Outcomes after endovascular versus open thoracoabdominal aortic aneurysm repair: A population-based study

Rodolfo V. Rocha, MD,[a] Thomas F. Lindsay, MDCM,[b] Peter C. Austin, PhD,[c] Mohammed Al-Omran, MD, MSc,[d] Thomas L. Forbes, MD,[b] Douglas S. Lee, MD, PhD,[c,e] and Maral Ouzounian, MD, PhD[a]

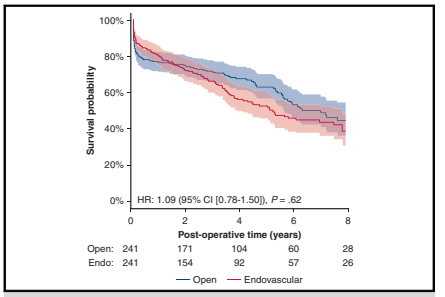
ABSTRACT

Objective: We sought to determine the early and late outcomes of endovascular versus open thoracoabdominal aortic aneurysm repair.

Methods: We performed a multicenter population-based study across the province of Ontario, Canada, from 2006 to 2017. The primary end point was mortality. Secondary end points were time to first event of a composite of mortality, permanent spinal cord injury, permanent dialysis, and stroke, the individual end points of the composite, patient disposition at discharge, hospital length of stay, myocardial infarction, and secondary procedures at follow-up.

Results: A total of 664 adults undergoing surgical repair of a thoracoabdominal aortic aneurysm (endovascular: n = 303 [45.5%] vs open: n = 361 [54.5%]) were identified using an algorithm of administrative codes validated against the operative records. Propensity score matching resulted in 241 patient pairs. Endovascular repairs increased during the study and currently comprise more than 50% of total repairs. In the matched sample, open repair was associated with a higher incidence of in-hospital death (17.4% vs 10.8%, $P = .04$), complications (26.1% vs 17.4%, $P = .02$), discharge to rehabilitation facilities (18.7% vs 10.0%, $P = .02$), and longer length of stay (12 [7-21] vs 6 [3-13] days, $P < .01$). Long-term mortality was not significantly different (hazard ratio, 1.09; 95% confidence interval, 0.78-1.50), nor were the other secondary end points, with the exception of secondary procedures, which were higher in the endovascular group (hazard ratio, 2.64; 95% confidence interval, 1.54-4.55). At 8 years, overall survival was 41.3% versus 44.6% after endovascular and open repair ($P = .62$).

Conclusions: Endovascular repair was associated with improved early outcomes but higher rates of secondary procedures after discharge. Long-term survival after thoracoabdominal aortic aneurysm repair is poor and independent of repair technique. (J Thorac Cardiovasc Surg 2021;161:516-27)



Long-term survival is similar and poor after endovascular or open TAAA repair in a population-based study from Ontario.

Central Message

Endovascular repair is associated with improved early outcomes but higher rates of secondary procedures. Long-term survival is poor and independent of repair technique.

Perspective

Compared with the open approach, endovascular TAAA repair was associated with decreased early mortality, length of stay, and postoperative adverse events, but an increased rate of secondary procedures. Long-term survival and adverse events were similar. TAAA repair remains challenging with considerable associated morbidity and mortality.

See Commentaries on pages 528, 530, and 532.

From the [a]Divisions of Cardiovascular Surgery; [b]Vascular Surgery; and [c]Cardiology, Peter Munk Cardiac Centre, University Health Network, University of Toronto, Toronto, Ontario, Canada; [c]Cardiovascular Program, Institute for Clinical Evaluative Sciences, Toronto, Ontario, Canada; [d]Division of Vascular Surgery, St Michael's Hospital, University of Toronto, Toronto, Ontario, Canada.

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Address for reprints: Maral Ouzounian, MD, PhD, Toronto General Hospital, 200 Elizabeth St 4N-464, Toronto, Ontario, Canada M5G2C4 (E-mail: maral.ouzounian@uhn.ca).

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Abbreviations and Acronyms

AAA = abdominal aortic aneurysm
CI = confidence interval
EVAR = endovascular aortic repair
HR = hazard ratio
ICES = Institute for Clinical Evaluative Sciences
MI = myocardial infarction
PSM = propensity score matching
SMD = standardized mean difference
TAAA = thoracoabdominal aortic aneurysm
TALE = thoracoabdominal aortic aneurysm life-altering event

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The introduction of endovascular aortic repair (EVAR) revolutionized the surgical treatment of infrarenal abdominal aortic aneurysms (AAAs).[1] Multiple randomized controlled trials[2,3] and meta-analyses[4,5] have demonstrated an early survival benefit with EVAR compared with open surgical repair, and despite the increased rate of late reintervention,[6] EVAR is firmly established as a less-invasive treatment option for patients with AAAs. A recent Medicare study from the United States reported that more than 60% of patients undergoing AAA repair were treated with EVAR.[7] The Society for Vascular Surgery recommends EVAR as first-line treatment of infrarenal AAA, reserving open surgical repair for patients who do not meet the anatomic requirements for EVAR.[8]

It is unclear whether this paradigm shift toward an endovascular approach will be replicated in more anatomically complex areas of the aorta. Thoracoabdominal aortic aneurysms (TAAAs) span the thorax and abdomen, and supply vital branches to the spinal cord, visceral, renal, and limb arteries. Surgical repair of TAAA remains one of the most complex operations in medicine, with substantial rates of morbidity and mortality even in centers of excellence.[9,10] Endovascular TAAA repair was described in 2001 by Chuter and colleagues[11] and was initially offered only to patients at prohibitively high surgical risk. Over the past decade, endovascular TAAA repair has become increasingly offered to a wider cohort of patients with TAAA with promising results.[12,13]

Unlike the robust infrarenal AAA literature, there is a paucity of outcome data after TAAA repair, for a variety of reasons: (1) There is a lower incidence and prevalence of patients with TAAA; (2) only a limited proportion of patients with the pathology are offered repair; and (3) most published reports are from highly specialized centers that excel in one or the other approach. No randomized controlled trials comparing endovascular with open TAAA repair are under way or have been published, and given the multitude of anatomic and technical considerations, a randomized study is unlikely. Only a few comparative observational studies have been published with heterogeneous unmatched populations and only reporting short-term results.[14] The objective of this study was to investigate the early and late outcomes of endovascular versus open TAAA repair at a population-based level in the province of Ontario.

MATERIALS AND METHODS

Study Design

Patients undergoing first-time endovascular (branched and fenestrated) or open TAAA repair performed in Ontario, Canada, from January 2006 to February 2017, were identified through administrative databases housed at the Institute for Clinical Evaluative Sciences (ICES). ICES is a not-for-profit research institute encompassing a community of research, data and clinical experts, and a secure and accessible array of Ontario's health-related data. ICES is a prescribed-entity of Ontario that houses all healthcare mandatory databases, in addition to government databases (eg, census, economic indicators).

An algorithm developed to accurately identify patients receiving each therapy was validated with each operative report and was found to have a positive predictive value of 0.90 and 0.98 for endovascular and open TAAA cases, respectively.[15] Table E1 presents the coding strategy for each type of repair, which used a combination of administrative codes from the Canadian Classification of Health Intervention, the Ontario Health Insurance Plan (physicians' billings), and the 10th revision of the International Statistical Classification of Diseases and Related Health Problems. Patients undergoing hybrid TAAA repair were not included in the study, because cases with both endovascular and open TAAA repair administrative codes on the same index procedure date were excluded. We were not able to differentiate branched and fenestrated endografts from parallel grafts.

Data Collection and Definitions

Patients were identified through the validated coding strategy at ICES, which houses all healthcare information delivered in a single-payer system in the province of Ontario. Patients were then linked through a unique patient identifier to 4 additional administrative databases (Table E2). The databases used in our study are administrative databases with mandatory data collection as per the Ontario Ministry of Health and Long-Term Care. Therefore, there were no missing data, and completeness of follow-up was 100%. We excluded patients who did not have a valid health card number because they could not be followed after surgery. The use of data in this project was authorized under section 45 of Ontario's Personal Health Information Protection Act, which does not require review by a Research Ethics Board.

Study End Points

The primary end point for this study was mortality. The complications associated with the greatest life-altering impact after TAAA repair are

death, permanent spinal cord injury, permanent dialysis, and stroke, which together have been termed "thoracoabdominal aortic aneurysm life-altering events" (TALEs). In addition to time to first event of a TALE, the individual end points of the composite were also analyzed, along with the following end points of interest: patient disposition at discharge, hospital length of stay, myocardial infarction (MI), and secondary procedures on the thoracoabdominal aorta and its branches after discharge. Validated diagnostic codes were used to identify spinal cord injury (permanent paraplegia), MI, and stroke.[16-18] All administrative database codes used in the study are reported in Table E3.

