

Improved survival after introduction of an emergency endovascular therapy protocol for ruptured abdominal aortic aneurysms

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Background: The study was conducted to demonstrate improved survival (30-day mortality) after the introduction of an emergency endovascular therapy protocol for ruptured abdominal aortic aneurysms (rAAA). Numerous authors have successfully demonstrated reduced mortality in patients with rAAA using endovascular techniques. Comparison of endovascular aneurysm repair (EVAR) with open repair for rAAA may be misleading, however, because EVAR cannot be performed on all patients, and selection bias may explain the superior performance of any given surgical or endovascular strategy. We developed a model to predict mortality in patients before the introduction of EVAR (preprotocol population), applied this model to predict 30-day mortality among prospective patients (postprotocol population), and compared observed vs expected results.

Methods: We assessed 126 patients with rAAA. Primary outcome was 30-day mortality. Potential confounding variables were age, sex, presurgical lowest recorded systolic blood pressure (SBP), and glomerular filtration rate (GFR). A logistic regression model incorporating significant confounders was used to evaluate changes in 30-day mortality for all patients with rAAA after introduction of the EVAR protocol. Separate logistic regressions were done to compare 30-day mortality for preprotocol vs patients receiving EVAR and preprotocol vs patients receiving postprotocol open repair. Cumulative sum (CUSUM) analysis was used to assess shifts in the performance of the rAAA program over time.

Results: Significant confounders were SBP, absence of SBP, and GFR. Logistic regression found evidence of lower mortality after the protocol was introduced, 17.9% vs 30.0% (odds ratio [OR], 0.385; 95% confidence interval [CI], 0.141 to 0.981; $P = .046$). Comparison of all open repairs (preprotocol and postprotocol) and EVAR demonstrated decreased risk for EVAR of 5.0% vs 28.3% (OR, 0.109; 95% CI, 0.013 to 0.906; $P = .0084$). Unstable patients (SBP ≤ 80) showed a trend towards improved survival with EVAR relative to open repair (14.3% vs 56.0%, $P = .061$). Comparison of preprotocol surgery with open repair after the introduction of the protocol found no evidence of a difference between mortality rates for the open procedures—30.0% (preprotocol) vs 25.0% (postprotocol; OR, 0.688; 95% CI, 0.335 to 1.415, $P = .3031$)—demonstrating that the improved performance observed with CUSUM analysis was related to the introduction of the EVAR protocol.

Conclusion: Our predictive model using “weighted” CUSUM analysis (a measure of performance over time) demonstrated that a predefined strategy of management of rAAA that includes EVAR is associated with improved ($P < .05$) mortality. Unstable patients with rAAA may be particularly benefited by EVAR and should not be excluded from repair. Appropriate patients with rAAA who are undergoing treatment in experienced vascular centers should be offered EVAR as the treatment of choice. (*J Vasc Surg* 2007;45:443-50.)

A meta-analysis published in 2002 of the last 50 years demonstrated that open surgical repair of ruptured abdominal aortic aneurysms (rAAA) continues to be associated with surgical mortality rates of 45 to 50%¹ and an overall

mortality of 75% to 90%, including prehospital deaths,² despite advances in aortic grafts and open surgical and anesthetic technique. Since the advent in 1991 of minimally invasive or endovascular aneurysm repair (EVAR) for the treatment of elective, asymptomatic AAA, large prospective registries and multicenter trials for EVAR have demonstrated significant reductions in perioperative mortality and in long-term aneurysm-related death.³⁻⁷ Minimally invasive EVAR may particularly benefit high-risk operative candidates, although this is still controversial.⁸

Our single institution experience includes treatment of >420 elective patients with AAA since May 1999 using EVAR, with an overall 30-day mortality rate of 1.4% (6

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Competition of interest: none.

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deaths). This compares favorably with the 3% to 11% mortality rate reported in other large series.^{3-6,9-10} The successful treatment of these elective AAA patients has led to the question of whether this technique may be extended to patients requiring emergency repair of rAAA in an effort to reduce the historically high mortality rates.

