Original article

Colonic ischaemia and intra-abdominal hypertension following open repair of ruptured abdominal aortic aneurysm

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Background: The aim was to investigate the association between colonic ischaemia and intra-abdominal pressure (IAP) after surgery for ruptured abdominal aortic aneurysm (rAAA).

Methods: Sigmoid colon perfusion was monitored with an intramucosal pH (pHi) tonometer. Patients with a pHi of 7·1 or less were treated for suspected hypovolaemia with intravenous colloids and colonoscopy. IAP was measured every 4 h. Patients with an IAP of 20 mmHg or more had neuromuscular blockade, relaparotomy or both.

Results: A total of 52 consecutive patients had open rAAA repair; 30-day mortality was 27 per cent. Eight patients died shortly after surgery. Fifteen were not monitored for practical reasons; mortality in this group was 33 per cent. IAP and pHi were measured throughout the stay in intensive care in the remaining 29 patients. Monitoring led to volume resuscitation in 25 patients, neuromuscular blockade in 16, colonoscopy in 19 and relaparotomy in two. One patient died in this group. Twenty-three of 29 patients had a pHi of 7·1 or less, of whom 15 had a pHi of 6·9 or less. Sixteen had an IAP of 20 mmHg or more, of whom ten also had a pHi below 6·90. Peak IAP values correlated with the simultaneously measured pHi ($r = -0{\cdot}39$, $P = 0{\cdot}003$).

Conclusion: Raised IAP is an important mechanism behind colonic hypoperfusion after rAAA repair. Monitoring IAP and timely intervention may improve outcome.

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Introduction

Colonic ischaemia is a serious complication after abdominal aortic aneurysm (AAA) repair. Longo and colleagues[1] reported an incidence of 1·2 per cent of 4957 operations from the US Veterans Affairs Registry, but they did not state whether any of those operations were for ruptured AAA (rAAA). In a study of 2930 aortoiliac operations prospectively registered in the Swedish Vascular Registry (Swedvasc), the frequency of clinically evident transmural bowel gangrene was 2·8 per cent, but it was 7·3 per cent in 412 patients who had repair of an rAAA with preoperative shock, and 23 per cent of the deaths were associated with colonic ischaemia[2]. Independent risk factors for colonic ischaemia in the entire cohort were preoperative shock, renal insufficiency, emergency surgery, age, type of hospital, aortobifemoral grafting, operating and cross-clamping times as well as ligation of the internal iliac arteries[3]. Becquemin and co-workers[4] studied 1174 patients who had AAA surgery between 1995 and 2005 (492 with endovascular repair (EVAR), 88 for rupture) with similar conclusions regarding both incidence and risk factors. Several investigators have performed routine postoperative sigmoidoscopy after surgery for rAAA, reporting colonic ischaemia in about half the patients studied[5,6]. Champagne and colleagues[6] reported 36 per cent colonic ischaemia at sigmoidoscopy in 62 patients who survived rAAA more than 24 h, but only 14·5 per cent required colonic resection.

The perfusion of the sigmoid colon can be monitored by measuring the carbon dioxide level in a balloon catheter placed there through a colonoscope; the method is usually referred to as tonometry or intramucosal pH (pHi) measurement. This method, pioneered by Fiddian-Green and coworkers[5,7], was used in two prospective studies

showing that prolonged hypoperfusion of the sigmoid colon, defined as pHi of 7·1 or less, was strongly associated with adverse outcome after AAA repair[8,9].

Intra-abdominal hypertension (IAH) has been recognized as an important factor contributing to postoperative organ dysfunction after AAA surgery[10–12]. Only one study has reported prospective monitoring of the intra-abdominal pressure (IAP) after AAA surgery[11]; the study reported a relationship between IAH and clinical colonic ischaemia. However, no study has addressed the possible relationship between IAH and colonic ischaemia measured prospectively with pHi.

The aim of this investigation was to study the association between colonic ischaemia and IAP after repair of an rAAA.

Methods

All patients who had a repair of an rAAA at Gävle County Hospital and Uppsala University Hospital between April 2003 and December 2005 were included in this study.

