

# Rate of change in abdominal aortic aneurysm diameter after endovascular repair

Yehuda G. Wolf, MD,<sup>a</sup> Bradley B. Hill, MD,<sup>a</sup> Geoffrey D. Rubin, MD,<sup>b</sup> Thomas J. Fogarty, MD,<sup>a</sup> and Christopher K. Zarins, MD,<sup>a</sup> *Stanford, Calif*

**Objective:** Untreated abdominal aortic aneurysms (AAAs) enlarge at a mean rate of 3.9 mm/y with great individual variability. We sought to determine the effect of endovascular repair on the rate of change in aneurysm size.

**Methods:** There were 110 patients who underwent endovascular AAA repair at Stanford University Medical Center and who were followed up for 1 to 30 months (mean, 10 months) with serial contrast-infused helical computed tomography (CT). Maximal aneurysm diameter was determined using two independent methods: (1) measured manually, from cross-sectional computed tomography (XSCT) angiograms and (2) calculated from quantitative three-dimensional computed tomography (3DCT) data as orthonormal diameter.

**Results:** Maximal cross-sectional aneurysm diameter measured by hand (XSCT) and calculated as orthonormal values (3DCT) correlated closely ( $r = 0.915$ ;  $P < .001$ ). The XSCT-measured diameter was larger by  $2.3 \pm 3.75$  mm ( $P < .001$ ), and the 95% CI for SE of the bias was 1.85 to 2.75 mm. Preoperative aneurysm diameter (XSCT  $59.1 \pm 8.4$  mm; 3DCT  $58.1 \pm 9.3$  mm) did not differ significantly from the initial postoperative diameter. Considering all patients, XSCT diameter decreased at a rate of  $0.34 \pm 0.69$  mm/mo, and 3DCT diameter decreased at a rate of  $0.28 \pm 0.79$  mm/mo. Aneurysms in patients without endoleaks had a higher rate of decrease, an XSCT diameter by  $0.50 \pm 0.74$  mm/mo, and 3DCT diameter by  $0.46 \pm 0.84$  mm/mo. In these patients, mean absolute decrease in diameter at 6 months was  $3.4 \pm 4.5$  mm (XSCT) and  $3.3 \pm 5.9$  mm (3DCT) and at 12 months,  $5.9 \pm 5.7$  mm (XSCT) and  $5.4 \pm 5.7$  mm (3DCT). Aneurysms in patients with persistent endoleaks did not change in mean XSCT diameter, and 3DCT diameter increased by  $0.12 \pm 0.52$  mm/mo (not significant). Aneurysm diameter remained within 4 mm of original size in 68% (3DCT) to 71% (XSCT) of patients. In one patient, aneurysm diameter increased (XSCT and 3DCT) more than 5 mm. Four patients who had a new onset endoleak had a much higher expansion rate than those with a chronic endoleak ( $P < .05$ ).

**Conclusions:** The rate of decrease in aneurysm size (annualized 3.4-4.1 mm/y) after endovascular repair of AAA approximates the reported expansion rate in untreated aneurysms. However, individual aneurysm behavior is unpredictable, and the presence of an endoleak is unreliable in predicting changes in diameter. New onset endoleaks are associated with an enlargement rate greater than that of untreated aneurysms. (*J Vasc Surg* 2000;32:108-15.)

From the Division of Vascular Surgery<sup>a</sup> and the Department of Radiology,<sup>b</sup> Stanford University Hospital.

Competition of interest: TJF and CKZ own shares of Medtronic and have been paid consultants of Medtronic.

Reprint requests: Christopher K. Zarins, MD, Division of Vascular Surgery, Stanford University Hospital, Stanford, CA 94305-5642 (e-mail: [zarins@stanford.edu](mailto:zarins@stanford.edu)).

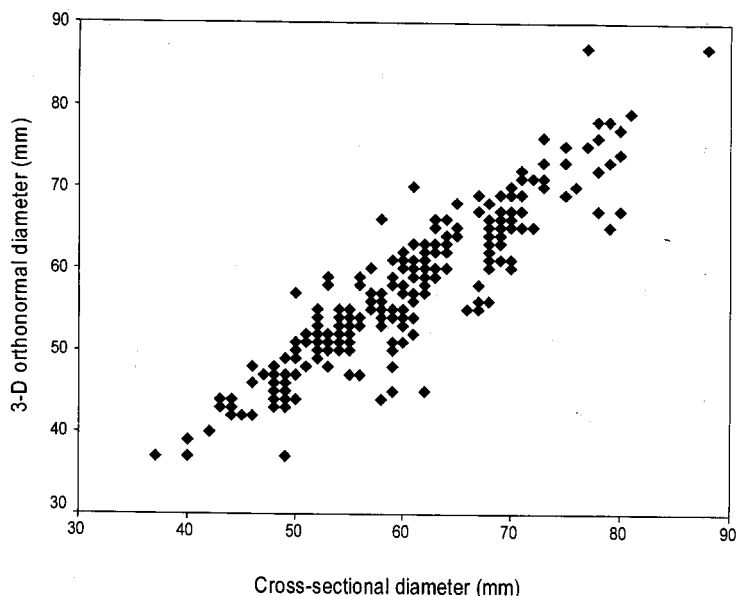
Copyright © 2000 by The Society for Vascular Surgery and International Society for Cardiovascular Surgery, North American Chapter.