Since the Nottingham group¹¹ reported the first case of successful EVAR for rAAA in 1994, numerous authors have demonstrated a strong correlation between the use of EVAR for rAAA and improved 30-day mortality outcomes compared with standard open repair.^{12,13} Most of these authors have concluded that EVAR is a viable treatment option in patients with appropriate anatomy. In 2005, Alsac et al¹² reported a review of the current literature describing EVAR of rAAA, which demonstrated that EVAR resulted in decreased procedure times, blood loss, and length of stay, including intensive care unit stay. The average postoperative mortality rate for this series was 24% (range, 9% to 45%).¹³

We agree with other authors¹⁴ that an analysis restricting the comparison of EVAR with previous or concurrent open procedures might be misleading owing to selection bias, including differences in patient risk factors, characteristics, or anatomy; for example, patients selected for EVAR may be at lower risk (more stable) than those selected for open repair, with technically easier anatomy to manage (eg, longer aortic necks). In addition, not all patients are anatomic candidates for EVAR. Rather than asking “Is EVAR better than open repair for rAAA?” we thought it was more appropriate to ask, “Can we improve the performance of our rAAA program by incorporating EVAR into our protocol?” recognizing the inherent selection bias and the inability to treat all patients with EVAR.

Our strategy was to develop a model that predicted mortality in patients before the introduction of EVAR (preprotocol population), to apply this model to predict 30-day mortality among prospective patients (postprotocol population), and then compare observed vs expected results.

METHODS

Patients treated with a contained or free-ruptured infrarenal AAA at a single university tertiary care center between March 10, 2001, and February 10, 2006, were prospectively enrolled into this study. Approval for the study was obtained from the Conjoint Health Research Ethics Board at the University of Calgary. Data were captured in a custom-designed Vascular Surgery Database (Access, Microsoft, Redmond, Wash). Charts were cross-referenced to validate data. To ensure that only those patients with documented blood outside of the aortic wall were included in the study, we excluded 26 patients with a coded diagnosis of rAAA and with symptoms of rAAA, but with no blood outside the aortic sac.

Patients enrolled before January 2004 were treated with standard open repair with a Dacron tube graft. After January 2004, an algorithm for endovascular treatment of rAAA was implemented (Fig 1). These patients underwent

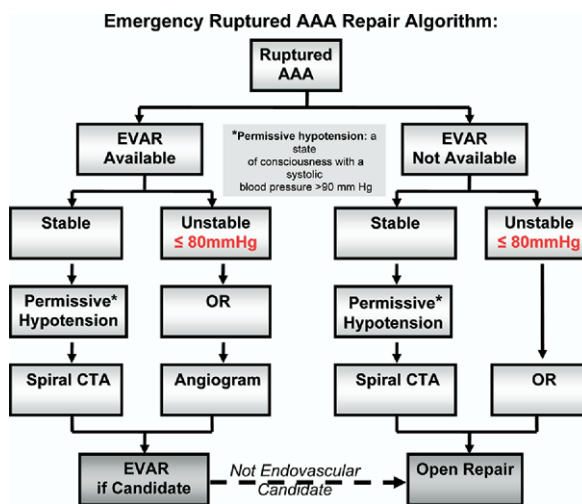


Fig 1. Emergency ruptured abdominal aortic aneurysm (rAAA) endovascular repair (EVAR) algorithm. *Permissive hypotension is defined as state of consciousness with a systolic blood pressure >80 mm Hg. Endovascular on call is defined as the availability of a vascular surgeon with endovascular privileges. CTA, Computed tomography angiography.

Table I. Criteria used to determine anatomic suitability for emergency endovascular aneurysm repair

Anatomic inclusion criteria for emergency endovascular aneurysm repair	
1.	Aortic neck ≤32 mm in diameter
2.	Infrarenal neck ≥10 mm in length
3.	Neck angulation ≤60°
4.	Calcification ≤40%
5.	Nonreverse funnel shaped neck
6.	Iliac diameter ≤20 mm, ≥6 mm
7.	Ability to preserve one internal iliac

eligibility assessment for an intention-to-treat protocol using a Zenith (Cook Inc, Bloomington, Ind.) aortouniliac or aortobiliac EVAR device.

