Abdominal aortic aneurysm repair and colonic infarction: a risk factor appraisal

P. Neary, C. Hurson, D. O. Briain, A. Brabazon, D. Mehigan, T. V. Keaveny and S. Sheehan

Department of Vascular Surgery, St Vincent's University Hospital, Dublin, Ireland

Received 28 January 2005; accepted 3 April 2006

Abstract

Objective Colonic infarction is a recognized complication of abdominal aortic aneurysm (AAA) surgery. The clinical difficulty in establishing the diagnosis combined with the patient's poor physiological status is usually associated with a fatal outcome. We assessed our experience with this problem to identify a possible risk factor profile for these patients.

Method Patients records were identified from the operative logs, intensive care unit, Hospital Inpatient Enquiry system and vascular unit databases over a 6-year period.

Results A total of 405 patients underwent AAA repair during this period; 140 as emergency ruptures. Nine patients were identified from the databases with known colonic infarction (2.2%). One was a woman. The mean age was 70 years. Seven patients had emergency ruptures (5%). Twenty independent risk factors were analysed using univariate and multivariate logistic regression models. Significant risk factors identified by using a multivariate analysis included the nature of the presenting patient, preoperative hypotension, prolonged cross-clamp time, intra-operative ischaemia and postoperative acidosis. Confirmatory diagnosis was made by colonoscopy in eight patients. One patient survived following the salvage surgery. The mean duration of survival was 10.5 days. The overall mortality was 89% of patients.

Conclusion In our unit infrarenal AAA repair has a 2.2% rate of colonic infarction. A definitive diagnosis is best made by colonoscopy. A risk factor profile for the development of colonic infarction may be constructed on the basis of specific clinical parameters. Earlier intervention on the basis of this profile may ultimately reduce the current excessive mortality.

Keywords Colonic infarction, aortic abdominal aneurysm, risk assessment

Introduction

Ischaemic colitis is a recognized and serious complication of abdominal aortic aneurysm (AAA) repair. It was first reported by Moore, 1 year after the first successful resection of an AAA [1]. The incidence of clinically manifested patients is between 0.2% and 10% [2]. This incidence has not changed with the introduction of endovascular aortic repair (EVAR) [3]. Dadian reported a 2.9% incidence of colonic infarction in 278 patients with EVAR [4]. The true incidence may indeed be far higher as prospective studies using routine sigmoidoscopy have reported incidences of 4.5–8.8% after elective surgery and 15–60% after surgery for ruptured aortic aneurysm [5,6]. Three forms of ischaemic colitis are described: (1) mucosal ischaemia which is transient; (2) mucosal and muscularis involvement which may result in healing with fibrosis and stricture formation and (3) transmural ischaemia or infarction, which results in gangrene and perforation. In over 60% of reported patients transmural ischaemia (transmural infarction) is described. In those patients with colonic infarction the mortality approaches 90% patients [7].

An early diagnosis of colonic infarction is paramount in instituting prompt emergency intervention. This has proved difficult in the postoperative setting as patients often have significant comorbidity present. The aim of this study was to identify a specific risk factor profile, based on preoperative assessment and perioperative findings, that would preferentially characterize a cohort of patients at risk of developing postoperative colonic infarction. We furthermore characterized the incidence and mortality of this condition and advocate the most reliable diagnostic modality.

Correspondence to: Mr P. Neary MD, FRCSI (Gen), Department of Colorectal surgery, Adelaide and Meath Hospital, AMNCH, Tallaght, Dublin 24, Ireland. E-mail: paulcneary@msn.com

Method

This was a retrospective case–control study of all patients undergoing aortic aneurysm repair in our institution over a 6-year period (January 1996 to February 2002). Patients were identified from the vascular surgical database, the operative theatre register, the intensive care unit record and the Hospital Inpatient Enquiry (HIPE) system. Patient's records were reviewed for preoperative, perioperative and postoperative risk factors to identify risk factors for colonic infarction. Other variables recorded included whether or not colonoscopy was performed, the time of diagnosis to colonic infarction, the procedure performed at reoperation, and the mean survival time.

