Long-term fate of the aneurysmal sac after endoluminal exclusion of abdominal aortic aneurysms

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Purpose: **Shrinkage of an abdominal aortic aneurysm (AAA) is the hallmark of successful endoluminal treatment. Our goal was to prospectively assess the midterm to long-term shrinkage of the AAA sac after endovascular repair.**

Methods: **A total of 123 patients with AAA underwent endoluminal treatment with the Ancure device at our institution between February 1996 and February 2000. At least a 1-year follow-up was available for 70 of the 123 patients. AAA sac size, presence of endoleaks, calcifications, and outcome data were collected on these patients at 6, 12, 24, and 36 months after repair and compared with the preoperative AAA size and characteristics. All endoleaks found at the 6-month follow-up visit were treated aggressively with embolotherapy. An AAA sac regression of 0.5 cm or more was considered the minimum measurable decrease. Regression of the sac diameter to 3.5 cm or less was considered a complete collapse of the sac.**

Results: **Successful endoluminal repair was accomplished in 119 of 123 patients. The mortality rate was 0.8% (1/123). There was a steady decrease in AAA sac size from baseline (5.56 ± 0.1 cm), to 6 months (5.0 ± 0.14 cm, $P = .0006$), to 12 months (4.65 ± 0.13 cm, $P = .04$), and to 24 months (4.26 ± 0.16 cm, $P = .03$). At 24 months, 74% (29/39) had a decrease in sac size of 0.5 cm or more, with 28% (11/39) complete collapse. Patients with initial endoleaks had the same likelihood of regression of sac size (≥ 0.5 cm) when compared with the group of patients with no endoleaks at the 24-month evaluation (64% vs 76%, $P = .09$).**

Conclusion: **Endoluminal AAA repair resulted in a significant reduction in sac size that continues up to 2 years. Significant shrinkage occurs as early as 6 months after placement. The initial presence of endoleaks does not predict the lack of sac regression.**

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Endovascular treatment for abdominal aortic aneurysms (AAAs) is now a widely accepted and available option for many patients.[1-3] Significant evolution of technology regarding this relatively new method of treatment has occurred over the past several years.[2-4] Currently, there are two Food and Drug Administration (FDA) approved devices on the market: Ancure (Guidant-Endovascular Technologies, Menlo Park, Calif) and AneuRx (Medtronic, Minneapolis, Minn). It is anticipated that at least two or three new devices will be approved within the next 2 years.

As expected with any new form of treatment, long-term or even midterm results have not yet been clearly defined.[5,6] Most available reports have detailed the early perioperative results or follow-up for up to 1 year. Of particular importance is that the definition of successful treatment has been a moving target. Technical implantation success was first reported; success was later modified to include the absence of endoleaks, and most recently the avoidance of rupture. The simple lack of increase in AAA

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size has been included as a sign of success on the guidelines of the advisory committee of The Society for Vascular Surgery/International Society of Cardiovascular Surgery, North American Chapter.[7] Shrinkage and total collapse of the aneurysmal sac, however, remain the undisputed sine qua non of successful treatment.

Decreasing AAA sac size implies the reduction of intra-aneurysmal pressure and may be the best marker of a successful treatment and reduced risk of rupture, the ultimate goal of any therapy for this disease. Documentation of progressive aneurysmal sac regression after device implantation is therefore considered a significant indicator of successful treatment.[8,9] Although many studies have reported the early shrinkage of aneurysmal sacs up to 1 year, few longer term reports are available on this important aspect of endovascular management of AAA.[1,2,5,6]

The goal of this study was to report the extent and variability of sac regression over a midterm to long-term follow-up period after the endoluminal repair of AAA with the Ancure device at a single institution.

METHODS

We reviewed 123 patients who underwent endoluminal AAA repair with the Ancure device at the University of Pittsburgh Medical Center over a 4-year period from February 1996 to February 2000. All endografts were deployed in the operating room with the patient under general anesthesia by a vascular surgeon using a 12-in digital C-arm fluoroscopy unit (Series 9600 OEC Medical Systems, Salt Lake City, Utah). An interventional radiologist was the first assistant on most cases. The procedural details have been previously reported.[10,11] Most patients were part of multicenter phase II or III FDA-approved clinical trials with around 25% implanted after marketing of the Ancure system. Three different configurations of the Ancure device were used: tube, bifurcated, and aortoiliac. All protocols were reviewed and approved by the University of Pittsburgh Institutional Review Board and reviewed annually. All patients in the clinical trials signed a research informed consent.