We broadened the anatomic inclusion criteria (Table I) compared with those recommended for elective EVAR. Specifically, we included those patients with a shorter neck length of 10 mm in the expectation that proximal seal of the graft from the aneurysm could be achieved using adjunctive techniques (ie, Palmaz stent, Cordis, Miami Lakes, Fla) at the discretion of the operating surgeon. We were willing to accept a less favorable neck length to achieve control of life-threatening hemorrhage as part of a damage control concept. Those who did not meet the anatomic inclusion criteria underwent a standard open repair.

Emergency ruptured abdominal aortic aneurysm repair algorithm. The algorithm for emergency rAAA repair was implemented in January 2004 (Fig 1). All patients with a diagnosis of a rAAA were first assessed clinically by the attending surgeon. The treatment algorithm was determined by whether EVAR was available and

whether the patient agreed to participate in the study once informed consent outlining the risks and benefits was obtained. The two treatment paradigms were as follows:

I. Endovascular aneurysm repair available

Clinical assessment determined whether the patient was hemodynamically stable or unstable, and thus assignment into one of these two subgroups. Hemodynamic stability was defined as a state of consciousness with a systolic blood pressure >80 mm Hg.

A. Stable endovascular aneurysm repair group. Patients who met the stability criteria were maintained in a state of permissive hypotension with resuscitation to a blood pressure of <90 mm Hg to maintain consciousness. These patients underwent a spiral computed tomography (CT) scan with intravenous contrast using 1.25-mm cuts. The patients were then taken to the operating room (OR) for EVAR performed under local, locoregional, or general anesthetic, provided CT or intraoperative angiographic anatomic inclusion criteria of the patient's rAAA were met (Table I). Groin cutdowns were performed to expose and control the common femoral arteries.

Standard EVAR repair using conventional techniques was performed, with the choice between aortouniiliac/occluder/femoral crossover and aortobiliac devices at the discretion of the operating surgeon. An aortic occlusion balloon (RELIANT, Medtronic, Minneapolis, Minn; PVL6, Cook, Queensland Australia; or CODA, Cook, Indianapolis, Ind) was placed above the level of the renal arteries during manipulation of the endovascular device only if signs of intraoperative hemodynamic instability developed, and was withdrawn immediately before to stent-graft deployment.

B. Unstable endovascular aneurysm repair group. Patients who were deemed unstable were taken directly to the OR from the emergency department. A groin cutdown was performed under a local anesthetic to expose and control a common femoral artery. An aortic occluding balloon was then inserted above the renal arteries and inflated to create a cross-clamp of the aorta, which allowed an angiogram to be performed to assess anatomic suitability.

Standard EVAR repair with bilateral groin cutdowns was then performed on patients who had amenable anatomy, with the choice between aortouniiliac/occluder/femoral crossover and aortobiliac devices at the discretion of the operating surgeon. The aortic occlusion balloon was deflated and withdrawn immediately before main body stent graft deployment.

For patients in whom the aortic anatomy was not amenable to EVAR, the intra-aortic occluding balloon was left in place in the proximal aorta above the rAAA to arrest ongoing bleeding. Once the patient had been appropriately resuscitated, a general anesthetic was administered and a standard open surgical cross-clamp placement and repair was performed with a Dacron tube graft.

Table II. Coefficients in the risk-adjustment model based on pre-endovascular aneurysm repair patients*

	Coefficient	SE	Z	P
(Intercept)	4.9287	1.6696	2.95	.0032
GFR	-0.0311	0.0130	-2.40	.0164
SBP	-0.0369	0.0133	-2.78	.0054
Absence of SBP	-2.7209	1.4570	-1.87	.0618

SE, Standard error; GFR, glomerular filtration rate; SBP, systolic blood pressure.

*The C statistic for this model was 0.82. The predicted log (odds) of mortality for patients with a systolic blood pressure (SBP) at the time of surgery is $\eta = (4.93 - 0.0311) \times (GFR - 0.0369) \times SBP$. For patients with no SBP at the time of surgery the formula is $\eta = (2.21 - 0.0311) \times GFR$.

II. Endovascular aneurysm repair not available (open repair)

Patients were included in this group if the on-call surgeon did not have EVAR privileges or if their workup in the EVAR group showed they did not have anatomic suitability for EVAR. The patient's clinical hemodynamic stability was ascertained as defined previously.

