Feature

Endoleaks Following Endovascular Aortic Aneurysm Repair: Clinical Significance and Treatment Modalities

MICHEL MAKAROUN, M.D., and ALBERT ZAJKO, M.D.

ABSTRACT **The Achilles heel of endovascular management of abdominal aortic aneurysms (AAA) remains the frequent development of endoleaks. We reviewed our experience over 4 years with two devices used to treat 158 patients with AAA. Forty-one endoleaks were noted for a total incidence of 27%. Two-thirds resolved spontaneously over a 6-month observation period. Proximal endoleaks were associated with neck angulation and most resolved on their own. Distal endoleaks were persistent and required coiling to obliterate the flow around the graft. Other endoleaks were self-limited in general. The incidence of endoleaks decreased significantly with experience and recent cases show no significant differences among the two devices. Long-term follow-up of treated or spontaneously sealed endoleaks reveals shrinkage of the AAA sac similar to patients with no endoleaks. We conclude that the incidence of endoleaks can be reduced with experience and an aggressive management policy can limit their potential danger.**

Keywords **Abdominal aortic aneurysm, endovascular repair, complications, endoleaks, treatment**

The last decade has witnessed an unprecedented revolution in the treatment of abdominal aortic aneurysms (AAA) using an endovascular, less invasive approach.[1–4] The technique has been embraced quickly by patients and physicians alike. The devices have been refined with the development of commer-

M.M., Professor of Surgery and Chief Division of Vascular Surgery, University of Pittsburgh School of Medicine, Pittsburgh, PA; A.Z., Professor of Radiology and Surgery, Department of Radiology, University of Pittsburgh School of Medicine, Pittsburgh, PA.

 0894-8046,p;2000,13,1,1,14,ftx,en;pvs000077

cially available products that are simple to use and smaller in profile. Early results have been very favorable, showing wide applicability, reduction of hospital stays, and high patient acceptance.[1–4]

The Achilles heel of this approach, however, remains the incomplete exclusion of the aneurysm from circulation in a significant number of patients.[5,6] Blood flow persists in the aneurysmal sac around the endograft and implies the continuous exposure of the sac to arterial pressure. The clinical significance of such "endoleaks" or perigraft flow remains unsettled: some aneurysms continue to expand and in some cases rupture while others remain stable or even shrink in size.[5–8] The proper management of this problem is also not well established, with conversion to open repair the only certain cure.

Endoleaks have been reported with all systems used and do represent an inevitable drawback of this approach. They have been classified into several types depending on the origin of the leak. A bloodtight seal is expected at the attachment sites proximal and distal to the aneurysm. This may not occur because of poor sizing, calcifications, poor deployment, or angulation—to name a few causes resulting in flow around the attachment sites (Type I). Intrinsic to the technique also, is the expectation that branches arising from the aneurysm, such as lumbars and the inferior mesenteric artery (IMA), would spontaneously occlude with time. These are usually easily controlled during an open procedure but are left alone with endografts, and may continue to perfuse the aneurysmal sac (Type II). Graft defects and poor sealing of modular components can also be the source of the endoleak (Type III). Finally, temporary graft porosity may appear radiographically as an endoleak (Type IV).

We have previously reported our early experience with endoleaks and described a method for management of persistent cases at 6 months.[9,10] In this report we update our experience with endoleaks arising over the last 4 years to identify their characteristics, compare their rate of development with the use of two different endograft systems, and assess the effect of experience on the incidence of this problem. We also review the results of our management strategy.

PATIENTS AND METHODS

From February 1996 to February 2000, we repaired 158 abdominal aortic aneurysms with three different commercially manufactured systems. The majority of our experience is with two systems: 121 patients received a unibody Ancure® endograft produced by Guidant Endovascular Technologies Inc. (EVT, Menlo Park, CA) and 35 patients received an Excluder® device produced by W.L. Gore and Associates (Flagstaff, AZ). Most procedures (91 Ancures and 35 Excluders) were part of multicenter phase II or III evaluation of these grafts with 30 performed following FDA approval of the Ancure

endograft in September 1999. All protocols were approved by the University of Pittsburgh institutional review board and reviewed annually.

All procedures were performed in the operating room with a mobile digital fluoroscopy unit by a vascular surgeon. An interventional radiologist acted as a first assistant on more than 70% of cases. Anatomic requirements varied somewhat among various grafts but all included a proximal neck that is at least 15 mm in length and no larger than 26 mm in diameter. Tube endografts required a minimum distal neck length of 12 mm while bifurcated devices required a suitable nondilated iliac artery landing zone. A unilateral aorto-iliac device was used in 6 patients with complicated iliac anatomy. No patient had the simultaneous exclusion of both internal iliac arteries.

The follow-up was performed according to the multicenter protocols and was somewhat different among various groups. All patients, however, had an intraoperative angiogram to assess the adequacy of device placement and aneurysm exclusion. Initial assessment of results was performed within 1 month on all patients using contrast enhanced Computer Tomography (CT). Duplex scanning was also used in the majority of patients. A 6-month CT scan was obtained on all phase II patients and all subsequent patients with a recognizable endoleak during the initial assessment. All patients also had yearly evaluations thereafter. Any patient with evidence of incomplete exclusion of the AAA on any study within 1 month of implantation was considered to have an endoleak. Late presence or absence of endoleaks was determined from contrast enhanced CT. In case of renal compromise or contrast allergy, duplex scanning was used as the determinant factor. The presence of endoleaks in this report was determined by the principal investigator after personal review of all studies.

Records of all patients and all pre- and postoperative imaging studies of patients with endoleaks were reviewed. Preoperative variables were compared between the group of patients who developed endoleaks and those who did not. Preoperative size of the AAA, size of the native artery at the attachment sites, degree of calcification (minimal, moderate or circumferential), or mural thrombus presence were noted. Angulation of the necks was measured and the status of preoperative patent branches of the AAA was determined. The site of the endoleak was classified from information obtained from all three diagnostic modalities used in the immediate postoperative evaluation. The classification was unchanged for the rest of the analysis. The size of the aneurysmal sac on follow-up was considered to be the minor axis on the largest axial slice on the CT scan. No volume analysis was performed.