Determination of eligibility for endoluminal repair was based on anatomic considerations. The two main exclusion criteria were the length of the proximal neck under 15 mm and inadequate access through the iliac arteries. Approximately 25% of all infrarenal AAAs evaluated at our institution qualified for endoluminal repair. All eligible patients underwent preoperative spiral computed tomography (CT) of the abdomen and pelvis with 3-mm cuts. Preprocedural aortography was obtained routinely during the first 2 years (phase II) and when the anatomy was unclear by CT evaluation for the most recent cases.

Matsumura et al[9] proposed that the size of the aneurysmal sac should be considered to be the minor diameter of the largest section of the AAA on an axial CT. Although this method may underestimate the true size of the AAA in certain cases, it is more reproducible, and it more accurately reflects changes over time, by preventing inaccurate sizing that results from the tortuosity of the AAA found in most patients. AAA size was measured preoperatively and repeated at 6-, 12-, 24-, and 36-month follow-up using the above technique. All measurements were performed prospectively by the senior author (M.S.M.) with a computer-aided digital-measuring tool.

Patients in the phase II evaluation of the Ancure graft with a documented endoleak (either at the time of implantation with angiography or with initial postoperative CT) were also reevaluated at 3 months. Patients with persistent endoleaks at 6 months underwent angiography and aggressive treatment of their endoleaks with percutaneous coil embolization with 0.038-in diameter coils and Tornado microcoils (Cook, Bloomington, Ind). The technique and results have been previously reported.[12]

The extent of aortic sac wall calcification was determined at its widest diameter. The following classification was used to determine the extent of circumferential involvement of the calcification: 0 (0%-25%), 1 (25%-50%), 2 (50%-75%), 3 (75%-100%). All patients were classified according to their preoperative CT. Because the accepted variability of the measurements can be as high as ± 0.5 cm,[13] AAA sac shrinkage was determined to be only significant when there was a 0.5-cm or less decrease in sac size from the preoperative CT. Complete collapse of the sac was defined arbitrarily to be a reduction of 3.5 cm or less. Measurements were expressed as the mean ± SE. The Student *t* test, the Fisher exact test, and χ^2 analysis were used to compare the various groups with respect to sac diameter. Statistical significance was defined as a *P* value less than .05.

RESULTS

There were 119 (97%) of the 123 total patients in the study who underwent successful implantation of an endoluminal device. Insertion technique was transfemoral (unilateral for tube grafts and bilateral for bifurcated grafts) in most cases. Three patients required transcommon iliac insertion because of diseased femoral or external iliac arterial systems. The perioperative mortality rate was 0.8% (1/123). One

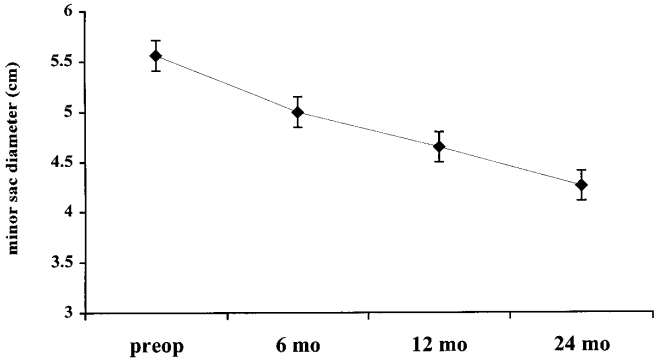

Fig 1. Minor diameter sac regression after endoluminal abdominal aortic aneurysm repair over a 24-month follow-up period.

Table I. Changes in the minor diameter of the aneurysmal sac over a 24-month follow-up period after endoluminal stent-graft repair of abdominal aortic aneurysms in 70 patients

	Baseline (n = 70)	*6 mo (n = 48)*	*12 mo (n = 55)*	*24 mo (n = 39)*
Sac size (cm)*	5.56 ± 0.1	5.0 ± 0.14*	4.65 ± 0.13*	4.26 ± 0.16*
Change in sac minor diameter (cm)	–	0.53 ± 0.1	–0.87 ± 0.11	–1.22 ± 0.15
No. of patients with decrease (%)†		21 (44%)	38 (69%)	29 (74%)
Complete collapse‡		2 (4%)	6 (11%)	11 (28%)

*$P < .05$.
†Size decrease from baseline of ≥ 0.5 cm.
‡Defined as sac size reduction to ≤ 3.5 cm.

high-risk patient from the early segment of the study died of an autopsy-proved myocardial infarction 3 days after the procedure. There were two intraoperative open conversions and two failures to access. Four patients returned to the operating room on the same day of treatment. One patient was bleeding around one of the proximal hooks, two patients had an atheromatous embolus to the tibioperoneal trunk, and one had an occlusion of one of the limbs that required thrombectomy and stenting.