Statistical Analysis

Continuous variables are reported as mean ± standard deviation or median (interquartile range). Categoric variables are reported as frequencies and percentages. All statistical analyses were conducted using R (version 3.5.1; R Foundation for Statistical Computing, Vienna, Austria). As per ICES policy, all cells with fewer than 6 events are presented as "5 or less" to avoid potential patient identification.

Propensity score matching (PSM) was used to adjust for prespecified clinically relevant baseline characteristics that were potentially confounding. Before matching, baseline demographics were compared using the Mann–Whitney *U* test (continuous data) or chi-square statistic (categoric data), as appropriate. Propensity scores were calculated using logistic regression models using all baseline variables listed in Table 1. Patients undergoing endovascular TAAA repair were matched 1:1 without replacement with patients undergoing open TAAA repair, using the logit of the propensity score with a caliper of 0.2 of the standard deviation of the logit of the propensity score.[19] Standardized mean differences (SMDs) were determined to compare baseline characteristics; an SMD less than 0.1 was considered as an acceptable indicator of balance between groups.[20]

In-hospital outcomes were compared between the matched cohorts using the McNemar test for categoric outcomes and paired *t* tests for continuous outcomes. We conducted 3 prespecified subgroup analyses to determine the effect of surgical approach (endovascular vs open TAAA repair) on in-hospital mortality according to patients' age at the time of intervention, surgical era, and institutional TAAA volume.

Time-to-event analyses were performed using Cox proportional hazards models to compare long-term freedom from TALE and mortality between groups. Long-term rates of permanent dialysis, permanent paraplegia, stroke, MI, and secondary procedures on the thoracoabdominal aorta or its branches were compared using a cause-specific hazard model adjusting for death as a competing risk.[21] Hazard ratios (HRs) were determined up to 1, 3, 5, and 8 years after surgery, with robust sandwich-type variance estimator for the matched groups to account for clustering in matched pairs when analyzing the PSM groups.[22] Kaplan–Meier survival functions were estimated for survival and freedom from TALE. Equality of these was tested using the stratified log-rank test.[23] Cumulative incidence functions were generated for the secondary outcomes of interest. Equality of these cumulative incidence functions was tested using the method suggested by Austin and Fine.[24]

Survival After Thoracoabdominal Aortic Aneurysm Repair Versus General Population

Each endovascular and open TAAA repair case was matched to 5 random individuals from Ontario, based on age, sex, income quintile, and area of residence, to compare survival after TAAA repair with a control population of those not undergoing TAAA repair. Kaplan–Meier curves of long-term survival and Cox proportional hazards models were generated for patients undergoing TAAA repair compared with the general population.

RESULTS

Population Characteristics

A total of 664 patients who underwent TAAA repair between January 2006 and February 2017 in the province of Ontario were identified and comprise the study population (endovascular: n = 303 [45.5%] vs open: n = 361 [54.5%]). The total procedure volume for TAAA repair increased over the 11 years of study, with a greater increase in endovascular repairs, which comprised more than 50% of all TAAA repairs performed in Ontario since 2011 (Figure 1).

Baseline demographics before and after PSM are described in Table 1. After PSM, 241 patient pairs were formed with SMDs all less than 0.1, indicating adequately balanced groups.

In-Hospital Outcomes

In-hospital outcomes before and after propensity matching are presented in Table 2. In the matched sample, open TAAA repair was associated with a higher incidence of in-hospital death (17.4% vs 10.8%, $P = .04$), TALE (26.1% vs 17.4%, $P = .02$), discharge to rehabilitation facilities (18.7% vs 10.0%, $P = .02$), and longer median length of stay (12 [7-21] vs 6 [3-13] days, $P < .01$). There were no differences in rates of dialysis, stroke, permanent paraplegia, or MI between the groups.

Subgroup analyses of in-hospital mortality stratified by age, surgical era, and institutional volume are presented in Table E4. The early mortality benefit of endovascular repair was most pronounced in patients who were aged more than 80 years, who underwent repair in the early surgical era, and who were in low-volume TAAA institutions. Outcomes were similar between groups in the more recent surgical era (2012-2017: endovascular: 10.0% vs open: 13.4%, $P = .45$), in high-volume institutions (endovascular: 9.7% vs open: 13.8%, $P = .25$), and for patients undergoing urgent repair (endovascular: 32.6% vs open 43.9%, $P = .38$). Institutional volume appeared to have a substantial effect on mortality after open TAAA repair (low volume: 36.0% vs high 13.8%, $P < .01$) but not endovascular repair (low volume: 12.6% vs high: 9.7%, $P = .52$).

Long-Term Survival

The early survival benefit of endovascular repair diminished over time and was no longer significant at 1 year after repair (Table 3). At a median follow-up of 3.5 years (interquartile range, 1.6-6.2 years), there was no significant difference in the primary outcome of all-cause mortality after endovascular versus open TAAA repair (HR, 1.09; 95% confidence interval [CI], 0.78-1.50; $P = .62$) (Figure 2, *A*). Compared with the general

TABLE 1. Baseline characteristics of endovascular versus open thoracoabdominal aortic aneurysm repair before and after propensity score matching*

Variable	Pre-PSM			Post-PSM		
	Open N = 361	Endovascular N = 303	*P* value	Open N = 241	Endovascular N = 241	*P* value
Age, mean ± SD	67.5 ± 11.2	71.3 ± 9.4	<.01	69.4 ± 10.0	70.1 ± 9.6	.75
Male, n (%)	269 (74.5)	205 (67.7)	.06	172 (71.4)	167 (69.3)	.37
Income quintile, n (%)			.38			.73
1	66 (18.3)	72 (23.8)		48 (19.9)	49 (20.3)	
2	77 (21.3)	61 (20.1)		50 (20.7)	46 (19.1)	
3	72 (19.9)	58 (19.1)		42 (17.4)	48 (19.9)	
4	71 (19.7)	62 (20.5)		49 (20.3)	51 (21.2)	
5	75 (20.8)	50 (16.5)		52 (21.6)	47 (19.5)	
Rural, n (%)	46 (12.7)	45 (14.9)	.50	30 (12.4)	37 (15.4)	.50
Year, n (%)			<.01			.68
2006-2008	99 (27.4)	39 (12.9)		45 (18.7)	39 (16.2)	
2009-2011	89 (24.7)	71 (23.4)		62 (25.7)	62 (25.7)	
2012-2014	93 (25.8)	105 (34.7)		74 (30.7)	77 (32.0)	
2015-2017	80 (22.2)	88 (29.0)		60 (24.9)	63 (26.1)	
Urgent or emergency, n (%)	96 (26.6)	47 (15.5)	<.01	41 (17.0)	46 (19.1)	.71
Charlson index, mean ± SD	2.4 ± 1.7	2.2 ± 1.7	.21	2.3 ± 1.7	2.3 ± 1.6	.30
Congestive heart failure, n (%)	59 (16.3)	50 (16.5)	1.00	36 (14.9)	37 (15.4)	1.00
Chronic kidney disease, n (%)	56 (15.5)	40 (13.2)	.44	33 (13.7)	33 (13.7)	1.00
Hypertension, n (%)	316 (87.5)	268 (88.4)	.81	210 (87.1)	211 (87.6)	1.00
Diabetes, n (%)	87 (24.1)	80 (26.4)	.53	55 (22.8)	59 (24.5)	.76
Chronic obstructive pulmonary disease, n (%)	138 (38.2)	126 (41.6)	.38	94 (39.0)	94 (39.0)	1.00
Coronary artery disease, n (%)	96 (26.6)	70 (23.1)	.32	59 (24.5)	55 (22.8)	.83
Previous stroke, n (%)	20 (5.5)	16 (5.3)	1.00	10 (4.1)	12 (5.0)	.92
Peripheral artery disease, n (%)	36 (10.0)	48 (15.8)	.03	27 (11.2)	30 (12.4)	.67
Previous coronary revascularization, n (%)	38 (10.5)	27 (8.9)	.51	24 (10.0)	22 (9.1)	.86
Previous malignancy, n (%)	42 (11.6)	43 (14.2)	.35	32 (13.3)	34 (14.1)	.92
Medications, n (%)						
Statins	205 (56.8)	193 (63.7)	.08	146 (60.6)	146 (60.6)	1.00
Beta-blockers	175 (48.5)	156 (51.5)	.48	117 (48.5)	119 (49.4)	.86
ACE inhibitors	210 (58.2)	184 (60.7)	.53	147 (61.0)	144 (59.8)	.72
Antiplatelets	46 (12.7)	52 (17.2)	.12	32 (13.3)	34 (14.1)	.86
Anticoagulants	38 (10.5)	35 (11.6)	.71	25 (10.4)	26 (10.8)	1.00
DOAC	7 (1.9)	11 (3.6)	.23	6 (2.5)	8 (3.3)	.80
Hospital admissions past 3 y (median [IQR])	1 [0-1]	1 [0-2]	<.01	1 [0-1]	1 [0-2]	.13
ED visits past 3 y (median [IQR])	2 [1-3]	2 [1-4]	.10	1 [1-3]	2 [1-4]	.27
Clinics visits previous 1 y, mean ± SD	15.4 ± 9.4	15.3 ± 9.2	.85	14.9 ± 8.5	15.3 ± 9.7	.32
Overall hospital volume,†n (%)			.10			.86
Low	7 (1.9)	≤5 (NA)		≤5 (NA)	≤5 (NA)	
Medium	36 (10.0)	(43-47)‡ (NA)		(22-25)‡ (NA)	(26-30)‡ (NA)	
High	318 (88.1)	253 (83.5)		213 (88.4)	209 (86.7)	

