10.4	43.2 ± 17.2	74.1 ± 12.5	<.001
Hypertension	143 (64)	79 (78)	46 (90)	8 (14)	10 (83)	<.001
Smokers	62 (28)	36 (36)	12 (24)	11 (19)	3 (25)	<.001
Diabetes	17 (8)	10 (10)	3 (6)	4 (7)	0 (0)	.95
Dyslipidemia	95 (43)	53 (52)	26 (51)	10 (17)	6 (50)	<.001
Coronary artery disease	58 (26)	40 (40)	10 (20)	3 (5)	5 (42)	<.001
Coronary artery revascularization	26 (12)	18 (18)	3 (6)	3 (5)	2 (17)	.08
Coronary artery bypass	6 (3)	5 (5)	0 (0)	1 (2)	0 (0)	.63
Ejection function <60%	32 (14)	25 (24)	4 (8)	1 (2)	3 (25)	<.001
Arrhythmia	31 (14)	19 (19)	8 (16)	2 (3)	2 (17)	<.001
Pacemaker	4 (2)	3 (3)	0 (0)	0 (0)	1 (8)	.19
Stroke	19 (9)	12 (12)	5 (10)	0 (0)	2 (17)	.02
Peripheral arterial disease	34 (15)	27 (27)	5 (10)	1 (2)	1 (8)	<.001
Renal insufficiency	28 (13)	21 (21)	2 (4)	2 (3)	3 (25)	<.001
Dialysis	5 (2)	5 (5)	0 (0)	0 (0)	0 (0)	.42
COPD	38 (17)	29 (28)	6 (12)	1 (2)	2 (17)	<.001
Restrictive respiratory insufficiency	4 (2)	2 (2)	2 (4)	0 (0)	0 (0)	.22
Neoplasia	34 (15)	18 (18)	9 (18)	2 (3)	5 (42)	.02
Previous aortic surgery	48 (22)	32 (32)	15 (29)	0 (0)	1 (8)	<.001
Valvular heart replacement	11 (5)	2 (2)	9 (18)	0 (0)	0 (0)	<.001
Ascending aorta	13 (6)	2 (2)	11 (22)	0 (0)	0 (0)	<.001
Descending aorta	12 (5)	11 (11)	0 (0)	0 (0)	1 (8)	.013
Abdominal aorta	29 (13)	23 (23)	5 (10)	0 (0)	1 (8)	<.001
ASA score						
≥3	187 (84)	83 (82)	45 (88)	47 (80)	12 (100)	.67
3	86 (39)	39 (39)	26 (51)	20 (34)	1 (8)	
4	83 (37)	37 (37)	17 (33)	22 (37)	7 (58)	
5	18 (8)	7 (7)	2 (4)	5 (8)	4 (33)	

Values are presented as n (%) or mean ± standard derivation. *COPD*, Chronic obstructive pulmonary disease; *ASA*, American Society of Anesthesiologists.

TABLE E3. Distribution of distal landing zone

Zone	Total (n = 223)	Aneurysm group (n = 101)	Dissection group (n = 51)	Traumatic group (n = 59)	Septic group (n = 12)
Zone 4	17 (8)	59 (58.4)	42 (82.4)	57 (96.6)	10 (83.3)
Emergent	6 (2.7)	19 (18.8)	24 (47.1)	48 (81.4)	10 (83.3)
Elective	11 (4.9)	40 (39.6)	18 (35.3)	9 (15.3)	0 (0)
Zone 5	17 (8)	35 (34.7)	8 (15.7)	2 (3.4)	2 (16.7)
Emergent	6 (2.7)	10 (9.9)	1 (2.0)	2 (3.4)	2 (16.7)
Elective	11 (4.9)	25 (24.8)	7 (13.7)	0 (0)	0 (0)
Zone 6	59 (26)	3 (3.0)	0 (0)	0 (0)	0 (0)
Emergent	29 (13)	2 (2.0)	0 (0)	0 (0)	0 (0)
Elective	30 (13.4)	1 (1.0)	0 (0)	0 (0)	0 (0)
Zone 7	81 (36)	2 (2.0)	0 (0)	0 (0)	0 (0)
Emergent	63 (28.2)	1 (1.0)	0 (0)	0 (0)	0 (0)
Elective	18 (8.1)	1 (1.0)	0 (0)	0 (0)	0 (0)
Zone 8	33 (15)	0 (0)	0 (0)	0 (0)	0 (0)
Emergent	11 (4.9)	0 (0)	0 (0)	0 (0)	0 (0)
Elective	22 (9.9)	0 (0)	0 (0)	0 (0)	0 (0)
Zone 9	16 (7)	2 (2.0)	1 (2.0)	0 (0)	0 (0)
Emergent	6 (2.7)	0 (0)	1 (2.0)	0 (0)	0 (0)
Elective	10 (4.5)	2 (2.0)	0 (0)	0 (0)	0 (0)

Values are presented as n (%).

TABLE E4. Nonfatal intraoperative complications

Complication	Result
Iliofemoral access trauma	11 (4.9)
Endovascular treatment	7 (3.1)
Open surgery treatment	4 (1.8)
Endograft migration	19 (8.5)
Distal migration	8 (3.6)
Supra-aortic trunks covering	11 (4.9)
Manual balloon traction	3 (1.3)
Endovascular treatment	5 (2.2)
Open surgery treatment	2 (0.9)
Surveillance	1 (0.4)
Intraoperative rupture	1 (0.4)

Values are presented as n (%).

