The effects of abdominal compartment hypertension after open and endovascular repair of a ruptured abdominal aortic aneurysm

Ragai R. Makar, MSc, FRCS, Stephen A. Badger, MD, MRCS, Mark E. O'Donnell, MD, DSEM, MRCS, William Loan, FRCR, Louis L. Lau, MD, FRCS, and Chee V. Soong, MD, FRCS, *Belfast, Northern Ireland, United Kingdom*

Objective: This study assessed if emergency endovascular repair (eEVR) reduces the increase in intra-abdominal compartment pressure and host inflammatory response in patients with ruptured abdominal aortic aneurysm (AAA). *Methods:* Thirty patients with ruptured AAA were prospectively recruited. Patients were offered eEVR or emergency conventional open repair (eOR) depending on anatomic suitability. Intra-abdominal pressure was measured postoperatively, at 2 and 6 hours, and then daily for 5 days. Organ dysfunction was assessed preoperatively by calculating the Hardman score. Multiple organ dysfunction syndrome, systemic inflammatory response syndrome, and lung injury scores were calculated regularly postoperatively. Hematologic analyses included serum urea and electrolytes, liver function indices, and C-reactive protein. Urine was analyzed for the albumin-creatinine ratio.

Results: Fourteen patients (12 men; mean age, 72.2 ± 6.2 years) underwent eEVR, and 16 (14 men; mean age, 71.4 ± 7.0 years) had eOR. Intra-abdominal pressure was significantly higher in the eOR cohort compared with the eEVR group. The eEVR patients had significantly less blood loss (P < .001) and transfused (P < .001) and total intraoperative intravenous fluid infusion (P = .001). The eOR group demonstrated a greater risk of organ dysfunction, with a higher systemic inflammatory response syndrome score at day 5 (P = .005) and higher lung injury scores at days 1 and 3 (P = .02 and P = .02) compared with eEVR. A significant correlation was observed between intra-abdominal pressure and the volume of blood lost and transfused, amount of fluid given, systemic inflammatory response syndrome score, multiple organ dysfunction score, lung injury score, and the length of stay in the intensive care unit and hospital.

Conclusion: These results suggest that eEVR of ruptured AAA is less stressful and is associated with less intra-abdominal hypertension and host inflammatory response compared with eOR. (J Vasc Surg 2009;49:866-72.)

Despite successful repair of ruptured abdominal aortic aneurysm (rAAA), many patients die as a result of multiple organ dysfunction syndrome (MODS). In contrast with elective repair, the operative mortality of rAAA has not improved significantly in recent years, with mortality rates still ranging from 32% to 80%.¹⁻⁶ However, endovascular repair (EVR) of rAAA is feasible and has been suggested to be associated with a better outcome than emergency open repair (eOR).^{7,8}

Although the pathogenesis of MODS after AAA surgery is multifactorial, the development of abdominal compartment hypertension may play a role. Abdominal compartment syndrome (ACS) is diagnosed when the abdominal pressure increases to >20 mm Hg in combination with end-organ dysfunction.^{9,10} The latter occurs in approximately 30% of rAAA patients¹¹ and is associated with a mortality rate of about 70%.¹² The natural precursor to MODS in patients

The Vascular Unit received an educational grant from Medtronic Inc. Competition of interest: none.

Reprint requests: R.R. Makar, Apartment 14, Shaftesbury Court, 32-36 Dublin Rd, Belfast BT2 7HN, Northern Ireland, UK (e-mail: ragaimakar@ hotmail.com).

0741-5214/\$36.00

Copyright @ 2009 by The Society for Vascular Surgery. doi:10.1016/j.jvs.2008.11.027

after rAAA appears to be the systemic inflammatory response syndrome (SIRS).¹³ Endovascular aneurysm repair may reduce the risk of MODS and improve outcome by minimizing the elevation of intra-abdominal pressure (IAP).

The aim of this study was to assess the relationship between abdominal compartment hypertension and the inflammatory response in patients after repair of rAAA, and whether EVR can reduce these responses.

PATIENTS AND METHODS

Study design. This study was a nonrandomized, controlled observational study comparing EVR of rAAA with the conventional transabdominal open repair. The study was approved by the Local Research Ethics Committee in Belfast, and the Belfast City Hospital Trust provided clinical indemnity.

