

Colon ischemia following abdominal aortic aneurysm repair in the era of endovascular abdominal aortic repair

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Objective: To review, in the era of endovascular abdominal aortic repair (EVAR), the clinical spectrum of colonic ischemia (CI) following abdominal aortic aneurysm (AAA) repair and to assess the rate, overall mortality, and associated factors of occurrence.

Methods: Between 1995 and 2005, 1174 patients with infrarenal AAA were treated either by open surgery (n = 682) or by EVAR (n = 492). Preoperative risk factors, clinical presentation, intraoperative data, and early postoperative outcomes were prospectively assessed. Overt colonic ischemia as proven by colonoscopy and/or by operation was considered as a validating event and was correlated to collected variables.

Results: CI occurred in 34 patients (2.9%). Eighteen out of 34 (53%) patients died within 1 month. At 2 years, the survival rate was 35% in the CI group vs 86% in the non-CI group. Associated factors of occurrence of CI were: type of operation (open group = 27/682 [4%] vs EVAR = 7/492 [1.4%] [$P = .01$]), aneurysm rupture (11/88 [12.5%] vs 23/1086 [2.1%], $P < .001$), preoperative renal insufficiency (4/30 [13.3%] vs 29/1133 [3.1%], $P = .01$), preoperative respiratory insufficiency (8/157 [7%] vs 23/1005 [2%], $P = .01$), duration of operation (<2 hours [518] = 1.7%, between 2 to 4 hours [558] 2.9%, more than 4 hours [66] 13.6%, $P = .001$). Mean blood loss was greater in patients with CI (CI = 2000 ml [650-3350] than in those without CI = 1000 ml [500-1800] $P = .008$). Logistic regression analysis showed that rupture (OR 6.03 [interval of confidence (IC) 95% 2.68-13.5] $P = .0001$), duration of operation (OR 5.73 [IC 95% 2.06-15.9] $P = .001$) and creatinin > 200 mol/l (OR 4.67 [IC 95% 1.39-15.7] $P = .028$) were independent factors of CI. The mortality due to colonic ischemia was not statistically different between open surgery 14/27 (52%) and EVAR 4/7 (57%).

Conclusion: CI remains a serious complication following AAA repair. In the univariate analysis, EVAR was associated with a lower rate of colonic ischemia. However, the logistic regression analysis showed that only rupture, long duration of operation, and prior renal disease were independently associated with CI. Within the two treatment modalities, the mortality rate remained identical. (J Vasc Surg 2008;47:258-63.)

Colonic ischemia is a major adverse event after abdominal aortic aneurysm (AAA) repair. Following open repair, the incidence of clinically significant colonic ischemia is in the range of 1 to 3% after elective surgery¹⁻³ and 10% in case of rupture.^{4,5} When routine postoperative colonoscopy is performed, the incidence reaches 5% to 9% after elective surgery and 15% to 60% following rupture.^{5,6}

Endovascular abdominal aortic repair (EVAR) has a lower rate of early postoperative mortality and of overall complications than open repair.^{7,8} EVAR has also been advocated for ruptured aneurysms with a seemingly decreased mortality rate.⁹⁻¹¹ However, ischemic complications such as CI has been reported since the early phase of development of EVAR¹² with a current incidence of 1.5% to 3%.^{13,14}

Presumed causes of CI are nonocclusive ischemia due to shock or vasopressive drugs, inferior mesenteric artery, and/or internal iliac arteries occlusion and/or athero-embolization.^{2,15-17} It has been suggested that EVAR may reduce the severity of colonic ischemia.¹⁸ However, firm comparative data are missing.

The current study was undertaken to review in the era of EVAR the clinical spectrum of CI and to assess the rate, mortality, and associated factors of occurrence.

METHODS

Since 1985, all vascular procedures performed in the Department of Vascular Surgery at Henri Mondor Hospital, University Paris XII, are stored prospectively in a specifically designed data base (Logit). The data base sheets include preoperative risk factors, anatomical features, intraoperative data, and early and late postoperative outcomes. Pre- and intraoperative data are filled by senior surgeons. In-hospital outcomes are filled by residents when patients leave the hospital. A weekly review of all cases allows a double check of the data and coded items, and finally, the medical staff reviews every 3 months the records of patients who have died or who had life threatening complications. In the data base, the definitions of preoperative risks factors fulfilled the criteria of the ad-hoc committee for reporting

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standards.¹⁹ The variables examined in this report were age, gender, preoperative risk factors (diabetes, coronary disease, ventricular dysfunction, renal insufficiency defined by serum creatinin >200 micromoles, pulmonary disease, previous colectomy), indication for surgery (ruptured vs elective), type of treatment (EVAR vs open), and operative details (duration of operation, blood loss, inferior mesenteric artery reconstruction, site of distal anastomoses, hypogastric artery embolization or coverage).