PSM, Propensity score matching; *SD*, standard deviation; *ACE*, angiotensin-converting enzyme; *DOAC*, direct oral anticoagulant; *ED*, emergency department; *IQR*, interquartile range; *NA*, not available. *All variables listed in Table 1 were used in the propensity score calculation. †All 17 centers that performed TAAA during our study period were divided into terciles (high, intermediate, and low volume) according to the number of TAAAs performed. ‡Required to avoid reidentification.

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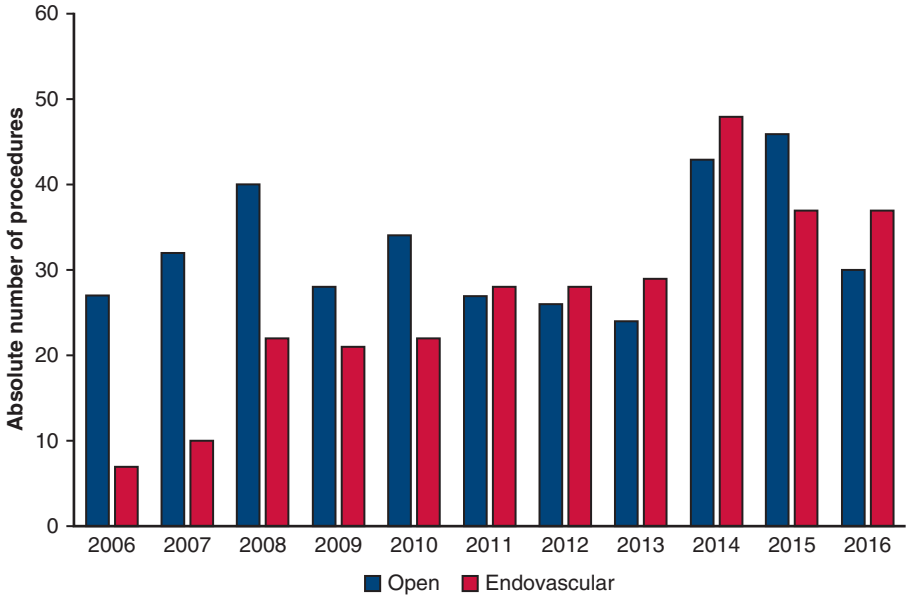

FIGURE 1. Endovascular and open thoracoabdominal repair volume in Ontario.

population, survival after endovascular or open TAAA repair was worse and the excess mortality worsened over time when matched for age, sex, income quintile, and area of residency (HR, 3.99; 95% CI, 3.40-4.70; $P < .01$) (Figure 2, *B*).

Long-Term Outcomes

Long-term outcomes of each of the clinical end points of interest are reported in Table 3. Freedom from TALE, the composite end point, was not different between the 2 groups (HR, 1.02; 95% CI, 0.76-1.38; $P = .88$) (Figure 3). The cumulative incidence of permanent dialysis, stroke, and MI was comparable between the 2 groups (Table 3 and Figures E1-E3). The long-term permanent paraplegia cases (events after discharge) were 5 or less in each group. The cumulative incidence of this outcome could not be described, because there would be a risk of patient reidentification.

Secondary Procedures on the Thoracoabdominal Aorta or Its Branches

Secondary procedures on the thoracoabdominal aorta or its branches were significantly higher after endovascular repair compared with open repair after a median of 3.5 years of follow-up (HR, 2.64; 95% CI, 1.54-4.55; $P < .01$) (Figure 4 and Table E5). At 8 years, 23.3% of the endovascular group required 1 or more secondary procedures on the thoracoabdominal aorta or its branches compared with 12.5% of the open group ($P < .01$, Table 3). After propensity matching, 90 secondary procedures were required in 55 patients after endovascular TAAA repair, with interventions of the aorta (15.6%), visceral (32.2%), and leg vessels (16.7%) being the most common. In the open repair group, 45 secondary procedures were required in 34 patients, with a large portion of these being abdominal wall procedures (35.6%), followed by intervention on leg arteries (22.2%). Few patients required any secondary procedures on the thoracic or abdominal aorta in the open TAAA repair group. Secondary procedures on the thoracoabdominal aorta or its branches were associated with a 30-day mortality of 14% versus 13% in the endovascular and open groups.

DISCUSSION

TAAAs are potentially fatal, and guidelines uniformly recommend repair of TAAA that meets treatment indications to prevent rupture.[8] Repair of TAAA has evolved to include an endovascular approach based on reported midterm results and data extrapolated from the infrarenal AAA literature. However, long-term outcomes after TAAA repair in relation to the general population are not known, with sparse data comparing endovascular repair with conventional surgery.[14] This study is the largest population-based comparative analysis reporting differences in early and late outcomes after endovascular versus open TAAA repair (Figure 5). Open repair was associated with significantly higher in-hospital mortality, longer length of stay, greater disposition to rehabilitation facilities, and more life-altering adverse events. There was no difference between the 2 groups in rates of paraplegia, dialysis, or stroke during index hospitalization. Long-term adverse events were similar between the 2 groups, with the exception of secondary procedures, which were higher after endovascular repair.

TABLE 2. In-hospital outcomes of endovascular versus open thoracoabdominal aortic aneurysm repair before and after propensity score matching

Outcome	Pre-PSM			Post-PSM		
	Open N = 361	Endovascular N = 303	*P* value	Open N = 241	Endovascular N = 241	*P* value
Death, n (%)	64 (17.7)	31 (10.2)	<.01	42 (17.4)	26 (10.8)	.04
Transient dialysis, n (%)	45 (12.5)	21 (6.9)	.02	27 (11.2)	20 (8.3)	.35
Permanent dialysis, n (%)	17 (4.7)	8 (2.6)	.22	14 (5.8)	≤5 (NA)	>.05
Permanent paraplegia, n (%)	13 (3.6)	13 (4.3)	.69	10 (4.1)	11 (4.6)	1.00
Stroke, n (%)	18 (5.0)	18 (5.9)	.61	10 (4.1)	12 (5.0)	.83
TALE, n (%)	93 (25.8)	54 (17.8)	.02	63 (26.1)	42 (17.4)	.02
MI, n (%)	22 (6.1)	18 (5.9)	1.00	13 (5.4)	17 (7.1)	.57
Disposition*			<.01			.02
Home, n (%)	160 (44.3)	163 (53.8)		110 (45.6)	133 (55.2)	
Home with services, n (%)	71 (19.7)	78 (25.7)		44 (18.3)	58 (24.1)	
Rehabilitation institution, n (%)	66 (18.3)	31 (10.2)		45 (18.7)	24 (10.0)	
Length of stay (median [IQR])	13 [7-22]	6 [3-13]	<.01	12 [7-21]	6 [3-13]	<.01

PSM, Propensity score matching; *NA*, not available; *TALE*, thoracoabdominal aortic aneurysm life-altering events; *MI*, myocardial infarction; *IQR*, interquartile range; *SD*, standard deviation. *Excluding patients who died in hospital.

Rates of TAAA repair increased throughout the study period, with a greater proportion of TAAAs being treated with an endovascular approach, particularly at higher-volume hospitals. This shift toward more endovascular repair has been observed in both the thoracic[25] and infrarenal aortas.[26] Several factors preclude the widespread use of endovascular repair in patients with TAAA: A high level of surgical expertise is required,[27] the use of "off-the-shelf" endografts for urgent cases is uncommon, most devices are custom-made and require individual special access permission from Health Canada,[28] and the device cost is high.[29] Furthermore, the long-term results of endovascular TAAA repair, particularly for patients with chronic dissection or connective tissue disorders, are unknown.