Patient cohort. All patients aged >50 years, presenting to the Vascular and Endovascular Surgery Unit of the Belfast City Hospital with a rAAA were recruited after informed written consent from the patient or next of kin. Rupture was confirmed preoperatively by computed tomography (CT) scan, either at the referring hospital or our center. All patients were assessed for suitability for EVR. The criteria for eEVR suitability included an aneurysmal neck diameter <32 mm, infrarenal segment length >10 mm, iliofemoral diameter >6 mm, and aortic neck angula-

From the Vascular and Endovascular Surgery Department, Belfast City Hospital.

Presented as an abstract at the British Society of Endovascular Therapy, Solihull, United Kingdom, Jun 8, 2007.

tion of $<60^\circ$, although shorter necks and those with greater angulation were considered suitable especially if the walls were parallel. Stable and unstable patients who were suitable underwent eEVR, otherwise a conventional eOR was performed. All patients were resuscitated using a permissive hypotensive regimen.⁷

Patients were excluded from the study if they had no evidence of rupture on CT scan, had juxtarenal AAA, required suprarenal aortic clamping, had dementia, were on chronic renal dialysis, refused to participate in the study, or died ≤ 2 hours of surgery.

Operative technique. All operations were done by one of four vascular consultants and, in cases of eEVR, with the assistance of one of four consultant radiologists. Exposure and control of the common femoral arteries in eEVR were performed under local anesthesia when possible. Guidewires and angiographic catheters were then introduced by a 7F introducer sheath to the level of the first or second lumbar vertebrae. The positions of the renal arteries, aortic bifurcation, and common iliac bifurcation were identified on digital subtraction angiography with contrast injected using a power injector.¹⁴

Once this was performed, the stent graft was deployed through the femoral artery while an occluder was passed into the common iliac artery on the contralateral side. A femorofemoral bypass was then performed to revascularize the contralateral lower limb. This latter part of the operation was performed under general anesthesia if required.

All patients undergoing eOR underwent routine endotracheal general anesthesia with a standardized induction protocol and a midline incision from xiphisternum to pubic symphysis and primary abdominal closure.

Measurement of IAP. Under aseptic technique, normal saline (50 mL) was infused through a Foley urinary catheter, which is routinely inserted in all the patients, to fill the catheter tubing. This eliminated air bubbles within the draining catheter system. The bladder catheter was lowered to facilitate gravity drainage and avoid any possible confounding increase in bladder pressure. The catheter tube was clamped distal to the sampling membrane.^{15,16} A 20gauge needle was subsequently inserted through the catheter sampling membrane, and the catheter pressure was measured using a transducer connected to a monitor. The symphysis pubis equated to a pressure of 0 mm Hg. Patients were lying flat during all measurements. Data were recorded as mean pressure at end-expiration. IAP was measured at 2, 6, and 24 hours postoperatively and then daily until day 5.

Clinical, operative details, and blood analyses. The volumes of blood lost and transfused were recorded. Fluid intake, urinary output, cardiac rhythm, and arterial and central venous pressures were monitored and maintained at an adequate level. The Hardman score¹⁷ was calculated preoperatively, and the SIRS,¹⁸ lung injury,¹⁹ and MODS scores²⁰ were calculated immediately after surgery and then daily for 5 days. Samples of blood were collected for measurements of renal and liver function, full blood count, C-reactive protein, and arterial blood gas. These indices

were assessed before surgery and then daily for 5 days thereafter.

The arterial blood gas was used to calculate the ratio of partial pressure of arterial oxygen to the fraction of inspired oxygen (Pao_2/Fio_2). This was used along with serum creatinine, bilirubin, and platelets, Glasgow coma score, and pressure-adjusted heart rate in the calculation of MODS scores.²⁰ The three components used in the lung injury score calculation were Pao_2/Fio_2 ratio, chest roentgenogram score, and positive end-expiratory pressure (PEEP) score.¹⁹

The American College of Chest Physicians (ACCP) and Society of Critical Care Medicine (SCCM) consensus conference proposed the concept of SIRS.¹⁸ The latter is defined by two or more of the following: temperature $>38^{\circ}$ C or $<36^{\circ}$ C; heart rate >90 beats/min, respiratory rate >20 breaths/min, or hyperventilation PacO₂ <4.3 Kpa (<32 mm Hg); white blood cell (WBC) count >12000 cells/mm³ or <4000 cells/mm³, or >10% immature neutrophils.¹⁸ SIRS scores derived from the latter criteria have been shown to be a useful quantitative measure of the proinflammatory response.²¹

Blood samples were obtained through a radial arterial or central venous line. Urine was collected for albumin/ creatinine ratio at days 1, 3, and 5.

