View metadata, citation and similar papers at core.ac.uk
brought to you by CORE
provided by Elsevier - Publisher Connector

Eur J Vasc Endovasc Surg **26**, 293–298 (2003)
doi:10.1053/ejvs.2002.1930, available online at http://www.sciencedirect.com on SCIENCE DIRECT®

The Physiological Effects of Elevated Intra-abdominal Pressure Following Aneurysm Repair

V. Papavassiliou[1], M. Anderton[1], I. M. Loftus[1], D. A. Turner[2], A. R. Naylor[1], N. J. M. London[1], P. R. F. Bell[1] and M. M. Thompson*[3]

Departments of [1]Vascular and Endovascular Surgery and [2]Intensive Care, Leicester Royal Infirmary, U.K. and [3]Department of Vascular Surgery and Cardiovascular Research Group, St George's Hospital Medical School, London, U.K.

Objectives: *elevated intra-abdominal pressure (IAP) may cause widespread organ dysfunction (abdominal compartment syndrome) through effects on the respiratory, cardiac, renal and gastro-intestinal systems. The aim of this study was to document IAP following aneurysm surgery, and to determine the effect of IAH on outcome.*
Design: *prospective observational study.*
Setting: *University Hospital.*
Subjects: *the patient cohort comprised 75 patients undergoing infra-renal aneurysm repair (53 non-ruptured [40 conventional – 1 death, 13 endovascular] and 22 conventionally repaired ruptured AAA – 8 deaths). IAP was quantified by bladder manometry at the termination of the procedure and at 24 h intervals in patients who remained intubated. Physiological indices of organ function were recorded. Statistical analysis utilized the unpaired* t*-test, Fischer's exact test and Pearson's correlation.*
Results: *IAP was significantly higher at abdominal closure following ruptured aneurysm repair (15.4 mmHg [SE 1.6]) than conventional (10.5 [0.89]) or endovascular elective repair (6.4 [1.0]) of non-ruptured AAA. The sensitivity and specificity of IAP to predict subsequent mortality was analysed using a receiver characteristic operating curve. This analysis demonstrated that a cut off of 15 mmHg was the most useful for indicating patients at risk (sensitivity 0.66, specificity 0.79).*

Physiological indices of organ dysfunction (pH[$p = 0.027$*], base excess [*$p = 0.005$*], peak inspiratory pressure [*$p = 0.0015$*], CVP and urine output [*$p = 0.0029$*]) were significantly impaired in patients with IAP* $\geqslant$ *15 mmHg, in comparison to patients with lower pressures. IAP correlated significantly with indices of cardiac (CVP* $p = 0.038$*), respiratory (*PaO_2/FiO_2*,* $p = 0.026$*), and renal function (urine output* $p = 0.046$*).*
Conclusions: *these data suggest that the management of IAH may have a role following repair of ruptured AAA. High intra-abdominal pressures rarely complicate elective or endovascular aneurysm repair.*

Key Words: Abdominal aortic aneurysm; Abdominal compartment syndrome; Multiple organ failure.

Introduction

Elevated intra-abdominal pressure (IAP) may affect visceral perfusion, cardiac output, respiratory function, renal function and cerebro-spinal pressure.[1] The combination of elevated IAP and physiological derangement is termed the abdominal compartment syndrome (ACS). The mainstay of treatment for the abdominal compartment syndrome is abdominal decompression, which may rapidly reverse organ dysfunction. A primary elevation of IAP may occur due to increased intra-abdominal or retroperitoneal volume, or conversely, a decrease in the volume of the intra-abdominal compartment. Less commonly, secondary elevation of IAP may accompany massive fluid resuscitation due to visceral oedema.[2]

Hypothetically, given the aetiology of this condition, the ACS might be expected to occur relatively frequently following surgery for ruptured abdominal aortic aneurysms (AAA). Surprisingly, there are relatively few reports of elevated IAP pressure complicating aortic surgery.[3,4] The prevalence of elevated IAP and ACS after elective and ruptured aneurysm surgery remains undefined.

