Colonic ischemia complicating open vs endovascular abdominal aortic aneurysm repair

Robert Jason T. Perry, MD, Matthew J. Martin, MD, Matthew J. Eckert, MD, Vance Y. Sohn, MD, and **Scott R. Steele, MD,** *Tacoma, Wash*

Objective: **Colonic ischemia (CI) is a known complication of both open abdominal aortic aneurysm (AAA) repair and endovascular aneurysm repair (EVAR). Despite a relatively low incidence of 1% to 6%, the associated morbidity and mortality are high. We sought to analyze factors that affect the development of CI on the basis of type of repair as well as associated outcomes from a large nationwide database.**

Methods: **All admissions undergoing AAA repair were selected from the 2003 and 2004 Nationwide Inpatient Sample. Univariate and logistic regression analyses were used to compare outcome measures and identify independent predictors of development of colonic ischemic complications.**

Results: **We identified 89,967 admissions for AAA repair (mean age, 69.9 years). Open elective repair was performed in 49% of cases, elective EVAR in 41%, and ruptured aneurysm repair in 9%. The overall incidence of CI was 2.2% (1941 cases); however, the incidence for specific procedures was significantly higher after repair of ruptured aneurysm (8.9%) and open elective repair (1.9%) than after EVAR (0.5%; both $P < .001$). Patients who developed CI were at increased risk for mortality (37.8% vs 6.7%), had longer hospital stays (21.5 vs 8.1 days), incurred higher hospital charges ($182,000 vs $77,000), and were less likely to be discharged home from hospital (36% vs 71%; all $P < .001$). Independent predictors of development of CI included ruptured aneurysm (odds ratio [OR] = 6.4), female gender (OR = 1.6) and, in the setting of elective repair, open operation (OR = 3.1). CI was found to be a strong independent predictor of mortality in evaluations of both the entire cohort (OR = 4.5) and the elective open repair and EVAR (OR = 2.4) subgroups.**

Conclusions: **CI is significantly more common after open AAA repair and is associated with increased morbidity and a two- to fourfold increase in mortality. (J Vasc Surg 2008;48:272-7.)**

Endovascular aortic aneurysm repair (EVAR) was first demonstrated as a feasible option for the repair of abdominal aortic aneurysm (AAA) by Volodos[1] in 1986 and Parodi in 1991.[2] Since that time multiple studies have documented the benefit of EVAR in reducing postoperative complications, hospital length of stay, and mortality.[3,4] One of the well-described complications of both open AAA repair and EVAR is colonic ischemia (CI), in which low blood flow can cause changes ranging from mild mucosal ulcerations to full-thickness necrosis of the bowel wall.[5] Although the incidence of CI after major vascular surgery is relatively low (approximately 1%-6%), development in this setting is associated with high mortality rates up to 90%, depending on both patient factors (advanced age, underlying medical illness, ruptured AAA [rAAA]) and the physiologic insult (fluid shifts, prolonged operative and cross-clamp time, sacrifice of inferior mesenteric or hypogastric artery collaterals, blood loss, sepsis) associated with repair.[5-9]

Relatively small reports of CI after EVAR have documented an incidence of approximately 1% to 6%.[10-15] Unlike open repair in which the onset is thought to occur from a global physiologic insult, the pathogenesis of CI after EVAR is thought to be due to dislodged debris from the aneurysm sac or from iliofemoral access.[16-19] Because of the relative rarity of this complication and the low numbers of cases for analysis in the literature, no well-validated epidemiologic or etiologic factors have been described to aid in identifying patients at increased risk for this devastating complication. Among potentially important variables predictive of the development of CI after aneurysm repair is the method of repair; it remains unclear whether the incidence of CI associated with EVAR differs significantly from that of open repair. Therefore the purpose of this study was to determine the incidence of CI after open AAA repair and EVAR using a large nationwide database, to identify risk factors predictive of CI, and to define its attendant mortality risk.