Only a limited number of late interventions were required. None of the patients needed explantation of the device; none had early or late sac ruptures. One patient with a cephalad migration of the distal attachment system of a tube device was treated successfully with a new endoluminal graft. All endoleaks were successfully treated between 8 and 16 months after initial implantation. Ten patients died of unrelated causes: myocardial infarction (5), cirrhosis (1), malignancy (2), cerebrovascular accident (1), renal failure (1).

Of the 123 patients treated with the Ancure device, 70 had at least a 1-year follow-up visit with a CT evaluation. They also had a mean follow-up period of 21.5 months and were analyzed for sac shrinkage. These patients comprised 61 men and nine women (age, 72 ± 0.82 years). The demographics, risk factors, and ASA classification were similar for all subgroups.

The mean preoperative AAA size was 5.56 ± 0.1 cm (range, 4.2-8.1 cm). Longer term follow-up was available on a smaller group of patients (39 with 24-month, 9 with 36-month, and 1 patient had a 4-year evaluation). Of the nine patients who had a 3-year follow-up, 67% had at least a 0.5-cm reduction in sac size. The only 4-year follow-up patient had regression of sac size from 4.8 cm (baseline) to 2.7 cm (48 months), which was essentially the diameter of the endograft.

Overall, there was a steady decrease in AAA sac size from baseline to 6 months (5.0 ± 0.14 cm, $P = .0006$), to 12 months (4.65 ± 0.13 cm, $P = .04$), to 24 months (4.26 ± 0.16 cm, $P = .03$) (Fig 1, Table I). At 24 months, 74% (29/39) had a decrease in sac size of 0.5 cm or more, with 28% (11/39) complete collapse (Fig 2, Table I). The mean sac diameter