Endoleak management decisions were not addressed in any protocol, including the type and timing of intervention. This decision was left to the individual investigators and their patients to determine. Our management strategy of endoleaks has been previously described.[9] In short, we initially observed our patients for spontaneous resolution for 6 months. Persistent endoleaks at 6 months were then examined by angiography and treated by coil embolization in

the majority of cases. Follow-up CT scans were used to assess the results of intervention with repeated coiling performed when necessary. Some exceptions were related to the medical condition of the patient or to shrinkage of the aneurysm.

RESULTS

One hundred fifty-eight grafts were attempted in 157 patients (129 men and 28 women). Graft deployment was successful in 152 for an immediate success rate of 97%. The highest rate of failure was in 3 of 18 women with an attempted Ancuret graft (16%) mostly because of access issues. One high-risk patient died from an autopsy proven, massive MI on the third postoperative day for an operative mortality of 0.6%. That patient had exhibited an endoleak on his completion angiography. Three patients were converted immediately and two more cases were aborted because of access problems. There has been no late ruptures or conversions to open repair. Ten patients have so far expired during follow-up from a variety of unrelated medical problems.

A. Incidence of Endoleaks

Endoleaks were noted at some time during the follow-up of 41 of 153 endografts for a total incidence of 27%. This included a late cephalad migration of the distal attachment of a tube endograft. Subgroup analysis showed no difference in aneurysm size, neck diameter, or distal attachment-site diameter among patients with or without endoleaks. Degree of calcification score and presence of mural thrombus were also similar between the two groups. The presence or absence of large branches on the preoperative angiogram, such as lumbars, accessory renals, or inferior mesenteric arteries (IMA) was not clearly predictive of endoleaks either.

Effect of Neck Angulation

We have previously reported the effect of neck angulation on proximal endoleak development with the Ancure® graft.[9] All patients with endoleaks associated with the proximal attachment system had an angulation of the proximal neck in excess of 35 degrees to the main flow channel of the AAA. The likelihood of developing a proximal attachment system endoleak was 50% if the angle on preoperative angiography exceeded 35 degrees. Curiously, the same is true for the Excluder® graft with 50% (4 of 8) of patients with a neck angulation exceeding 35 degrees on preoperative angiography, exhibiting an initial endoleak close to the proximal portion of the device.

Effect of Graft Configuration

The Ancure® system is provided with three configurations. The "bifurcated" configuration was associated with a much lower incidence of

endoleaks 18/85 (21%) when compared with the "Tube" 12/24 (50%) or "Aorto-iliac" 3/7 (43%).

Effect of Graft Type

Endoleaks were noted in 33/116 (28%) patients with the Ancure® system. Patients receiving the Excluder® device, which is a bifurcated modular system, had a lower incidence of endoleaks noted in 7/35 (20%) cases. This was similar, however, to the incidence of the bifurcated Ancure® mentioned above.

Effect of Experience

The Ancure® system has been used in our center for all 4 years of our program. Patients in Phase II (tube, bifurcated and aorto-iliac) treated from February 1996 to May 1998 developed endoleaks in 20/54 (37%) of cases. Patients treated from August 1998 to February 2000 in Phase III and following FDA approval (tube or bifurcated only) had a lower incidence of endoleaks that were diagnosed in only 13/62 (21%) of cases. Only 7 of the last 50 bifurcated cases were noted to have an endoleak. The difference is presumed to be due to better case selection and increasing experience.

B. Spontaneous Resolution

Six patients with the Ancure® and three with the Excluder® device had their endoleaks identified only on the postdeployment intraoperative angiography. The initial postoperative CT or Duplex scan could not identify the leaks. The leaks are presumed to have sealed spontaneously in the interim. Subsequent follow-up has not identified any recurrence.

Spontaneous resolution of endoleaks was very significant by 6 months. Only 9/88 (10%) patients with Ancure® and 1/28 (3%) with Excluder® devices still had a noticeable leak. Follow-up of AAA size over time in patients with an endoleak that sealed spontaneously was similar to those who never had an endoleak. At 24-month follow-up, 80% of patients with endoleak that sealed and 76% of patients with no endoleak had shown a significant reduction in size of at least 5 mm by CT scan.

C. Persistent Endoleaks

The 10 endoleaks still evident at the 6-month follow-up were considered to be persistent. One Excluder® patient is being observed for another 6 months, since the AAA is slightly smaller in size and the technique of coiling cannot be easily applied to a fully externally supported graft. Another patient with an Ancure® graft also had a smaller AAA by 6 months. He was developing congestive heart failure (CHF) and was not treated. He died at 14 months from progressive CHF.

The remaining eight patients with Ancure® devices, and a persistent endoleak at 6 months, underwent angiography and attempted coiling. The technique used has been previously described.[10] In short, the AAA cavity is accessed around the graft and coils deposited in the outflow vessels from the sac. Large coils are next packed in the sac down to the attachment frame. A follow-up CT is obtained at 6 to 8 weeks and if a residual endoleak was detected, the procedure was repeated.

Only one patient had a Type II endoleak with an IMA feeding the sac. The leak was obliterated with coils at the IMA origin through a superior mesenteric artery approach. All seven other patients had a Type I endoleak demonstrated around the distal attachment system: 2 tube and 5 bifurcated (Fig. 1A, B). One patient had a combination endoleak around the proximal and distal attachment sites and was approached around the proximal site (Fig. 2A, B). He required a two-staged treatment of a complex endoleak. Another patient required three treatment sessions before complete obliteration of the leak. The technique was successful in angiographic exclusion of the AAA in all patients except one, in whom access to the sac was hindered by a Wall stent overlying the distal attachment system. This patient was treated successfully at 13 months with a stented graft extension.

CT follow-up of AAA size in the 10 patients with persistent endoleaks showed quite a variable response. Three patients showed a decrease in AAA size, four patients remained stable and three increased in size as long as the endoleak was present. Following successful coiling, all seven patients showed a decrease in AAA size on follow-up by CT scans from 6 to 18 months after obliteration of the leak (Fig. 3A, B). Pressure measurements in the sac were obtained in three patients during the coiling procedure and were near systemic arterial pressures.

D. Endoleaks by Sites

The behavior of different endoleaks was previously reported and will only be summarized here.