For the purpose of this study, we reviewed the 1174 consecutive infrarenal AAA patients who were treated between January 1995 and December 2005. A total of 682 (58%) were treated by open surgery and 492 (42%) by EVAR. When performing EVAR, we used commercially available stent grafts: Stentor (Mintec, Bahamas) n = 8 (1.6%), Talent (Medtronic, Santa Rosa, Calif) n = 8 (1.6%), Ancure (EVT Guidant, San Francisco, Calif) n = 14 (2.8%), AneurX (Medtronic) n = 24 (4.9%), Excluder (Gore, Flagstaff, Ariz) n = 25 (7%), Vanguard (Boston Scientific, Natick, Mass) n = 69, (13%), and Zenith (Cook) n = 334 (68%). All were EEC marked. Since 2001, the date of creation of this agency, all were approved by the AFS-SAPS (Agence Française de Sécurité Sanitaire des Produits de Santé), the French version of the FDA in the United States.

In the open repair group, we used standard Dacron grafts (Datascope La Ciotat France or Edwards Maurepas 78000 France). Treatment allocation was influenced by anatomical factors and followed the French regulation, which so far, prohibits EVAR in relatively good risk patients unless they are enrolled in clinical trials. From a technical point of view, in open repair patients, we reattached fully patent IMA when the back flow was poor or when intraoperative Doppler ultrasound study of the colonic arcade failed to demonstrate pulsatile flow. In EVAR patients whose aneurysm extended below the iliac bifurcation, the internal iliac artery was covered and coil embolized prior to stent graft placement. However, in a few cases with occluded contralateral internal iliac, we bypassed the internal iliac artery from the ipsilateral common femoral artery. When coil embolization was performed, we strove, whenever feasible, for blocking the origin of the internal iliac artery.

Postoperatively, we did not perform systematic colonoscopy; however, our indications for colonoscopy were relatively liberal: patients with postoperative severe abdominal pain, early diarrhea, rectorrhagia, unexplained hemodynamic disturbances, unexplained organ failures, and/or elevated lactate enzymes were assessed for colonic ischemia either by colonoscopy and/or by redo surgery.

Colectomy was performed (1) at the time of AAA repair, in case of obvious transmural necrosis of the colon and (2) postoperatively, when colonic ischemia upon colonoscopy was either extensive or transmural or when CI was associated with clinical and/or biological symptoms. Observation with repeat colonoscopy was done in pauci symptomatic cases with ischemia seemingly limited to the mucosa. The records of patients identified with colonic ischemia in the data base were reviewed to assess clinical

presentations and specific outcomes. Statistics were performed by the Department of Biomedical statistics of Henri Mondor Hospital (Dr Roudot-Thoraval). Statistical comparisons were made by means of χ^2 test or Fisher exact test if appropriate for qualitative data, and Student *t* test or Mann Whitney nonparametric test if appropriate for continuous data. *P* values < .05 were considered significant. Variables associated with colonic ischemia in univariate analysis were tested in a stepwise logistic regression analysis. Adjusted odds ratios (ORs) with 95% confidence intervals were estimated from the model. Statistical analysis was performed with SPSS 13.0 and BMDP statistical software.

RESULTS

Among the 1174 consecutive infrarenal AAA patients, they were 1097 (93, 4%) males and 77 females. Mean age was 70.8 ± 9.0 years. Six hundred and eighty-two were treated by open surgery and 492 by EVAR. Postoperative colonic ischemia occurred in 34 patients (2.9%). Colonic ischemia was diagnosed at the time of aneurysm repair in five cases (15%): three were ruptured aneurysms and two were elective difficult cases. Colectomy was performed at the end of the intervention due to obvious transmural necrosis. Postoperative colonic ischemia was diagnosed in 29 patients (85%): by colonoscopy in 21/29 cases (72%) and by early reoperation in eight cases. For those patients with colonic ischemia diagnosed postoperatively, diarrhea was present in 15/29 (53%), rectorrhagia in 9 (31%), septic shock in 16/29 (57%), persistent low blood pressure with multiple organ failure in 24/29 (84%), and fever (temperature >38° Celsius) in 20/29 (69%). Review of biological parameters showed a high level of lactates in 23/29 cases (81%), elevated creatinin (ie, increase by at least 30% of the preoperative value) in 18/29 (65%), and elevated leucocytes (>10,000 c/mm³) in 20/29 (72%).