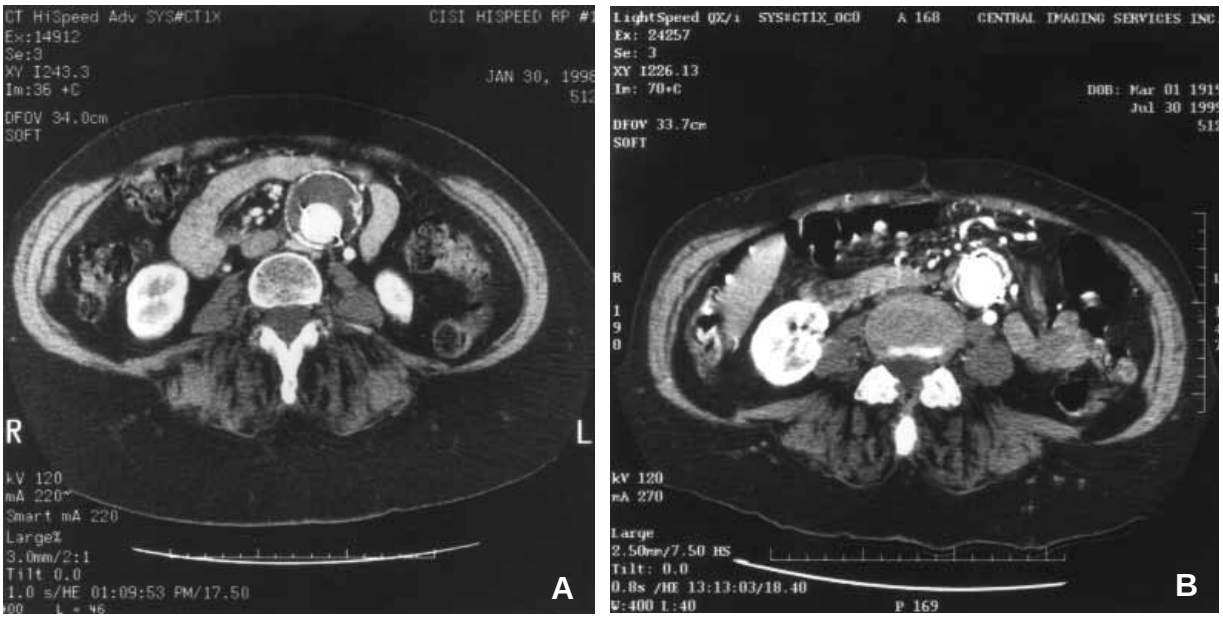

Fig 2. A, Computed tomography of 76-year-old patient 1 month after endoluminal repair of a 6-cm abdominal aortic aneurysm. Despite the severe calcification, the sac has decreased to 5 cm. **B,** Computed tomography of same patient 13 months after endoluminal repair. Note sac has essentially collapsed around the stent-graft and now measures 3 cm.

Table II. Effect of the type of endoluminal stent-graft used in determining the minor diameter of the aneurysmal sac over a 24-month follow-up period

	Preop	*6 mo*	*12 mo*	*24 mo*
Tube (n)	17	14	13	10
Size (cm)	5.1 ± 0.14	4.58 ± 0.24	4.2 ± 0.2	4 ± 0.3
No. of patients with decrease (%)*		5 (36%)	9 (69%)	7 (70%)†
Complete collapse‡		2 (14%)	2 (15%)	4 (40%)
Bifurcated(n)	47	30	38	25
Size (cm)	5.69 ± 0.13	5.17 ± 0.17	4.7 ± 0.17	4.3 ± 0.2
No. of patients with decrease (%)*		15 (50%)	28 (74%)	20 (80%)†
Complete collapse‡		1 (3%)	4 (11%)	4 (16%)

*Size decrease from preop of ≥ 0.5 cm.
†$P > .05$.
‡Defined as sac size reduction to ≤ 3.5 cm.

reduction was reduced by 0.53 ± 0.1 cm at 6 months and 1.22 ± 0.15 cm at 36 months (Table I). There was documented shrinkage of the sac at all intervals.

Only three patients had a significant increase in size (> 0.5 cm) during follow-up. All of these patients had endoleaks and were subsequently treated. Decrease in the sac size was noted in all successfully treated endoleaks (complete obliteration of the communication between the sac and the native blood artery) during the 2-year follow-up. The three patients who had an increase in sac size had appropriate sac reduction 2 years after treatment (patient 1, 5.5 cm from 5.7 cm; patient 2, 3.2 cm from 5.2 cm; patient 3, 4.4 cm from 5.5 cm).

Graft configuration was primarily bifurcated (47/70 [67%]). The tube (17/70 [24%]) and aortoiliac (6/70 [9%]) grafts were used in one third of cases. Tube grafts were associated with more endoleaks (10/17 [58%]) than the bifurcated devices (12/47 [25%]), ($P = .01$). No significant difference in sac

Table III. Effect of calcification in determining the minor diameter of the aneurysmal sac over a 24-month follow-up period

CA class	*Preop*	*6 mo*	*12 mo*	*24 mo*
CA = 3 (n)	12	9	12	9
Size (cm)	5.38 ± 0.24	5.1 ± 0.32	4.74 ± 0.3	4.4 ± 0.4
No. of patients with decrease (%)*		4 (44%)	9 (75%)	6 (66%)†
Complete collapse‡		0 (0%)	1 (8%)	3 (33%)
CA = 0 (n)	24	21	17	19
Size (cm)	5.62 ± 0.19	4.99 ± 0.23	4.74 ± 0.21	4.22 ± 0.21
No. of patients with decrease (%)*		8 (38%)	12 (71%)	15 (79%)†
Complete collapse‡		1 (5%)	2 (12%)	6 (32%)

CA 3 represents severe (75%-100%) circumferential calcification. CA 0 represents minimum (0%-25%) calcification.
*Size decrease from preop of ≥ 0.5 cm.
†$P < .05$.
‡Defined as sac size reduction to ≤ 3.5 cm.

shrinkage was noted between configuration at 6 months or 1 year. At 24 months, however, more patients with the bifurcated device had a significant reduction in size than the tube grafts (Table II).

Twenty-four patients were determined to have a calcification index of 0 (0%-25%). The majority of the group (34/70, 49%) had only partial calcification: an index of 1 (25%-50%) and 2 (50%-75%). More patients in the 0 group had measurable decreases in sac size at the 2-year interval than those patients with severe calcification (index of 3, 66% vs 79%, $P < .02$, Table III). However the rate of complete collapse of the sac in both groups was similar (Table III).