The parameters selected for assessment were based on a literature search using the PubMed internet search engines inclusive of 2004. In all, 118 abstracted reports involving documented intestinal ischaemia following AAA repair were identified (key word search parameters included: 'intestinal/colonic ischaemia, ischaemic colitis, abdominal aortic surgery, aneurysm repair, risk factors'. We eliminated case reports and reviews. This identified 35 publications that were relevant to investigating intestinal ischaemia [2,3,5,7-38]. In these studies, a total of 22 075 patients had been analysed for potential indicators of ischaemic colitis following aortic aneursym surgery. There were only 12 reports with series numbers equal or greater than this study. The 20 most frequently assessed indices that were considered significant from these 22 075 patients were incorporated into our study for univariate and multivariate analyses. As eight of the series account for over 80% of the patients we accepted that this may have skewn the population indices studied.

A total of 405 patients underwent AAA repair during this period. Nine patients were identified with known colonic infarction. A cohort study was then performed on the study population. The data for the cohort study consisted of 107 patients, representing the nine patients and 98 controls selected at random from the remaining patient records. The cohort study data were split as shown in Table 1. Based on a literature review, we used 20 preselected parameters in assessing our population (Table 2). Two emergency cases were dropped from the study as the patients did not survive their AAA repair operation, leaving 105 patients in the study.

Table I Table of patients in the case-control study.

	Elective	Emergency	Total
Colonic infarction absent	57	41	98
Colonic infarction present	2	7	9
Total	59	48	107

Preoperative	Intra-operative	Postoperative
Age	Hypovolaemia	Renal failure
Cardiovascular disease	Systolic blood pressure	Temperature
Emergency/ elective case	Blood loss (litres)	ICU neutrophilia
Hypertension	Transfusion (units)	Acidosis
Renal disease	Clamp time (mins)	Ionotropic requirement
Smoker	Operative time (mins)	Bloody diarrhoea
Diabetes mellitus	Bifurcated graft	
	Ischaemia noted at time of surgery	

Table 2 List of variables utilized for the case-control study.

All variables, except those in bold, are binary indicating the presence or absence of a potential risk factor. Variables in bold represent interval data.

Statistics

Both univariate and multivariate analyses of the potential risk factors were performed. For binary variables, odds ratios were calculated which refer to the risk of a patient developing colonic infarction after AAA surgery, contrasted between patients in whom that risk factor was present or absent. In patients in whom the odds ratio could not be meaningfully calculated, the Pearson chi-squared test was applied to determine whether the risk factor and colonic infarction outcome were independent [39]. For interval variables, the significance of a difference in the means was tested using Student's t-test. Depending on the outcome of Levene's test for the equality of variances, an equal or unequal variance t-test was performed. The mean values were reported for each comparison and a significance level of 5% (two-tailed) was adopted. A nonparametric Mann-Whitney test was also performed to lend additional assurance to the reported results. In the multivariate analysis, the logistic regression model was utilized as the dependent variable was binary (absence, presence of CI). The logistic model was constructed using a forward, stepwise method, using the likelihood ratio for variable selection. The analysis was performed using the SPSS package.

Results

A total of 405 patients underwent AAA repair during this period and nine patients were identified with known colonic infarction. The overall incidence of colonic infarction within our operative population was 2.2% of cases. Eight of the nine patients were men. The mean age

Presenting case	No. at risk	No. with infarction	Percentage
Emergency surgery	140	7	5.00
Elective surgery	265	2	0.75
Total	405	9	2.22

Table 3 Nature of presenting case.

of colonic infarction patients was 70.57 (59-82 years). One hundred and forty patients had emergency ruptures (34.56%). There were seven patients with colonic infarction in this group (5%). In patients operated on an elective basis the incidence of colonic infarction was 0.8% (2/265). In total, the mortality associated with colonic infarction was 89% (8/9) with one patient surviving salvage surgery. The diagnosis of infarction was made on day 5 (mean) following initial surgery. The mean duration of survival for these patients was 10.5 days. Of the nine patients, the diagnosis was confirmed with colonoscopy in eight of the patients and through relaparotomy for progressive sepsis in one patient. The definitive surgery performed on relaparotomy consisted of a formal resection of the left colon and formation of an end colostomy (Hartman's procedure) in all patients. A breakdown of this patient group between emergency and elective cases is presented in Table 3.

