Endoleaks after endovascular graft treatment of aortic aneurysms: Classification, risk factors, and outcome

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Purpose: **Incomplete endovascular graft exclusion of an abdominal aortic aneurysm results in an endoleak. To better understand the pathogenesis, significance, and fate of endoleaks, we analyzed our experience with endovascular aneurysm repair.**

Methods: **Between November 1992 and May 1997, 47 aneurysms were treated. In a phase I study, patients received either an endovascular aortoaortic graft (11) or an aortoiliac, femorofemoral graft (8). In phase II, procedures and grafts were modified to include aortofemoral, femorofemoral grafts (28) that were inserted with juxtarenal proximal stents, sutured endovascular distal anastomoses within the femoral artery, and hypogastric artery coil embolization. Endoleaks were detected by arteriogram, computed tomographic scan, or duplex ultrasound. Classification systems to describe anatomic, chronologic, and physiologic endoleak features were developed, and aortic characteristics were correlated with endoleak incidence.**

Results: **Endoleaks were discovered in 11 phase I patients (58%) and only six phase II patients (21%; $p < 0.05$). Aneurysm neck lengths 2 cm or less increased the incidence of endoleaks ($p < 0.05$). Although not significant, aneurysms with patent side branches or severe neck calcification had a higher rate of endoleaks than those without these features (47% vs 29% and 57% vs 33%, respectively), and patients with iliac artery occlusive disease had a lower rate of endoleaks than those without occlusive disease (18% vs 42%). Endoleak classifications revealed that most endoleaks were immediate, without outflow, and persistent (71% each), proximal (59%), and had aortic inflow (88%). One patient with a persistent endoleak had aneurysm rupture and died.**

Conclusions: **Endoleaks complicate a significant number of endovascular abdominal aortic aneurysm repairs and may permit aneurysm growth and rupture. The type of graft used, the technique of graft insertion, and aortic anatomic features all affect the rate of endoleaks. Anatomic, chronologic, and physiologic classifications can facilitate endoleak reporting and improve understanding of their pathogenesis, significance, and fate. (J Vasc Surg 1998;27:69-80.)**

Endovascular grafts are being scrutinized as possible alternatives to conventional grafts for the treatment of abdominal aortic aneurysms.[1-14] Incomplete exclusion or endoleaks are among the most common complications of endovascular aneurysm repair and result in ongoing perfusion of the aneurysm sac. Endoleaks usually indicate procedural failure and when untreated may cause aneurysm rupture. To better understand the uncertain pathogenesis, fate, and significance of

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Table I. Endovascular graft and technique-related differences between phase I and phase II

	Phase I			*Phase II*
Graft type	Aortoaortic (tube)	EVT (tube)	Aortoiliac, femorofemoral	Aortofemoral, femorofemoral
Graft material	Knitted Dacron	Woven Dacron	PTFE	PTFE
No. of patients	7	4	8	28
Proximal stent placement	Infrarenal aorta	Infrarenal aorta	Infrarenal aorta	Juxtarenal aorta
Distal graft placement	Distal aorta	Distal aorta	Iliac artery	Femoral artery
Method of distal graft fixation	Stent	Stent	Stent	Sutured endoluminal anastomosis
Preoperative hypogastric artery coil embolization	Not applicable	Not applicable	No	Yes

PTFE, Polytetrafluoroethylene.

endoleaks, we reviewed our experience with endovascular abdominal aortic aneurysm repair. We identified graft-, patient-, and technique-related factors associated with endoleaks and developed anatomic, chronologic, and physiologic classification systems to facilitate their reporting and analysis.

MATERIALS AND METHODS

Patients. All patients who had abdominal aortic aneurysms larger than 5 cm diagnosed between November 1992 and May 1997 were eligible for endovascular repair. High-risk patients with severe cardiopulmonary disease, a hostile abdomen, or other major coexisting medical problems were offered treatment with Parodi-type balloon-expandable endovascular grafts. Good-risk patients were asked to participate in the phase I North American trial of the Endovascular Technologies (EVT) endovascular graft[7] (Menlo Park, Calif.). Patients who did not meet the inclusion criteria for these studies or who refused to participate were treated with standard open surgery or were observed.

Endovascular procedures. A detailed description of the endovascular grafts used in this study has been reported elsewhere.[5] Briefly, the EVT grafts were constructed from self-expanding hooked "Z" stents and woven Dacron prosthetic tubes. The Parodi-type devices were originally constructed from thin-walled knitted Dacron grafts (Barone, Inc., Buenos Aires, Argentina) and more recently from 8 mm polytetrafluoroethylene grafts (Impra, Inc., Tempe, Ariz.) predilated proximally to 30 mm. These grafts were sutured to Palmaz balloon-expandable aortic stents with four sutures (P5014 Johnson and Johnson Interventional Systems, Warren, N.J.) so that one-half the length of each stent was overlapped by graft material.

In the first phase of our study, high-risk patients who had proximal and distal aneurysm neck lengths 1.5 cm or greater were treated with endovascular aortoaortic tube grafts. Patients who had distal neck lengths less than 1.5 cm or common iliac artery involvement were treated with aortoiliac endovascular grafts and femorofemoral bypass grafts with contralateral common iliac artery occluder devices. These grafts were anchored proximally by balloon-expandable stents placed beneath the renal arteries. The grafts were anchored distally in the aorta, common, or external iliac artery with a second stent. Good-risk patients treated under the EVT protocol also had their grafts anchored by two stents; one deployed beneath the renal arteries and one proximal to the aortic bifurcation.

In phase II, all of the patients were treated with tapered aortofemoral endovascular grafts and femorofemoral bypass grafts with occlusion of the opposite common iliac artery. The proximal stent of these grafts was deployed near or across the orifice of one or both renal arteries so that the proximal end of the prosthetic graft was affixed just inferior to the orifice of the lowest renal artery. The graft ended distally in the common femoral artery with a sutured endoluminal anastomosis. At the time of surgery, these patients had already undergone coil embolization of the intended ipsilateral hypogastric artery on completion of the preoperative angiogram (Table I).

Devices were inserted under Institutional Review Board supervision and in phase II under an investigator-sponsored investigational device exemption (IDE) from the Food and Drug Administration.