METHODS

Data were collected from the 2003 and 2004 Nationwide Inpatient Sample (NIS) databases, a product of the Health Care Cost & Utilization Project, Agency for Healthcare Research and Quality. NIS is a nationwide hospital discharge database consisting of a 20% random sample of all discharged patients in the United States, which reflects 8 million annual hospital stays.[20] The database includes both admission and discharge diagnoses as well as procedures performed during hospitalization. Complications associated with a particular operation or procedure can thus be analyzed. The NIS also contains patient demographic and medical comorbidity information, allowing multivariate analyses of outcomes based on these factors. Additionally, hospital charges and lengths of stay as well as discharge status can be obtained.

From the Department of General Surgery, Madigan Army Medical Center.
Competition of interest: none.
Presented at the Twenty-second Annual Meeting of the Western Vascular Society, Kona, Hawaii, Sept 9, 2007.
Reprint requests: Scott R. Steele, MD, FACS, Madigan Army Medical Center, Department of Surgery, Fort Lewis, WA 98431 (e-mail: docsteele@hotmail.com).
CME article
0741-5214/$34.00

doi:10.1016/j.jvs.2008.03.040

Table I. Demographics and presentation: open AAA repair, EVAR, and ruptured AAA repair

	All (n = 89,867)	*Open (n = 44,184; 49%)*	*EVAR (n = 37,172; 41%)*	*Rupture (n = 8511; 9%)*
Age (y) (mean ± SD)	69.9 ± 14.9	66.4 ± 18.6	73.5 ± 8.6	72.6 ± 9.2
Disease severity (mean ± SD)	409 ± 703	331 ± 539	153 ± 308	1944 ± 808
Caucasian (n [%])	58915 (87.6)	28191 (85.4)	24568 (90.5)	5364 (86.6)
Female (n [%])	20365 (22.7)	11736 (26.6)	6636 (17.9)	1994 (23.4)
Elective admission (n [%])	65384 (72.8)	32499 (73.7)	32160 (86.6)	725 (8.5)

AAA, Abdominal aortic aneurysm; *EVAR*, endovascular aneurysm repair.
All $P < .01$.

Patients were initially identified according to the *International Classification of Disease, Ninth Revision, Clinical Modification* (*ICD-9-CM*) procedure codes for open (38.44) or endovascular (39.71) AAA repair. Patients were further classified according to *ICD-9-CM* diagnostic codes for non-ruptured AAA (441.4) and rAAA (441.3). The *ICD-9-CM* diagnostic code for acute vascular insufficiency of the intestine (557.0) was used to identify our primary study variable of CI. Patients with chronic CI (indicated by an admission diagnosis of CI) were excluded from analysis. Patient data for age, gender, race, comorbid illness, and disease severity score were extracted from the NIS. The disease severity score is obtained by using a proprietary Disease Staging software package (Medstat Disease Staging Software, Ann Arbor, Mich) that includes baseline variables and all available diagnostic codes among 15 possible diagnostic codes per admission. It is the best validated variable contained in the data set for comparing the overall illness or disease "severity" of patient groups. Much like the Injury Severity Score commonly used in trauma, the disease severity score is extremely useful for adjusting between groups for injury severity. We performed an internal validation analysis of this variable for our specific data set and found the score to be strongly predictive of mortality for the entire data set and the open and EVAR subgroups.

To assess outcomes, we also obtained in-hospital mortality, hospital charges, length of stay, and discharge status (home vs other). To better approximate a normal distribution for both univariate and multivariate analysis, we performed log transformations for the disease severity score, hospital charges, and length of stay, all of which demonstrated significant skew.