The relevance of determining the frequency of intra-abdominal hypertension following aortic surgery relates to the high incidence of systemic inflammation and organ dysfunction following repair of ruptured

* Please address all correspondence to: M. M. Thompson, Professor of Vascular Surgery, Department of Vascular Surgery, 4th Floor St James Wing, St George's Hospital, Blackshaw Rd, London SW17 0QT, U.K.

1078–5884/03/030293 + 06 $35.00/0 © 2003 Elsevier Science Ltd. All rights reserved.

AAA,[5] which may affect virtually all patients. An acute increase in IAP has previously been shown to cause multi-organ failure and systemic inflammation[6,7] and there may be a relationship between IAP and the clinical outcome of aortic surgery.

The aim of the present study was to quantify IAP after aneurysm surgery, and to define any relationship between intra-abdominal hypertension and physiological dysfunction.

Methods

Study design

The study was designed as a prospective observational investigation into the effects of raised IAP in patients undergoing infra-renal aneurysm repair, and was approved by the Leicestershire Research Ethics Committee. A non-consecutive cohort of patients having repair of ruptured or non-ruptured AAA was studied between 25/1/2000 and 5/10/2001. All patients were admitted to the intensive care unit (ITU) for at least 24 h following surgery.

Patients underwent IAP measurement by bladder manometry at the termination of the surgical procedure and selectively, 24 h post-operatively and at daily intervals.[8] Bladder manometry was utilized as the optimum method for quantifying IAP as it closely correlates with abdominal pressures between 5 and 50 mmHg.[9] Standard physiological measurements of post-operative care were recorded on the intensive care unit, within one hour of bladder manometry (Table 1). Patient outcome was prospectively recorded.

Patient cohort

Patients undergoing conventional ($n = 40$, age 72.5 [1.2], five females) or endovascular ($n = 13$, age 72.2 [2.2], 3 females) repair of non-ruptured AAA, and patients having conventional repair of ruptured AAA ($n = 22$, age 71.5 [1.1], two females) were included in the study. Overall, there were nine deaths, eight following aneurysm rupture (38%) and one after conventional elective surgery (2.5%). All patients undergoing repair of ruptured AAA were conscious prior to surgery and none had a pre-operative cardiac arrest. All patients in the study undergoing conventional open repair of abdominal aneurysms had a standard abdominal closure. None had a temporary or mesh closure within the timeframe of the study.

Mortality from ruptured aneurysm surgery was due to ongoing bleeding (two patients), renal failure (three), pulmonary oedema (one) and cardiac failure (two). The death in the elective group was secondary to aspiration pneumionia and subsequent multiple organ failure.

Measurement of intra-abdominal pressure

Intra-abdominal pressure was quantified using urinary bladder manometry as described by Kron *et al*.[8] Initially the bladder was drained using a Foley urinary catheter. Subsequently, 50 ml of sterile saline were instilled into the bladder. The catheter was clamped beyond the aspiration port, and a 16-guage needle used to connect the aspiration port to a pressure transducer, using the symphysis pubis as zero. Several studies have shown a close correlation between intra-vesical pressure and the true IAP.[9,10]

Bladder pressure measurements were taken immediately following surgery and selectively at 24 h intervals. IAP was only quantified in patients who were intubated and sedated, as unpublished data from our group have demonstrated that IAP measurements in patients who have been recently extubated are not reproducible due to the effects of respiration and abdominal contraction due to pain.

Statistical analysis

Results of IAP measurement and organ dysfunction were initially analysed using Kolmogorov–Smirnov test for normal distribution. The data passed this test

Table 1. Physiological variables measured on patient cohort.

Cardiac	Respiratory	Renal	Metabolic
Systemic arterial pressure	Peak inspiratory pressure (PIP)	Serum creatinine	pH (arterial blood)
Central venous pressure	Peak end expiratory pressure (PEEP)	Urine output (24 h)	Base excess (arterial blood)
	PaO_2/FiO_2		Platelet count
	PaO_2		

PaO_2 – Partial pressure of oxygen in arterial blood (kPa). FiO_2 – Inspired oxygen concentration.

and were presented as mean values with standard errors. Continuous variables were compared with the unpaired *t*-test, and discrete variables using Fischer's exact test. Analysis of correlation was performed using Pearson's correlation co-efficient.