Patient follow-up. Completion angiograms were obtained in the operating room to discover endoleaks. Spiral computed tomographic (CT) scans were performed within the first postoperative week unless the patient had renal insufficiency or another contraindication to the early readministration of intravenous contrast material. Color flow duplex studies were also obtained within a week after graft insertion. Follow-up CT scans and duplex studies were obtained at 3 months, 6 months, and yearly

thereafter. The presence of perigraft, intrasaccular contrast material on a CT scan or angiogram or flow in this location on a duplex study indicated the presence of an endoleak.

Data collection and analysis. Aneurysm characteristics, including maximum diameter, neck length, and degree of calcification; presence of patent side branches (e.g., lumbar, hypogastric, or inferior mesenteric arteries); and association with iliac aneurysms or occlusive disease were assessed before operation with spiral CT scans and arteriography. CT scan images were acquired using 3-mm-thick transverse cuts obtained at 6 mm intervals. The degree of aneurysm neck calcification was recorded as the number of aortic quadrants (0 to 4) that contained calcification on the first transverse noncontrast CT image below the lowest renal artery. Patients were divided into two groups on the basis of the degree of neck calcification: those with severe (four quadrant) calcification and those with less-extensive involvement. Aneurysm neck length was the distance measured on CT scan between the most inferior renal artery and the start of the aneurysm. Extent of aneurysm involvement was graded according to previously described reporting standards for infrarenal endovascular abdominal aortic aneurysm repair.[15] Data were analyzed on a personal computer using Microsoft Excel v. 5.0 and Fisher's exact test.

RESULTS

Forty-six patients underwent 47 endovascular procedures to treat their aneurysms. The average age of the patients was 77.6 years, 42 were men, and the mean preoperative aneurysm sac diameter was 6.4 cm. Nineteen patients (41%) were treated in phase I of the trial. These patients received Parodi-type aortoaortic (7) or aortoiliac, femorofemoral grafts (8), or an EVT graft (4). In phase II all 28 patients received aortofemoral, femorofemoral grafts. Patent aneurysm side branches were observed in 19 patients. Eleven patients (24%) had iliac occlusive disease, 16 (35%) had iliac aneurysmal disease, 28 (61%) had a proximal neck length 2.0 cm or less, and seven (15%) had severe neck calcification. The percentage of patients with complex (grade IV) aneurysms was similar in the first and second phases of the study (16% and 14%, respectively).

A total of 17 aneurysm repairs (36%) were complicated by endoleaks. These were discovered in 11 patients treated in phase I of the study (58%) and in only six patients treated in phase II (21%). Of the phase I patients, endoleaks complicated four aortoaortic grafts (57%), four aortoiliac, femorofemoral grafts (50%), and three EVT grafts (75%). All of the patients who had grade IV aneurysms treated in phase I of the study had endoleaks, compared with only 50% of those treated in phase II.

Aortic characteristics and endoleak incidence. Aneurysms with neck lengths 2 cm or less experienced a significantly higher rate of endoleaks than those that had longer necks (50% vs 16%; $p < 0.05$). The rate of endoleaks was also increased, but not in a statistically significant fashion in patients with patent aneurysm side branches and severe aneurysm neck calcification compared with that in patients who did not have these features (47% vs 29%, $p = 0.10$ and 57% vs 33%, $p = 0.15$, respectively). Patients with iliac occlusive disease had fewer (but not significantly fewer) endoleaks than those without occlusive disease (42% vs 18%; $p = 0.11$), and aneurysm sac diameter did not significantly influence the rate of endoleaks.

Endoleak classification. A three-part classification system was developed to describe aortic aneurysm endoleaks. An *anatomic* classification describes the site of origin and outflow status of the endoleak. Endoleaks can originate from the proximal, distal, or midgraft segment of an endovascular device or from the contralateral iliac artery occluder (Fig. 1). Midgraft endoleaks can begin at an arterial side branch, such as the inferior mesenteric, hypogastric, lumbar, or accessory renal artery, at a graft defect, or between components of a modular device. Outflow is present when an endoleak exits the aneurysm sac (Fig. 2). Outflow is absent when an endoleak terminates as a "pseudoaneurysm" within the aneurysm sac (Table II).

A *chronologic* classification stratifies endoleaks according to their time of onset and fate. Endoleaks are immediate when they are discovered on the first postoperative imaging study; delayed when they are not present on the initial study but are visualized on a subsequent study; or recurrent when they are initially seen but seal, only to "reopen" during follow-up (Fig. 3). Fate describes the history of an endoleak: an endoleak can be persistent throughout the follow-up period; seal spontaneously; or seal with additional intervention (Table III).

A *physiologic* classification describes endoleaks according to their source of inflow. Endoleaks can originate directly from the aorta or indirectly via collateral channels (Table IV).

Management of endoleaks. An attempt was made to treat all endoleaks unless the patient's medical condition or the technical feasibility of a sec-