Definition of variables. The primary variable in this study was the method of repair, defined by open versus endovascular repair as classified by their *ICD-9-CM* codes, along with the development of CI. Other variables included rAAA versus non-ruptured AAA, elective versus emergent presentation, age (years), gender, race, and comorbidity. The NIS database categorizes race as Caucasian, African American, Hispanic, Asian, Native American, and other. Race was dichotomized as either Caucasian or non-Caucasian for the purpose of this analysis. Age was analyzed as a continuous variable in univariate and multivariate analysis, and was then dichotomized at age greater than 78 years (the 75th percentile for the study population) for the final multivariate model.

Main outcome measures. Total hospital charges were used with the NIS variable TOTCHG (total charges cleaned). In general, these charges do not include professional fees and noncovered charges but do include emergency department charges incurred before admission to the hospital. The length of the hospital stay was measured in days from the time of admission to the time of discharge. The NIS database provides the following information about the patient's discharge status: routine discharge, short-term hospital stay, skilled nursing facility, intermediate care facility, discharge to another type of facility, home health care, left against medical advice, and died during hospitalization. Patients who died during the hospitalization (n = 6572) were evaluated in the mortality analysis but were excluded in evaluation of this specific end point only. Patients requiring disposition to another facility were also categorized together and evaluated separately (NIS variables DISPUniform 2, 3, 4, and 5).

Statistical analysis. All statistical analyses were performed with commercially available software (SPSS for Windows version 14.0; SPSS Inc, Chicago, Ill). Because the NIS database is an approximately 20% sample of the United States yearly inpatient admissions, we performed all analyses using weighted variables (NIS variable DISCWT) to produce national estimates. Patients with invalid or missing data for the primary variables of interest were analyzed for any significant variance from the population and excluded for evaluation of that data element. We compared continuous variables using the Student *t* test, Mann-Whitney *U* test, analysis of variance (ANOVA), and Kruskal-Wallis test, as appropriate. Categorical variables were analyzed by χ^2 or Fisher exact test, as appropriate. All tests were two-tailed with $P < .05$ considered significant. Selected variables identified as significant by univariate analysis were entered into a block multivariate logistic regression model to determine independent predictors of development of CI as well as mortality. Adjusted ORs are reported with 95% confidence intervals.

RESULTS

The cohort consisted of 89,867 admissions, of whom 49% underwent open repair of non-rAAA, 41% underwent non-ruptured EVAR, and 9% underwent repair of rAAA. The rAAA group included patients treated with both open repair and EVAR. Patient demographics, disease severity scores, and admission information are presented in Table I. In comparison with non-ruptured open repair, EVAR pa-

Table II. Demographics and presentation: colonic ischemia vs no colonic ischemia

	Colonic ischemia (n = 1941; 2.2%)	*No colonic ischemia (n = 87,926; 97.8%)*	P
Age (y) (mean ± SD)	72.4 ± 10.0	69.9 ± 14.9	<.001
Disease severity (mean ± SD)	1311 ± 1095	389 ± 678	<.001
Caucasian (n [%])	1297 (87.0)	57618 (87.6)	NS
Female (n [%])	623 (32.1)	19742 (22.5)	<.001
Elective admission (n [%])	883 (45.6)	64500 (73.5)	<.001
Ruptured AAA (n [%])	756 (8.9)	1185 (1.5)	<.001

NS, Not significant; *AAA,* abdominal aortic aneurysm.

Table III. Colonic ischemia hospital and discharge outcomes

	Colonic ischemia	*No colonic ischemia*	P
Mortality (n [%])	731 (37.8)	5841 (6.7)	<.001
LOS (d) (mean ± SD)	21.5 ± 23.1	8.1 ± 11.0	<.001
Hospital charges (US$) (mean ± SD)	181868 ± 167919	76597 ± 79659	<.001
Discharge home (n [%])	430 (36.0)	57885 (70.8)	<.001

LOS, Hospital length of stay.