A. Stable open repair group. Patients who were stable were maintained in a state of permissive hypotension and underwent a spiral CT scan to confirm the diagnosis and assist with preoperative planning before they were taken to the OR for a standard open rAAA repair with a Dacron tube graft under a general anesthetic. Whenever possible, an intra-aortic occluding balloon was placed through a common femoral artery cutdown to arrest ongoing bleeding before administration of the general anesthetic and laparotomy.

B. Unstable open repair group. Patients who were unstable were taken directly to the OR from the emergency department and underwent open rAAA repair without prior imaging. Whenever possible, an intra-aortic occluding balloon was placed through a common femoral artery cutdown to arrest ongoing bleeding before administration of the general anesthetic and laparotomy.

Statistical methods. The primary outcome was 30-day all-cause mortality. Potential confounding variables were age, gender, presurgical lowest SBP, and glomerular filtration rate (GFR). The GFR ($\text{mL}/[\text{min} \cdot 1.73\text{m}^2]$) was calculated on the basis of serum creatinine level ($\mu\text{mol}/\text{L}$), age, sex, and race using the Modification of Diet in Renal Disease Study Group formula.¹⁵ For four patients with missing creatinine values, the missing value was conservatively calculated as the mean serum creatinine value for pre-EVAR patients ($95.73 \mu\text{mol}/\text{L}$) before the calculation of GFR.

If continuous variables were normally distributed, we used *t* tests to compare the means of these variables of the patients treated before and after the introduction of the protocol, otherwise Wilcoxon tests were used. The Fisher exact test was used to compare binary variables. A two-sample test of proportion was done to compare mortality of open and EVAR patients who were unstable. After review-

Table III. Preoperative clinical characteristics of patients before and after introduction of the endovascular protocol

Variable	Preprotocol mean (% total)	Postprotocol mean (% total)	Test statistic	P
Age	71.01 (8.14)	72.66 (7.91)	$t_{124} = -1.14$.2554
Female (n)	11 (15.7)	13 (23.2)		.3624*
Hgb	109.69 (30.85)	111.66 (23.33)	$t_{122} = -0.39$.6944
WBC	13.86 (12.64)	11.65 (4.63)		.9606*
Sodium	137.30 (16.85)	139.79 (4.98)		.3218*
Potassium	4.16 (0.72)	4.16 (0.71)	$t_{121} = -0.01$.9919
GFR	83.07 (36.35)	65.39 (28.86)	$t_{124} = 2.97$.0036
SBP	92.51 (45.80)	94.07 (44.68)	$t_{124} = -0.19$.8483
SBP ≤ 80 (n)	25 (35.7)	20 (35.7)		1.0000*
No BP (n)	8 (11.4)	6 (10.7)		1.0000*

WBC, White blood cell; GFR, glomerular filtration rate; SBP, systolic blood pressure.

*Fisher's exact test.

ing the preprotocol data, the three variables of SBP, absence of SBP, and GFR were determined to significantly predict 30-day mortality. These variables were used to derive a logistic regression model to evaluate changes in the 30-day mortality postprotocol (Table II).

We used a logistic regression model including the significant variables identified in the preprotocol data (SBP, absence of SBP, and GFR) to evaluate changes in 30-day mortality after the introduction of the EVAR protocol. This model included patients before the introduction of the protocol as well as all patients receiving open or EVAR procedures after the introduction of the protocol. We also used separate logistic regression models to compare pre-EVAR 30-day mortality with 30-day mortality for all patients receiving EVAR procedures and patients receiving open procedures after the introduction of the protocol.

We used risk-adjusted cumulative sum (CUSUM) plots to examine shifts in 30-day mortality after the introduction of the EVAR protocol. Risk-adjusted CUSUM plots are highly sensitive to shifts in surgical performance.¹⁶⁻²⁶ These plots use two CUSUM lines. The upper line detects poorer-than-expected performance over time and is constrained so that it cannot go below zero. A horizontal "alert" line is crossed if outcome performance was worse than expected according to the risk-adjustment model. For surgeons operating at the level of pre-EVAR performance, this line would be crossed every 100 surgeries (ie, the horizontal alert line corresponds to an average run length of 100). The lower line detects better-than-expected performance and is constrained so that it cannot go above zero. A horizontal "superior" line is crossed if outcome performance is better than expected according to the risk-adjustment model. This line would be crossed every 100 surgeries by surgeons operating at the level of pre-EVAR protocol surgeons. The constraints on the lines (upper line constrained to be above zero and the lower line constrained to be below zero) ensure that the CUSUM lines will be sensitive to clusters of bad or good events.