A balloon catheter was placed in the lumen of the sigmoid colon with the aid of a colonoscope in the operating theatre at the end of the aneurysm surgery or immediately after admission to the intensive care unit. Intraluminal partial pressure of carbon dioxide (PCO_2) was measured automatically every 10 min with a Tonocap® device (GE Healthcare, Helsinki, Finland) and pHi was calculated by entering the tonometrically measured PCO_2 and the arterial bicarbonate concentration into the Henderson–Hasselbalch equation[7] every 4 h for 72 h, then every 6–8 h for as long as the patient was treated in intensive care, or until the catheter was expelled by return of intestinal function. If the catheter was expelled within the first 72 h, it was repositioned after a diagnostic colonoscopy, as early bowel movement may be a sign of colonic ischaemia. When low pHi values were registered, more frequent measurements were performed. Colonoscopy was indicated when persistent low pHi values were recorded, to make sure that the balloon catheter was in contact with the mucosa (to rule out that faecal contamination affected the readings) and to assess the grade of ischaemic injury. IAP was measured in the bladder every 4 h after infusion of 50 ml of saline solution, as described by Kron and colleagues[13].

A pHi of 7·1 or less and an IAP of at least 20 mmHg were considered thresholds for intervention. Experimental studies have shown that supply-dependent oxygen consumption develops at a pHi of 7·1, and below this level an anaerobic metabolism prevails[14]. Clinical studies have suggested that this threshold is associated with colonic ischaemia and major complications[5,7–9]. Abdominal compartment syndrome was defined as an IAP of at least 20 mmHg accompanied by organ dysfunction (cardiac, respiratory or renal)[15].

The study was open; monitoring results were known to the investigators and staff, who based their interventions on the results. Patients with a pHi of 7·1 or less were treated for suspected hypovolaemia with intravenous colloids (mainly plasma), and those who continued to have low pHi despite volume resuscitation underwent colonoscopy. Patients with an IAP of at least 20 mmHg were treated with diuretics, colloids and neuromuscular blockade (as described in the recommendations from the consensus conference[16]) and decompression laparotomy when indicated. Demographic details and risk factors were recorded prospectively and conventional intensive care monitoring was undertaken. All patients were followed until death or a minimum of 3 years after hospital discharge. In November 2008 survival was cross-checked against the Swedish population registry.

The study was approved by the Research Ethics Committee of Uppsala University Hospital.

Statistical analysis

Statistical analysis was carried out using SPSS® version 14 (SPSS, Chicago, Illinois, USA). The Spearman rank test was used to analyse the correlation between pHi and IAP. Fisher's exact test was used for comparison of two proportions, Kendall's tau-b test to measure associations of ordinal variables, and Wilcoxon's rank sum test for comparison of age. $P < 0{\cdot}050$ was considered significant.

Results

A total of 52 patients underwent open repair for rAAA at the two hospitals during the study. None died during surgery, but eight died shortly afterwards (mean 5·5 (range 1–13) h). They were all at least 80 years of age and with multiple co-morbidities (*Table 1*). The short postoperative period precluded any meaningful postoperative monitoring.

Among the remaining 44 patients, 15 did not undergo pHi monitoring for the following reasons: the pHi measuring device was occupied by another patient (two patients), the tonometer could not be placed because of large amounts of faeces (two), previous sigmoid colon resection with colostomy (one), previous cystectomy precluded IAP monitoring (one), no room in the intensive care unit (one) and unavailability of a researcher (eight). The 30-day mortality was five of 15 (33 per cent) in those not monitored compared with one of 29 (3 per cent) in the patients who underwent the monitoring protocol.