Our results are similar to those in a recent meta-analysis of comparative studies, in which endovascular TAAA was associated with a lower risk of complications in unadjusted analysis.[14] Most comparative studies used population-based data without a validated coding strategy, reported only early outcomes, and did not attempt to adjust for potential confounders.[14]

The in-hospital mortality for both endovascular and open repair observed in this study was higher than in single-center reports,[9,10,12,13] highlighting the differences between population-based outcomes and reports from highly specialized centers. In a recent population-based study from Germany, in-hospital mortality was 23.9% for open TAAA repair and 10.6% for endovascular TAAA repair.[30] Rigberg and colleagues[31] reported 30-day mortality rates of 19.2% and 48.4% after elective and ruptured open TAAA repair in California, respectively. Similar to our findings, Marzelle and colleagues[32] reported an in-hospital mortality of 11.9% for endovascular TAAA repair in patients enrolled in the multicenter WINDOWS trial.

The volume-outcome relationship has been observed repeatedly for complex surgical procedures, including TAAA repair. In an early report of TAAA repair in 1542 patients in the United States, mortality was 22.3%, and both hospital and surgeon volume were significant predictors of mortality.[33] Median annual TAAA repair volumes for high-volume surgeons and hospitals were only 7 and 12, respectively, with an associated reduction in mortality of 58% and 42% compared with low-volume surgeons and centers. More recently, Moulakakis and colleagues[34] observed an inverse association between in-hospital mortality and institutional volume in a meta-analysis of 30 studies of open TAAA repair ($P = .005$). Our study confirms these findings and the effect of institutional volume on outcomes after TAAA repair, particularly in the open surgical cohort. We hypothesize that the observed improvement in mortality after open repair in the recent era reflects greater centralization of these patients. Given the clear volume-outcome relationship for complex procedures including TAAA repair (particularly open repair), we suspect that this is the reason why the outcome gap between the 2 procedures narrowed over time. These data support regionalization of care to high-volume centers.

The early benefit of endovascular TAAA repair did not translate into improved survival or freedom from adverse events at 1 year. A similar loss of the early survival benefit has been observed with EVAR.[4] The long-term survival observed in our study is far lower than in the general population and lower than what is reported after AAA repair, likely reflecting the high burden of atherosclerosis and

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TABLE 3. Long-term outcomes after endovascular versus open thoracoabdominal aortic aneurysm repair before and after propensity score matching

	Pre-PSM			Post-PSM		
	Open N = 361	Endovascular N = 303		Open N = 241	Endovascular N = 241	
Outcome	% probabilities (95% CI)	% probabilities (95% CI)	*P* value	% probabilities (95% CI)	% probabilities (95% CI)	*P* value
Survival						
1 y	77.8 (73.7-82.2)	79.5 (75.1-84.2)	.48	77.2 (72.1-82.7)	80.5 (76.6-85.7)	.30
3 y	72.0 (67.5-76.8)	67.8 (62.6-73.4)	.45	70.5 (65.0-76.6)	67.5 (61.7-73.9)	1.00
5 y	63.8 (58.7-69.3)	55.3 (49.4-62.0)	.12	63.2 (56.9-70.1)	54.4 (47.9-61.9)	.73
8 y	48.1 (42.0-55.0)	41.7 (34.6-50.4)	.17	44.6 (37.6-55.3)	41.3 (33.6-50.7)	.62
Freedom from TALE						
1 y	69.5 (64.9-74.4)	71.6 (66.7-76.9)	.44	68.0 (62.4-74.2)	72.6 (67.2-78.5)	.34
3 y	64.0 (59.2-69.2)	61.1 (55.7-66.9)	.74	61.5 (55.6-68.0)	60.2 (54.2-66.9)	.81
5 y	55.9 (50.7-61.6)	48.1 (42.2-54.8)	.26	53.9 (47.6-61.0)	47.3 (40.8-54.8)	.88
8 y	39.9 (34.2-46.5)	34.9 (28.2-43.3)	.32	35.6 (28.3-44.9)	34.8 (27.6-43.8)	.88
Dialysis						
1 y	6.1 (3.6-8.6)	5.1 (2.6-7.6)	.54	7.1 (3.8-10.3)	5.1 (2.3-8.0)	.23
3 y	8.1 (5.2-10.9)	6.3 (3.5-9.1)	.40	8.7 (5.0-12.4)	6.7 (3.4-10.0)	.35
5 y	10.7 (7.2-14.2)	8.1 (4.7-11.5)	.31	11.5 (7.0-16.0)	8.7 (4.8-12.6)	.31
8 y	15.5 (10.8-20.1)	9.3 (5.0-13.7)	.10	14.6 (7.8-20.4)	10.3 (5.3-15.2)	.30
Stroke						
1 y	5.3 (3.0-7.6)	7.7 (4.7-10.8)	.13	4.6 (1.9-7.2)	6.3 (3.2-9.5)	.25
3 y	6.3 (3.7-8.9)	9.9 (6.4-13.4)	.12	5.6 (2.6-8.6)	9.0 (5.2-12.8)	.13
5 y	7.2 (4.4-10.1)	12.5 (8.2-16.7)	.06	6.4 (3.1-9.8)	11.2 (6.7-15.7)	.10
8 y	7.8 (4.7-10.9)	14.8 (9.6-20.1)	.03	7.7 (3.5-12.0)	13.7 (8.1-19.4)	.11
Secondary procedures*						
1 y	3.4 (1.5-5.3)	11.4 (7.2-15.6)	<.01	3.0 (0.8-5.3)	10.5 (6.5-14.4)	<.01
3 y	6.5 (3.8-9.2)	16.4 (11.9-20.8)	<.01	6.3 (2.9-9.7)	16.7 (11.7-21.8)	<.01
5 y	8.2 (5.1-11.4)	20.8 (15.5-26.0)	<.01	7.9 (3.9-11.9)	21.2 (15.3-27.2)	<.01
8 y	13.5 (8.6-18.3)	23.9 (18.0-29.7)	<.01	12.5 (6.0-18.9)	23.3 (16.8-29.7)	<.01
MI						
1 y	30.7 (25.9-35.5)	26.8 (21.6-31.9)	.11	29.0 (23.2-34.7)	28.9 (23.1-34.6)	.77
3 y	39.5 (34.2-44.7)	35.8 (30.0-41.6)	.22	37.1 (30.7-43.5)	38.9 (32.3-45.4)	.71
5 y	42.2 (36.8-47.7)	39.1 (33.0-45.3)	.23	39.3 (32.6-46.0)	41.9 (35.3-48.5)	.79
8 y	51.3 (45.1-57.5)	41.0 (34.5-47.4)	.06	50.5 (41.0-59.1)	43.8 (36.7-51.0)	.58

PSM, propensity score matching; *CI*, Confidence interval; *TALE*, thoracoabdominal aortic aneurysm life-altering events; *MI*, myocardial infarction. *Secondary procedures on the thoracic or abdominal aorta, abdominal arteries, pelvic vessels, leg arteries, and redo open or endovascular TAAA.

comorbidities in this patient population. In our series, only 50% of patients were alive and free from permanent paraplegia, permanent dialysis, or stroke at 5-year follow-up, regardless of type of repair. Further study is required to determine the reasons for this observed excess mortality and how to improve long-term outcomes after TAAA repair.

Secondary procedures were significantly higher in the endovascular group and predominantly related to aortic and visceral arteries compared with abdominal wall procedures (hernia repair) in the open TAAA repair group. This is consistent with Schermerhorn and colleagues,[35] who reported a higher aneurysm-related reintervention rate after EVAR compared with open AAA repair and more laparotomies and readmissions in the open cohort. The high rate of reintervention after endovascular TAAA repair has also been observed by Oderich and colleagues,[13] who reported that 29% of cases required reintervention at 5 years. The high mortality rate after secondary procedures may have contributed to the loss of the early survival benefit after endovascular TAAA repair observed in this study.