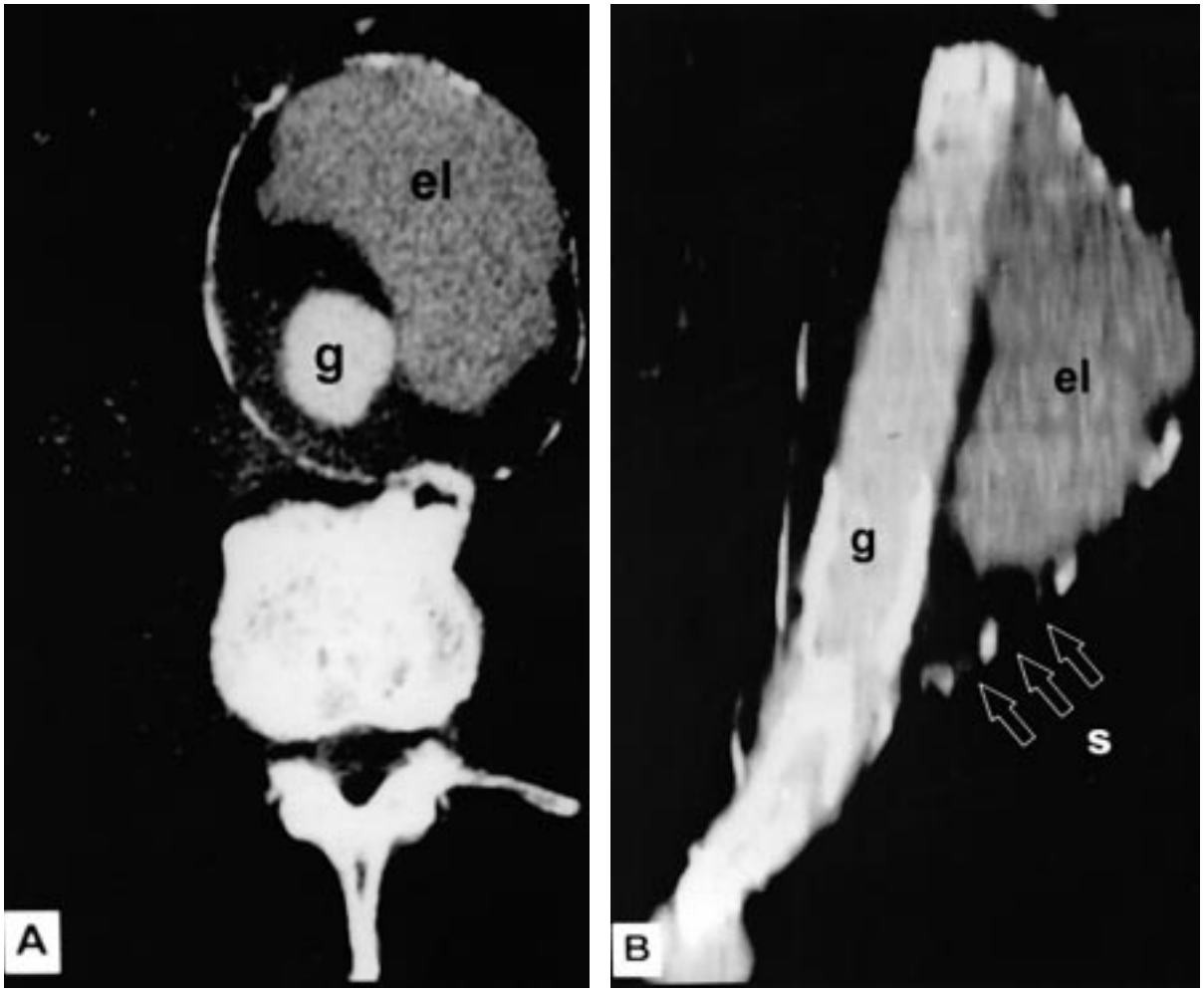

Fig. 1. An 88-year-old man underwent elective repair of an 8 cm abdominal aortic aneurysm with an aortoiliac, femorofemoral graft. **A,** Postoperative CT scan demonstrates large endoleak *(el)* adjacent to endovascular graft *(g)*. **B,** Two-dimensional CT reconstruction shows that endoleak *(el)* originates at proximal portion of endovascular graft *(g)* and ends within distal aneurysm sac *(s)*.

ondary procedure precluded treatment. Three patients underwent additional procedures to seal their endoleaks. One patient had undergone successful implantation of an EVT tube graft that developed a delayed endoleak as a result of a hook fracture within its attachment system. When the aneurysm became pulsatile, the patient's endoleak was sealed by an aortofemoral, femorofemoral graft deployed within the lumen of the EVT graft (Fig. 4). A second patient experienced an endoleak 5 months after placement of an aortoaortic tube graft. A postoperative CT scan demonstrated perigraft, intrasaccular contrast material that we thought was caused by implantation of the distal aspect of the graft within aortic thrombus. This patient underwent an open sutured extension to the aortic bifurcation to repair the endoleak. The final patient experienced an immediate occluder device endoleak after treatment with an aortofemoral, femorofemoral graft. He had multiple coils angiographically placed within the occluder device on the first postoperative day, which resulted in closure of the endoleak (Fig. 5).

DISCUSSION

The successful treatment of an aneurysm depends on its complete exclusion from the arterial circulation.

Table II. Anatomic classification of endoleaks

Endoleak feature	*No.*	*(%)*
Site		
Proximal end of graft	10	(59)
Distal end of graft	3	(17)
Midgraft	2	(12)
Arterial side branch*	2	(12)
Graft defect	0	(0)
Between modular device components	0	(0)
Occluder device	2	(12)
Outflow		
Present†	5	(29)
Absent	12	(71)

*One of the side branch endoleaks originated at the hypogastric artery and the other from a lumbar artery.
†Of those endoleaks with outflow, the vessel involved was a lumbar artery in three patients and a hypogastric artery in two.

Table III. Chronologic classification of endoleaks

Endoleak feature	*No.*	*(%)*
Time of onset		
Immediate	12	(71)
Delayed*	4	(24)
Recurrent†	1	(5)
Fate		
Persistent‡	12	(71)
Sealed	5	(29)
Spontaneously§	2	(12)
With intervention	3	(17)

*One medically ill patient with a delayed endoleak died of a ruptured aneurysm 18 months after his repair.
†Patient had an immediate endoleak that sealed spontaneously and then recurred.
‡Of the 12 persistent endoleaks, four had evidence of outflow and eight did not.
§ One endoleak that sealed spontaneously did so 1 month after surgery and was a proximal endoleak without outflow. The other was a midgraft endoleak with outflow that sealed 2 months after surgery.

Incomplete fixation of an endovascular graft may result in persistent flow within the aneurysm sac. This flow, which may also arise from patent inferior mesenteric, lumbar, hypogastric, or accessory renal arteries, is called an endoleak.[16] Endoleaks have been reported to complicate between 8% and 44% of all endovascular abdominal aortic aneurysm repairs. To date, at least seven patients who had endoleaks after these procedures have had aneurysm rupture.[3,12,17,18] These cases, as well as experimental studies and data on patients whose aneurysms were treated with nonresective procedures, suggest that endoleaks can cause aneurysm rupture.