Table I shows the statistically significant variables identified in the univariate analysis. In patients without CI and in patients with CI, the median (quartiles range) blood loss was 1000 ml (500-1800) and 2000 ml (650-3350), respectively (*P* = .008). The median (quartiles range) operating time was 150 mn (120-180) and 180 mn (120-255), respectively (*P* = .0005). Analysis of technical factors with regard to CI occurrence is shown in Table II.

Table III shows the results of the multivariate analysis. Aneurysm rupture, length of operation (>4 hours), and preoperative renal insufficiency were independently associated with postoperative colonic ischemia. The prognosis of colonic infarction was poor. Eighteen patients (52%) died within the first postoperative month. The mortality due to colonic ischemia was not statistically different between open surgery 14/27 (52%) and EVAR 4/7 (50%).

Twenty patients (59%) were treated by colectomy. Twelve of them died (60%). Colon resection was performed between 0 and 13 days with the majority at day 3. In six patients, colectomy was not performed because of extensive necrosis and multiple organ failure. All of them died. In the eight remaining cases, conservative therapy was attempted because ischemia was deemed less extensive. None of these

Table I. Predictive risk factor of colonic ischemia following AAA repair (univariate analysis)

(N° at risk)	% Colon ischemia	P value
Gender:		
Male (1097)	2.8	.59
Female (77)	3.9	
Diabetes:		
No (1046)	2.9	.76
Yes (116)	1.7	
Coronary disease:		
No or asymptomatic (874)	2.3	.27
Angina pectoris stable or unstable (254)	3.5	
Left ventricular function:		
Normal or mild (1002)	2.5	.66
Severely impaired (59)	3.4	
Kidney:		
Creatinin < 200 µmol (1133)	2.7	.01
Creatinin > 200 µmol (30)	13.3	
Respiratory function:		
Normal or moderately impaired (1005)	2.5	.07
Severely impaired (157)	5.1	
Previous colectomy: (44)	4	.89
Presentation:		
Nonruptured (1086)	2.1	<.0001
Ruptured (88)	12.5	
Operative duration:		
≤2 h (518)	1.7	
2-4 h (558)	2.9	.001
>4 h (66)	13.6	
Procedure:		
Open surgery (682)	4	.01
EVAR (492)	1.4	

AAA, Abdominal aortic aneurysm; EVAR, endovascular abdominal aortic repair.

Table II. Technical details of patients treated by open repair and by EVAR regarding the occurrence of colon ischemia

Open repair (N° at risk)	% Colon ischemia	P value
Tube graft (239)	4.02	
Aorto uni or bi iliac graft (395)	2.7	
Aorto bifemoral graft (21)	22	.001
IMA reimplantation		
Yes (39)	4	
No (602)	3.9	ns
EVAR		
Hypogastric artery loss		
No (379)	.7	
Yes (106)	3.6	.046

EVAR, Endovascular abdominal aortic repair; IMA, inferior mesenteric artery.

patients died. Colonic continuity was re-established in five out of six surviving patients after colonic resection. Major postoperative complications were seen in the vast majority of patients with colonic ischemia. Cardiac complications occurred in 18/34 (53%), pulmonary complications in 20/34 (61%), and renal insufficiency in 20/34 (61%) of whom 14 (41%) required hemodialysis.

Table III. Multivariate analysis of factors associated with colonic ischemia following AAA repair

Risk factors	Odd ratio	IC 95% Odds ratio	P value
Presentation:			
Nonruptured	1*	—	
Ruptured	6.03	2.68–13.5	.0001
Operative duration:			
≤2 h (518)	1*		
2-4 h (558)	1.41	.60–3.28	.43
>4 h (66)	5.73	2.06–15.9	.001
Kidney:			
Creatinin ≤ 200 µmol	1*	—	
Creatinin > 200 µmol	4.67	1.39–15.7	.028

*Reference category.