Table 1 Baseline characteristics, preoperative risk factors and outcomes in patients who had repair of ruptured abdominal aortic aneurysm

	Monitored ($n = 29$)	Not monitored ($n = 15$)	P‡	Early death ($n = 8$)
Sex ratio (M : F)	24 : 5	12 : 3		6 : 2
Age (years)*	73 (58–86)	78 (64–87)	0·072§	83 (80–88)
Preoperative risk factors				
Cardiac disease	12	9	0·342	8
Hypertension	17	9	1·000	7
Renal disease	3	2	1·000	2
Pulmonary disease	10	4	0·738	4
Cerebrovascular disease	1	2	0·264	1
Diabetes mellitus	6	6	0·284	3
Preoperative shock	20	7	0·738	3
Outcome				
Colonic ischaemia	2	2	0·596	1
Inotropic support > 48 h	7	9	0·026	5
Renal replacement therapy	7	4	1·000	2
Reoperation	5	7	0·071	2
Ventilator support > 48 h	12	8	0·532	4
In-hospital mortality†	2	5	0·036	8

*Values are mean (range). †One monitored patient died on day 39; all other deaths were within 30 days of surgery. ‡Comparison between monitored and non-monitored patients, excluding those who died early; Fisher's exact test unless indicated otherwise; §Wilcoxon's rank sum test.

Table 2 Clinical details in 29 monitored patients, grouped according to minimum intramucosal pH

	Minimum intramucosal pH			
	≥ 7·1	> 6·9 < 7·1	≤ 6·9	P†
No. of patients	6	8	15	
Age (years)*	75(59–86)	71(58–83)	74(64–85)	
Perioperative risk factors				
Preoperative shock	4	4	12	0·309
Cross-clamping > 120 min	2	2	10	0·049
Perioperative bleeding ≥ 5 litres	1	2	9	0·018
Postoperative renal failure	1	4	2	0·346
Postoperative pulmonary failure	1	3	8	0·096
Postoperative cardiac failure	1	1	5	0·061
Intra-abdominal pressure ≥ 20 mmHg	1	5	10	0·066
Treatment and outcome				
Volume resuscitation	2	8	15	0·011
Neuromuscular blockade	1	5	10	0·066
Colonoscopy	1	5	13	0·001
Mucosal or transmural colonic gangrene	0	0	2	0·129
In-hospital mortality	1	1	0	0·158

*Values are mean (range). †Kendall's tau-b test.

A second monitored patient died in hospital on day 39 from urosepsis and organ failure, thus in-hospital mortality in the monitored patients was 7 per cent. Baseline characteristics and clinical details of the three groups are shown in *Table 1*.

Twenty-nine patients were monitored throughout the postoperative period. They were monitored for a median of 68 (range 24–152) h. A pHi of 7·1 or less occurred in 23 and a pHi of 6·9 or less in 15. Among these 15 patients, the pHi of less than 6·9 was detected on the very first measurement on arrival to the intensive care unit in ten, on day 2 after surgery in three, on day 3 in one and on day 4 in one.

Sixteen patients had an IAP of 20 mmHg or more, of whom ten also had a pHi below 6·90. An IAP of 20 mmHg or more was detected on day 1 after surgery in ten, on day 2 in four, and on day 3 in two. The temporal relationships of IAH and colonic ischaemia in the ten patients who developed both and IAP of 20 mmHg or more and pHi below 6·90 are shown in *Fig. 1*.

There was a significant correlation between all simultaneously measured pHi and IAP values (−0·19, $P < 0·001$). The correlation between the lowest pHi registered for each patient and the corresponding, simultaneously measured, IAP was −0·27 ($P = 0·047$) (*Fig. 2*). The correlation between the highest IAP registered for each patient and the simultaneously measured pHi was −0·39 ($P = 0·003$) (*Fig. 3*).

Five patients (10 per cent) developed clinically significant colonic ischaemia, only two in the monitored group. One had a pHi below 6·7 for 12 h, and the IAP was 28 mmHg for 1 h, on the first postoperative day (*Fig. 1a*). Colonoscopy revealed mucosal gangrene and the patient was treated conservatively for hypovolaemia and with neuromuscular blockade to reduce IAP, successfully. The correlation between pHi and IAP in this particular patient was −0·71 ($P < 0·001$). The other patient developed sigmoid colon gangrene and had sigmoid resection. The pHi was 6·8 for 2 h and the IAP was 24 mmHg for 4 h, on day 2 after surgery (*Fig. 1e*). The correlation between pHi and IAP in this patient was −0·57 ($P = 0·061$). Both patients recovered after treatment.