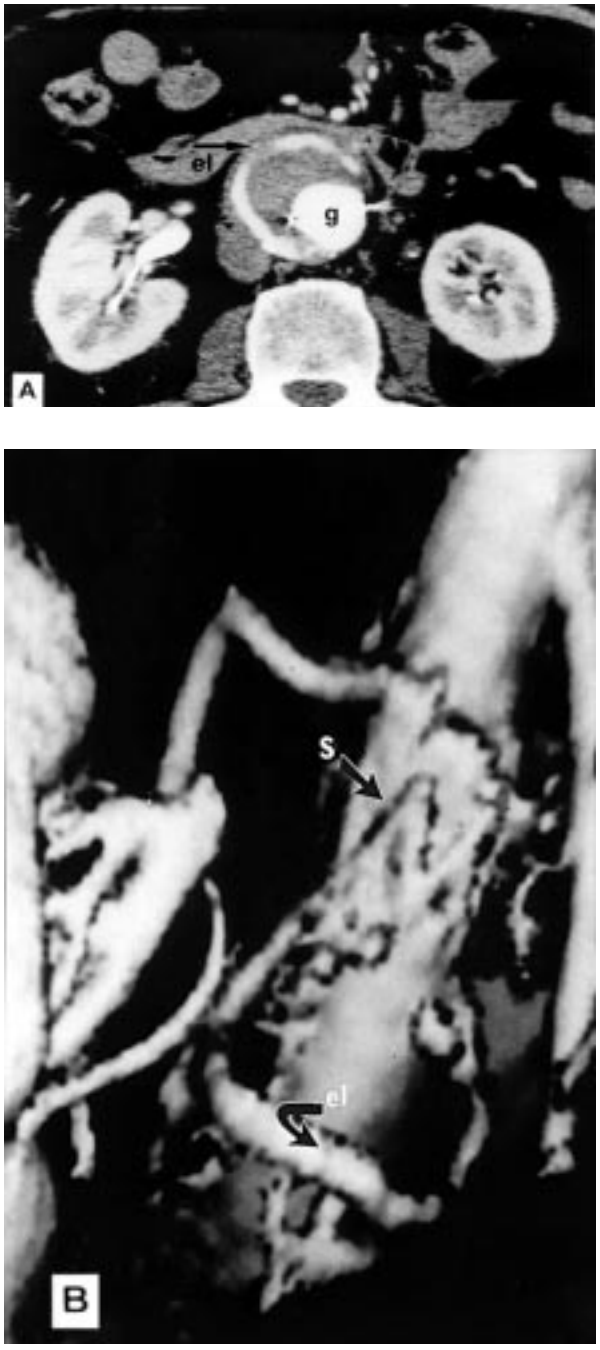

Fig. 2. Patient with endoleak after placement of EVT tube graft. **A,** Transverse CT scan image shows that endoleak *(el)* takes on C-shaped configuration within aneurysm sac. Endovascular graft *(g)* is also shown. **B,** Three-dimensional reconstruction shows zig-zag configuration of proximal stent *(s)* and endoleak *(el)* originating adjacent to stent. Endoleak travels around aneurysm sac and has outflow through a patent lumbar vessel.