Proximal Attachment System

Endoleaks arising at this location were mostly associated with neck angulation and all resolved without intervention (Fig. 4A, B). They were diagnosed usually during completion angiography as a small amount of contrast fills the sac around the proximal portion of the endograft. Ten such endoleaks with the Ancure® and five with the Excluder® were noted to resolve spontaneously. A leak initially classified as distal was also shown to have a proximal component on angiography when it persisted at 6 months. The patient was treated successfully with coils.

Distal Attachment System

Distal endoleaks with the bifurcated Ancure® system were mostly associated with ectatic iliac arteries that were somewhat larger than the limbs.

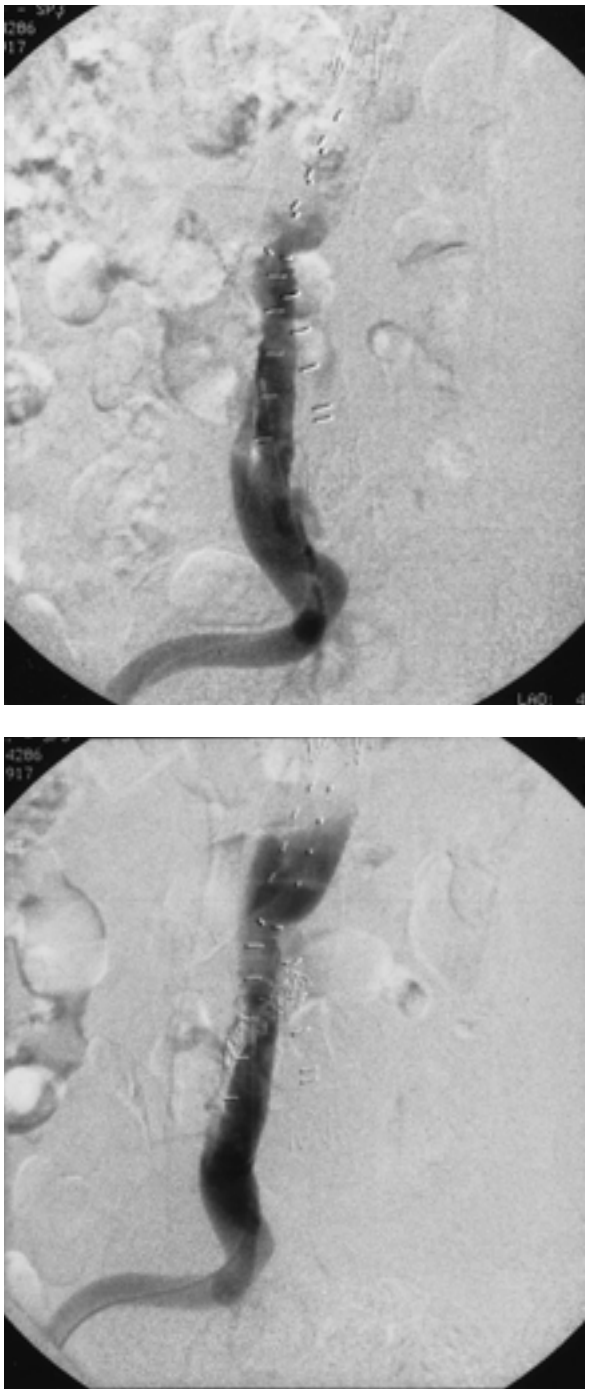

Fig. 1 **(A)** Persistent endoleak noted around the distal iliac attachment of a bifurcated graft. **(B)** Coils have been placed to obliterate the leak in January 1998.

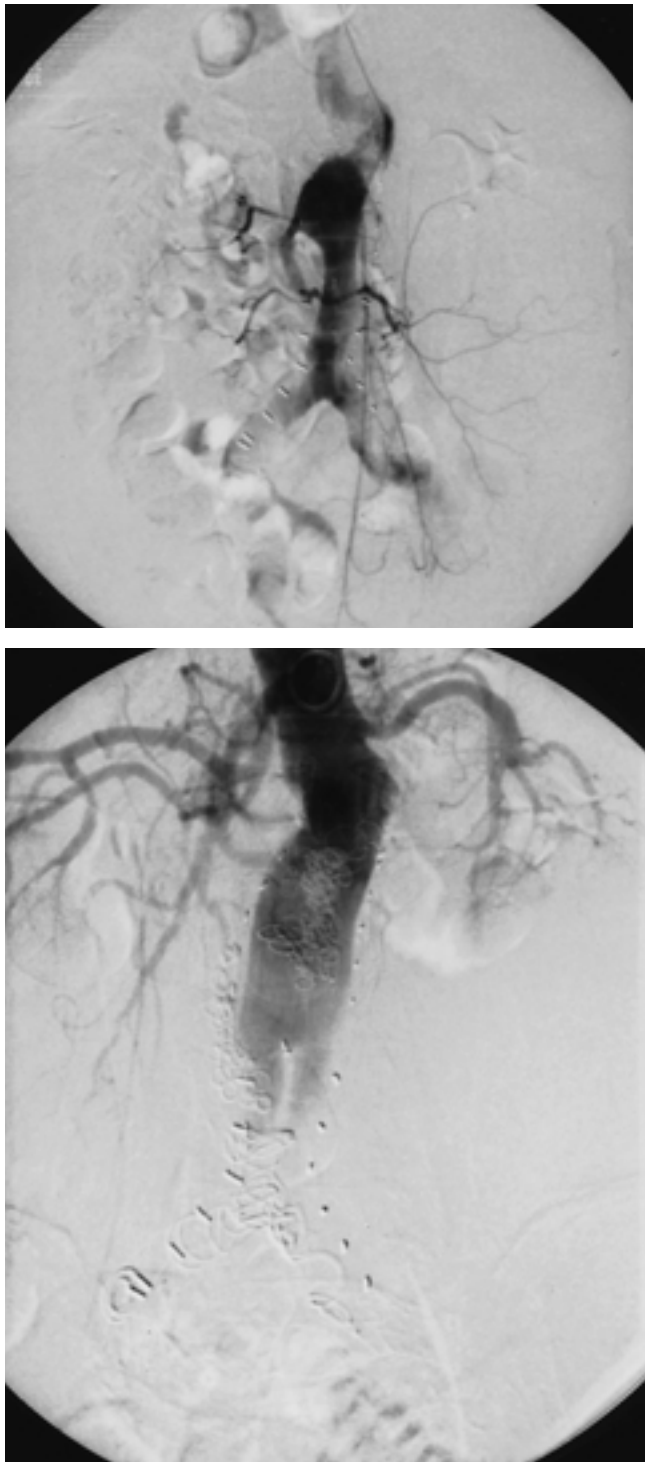

Fig. 2 **(A)** Opacification of a complex cavity through a trans catheter injection around the proximal attachment showing outflow around both distal iliacs and lumbar vessels. **(B)** Leak obliterated with multiple coils. Reprinted with permission.