The risk-adjustment model for the CUSUM lines was developed using SBP, absence of SBP, and GFR to predict the 30-day mortality for patients before the introduction of the protocol (Table II). This model was then used to

predict the probability of death for each patient according to the covariate values. The outcome for each patient (lived or died) and the predicted probability of death determined the amount of upward or downward movement in the CUSUM lines. Data points were therefore "weighted," in that mortality in patients with a low probability of death produced greater negative impact than did mortality in patients with a high probability of death. Conversely, survival in patients with a high predicted mortality had greater positive impact than did survival in patients with a low predicted mortality. This helped to eliminate "survivor bias," whereby low-risk patients artificially improved outcomes.

RESULTS

Clinical characteristics of patients before and after the introduction of the EVAR protocol are presented in Table III. EVAR was done in 20 (36%) of the 56 patients in the postprotocol period. Procedures were completed in the OR with portable fluoroscopic support. Two patients had local anesthesia, and the others had general anesthesia. Twelve patients had aortobiiliac/occluder/femoral-femoral bypass procedures, six patients had aortobiiliac procedures, and two patients with ruptured saccular aneurysms had tube endograft repair. Postprotocol patients were found to have decreased renal function as indicated by lower presurgical GFR ($t_{124} = 2.967$, $P = .0036$). Without adjustment for clinical characteristics, there was no evidence of lower mortality for all patients (combined open and EVAR procedures) after the introduction of the EVAR protocol ($P = .146$, Fisher exact test).

Logistic regression: (risk-adjusted outcomes). The results of risk-adjusted logistic regression are presented in Table IV. After adjusting for SBP, absence of SBP, and GFR, there was evidence of a lower mortality rate after the introduction of the protocol: 21 (30.0%) of 70 vs 10 (17.9%) of 56 (OR, 0.385; 95% CI, 0.141 to 0.981; $P = .046$). A similar comparison of all open surgeries (preprotocol and postprotocol) and EVAR procedures demonstrated evidence of decreased risk for EVAR procedures: 28.3% vs 5.0% (OR, 0.109; 95% CI, 0.013 to 0.906; $P = .0084$), and a logistic regression comparing preprotocol

Table IV. Comparison of mortality with odds ratios after adjusting for patient risk factors

Cohort	Mortality %	Cohort	Mortality %	OR*	CI	P
Preprotocol (All)	21/70 (30)	Postprotocol (All)	10/56 (18)	0.385	0.141-0.981	.046
All open	30/106 (28)	EVAR	1/20 (5)	0.109	0.013-0.906	.0084
Preprotocol open	21/70 (30)	Postprotocol Open	9/36 (25)	0.688	0.335-1.415	.3031

CI, Confidence interval; EVAR, endovascular aneurysm repair.

*Odds ratio from logistic regression that adjusted for systolic blood pressure, no blood pressure, and glomerular filtration rate.