One patient had a perforation of the sigmoid colon, probably a complication relating to placement of the

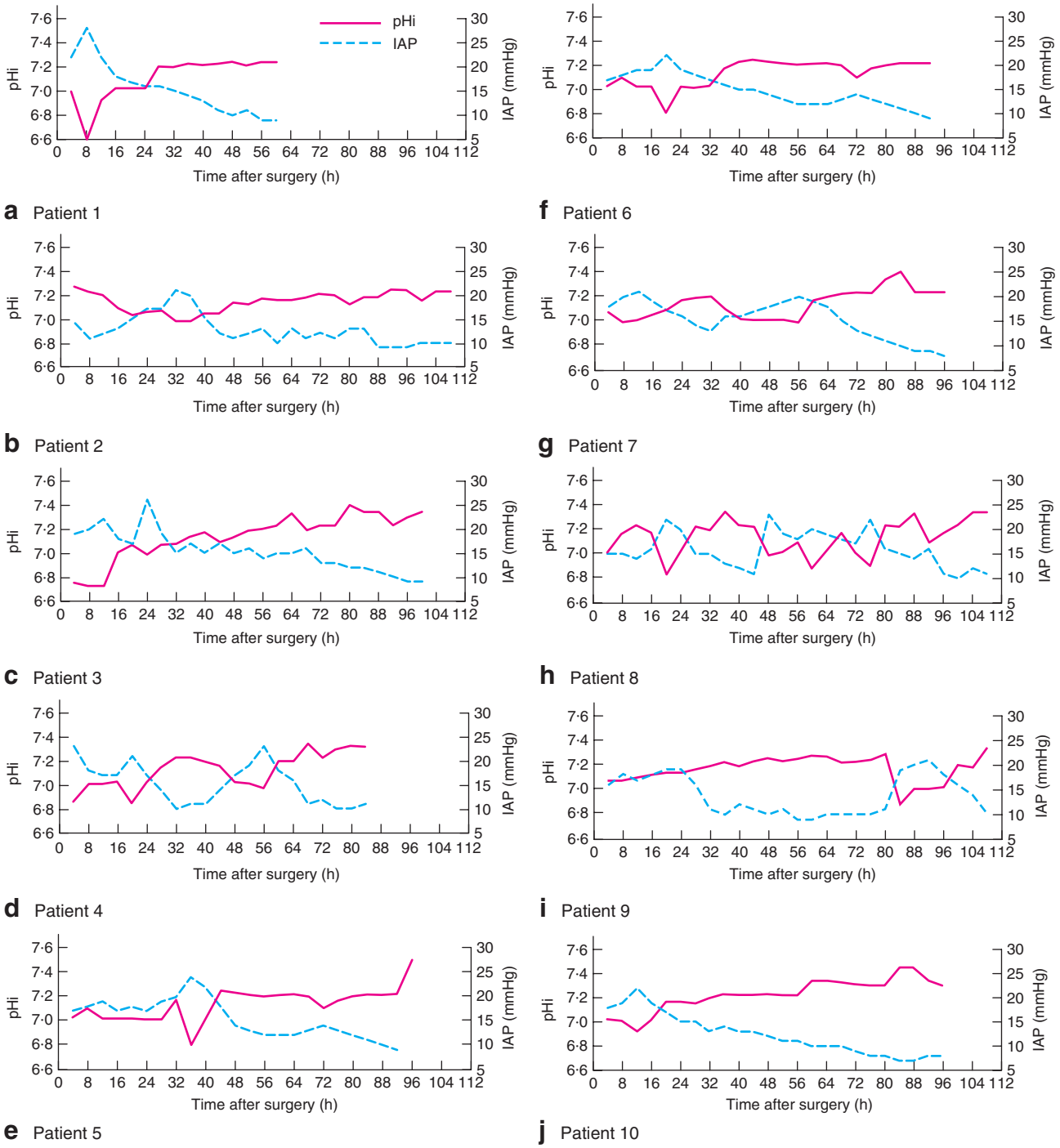

Fig. 1 The temporal relationship between intra-abdominal hypertension (IAH) and intramucosal pH (pHi) in ten patients who developed both intra-abdominal pressure (IAP) of atleast 20 mmHg and pHi below 6·90