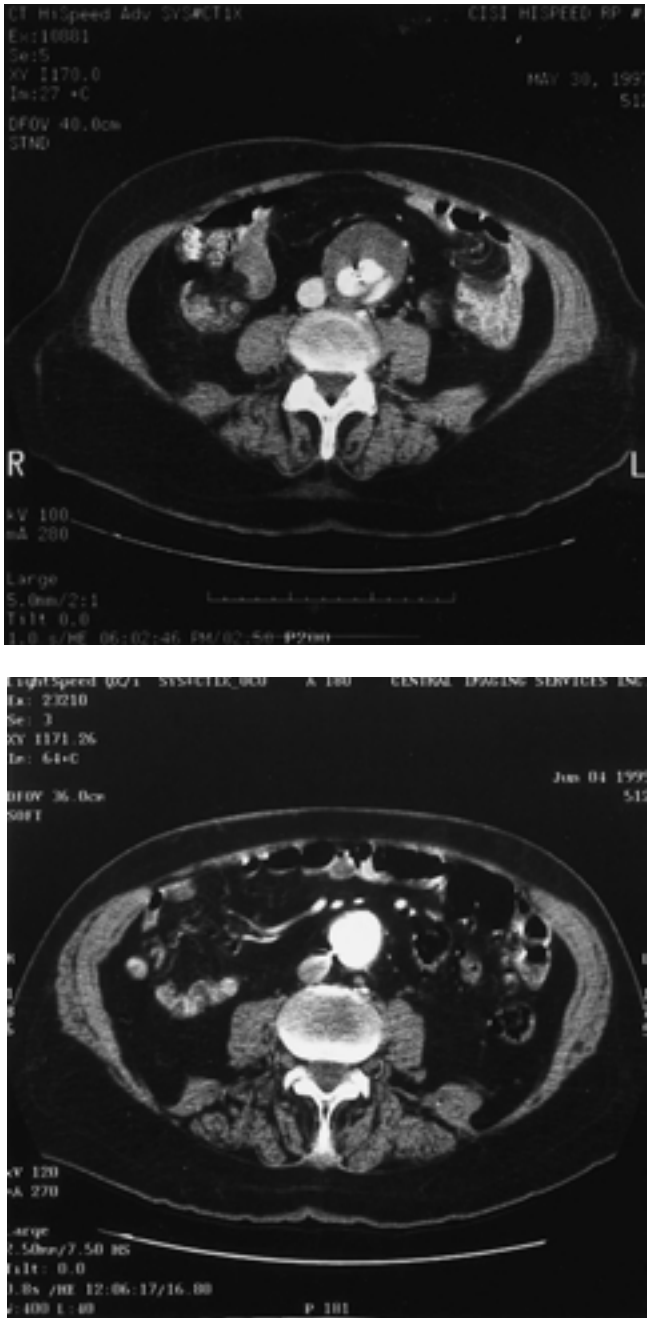

Fig. 3 **(A)** CT scan of patient in Fig. 1 showing endoleak postoperatively in May 1997. **(B)** One year after the coiling in January 1999, the AAA is nearly completely collapsed.