Statistical analysis. Nonpaired continuous variables were analyzed by the Mann-Whitney *U* test or the *t* test, depending on data distribution. Categoric variables were analyzed with the χ^2 test and Fisher exact test, as appropriate. The Spearman correlation was used to assess the relationships between IAP and clinical outcome variables. A two-tailed *P* < .05 was considered statistically significant. All analyses were performed using SPSS 13 statistical software (SPSS Inc, Chicago, Ill).

RESULTS

Patient characteristics. Between October 2004 and January 2007, 40 consecutive rAAA patients who reached the hospital alive were considered for the study. The study excluded 10 patients. Two patients sustained a cardiac arrest on arrival to the operating theater, and a third patient died of a severe myocardial infarction (MI) after aneurysm exclusion by deployment of the aortouniiliac stent. Two further patients were excluded because they were hemodialysis dependent preoperatively, with one in each group. The remaining excluded patients (n = 5) had eOR because of unavailability of the facility or the required staff for eEVR, or because of an inability to get consent.

The study comprised 30 patients, of whom 14 had eEVR and 16 underwent an eOR. The mean age \pm standard deviation was 72.2 \pm 6.2 years for the eEVR group and 71.4 \pm 7.0 years for the eOR group (Table I). The male/female ratios were 12:2 in the eEVR group and 14:2 in the eOR group. The baseline comorbidities, cardiovascular risks, and preoperative blood pressure were comparable for the two groups (Table I). No difference was identified between patient groups based on the Hardman score. One patient in each group was receiving aspirin and clopi-

Table I. Baseline preoperative patient characteristics

Characteristics ^a	eEVR (n = 14)	$eOR \ (n = 16)$	Р
Age, y	72.2 (6.2)	71.4 (7)	.75
Male/female ratio	12:2	14:2	>.999
Risk factors, No.			
Diabetes mellitus	0	0	_
Hypertension	11	12	>.999
Hyperlipidemia	5	9	.29
Comorbidities, No.			
Ischemic heart disease	8	11	.70
Myocardial infarction	6	6	>.999
Carotid artery disease	4	0	.07
Peripheral vascular	2	0	.20
disease			
COPD	2	3	>.999
Creatinine >150	8	10	>.999
mmol/L			
SBP at admission, mm	101 (30.5)	108 (28.2)	.56
Hg	· · · · ·	. ,	
Hardman Index score			
0	4	4	1.0
1	5	2	.20
2	3	2 5	.40
3	3 2	4	.65
4	0	1	>.999
Infrarenal AAA diameter, mm	82 (17.7)	89 (15.8)	.39

AAA, Abdominal aortic aneurysm; *eEVR*, emergency endovascular repair; *eOR*, emergency open repair; *COPD*, chronic pulmonary obstructive disease; *NS*, not significant; *SBP*, systolic blood pressure; *SD*, standard deviation.

^aContinuous data are presented as the mean (standard deviation).

dogrel, and one patient in the eEVR group was taking warfarin.

In the 16 patients who underwent eOR, seven had a conical infrarenal neck that measured <10 mm long, six had short necks with angulation $\geq 60^{\circ}$, and one had a neck diameter >32 mm. Two patients had iliac arteries ≤ 6 mm in diameter, and three patients had aneurysmal and tortuous iliac arteries in addition to other unsuitable criteria.

Surgical and postoperative data. All patients who had eOR received general anesthesia. In the eEVR group, however, local anesthesia with or without sedation was used in 11 patients. One patient had general anesthesia, and two patients started with local anesthesia but were converted to general anesthesia during the femorofemoral bypass grafting. All patients received intravenous teicoplanin (400 mg; Aventis Pharma Ltd, Kent, UK) and cefuroxime (1.5 gm; Glaxo-Wellcome, Middlesex, UK) at the start of the operation, but none were given intraoperative anticoagulation therapy. However, all patients received postoperative subcutaneous low-molecular-weight heparin was for deep venous thrombosis prophylaxis.

All patients in the eEVR group received an aortouniiliac stent graft (Talent, Medtronic Ave, Santa Rosa, Calif), with the exception of one patient who received a bifurcated Talent stent graft. A bifurcated graft was used in seven eOR patients and a straight graft in nine. All eOR patients had primary abdominal closure. There was no intraoperative conversion from eEVR to eOR. Six eEVR patients were found to have intraoperative type 1 endoleak, which was controlled with ballooning and Palmaz stent (Cordis Corp, Miami, Fla) in four patients, aortic cuff extension (Talent, Medtronic Ave, Santa Rosa, Calif) in one patient, and both aortic cuff and Palmaz stent in another. One patient had an intraoperative type 2 endoleak, and a second patient had minor retrograde flush from the occluded site. Both were left untreated because the patients had remained stable. The latter had resolved spontaneously at follow-up.