DISCUSSION

Colonic necrosis remains an infrequent but devastating complication following AAA repair. In the current series, which included open and EVAR patients, the overall rate of colonic ischemia was 2.9%. This rate is similar to previous reports investigating open surgery before EVAR was introduced. It was 1% out of 1420 patients in Brewster's series,¹ and 2.6% out of 1800 patients in the SWEDAC registry.² In EVAR series, the rate of colonic ischemia was 2.9% out of 278 patients for Dadian¹⁴ and 1.2% out of 311 patients for Maldonado.¹³ In the univariate analysis, we found more colonic ischemia following open surgery (4%) than after EVAR (1.4%). However, in the multivariate analysis the type of AAA repair was not an independent variable, underlining the weight of stronger parameters such as rupture, length of operation, and renal insufficiency.

Associated factors

In our series the univariate analysis found that type of treatment, rupture, duration of operation, renal disease, pulmonary dysfunction, blood loss, femoral anastomosis, and hypogastric artery loss were statistically associated with the onset of CI. Logistic regression analysis showed that rupture, duration of operation, and renal disease were independently associated with CI. Associated factors of colonic ischemia were screened in previous reports of open repair and in scarce report of EVAR.

Björk,⁵ in a multicenter registry, found that among 2824 patients operated between 1987 and 1993, 62 (2.1%) presented postoperative intestinal ischemia. In this cohort, 1239 patients had elective AAA repair, 561 had emergent AAA repair, and 1014 had surgery for occlusive disease and miscellaneous indications. Multivariate analysis found that shock, emergent surgery, renal disease, and aortofemoral grafts were significantly associated with colonic ischemia. Due to relative small number, in EVAR series, associated factors of colonic ischemia were seldom investigated per se, although anecdotal reports^{13,14} shed some lights on this issue.

Mechanisms

Colonic ischemia following AAA repair has a multifactorial origin.

AAA rupture. AAA rupture has previously been identified^{1,5,6,20} as a major predictor of colonic ischemia. In the current series, the incidence of colonic ischemia was 2% for nonruptured aneurysms, 7.8% from symptomatic aneurysms without peri-aortic hematoma, and 14% for true ruptured AAA. From the current series, we cannot draw strong recommendations of EVAR in ruptured AAA for two reasons: (1) So far, the fact that our group performed EVAR for ruptured AAA for patients with a relative stable hemodynamical status⁹ may induce a bias; and (2) The single CI after EVAR for ruptured AAA had both hypogastric arteries blocked. Although large series are lacking, they are clues that EVAR may be beneficial in that setting. Coppi¹¹ reported a series of 124 AAA rupture, 33 of whom were treated by EVAR. The rate on CI was 3% in EVAR patients and 8.7% in the open repair patients.

Duration of operation. In the current series, operations which lasted more than 4 hours were followed by a much higher rate of CI (13.6% vs 2%). This finding confirms the previous report from the Swedac registry,⁵ in which patients who presented with colon ischemia had a longer duration of operation. Although rarely investigated per se, length of operation is an indirect clue of technical difficulties. It is also related with more intraoperative hemodynamic disturbances and more blood loss as we observed in patients who presented postoperative CI.

Anatomical factors

IMA patency. In our series, the rate of colonic ischemia was similar whether the IMA was reattached or not during open repair. It is our policy to reattach a fully patent IMA when the back flow is poor or when intraoperative Doppler ultrasound study of the colonic arcade fail to demonstrate pulsatile flow. However, from our study alone, we cannot draw firm conclusion regarding the utility of IMA reimplantation since we did not collect the status of the IMA at operation (patent with back flow, patent without back flow or thrombosed). None of the studies evaluating the influence of IMA ligation found an increased risk of colonic ischemia.³ A randomized study assessing the influence of reimplantation of the IMA did not find any reduction in colonic ischemia.²¹ In our series, EVAR patients, in whom the IMA is constantly covered, did not experience an increased risk of colonic ischemia.

Hypogastric arteries. The need to maintain the patency of the hypogastric arteries remains debated. During open surgery, ligation of both hypogastric arteries have been shown to increase the rate of colonic ischemia.^{1,5,21} In a previous report,²² we have found that in the presence of unilateral or bilateral hypogastric aneurysms, AAA open repair was associated with higher rate of postoperative complications. We found a higher rate of CI when the distal anastomosis was performed on the common femoral arteries compared with the iliac or aortic level. However, we

cannot draw firm conclusions since most femoral anastomoses actually preserved internal artery flow.