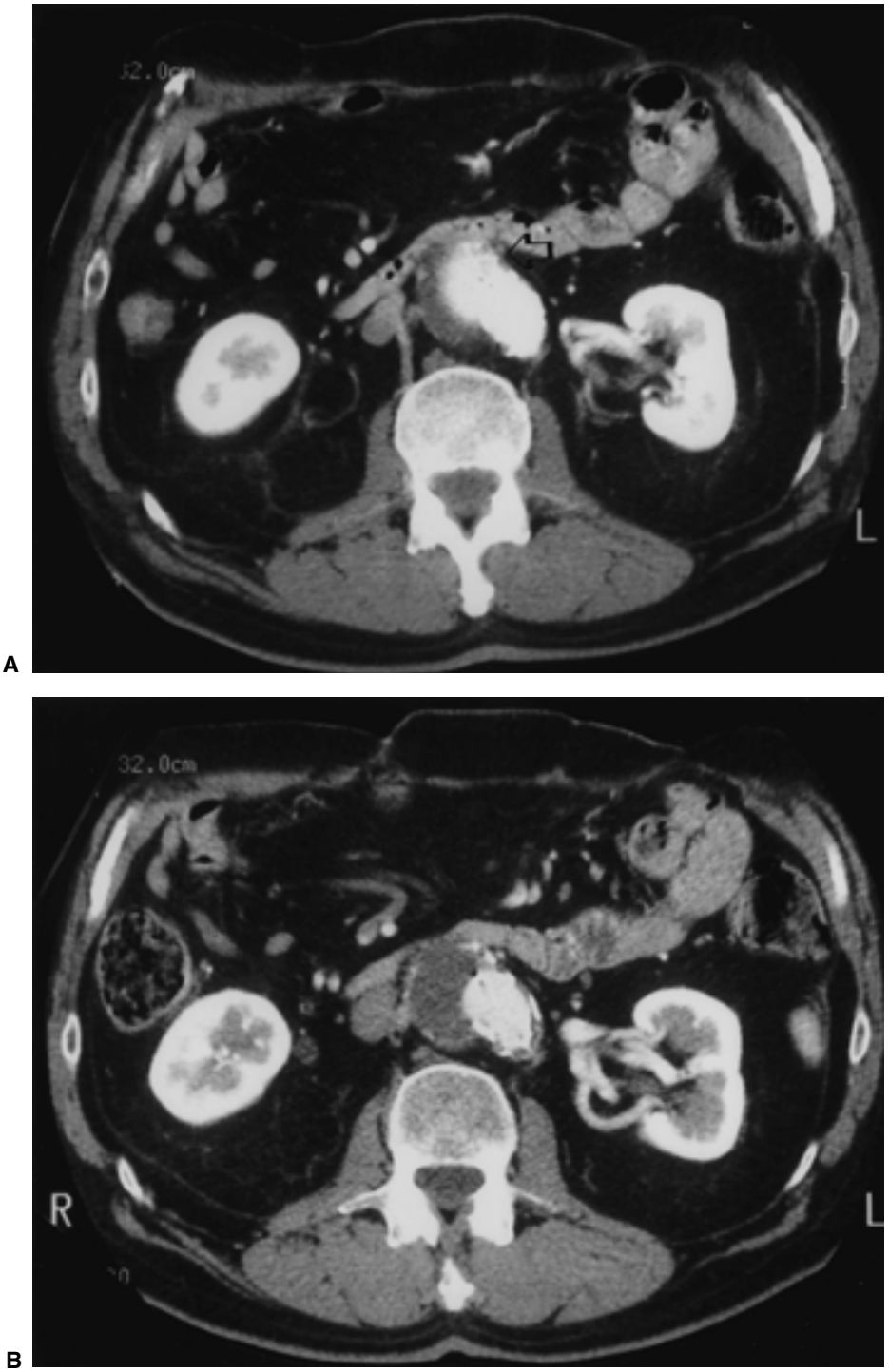

Fig. 4 **(A)** Proximal endoleak associated with angulation of the neck (arrow). **(B)** The endoleak has subsided 3 months later. Reprinted with permission.

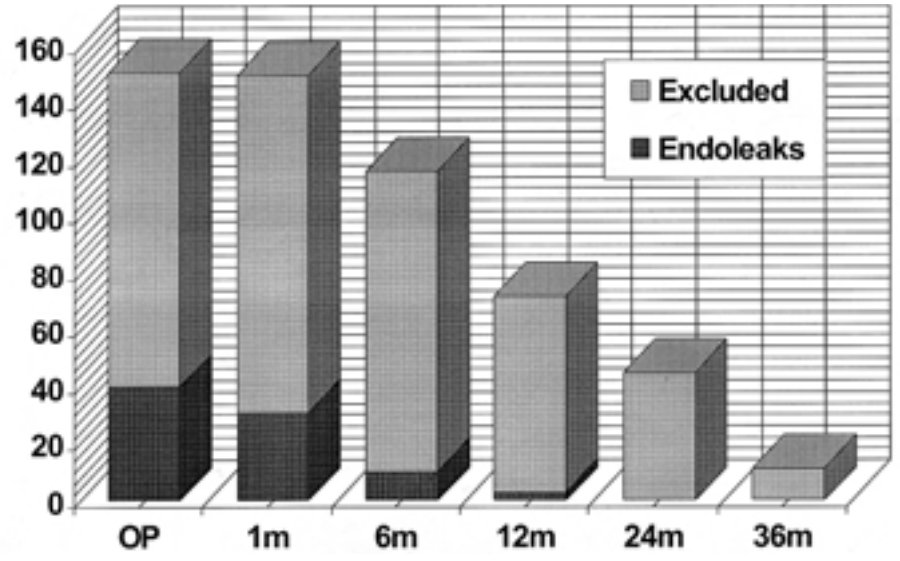

Fig. 5 The rate of endoleaks at different time intervals during follow-up.

They tended to be persistent with six of 10 coiled and one treated with an extension endograft. Only one endoleak stopped spontaneously and two patients died during follow-up in under a year from medical problems. Only one of three distal leaks with a tube graft resolved on its own. The other two leaks were coiled with success. A late endoleak that developed at 2 years with migration of the distal attachment of a tube endograft was treated with an aorto-iliac endograft inside the tube and a fem-fem bypass.

Other Endoleaks

Twelve endoleaks that are either branch flow or indeterminate have mostly subsided spontaneously. One Excluder® remains open at 6 months and two Ancure patients have not had their 6 month follow-up yet. One IMA was coiled.

The fate of endoleaks in our experience, combining spontaneous resolution with treatment of persistent perigraft flow, has been effective in excluding nearly all patients treated by 1 year follow-up (Fig. 5). Of the three patients still exhibiting the problem, two were treated successfully shortly after 1 year and one patient died from CHF.

DISCUSSION

The clinical significance of endoleaks remains under debate. Some patients with a clear endoleak have a shrinking aneurysmal sac while others have an

expanding AAA in the presence of what appears to be good exclusion by the endograft. This has led some to use "endotension" as a more descriptive term of the clinical situation associated with AAA enlargement and the risk of rupture. However, until this can be measured reliably we are left with endoleaks as the most obvious indicator of treatment failure and residual risk of rupture.

Several reports have established endoluminal repair of AAA as a viable alternative to the standard open procedure.[1–7] Endoleaks remain the major drawback and have been reported with all systems used. The etiology is not uniform and neither is their natural history or management. Branch flow backbleeding into the sac is an unavoidable side effect of the technique, unless all branches are embolized preoperatively, a somewhat daunting and potentially undesirable task. Although we attempted to embolize large branches initially, we quickly abandoned it because of additional cost, patient burden and most importantly, ineffectiveness, since our first two embolizations did not prevent the development of endoleaks. It is of note that our review failed to identify a direct relationship between patent branches on the preoperative angiogram and the development of endoleaks. This finding has been documented by others.[11,12]

Other types of endoleaks are associated with particular devices and are related to the attachment systems, modular joint interface or fabric of the device. Our experience with two systems seems to indicate on the surface that the Excluder® has a significantly lower incidence of endoleaks compared to the Ancure®. However, when a similar time frame is used for the comparison, the difference is nonexistent, with both having an incidence around 20% over the last 2 years. This most likely reflects maturation in the case selection process and a better appreciation of the causes of endoleaks. This finding implies that the incidence of endoleaks might be similar between systems and the choice of devices has to be governed by other characteristics. Interestingly enough, the reported rate of endoleaks with the AneuRx® device (Medtronic, Santa Rosa, CA) is also around 20%.[4]

The configuration of the device used seems to influence the endoleak rate with the tube configuration carrying a higher rate than the bifurcated. This fact should alert users to use the tube configuration only in select cases where there is a long and narrow distal neck that may not be suited for the two limbs of a bifurcated device. We presently require 20 mm of distal neck for a tube configuration rather the initial requirement of 12 mm.