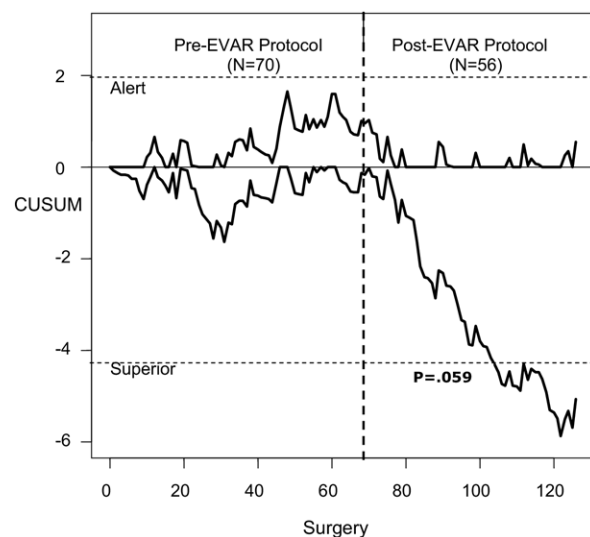


Fig 2. Cumulative sum (CUSUM) plot for all repairs. Vertical dashed line indicates the introduction of the ruptured abdominal aortic aneurysm endovascular aneurysm repair (EVAR) protocol. After the introduction of the protocol, the lower CUSUM line crosses the “superior” line within 35 surgeries ($P = .059$).

(open) surgeries with open procedures after the introduction of the protocol found no evidence of a difference between mortality rates for the open procedures: 30.0% vs 25.0% (OR, 0.688; 95% CI, 0.335 to 1.415; $P = .3031$).

Unstable patients (systolic blood pressure ≤ 80 mm Hg). No difference was evident between groups with respect to unstable patients between preprotocol (25/70 [35.7%]) and postprotocol (20/56 [35.7%]; $P = 1$, two-sample test of proportion), or between all preprotocol and postprotocol open (38/106 [35.9%]) and EVAR patients (7/20 [35%]; $P = 1$).

The mortality was 20 (53%) of 38 among unstable open patients, 14 (56%) of 25 among pre-EVAR protocol patients, and one (14.3%) of seven among unstable EVAR patients. There was a trend towards improved survival in unstable rAAA patients with EVAR compared with open repair, although not statistically significant ($P = .0617$, test of proportion).

Cumulative sum plots. The coefficients for the risk-adjustment model based on the pre-EVAR protocol patients are presented in Table II. The C statistic for this model was 0.82. Fig 2 presents a CUSUM plot showing the

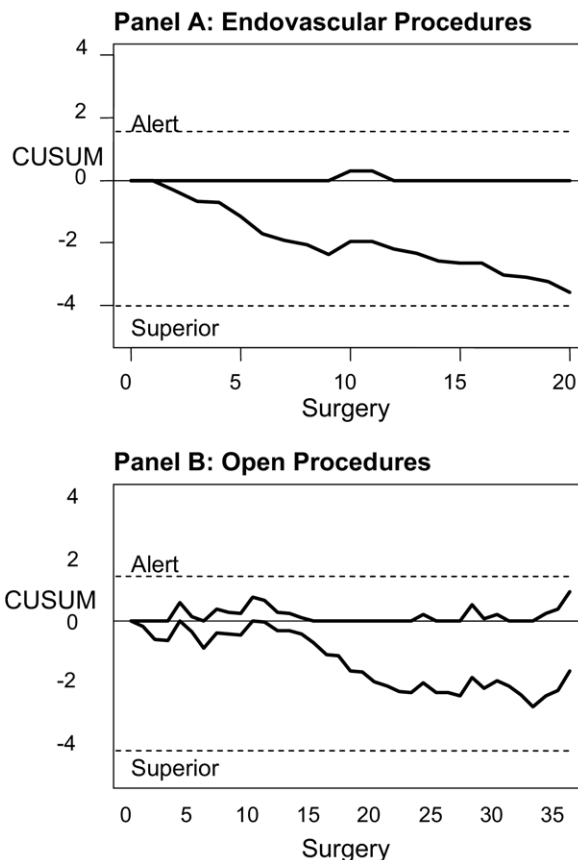


Fig 3. Cumulative sum (CUSUM) plots for (A) endovascular aneurysm repair and (B) open repairs after the introduction of the EVAR protocol. Note the stable performance of open repairs, with a trend towards improvement in the performance of endovascular repairs.

shift in performance for all patients pre-EVAR and post-EVAR protocol. The lower CUSUM line crosses the “superior” line in the first 35 repairs after the introduction of the protocol. The CUSUM line for surgeons with a pre-EVAR level of performance would, on average, cross this line every 100 surgeries. The probability that pre-EVAR surgeries would cross this line within 35 surgeries is 0.059. CUSUM plots showing shifts in performance associated with open and closed procedures are presented in Fig 3. EVAR surgical performance improved during the study, whereas open surgical performance remained relatively stable.