An initial statistical analysis was performed to determine the strength of the association between the nature of the presenting patient and the occurrence of colonic infarction using all 405 patients. As the event of interest (occurrence of colonic infarction) is of low probability and because of the case–control nature of the study, the risk estimate is reported as an odds ratio. The odds that an emergency surgery case would result in colonic infarction was 5%/95% = 0.05263. The odds that an elective case would result in colonic infarction was 0.7547%/99.245% = 0.007604. The odds ratio was 0.0526/0.0075 = 6.92, with a 95% confidence interval of (1.418–33.778). This confirmed that patients undergoing emergency repair have an increased risk of developing colonic infarction.

All of the interval data with the exception of age were collected intra-operatively. Analysis of the interval data of patients and controls is reported in Table 4. In a number of patients, data were not available, and hence the number of data observations varies between the variables. Full data were available for all nine patients with colonic infarction.

Taking all of the patients in the cohort together, three potential risk factors were identified [mean preoperative systolic blood pressure (80.67 *vs* 126.53 P < 0.001), blood loss (6.489 l *vs* 3.793 l P < 0.029) and clamp time (186.67 min *vs* 91.08 min P < 0.001)]. Examining emergency cases only, the risk factors identified also included preoperative systolic blood pressure and clamp time (P < 0.003, P < 0.002). Elective cases did not exhibit any strong statistical difference between the two groups and this, however, may be reflective of the small

	Infarction present	n (all)	Mean (all)	<i>n</i> (emergency)	Mean (emergency)	n (elective)	Mean (elective)
Age (years)	0	96	72.68	39	72.54	57	72.77
	1	9	70.44	7	70.57	2	70.0
Blood pressure	0	94	126.53	37	117.73	57	132.25
preoperatively (mmHg)	1	9	80.67*† (<i>P</i> < 0.001; <i>P</i> < 0.001)	7	$69.43*\dagger (P < 0.001;$ P = 0.003)	2	120
Blood loss (l)	0	88	3.793	37	5.647	51	2.448
	1	9	6.489*† ($P = 0.018$; P = 0.029)	7	7.271	2	3.75
Transfusion (l)	0	85	6.67	35	12.14	50	2.84
	1	9	11.0† ($P = 0.024$)	7	13.0	2	4.0
Clamp time (min)	0	80	91.08	32	96.09	48	87.73
	1	9	186.67*† ($P = 0.010$; P < 0.001)	7	171.43*† ($P = 0.003$; P = 0.002)	2	240† ($P = 0.033$)
Surgical time (min)	0	87	260.23	37	267.97	50	254.5
	1	9	266.67	7	248.57	2	330.0

Table 4 Comparison of mean values for interval data.

Where two *P*-values are reported, the first value refers to the *t*-test result, the second to the Mann–Whitney test.

*Difference between mean values is significant using *t*-test.

†Difference between means is significant using Mann-Whitney test.

incidence of colonic infarction recorded in these patients. It was noted that clamp time was also significantly longer in this group (240 min *vs* 87.73 min P < 0.03).

A comparison of the odds ratios for the risk factors collected pre-, intra- and postoperatively was performed. The data set was binary and the odds ratio or Pearson chisquared test was used for the univariate analysis. For the preoperative data set, the increased risk factor for undertaking the procedure as an emergency case (n =7/9) was again identified (odds ratio 5.115, 95% CI 1.009–25.936, P < 0.049). Other preoperative parameters assessed included the presence of diabetes mellitus, hypertension, preoperative renal impairment, coronary heart disease and whether the patient was a smoker. There is no statistical evidence that any of these preoperative risk factors were associated with the occurrence of colonic infarction. This finding held true for the two subgroups, elective and emergency cases. Multivariate analyses were performed on the six preoperative potential risk factors. The results from the multivariate analysis concurred with those of the univariate analysis in that the only risk factor which was of significance was the nature of the presenting patient [OR 5.25 (1.035-26.638)].

The comparison of the odds ratios for the binary risk factors collected intra-operatively identified the presence of intra-operative hypovolaemia (odds ratio 19.5, 95% CI 3.666–103.719, P < 0.001) and a subjective peroperative ischaemic appearance of the bowel as risk factors. This finding was consistent for the cohort taken together (emergency and elective) and examining the two subgroups of patients, the presence of ischaemia and hypovolaemia significantly correlated with colonic infarction in the emergency group (7/7 patients in each patient)exhibiting the risk factor). Only ischaemia was included in the multivariate analysis model of the eight intraoperative risk factors. This was not statistically significant. A similar result emerged when the preoperative and intraoperative risk factors were selected for potential entry into the model.