The site of origin of the endoleak may be of some predictive value when it comes to prognosis. Most leaks associated with the proximal end of an endograft, unless major in nature due to poor deployment, are usually associated with angulated necks to the flow channel of the AAA exceeding 35 degrees. Contrary to conventional wisdom, these appear to be rather benign and spontaneously resolving in just about all cases. The persistent endoleaks, at least with the Ancure® device, seem to be associated with the distal attach-

ment system deployed in an ectatic iliac segment. Branch flow and indeterminate endoleaks also appear to be benign, resolving in the majority of cases on their own.

Treatment protocols for correcting endoleaks should be in place at all centers engaging in this new treatment modality of AAA. Careful observation for 6 months may be warranted because more than two thirds of the endoleaks will spontaneously resolve without any long-term disadvantage. This has been reported previously by others and confirmed in our review.[13] Observation beyond that point however has to be very carefully evaluated. Patients with a shrinking aneurysmal sac, especially with worsening medical condition, can be closely watched further, with evaluations every 6 months. However, the majority of patients should be treated aggressively to eliminate the source of the endoleak and prevent a potentially disastrous rupture of the AAA.

The persistent endoleaks associated with the Ancure® endograft seem to be particularly suited for the coiling technique. The graft is unsupported and the leaks are generally around the iliac limbs making access to the cavity somewhat simple. This method has been very successful in our hands with a radiographic seal achieved in all but one patient where a wall stent limited access to the cavity. Long term follow-up of these treated endoleaks has so far shown a decrease in the AAA size in all patients. Experimental evaluation of a similar approach for treatment of endoleaks had implied that coiling might not be effective in reducing intraaneurysmal pressures.[14] The model used however a sizable direct rent in the graft with acute pressure measurements that might not be comparable with the clinical situation. Our clinical follow-up of the patients now confirms that this is an effective treatment for the endoleaks associated with the Ancure® endograft. Others also have had success with a similar approach.[15]

Coiling, however, may not be uniformly successful, and is not well suited in fully supported modular grafts. The exoskeleton may hamper the attempts to access the cavity around the graft and other modalities may have to be considered. Stent graft extensions or a new graft in graft may have to be used. We have used each method once. This has been effective in both cases and is the basis of endovascular repairs with most modular systems. Of course, if everything else fails, the standard open procedure can be always used to finally treat the AAA appropriately.

CONCLUSIONS

Endoleaks remain a significant problem of endovascular repair of AAA with all systems available. The incidence, however, seems to be decreasing with experience and better case selection. Most endoleaks resolve spontaneously and persistent cases should be aggressively treated. This problem highlights the necessity of long-term follow-up with this modality of treatment.

ACKNOWLEDGEMENTS

The authors would like to acknowledge the invaluable help of Nita Missig R.N., and Mr. Hiroatsu Sugimoto in collecting the data.

REFERENCES

1. Makaroun M, Zajko A, Orons P, et al. The experience of an academic medical center with endovascular treatment of abdominal aortic aneurysms. Am J Surg 1998;176:198–202
2. May J, White GH, Yu W, et al. Concurrent comparison of endoluminal versus open repair in the treatment of abdominal aortic aneurysms: Analysis of 303 patients by life table method. J Vasc Surg 1998;27:213–221
3. Blum U, Voshage G, Lammer J, et al. Endoluminal stent-grafts for infrarenal abdominal aortic aneurysms. NEJM 1997;336:13–20
4. Zarins CK, White RA, Schwarten D, et al. AneuRx stent graft versus open surgical repair of abdominal aortic aneurysms: Multicenter prospective clinical trial. J Vasc Surg 1999;29:292–305
5. White GH, Yu W, May J. "Endoleak": A proposed new terminology to describe incomplete aneurysm exclusion by an endoluminal graft. J Endovasc Surg 1996;3:124–125
6. Wain RA, Marin ML, Ohki T, et al. Endoleaks after endovascular graft treatment of aortic aneurysms: Classification, risk factors and outcome. J Vasc Surg 1998;27:69–80
7. Resch T, Ivancev K, Lindh M, et al. Persistent collateral perfusion of abdominal aortic aneurysm after endovascular repair does not lead to progressive change in aneurysm diameter. J Vasc Surg 1998;28:242–249
8. May J, White GH, Yu W, et al. Endovascular grafting for abdominal aortic aneurysms: Changing incidence and indication for conversion to open operation. Cardiovasc Surg 1998;6:194–197
9. Makaroun M, Zajko A, Sugimoto H, et al. Fate of endoleaks after endoluminal repair of abdominal aortic aneurysms with the EVT device. Eur J Vasc Endovasc Surg 1999;18: 185–190
10. Amesur N, Zajko A, Makaroun M. Embolotherapy of persistent endoleaks after endovascular repair of abdominal aortic aneurysm with the Ancure-Endovascular Technologies endograft system. J Vasc Interv Rad 1999;10:1175–1182
11. Walker SR, Halliday K, Yusuf SW, et al. A study on the patency of the inferior mesenteric artery and lumbar arteries in the incidence of endoleak following endovascular repair of infra-renal aortic aneurysms. Clin Rad 1998;53:593–595
12. Broeders IA, Blankensteijn JD, Eikelboom BC. The role of infrarenal aortic side branches in the pathogenesis of endoleaks after endovascular aneurysm repair. Eur J Vasc Endovasc Surg 1998;16:419–426
13. Matsumura JS, Pearce WH, McCarthy WJ. Reduction in aortic aneurysm size: Early results after endovascular graft placement. J Vasc Surg 1997;25:113–123
14. Marty B, Sanchez L, Ohki T. Endoleak after endovascular graft repair of experimental aortic aneurysms: Does coil embolization with angiographic "seal" lower intraaneurysmal pressure? J Vasc Surg 1998;27:454–462
15. Golzarian J, Struyven J, Abada HT, et al. Endovascular aortic stent-grafts: Transcatheter embolization of persistent perigraft leaks. Rad 1997;202:731–734

Expert Commentary

Takao Ohki, M.D.
Frank J. Veith, M.D.