When compared with eOR, eEVR resulted in significantly less blood loss (P < .001), less blood transfusion (P < .001), and less total intraoperative intravenous fluid infusion (P = .001; Table II). All patients in the open group were admitted to the intensive care unit (ICU) postoperatively, compared with only eight patients in the eEVR group (P = .005). The eOR group required longer ICU or combined ICU and high dependency unit (HDU) care than the eEVR group (P = .006 and P = .01); however, the difference between the groups in total HDU or duration of hospital stay was not significant (Table II).

IAP was significantly higher at all time points in the eOR group except at day 2 and 3 postoperatively (Fig 1). SIRS developed in nine of the 16 patients in the eOR group compared with only one of 14 in the eEVR group (P = .005) at day 5. A significantly higher lung injury score at days 1 and 3 (P = .02 and P = .02; Fig 2) and PEEP score immediately postoperatively and at days 1, 2, and 3 (P < .05) were observed in the eOR group compared with the eEVR group (Fig 3). However, no significant difference in MOD score or SIRS score existed between the groups except for the SIRS score at day 5 (P < .01).

Correlation between IAP and other variables. IAP correlated significantly with the length of ICU stay at all time points. Similarly, IAP correlated with the PEEP score at the all time points except for day 1 (Table III). There was a stronger association between the MOD score and the IAP at the preceding 24 hours than that of the same day, as demonstrated by the correlation between the IAP at days 1, 2, 3, and 4 and MODS at days 2, 3, 4, and 5 (r = 0.46, 0.55, 0.57, and 0.54; P = .01, .002, .001, and .003, respectively) and between IAP at days 2, 3, 4, and 5 and MODS at days 2, 3, 4, and 5 (r = 0.43, 0.45, 0.49, and 0.53; P = .02, .01, .007, and .003, respectively).

IAP at day 4 correlated with the SIRs score at day 5 (r = 0.44; P = .02). No correlation between IAP and the albumin/creatinine ratio or C-reactive protein was noted during the 5-day postoperative period. The IAP at days 3 and 4 was correlated with the lung injury score at days 4 and 5 (r = .45 and .37; P = .01 and .04). IAP at 2 and 6 hours correlated with blood loss (r = 0.57 and 0.65; P = .001 and <.001), platelet transfusion (r = 0.41 and 0.47; P = .02 and .009), and total intravenous fluids (r = .43 and 0.52; P = .01 and .003). IAP also correlated with packed cells received at 6 hours (r = 0.49, P = .006).

Complications. Four patients died postoperatively, with two in each group. Three of these patients had a Hardman score of \geq 3. In the eOR group, ACS developed

Variable ^a	eEVR (n = 14)	$eOR \ (n = 16)$	Р
Blood loss, mL	862 (297-1183)	3767 (2275-6284)	<.001
Packed cells transfused, U	3 (2-4)	9 (5-11)	< .001
Intra-op IV fluid, mL	2250 (1500-3125)	4250 (3123-7500)	.001
ICU			
Admission, No.	8	16	.005
LOS, h	22 (0-78)	90 (48-168)	.006
HDU			
Admission.	9	4	.06
LOS, h	10 (0-25)	0 (0-3)	.08
ICU and HDU LOS stay, h	38 (9-102)	138 (49-168)	.01
Hospital LOS, d	13.5 (9.7-22.2)	19 (9.2-29)	.3
Complications			
Deaths	2	2	>.999
ACS	1	1	>.999
Cardiac	2	2	>.999
Respiratory	2	7	.08
Dialysis	2	2	>.999
Stroke	0	1	>.999
Bowel ischemia	1	1	>.999
Wound problems	1	3	.6

Table II. Intraoperative and postoperative details, and perioperative complications

ACS, Abdominal compartment syndrome; HDU, high dependency unit; ICU, intensive care unit.

^aContinuous data are presented as the median interquartile range (IQR); categoric data are presented as the number.

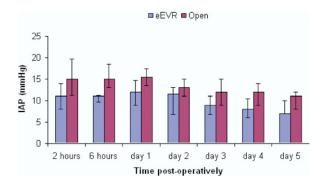


Fig 1. Intra-abdominal pressure *(IAP)* was significantly higher in the open group at 2 hours (P < .05), 6 hours ($P \le .001$), 1 day (P < .05), 4 days (P < .05), and 5 days (P < .05) compared with the emergency endovascular repair (*eEVR*) group. Results are presented as median and interquartile range.

in one patient 6 hours postoperatively, necessitating a laparotomy and left hemicolectomy for ischemic large bowel, followed by overwhelming sepsis. The ACS diagnosis in this patient was based on IAP of 35 mm Hg that was associated with dysfunction of the cardiovascular, renal, and respiratory systems, in that sequence. Another eOR patient had diffuse brain injury thought to be secondary to preoperative hypotension.