Twenty-five patients were documented to have an endoleak. The majority of these were type II, but the exact type or origin of the endoleaks was difficult to ascertain on the basis of CT scans. Sixteen sealed spontaneously, and eight were treated successfully with percutaneous coil embolization and obliteration of the endoleak. One patient with a shrinking AAA, a persistent endoleak, and progressive heart failure was not treated. He died of cardiac problems at 14 months. This group of patients with an initial endoleak had similar regression of sac size (≥ 0.5 cm) at 24 months, when compared with the group of patients who never exhibited an endoleak (64% and 76%, respectively; $P = .09$; Table IV). There was no difference in sac shrinkage between the treated endoleak group and the untreated endoleak group, who spontaneously sealed ($P > .05$).

DISCUSSION

This study represents a prospective evaluation of AAA sac morphology after endoluminal repair with one of the first two FDA-approved devices to be released for general use. It is one of the earliest reports of AAA sac regression extended to 2 years now that a significant number of patients treated between 1996 and 1998 are returning for 2- and 3-year follow-up. Shrinkage of the AAA sac has been demonstrated to occur as early as 6 months after implantation of the Ancure endograft with a steady regression up to 2 years.

Malian et al,[14] in one of the few reports to go beyond 1 year of follow-up, proposed that most of the regression of the AAA sac occurred in the first year with minimal changes thereafter. Our data, however, reveal a progressive reduction in the mean size of the AAA by an additional 0.4 cm between 12 and 24 months. A complete collapse of the AAA is also much more likely during the second year of follow-up when compared with the first. In addition, a small percentage of patients who had not yet shown a reduction in size by 12 months will show regression at the 2-year follow-up. These results are very encouraging for long-term efficacy of endoluminal repair of AAA.

The definition of significant reduction in size was arbitrarily set at a minimum of 0.5 cm. This was in keeping with the definitions used in the multicenter phase II trials approved by the FDA. Large-scale analyses of interobserver and intraobserver variability in evaluating AAA size from CT scans has shown that a 5-mm threshold would represent an actual change in the aneurysm rather than measurement error.[13] The use of the minor diameter on the axial slices also ensures the reduction in measurement variability due to tortuosity of the AAA and positioning of the patient.

Table IV. Effect of endoleak in determining the minor diameter of the aneurysmal sac over a 24-month follow-up period. Note that all patients with a persistent endoleaks were treated with embolotherapy at the 6-month follow-up visit.

	Preop	*6 mo*	*12 mo*	*24 mo*
No leak (n)	45	27	38	25
Size (cm)	5.49 ± 0.12	4.89 ± 0.22	4.56 ± 0.16	4.25 ± 0.2
No. of patients with decrease (%)*		12 (44%)	27 (71%)	19 (76%)
Total leak (n)	25	21	17	14
Size (cm)	5.63 ± 0.17	5.14 ± 0.14	4.84 ± 0.22	4.23 ± 0.25
No. of patients with decrease (%)*		8 (38%)	9 (53%)	9 (64%)
SS group (n)	16	13	13	10
Size (cm)	5.64 ± 0.26	4.91 ± 0.2	4.6 ± 0.23	4.1 ± 0.28
No. of patients with decrease (%)*		6 (46%)	9 (69%)†	8 (80%)†
CE group (n)	9	8	7	7
Size (cm)	5.61 ± .07	5.59 ± .08	5.63 ± 0.38	4.68 ± 0.56
No. of patients with decrease (%)*		1 (13%)	2 (29%)†	3 (43%)†

*Size decrease from preop of ≥ 0.5 cm.
†$P < .05$ cm.
CE, Coil embolization; *SS,* spontaneous seal.