TABLE E5. Postoperative cause of death

Cause of death	All patients (n = 223)	Aneurysm group (n = 101)	Dissection group (n = 51)	Traumatic group (n = 59)	Septic group (n = 12)
Multisystem organ failure	12 (5.4)	8 (7.9)	4 (7.8)	0 (0)	0 (0)
Cardiac	5 (2.2)	4 (4.0)	0 (0)	0 (0)	1 (8.3)
Respiratory	3 (1.3)	3 (3.0)	0 (0)	0 (0)	0 (0)
Bowel ischemia	3 (1.3)	2 (2.0)	1 (2.0)	0 (0)	0 (0)
Neurologic	2 (0.9)	1 (1.0)	0 (0)	1 (1.7)	0 (0)
Retrograde aortic dissection	1 (0.4)	1 (1.0)	0 (0)	0 (0)	0 (0)

Values are presented as n (%).

TABLE E6. Postoperative moderate to severe complications

Complication	All patients (n = 223)	Aneurysm group (n = 101)	Dissection group (n = 51)	Traumatic group (n = 59)	Septic group (n = 12)
None	161 (72.2)	65 (64.4)	36 (70.6)	51 (86.4)	9 (75)
Respiratory	26 (11.7)	10 (9.9)	4 (7.8)	8 (13.6)	2 (16.7)
Hemodialysis	20 (9.0)	11 (10.9)	6 (11.8)	3 (5.1)	0 (0)
Cardiac	15 (6.7)	8 (7.9)	2 (3.9)	3 (5.1)	2 (16.7)
Stroke	10 (4.5)	4 (4.0)	5 (9.8)	1 (1.7)	0 (0)
Wound	7 (3.1)	4 (4.0)	3 (5.9)	0 (0)	0 (0)
Spinal cord ischemia	6 (2.7)	4 (4.0)	2 (3.9)	0 (0)	0 (0)
Bowel ischemia	6 (2.7)	3 (3.0)	3 (5.9)	0 (0)	0 (0)
Retrograde aortic dissection	6 (2.7)	5 (5.0)	1 (2.0)	0 (0)	0 (0)
Limb ischemia	5 (2.2)	3 (3.0)	2 (3.9)	0 (0)	0 (0)
Hemothorax	4 (1.8)	2 (2.0)	2 (3.9)	0 (0)	0 (0)
Endograft collapse	1 (0.4)	0 (0)	0 (0)	1 (1.7)	0 (0)

Values are presented as n (%).

TABLE E7. Postoperative type of early reintervention

Type of reintervention	All patients (n = 223)	Aneurysm group (n = 101)	Dissection group (n = 51)	Traumatic group (n = 59)	Septic group (n = 12)
Aortic	12 (5.4)	7 (6.9)	3 (5.9)	2 (3.4)	0 (0)
Endovascular	7 (3.1)	3 (3.0)	2 (3.9)	2 (3.4)	0 (0)
Open surgery	5 (2.2)	4 (4.0)	1 (2.0)	0 (0)	0 (0)
Supra aortic trunks	4 (1.8)	2 (2.0)	1 (2.0)	0 (0)	1 (8.3)
Endovascular	2 (0.9)	1 (1.0)	0 (0)	0 (0)	1 (8.3)
Open surgery	2 (0.9)	1 (1.0)	1 (2.0)	0 (0)	0 (0)
Wound	4 (1.8)	3 (3.0)	1 (2.0)	0 (0)	0 (0)
Bowel resection	4 (1.8)	3 (3.0)	1 (2.0)	0 (0)	0 (0)
Hemothorax	4 (1.8)	2 (2.0)	2 (3.9)	0 (0)	0 (0)
Cervical hematoma	3 (1.3)	1 (1.0)	2 (3.9)	0 (0)	0 (0)
Minor amputation	1 (0.4)	0 (0)	1 (2.0)	0 (0)	0 (0)

Values are presented as n (%).

TABLE E8. Late cause of death

Cause of death	All patients (n = 197)	Aneurysm group (n = 82)	Dissection group (n = 46)	Traumatic group (n = 58)	Septic group (n = 11)
Neoplasia	9 (4.6)	5 (6.1)	1 (2.2)	0 (0)	3 (27.3)
Respiratory	3 (1.5)	2 (2.4)	1 (2.2)	0 (0)	0 (0)
Endograft infection	3 (1.5)	1 (1.2)	0 (0)	0 (0)	2 (18.2)
Retrograde aortic dissection	2 (1.0)	1 (1.2)	1 (2.2)	0 (0)	0 (0)
Gastrointestinal	2 (1.0)	1 (1.2)	1 (2.2)	0 (0)	0 (0)
Stroke	1 (0.5)	1 (1.2)	0 (0)	0 (0)	0 (0)
Acute limb ischemia	1 (0.5)	1 (1.2)	0 (0)	0 (0)	0 (0)
AAA rupture	1 (0.5)	1 (1.2)	0 (0)	0 (0)	0 (0)
TAA rupture	1 (0.5)	1 (1.2)	0 (0)	0 (0)	0 (0)
Cardiac	1 (0.5)	0 (0)	1 (2.2)	0 (0)	0 (0)
MSOF	1 (0.5)	0 (0)	1 (2.2)	0 (0)	0 (0)
Unknown	2 (1.0)	2 (2.4)	0 (0)	0 (0)	0 (0)

Values are presented as n (%). *AAA*, Abdominal aortic aneurysm; *TAA*, thoracic aortic aneurysm; *MSOF*, multisystem organ failure.