Results

Intra-abdominal pressure following aneurysm repair

The intra-abdominal pressure at the end of surgery for conventional repair of ruptured (mean IAP 15.4 mmHg [SEM 1.6]) and non-ruptured (10.5 mmHg [0.89], $p = 0.0057$, 95% CI 1.46–8.22) aneurysms is illustrated in Figure 1. IAP following endovascular repair of non-ruptured aneurysms was included as a control (6.3 mmHg [1.04]). The mean IAP 24 and 48 h following ruptured aneurysm repair was 15.7 mmHg [1.4] and 12.9 mmHg [1.1] respectively. There were insufficient patients who remained intubated and sedated after 24 h in the elective or endovascular groups for comparative pressure measurements at 24 h.

Prediction of mortality – sensitivity and specificity of IAP

The ability of IAP measured immediately post-operatively, to predict subsequent mortality was investigated in the cohort of 62 patients undergoing conventional aneurysm surgery (22 ruptured AAA, 40 non-ruptured). Overall, there were nine deaths, eight in patients having ruptured aneurysms. The sensitivity, specificity, positive and negative predictive values for IAP between 6 and 24 mmHg were calculated and are illustrated in Table 2.

The optimum pressure for predicting mortality was determined by a receiver operating characteristic (ROC) curve (Fig. 2). The best cut-off is that which maximizes both sensitivity and specificity. Graphically this is represented by the point nearest the left-hand top corner of the graph, 15 mmHg. The area under the OC curve was 0.68 compared to 0.5 for the control line.

Overall 17 of the 62 patients (27%) had IAP $\geqslant$ 15 mmHg. In the ruptured group 54% (12/22) of the patients had a pressure above 15 mmHg in comparison to 13% (5/40) of the elective group. The mortality for the various groups and abdominal pressures are documented in Table 3.

Table 2. Diagnostic parameters for the ability of IAP immediately post-operatively to predict mortality. Sensitivity, specificity, positive predictive values (PPV) and negative predictive values (NPV) were calculated for a range of intra-abdominal pressures (IAP) from 6 to 24 mmHg.

IAP (mmHg)	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
6	100	11	16	100
8	77	19	14	83
10	78	41	18	92
12	78	54	23	94
14	66	73	30	93
15	66	79	35	93
16	55	81	33	91
18	33	85	27	88
20	11	87	13	85
22	11	91	17	85
24	11	94	25	86

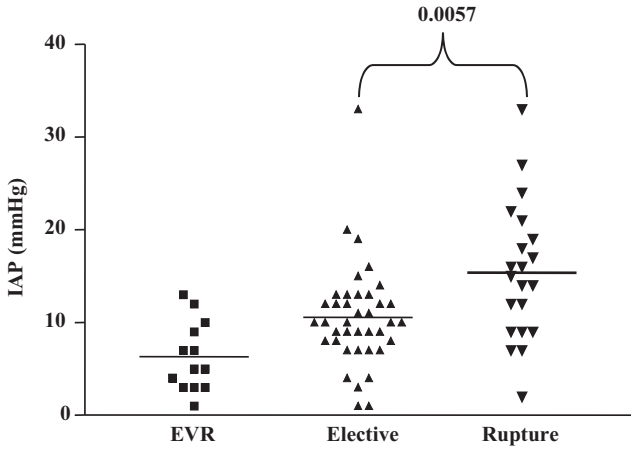

Fig. 1. Graph illustrating intra-abdominal pressure immediately following conventional repair of non-ruptured (elective) and ruptured abdominal aortic aneurysms (rupture), and for endovascular repair of non-ruptured aneurysm (EVR).

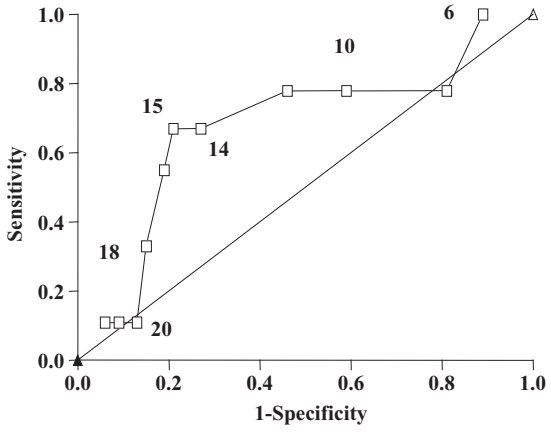

Fig. 2. Receiver operating characteristic (ROC) curve, plotting the sensitivity of post-operative IAP to predict mortality against 1-specificity. Pressures between 6 and 24 mmHg were investigated. Individual data points are annotated with the IAP. A control line passing through 0 and 1 is given for comparison. The optimum cut-off for mortality prediction is 15 mmHg.