DISCUSSION

The incidence of rAAA continues to increase despite a 100% increase in elective repairs during the past 20 years, the development of screening programs, and overall improvements in public and physician awareness of the pathology.²⁷ EVAR repair for rAAA, with a reduced physiologic insult,^{13,28-30} offers a less invasive procedure for these often moribund patients and has been successfully applied in this emergency setting.³¹ The current use of EVAR for rAAA is still low, however, accounting for only 6% of repairs for rAAA in a study involving nearly 30% of the United States population.³² Interpretation of results for “like patients” has been limited by selection bias and the anatomic restrictions of endovascular technology.

Similar to other investigators, we have demonstrated an improved survival (5.0% mortality) in both stable and unstable patients undergoing EVAR for rAAA compared with those undergoing open repair (28.3% mortality). However, the primary objective of the study was to determine if a new protocol incorporating endovascular technology for anatomically suitable patients would significantly improve the odds of patients surviving their illness at presentation. As a result of the implementation of our intent-to-treat EVAR protocol, we report an improved performance of our rAAA program with respect to 30-day mortality (30.0% vs 17.9%) for all patients presenting with rAAA.

Other investigators have also demonstrated the utility of the development of a protocol-based approach to EVAR for rAAA. Arya et al³³ demonstrated a reduced mortality (39% vs 59%) for rAAA after introduction of an intent-to-treat EVAR protocol. Mehta et al³⁴ reported an 18% mortality for rAAA patients treated with EVAR alone. A subanalysis of their overall results demonstrated a combined postprotocol rAAA mortality for open surgical and EVAR repair of 35%. Our 17.9% mortality rate postprotocol and our 5.0% EVAR mortality rate for rAAA is lower than that reported in this and other series,¹⁴ as is our 30% mortality rate for index open aortic repair. Single institution reports of mortality post EVAR for rAAA are 8% to 14%.^{13,31,35,36} A recent systematic review and meta-analysis of EVAR observational studies for rAAA involving 400 patients demonstrated a pooled 30-day mortality rate of 20%.³⁷

Typical reported mortality rates for open repair are 32% to 50%.^{1,2,14} Our center receives referrals from a large geographic area and represents the only tertiary care center for vascular repair for a catchment population of >2 million. The patients that arrived to undergo repair at our center were sick: 35% patients were unstable (SBP <80 mm Hg), and 11% of these had no recordable blood pressure. Those patients receiving EVAR repair had a comparable SBP to those receiving open repair (101.2 vs 93.32 mm Hg, respectively; $P = .476$, t test). A subgroup analysis demonstrated no difference in the proportion of unstable patients in either the preprotocol vs postprotocol, or open vs EVAR groups.

Unlike other series of EVAR for rAAA,³⁸ we did not use hemodynamic instability to exclude patients. However,

that these patients survived prolonged transport to present alive in the emergency department for surgical repair suggests that they were physiologically more robust. The resultant self-selection of our population may therefore limit the applicability of our results to centers with decreased transport time and distances, as 28 (26%) of our patients for whom data were available were transferred from “out of province” or “out of city.”

Nevertheless, the lower preprotocol mortality rate also effectively raised the bar in terms of our group having to demonstrate the significant improvements observed in the postprotocol cohort. The low postprotocol and EVAR mortality rates in our series may also reflect the experience of our endovascular team and our large inventory of readily available commercial devices. In addition, we preferentially used the aortouniiliac device for unstable patients, which is more rapidly placed and thus shortens the time to endoseal owing to the elimination of the need to cannulate the contralateral limb. The benefits of the aortouniiliac system for rAAA have been noted by other investigators.^{14,39} It has also been our observation that the routine use of early balloon control of the aorta, even with open surgical cases, reduces the incidence of prolonged shock and hemodynamic instability.

We use the term *endovascular spillover* to describe the application of endovascular techniques to improve the performance of open techniques. Our use of these balloon techniques to control the aorta during open repair antedated our protocol and also contributed to reduced baseline and postprotocol mortality. And finally, the elimination of routine preoperative CT scanning (and the associated delay to OR) may have benefited our patient population postprotocol and reduced the selection bias observed in other studies,^{13,31,40} whereby patients selected for EVAR had to be stable enough to undergo CT scanning.