One patient in the eEVR group had ACS at 8 hours postoperatively, which required decompression by a laparotomy, followed by Hartmann operation for ischemic colon 24-hours later. The ACS diagnosis in this patient was based on an IAP of 25 mm Hg, resulting in respiratory compromise that required endotracheal intubation and assisted ventilation, followed by renal impairment. He died of sepsis and pneumonia on day 25. Another patient died of

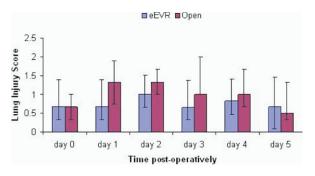


Fig 2. The open group had a significantly higher lung injury score at day 1 and day 3 (P = .02 and P = .02, respectively). Results are presented as median and interquartile range. *eEVR*, emergency endovascular repair.

exacerbation of respiratory and acute renal failure at day 4 postoperatively. The patient had been on home oxygen for chronic obstructive pulmonary disease and had previously been denied elective repair.

DISCUSSION

For decades, the mortality rate of eOR for rAAA has shown only minor improvement, despite widespread advances in perioperative critical care.¹⁻⁶ This could be explained by a limited change in the actual operative open techniques over the years, with the exception of the use of a retroperitoneal approach in rAAA repair.²² Initial reports on the use of eEVR for rAAA suggested significant improvement in the operative mortality rate, which ranges between 0% and 23%.^{7,23-26} However, other studies showed a 30-day mortality of 37% to 53% in eEVR patients compared with 39% to 53% for eOR patients.^{27,28} Some

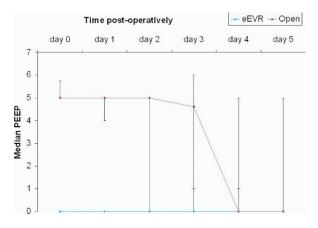


Fig 3. A significantly higher positive end-expiratory pressure (*PEEP*) was observed immediately postoperatively and at 1, 2, and 3 days (P < .05) in the open group. Results are presented as median and interquartile range. *eEVR*, emergency endovascular repair.

 Table III. Correlation between intra-abdominal pressure and intensive care unit stay or positive end expiratory pressure

A, Correlation between	intensive	care	unit	stay	and	intra-
abdominal pressure						

IAP time points	Correlation coefficient	Р	
2 hours	0.54	.002	
6 hours	0.61	<.001	
Day 1	0.49	.006	
Day 2	0.74	<.001	
Day 3	0.72	<.001	
Day 4	0.73	<.001	
Day 5	0.74	<.001	

B, Correlation between intra-abdominal pressure and positive endexpiratory pressure

IAP	PEEP	Correlation coefficient	Р
2 hours	Day 0	0.53	.002
Day 1	Day 1	0.3	.09
Day 2	Day 2	0.62	<.001
Day 3	Day 3	0.58	.001
Day 4	Day 4	0.57	.001
Day 5	Day 5	0.63	.001

IAP, Intra-abdominal pressure; *ICU*, intensive care unit; *PEEP*, positive end-expiratory pressure.

units suggest on-table angiography to overcome the delay for preoperative CT scaning.²⁹

After eOR or eEVR of rAAA, a bimodal distribution in mortality has been described at the first 24 hours and >7 days.³⁰ The first peak is due to hypovolemic shock and the second peak is secondary to MODS, although studies on the pathophysiology of ACS suggest that hypovolemic shock is the precipitating factor for the rise of IAP.³¹ In the current study, the IAP was at its highest during the first 48 hours postoperatively. This could be related to the volume

of blood lost and transfused, and amount of fluid administered. Patients with ACS tend to receive more pre-ICU crystalloids and packed red blood cells.³² Excessive fluid resuscitation is associated with increased frequency of intraabdominal hypertension, ACS, MODS, and mortality.³² The increase in the microvascular permeability could also play an important role in these patients.^{12,32-34}

The mortality rate in both groups was <15%, or 23% when all patients were included who arrived at the hospital alive. This decrease in death relative to our retrospective published data follows the application of an intention-to-treat by eEVR protocol and the use of hypotensive hemostasis.³⁵ Both groups in this study had comparable Hardman scores, mean systolic blood pressure, and comorbidities that should have minimized the selection bias between the groups. Although eEVR resulted in reduced blood loss, blood transfusion, and fluid requirements, its impact on hospital stay was probably masked by the major trauma effect of the rAAA.³⁶ However, more patients required ICU admission and had prolonged intensive care after eOR compared with eEVR.