0741-5214/2000/\$12.00 + 0 24/6/107754

doi:10.1067/mva.2000.107754

The natural history of abdominal aortic aneurysms (AAAs) is to enlarge and rupture. Numerous investigations have shown that the mean rate of aneurysm enlargement is between 2.6 and 6.3 mm/y.<sup>1-4</sup> However, there is a great variability in individual expansion rate, with up to 50% of aneurysms showing no change over periods of time. Individual rates vary from 0 to 10 mm/y.

Endovascular aneurysm repair (EVR) is expected to modify this progression, and at this time, the nat-



**Fig 1.** Scatterplot (n = 274) showing the maximal transverse diameter of the aneurysm by manual axial measurement (XSCT) and 3-D orthonormal values (3DCT). The values were highly correlated ( $r = 0.915$ ;  $P < .001$ ), and 83.2% of the values were within 5 mm of each other. The XSCT was larger by  $2.3 \pm 3.75$  mm ( $P < .001$ ), and the 95% limits of agreement were  $-5.2$  to  $9.8$  mm. The 95% CI for the SE of the bias was 1.85 to 2.75 mm.<sup>8</sup>

ural history of AAA after EVR is inadequately defined. A number of investigators have shown that aneurysms after EVR decrease in diameter, and some have shown that aneurysms with endoleak do not decrease in size and even expand.<sup>5-7</sup> Although small retrograde endoleaks have not been shown to increase the likelihood of rupture, some believe that the presence of an endoleak constitutes failure of an otherwise successful EVR.

The purpose of this investigation was to determine the rate of change in aneurysm diameter after EVR in the presence and absence of endoleak and relate it to the documented rate of change in untreated aneurysms. We used two methods for measuring aneurysm diameter that are based on periodic contrast-infused helical computed tomography (CT) scans.

### PATIENTS AND METHODS

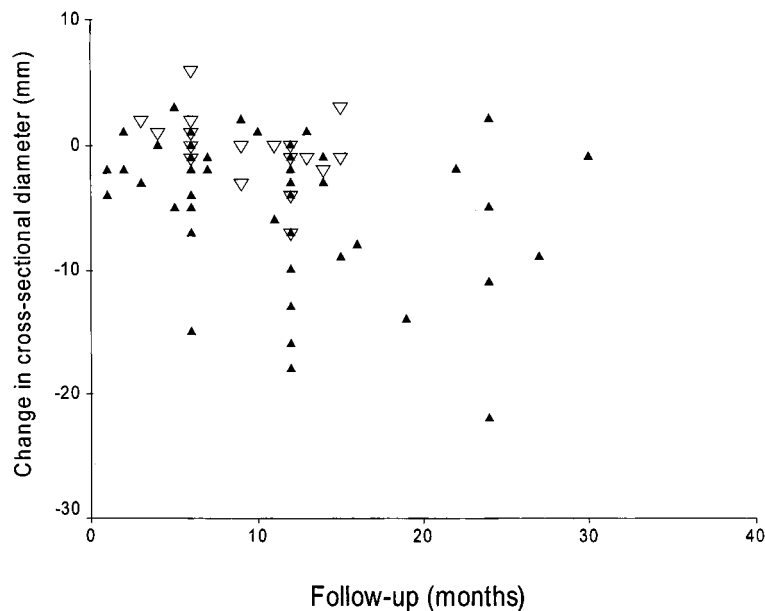
During a 3-year period, 110 consecutive patients underwent endovascular AAA repair at Stanford University Medical Center with a bifurcated modular AneuRx stent graft. All patients underwent preoperative CT angiography for quantitative assessment of aneurysm morphology, except for two patients who underwent magnetic resonance angiography because of renal failure. Postoperative

**Table I.** Direction of change of cross-sectional diameter (XSCT) in patients with radiologic follow-up of 3 months or longer

	Endoleak	No endoleak
Increase ( $\geq 5$ mm)	4.5% (1)	0% (0)
No change ( $\pm 4$ mm)	91% (20)	63% (32)
Decrease ( $\geq 5$ mm)	4.5% (1)	37% (19)
Total	22	51

CT angiography was performed within the first month, after 6 and 12 months, and once a year thereafter. After each of these examinations, the patients were seen in the outpatient clinic.