tonometer catheter. The patient had diverticular disease, and placing the catheter by colonoscopy was technically difficult. IAP and pHi were normal for 3 days. On day 4 the patient developed fever and abdominal pain. At relaparotomy a perforation of the sigmoid colon was identified. The sigmoid colon was resected, and the specimen showed no signs of ischaemia. Further recovery was uneventful.

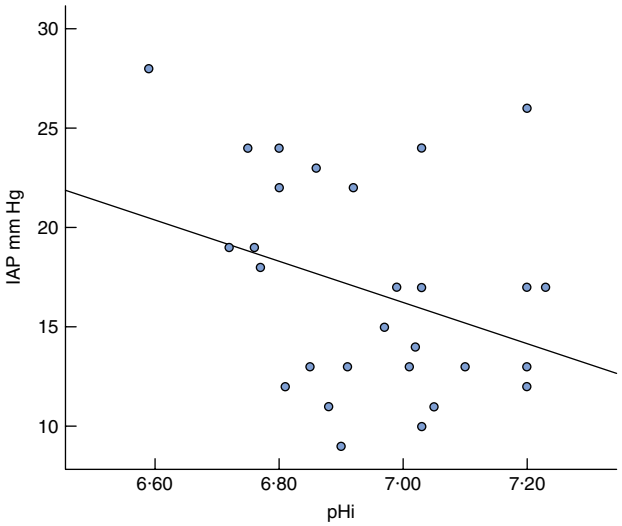

Fig. 2 The lowest intramucosal pH (pHi) value registered for each patient against its corresponding intra-abdominal pressure (IAP), with line of regression

There were two patients who were monitored and died during their hospital stay without colonic ischaemia. The first was 80 years old with a history of cardiac disease and hypertension, in shock on arrival. There was prolonged clamping (120 min) and heavy blood loss (6·5 litres). There was no evidence of colonic ischaemia or IAH after surgery: the lowest pHi was 7·10 and the highest IAP was 17 mmHg. The patient developed urosepsis, renal and cardiac failure, and died on day 39.

The second patient was 83 years old and in shock on arrival to hospital. Clamp time was 60 min and perioperative bleeding was 3 litres. A pHi of 6·97 and IAP of 19 mmHg were registered 6 h after surgery. The patient received volume resuscitation with colloids, and 1 hour later the values were 7·39 and 13 mmHg respectively. After the development of cardiac and respiratory failure, the patient died on day 5.

Survival after 1, 2 and 3 years was 90, 79 and 66 per cent respectively in the monitored group compared with 67, 60 and 60 per cent in the non-monitored group.

Table 2 shows the various interventions undertaken in these patients according to lowest pHi value. The 15 patients with the lowest pHi had more preoperative shock, longer cross-clamp times and greater blood loss. They also had more interventions. None of the patients in this group died after treatment.

Discussion

This study investigating the role of IAH in adverse outcome after open repair of rAAA established an association between IAH and colonic hypoperfusion. When all measuring points were included the correlation was rather weak, but this is not surprising as most of the measurements were normal. As *Fig. 1* shows, episodes of IAH were often short-lived in relation to the long period of monitoring. When the highest IAP registered for each patient was compared with the simultaneously measured colonic pHi, however, the correlation was strong ($r = -0{\cdot}39$, $P = 0{\cdot}003$).

The inverse correlation between the lowest pHi registered for each patient and the corresponding, simultaneously measured, IAP was also significant, but weaker. Many patients had low pHi measurements early, often the very first recorded values after completion of the laparotomy, as a result of preoperative shock and clamping but before the development of IAH. It appears that, although colonic hypoperfusion after rAAA repair may have various aetiologies, IAH is always a threat to the colonic circulation, and monitoring IAP is justified. This is also supported by the general guidelines in the consensus document on abdominal compartment syndrome[16].