Organ function and intra-abdominal pressure

The optimum pressure for predicting mortality was then used to analyse differences in physiology between patients with IAP < 15 mmHg and those with IAP 15 mmHg. Pressures determined immediately postoperatively and in subsequent days were used, with the corresponding physiological variables. The analysis of physiological function of the metabolic, renal, respiratory and cardiac systems is illustrated in Table 4. These data demonstrated that patients with IAP ⩾ 15 mmHg had significantly impaired indices of organ function when compared to patients with IAP < 15 mmHg. The table contains data from all the IAP measurements determined at abdominal closure. As IAP was only quantified in patients who were intubated and sedated, at the 24 h time period, all the ruptured group were included, but only 19 of the elective patients had their IAP quantified. Independent analysis of physiological variables at abdominal closure and 24 h time periods is not presented as the numbers in each group are too small.

The relationship between IAP and organ function was further examined by correlation analysis (Table 5). There were significant correlations between IAP and many indices of organ function, although not with pH or peak inspiratory pressure. Despite the statistical significance of these results, most of the correlations were relatively weak, between 20 and 34%. This may not be surprising in view of the multiple factors responsible for organ dysfunction following aneurysm surgery.

Table 3. Mortality in the different patient categories according to IAP at abdominal closure.

	IAP ⩾ 15 mmHg	IAP < 15 mmHg
Elective-survived	5	34
Elective-died	0	1
Ruptured-survived	6	8
Ruptured-died	6	2

Discussion

The influence of the abdominal compartment syndrome on the outcome of aortic aneurysm repair has received relatively little attention. Kron *et al*.[8] reported four cases of ACS in patients with aortic aneurysms in 1984. This small series demonstrated that decompression of the abdomen when the IAP exceeded 25 mmHg improved renal function, and may have influenced survival. Similarly, Fietsam[3] and colleagues reported a 4% incidence of ACS after repair of ruptured AAA.

Intuitively, it would be expected the IAP would be elevated in patients following repair of ruptured AAA, due to the volume of fluid resuscitation and the presence of retroperitoneal heamorrhage. The present study demonstrated a significant elevation of IAP following ruptured AAA surgery when compared to transperitoneal or endovascular repair of elective aneurysms.

The influence of IAP measured immediately after abdominal closure, to predict subsequent mortality was examined using sensitivity/specificity analysis. Interestingly, although the positive predictive value of high IAP's was not great, the negative predictive

Table 5. The correlation between intra-abdominal pressure and physiological variables.

Parameter	Pearson *r*	*p*	95% CI
pH	−0.13	0.19	0.31 to 0.064
BE	−0.20	0.034	−0.37 to −0.16
CVP (mmHg)	0.21	0.038	0.012 to 0.39
PIP (mmHg)	0.15	0.24	−0.097 to 0.37
Urine output (ml/24 h)	−0.24	0.046	−0.44 to −0.005
Creatinine	0.30	0.0018	0.12 to 0.47
Platelets	−0.34	0.0005	−0.50 to −0.16
PaO_2	−0.23	0.016	−0.40 to −0.045
PaO_2/FiO_2	−0.22	0.026	−0.40 to −0.035

BE (base excess), CVP (central venous pressure), PIP (peak inspiratory pressure), PaO_2 (partial pressure oxygen), PaO_2/FiO_2 (oxygenation index). Pearson *r* (Pearson's correlation coefficient). 95% CI (95% confidence intervals).

Table 4. The difference in pH, base excess (BE), central venous pressure (CVP), peak inspiratory pressure (PIP), urine output (24 h), and creatinine in two groups of patients with intra-abdominal pressure (IAP) above and below 15 mmHg.