Drs. Makaroun and Zajko, who are pioneers in the field of endovascular repair of abdominal aortic aneurysms (AAAs), have nicely described the significance of and treatment options for endoleaks following endovascular graft (EVG) repair of AAAs. Endoleaks have been considered the "Achilles' heel" of EVG repair and the clinical significance as well as treatment modalities are yet to be defined. This article has clarified several important points and has made it possible for us to better understand this problem. Two endovascular grafts have recently been approved for general use by the FDA, and, since there is no standard treatment protocol for the management of endoleaks, this article is a very timely and valuable one.

The authors have reported the early and mid-term results of endovascular repair of AAAs in 158 patients treated and followed over a 4-year period. The technical success rate was 97% with an operative mortality rate of 0.6%. These excellent results, however, cannot be used to support the routine use or abuse of EVGs. One must realize that these results were possible due to (1) the authors' extensive experience with EVG repair, and (2) careful patient selection adhering to the study protocol or the instructions for use. One should not assume that these results can be reproduced at hospitals that are inexperienced with EVG repair or if the EVG is used to treat patients who have unfavorable anatomic characteristics.

We cannot agree more with the various points that the authors have made. These include the following: (1) For most endoleaks, an observation policy for 6 months prior to performing some form of interventional treatment, except in unusual circumstances; (2) the fact that the presence or absence of patent side branches (IMA or lumbars) was not predictive of postoperative endoleaks; (3) bifurcated grafts should preferentially be used over tube or aortouni-iliac grafts; (4) the vast majority of type 2 endoleaks seal spontaneously and are benign in nature; (5) not to forget about an open surgical repair as a treatment option, if endovascular methods to treat an endoleak fail.

Although we generally agree with the authors' messages, we were surprised that most proximal type 1 endoleaks resolved spontaneously and were accompanied by shrinkage of the aneurysm sac. We and others have long believed that regardless of whether a type 1 endoleak seals or not, it needs to be treated with either placement of a covered stent (extension cuff) or surgically. In Dr. Makaroun's and Dr. Zajko's experience, most if not all type 1

T.O., Assistant Professor and Chief of Endovascular Programs; F.J.V., Professor and Chief, Vascular Surgery Services, Department of Surgery, Division of Vascular Surgery, Montefiore Medical Center, Bronx, NY.
 0894-8046,p;2000,13,1,15,18,ftx,en;pvs000078

endoleaks resolved spontaneously with shrinkage of the aneurysm sac. Not all type 1 endoleaks are equal, and one has to assume that the endoleaks the authors are describing are minor proximal endoleaks that accompany an EVG that is placed with precision in a good quality proximal neck, as opposed to large proximal endoleaks secondary to a malpositioned EVG in poor quality necks.[1] Although we are not in a position to challenge the authors' observation, one must be careful in interpreting their finding. In addition, one should not use their observation to raise the threshold during the initial operation for taking immediate action to seal the proximal endoleak with either placement of a proximal cuff or by using a balloon-expandable stent to better appose the EVG to the neck. The best time to address a proximal endoleak is when one is performing the initial procedure, and every effort should be made to correct it before the patient leaves the operating room. It is also clear that not all spontaneously sealed endoleaks are benign. Some large type 1 endoleaks will continue to pressurize the aneurysm, despite the lack of CT or angiographic visualization of a leak. In the future, it may be useful to develop a subclassification of type 1 endoleaks so that one can differentiate between those that need additional intervention or surgical treatment despite spontaneous sealing, and those that can be considered resolved. Similarly, not all endoleaks can be treated with coil embolization, and some will need more secure mechanical fixation. Better understanding of the nature of endoleaks may help guide the optimal methods of treatment.[1]

The fact that proximal endoleaks occurred in 50% of patients with proximal neck angulations greater than 35 degrees, irrespective of the EVG used, is alarming. This underscores the need for a better EVG with the ability to accommodate a proximal neck angulation. For this reason, we have been using the Montefiore Endovascular Graft System (MEGS) for aneurysms with severe angulation. Among the 50 cases recently treated with the MEGS device, we have encountered only 1 case (2%) of a type 1 endoleak, despite the fact that it was used for complex AAAs that were not suitable for any of the 6 other industry-made EVGs that are available at our institution. The reason for this low endoleak rate is related to the fact that the MEGS utilizes an extra large Palmaz stent for proximal fixation. This stent exerts the strongest radial force among all the EVGs, and therefore it is better able to handle angulated necks.

During the authors' follow-up period ranging from 2–48 months, there has been no late rupture or conversion to open repair, despite the fact that 41 of the 153 aneurysms (27%) had some type of endoleak. The authors are to be commended for achieving such an excellent result. However, one must be careful in interpreting this observation. It is our concern that this kind of observation suggests that endoleaks were "no big deal." One should not get the wrong message. Their excellent results were achieved because the authors employed a strict follow-up protocol and aggressively treated all endoleaks that were persistent or those that resulted in enlargement of the AAA.

The bottom lines of this paper are: (1) patient selection and fine surgical/endovascular techniques are important; (2) strict, lifelong follow-up is essential; (3) an aggressive treatment policy should be employed for persistent endoleaks; (4) with all of the above, excellent results can be achieved.

REFERENCES

1. Mehta M, Ohki T, Veith FJ, et al. Are all endoleaks equal? Eur J Vasc Endovasc Surg 2000 (in press).

Expert Commentary

Jon Matsumura, M.D.

Drs. Makaroun and Zajko have reviewed their four-year experience with 158 patients undergoing endovascular treatment for abdominal aortic aneurysm. In this paper they specifically address the frequency of endoleaks and subsequent clinical course and reintervention. Their results are excellent with low perioperative mortality and only three early conversions and two aborted procedures in the entire group. There were no late conversions or known aneurysm ruptures. These results are impressive and reflect upon a combination of excellent medical judgment and technical abilities. Specifically, the authors remark on their 15 mm selection threshold for proximal neck length. In addition, the authors have adopted a routine for management of postoperative endoleaks, which is to reintervene at six months for any persistent endoleak at that time point. The authors present data that most endoleaks resolved by the six-month point, and, of the ten that did not, they were successful with reinterventions in eight of the patients. The other two patients were observed, one because the aneurysm was smaller in size and coiling could be more difficult, and the second because the patient was dying of congestive heart failure.