Study Strengths and Limitations

This is the largest multicenter population-based analysis of the short- and long-term outcomes of endovascular versus open TAAA repair. In contrast to highly specialized single-institution studies whose results may not be generalizable, this report provides the contemporary results of a ''real-world'' experience with TAAA repair. Unlike previously published population-based reports, this study is the first to use validated codes to identify repair technique in

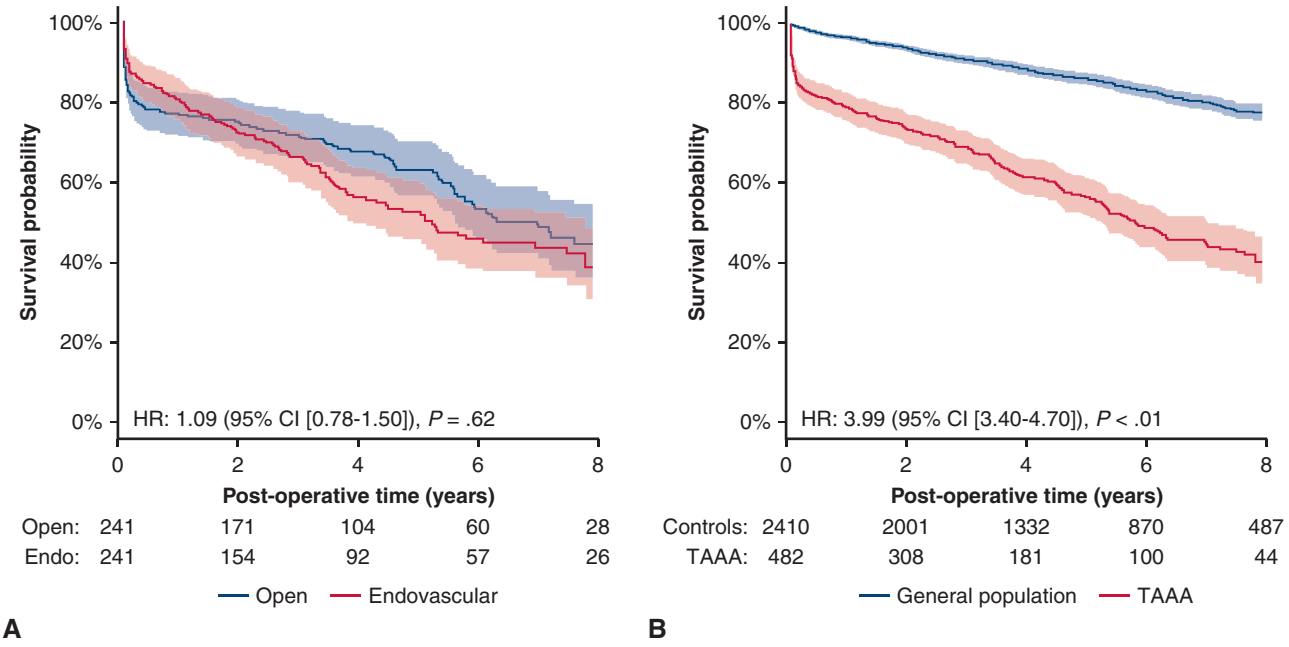


FIGURE 2. Long-term outcomes of patients undergoing TAAA repair. A, Kaplan–Meier curves for survival after endovascular versus open TAAA repair in a PSM cohort. B, Kaplan–Meier curves for survival after TAAA repair compared with general population controls. *HR*, Hazard ratio; *CI*, confidence interval; *TAAA*, thoracoabdominal aortic aneurysm. Controls are individuals without TAAA repair, matched for age, sex, income quintile, and area of residence.

patients undergoing TAAA repair. Despite the high predictive value of the codes used,[15-18] this report reflects the limitations of administrative data. Despite the use of PSM, important unmeasured confounders were not considered. Important prognostic factors, including patient characteristics (pulmonary function, ejection fraction, hereditary aortopathy) and TAAA characteristics

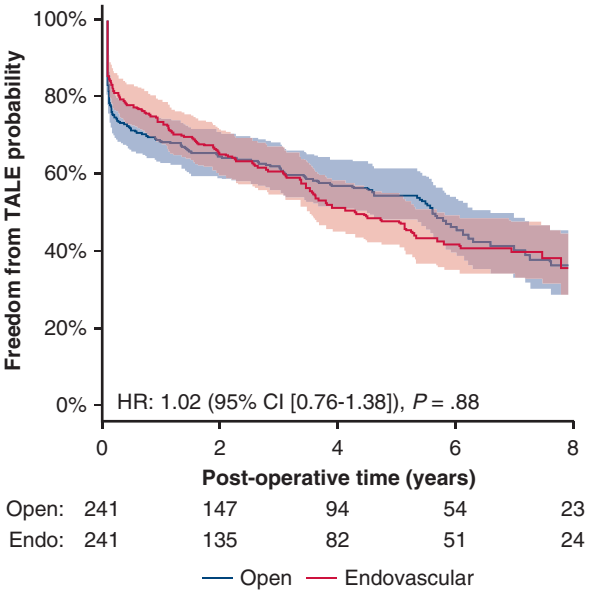


FIGURE 3. Kaplan–Meier curves for freedom from TALE after endovascular versus open TAAA repair in a PSM cohort. *HR*, Hazard ratio; *CI*, confidence interval; *TALE*, thoracoabdominal aortic aneurysm life-altering events.

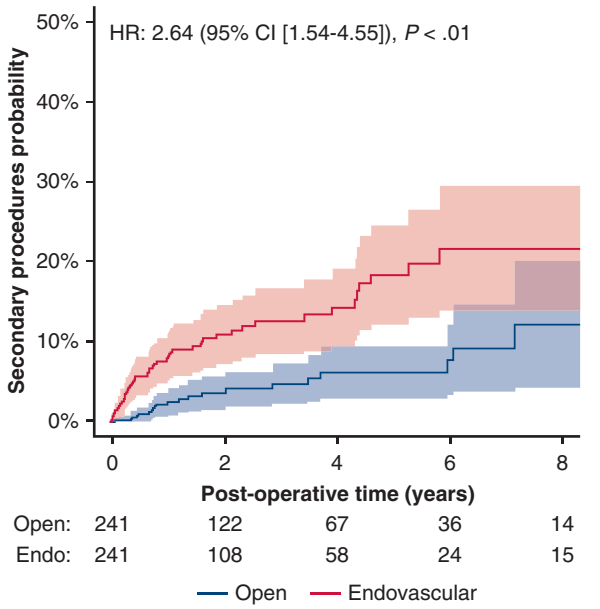
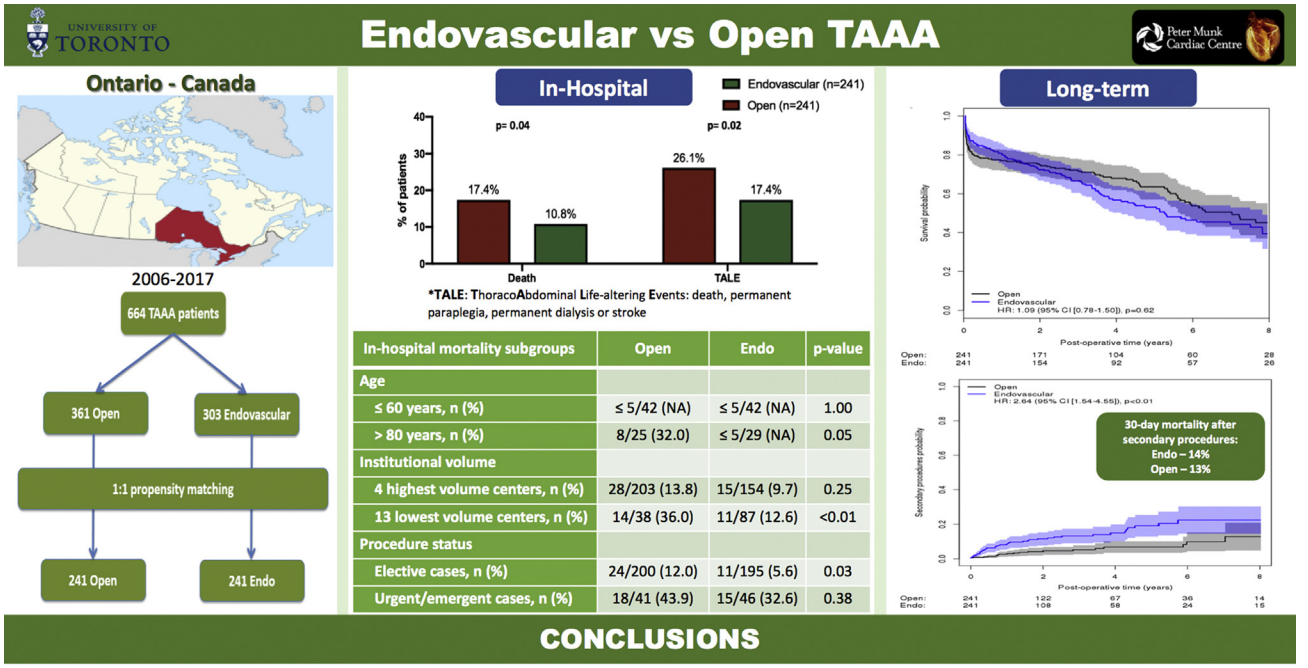


FIGURE 4. Cumulative incidence *curves* for secondary procedures on the thoracoabdominal aorta and its branches after endovascular versus open thoracoabdominal aortic repair in a PSM cohort. *HR*, Hazard ratio; *CI*, confidence interval.