The primary limitation of our study is its lack of true randomization. We expect a major impediment to randomization in future studies will be an exceptionally high rate of treatment arm crossover owing to the strict anatomic limitations imposed by current endovascular technology and the current limited availability of skilled endovascular teams comfortable with emergency EVAR for rAAA. Our liberal application of anatomic inclusion criteria (eg, neck length of 10 cm) and our lack of medical exclusion criteria allowed for EVAR repair in 36% of our postprotocol cohort, which is similar to the 23% to 50% EVAR rates for rAAA reported by experienced centers.^{14,36,41-46}

Our bias is that medical exclusion of EVAR for patients with rAAA is inappropriate and that the greatest benefits of this technology will be observed in the sickest of the patients. Although not statistically significant, likely because of the small numbers in our series, we did see a trend ($P = .06$) towards improved survival with EVAR in those patients with either no recordable SBP at presentation or with SBP <80 mm Hg.

Recognizing that long-term durability of an aneurysm repair may be as important as short-term outcome, we

support the philosophy that endovascular damage control is an appropriate goal in treating a rAAA and that this should increase the applicability of this technology. In this setting, EVAR may also serve as a bridge to definitive surgical or secondary EVAR interventions performed under elective circumstances. Early in our series, only one surgeon was performing EVAR for rAAA. With experience, all members of the surgical group became involved, and exclusion of EVAR for rAAA because of lack of availability disappeared. We believe, therefore, that our nonrandomized experience most closely approximates the realities faced by most endovascular centers worldwide.

Patient risk model. Our initial strategy was to rely on the established predictive criteria for mortality in vascular patients using the Portsmouth modification of the Physiological and Operative Severity Score for Enumeration of Mortality and Morbidity (P-POSSUM) methodology to develop a risk-adjustment model for our postprotocol cohort.⁴⁷ This mortality model includes variables for age, blood urea nitrogen, sodium, potassium, hemoglobin, and white cell count and has been shown to be highly predictive of mortality in a United Kingdom rAAA patient population. The ability of this model to predict mortality in our preprotocol patients was suboptimal, however, with a *C* statistic of only 0.66 (completely random would be 0.50). This was likely due to the lack of available blood urea nitrogen data in our patients because this was rarely obtained preoperatively.

Further analysis was performed on additional patient variables to validate a risk-adjustment model specific for our patient population that had better discrimination in predicting 30-day mortality (*C* statistic = 0.82). The strong association between GFR, SBP, and death observed in our preprotocol cohort has been described by other investigators⁴⁸ and allowed for the application of our predictive model to our postprotocol group to assess risk-adjusted performance.

The use of CUSUM analysis to assess performance has been validated in multiple series looking at surgical performance, learning curves, and endovascular learning curves, in particular.³⁹ The most important feature of our risk-adjusted CUSUM analysis of mortality after treatment for rAAA is the ability to determine a weighted impact to stratify the patients according to their predicted risk of death. This is a unique feature of our trial and, to our knowledge, has not been previously reported. This feature eliminated the bias observed in other studies whereby all deaths and all survivals are equivalent, without any estimation of the predicted risk of death for any particular patient. This is particularly important during the assessment of endovascular technology, where selection bias or anatomic restrictions may limit application.

CONCLUSION

Our predictive model using weighted CUSUM analysis, which is a measure of performance over time, demonstrated that a predefined strategy of management of rAAA that includes EVAR is associated with improved ($P < .05$)

mortality. EVAR may be of particular benefit to unstable patients with rAAA, and they should not be excluded from repair. Appropriate patients with rAAA who are undergoing treatment in experienced vascular centers should be offered EVAR as the treatment of choice.

AUTHOR CONTRIBUTIONS

Conception and design: RM, MN
Analysis and interpretation: RM, CC, MN, PF
Data collection: RD, MM, MN, WA
Writing the article: RD, MN
Critical revision of the article: RM, CC, MM, PF, WA
Final approval of the article: RD, CC, MM, PF, WA
Statistical analysis: PF, MM
Obtained funding: Not applicable
Overall responsibility: RD

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