In this study, the development of abdominal hypertension correlated with the MODS score and prolonged ICU stay. Most patients recovered, but there is a potential of progressing to ACS. A contributing factor for the rise in IAP in eOR could be intestinal manipulation and mesenteric traction. Previous research observed higher SIRS and MODS scores in elective transperitoneal repair compared with the retroperitoneal approach.³⁷ This was thought to be due to less bowel manipulation and mesenteric traction in the retroperitoneal approach.³⁷ Others showed a decrease in inflammatory mediators with eEVR compared with OR,³⁸ although in our study the eEVR group did not have significantly better MODS scores compared with the eOR group. This may be due to the small number of patients, leading to a type II error. However, the eEVR group demonstrated lower SIRS scores at day 5, whereas the eOR group had a significant deterioration in SIR scores at days 4 and 5.

Although ACS is a potential complication after rAAA surgery, there are few reports on its incidence. Elevation of IAP was reported in a retrospective study of open repair of 17 rAAA patients: nine patients had an IAP >20 mm Hg and ACS developed in seven. Five of the six patients who had decompression survived.¹¹ ACS complicates 8% to 20% of patients after eEVR with an associated 67% mortality.^{7,31} In a study of 53 eEVR and eOR patients and 22 rAAA undergoing OR, the latter had a higher IAP compared with the elective patients.³³ An IAP of <15 mm Hg was postulated as a negative predictor of mortality.³³

In our study, both patients with ACS diagnosed at 6 hours underwent decompression, which is comparable to the timing of ACS in trauma patients after resuscitation.³² Both patients had colonic ischemia requiring resection. This association between IAP and bowel ischemia is supported by other studies.¹¹ Colon ischemia in patients who require delayed decompression laparotomy and mesh closure is reported at 40%, compared with 6% in patients with early mesh abdominal closure for open rAAA repair.¹² An

IAP of ≥ 20 mm Hg is associated with a reduction in venous return to the heart and a decreased cardiac output.³⁴ The increased abdominal pressure also reduces venous flow to the various intraperitoneal organs.^{9,39} This reduction in cardiac output and venous flow can therefore lead to a reduction in perfusion of the various visceral organs. This may cause the development of bowel ischemia, which in mild cases can increase intestinal permeability but in extreme cases can result in bowel infarction.

The limitations of this study were the small number of patients and lack of randomization. A randomized study would be logistically difficult for a single center, bearing in mind that only \leq 50% of the patients with rAAA would be suitable for eEVR. This would have diminished even further the number of patients recruited into each group of the study. Although it may be argued that the two groups are not entirely comparable, the risk profile and Hardman scores were similar between the two groups, thus minimizing potential confounding factors. We acknowledge the potential bias associated with the application of anatomic suitability for selecting patients for eEVR or eOR. However, we included in our patient cohort only those who had infrarenal clamping to minimize the impact of anatomic selection.

CONCLUSION

Our results demonstrated that eEVR for rAAA is associated with lower IAP and systemic inflammatory response than eOR repair. We have also shown that recovery is quicker with the eEVR group. Although it would be tempting to suggest that the reduction in IAP may account for these improvements in the eEVR group, it is difficult to be certain that the elevation is the cause and not the effect of MODS. However, the fact that IAP elevation seems to precede dysfunction of the various organs would support it playing a causative role.

We thank Bernard Lee, FRCS, Ray Hannon, MD, FRCS, L. Johston, and C. Boyed for provision of patient data and their operative and perioperative management.

AUTHOR CONTRIBUTIONS

Conception and design: RM, CS Analysis and interpretation: RM, SB, MO, WL, LL, CS Data collection: RM, SB, MO Writing the article: RM, SB, CS Critical revision of the article: RM, SB, MO, WL, LL, CS Final approval of the article: RM, SB, MO, WL, LL, CS Statistical analysis: RM, MO Obtained funding: CS Overall responsibility: CS

REFERENCES

- Bown MJ, Sutton AJ, Bell PR, Sayers RD. A meta-analysis of 50 years of ruptured abdominal aortic aneurysms. Br J Surg 2002;89:714-30.
- Johansen K, Kohler TR, Nicholls SC, Zierler RE, Clowes AW, Kazmers A. Ruptured abdominal aortic aneurysm: the Harbor view experience. J Vasc Surg 1991;13:240-5.