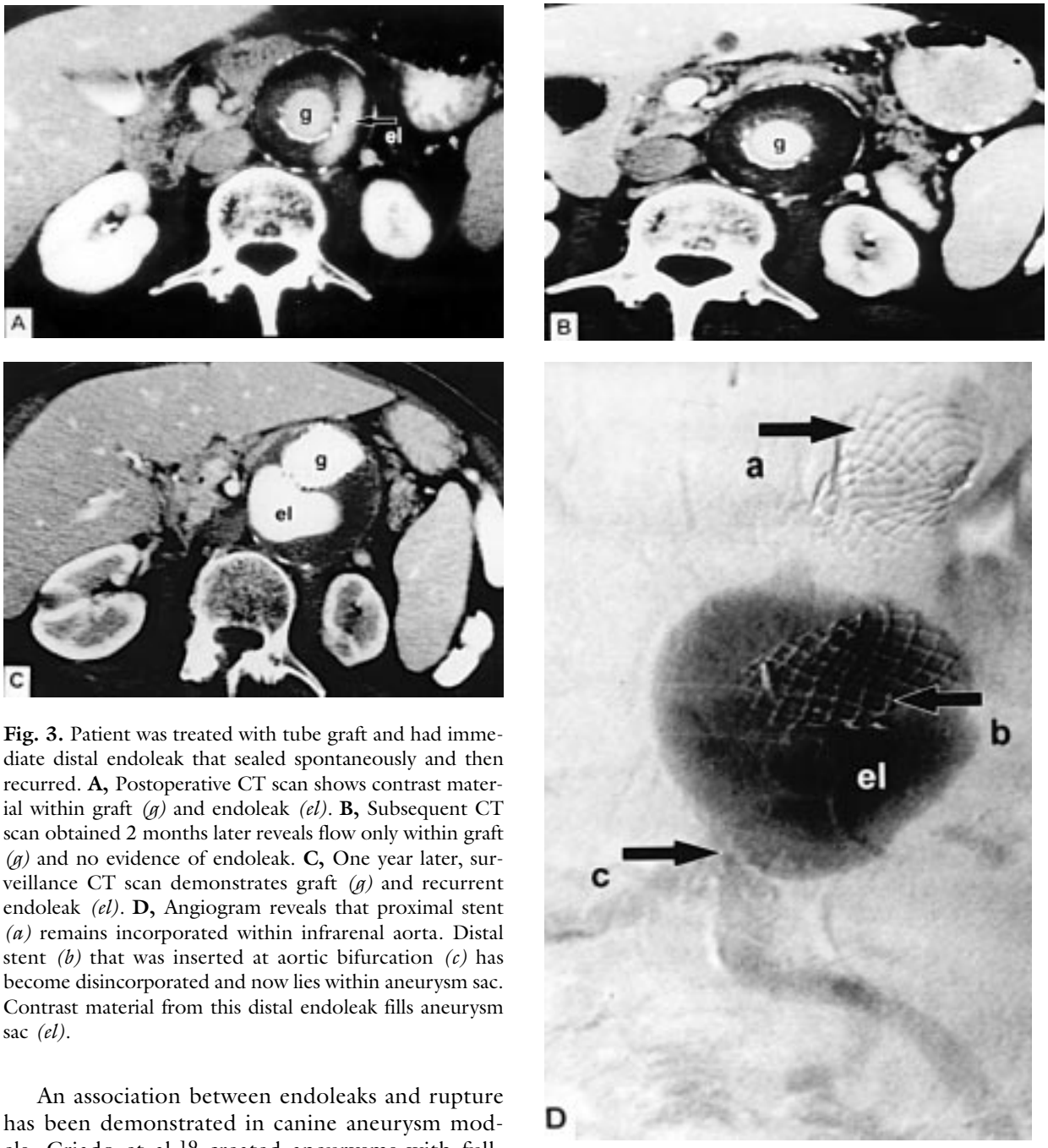

Fig. 3. Patient was treated with tube graft and had immediate distal endoleak that sealed spontaneously and then recurred. **A,** Postoperative CT scan shows contrast material within graft *(g)* and endoleak *(el)*. **B,** Subsequent CT scan obtained 2 months later reveals flow only within graft *(g)* and no evidence of endoleak. **C,** One year later, surveillance CT scan demonstrates graft *(g)* and recurrent endoleak *(el)*. **D,** Angiogram reveals that proximal stent *(a)* remains incorporated within infrarenal aorta. Distal stent *(b)* that was inserted at aortic bifurcation *(c)* has become disincorporated and now lies within aneurysm sac. Contrast material from this distal endoleak fills aneurysm sac *(el)*.

An association between endoleaks and rupture has been demonstrated in canine aneurysm models. Criado et al.[19] created aneurysms with full-thickness jejunal patches, which, in contrast to prosthetic patches, were susceptible to rupture. After endovascular graft treatment of these aneurysms, the aorta-aneurysm sac pressure differential was measured. In dogs without endoleaks, the pressure differential was high and there were no reports of aneurysm rupture. In dogs with endoleaks, there was no significant pressure differential and all of the aneurysms ruptured within 5 days of surgery.

Aneurysm sac pressure was also measured in a canine endoleak model developed in our laboratory using polytetrafluoroethylene interposition aneurysms.[20] When these aneurysms were successfully excluded by an endovascular graft, a large aorta-aneurysm sac pressure gradient was observed. When an endovascular graft containing a fenestration (simulating an endoleak) was implanted, pres-

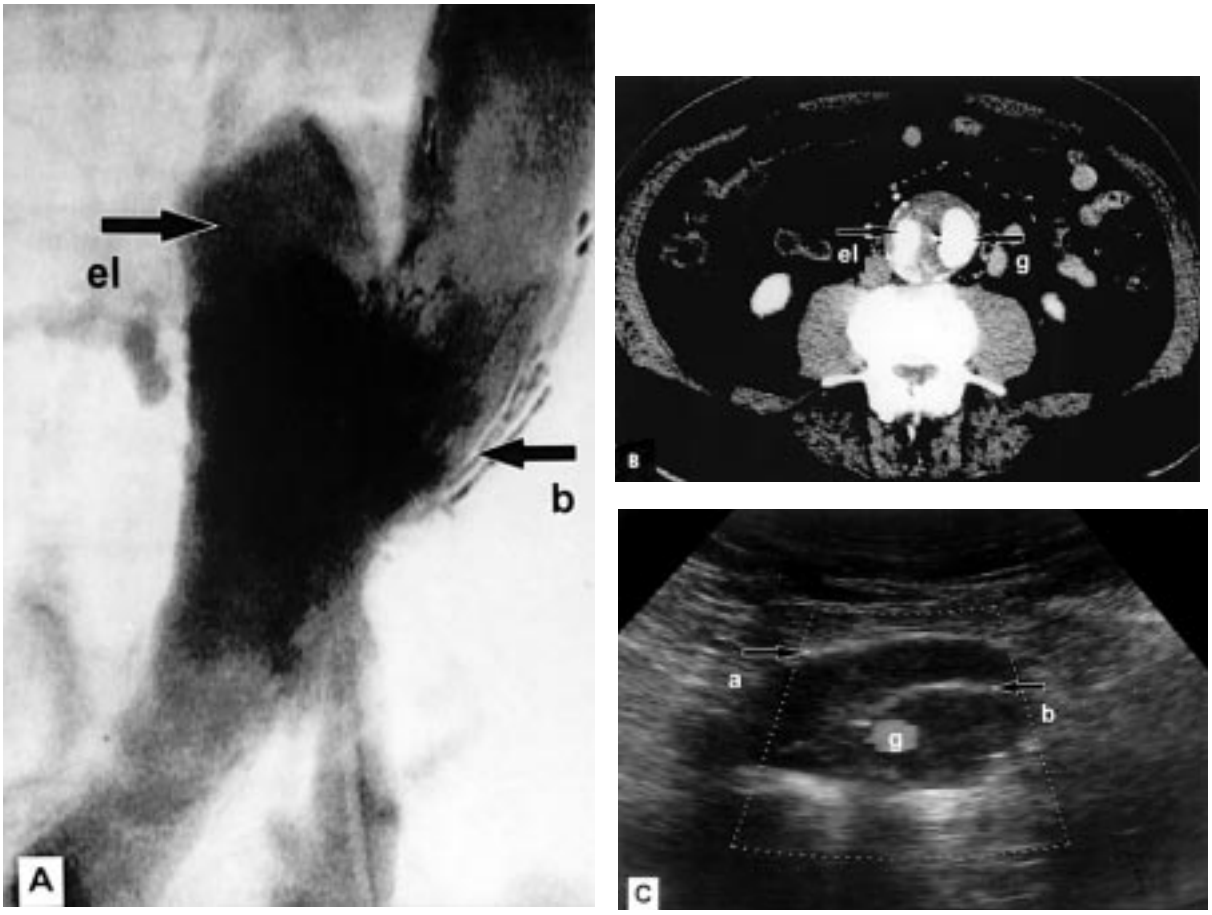

Fig. 4. Endovascular repair of endoleak caused by structural failure of previously inserted endovascular graft. Patient was treated with EVT tube graft and experienced delayed distal endoleak 1.5 years after operation when metallic hooks on distal attachment device fractured. **A,** Arteriogram demonstrates large endoleak *(el)* adjacent to broken distal stent *(b)*. **B,** Endoleak *(el)* can be seen on follow-up CT scan next to endovascular graft *(g)*. **C,** Patient underwent a reparative procedure in which a second endovascular graft was deployed within the first graft. Follow-up duplex scan shows flow in new graft *(g)*, which is seen within aneurysm sac *(a)* and within old graft *(b)*. There is no evidence of ongoing endoleak.