Table IV. Independent predictors of colonic ischemia

Patient population	*Risk factor*	*Adjusted OR*	*95% CI*	P
All patients	Ruptured AAA	6.4	5.8-7.0	<.001
	Log DSS	2.6	2.3-3.0	<.001
	Female gender	1.6	1.5-1.8	<.001
Non-ruptured patients	Open	3.1	2.7-3.7	<.001
	Log DSS	2.6	3.1-3.3	<.001

OR, Odds ratio; *CI,* confidence interval; *AAA,* abdominal aortic aneurysm; *DSS,* disease severity score; *open,* elective open AAA repair; *EVAR,* elective endovascular AAA repair.
The ORs provided are the results of multivariate logistic regression analysis.

tients were older, had lower disease severity scores, were more likely to be Caucasian and to be admitted electively, and were less likely to be female. Patients undergoing repair of rAAA had much higher disease severity scores than patients undergoing elective repair, open or EVAR. Mortality rates were higher among patients undergoing repair of ruptured aneurysms (38.5%, 3262 patients) and elective open repair (6.5%, 2865 patients) than among patients undergoing elective EVAR (1.2%, 445; both $P < .001$).

Overall, 1941 (2.2%) patients developed CI (Table II). Patients who developed CI were older, had higher disease severity scores, were more likely to be female and to have undergone operation for a rAAA, and were less likely to present electively. Although the overall incidence was 2.2%, CI was more commonly associated with repair of ruptured aneurysms (8.9%, 756 patients) and elective open AAA repair (2.2%, 983 patients) than with EVAR (0.5%, 202 patients; $P < .001$). Patients who developed CI were at increased risk of mortality, incurred longer hospital stays and hospital charges, and were less likely to be discharged home than those who did not develop CI (Table III).

Of 1941 patients who developed CI, 692 (35.7%) underwent colectomy and, of those, 370 (53.5%) died. Patients who developed CI after repair of rAAA were more likely to undergo colectomy than patients who had elective repairs, open or EVAR (41% vs 31% and 27%, respectively; $P < .01$). Patients who underwent colectomy after EVAR had higher mortality than patients who underwent colectomy in the setting of either open elective or rAAA repair (73% vs 51% and 48%, respectively; $P < .01$). Patients with rAAA who developed CI and were managed non-operatively exhibited lower survival than that of elective open repair and EVAR patients (60% vs 78% and 84%; $P < .01$).

Key variables of interest for CI and mortality were subjected to multivariate regression analysis to identify independent predictors of CI and mortality (Tables IV and V, respectively). Separate analyses were conducted for all patients and only those undergoing elective repairs (open and EVAR). Because the presence of rAAA demonstrated significant multicollinearity with the NIS disease severity score, the multivariate analyses were conducted with and without the disease severity score.

In analyzing all patients (those with rAAA and nonruptured AAA), the strongest predictor of the development of CI was rAAA. In the setting of elective repair, open repair was the strongest predictor of the development of CI. When the disease severity score was included in the analyses, it too was a predictor of CI (Table IV). When the entire

Table V. Independent predictors of mortality

Patient population	*Risk factor*	*Adjusted OR*	*95% CI*	P
All patients	Ruptured AAA	13.2	12.5-14.0	<.001
	Log DSS	7.5	6.7-8.5	<.001
	Colonic ischemia	4.5	4.0-5.0	<.001
	Age > 78 y	1.5	1.4-1.6	<.001
	Female gender	1.4	1.3-1.5	<.001
Non-ruptured patients	Log DSS	7.0	6.2-7.9	<.001
	Colonic ischemia	2.4	2.1-2.7	<.001
	Open	2.3	2.1-2.6	<.001
	Age > 78 y	1.2	1.1-1.3	<.001

OR, Odds ratio; *CI,* confidence interval; *AAA,* abdominal aortic aneurysm; *DSS,* disease severity score; *open,* elective open AAA repair; *EVAR,* elective endovascular AAA repair.
The ORs provided are the results of multivariate logistic regression analysis.

cohort was analyzed, female gender increased the risk of developing CI by 60%.