A univariate analysis of the postoperative parameters was performed taking both patient groups together. Patients who exhibited any of the postoperative risk factors, with the exception of temperature, all had a statistically significant increased risk of developing colonic infarction (renal failure P = 0.019, neutrophilia P =0.036), acidosis P < 0.001, ionotropic support P =0.003, bloody diarrhoea P = 0.014). In the subgroup assessment, however, the only significant result was the presence of acidosis in patients having undergone emergency surgery. In the multivariate analysis, only acidosis was therefore included in the model. This produced a significant OR of 23.142 (4.736–113.084). Of note, we acknowledge that little can be said 'statistically' about the two subgroups of patients because of the small numbers involved.

Finally, we performed a multivariate analysis using all the significant risk factors (pre/intra/postoperative) identified in the univariate analysis. The initial factors selected for inclusion in the model were blood loss and the presence of intra-operative ischaemia. Neither exhibited a statistically significant coefficient. With the exclusion of the subjective intra-operative appearance of ischaemia, however, both the presence of acidosis $[OR = 25.594 \quad (3.498-187.275)]$ and a prolonged clamp time (P = 0.003) were significant overall risk factors in the multivariate analysis model.

Discussion

The most severe form of intestinal ischaemia is transmural necrosis or colonic infarction. This is a recognized and often fatal complication of AAA repair. The mortality associated with a second laparotomy is very significant in these patients with high co-morbidities [40]. We do not employ a policy of a routine second look laparotomy in all our patients and with this in mind we have endeavoured to identify possible risk factors in those patients undergoing AAA repair specific for developing colonic infarction. The design was that of a retrospective case–control study with univariate and multivariate analyses.

As in other reports, patients who underwent an emergency surgery had a significantly increased odds ratio of developing colonic infarction over patients who underwent elective surgery [41]. In our unit, 34.5% of patients underwent an emergency surgery and the incidence of colonic infarction within this subgroup was 5%. Other preoperative parameters assessed included a history of smoking, renal impairment, hypertension, diabetes mellitus, advanced age and cardiovascular disease (a history of myocardial infarction, angina or a coronary arterial bypass graft). Renal disease, hypertension and advanced age have previously been reported as independent risk factors for the development of ischaemic colitis [2]. These findings have not, however, been a universal finding in the literature [32]. Preoperative renal insufficiency occurred in 15.2% of our study population, 45% had a history of hypertension and there was no significant difference in their age profile. We were unable to demonstrate any significantly increased risk for patients with any of these medical comorbidities. Based on our data, we do not believe that these parameters represent significant 'players' in identifying an accurate preoperative profile for the development of colonic infarction.

The intra-operative factors identified as being significant included preoperative hypotension, perioperative

blood loss, ischaemic appearance, hypovolemia and crossclamp time. A low mean preoperative systolic blood pressure of 80 mmHg was highly significant (P < 0.001). Preoperative hypotension has been identified previously as a risk factor using a multivariate analysis and neural network matrices [37,42]. The possibility of a reduction in the marginal arterial supply or indeed a low-flow state to the left colon would inherently seem to increase the risk of *in situ* thrombosis and possible infarction. This is confirmed by our analysis. In our series, the mean blood loss incurred in emergency cases differed significantly from elective cases. This difference, however, was not significant in the subgroup analysis of the emergency cases alone. Multivariate analyses from other authors have identified a blood loss more than 10 l as an independent risk factor. These authors suggest that tenting of the enteric blood supply from a large retroperitoneal haematoma may account for the increased incidence of ischaemia recorded [43]. The small number of patients with a blood loss more than 101 in our series was insufficient to draw any such meaningful conclusions.

A prolonged aortic cross-clamp time was identified as an independent risk factor. This difference was maintained in the subgroup analysis of emergency cases. The data set to differentiate suprarenal vs infrarenal clamping was unavailable; however, suprarenal clamping has not been identified as an independent risk factor by other groups [2]. Hypovolaemia was also identified as being an independent significant risk factor for developing colonic infarction (odds ratio 19.5). Similarly, intra-operative ischaemic-appearing bowel was significantly higher in those who went on to develop colonic infarction. This was true of all nine patients and should be considered a definitive marker for high-risk patients. The intra-operative evaluation of the marginal artery supply to the descending colon has been made by several authors [44]. They advocate the routine use of laser Doppler flowmetry. As the intra-operative appearance of colonic ischaemia has been confirmed to be a significant risk factor for the subsequent development of infarction, using such modalities as laser Doppler flowmetry in selected or indeed routine patients has some evidence-based rationale. The intra-operative use of laser Doppler in selected patients should therefore be a part of a vascular surgeon's technical armamentarium. The type of graft inserted (bifurcated vs tube graft), operative time and transfusion requirement were not independent risk factors in our study.