In-hospital mortality subgroups	Open	Endo	p-value
Age			
≤ 60 years, n (%)	≤ 5/42 (NA)	≤ 5/42 (NA)	1.00
> 80 years, n (%)	8/25 (32.0)	≤ 5/29 (NA)	0.05
Institutional volume			
4 highest volume centers, n (%)	28/203 (13.8)	15/154 (9.7)	0.25
13 lowest volume centers, n (%)	14/38 (36.0)	11/87 (12.6)	<0.01
Procedure status			
Elective cases, n (%)	24/200 (12.0)	11/195 (5.6)	0.03
Urgent/emergent cases, n (%)	18/41 (43.9)	15/46 (32.6)	0.38

FIGURE 5. Endovascular versus open thoracoabdominal aortic repair in Ontario. This propensity score–matched study found that endovascular repair was associated with improved early outcomes but higher rates of secondary procedures. Long-term survival was poor and independent of technique. *TAAA*, Thoracoabdominal aortic aneurysm; *NA*, not available.

(aneurysm size, Crawford extent, presence of dissection), could not be reliably obtained. Surgical details, including surgeon experience, spinal fluid drainage, neuromonitoring, distal aortic perfusion, and management of branch vessels, were also not captured. Furthermore, we were not able to ascertain if a procedure was staged. However, because our coding strategy identified endovascular TAAA procedures using branched and fenestrated grafts, if the procedure was staged, the index case would likely be the final step of the staged procedure. The indications for reintervention (endoleak, branch vessel occlusion, progression of disease, planned procedure vs failure of endograft) are unknown, and those that occurred during the index hospitalization were not captured.

CONCLUSIONS

Despite incremental improvement in outcomes after TAAA repair from 2006 to 2017, overall mortality in Ontario continues to be higher than commonly published in single-center expert series. In this first population-based study comparing short- and long-term outcomes after endovascular with open TAAA repair, an endovascular approach was associated with improved early mortality but similar rates of dialysis, stroke, and paraplegia. The early mortality benefit after endovascular repair is not evident within 1 year of follow-up, with a higher rate of secondary procedures on the thoracoabdominal aorta and its branches in the endovascular group. Long-term survival and adverse events after TAAA repair were poor and independent of repair technique, likely reflecting the high burden of atherosclerosis and comorbidities in this patient population.

Webcast

You can watch a Webcast of this AATS meeting presentation by going to: https://aats.blob.core.windows.net/media/19%20AM/Sunday_May5/205BD/205BD/S51%20-%20Arch%20and%20descending%20aorta/S51_6_webcast_023511996.mp4.

Conflict of Interest Statement

Dr Ouzounian serves as a consultant for Medtronic and is on their North American advisory board. All other authors have nothing to disclose with regard to commercial support.

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Key Words: thoracoabdominal aortic aneurysm, survival, population-based

Discussion

Dr Marek Ehrlich *(Vienna, Austria)*. Your group from Toronto performed a multicenter, population-based, propensity-scored study across the province of Ontario comparing early and late outcomes of endovascular versus open TAAA repair. Although the 2 groups are relatively small, this article provides further information on the ongoing debate of TEVAR versus open repair. I would like to raise a few questions related to your patients.

First, I didn't find any information on the indication of the disease. Second, 44% of patients in the open group were

operated on an urgent or emergency basis. What was the reason for this? Third, what are the group's evolving strategies to reduce perioperative complications after TAA repair? How have these data influenced the Ontario group's decision on which patients get TEVAR and which open repair?

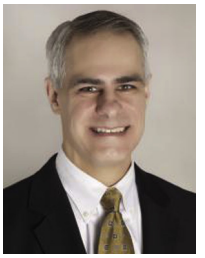
Dr Maral Ouzounian *(Toronto, Ontario, Canada)*. Because this study was based on administrative data and not detailed clinical data, we do not have information on several important variables, including the indication for surgery, size of the aneurysm, Crawford extent, and technical details of the operation such as whether or not we used circulatory arrest or left heart bypass. Therefore, we can't answer those questions because of the limitations of the data. The proportion of patients who had urgent or emergency operations in the open repair group was in fact 19%, not 44%. It's still a high proportion, higher than most expert series.

In looking for an explanation, when we looked at our own patients at Toronto General Hospital, we found that the average aneurysm size for patients undergoing TAAA repair was 7 cm, so quite a bit larger than the threshold for surgery. We suspect that patients may be getting referred late for evaluation and repair. In the more recent era, we have found greater regionalization of care such that in the last 6 years, 4 hospitals are doing 90% of the open procedures. We believe that this centralization of cases to higher-volume centers may be contributing to the improved outcomes we have observed, but we can't say for certain.

In terms of what we are doing to improve outcomes, at our hospital we have a multidisciplinary team approach to patients with complex aortic disease. We have a multidisciplinary clinic where these patients are seen by cardiac surgery, vascular surgery, and anesthesia teams, and the decision regarding which modality to use is made as a team. We selectively perform preoperative spinal cord embolization when we think it would be helpful, and we use prophylactic spinal drains and left heart bypass liberally in these patients. We also use intraoperative neuromonitoring and rescue therapy with hyperbaric oxygen in the event of spinal cord ischemia. Those are the few steps that we have recently taken.

Dr John Elefteriades *(New Haven, Conn)*. Maral, that was a beautiful article. Every question that came to my mind was answered in the next slide. The only major area that I could see that was not analyzed has to do with how often the aneurysm was fully controlled by the endovascular means and how often there may have been major endoleaks. You may not have access from your administrative database, but could you possibly do that now from a clinical chart and other avenues?

Dr Ouzounian. We were not able to identify exactly what type of secondary procedures were performed in these patients. We are unable to tell from administrative data whether it was for preexisting disease or if disease developed after the index repair. What we did observe was that the endovascular group had more interventions on the aorta—the thoracic aorta, abdominal aorta, and branches. The open repair group seemed to have a more definitive operation, as we would suspect. With our local patients at Toronto General, we are currently analyzing how many have late endoleaks and what the long-term outcomes are in those that do.



Dr Scott A. LeMaire *(Houston, Tex)*. This points out one of the problems with administrative data. Could you comment on your propensity score analysis? In addition to not having data to describe the groups and their outcomes, you also don't have all of the variables you would like to use for balancing the groups in your propensity score analysis. So, without those data, your groups may not be as well matched as you might hope. What were the challenges with this aspect of the analysis?

Dr Ouzounian. We did an exhaustive PSM based on the variables we had, but the variables we had were limited to mostly baseline demographics, clinical comorbidities, the Charlson index, and those types of things, not on more pertinent issues related to the thoracoabdominal repair. We can't say, for example, what proportion of patients were even eligible for an endovascular repair based on anatomic considerations. We are starting out with a heterogeneous group with significant selection bias.

From the original cohort of 664, we ended up with 241 in each group, and the patients who we lost were mostly younger patients in the open group. We just couldn't match them to the endovascular group; the age distribution was different. We suspect that many of those unmatched patients are those who are younger with connective tissue disorders and chronic dissections who almost exclusively receive open repair.

Dr LeMaire. How did you define the lower-volume centers versus the higher-volume centers, and what is the takeaway about volume, at least from the Ontario perspective?

Dr Ouzounian. We found this observation to be interesting. Four centers in Ontario were doing the bulk of the volume in both endovascular and open repair during the study period. The mean volume in the high-volume centers is still not high; for open repairs, it was 17 per year and for endovascular it was 14 to 15 per year. The low-volume centers were doing less than 5 per year. Because of privacy

rules, we aren't able to disclose numbers less than 5; we have to suppress those small cells.

We did find that over time, however, more patients in Ontario are being repaired in high-volume centers. In the recent era, 90% of patients undergoing open repair are being done in high-volume centers. Endovascular repair seems to be distributed widely with 70% of cases being currently done in higher-volume centers.

We also found that the volume effect on mortality was more pronounced in the open group than in the endovascular group. Results were better after endovascular repair in the higher-volume centers, but the difference was really not as pronounced than in patients undergoing open TAAA repair. Centers that were doing a handful of patients, potentially ruptured and too sick to be transferred. Well those patients were essentially nearly all dying.

Dr Malakh Lal Shrestha *(Hannover, Germany).* I have 2 short questions. One would be that in your series I don't see the adverse events normally associated with open surgery like, let's say, bleeding or length of stay in the intensive care unit has been documented, but adverse events and possible adverse events associated with endovascular group have not been documented, namely, the amount of radiation given to these patients.

The amount of radiation that the patient received, not only in the initial one, but also if you say that because of endoleaks you have to go back in again, you have to document that also, and in the long run the cause of mortality in these patients, whether it was aortic-related problems or malignancy due to the radiation, which has been documented as a possibility.

Dr Ouzounian. We don't have data about endoleaks or radiation dose in the endovascular group. We are looking at those end points within our own institutional series.

In terms of cause of death, we suspect that these patients have a high burden of atherosclerotic disease. When we looked at long-term adverse events at 10 years, for example, 40% of patients had an MI and 10% of patients had a stroke in follow-up. We suspect that these patients may be dying of atherosclerotic events, including coronary events and strokes, but we don't know for sure.