Similarly, the strongest predictor of mortality among all patients was rAAA. Disease severity score, CI, advanced age, and female gender all contributed to mortality. Similar trends were noted in elective repairs, with disease severity score, CI, open repair, and advanced age all contributing significantly to mortality (Table V). Female gender was associated with a modest reduction in mortality risk.

DISCUSSION

This series identified nearly 90,000 patients who underwent open and endovascular AAA repair. The cohort presented is similar with regard to baseline demographics and distribution of open and endovascular repair to those reported by previous investigators using similar methodology.[3,4] The use of the NIS for evaluating outcomes after aneurysm repair has been previously validated by other investigators.[21] In contrast to previously published reports, patients undergoing EVAR in the current series, despite their more advanced age, appeared to manifest fewer underlying medical illnesses than open repair patients. This finding may be due to using the disease severity score as a global marker of comorbid illness, rather than tabulating individual comorbidities as has been previously done.[3,4]

CI has long been recognized as a life-threatening complication of open AAA repair. The incidence of clinically evident CI after elective open AAA repair is almost uniformly reported at 1% to 6% and varies depending on sample size, patient presentation (elective vs emergent), and definition of CI.[22-24] Our incidence rate of 2.2% is in agreement with previously published results.

As in elective open AAA repair, previous investigators[16-19] have noted a CI incidence of 1% to 6% and associated mortality of 38% to 50% after elective EVAR. In single-institution comparisons of elective EVAR and open AAA repair, several investigators have reported apparent differences in the incidence of CI. Moore et al[25] found no CI in patients undergoing EVAR compared with 2% in patients undergoing open repair. Similarly, Mehta and colleagues[12] reported CI requiring colectomy in 2.6% of open repair patients but only 0.6% of EVAR patients. Despite these apparent differences, neither study achieved statistical significance because of their small sample sizes. Our study demonstrated a similar risk of CI after EVAR (0.5%) compared with open AAA repair (1.9%) as has been previously reported, but our study overcomes the limitations of smaller series in that its large sample size minimizes the likelihood of missing a true difference.

Rates of CI after open AAA repair increase dramatically in the setting of rAAA. In this situation, the incidence of CI is approximately 10% to 36% and, again, is dependent on whether the diagnosis is made on the basis of clinical or endoscopic findings.[26-29] The incidence of CI after repair of rAAA in our report (8.9%) is somewhat lower than average. Overall mortality rate with rupture, lack of endoscopic evaluation in all patients, lack of postmortem confirmatory data in patients who developed multiorgan system failure before the diagnosis of CI, and the population of rAAA treated with EVAR may all have contributed to a lower incidence.

The development of CI after AAA repair is associated with significant morbidity. This finding is reflected in the differences we found in length of stay, hospital charges, and discharge disposition between those patients who did and did not develop CI. CI was associated with an average of 13 additional hospital days and, in light of the number of patients developing the complication, an overall increase in 25,233 hospital days. Additionally, CI was associated with a greater than twofold increase in hospital charges, with a mean difference of $105,271 for overall excess hospital charges of more than $200 million. Patients developing CI were nearly twice as likely to require skilled nursing as those who did not, further increasing the economic burden associated with the development of this complication.

In addition to the morbidity associated with CI, this complication also confers a high mortality of approximately 25% to 90%.[6,8,22,23,28,30] Chen and colleagues[30] have identified CI as a strong independent predictor of mortality in patients undergoing open AAA repair for rAAA. Our CI-associated mortality rate of 37.8% and the identification of CI as an independent mortality risk factor (fourfold with inclusion of repair of rAAA, twofold in the setting of

elective repair) are in agreement with these previously published results.