The postoperative parameters of progressive renal failure, unexplained neutrophilia, persistent ionotroph requirement, the presence of bloody diarrhoea and resistant metabolic acidosis all were independent risk factors on a univariate analysis when the population was considered as a whole. Persistent acidosis alone was identified on a multivariate analysis as representing an increased risk. This was also valid for a subgroup analysis and should therefore be considered indicative of high risk. The possibility of underlying colonic infarction should be considered in those patients in whom the other independent risk factors are present. This may lead to a more prompt intervention being instituted.

We would recommend a high index of suspicion and a low threshold for endoscopy in suspected patients. Colonoscopy confirmed the diagnosis in all the patients in whom it was used, emphasizing its reliability as a diagnostic tool for this condition. The use of flexible sigmoidoscopy has been advocated to follow 'at-risk' patients after emergency AAA repair [45]. Some difficulty may arise in patients over dubious levels of ischaemia and in patients with more proximally located infarction. In our experience, the appearance of colonic infarction is unmistakable. The appearance of more subtle degrees of ischaemia may, however, be more challenging. In patients with incomplete transmural ischaemia and in the presence of the risk factors, we have identified that an early and aggressive reoperative approach should be considered. This is because of the concomitant mortality of delayed intervention being so catastrophic for the patient.

Attempts in the literature to identify risk factors for the subsequent development of colonic infarction are limited. The incidence of colonic infarction in our experience following AAA repair was 2.2%. Patients with ruptured AAAs repaired as an *emergency*, having evidence of *preoperative hypotension* and a *prolonged aortic crossclamping time* are at increased risk of developing colonic infarction. These patients should be closely monitored for evidence of *resistant postoperative metabolic acidosis*. A left-sided colonoscopy should be performed as a matter of urgency in these patients with acidosis. The early diagnosis of colonic infarction using these three parameters alone may result in an improved outcome.

References

- Moore SW. Resection of the abdominal aorta with defect replaced with homologous graft. *Surg Gynecol Obstet* 1954; 99: 745–55.
- 2 Bjorck M, Bergqvist D, Troeng T. Incidence and clinical presentation of bowel ischaemia after aortoiliac surgery– 2930 operations from a population-based registry in Sweden. *Eur J Vasc Endovasc Surg* 1996; 12: 139–44.
- 3 Van Damme H, Creemers E, Limet R. Ischaemic colitis following aortoiliac surgery. *Acta Chir Belg* 2000; 100: 21–7.
- 4 Dadian N, Ohki T, Veith FJ, Edelman M, Mehta M, Lipsitz EC, Suggs WD, Wain RA. Overt colon ischemia after endovascular aneurysm repair: the importance of microembolization as an etiology. *J Vasc Surg* 2001; 34: 986–96.