Not all reports of size changes have used the same standards. The French Vanguard trial reported a reduction of at least 0.3 cm for 27% of the patients at 6 months.[3] Despite a more rigid criterion, we noted that 44% of the patients had at least a 0.5-cm decrease in AAA sac diameter at 6 months. Although the French trial reported that 75% of the patients with a follow-up period between 12 and 24 months had a reduction, the magnitude of the change was not specified. Mean sac shrinkage at 1 year has been uniform across studies using the Ancure endograft. May et al[15] reported a 0.93-cm maximal AAA sac diameter reduction after 1 year in 30 patients similar to the 0.87 cm in the current report. At 2 years, the mean reduction in our study reached 1.22 cm. Other reports only reveal slight changes in AAA sac morphology during the early follow-up periods.[2,6,8,16]

Although the significance of endoleaks after endoluminal AAA repair is still unknown to a large extent, an increasing number of reports are proposing that endoleaks are in fact responsible for failure of treatment.[8,15,17] It is well accepted that the presence of an endoleak has the potential to increase AAA sac size, intramural pressure, and possibly lead to late rupture.[9,18] Therefore, we evaluated our study group with respect to this problem. Contrary to what is reported in the literature,[8,9,15] we found that the endoleak group had comparable sac shrinkage especially after long-term follow-up. This result was most likely the result of our aggressive stance to treat all patients with persistent endoleaks with coil embolization. Only three patients still exhibited an endoleak at 1 year and none after 18 months. All three of these patients eventually underwent coil embolization and successful treatment of the endoleak. This obviously implies that patients with an initial endoleak can expect sac regression when the endoleak seals spontaneously or with treatment. The group who underwent coil embolotherapy had outcomes similar to the spontaneously sealed group at 2 years (Table IV). These data reveal that embolotherapy is an important adjunct for successful endoluminal AAA repair and should be available to surgeons who perform this operation.

The calcification at the level of the aortic wall was also investigated in our study as a possible negative etiologic factor in sac regression. The classification scheme provided a method of quantification for circumferential calcium deposits. Interestingly, there were no significant differences among the groups with respect to size, but there was a difference between group 3 and group 0 in the number of patients who had sac reduction of 0.5 cm or more at the end of the 2-year follow-up period. It can be speculated that even the heavily calcified AAA sac may ultimately undergo remodeling when the pressure and flow are redirected. Thus, calcification of the aortic wall, though not a strong predictor of endograft failure, should be considered a factor in lack of sac regression if there is 75% to 100% circumferential involvement.

The concept of AAA sac regression after endoluminal repair appears intuitive. When the arterial pressure is redirected through the endoluminal device, the AAA sac should collapse around the graft.[19,20] However, this is not always the case.

When AAAs are excluded by simple ligation and bypass graft, leaving patent lumbar arteries and the inferior mesenteric artery, a number of these patients can continue to expand and even rupture.[21] Another proposed theory implicates a neointimal hyperplastic reaction over the endograft surface, which in turn causes a fibrotic shrinkage of the AAA sac.[19] This fibrotic process has not been confirmed clinically but is reported to occlude small arterioles and even lumbar arteries, helping obliterate the sac.

In summary, our review supports the notion that endoluminal AAA repair is very effective in reducing the size of the AAA, a process that continues past the first year of treatment. The threat of rupture and continued expansion were minimal. Patients with endoleaks after treatment behave similarly to those who have initial complete AAA exclusion. Three of four patients treated with the Ancure device can be expected to have at least a 0.5-cm reduction in sac size by 2 years with minimal or no risk of rupture.

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DISCUSSION

Dr William J. Quinones-Baldrich (Los Angeles, Calif). I would like to thank the Society for the opportunity to discuss this paper and congratulate Dr Rhee and his colleagues on an excellent presentation. Decreasing the size of the aneurysm sac after endoluminal repair is intuitively the best evidence of an effective treatment. The series presented by Dr Rhee today is an extremely encouraging report documenting that decrease in the aneurysm sac by more than 5 mm occurs in 74% of patients treated with the Ancure endovascular graft at 2 years. Twenty-eight percent of patients have complete collapse defined by regression of the aneurysm sac less than 3.5 cm. Importantly, the decrease in the aneurysm sac appears to continue as the time of follow-up is increased. The presence or absence of calcification in