The authors state that the aortic aneurysm size in the ten patients with persistent leaks had a variable response with three showing a decrease, four remaining stable, and three increasing in size. This is an important observation because others have suggested that presence of a persistent endoleak is strongly correlated with continued aneurysm expansion.[1–4] Some differences in findings may be attributable to inadequate statistical power to detect the correlation. Depending on the methods used to measure aneurysm diameters or volumes, inter- and intra-observer variability increases the statistical sample necessary to detect real causal relationships. I believe that some of the discrepancy between literature reports also is related to our inability to detect and define all endoleaks. Specifically, some patients may have a Type 1 endoleak, which is diagnosed as another type of endoleak, and vice versa. Calcified thrombus may be mistaken for endoleak if pre-contrast CT scans are not performed. Other patients may have a slow flowing endoleak that is missed on postoperative imaging. Last, some studies have applied selective reintervention for persistent endoleaks or endoleaks which are associated with aneurysm growth and, therefore, the natural history of unselected endoleaks and aneurysm size change is altered.

Another important observation of the authors is the finding that in the three patients who underwent pressure measurements during a reinterven-

J.M., Assistant Professor of Surgery, Division of Vascular Surgery, Northwestern Memorial Hospital, Chicago, IL.

Tel. +1 (212) 584-4662. 0894-8046,p;2000,13,1,19,20,ftx,en;pvs000079

tion for coiling, sac pressures were found to be systemic. It would be interesting to know what the sac pressures were like in the three patients whose aneurysms were shrinking during the six-month interval. If they are systemic, then the usually reassuring finding of aneurysmal sac decrease loses much of its luster. This observation suggests that we have a lot to learn about which endoleaks need reintervention and which should be safely monitored.

In the future, randomized controlled studies need to compare surveillance strategies and reintervention strategies to determine the optimal program for follow-up and subsequent therapy. Until then, I agree with the authors that treatment protocols must be in place at all centers engaging in endovascular grafting. Drs. Makaroun and Zajko have drawn upon their extensive experience to provide us with a treatment protocol that is an excellent choice for a treatment arm in these future controlled studies.

REFERENCES

1. Malina M, Ivancev K, Chuter TAM, et al. Changing aneurysmal morphology after endovascular grafting: Relation to leakage or persistent perfusion. J Endovasc Surg 1997;4:23–30
2. May J, White GH, Yu W, Waugh RC, Stephen MS, Harris JP. Prospective study of anatomic and pathological changes in abdominal aortic aneurysms following endoluminal repair: Is the aneurysmal process reversed? Eur J Vasc Endovasc Surg 1996;11:1–7
3. Broeders IAMJ, Blankensteijn JD, Gvakharia A, et al. The efficacy of transfemoral endovascular aneurysm management: A study on size changes of the abdominal aorta during mid-term follow-up. Eur J Vasc Endovasc Surg 1997;14:84–90
4. Matsumura JS, Moore WS, EVT Investigators. Clinical consequences of periprosthetic leak following endovascular repair of abdominal aortic aneurysms. J Vasc Surg 1998;27:606–613

The Last Word

Michel Makaroun, M.D.
Albert Zajko, M.D.

We agree wholeheartedly with essentially all the comments of Drs. Ohki, Veith, and Matsumura. Their extensive experience is evident in their commentaries. Drs. Ohki and Veith appropriately point out that not all endovascular aneurysm treatment options are equivalent, and care should be exercised in comparing data. They are very perceptive in noting that our results were obtained with "good risk" patients, physiologically and, most importantly, anatomically, since most were enrolled in tightly controlled clinical trials. Dr. Matsumura wisely points out that not all endoleaks are equal and much remains to be learned.

Two points are worthy of some emphasis. First, not all graft systems are equivalent, and subsequently not all type I endoleaks are the same. We could not agree more with Drs. Ohki and Veith that generalizations should be avoided. Most of our experience is with the Ancure system that provides an active fixation to the aortic wall with all patients preselected with appropriate proximal necks, although some were very angulated. A faint endoleak at completion angiography at that level in this clinical setting has proven to be very benign, but this should not be misconstrued to mean that proximal Type I endoleaks are benign in all settings and are to be ignored. We believe our peculiar finding is due to the totally unsupported body of the Ancure graft distal to the fixation systerm allowing the graft to realign with the aortic flow, sometimes sealing the endoleak within 24 hours. Results with the Montefiore endovascular grafting system (MEGS) are probably due to similar mechanics. Fully supported systems do not remodel with the aortic flow and may have more significant problems when a proximal endoleak exists.

Treatment protocols for endoleaks are essential and should be a part of any endovascular program as suggested by both commentaries. Drs. Ohki and Veith correctly point out that our late results are excellent because of an aggressive approach to endoleak management. We agree. Treatment protocols, however, should also be sytem specific. Our coiling technique works well with the Ancure system, as we have documented with long-term follow-up, but may have to be modified with other systems. The metallic scaffolding of fully supported grafts may interfere with the approach to the cavity of the sac that we have used and endoleak source and response may be different.

We also strongly agree with the message of Drs. Ohki and Veith that endoleaks are "a big deal." Any suggestion to the opposite ignores all the lessons we have learned so far about aneurysm exclusion. However, we

should also find a way to categorize and understand endoleaks better, as Dr. Matsumura suggests. Endoleaks with different systems behave differently. Tension exerted on the wall is variable. The classification system we are familiar with is excellent in neatly separating sources of endoleaks. Unfortunately, the means of identifying that source on CT or ultrasound leaves a lot to be desired. Many of our persistent endoleaks documented by angiography to be Type I around the distal limbs were called Type II by independent observers reviewing CT and ultrasound exams.

We have greatly increased our knowledge since the early pioneering work of Dr. Parodi, but we have to recognize that even more remains to be learned.