It is clear that the degree and extent of ischemic injury to the colon have a large impact on overall outcome, as is evident in differences in management and outcome among patients who developed CI. Patients who developed CI and did not undergo colectomy, in general, did better than patients who underwent colectomy. Patients who develop CI and do not undergo colectomy likely comprise two distinct subgroups: those with mild ischemia limited to the mucosa in whom non-operative management is appropriate and moribund patients in whom colectomy is unlikely to yield any survival benefit. Presumably, because mortality was lower in the non-operative group, the majority of those patient managed non-operatively in the current series had milder and less extensive injury than those managed operatively, as has been reported by others.[16]

Patients who underwent repair of rAAA and subsequently developed CI were more likely to undergo colectomy. Although our data set does not provide a clear reason for the increased likelihood of colectomy in this patient subgroup, the increased physiologic insult associated with ruptured aneurysm and the more liberal use of surveillance endoscopy in patients with ruptured aneurysm may explain this finding.[28] Unfortunately, it was not possible with the present data to further differentiate those patients who suffered ischemic injury limited to the mucosa from those with transmural necrosis, and those with segmental colitis from those with more extensive injury, both of which predict higher mortality.[28,31] Interestingly, in patients who underwent elective EVAR and subsequently developed CI, mortality associated with colectomy was significantly greater than that associated with either rAAA or elective open repair. There are two possible explanations for this difference. There may have been a lower index of suspicion for CI in patients treated with EVAR; therefore, the presentation of CI may have been more advanced at the time of diagnosis. Alternatively, CI may represent only one component of visceral ischemia among EVAR patients. As has been reported previously, there is a difference in the pathogenesis of CI in open repair and EVAR and an increased likelihood of concomitant small bowel ischemia associated with EVAR.[16-19] Our data set did not include information on the timing of the development of CI in relation to aneurysm repair nor did it include the presence of associated small bowel ischemia, so these explanations remain speculative.

Although previous studies have suggested that a higher incidence of CI is associated with open repair than with EVAR,[12,25] in this study we identified open repair as an independent risk factor for the development of CI. We confirmed previous investigators' findings that rAAA is an independent CI risk factor.[6,7,9] Female gender has been associated with rAAA at presentation and increased mortality risk, but gender has not previously been associated with an increased risk of CI.[32] Explanations for the increased risk of CI associated with female gender are not forthcoming from the current literature, but the increased risk may reflect differences in case difficulty, aneurysm anatomy, thrombus characteristics, or the degree of visceral collateral circulation. It should be pointed out, however, that none of these potentially important variables is contained in the NIS.

We should point out several limitations of the present study. Large databases like the NIS provide a wealth of information but lack specific data that could allow more definitive conclusions such as surgeon experience with EVAR, lack of detailed patient anatomic and operative information, and long-term follow-up. In particular, because all abdominal aneurysms are uniformly coded by *ICD-9-CM*, there is no possibility of delineating juxtarenal from infrarenal aneurysms. In light of the current state of endovascular technology, the open repair group is undoubtedly enriched, to some degree, with complex aneurysm types, the repair of which increase the risk of visceral ischemia.[33]

Because this is purely a large observational study, it is not and should not be interpreted as the gold standard. In addition, NIS provides no information on appropriateness of operative and perioperative management or readmission rates, and no data beyond the in-hospital complication or mortality data. It also does leave open the possibility of coding errors that may not only affect the type of procedure and perioperative data but also outcomes.[34] Our goal, however, was to identify—as nearly as possible—national trends regarding ischemic complications of aneurysm repair. The benefits of this large sample size allow us to draw our conclusions on the basis of all different levels of experience, skill, and institution size and not, as is often the case, on the findings of smaller randomized trials performed by experts in select institutions.

CONCLUSION

Colonic ischemia is a relatively uncommon but devastating complication after AAA repair. We found that a higher incidence of CI is associated with open AAA repair than with EVAR regardless of the indication for operation. Open AAA repair remained a significant risk factor for CI after adjusting for potential confounding factors including rAAA. When it does occur, CI is associated with a twofold increase in mortality and significant resource consumption, with an estimated 25,233 additional hospital days and $200 million in excess hospital charges. CI incidence after AAA repair may likely decrease because EVAR is more broadly applied to the treatment of AAA.