sure remained elevated in the aneurysm sac and minimal aorta-aneurysm pressure gradients were measured.[21]

Retroperitoneal exclusion for the treatment of abdominal aortic aneurysms is an alternative to transabdominal endoaneurysmorrhaphy. Resnikoff et al.[22] reported on the retroperitoneal exclusion of 831 aneurysms and found that 17 patients who underwent this procedure had residual flow within their aneurysm sacs. Of these, 14 patients underwent a second operation to treat abdominal or back pain related to the aneurysm or aneurysm rupture. The residual flow originated from iliac, lumbar, or inferior mesenteric arteries. Other cases of aneurysm rupture after nonresective repairs

Table IV. Physiologic classification of endoleaks

Endoleak feature	*No. (%)*
Inflow	
Aortic	15 (88)
Proximal end of graft	10 (59)
Distal end of graft	3 (17)
Graft defect	0 (0)
Occluder device*	2 (12)
Collateral†	2 (12)
Arterial side branch (hypogastric, lumbar, accessory renal, or inferior mesenteric artery)	

*By definition, occluder device endoleaks are considered in this group even though reperfusion of the aneurysm sac occurs in an indirect fashion through the femorofemoral bypass graft. †These originated from a hypogastric and a lumbar artery in patients who were treated during phase II of the study.

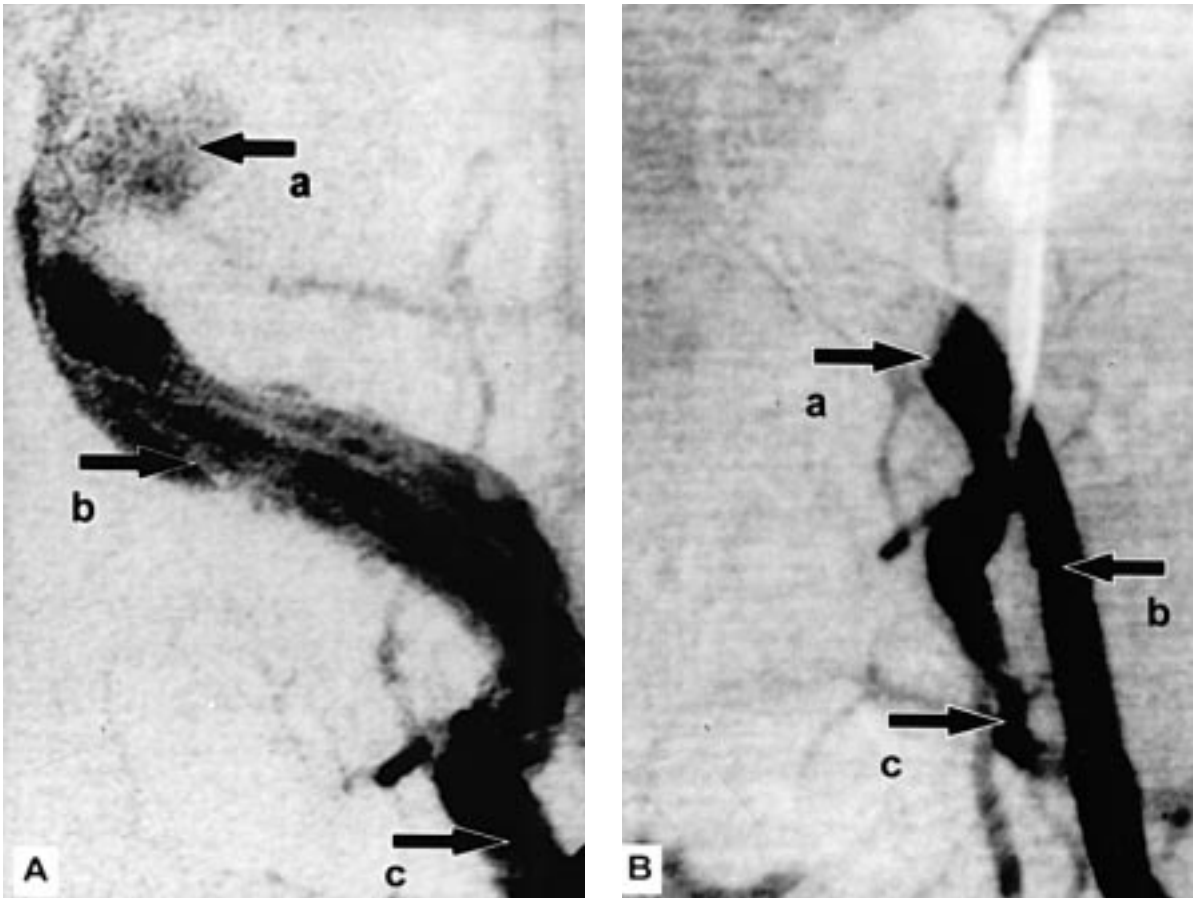

Fig. 5. This 68-year-old man had an occluder device endoleak after treatment with an aortofemoral, femorofemoral graft. **A,** Angiogram reveals that aneurysm sac *(a)* is filling with contrast material from endoleak *(b)* originating in iliac occluder device. Angiogram was performed in retrograde manner through external iliac artery *(c)*. **B,** Occluder device has been packed with metallic coils, and angiogram now demonstrates an abrupt cut-off of contrast material where endoleak had been *(a)*. Endoleak has sealed, and contrast material is seen only within external iliac *(b)* and internal iliac *(c)* arteries and not within aneurysm sac.

have also occurred.[23,24] Residual intrasaccular flow after nonresective aneurysm repair is analogous to endoleaks originating from collateral sources after endovascular abdominal aortic aneurysm repair. These experiences provide further evidence that rupture of incompletely excluded aneurysms after endovascular repair can occur.