When EVAR is associated with hypogastric artery loss, the risk of pelvic or colonic ischemia is dubious. In several studies, hypogastric artery coverage with or without coil embolization was advocated as a relatively innocuous procedure.^{14,15,23,24} On the contrary, other authors^{17,25-27} found a higher incidence of colonic and pelvic ischemia as we found in the current series. It is difficult to understand why there could be a different outcome between EVAR and open surgery following loss of the hypogastric artery. Further studies are necessary to clarify this point. The following factors, which may play a role, have not yet been extensively investigated: prior patency of the hypogastric arteries, uni versus bilateral occlusion, proximal versus distal coil embolization, the use of coil or simple coverage by the limb of the graft, and evidence of an endoleak which may preserve some flow in the colonic arcade.

Collateral pathways between the superior mesenteric artery (SMA) and the IMA. Preoperative study of this arterial supply is rarely performed. Occlusion of the SMA is a recognized contraindication for EVAR. However, previous ligation of collaterals between the SMA and the inferior MI are probably as much important. In Dadian's series,¹⁴ in eight patients with colonic ischemia following EVAR one patient had a previous colectomy. Maldonado¹³ reported a series of seven patients with pelvic ischemia, four of whom had colonic ischemia. One of these patients had a previous colectomy. In our series, among patients who had colonic ischemia following EVAR, two had a previous colectomy and one had a duodeno-pancreatectomy. Of note, EVAR was chosen in these three patients on the basis of a hostile abdomen. However, as shown in Table I, the role of previous colectomy on CI occurrence, was not confirmed by statistical analysis.

Embolisms. The role of microembolization, which was emphasized as a cause of colonic ischemia after EVAR as well as after open surgery,^{13,14,26,28-30} was not fully investigated in our series.

Renal disease. Renal insufficiency represents a major risk factor of death in elective as well as in urgent AAA repair.^{20,22} It has also been previously identified as a factor of colonic ischemia.^{1,5} In the current study, renal disease was found an independent risk factor of CI. Renal disease is associated with severe arterial disease including medium and small size arteries. What is observed in legs arteries is probably applicable to colon circulation. It is fairly understandable that, in renal insufficiency patients, colon may be more affected by circulatory instability during open repair and during EVAR.

Treatment and outcome

Once diagnosis of CI is made, colectomy should be considered. Conservative treatment can only be advocated in patients with no transmural necrosis and no organ failure. However, a strict surveillance based on clinical evaluation and repeated colonoscopy is mandatory since the severity of colonic ischemia may worsen

with time. Menegaux et al³¹ reported 49 cases of CI following aortic repair, 19 had a delayed colectomy 1 to 11 days postoperatively. In our series, the majority of colon resections were performed within the first three postoperative days. Mortality following colectomy remained considerable. In prior reports, mortality was between 37% and 53%.^{6,13,14,31} It was 60% in our series. Our data are too small to allow subgroup analysis of prognostic factors. However, multiple emboli,¹³ transmural necrosis and associated organ failures,³¹ have been shown to be associated with a higher mortality rate. In our series, there was no difference in mortality whether EVAR or open repair was the primary operation. In surviving patients, reestablishment of the digestive continuity was often possible and successful, as observed in five out of seven surviving patients of the series.

CONCLUSION

Colonic ischemia remains a serious complication following AAA repair. Although in univariate analysis, EVAR was associated with a lower rate of colonic ischemia, multivariate analysis showed that the sole independent factors of CI were rupture, long duration of operation, and prior renal disease. Of note, within the two treatment modalities, the mortality rate was similar.

AUTHOR CONTRIBUTIONS

Conception and design: JPB
Analysis and interpretation: JPB, MM
Data collection: NF, MM
Writing the article: JPB, JM
Critical revision of the article: JM, PD, EA
Final approval of the article: JPB, MM, NF, JM, PD, EA
Statistical analysis: FRT, JPB
Obtained funding: Not applicable
Overall responsibility: JPB

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DISCUSSION

Dr Frank Veith (*Riverdale, NY*). Did you look at any of your excised specimens to see if there were microemboli, atheroemboli, in the colonic vessels? We agree with you that it is good to save hypogastrics, but together with Dr Mehta, we have collected more than 200 patients with unilateral hypogastric occlusion and more than 60 with bilateral hypogastric occlusion, and we have had no incidence of severe colonic ischemia. The patients who have had the colonic ischemia have all been without hypogastric occlusion, and they have all had the microemboli, so we think that is more important than just saving the hypogastric.

Dr Becquemín. Thank you, Dr Veith, for your comment. I am well aware of your paper on the topic and we also looked at the mechanisms of colonic ischemia. Unfortunately, this series is retrospective, and we have not specifically looked at microembolism in the excised specimens. However, 13 patients in the series had multiple microemboli in the legs, pelvis, and probably in the colon as well. We then agree that it is a very important issue.