Dr D. Craig Miller *(Stanford, Calif).* You practice in a socialized environment, albeit one without a NICE Committee, so you must know all the costs incurred for both groups of patients. I wonder if anyone has calculated a Canadian dollar per quality-adjusted life year (QALY) quotient on this and derived an economic ICER. Can the Canadian healthcare system or Canadian society really afford treatment of thoracoabdominal pathology in these patients given their limited life expectancy? As a corollary, what is the Canadian society's ''willingness to pay'' threshold for medical care today? In the United States, this benchmark has hovered around the annual cost (or cost per QALY) of permanent dialysis therapy, something in the range of $55,000 to $70,000/QALY.

Dr Ouzounian. In Canada, we try to take care of every patient in the best way possible. The endovascular repair patients had a custom-made fenestrated or branch graft. These are expensive, about $60,000 per graft. The open repair cases have a very high cost in the hospital because of the length of stay in the intensive care unit. Our PhD student is actually doing a cost analysis on cost that will be presented at the Society of Vascular Surgeons. Overall, endovascular repair is more expensive in terms of cost in the early phase.

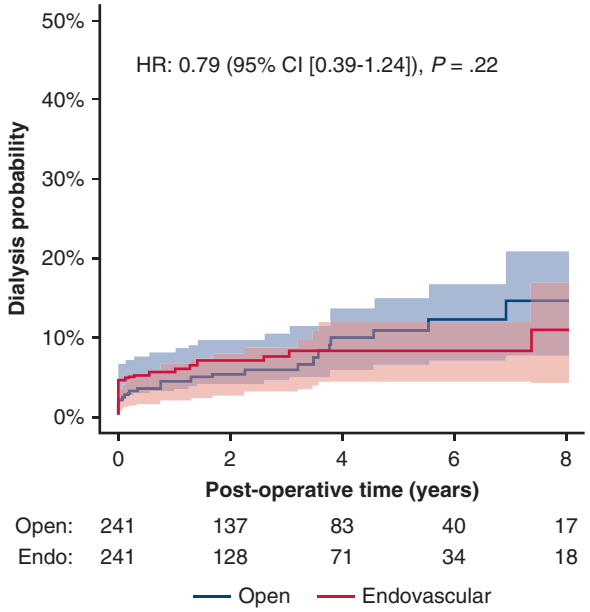


FIGURE E1. Cumulative incidence curves for permanent dialysis after endovascular versus open TAAA repair in a PSM cohort. *HR*, Hazard ratio; *CI*, confidence interval.

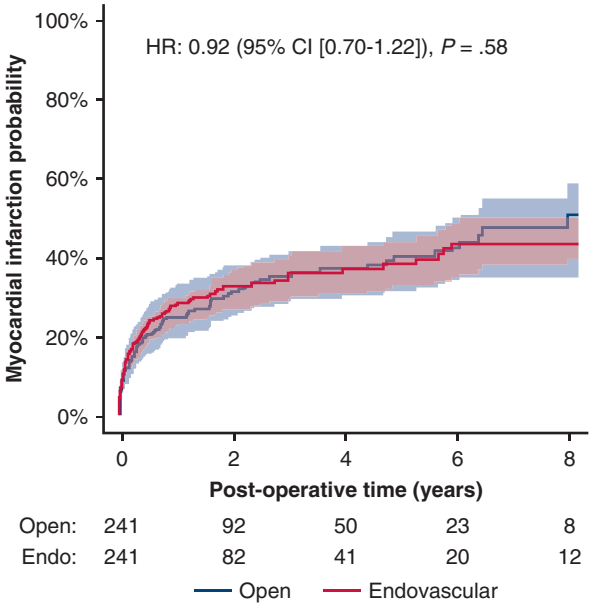


FIGURE E3. Cumulative incidence curves for MI after endovascular versus open TAAA repair in a PSM cohort. *HR*, Hazard ratio; *CI*, confidence interval.

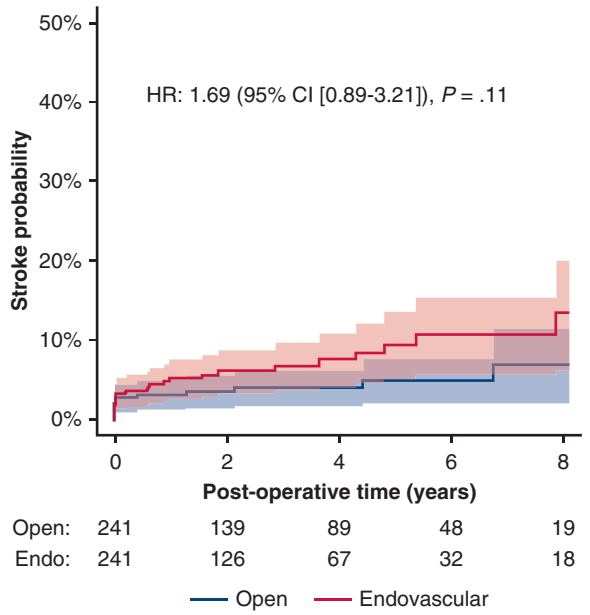


FIGURE E2. Cumulative incidence curves for stroke after endovascular versus open TAAA repair in a PSM cohort. *HR*, Hazard ratio; *CI*, confidence interval.

TABLE E1. Validated identification algorithm for endovascular and open thoracoabdominal aortic aneurysm repair using Canadian Classification of Health Intervention and Ontario Health Insurance Plan codes through chart abstraction method

Procedure code*	PPV (95% CI)
- Endovascular TAAA repair using CCI code	
1ID80GQNRN	0.90 (0.78-0.90)
- Open TAAA repair using a combination of CCI and OHIP codes	
[(1ID80LAXXQ OR 1ID80LAXXN OR 1ID80LAXXK OR 1ID80LAXXA OR 1ID80LA OR 1ID80QFXXQ OR 1ID80QFXXN OR 1ID80QFXXK OR 1ID80QFXXA) AND R803]	0.98 (0.87-1.00)

PPV, positive predictive value; *CI*, confidence interval; *TAAA*, thoracoabdominal aortic aneurysm; *CCI*, Canadian Classification of Health Interventions; *OHIP*, Ontario Health Insurance Plan. *All cases must present a concomitant I71 code (aortic aneurysm) from the ICD-10-CA, at the index procedure of up to 4 years before the procedure.

TABLE E2. Administrative databases used to identify study end points

Administrative database	End points
Canadian Institute for Health Information Discharge Abstract Database	In-hospital adverse events Long-term clinical outcomes
Registered Persons Database	All-cause mortality
Ontario Health Insurance Plan (physicians' billings)	Secondary procedures
Canadian Organ Replacement Register	Dialysis activity in Canada

ADULT

TABLE E3. Diagnostic codes used in this study

Variable/condition	CCI	ICD-10-CA	OHIP codes	Other databases
Procedural codes for index event				
Open	CCI codes: (1ID80QFXXA OR 1ID80QFXXK OR 1ID80QFXXN OR 1ID80QFXXQ OR 1ID80LA OR 1ID80LAXXA OR 1ID80LAXXK OR 1ID80LAXXN OR 1ID80LAXXQ) AND OHIP code: R803 AND ICD-10-CA code: I71 within 4 y before the procedure			
Endovascular	CCI code: 1.ID.80.GQ-NR-N AND ICD-10-CA code: I71 within 4 y before the procedure			
Outcomes				
Death				RPDB
Any stroke		I60 I61 I62 I63.x (excluding I63.6) I64.x H34.1		
Dialysis				CORR database (treatment code): Hemodialysis: 111, 112, 113, 121, 122, 123, 131, 132, 133, 211, 221, 231, 311, 312, 313, 321, 322, 323, 331, 332, 333, 413, 423, 433 Home HD: 413, 423, 433 Peritoneal dialysis: 141, 151, 152, 241, 242, 251, 252, 443, 453
Nontraumatic spinal cord injury		G82.0 G82.1 G82.2 G83.1 G83.3 G83.4 G95.1 G95.2 G95.8 G95.9 S24.18 S24.19 S34.18 S34.19 S34.38		
Hospital length of stay				DAD: Number of days between index procedure and discharge date, subtracting the number of ALC days (ALC LOS). Discharge is defined as discharge/transfer to any nonhospital/nonacute care facility (DISCHDISP = 2, 3, 4, 5, 6, 7, 12).