Endoleaks can be multifactorial in cause and result from aneurysm anatomic features, endovascular graft design characteristics, or graft deployment techniques. In this series, we evaluated aortic morphologic features and their effect on endoleak incidence. Aneurysms with short necks were found to have a higher rate of endoleaks than those with longer necks. Some of the modifications implemented in the second phase of our study attempted to address this issue.

Aneurysms with short infrarenal necks require more exact placement of an endovascular graft's proximal stent and graft than those with longer necks. If the stent and graft are deployed too low, they may be located in aneurysmal or thrombus-lined aorta. In these cases, progressive dilatation of the aneurysm neck or dissolution of the thrombus can result in stent disincorporation and an endoleak. Moreover, there is less overlap between the graft and the aorta to provide an effective seal. Finally, large-diameter proximal necks may be more prone to dilatation than smaller necks, leading to stent disincorporation and endoleaks.

Deployment of the proximal stent of an endovascular graft in the juxtarenal versus infrarenal aorta can potentially prevent these problems. The juxtarenal aorta is less susceptible to aneurysmal dilatation and is almost never involved with thrombus. Such deployment assures maximal overlap of the graft and

normal nondilated aortic wall. These characteristics make the juxtarenal position a better fixation site for the stent and assure a more hemostatic and durable seal between the aorta and the proximal portion of the graft.

Other changes in the second phase of our study involved modifications to the endovascular grafts. In contrast to the grafts inserted in phase I, the modified grafts did not require prospective determination of graft length for each patient and did not rely on the deployment of a second, distal stent for their successful insertion. Instead, these grafts were long enough so that they could be retrieved from within the femoral arteriotomy. After customizing the graft's length, the distal anastomosis could then be performed in a hand-sewn endoluminal fashion. These changes allowed us to successfully treat patients who had infrarenal aneurysms regardless of their distal aneurysm neck lengths or the presence of iliac artery aneurysmal disease. In addition, precise preoperative graft/aneurysm length measurements, crucial to the deployment of aortoaortic tube grafts and aortoiliac grafts, were of much less consequence. Therefore, these changes could be expected to decrease the incidence of endoleaks that result from inexact length measurements or misdeployed endovascular grafts.

Although not significant, aneurysms with patent side branches also had a higher rate of endoleaks than those without such branches. Preoperative coil embolization of the hypogastric artery was implemented to eliminate endoleaks of hypogastric artery origin. In the future, the potential use of preoperative or intraoperative coil embolization could prevent large lumbar arteries or patent inferior mesenteric arteries from causing endoleaks. When this will be necessary remains to be determined because some patent side branches thrombose and do not cause endoleaks.

To date, there has been little consensus on how endoleaks should be reported. The currently described anatomic, chronologic, and physiologic classifications group endoleaks on the basis of features whose presence or absence is easy to ascertain. The anatomic classification facilitates our understanding of the pathogenesis of endoleaks. In addition, it may help lead to improvements in endovascular graft design and patient selection and therefore decrease the endoleak rate.

The chronologic and physiologic classifications may give us insight into the behavior of endoleaks and how they are best treated. Current clinical and experimental evidence suggests that aortic endoleaks have high pressures and can cause aneurysm enlargement or rupture and should be routinely treated by revision, surgical conversion, or other endovascular techniques. Based on the Albany series of aneurysms treated with retroperitoneal exclusion,[22] we believe that the collateral inflow endoleaks are also potentially dangerous, although they may be associated with lower or more variable pressures. However, because collateral vessels often thrombose after aneurysm repair rather than cause endoleaks, their significance is less certain. Endoleaks that originate from a hypogastric artery crossed by an endovascular graft can and should be prevented by preoperative coil embolization. Although more difficult to accomplish, secondary embolization of collateral endoleaks may also be warranted. In the future, it may be possible to choose which collateral endoleaks require treatment and which can be safely observed. Finally, because endoleaks can recur, lifelong surveillance must be recommended at this time for all patients who undergo treatment with endovascular grafts.

CONCLUSION

Endoleaks complicate a significant number of endovascular abdominal aortic aneurysm repairs and can lead to aneurysm rupture. The cause of endoleaks is multifactorial and includes anatomic as well as graft design and technique-related characteristics. In particular, we found that aneurysm neck lengths 2 cm or less increased the incidence of endoleaks. The combination of juxtarenal stent placement, preoperative hypogastric artery coil embolization, and aortofemoral, femorofemoral graft insertion appears to have decreased their incidence. Furthermore, endoleaks can be classified according to their anatomic, chronologic, and physiologic features to facilitate their reporting. These classifications can also improve our understanding of the pathogenesis, clinical significance, and fate of endoleaks and help decide how they are best treated.

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DISCUSSION

Dr. David C. Brewster (Boston, Mass.). Obviously, the problem of endoleaks is a very important one because, as you and others have emphasized, this basically represents a failure of the goal of endovascular repair, which is total exclusion of the aneurysm. Therefore, I think your studies and observations are clearly important. You mentioned that the incidence of endoleaks in phase II patients was quite a bit lower, and I wonder whether you could clarify whether you believe that this is because of the difference in graft configuration, more stringent patient selection, or perhaps simply your increasing experience with endovascular stent-graft placement?

Second, our own bias in an initial experience with approximately 40 endovascular stent-graft repairs of aortic aneurysms has been to use the adjunct of placing coils within the aneurysm sac at the time of stent-graft placement, hoping thereby to further promote thrombosis within the sac and by this additional maneuver decrease the incidence of branch-to-branch endoleaks. Could you comment on this theory or strategy?

Dr. Reese A. Wain. As you pointed out, the incidence of endoleaks in the second phase of our study was considerably less than that in our earlier experience. Because patient selection did not change significantly throughout our study, I believe that the graft design and deployment strategies used in phase II were largely responsible for the improved results. Although the small sample size does not permit quantitative analysis, I believe that juxtarenal graft placement was the single most important factor in limiting the incidence of endoleaks. I am sure that our increasing experience with endovascular graft procedures was a positive influence, as well.