Parameter	IAP ⩾ 15 mmHg ($n = 50$)	IAP < 15 mmHg ($n = 90$)	*p*	95% CI
pH	7.27 (0.025)	7.33 (0.015)	0.028	−0.12 to −0.007
BE	−3.13 (0.48)	−1.15 (0.3)	0.0005	−3.05 to −0.89
CVP (mmHg)	12.1 (0.61)	8.26 (0.41)	<0.0001	2.43 to 5.23
PIP (mmHg)	24.9 (0.87)	21.4 (0.62)	0.0015	1.37 to 5.54
Urine output (ml/24 h)	1526 (180.4)	2104 (100.5)	0.0029	−956 to −201
Creatinine	178.2 (13.2)	115.8 (5.3)	<0.0001	38.7 to 85.9

Physiological data passed the Kolmogorov–Smirnov test for normal distribution, and were presented as mean values with SEM in parentheses. Data were analysed using the unpaired *t*-test.

value of a low IAP appeared to be a reasonable guide of a favorable outcome. Using the ROC analysis, the optimum combination for sensitivity/specificity in predicting mortality was observed at 15 mmHg. This pressure was therefore used to stratify patients into two groups for subsequent physiological analysis. The critical pressure of 15 mmHg was lower than observed in studies on trauma patients, where the threshold for diagnosis and intervention was often above 20 mmHg.[11,12]

These differences in threshold pressure for ACS in trauma and aneurysm patients may well reflect a lower tolerance to elevated IAP during aneurysm surgery due to the additional insults of aortic cross clamping and increased age. Platell *et al*.[4] described a group of patients who required decompressive laparotomy following aortic reconstruction and suggested that an IAP greater than 18 mmHg was a significant risk factor for the development of renal dysfunction.

Previous experimental and clinical studies have documented convincing relationships between raised IAP and physiological dysfunction in respiratory, renal, cardiovascular, metabolic and gastrointestinal systems. In the present study, the effect of raised IAP after aortic surgery was assessed in two cohorts of patients separated by an IAP of 15 mmHg. The data demonstrated that patients with an IAP $\geqslant$ 15 mmHg had significant physiological impairment in a range of organ systems.

To ensure that the division of the patients by an IAP of 15 mmHg was not solely responsible for these findings, the IAP was correlated with physiological function. Again, a convincing relationship was described between IAP and metabolic (base excess), cardiovascular (CVP), renal (urine output/creatinine), and respiratory (oxygenation index) function. These data revealed that IAP plays a demonstrable role in physiologic function following aortic reconstruction.

The correlations described were statistically significant but were relatively weak with coefficients of approximately 25%. The low correlation coefficients might be expected from the composition of the study group, which included both ruptured and non-ruptured aneurysms, and from other factors that would influence organ function following aortic surgery (age, pre-existing co-morbidity, type of reconstruction, clamp time, blood transfusion etc.).

Data from this investigation have suggested that the IAP following aortic surgery may play a role in subsequent physiological function and therefore outcome. There are however, significant limitations to the study. The principle concern is the diverse patient mix in the study cohort, which included both ruptured and non-ruptured aneurysms, and the low numbers of patients with ruptured aneurysms. Patients with ruptured aneurysms had elevated IAP when compared to the non-ruptured group. Clearly, patients with ruptured AAA would also be expected to have impaired physiologic function. The relationship between IAP and physiologic function might therefore be related to the patient mix rather than a true reflection of the effect of IAP on organ function.

Unfortunately, there were insufficient patients with ruptured aneurysms in this study to allow analysis of this group in isolation or to justify a multiple regression analysis. Further multi-center investigations to observe the effect of IAP on mortality following ruptured AAA repair, will be needed to define the precise relationship between IAP and outcome. Hopefully such a study might allow a clinical trial of IAP management to be considered following ruptured aneurysm repair.

In addition, the relationship between the physiological variables and IAP have been analysed using data from the entire time course of the study and not for individual days, although all data points are matched for IAP and the corresponding physiological variable. Again, larger scale studies will be required to determine the time course of the IAP/physiological response.