In a recent analysis from the Swedish Vascular Registry, 30-day mortality after rAAA repair had decreased from 38·4 per cent in 1994–1999 to 32·9 per cent in 2000–2005[17]. EVAR for rAAA is a promising technique, but few centres have the organization to permit emergency EVAR, and the only controlled trial comparing EVAR with open repair showed no difference in mortality[18].

Sugrue and co-workers[19] demonstrated a strong association between abnormally low gastric pHi and increased IAP in 73 patients undergoing major abdominal surgery, but the possible relation between colonic pHi and IAP has not previously been studied.

The aetiology of colonic ischaemia after rAAA repair is multifactorial. Preoperative shock, prolonged cross-clamping and major bleeding are the strongest risk factors[3,4]; they may all create a situation of hypovolaemia. The observation in this study that many patients suffered colonic hypoperfusion during the first few hours after surgery is important when considering preventive actions. Capillary leakage due to the inflammatory response following trauma results in the need for volume resuscitation. This active approach, however, can precipitate IAH.

Optimum fluid resuscitation is controversial. Balogh and colleagues[20] compared two different trauma resuscitation strategies (500 and 600 ml per min per m^2). They concluded that supranormal resuscitation doubled the

risk of IAH, abdominal compartment syndrome, organ dysfunction and death. Specific studies on patients after surgery for rAAA have not been published, but studies including mixed non-trauma surgical patients have shown that resuscitation with isotonic crystalloids increases the risk, compared with resuscitation with hypertonic crystalloid or colloid solutions[16].

An interesting observation in this study was that the great number of interventions among the sickest patients, those with lowest colonic pHi, was associated with a low subsequent mortality.

There are other risk factors for colonic ischaemia, including improper ligation of the inferior mesenteric artery or both internal iliac arteries[3,4] and renal failure[3,4,21,22].

The study does have limitations. Colonic tonometry is cumbersome, particularly in the emergency set-up, when the patient's bowel is not prepared: some patients were excluded for this reason. Eight (15 per cent) of the 52 patients were not included because of the lack of availability of the research team. The monitored and non-monitored patients had similar preoperative risk factors. The non-monitored patients tended to be older, but preoperative shock was more common among monitored patients. The non-monitored patients had a significantly higher rate of postoperative cardiac failure and mortality. It is possible that monitoring and the interventions undertaken (colloid volume substitution, neuromuscular blockade and colonic resection) might have improved outcomes in the group.

A serious complication occurred after placement of the tonometry catheter: a perforation of a colonic diverticulum. Despite having placed tonometers in more than 100 patients[8,9] this complication had never occurred before. Thus, the risk of this complication is low, but must be recognized and avoided.

The findings of this study suggest that it may be beneficial to use colonic tonometry more often after rAAA repair, particularly when multiple risk factors for colonic ischaemia exist. Work is in progress to determine whether a tonometric balloon catheter placed in contact with the outer wall of the sigmoid colon at the end of surgery could be an alternative monitoring device. The authors' experience is that IAH is rare after elective AAA repair and so monitoring of pHi and IAP is unnecessary, unless there is major blood loss, prolonged cross-clamping, or internal iliac artery ligation.

Colonic tonometry can identify ischaemia early. It is now recognized that colonic ischaemia often follows hypovolaemia. IAH may partly be the result of active fluid resuscitation. Monitoring pHi and IAP may help to control these two mechanisms and reduce the risk of colonic ischaemia.

Acknowledgements

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Authors' reply: Randomized clinical trial of anal fistula plug *versus* endorectal advancement flap for the treatment of high cryptoglandular fistula *in ano* (*Br J Surg* 2009; 96: 608–612)

Sir

In response to the letter, I would kindly suggest the authors read carefully the paper before commenting on it. Patients with fistula *in ano* discharging a drop of pus from time to time were not excluded. Exclusion criteria included patients in whom incision and drainage and placement of a seton is recommended in the medical literature[1–2].