We have not tried placing metallic coils within the aneurysm sac in an attempt to promote thrombosis, although the concept is intriguing. As you know, this practice was attempted in the early 1900s to treat inoperable aneurysms when yards and yards of metallic wire were introduced into the sac to promote thrombosis. This practice never achieved widespread appeal and was abandoned

as the sole treatment for aneurysmal disease. However, as an adjunct to endovascular grafting, this technique may be resurrected.

Dr. Frank W. LoGerfo (Boston, Mass.). I enjoyed your presentation and compliment you on it. We, like many places, have been asked to participate in these trials and are trying to get organized to do that. I have a question related to that particular issue.

I noticed that these are straightforward aneurysms, 5 cm, long neck, and so forth. With the endovascular techniques there is a stent across the renal artery orifices, there are coils in the hypogastric, and there is a femorofemoral graft. I am wondering how you deal with the issue of informed consent to get patients to participate in a trial like this when you know that surgically this is a piece of cake. That particular aneurysm, you have to guess, is in the 1% mortality range, no issues of sexual dysfunction after the operation, a simple tube graft, and no concerns about the renal arteries. How do you deal with that in informing a patient to accept the endovascular alternative?

Dr. Wain. The majority of the patients in our series were high-risk patients. These patients were treated only after a cardiologist and pulmonologist independently concluded that the patient could not tolerate conventional aneurysm repair. The typical endovascular graft was not inserted in a 50-year-old with a 5 cm aneurysm and mild hypertension. Instead, our patients were often octogenarians with 7 cm or larger aneurysms, multiply reoperated abdomens, severely depressed ejection fractions, and marked pulmonary dysfunction. These patients were not candidates for open repair, and their best hope for treatment was an endovascular procedure.

The good-risk patients underwent extensive counseling in the preoperative period. They were offered treatment with an endovascular graft and were told explicitly that it remains an experimental procedure. These patients were aware of the good results that have been reported with open repair but have nonetheless arrived at an educated decision to proceed with placement of an endovascular graft.

Dr. Mark F. Fillinger (Lebanon, N.H.). I enjoyed your presentation. I have a question about the number of endoleaks that had no apparent outflow. As I understand it from your presentation, the large majority of endoleaks had no apparent outflow. I was wondering whether you could describe your CT techniques in terms of timing and volume of the contrast load, the beam collimation, and interval of reconstruction. In our experience, it is very unusual to see an endoleak without both inflow and outflow.

I would also like to know whether endoleaks that had no apparent outflow were more likely to seal spontaneously than endoleaks that did have an outflow?

Dr. Wain. I would be interested to know how you image your patients, because in our experience most endoleaks have not been found to have outflow. Twelve (71%) endoleaks had no outflow compared with only five (29%) with outflow. Our CT scans are performed according to a standard vascular protocol using 3 mm cuts and contrast boluses timed to highlight the arterial anatomy. One of the techniques we are currently evaluating is that of a delayed-phase CT scan study, which may prove more sensitive for detecting endoleaks.

Because of the small sample size, a comparison between the number of endoleaks that sealed with and without outflow would not be meaningful.

Dr. Thomas S. Riles (New York, N.Y.). I have two questions. One is about the classification of that one type of leak as a low pressure leak. I'm curious to know whether you've actually measured pressures in the sac to determine that it was low pressure. From my understanding of hydraulics, a very small artery can maintain systemic pressure, especially if there is not outflow. Are you justified in using the term "low pressure" in this context?

Second, have you classified the leaks according to the types of grafts that you have used? I've been involved only with the EVT grafts. Of the 17 successful implants we have performed, we have had only one leak, and that occurred 2 years after implantation because of the fracture of a hook. I'm not sure whether it's because of the type of graft that I'm using or selection of patients. Could you correlate the complications with the different types of grafts you use?

Dr. Wain. In answering your first question, I must say that we did not actually measure the pressures inside the aneurysm sac. I tried to be careful in the presentation to distinguish between high-pressure (aortic) inflow endoleaks and those endoleaks with low- or variable-pressure inflow, which is transmitted through a collateral bed and is therefore in some way damped. Even so, we know that collateral vessels can harbor significant pressures, although I doubt that they can achieve systemic pressure, and systolic and pulse pressure is always less than in the aorta.

Endoleaks were discovered in three of four of our EVT tube grafts, four of seven Parodi-type tube grafts, four of eight aortoiliac, femorofemoral grafts, and only six of 28 patients who were treated with aortofemoral, femorofemoral grafts.

Dr. Carlos E. Donayre (Torrance, Calif.). I compliment your analysis of your data. We heard earlier about the value of intravascular ultrasound in stent deployment, and we have also been very impressed with its use in the deployment of stented grafts at Harbor-UCLA. Could you comment on the use of intravascular ultrasound on balloon-expandable covered stents? I know you have had experience with it. Can you comment on the endoleak rate difference between your phase I and phase II groups? Did you use intravascular ultrasound in the phase I group or both groups? Do you think that its use attributed to your change in endoleak rate, especially with regard to the proximal neck?

Dr. Wain. Thank you for raising that issue, Dr. Donayre. We have used intravascular ultrasound in almost all of the patients that we treated in this study, so I do not think that the findings on ultrasound could account for the difference in endoleak incidence. We used intravascular ultrasound to assure that the proximal stent had the

appropriate apposition to the aortic wall and that the graft was completely unfolded along its course. It is interesting to note that in a study on imaging techniques for endovascular grafts by Lyon et al. from our institution that intravascular ultrasound was a poor technique for detecting endoleaks, although it was more useful than fluoroscopy in a number of other ways, particularly in detecting graft compression or narrowing.

Dr. George J. Kretschmer (Vienna, Austria). Is it possible to distinguish primary leaks, which are observed immediately after the procedure, and secondary leaks that develop during late follow-up? You observed two spontaneously thrombosed leaks. Were these primary or secondary leaks?

Dr. Wain. Immediate or primary endoleaks are distinguished from delayed endoleaks by when they are discovered in the postoperative period. Immediate endoleaks are seen on the first postoperative imaging study. Delayed endoleaks are not seen on the first postoperative study but are visualized on a subsequent study.

Both of the endoleaks that sealed spontaneously were primary endoleaks. One of these was a proximal endoleak without outflow that sealed 1 month after operation, and the other was a midgraft endoleak without outflow that sealed within 2 months.