Two contemporary studies have also suggested that management of IAP may be crucial in determining the outcome of ruptured AAA repair. In 1997, Oelschlager *et al*.[13] reviewed 23 patients who survived ruptured aneurysm surgery. In a sub-group of eight patients whose abdominal wounds were left open at the time of surgery and underwent delayed closure, there was improved oxygenation and a trend to increased survival. More recently, Rasmussen *et al*.[14] reported a case control study of 45 patients who required mesh closure following ruptured AAA repair, in comparison to 90 patients who underwent traditional abdominal closure. The decision to utilise a mesh closure was made on clinical grounds. The most significant findings related to a comparison of patients who underwent mesh closure at the original operation and those requiring decompressive laparotomy following development of ACS. Patients undergoing initial mesh closure had lower mortality rates and a lower incidence of multiple organ failure, than those requiring a second operation for ACS.

The pressure of 15 mmHg identified in the present study is not suggested as a criterion for abdominal decompression in the post-operative management of ruptured AAA. Further, larger studies concentrating solely on aortic ruptures will be required to define a threshold for intervention, the time course of the pressure changes, and to determine whether high

abdominal pressures following repair of ruptured AAA are merely an inevitable consequence of aortic rupture or an important cause of post-operative mortality. However, the physiological findings of the present study, suggest that management of IAP and the ACS might become be a routine facet of peri-operative care following repair of ruptured AAA.

References

1 Saggi BH, Sugerman HJ, Ivatury RR, Bloomfield GL. Abdominal compartment syndrome. *J Trauma* 1998; **45**: 597–609.
2 Ivatury RR, Sugerman HJ. Abdominal compartment syndrome: a century later, isn't it time to pay attention? *Crit Care Med* 2000; **28**: 2137–2138.
3 Fietsam R, Jr, Villalba M, Glover JL, Clark K. Intra-abdominal compartment syndrome as a complication of ruptured abdominal aortic aneurysm repair. *Am Surg* 1989; **55**: 396–402.
4 Platell CF, Hall J, Clarke G, Lawrence-Brown M. Intra-abdominal pressure and renal function after surgery to the abdominal aorta. *Aust N Z J Surg* 1990; **60**: 213–216.
5 Bown MJ, Nicholson ML, Bell PR, Sayers RD. Cytokines and inflammatory pathways in the pathogenesis of multiple organ failure following abdominal aortic aneurysm repair. *Eur J Vasc Endovasc Surg* 2001; **22**: 485–495.
6 Sugerman HJ, Bloomfield GL, Saggi BW. Multisystem organ failure secondary to increased intra-abdominal pressure. *Infection* 1999; **27**: 61–66.
7 Doty JM, Oda J, Ivatury RR *et al*. The effects of hemodynamic shock and increased intra-abdominal pressure on bacterial translocation. *J Trauma* 2002; **52**: 13–17.
8 Kron IL, Harman PK, Nolan SP. The measurement of intra-abdominal pressure as a criterion for abdominal re-exploration. *Ann Surg* 1984; **199**: 28–30.
9 Fusco MA, Martin RS, Chang MC. Estimation of intra-abdominal pressure by bladder pressure measurement: validity and methodology. *J Trauma* 2001; **50**: 297–302.
10 Ridings PC, Bloomfield GL, Blocher CR, Sugerman HJ. Cardiopulmonary effects of raised intra-abdominal pressure before and after intravascular volume expansion. *J Trauma* 1995; **39**: 1071–1075.
11 Meldrum DR, Moore FA, Moore EE, Franciose RJ, Sauaia A, Burch JM. Prospective characterization and selective management of the abdominal compartment syndrome. *Am J Surg* 1997; **174**: 667–672.
12 Burch JM, Moore EE, Moore FA, Franciose R. The abdominal compartment syndrome. *Surg Clin North Am* 1996; **76**: 833–842.
13 Oelschlager BK, Boyle EM, Jr, Johansen K, Meissner MH. Delayed abdominal closure in the management of ruptured abdominal aortic aneurysms. *Am J Surg* 1997; **173**: 411–415.
14 Rasmussen TE, Hallett JW, Jr, Noel AA *et al*. Early abdominal closure with mesh reduces multiple organ failure after ruptured abdominal aortic aneurysm repair: guidelines from a 10-year case-control study. *J Vasc Surg* 2002; **35**: 246–253.

Accepted 6 March 2003