- 5 Bast TJ, Van Der Biezen JJ, Scherpenisse J, Eikelboom BC. Ischaemic disease of the colon and rectum after surgery for abdominal aortic aneurysm. A prospective study of the incidence and risk factors. *Eur J Vasc Surg* 1990; 4: 253–7.
- 6 Hagihara PF, Ernst CB, Griffen WO. Incidence of ischaemic colitis following abdominal aortic reconstruction. *Surg Gynecol Obstet* 1979; **149:** 571–3.
- 7 Longo WE, Lee TC, Barnett MG, Vernava AM, Wade TP, Peterson GJ, Jacobs DL, Virgo KS, Johnson FE. Ischemic colitis complicating abdominal aortic aneurysm surgery in the US veteran. J Surg Res 1996; 60: 351–4.
- 8 Vohra R, Abdool-Carrim AT, Groome J, Pollock JG. Ruptured aortic aneurysms: postoperative complications and their management. *Ann Vasc Surg* 1988; **2**: 114–9.
- 9 Noirhomme P, Buche M, Louagie Y, Verhelst R, Matta A, Schoevaerdts JC. Ischemic complications of abdominal aortic surgery. J Cardiovasc Surg (Torino) 1991; 32: 451–5.
- 10 Kogel H, Vollmar JF, Zelesny T, Witter B. Prevention of postoperative ischemic colitis in aorto-iliac vascular reconstruction. *Chirurgia* 1992; 63: 44–9.
- 11 Giordanengo F, Boneschi M, Vandone PG, Erba M. Intestinal infarction after ruptured aneurysm of the abdominal aorta. Clinical contribution. *Minerva Cardioangiol* 1995; **43**: 429–34.
- 12 Guivarc'h M, Roullet-Audy JC, Mosnier H, Boche O. Ischemic colitis. A surgical series of 88 cases. J Chir (Paris) 1997; 134: 103–8.
- 13 Hsiang YN, Turnbull RG, Nicholls SC, McCullough K, Chen JC, Lokanathan R, Taylor DC. Predicting death from ruptured abdominal aortic aneurysms. *Am J Surg* 2001; 181: 30–5.
- 14 Alvarez-Tostado-Fernandez F, Martinez-Hernandez-Magro P, Villanueva-Saenz E. Ischemic colitis in patients submitted to aortic replacement surgery. Risk factors. *Rev Gastroenterol Mex* 2002; 67: 171–8.
- 15 Durrani NK, Trisal V, Mittal V, Hans SS. Gastrointestinal complications after ruptured aortic aneurysm repair. Am Surg 2003; 69: 330–3.
- 16 Kalman PG, Johnston KW, Lipton IH. Prevention of severe intestinal ischemia following reconstruction of the abdominal aorta. *Can J Surg* 1981; 24: 634–7.
- 17 Jonung T, Ribbe E, Norgren L, Thorvinger B, Thorne J. Visceral ischemia following aortic surgery. *Vasa* 1991; 20: 125–31.
- 18 Urayama H, Ueyama K, Takahashi A, Kato A, Tokuraku M, Tsuchida K, Watanabe Y. Effect of surgery for combined abdominal aortic and internal iliac artery aneurysm on postoperative intestinal ischemia and sexual dysfunction. *Nippon Geka Gakkai Zasshi* 1991; **92**: 1719–22.
- 19 Redaelli CA, Carrel T, von Segesser LK, Turina M. Intestinal ischemia following replacement of the infrarenal aorta and aorto-iliac bifurcation. *Helv Chir Acta* 1992; 58: 589–94.
- 20 Porcellini M, Renda A, Selvetella L, Bernardo B, Baldassarre M. Intestinal ischemia after aortic surgery. *Int Surg* 1996; 81: 195–9.
- 21 Jarvinen O, Laurikka J, Sisto T, Tarkka MR. Intestinal ischemia following surgery for aorto-iliac disease. A review of

502 consecutive aortic reconstructions. Vasa 1996; 25: 148–55.