- Gloviczki P, Pairolero PC, Mucha PJ Jr, Farnell MB, Hallett JW Jr, Ilstrupb DM, et al. Ruptured abdominal aortic aneurysms: repair should not be denied. J Vasc Surg 1992;15:851-7.
- 4. Kniemeyer HW, Kessler T, Reber PU, Ris HB, Hakki H, Widmer MK. Treatment of ruptured abdominal aortic aneurysm, a permanent challenge or waste of resources? Prediction of outcome using a multi-organ dysfunction score. Eur J Vasc Endovasc Surg 2000;19:190-6.
- Noel AA, Gloviczki P, Cherry KJ Jr, Bower TC, Panneton JM, Mozes GI, et al. Ruptured abdominal aortic aneurysms: the excessive mortality rate of conventional repair. J Vasc Surg 2001;34:41-6.
- Prance SE, Wilson YG, Cosgrove CM, Walker AJ, Wilkins DC, Ashley S. Ruptured abdominal aortic aneurysms: selecting patients for surgery. Eur J Vasc Endovasc Surg 1999;17:129-32.
- Veith FJ, Ohki T, Lipsiotz EC, Suggs WD, Cynamon J. The treatment of ruptures abdominal aneurysm with stent grafts: a new gold standard? Semin Vasc Surg 2003;16:171-5.
- Larzon T, Lindgren R, Norgen L. Endovascular treatment of ruptured abdominal aortic aneurysm: a shift of the paradigm? J Endovasc Ther 2005;12:548-55.
- Sanchez NC, Tenofsky PL, Dort JM, Shen LY, Helmer SD, Smith RS. What is normal intra-abdominal pressure? Am Surg 2001;67:243-8.
- Diebel LN, Wilson RF, Dulchavsky SA, Saxe J. Effect of increased intra-abdominal pressure on hepatic arterial, portal venous, and hepatic microcirculatory blood flow. J Trauma 1992;33:279-83.
- Djavani K, Wanhainen A, Bjorck M. Intra-abdominal hypertension and abdominal compartment syndrome following surgery for ruptured abdominal aortic aneurysm. Eur J Vasc Endovasc Surg 2006;31:581-4.
- Rasmussen TE, Hallett JW, Noel AA, Jenkins GJ, Bower TC, Kenneth JC, et al. Early abdominal closure with mesh reduces multiple organ failure: guidelines from a 10-year case-control study. J Vasc Surg 2002; 35:246-53.
- Bown MJ, Nicholson ML, Bell PRF, Sayers RD. The systemic inflammatory response syndrome, organ failure and mortality after abdominal aortic aneurysm repair. J Vasc Surg 2003;37;600-6.
- O'Donnell ME, Badger SA, Makar RR, Loan W, Soong CV. Techniques in occluding the aorta during endovascular repair of ruptured abdominal aortic aneurysms. J Vasc Surg 2006;44:211-5.
- Fusco MA, Martin RS, Chang MC. Estimation of intra-abdominal pressure by bladder pressure measurement: validity and methodology. J Trauma 2001;50:297-302.
- Kron II, Harman PK, Nolan SP. The measurement of intra-abdominal pressure as a criterion for abdominal reexploration. Ann Surg 1984; 199:28-30.
- Hardman DT, Fisher CM, Patel MI, Neale M, Chambers J, Lane R, et al. Ruptured abdominal aortic aneurysm: who should be offered surgery? J Vasc Surg 1996;23:123-9.
- Bone RC, Balk R, Cerra FB, Dellinger RP, Fein AM, Knaus WA, et al. Definition for sepsis and organ failure and guidelines for the use of innovative therapies in sepsis. The ACCP/SCCM consensus conference committee. American College of Chest Physicians/Society of Critical Care Medicine. Chest 1992;101:1644-55.
- Murray JF, Matthay MA, Luce JM, Flick MR. An expanded definition of the adult respiratory distress syndrome. Am Rev Respir Dis 1988; 138:720-3.
- Marshall JC, Cook DJ, Christou NV, Bernard GR, Sprung CL, Sibbald WJ. Multiple organ dysfunction score: a reliable description of a complex clinical outcome. Crit Care Med 1995;23:1638-52.
- Talmore M, Hydo L, Barie PS. Relationship of systemic inflammatory response syndrome to organ dysfunction, length of stay, and mortality in critical surgical illness. Arch Surg 1999;134:81-7.
- Darling RC, Cordero JA, Chang BB, Shah DM, Paty PS, Lloyd WE, et al. Advances in surgical repair of ruptured abdominal aortic aneurysms. Cardiovasc Surg 1996;4:720-3.
- 23. Greenberg RK, Srivactava SD, Ouriel K, Waldman D, Ivancev K, Shortell C, et al. An endoluminal method of haemorrhage control and repair of ruptured abdominal aortic aneurysms. J Endovasc Ther 2000; 7:1-7.
- Brian G, Sanchez LA, Choi ET, Sicard GA. Endoluminal repair of ruptured abdominal aortic aneurysms under local anaesthesia: initial experience. Vasc Endovasc Surg 2004;38:203-7.