(Continued)

TABLE E3. Continued

Variable/condition	CCI	ICD-10-CA	OHIP codes	Other databases
Discharge location				DAD: The value of DISCHDISP variable
Secondary procedures:	-1.KA.^			
- Aortic	- 1.KE.^			
- Abdominal arteries	-1.KT.^			
- Pelvic vessels	- 1.KG.^			
- Leg arteries	- 1.JM.MI-XXN			
- Axillofemoral bypass	- 1.SY.80.^			
- Abdominal wall	- (1ID80QFXXA OR 1ID80QFXXK OR 1ID80QFXXN OR 1ID80QFXXQ OR 1ID80LA OR 1ID80LAXXA OR 1ID80LAXXK OR 1ID80LAXXN OR 1ID80LAXXQ)			
- Redo open TAAA				
- Redo endo	- 1.ID.80.GQ-NR-N			
Comorbidities				
Stroke index procedure		I63.^, I64.^, G45.^ (except G45.4), H34.1		
Dialysis index procedure				CORR database (treatment code): Hemodialysis: 111, 112, 113, 121, 122, 123, 131, 132, 133, 211, 221, 231, 311, 312, 313, 321, 322, 323, 331, 332, 333, 413, 423, 433 Home HD: 413, 423, 433 Peritoneal dialysis: 141, 151, 152, 241, 242, 251, 252, 443, 453
Coronary artery disease	1IJ26, 1IJ27, 1IJ54, 1IJ57, 1IJ50, 1IJ76	I21, I22, I23, I24, I25, Z955, Z958, Z959, R931, T822	R741, R742, R743, G298, E646, E651, E652, E654, E655, G262, Z434, Z448, 410, 412	
MI		I21.x I22.x		
Congestive heart failure	*Diagnosis date in Ontario Congestive Heart Failure Database that precedes index date			
Peripheral artery disease		I70.2 I73.9 I74.3 I74.4 I79.2 E10-E14 (with common fourth character .5) *1.WK.93.^, *1.WL.93.^, *1.WM.93.^, *1.WN.93.^ *(exclude if accompanied by ICD-10 code C40.^, D16.^, D48.0, D48.1, D48.2, Q65.^-Q79.^, S70.^ – S99.^, T20.^ – T32.^), 3.KG.10.^, 3.KG.20.^, 3.KG.30.^, 3.KG.40.^		

(Continued)

TABLE E3. Continued

Variable/condition	CCI	ICD-10-CA	OHIP codes	Other databases
Diabetes	*Diagnosis date in Ontario Diabetes Database that precedes the index date			
Hypertension	*Diagnosis date in Ontario Hypertension Database that precedes the index date			
COPD	*Diagnosis date in Ontario COPD Database that precedes the index date			
Chronic kidney disease		Per Charlson score definition: N032-N037, N052-N057, N18, N19, N250, Z490-Z492, Z940, Z992	OHIP-diagnostic: 403, 585	
Coronary revascularization	1IJ50 1IJ54 1IJ57GQ 1IJ76			
Cerebrovascular disease		I65.ˆ, I.66ˆ		
Malignancy		C15.ˆ, C18.ˆ, C19.ˆ, C20.ˆ, C22.ˆ, C25.ˆ, C34.ˆ, C50.ˆ, C56.ˆ, C61.ˆ, C82.ˆ, C83.ˆ, C85.ˆ, C91.ˆ, C92.ˆ, C93.ˆ, C94.ˆ, C95.ˆ, D00.ˆ, D05.ˆ	203, 204, 205, 206, 207, 208, 150, 154, 155, 157, 162, 174, 175, 183, 185.	
Medications				
Statins	All drugs from ODB with SUBCLNAM "ANTI-LIPEMIC: STATINS."			
Beta-blockers	All drugs from ODB with SUBCLNAM "BETA-BLOCKERS," "BETA-BLOCKERS COMBINATION," "BETA-BLOCKING AGENTS."			
ACEi/ARB	All drugs from ODB with SUBCLNAM "ACE INHIBITORS," "ACE INHIBITORS COMBINATION," "ANGIOTENSIN II ANTAGONIST," "ANGIOTENSIN II COMBINATION."			
Antiplatelet agents	All drugs from ODB with SUBCLNAM "PLATELET-REDUCING AGENT," FIBRINOGEN-PLATELET BINDING INHIBITORS," "PLATELET AGGREGATION INHIBITORS," "ADENOSIDE DIPHOSPHATE INHIBITORS."			
Anticoagulants	All drugs from ODB with SUBCLNAM "HEPARINS," "VITAMIN K ANTAGONISTS," "ANTICOAGULANTS," "ANTICOAGULANTS MISCELLANEOUS," "SYNTHETIC ANTITHROMBOTIC AGENTS," "LOW MOLECULAR WEIGHT HEPARINS (LMWH)."			

CCI, Canadian Classification of Health Interventions; *ICD-10-CA*, International Classification of Diseases, 10th Revision, Canada; *OHIP*, Ontario Health Insurance Plan; *RPDB*, Registered Persons Database; *TAAA*, thoracoabdominal aortic aneurysm; *CORR*, Canadian Organ Replacement Register; *LOS*, length of stay; *ALC*, alternate level of care; *MI*, myocardial infarction; *COPD*, chronic obstructive pulmonary disease; *ACEi*, angiotensin-converting enzyme inhibitor; *ARB*, angiotensin receptor blocker.

TABLE E4. In-hospital mortality after endovascular versus open thoracoabdominal aortic repair stratified by age, era, and institutional volume

In-hospital mortality	Pre-PSM			Post-PSM		
	Open	Endovascular	*P* value	Open	Endovascular	*P* value
Overall	64/361 (17.7)	31/303 (10.2)	<.01	42/241 (17.4)	26/241 (10.8)	.04
Stratified by age						
≤60 y, n (%)	8/77 (10.4)	≤5/44 (NA)	1.00	≤5/42 (NA)	≤5/42 (NA)	1.00
61-70 y, n (%)	21/124 (16.9)	(6-9) */81 (NA)	.22	(11-14)*/78 (NA) (NA)	8/72 (11.1)	.35
71-80 y, n (%)	25/132 (18.9)	13/130 (10.0)	.05	16/96 (16.7)	11/98 (11.2)	.31
>80 y, n (%)	10/28 (35.7)	6/48 (12.5)	.02	8/25 (32.0)	≤5/29 (NA)	≤.05
Stratified by era						
2006-2011, n (%)	37/188 (19.7)	12/110 (10.9)	.05	24/107 (22.4)	12/101 (11.9)	.05
2012-2017, n (%)	27/173 (15.6)	19/193 (9.8)	.12	18/134 (13.4)	14/140 (10.0)	.45
Stratified by institutional volume						
4 highest TAAA volume† centers, n (%)	43/299 (14.4)	19/195 (9.7)	.16	28/203 (13.8)	15/154 (9.7)	.25
13 lowest TAAA volume centers, n (%)	21/62 (33.9)	12/108 (11.1)	<.01	14/38 (36.0)	11/87 (12.6)	<.01
Stratified by procedure status						
Elective cases, n (%)	29/265 (10.9)	15/256 (5.9)	.04	24/200 (12.0)	11/195 (5.6)	.03
Urgent/emergency cases, n (%)	35/96 (36.5)	16/47 (34.0)	.85	18/41 (43.9)	15/46 (32.6)	.38

PSM, Propensity score matching; *NA*, not available; *TAAA*, thoracoabdominal aortic aneurysm. *Required to avoid reidentification. †Highest volume definition: institutions that have performed more than 60 cases of TAAA repair during our study period.

TABLE E5. Type of secondary procedures required after endovascular versus open thoracoabdominal aortic aneurysm repair

Secondary procedures	Pre-PSM		Post-PSM	
	Open	Endovascular	Open	Endovascular
Patients requiring secondary procedures	n = 51 (14.1%)	n = 64 (21.7%)	n = 34 (14.1%)	n = 55 (22.8%)
No. of secondary procedures	n = 73	n = 102	n = 45	n = 90
Thoracic or abdominal aorta, n (%)	8 (11.0)	15 (14.7)	≤5 (NA)	14 (15.6)
Abdominal arteries, n (%)	12 (16.4)	32 (31.4)	7 (15.6)	29 (32.2)
Pelvic vessels, n (%)	≤5 (NA)	(5-8)* (NA)	≤5 (NA)	≤5 (NA)
Leg arteries, n (%)	13 (17.8)	16 (15.7)	10 (22.2)	15 (16.7)
Abdominal wall procedures, n (%)†	24 (32.9)	13 (12.7)	16 (35.6)	11 (12.2)
Redo open TAAA, n (%)	7 (9.6)	≤5 (NA)	≤5 (NA)	≤5 (NA)
Redo endovascular TAAA, n (%)	≤5 (NA)	14 (13.7)	≤5 (NA)	13 (14.4)

PSM, Propensity score matching; *NA*, not available; *TAAA*, thoracoabdominal aortic aneurysm. *Required to avoid re-identification of TAAA endovascular cases. †Abdominal wall procedures were not included in the aortic repair related reintervention cumulative incidence in Table 3 and Figure 2, *D*.