- 22 Bjorck M, Troeng T, Bergqvist D. Risk factors for intestinal ischaemia after aortoiliac surgery: a combined cohort and case-control study of 2824 operations. *Eur J Vasc Endovasc Surg* 1997; 13: 531–9.
- 23 Pargger H, Hampl KF, Christen P, Staender S, Scheidegger D. Perioperative gastrointestinal ischemia in abdominal aortic aneurysm operations. *Schweiz Med Wochenschr* 1997; 127: 1511–8.
- 24 Kim MW, Hundahl SA, Dang CR, McNamara JJ, Strachley CJ, Whelan TJ Jr. Ischemic colitis after aortic aneurysmectomy. Am J Surg 1983; 145: 392–4.
- 25 Ernst CB, Hagihara PF, Daughtery ME, Sachatello CR, Griffen WO. Ischemic colitis incidence following abdominal aortic reconstruction: a prospective study. *Surgery* 1976; 80: 417–21.
- 26 van Vroonhoven TJ, Verhagen HJ, Broker WF, Janssen IM. Transmural ischaemic colitis following operation for ruptured abdominal aortic aneurysm. *Neth J Surg* 1991; **43**: 56– 9.
- 27 Maupin GE, Rimar SD, Villalba M. Ischemic colitis following abdominal aortic reconstruction for ruptured aneurysm. A 10-year experience. *Am Surg* 1989; 55: 378–80.
- 28 Meissner MH, Johansen KH. Colon infarction after ruptured abdominal aortic aneurysm. Arch Surg 1992; 127: 979–85.
- 29 Noirhomme P, Buche M, Louagie Y, Verhelst R, Matta A, Schoevaerdts JC. Ischemic complications of abdominal aortic surgery. J Cardiovasc Surg 1991; 32: 451–5.
- 30 Dahan P, Roseau G, Duchatelle JP, Andreassian B, Paolaggi JA. Intestinal ischemia after surgery of the infrarenal aorta. Apropos of 13 cases. *Ann Chir* 1991; 45: 402–7.
- 31 Fiddian-Green RG, Amelin PM, Herrmann JB, Arous E, Cutler BS, Schiedler M, Wheeler HB, Baker S. Prediction of the development of sigmoid ischemia on the day of aortic operations. Indirect measurements of intramural pH in the colon. *Arch Surg* 1986; **121**: 654–60.
- 32 Brewster DC, Franklin DP, Cambria RP, Darling RC, Moncure AC, Lamuraglia GM, Stone WM, Abbott WM. Intestinal ischemia complicating abdominal aortic surgery. *Surgery* 1991; 109: 447–54.
- 33 Welch M, Baguneid MS, McMahon RF, Dodd PD, Fulford PE, Griffiths GD, Walker MG. Histological study of colonic ischaemia after aortic surgery. *Br J Surg* 1998; 85: 1095–8.
- 34 Reddy E, Robbs JV, Rubin J. Abdominal aortic aneurysm resection – operative risk and long-term results. S Afr Med J 1985; 67: 921–3.
- 35 Jarvinen O, Laurikka J, Salenius JP, Lepantalo M. Mesenteric infarction after aortoiliac surgery on the basis of 1752 operations from the National Vascular Registry. *World J Surg* 1999; 23: 243–7.
- 36 Piotrowski JJ, Ripepi AJ, Yuhas JP, Alexander JJ, Brandt CP. Colonic ischemia: the Achilles heel of ruptured aortic aneurysm repair. *Am Surg* 1996; 62: 557–60.
- 37 Levison JA, Halpern VJ, Kline RG, Faust GR, Cohen JR. Perioperative predictors of colonic ischemia after ruptured abdominal aortic aneurysm. J Vasc Surg 1999; 29: 40–5.

- 38 Schiedler MG, Cutler BS, Fiddian-Green RG. Sigmoid intramural pH for prediction of ischemic colitis during aortic surgery. A comparison with risk factors and inferior mesenteric artery stump pressures. *Arch Surg* 1987; 122: 881–6.
- 39 Ott L, Longnecker M (6th edition, 2006). An Introduction to Statistical Methods and Data Analysis. Duxbury Press, Belmont, CA.
- 40 Eliason JL, Wainess RM, Dimick JB et al. The effect of secondary operations on mortality following abdominal aortic aneurysm repair in the United States: 1988–2001. Vasc Endovasc Surg 2005; 39: 465–72.
- 41 Bjorck M, Troeng T, Bergqvist D. Risk factors for intestinal ischaemia after aortic-iliac surgery: a combined cohort and case control study of 2824 operations. *Eur J Vasc Endovasc Surg* 1997; **13**: 531–9.
- 42 Turton EP, Scott DJ, Delbridge M, Snowden S, Kester RC. Ruptured abdominal aortic aneurysm: a novel method of

outcome prediction using neural network technology. Eur J Vasc Endovasc Surg 2000; 19: 184–9.

- 43 Farooq MM, Freischlag JA, Seabrook GR, Moon MR, Aprahamian C, Towne JB. Effect of the duration of symptoms, transport time, and length of emergency room stay on morbidity and mortality in patients with ruptured abdominal aortic aneuysms. *Surgery* 1996; 119: 9–14.
- 44 Redaelli CA, Schilling MK, Carrel TP. Intraoperative assessment of intestinal viability by laser Doppler flowmetry for surgery of ruptured abdominal aortic aneurysms. *World J Surg* 1998; 22: 283–9.
- 45 Brandt CP, Piotrowski JJ, Alexander JJ. Flexible sigmoidoscopy. A reliable determinant of colonic ischemia following ruptured abdominal aortic aneurysm. *Surg Endosc* 1997; 11: 113–5.