AUTHOR CONTRIBUTIONS

Conception and design: JP, MM, ME, VS, SS
Analysis and interpretation: JP, MM, ME, VS, SS
Data collection: JP, MM, SS
Writing the article: JP, SS
Critical revision of the article: JP, MM, ME, VS, SS
Final approval of the article: JP, MM, ME, VS, SS
Statistical analysis: JP, MM, SS
Obtained funding: JP, SS
Overall responsibility: SS

REFERENCES

1. Volodos' NL, Shekhanin VE, Karpovich IP, Troian VI, Gur'ev I. [A self-fixing synthetic blood vessel endoprosthesis]. Vestn Khir Im I I Grek 1986;137:123-5.
2. Parodi JC, Palmaz JC, Barone HD. Transfemoral intraluminal graft implantation for abdominal aortic aneurysms. Ann Vasc Surg 1991;5: 491-9.
3. Dillavou ED, Muluk SC, Makaroun MS. Improving aneurysm-related outcomes: nationwide benefits of endovascular repair. J Vasc Surg 2006;43:446-51.
4. Lee WA, Carter JW, Upchurch G, Seeger JM, Huber TS. Perioperative outcomes after open and endovascular repair of intact abdominal aortic aneurysms in the United States during 2001. J Vasc Surg 2004;39: 491-6.
5. Marston A, Pheils MT, Thomas ML, Morson BC. Ischaemic colitis. Gut 1966;7:1-15.
6. 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.
7. 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.
8. Longo WE, Lee TC, Barnett MG, Vernava AM, Wade TP, Peterson GJ, et al. Ischemic colitis complicating abdominal aortic aneurysm surgery in the U.S. veteran. J Surg Res 1996;60:351-4.
9. Sandison AJ, Panayiotopoulos Y, Edmondson RC, Tyrrell MR, Taylor PR. A 4-year prospective audit of the cause of death after infrarenal aortic aneurysm surgery. Br J Surg 1996;83:1386-9.
10. Axelrod DJ, Lookstein RA, Guller J, Nowakowski FS, Ellozy S, Carroccio A, et al. Inferior mesenteric artery embolization before endovascular aneurysm repair: technique and initial results. J Vasc Interv Radiol 2004;15:1263-7.
11. Lee C, Dougherty M, Calligaro K. Concomitant unilateral internal iliac artery embolization and endovascular infrarenal aortic aneurysm repair. J Vasc Surg 2006;43:903-7.
12. Mehta M, Roddy SP, Darling RC III, Ozsvath KJ, Kreienberg PB, Paty PS, et al. Infrarenal abdominal aortic aneurysm repair via endovascular versus open retroperitoneal approach. Ann Vasc Surg 2005;19:374-8.
13. Mehta M, Taggert J, Darling RC III, Chang BB, Kreienberg PB, Paty PS, et al. Establishing a protocol for endovascular treatment of ruptured abdominal aortic aneurysms: outcomes of a prospective analysis. J Vasc Surg 2006;44:1-8.
14. Nevelsteen I, Duchateau J, De VP, De LJ. Ischaemic colitis after endovascular repair of an infrarenal abdominal aortic aneurysm: a case report. Acta Chir Belg 2006;106:588-91.
15. Verhoeven EL, Prins TR, van den Dungen JJ, Tielliu IF, Hulsebos RG, van SR. Endovascular repair of acute AAAs under local anesthesia with bifurcated endografts: a feasibility study. J Endovasc Ther 2002;9: 729-35.
16. Dadian N, Ohki T, Veith FJ, Edelman M, Mehta M, Lipsitz EC, et al. Overt colon ischemia after endovascular aneurysm repair: the importance of microembolization as an etiology. J Vasc Surg 2001;34: 986-96.
17. Geraghty PJ, Sanchez LA, Rubin BG, Choi ET, Flye MW, Curci JA, et al. Overt ischemic colitis after endovascular repair of aortoiliac aneurysms. J Vasc Surg 2004;40:413-8.