- Alsac JM, Desgranges P, Kobeiter H, Becquemin JP. Emergency endovascular repair for ruptured abdominal aortic aneurysms: feasibility and comparison of early results with conventional open repair. Eur J Vasc Endovasc Surg 2005;30:632-9.
- Hechelhammer L, Lachat ML, Wildermuth S, Bettex D, Mayer D, Pfammatter T. Midterm outcome of endovascular repair of ruptured abdominal aortic aneurysms. J Vasc Surg 2005;41:752-7.
- Peppelenbosch N, Gelkerken RH, Soong CV, Cao P, Steinmetz OK, Teijink JAW, et al. Endovascular treatment of ruptured abdominal aortic aneurysms using the Talent aortouniiliac system: an international multicenter study. J Vasc Surg 2006;43:1111-22.
- Hinchliffe RJ, Bruijstens L, MacSweeney STR, Braithwaite BD. A randomised trial of endovascular and open surgery for ruptured abdominal aortic aneurysm—results of a pilot study and lessons learned for rupture studies. Eur J Vasc Endovasc Surg 2006;32:506-13.
- Veith FJ, Ohki T, Lipsitz EC, Suggs WD, Cynamon J. Endovascular grafts and other catheter-directed techniques in the management of ruptured abdominal aortic aneurysms. Sem Vasc Surg 2003;16:326-31.
- Greco G, Rgorova N, Anderson PL, Gelijins A, Moskowitz A, Nowygrod R, et al. Outcomes of endovascular treatment of ruptured abdominal aortic aneurysm. J Vasc Surg 2006;43:453-9.
- Mahta M, Darling C, Roddy SP, Fecteau S, Ozsvath KJ, Kreienberg PB, et al. Factors associated with abdominal compartment syndrome complicating endovascular repair of ruptured abdominal aortic aneurysms. J Vasc Surg 2005;42:1047-51.
- 32. Balogh Z, McKinley BA, Cocanour CS, Kozar RA, Cox CS, Moore FA. Patients with impending abdominal compartment syndrome do not respond to early volume loading. Am J Surg 2003;186:602-6.

- Papavassilliou V, Anderton M, Loftus IM, Turner DA, Naylor AR, London NJM, et al. The physiological effects of elevated intra-abdominal pressure following aneurysm repair. Eur J Vasc Endovasc Surg 2003;26: 293-8.
- Cullen DJ, Coyle JP, Teplick R, Long MC. Cardiovascular, pulmonary, and renal effects of massively increased intra-abdominal pressure in critically ill parients. Crit Care Med 1989;17:118-21.
- 35. Arya N, Makar RR, Lau LL, Loan W, Lee B, Hannon RJ, et al. An intention-to-treat by endovascular repair policy may reduce overall mortality in ruptured abdominal aortic aneurysm. J Vasc Surg 2006;44: 467-71.
- 36. Hinchliffe RJ, Yusuf SW, Macierewicz JA, MacSweeney STR, Wenham PW, Hopkinson BR. Endovascular repair of ruptured abdominal aortic aneurysm–a challenge to open repair? Results of a single centre experience in 20 patients. Eur J Vasc Endovasc Surg 2001;22:528-34.
- Lau LL, Gardiner KR, Martin L, Halliday MI, Hannon RJ, Lee B, et al. Extraperitoneal approach reduces neutrophil activation, systemic inflammatory response and organ dysfunction in aneurysm surgery. Eur J Vasc Endovasc Surg 2001;21:326-33.
- Junnarkar S, Lau LL, Edrees WK, Underwood D, Smye MG, Lee B, et al. Cytokine activation and intestinal mucosal and renal dysfunction are reduced in endovascular AAA repair compared to surgery. J Endovasc Ther 2003;10:195-202.
- Burch JM, Moore EE, Moore FA, Franciose R. The abdominal compartment syndrome. Surg Clin North Am 1996;76:833-42.

Submitted Aug 8, 2008; accepted Nov 9, 2008.