Concerning the blockage of hypogastric artery, our figures are different, but contrary to your own study, our series included both open repair and EVAR, elective and rupture cases, and we concluded that it is better to save at least one hypogastric artery.

Dr Peter Gloviczki (*Rochester, Minn*). I enjoyed the presentation very much and thank you for calling attention to this very important complication. My question is, what do you use for intraoperative assessment of the colon in this type of patient and whether we should use it routinely or only selectively in patients with ruptured aneurysm? And my second question is, what should we do to decrease this complication? Should all patients with ruptured aneurysm undergo, within 24 hours, a sigmoidoscopy? I mean, the incidence that you show is very high, over 10%, so the yield may be quite high. So how do you assess the circulation postoperatively and what do you recommend to decrease this problem?

Dr Becquemín. During open repair, we routinely assess the colon with a Doppler probe following the repair of the aneurysm to decide whether the IMA should be reattached or left alone. Regarding rupture, it was our policy to use colonoscopy liberally, if not routinely. Unfortunately, once the colon is necrotic or severely ischemic, it is very rare to be able to save it with a delayed revascularization of the IMA or internal iliac artery. Again, as far as colon circulation is concerned, we inspect the back bleeding from the IMA, the gross aspect of the colon following restoration of flow, and the flow in the colon and ileal arteries with a Doppler probe.

Dr Roy Greenberg (*Cleveland, Ohio*). I have three questions for you. The first relates to whether prior colon surgery was included as one of the variables you assessed in your multivariable analysis, or were there not enough patients to really assess that?

The second question goes back to Dr Veith's question, which is, were you able to categorize the etiology of the ischemia in any of these patients? For example, the patient you showed in your example probably had low flow as opposed to an embolic problem, given the proximity of the ischemia to the colonic anastomosis. If one were to hypothesize that hypogastric flow is important for colonic ischemia, the etiology would likely be flow/perfusion in

nature rather than embolic, unless the management of such aneurysms requires undo manipulation.

My last is whether you looked at whether the left or right hypogastrics were involved in ischemia? Were they all the left side that was embolized or was it a mixture?

Dr Becquemín. Unfortunately, we had not prior colon surgery in the database, so we could not assess this item in the multivariate analysis. In the group of patients who had colonic ischemia, we found that three of them have a previous abdominal surgery.

The second question?

Dr Greenberg. Did you actually categorize patients with colonic ischemia where you had a specimen into low flow versus embolic?

Dr Becquemín. No, it was not done, and we think it was a very difficult question to answer since colon ischemia is probably multifactorial in origin. Your final question concerned the left side of the internal iliac occlusion. We did not specifically look at the side and I have not the answer to provide you with.

Dr Robert W. Hobson, II (*Newark, NJ*). I would like to return to the question regarding intraoperative testing. We also use the Doppler ultrasound methodology. Do you ever measure inferior mesenteric back pressure? And if you use Doppler or IMA back pressure measurements, are there characteristics of the Doppler method or pressure levels at which you would recommend reimplantation of the IMA?

Dr Becquemín. We have not measured the pressure. But we looked at the flow coming from the inferior mesenteric artery whether it was strong or weak or absent, and whatever the decision to reimplant or not the IMA, to assess the flow with a Doppler probe. We do not have levels to recommend except that in the absence of flow, every attempt must be made to restore the IMA. It is, however, difficult on this retrospective study to draw firm conclusions. We found no difference whether the inferior mesenteric artery was reattached or not. But since we have reimplanted the inferior mesenteric artery when the backflow was small and the flow weak, we have saved some colons.

Dr Alan B. Lumsden (*Houston, Tex*). One of the things we are unanimous, in addition to being surgeons, is that there are certain patients which are easy to predict, the patient who has got a patent IMA, who has got a celiac and an SMA stenosis is a setup for getting ischemia. Were there any direct anatomical risk factors that you could identify in these patients? A patent IMA, for example, would be an easy one which we exclude. Just the presence of a patent inferior mesenteric artery with an endograft would be a patient who we would consider more at risk and would need closer observation, for example.

Dr Becquemín. I agree that a fully patent IMA with stenotic celiac and superior mesenteric artery is at an increased risk of colon ischemia with EVAR. Unfortunately, it may be difficult to assess the IMA's patency on CT scan alone. Sometimes, you think that it is patent because the trunk of the inferior mesenteric artery is visible on the CT scan. However, the ostium itself may be occluded. A preoperative angiogram may be useful but it is not in our routine practice.