Regarding the follow-up period, the consensus conference unanimously agreed that the decision whether the operation should be considered a failure rests with the individual surgeon, but should not be taken for a minimum of 3 months[3]. In this study, patients have been followed up 1 year after surgery. Therefore, the 3 months period has been extensively exceeded.

In the study design, the primary endpoint was effectiveness in fistula healing. Secondary endpoints were continence disturbances, quality of life, etc. Taking into account that the comparison between the results obtained in two and 12 patients respectively would be meaningless, these results were not reported.

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Colonic ischaemia and intra-abdominal hypertension following open repair of ruptured abdominal aortic aneurysm (*Br J Surg* 2009; 96: 621–627)

Sir

Djavani and colleagues are to be commended for highlighting the importance of raised intra-abdominal pressure (IAP) and the development of abdominal compartment syndrome (ACS) as a preventable cause of postoperative morbidity in critically ill patients. However, we are concerned that the technique for determining intravesical pressure described by the authors (using a bladder infusion of 50 mL saline solution) has been superseded, as it is now recognised that the use of such a large infusion volume may lead to erroneously high IAP readings, and hence risks an overestimation of the true incidence of ACS[1]. Recent studies have shown that a bladder infusion of just 2 mL of saline may be adequate for IAP signal transduction[2], and in a modification of Kron's original technique[3] the current consensus guidelines from the World Society for the Abdominal Compartment Syndrome state that the reference standard for intermittent IAP measurement via the bladder is with a maximal instillation volume of 25 mL saline (http://www.wsacs.org/consensus_summary.php). Standardized IAP measurements should be recorded at end-expiration, with the patient in the supine position and with a pressure transducer zeroed at the iliac crest in the mid-axillary line. These readings should be taken after a brief period of equilibration to allow for detrusor muscle relaxation, and in the absence of active abdominal muscle contractions, both of which may otherwise contribute to falsely elevated bladder pressures[4,5].

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Authors' reply: Colonic ischaemia and intra-abdominal hypertension following open repair of ruptured abdominal aortic aneurysm (*Br J Surg* 2009; 96: 621–627)

Sir

We thank Mr Watson and colleagues for their interest in the correct technique of intra-abdominal pressure (IAP) measurements, and in our investigation.

When we started our study, in April 2003, the gold standard of IAP measurement was Kron's technique, measuring in the bladder with intermittent infusion of 50–100 mL. In the consensus document[1], published in 2006, one of the recommendations is 'The reference standard for intermittent IAP measurements is via the bladder with a maximum installation volume of 25 ml sterile saline'. These things happen in prospective studies, standards change over time. The relevant question raised is if the findings of our investigation, based on a technique that is no longer in use, are still valid?

We have been measuring IAP after AAA surgery consistently since 1998[2]. One of our early observations was the importance of installing pre-warmed saline slowly, and to wait 2 minutes from installation to measurement, otherwise the detrusor activity may create a falsely elevated IAP. These routines have been in practice during the investigation. De Waele *et al*. compared IAP in 20 patients with different installation volumes, from 10 to 100 mL[3]. Although there were no significant differences, there was a trend towards higher IAP with higher volumes. The mean λIAP between 10 and 50 mL installation volume was 2·2 mm Hg. We have analysed our results in the light of the new standard of IAP measurements and concluded that our conclusions would not have been altered if the mean IAP was falsely elevated by 1–2 mmHg. The aim of our investigation was to study the association between colonic ischaemia and IAP, and that association is not changed by a possible error of this magnitude.

Two new methods have been developed that do not require any filling of the bladder at all. We first tried the continuous method using a 16 Ch three-way catheter[4], but experienced haematuria in two patients. We currently use the Foley Manometer method[5], having the advantage of being feasible not only at the ICU, but also in a normal ward, which is an important advantage when treating patients with ruptured AAA with EVAR, who often can be nursed outside of the ICU.

The important message from our investigation is that colonic ischaemia after ruptured AAA repair often follows hypovolaemia. Active fluid resuscitation often results in intra-abdominal hypertension, which also results in colonic ischaemia. Controlling these two mechanisms may improve outcome.

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