the aneurysm wall did not appear to significantly alter the outcome. There was no difference in the sac shrinkage between those patients that had a treated endoleak and those that were untreated. It's still unclear to me after your presentation and reading the manuscript whether or not you had any endoleaks at the end of 1 year and you may want to clarify this issue. In a recent presentation at the Pacific Coast Surgical Association, the Stanford group reported their experience with the AneuRx system and only about 15% to 20% of patients had any significant decrease in the aneurysm sac greater than 5 mm at the end of the 1-year follow-up. This is significantly different than the 1- or 2-year data reported today with the Ancure/EVT device. Can you explain the difference in the reported incidence of sac shrinkage between the AneuRx and the Ancure system? Could it be related to the particular characteristics of the devices? The authors were not able to establish the amount of calcification in the aneurysm wall as a significant predictor of the behavior of the sac following endoluminal repair. They really only had nine patients at 24 months, which is where they achieved statistical significance, and six of the nine patients actually had sac shrinkage. They also proposed that aggressive treatment of an endoleak was responsible for eventual sac shrinkage in some of their patients; however, they were not able to establish the presence or absence of an endoleak as a definite predictor. In my opinion, one factor that may have significant influence in the behavior of the aneurysm sac is the amount of chronic thrombus already present within the aneurysm at the time of graft implantation, which leads to my second question. Did the authors look at the amount of chronic thrombus within the aneurysm on the preoperative CT to see if this had any predictive value on the actual behavior of the sac after repair? One of the more disturbing aspects that has been documented during follow-up of patients after endovascular aneurysm repair is the occasional patient that has an increase in the aneurysm sac in spite of the absence of a demonstrable endoleak. This has led some to postulate the concept of endotension within the aneurysm sac in spite of what appears to be complete exclusion with the endovascular graft. Again, after listening to your presentation and reading the manuscript, I am still unclear as to how many patients had an endoleak at the one year interval. Could you please clarify this? And this leads me to my last question. In how many patients did you observe an increase in the aneurysm sac and correlate this with the presence or absence of an endoleak? The data presented here today are extremely encouraging and support the notion that endovascular aneurysm repair can be an effective form of treatment. We certainly do not understand all the factors that influence the behavior of the aneurysm sac, particularly as it relates to the characteristics of the device itself. I am encouraged and repair enthusiastic about the endoluminal approach for aneurysm repair, and I congratulate the authors on an important contribution and an excellent presentation.

Dr Robert Y. Rhee (Pittsburgh, Pa). Thank you Dr Quinones-Baldrich for reviewing our paper. We, too, were very encouraged by the results of the study. The answer to the first question is one patient. This patient was not treated because of medical problems. Interestingly, the sac in this patient still regressed in size in spite of the endoleak. The remainder of the 25 patients with an endoleak were successfully treated at the 6-month or the 1 year follow-up. The second question regarding the possible differences between the Ancure and AnueRx devices is difficult to answer. One could only speculate that maybe the exoskeleton of the stent-graft devices may prevent significant sac regression. The issue of the role of thrombus was investigated in our study but not included in the data because it had no significant effect in sac regression. Finally, most of the patients with persistant endoleaks were noted to have sacs either that changed little in size or even increased over the 1-year follow-up period. However, all of these patients with endoleaks after 6 months were treated and subsequently experienced appropriate decrease in sac size.

Dr Christopher Zarins. (Stanford, Calif). I'd like to congratulate Dr Rhee on a beautiful presentation of very important data. I rise to ask the question about outcome end points. We frequently use endoleaks and changes in aneurysm size as the end point of analysis, and we're very happy when we have no endoleak and we're happy when the aneurysm decreases in size. And you've shown that you can achieve both of these. We have looked at our data at Stanford and looked at more than 100 patients over a year out, and we found that the rate of decrease in aneurysm size pretty much equals the reported rate of increase in aneurysm size in the natural history of study of aneurysms. But the interesting thing was that some, just as in aneurysm enlargement, enlarge very rapidly and rupture, and we also found that some aneurysms decrease very greatly in size. Now your paper suggested that that is a favorable and a good thing. But what we have found is that in a couple of patients who have late-onset acute endoleaks, even though the aneurysm has collapsed like a balloon, once you repressurize it, it reexpands like a balloon and perhaps has an increased chance of rupture. In the large AneuRx trial of over 1000 patients there was one patient who decreased in size from 9 cm to 6 cm and then ruptured. Never had an endoleak. So all of the favorable outcome analyses, no endoleak, and decreasing aneurysm size do not guarantee that you'll have a long-term outcome and no aneurysm rupture, so I think that in considering these end points that is really not the purpose of treatment (that is to prevent aneurysm rupture). Do we have a sense of security that we have indeed cured the patient if we have a decreasing-size aneurysm and no endoleak?

Dr Rhee. Thank you Dr Zarins for your thoughts. We, too, feel that regression of the sac is only one of the indicators of success for endovascular AAA treatment. However, we also feel that it is the most important objective evidence that we have to date that this new method of treatment is actually preventing AAA expansion and possibly rupture. Obviously, longer term follow-up is needed before these data can be put in perspective.