18. Maldonado TS, Rockman CB, Riles E, Douglas D, Adelman MA, Jacobowitz GR, et al. Ischemic complications after endovascular abdominal aortic aneurysm repair. J Vasc Surg 2004;40:703-9.
19. Zhang WW, Kulaylat MN, Anain PM, Dosluoglu HH, Harris LM, Cherr GS, et al. Embolization as cause of bowel ischemia after endovascular abdominal aortic aneurysm repair. J Vasc Surg 2004;40: 867-72.
20. Agency for Health Care Research and Quality, Healthcare Cost & Utilization Project. Overview of the Nationwide Inpatient Sample. Available at: http://www.hcup-us.ahrq.gov/nisoverview.jsp. Accessed January 2007.
21. Huber TS, Wang JG, Derrow AE, Dame DA, Ozaki CK, Zelenock GB, et al. Experience in the United States with intact abdominal aortic aneurysm repair. J Vasc Surg 2001;33:304-10.
22. Brewster DC, Franklin DP, Cambria RP, Darling RC, Moncure AC, Lamuraglia GM, et al. Intestinal ischemia complicating abdominal aortic surgery. Surgery 1991;109:447-54.
23. Cohen JD, Singer P, Grunberg G, Grozovski E, Sulkes J, Zelikovski A. Outcome after elective infrarenal aortic aneurysm surgery. World J Surg 1998;22:278-82.
24. Johnston KW. Multicenter prospective study of nonruptured abdominal aortic aneurysm, part II: variables predicting morbidity and mortality. J Vasc Surg 1989;9:437-47.
25. Moore WS, Kashyap VS, Vescera CL, Quinones-Baldrich WJ. Abdominal aortic aneurysm: a 6-year comparison of endovascular versus transabdominal repair. Ann Surg 1999;230:298-306.
26. Alonso-Perez M, Segura RJ, Pita S, Cal L. Surgical treatment of ruptured abdominal aortic aneurysms in the elderly. Ann Vasc Surg 1999;13:592-8.
27. Alonso-Perez M, Segura R, Pita S, Cal L. Operative results and death predictors for nonruptured abdominal aortic aneurysms in the elderly. Ann Vasc Surg 2001;15:306-11.
28. Champagne BJ, Darling RC III, Daneshmand M, Kreienberg PB, Lee EC, Mehta M, et al. Outcome of aggressive surveillance colonoscopy in ruptured abdominal aortic aneurysm. J Vasc Surg 2004;39:792-6.
29. Hsiang YN, Turnbull RG, Nicholls SC, McCullough K, Chen JC, Lokanathan R, et al. Predicting death from ruptured abdominal aortic aneurysms. Am J Surg 2001;181:30-5.
30. Chen JC, Hildebrand HD, Salvian AJ, Taylor DC, Strandberg S, Myckatyn TM, et al. Predictors of death in nonruptured and ruptured abdominal aortic aneurysms. J Vasc Surg 1996;24:614-20.
31. Longo WE, Ward D, Vernava AM III, Kaminski DL. Outcome of patients with total colonic ischemia. Dis Colon Rectum 1997;40: 1448-54.
32. Dillavou ED, Muluk SC, Makaroun MS. A decade of change in abdominal aortic aneurysm repair in the United States: have we improved outcomes equally between men and women? J Vasc Surg 2006;43: 230-8.
33. Back MR, Bandyk M, Bradner M, Cuthbertson D, Johnson BL, Shames ML, et al. Critical analysis of outcome determinants affecting repair of intact aneurysms involving the visceral aorta. Ann Vasc Surg 2005;19: 648-56.
34. Guller U, Jain N, Hervey S, Purves H, Pietrobon R. Laparoscopic vs open colectomy: outcomes comparison based on large nationwide databases. Arch Surg 2003;138:1179-86.

Submitted Jan 2, 2008; accepted Mar 18, 2008.