The CT angiography with 2.5 to 3 mm nominal section thickness was performed using both single and multidetector-row CT after a rapid intravenous bolus injection of iodinated contrast material with timed breath-held helical CT acquired during peak opacification. During follow-up examinations, late acquisitions (70 seconds postinjection) were added to rule out small endoleaks. Cross-sectional computed tomography (XSCT) images were reconstructed at 1.25 to 1.5 mm increments. All CT scans were acquired using the same CT angiography acquisition protocol. In addition, helical CT scans done at



**Fig 2.** Scatterplot of change in the cross-sectional maximal diameter of the aneurysm (XSCT) after EVR in patients with clinical follow-up of 3 months ( $n = 77$ ). The *empty triangles* represent patients with persistent endoleak ( $n = 22$ ), and the *black triangles* represent patients without endoleak ( $n = 55$ ). There is a wide dispersion of rates for the entire group. The graph for the orthonormal values (3DCT) exhibited similar variability. For patients with a chronic endoleak there was no linear change with time. For those without an endoleak such correlation was found ( $r = 0.321$ ;  $P = .017$ ).

Stanford University Hospital were processed with three-dimensional (3-D) reconstruction, which included shaded surface display, maximal intensity projection, and curved planar reformation. Measurements of the orthonormal maximal diameter were performed by a radiologist by constructing a median centerline through the lumen and measuring the transverse aortic diameter in a plane perpendicular to it. Manual measurements of the maximal diameter were performed by the surgeons on CT cross sections and related to the length scale on the film. On preoperative scans, the amount of thrombus within the aneurysm was graded on a discrete scale of 1 to 3 according to subjective impression of minimal, moderate, or large amount of thrombus seen on multiple scan sections. On follow-up CT scans, the presence or absence of endoleaks was determined by a senior radiologist and reviewed by a panel of radiologists and vascular surgeons. Aneurysm size was related to the state of endoleak, which was defined as positive if it persisted beyond the first postoperative month and was present for most of the patient's follow-up. In four patients a late new endoleak appeared 9 to 12 months after the procedure, and in one patient an endoleak stopped

after 12 months. In these five patients, the follow-up period was divided into two separate periods and evaluated separately.

Statistical analysis included the  $\chi^2$  test, paired and unpaired  $t$  test, calculation of Pearson correlation coefficient, and analysis of agreement according to Bland and Altman.<sup>8</sup> A  $P$  value less than .05 was considered significant.

## RESULTS

A total of 108 preoperative and 297 follow-up helical CT scans were performed on 110 patients. Mean follow-up was 10 months (range, 1-30 months), median follow-up was 9 months, and 37 patients had a follow-up of at least 1 year. The 3-D reconstruction was performed on 69 preoperative and 222 follow-up scans on 68% of the patients. The first postoperative CT scan was performed within 48 hours in 65% of patients, within 1 week in 84% of patients, and within 1 month in all patients. Maximal aneurysm diameter on cross-sectional CT (XSCT) and three-dimensional computed tomography (3DCT), including all studies, correlated closely ( $r = 0.915$ ;  $P < .001$ ) (Fig 1). The XSCT-measured diameter was larger by  $2.3 \pm 3.75$  mm ( $P < .001$ ),

the 95% limits of agreement were -5.2 to 9.8 mm, and 83.2% of measurements were within 5 mm of each other. The 95% CI for the SE of the bias was 1.85 to 2.75 mm.<sup>8</sup>

The preoperative maximal diameter was 59.1 ± 8.4 mm on XSCT and 58.1 ± 9.3 mm on 3DCT, and the initial postoperative values for the maximal diameter were not different from the preoperative values. After EVR, the mean absolute decrease in diameter at 6 months was 2.4 ± 4.2 mm (XSCT) and 2.8 ± 5.2 mm (3DCT), and at 12 months it was 4.5 ± 5.4 mm (XSCT) and 4.2 ± 5.8 mm (3DCT). The mean overall rate of decrease in aneurysm diameter was 0.34 ± 0.69 mm/mo on XSCT and 0.28 ± 0.79 mm/mo on 3DCT with considerable individual variability in the rate of change (Fig 2).

Our policy with regard to endoleaks has been to investigate and treat those that appear to be related to the graft or the attachment sites (Type 1) and observe those that are related to branch vessel retrograde flow (Type 2) unless associated with increasing size. Although we did not investigate every endoleak with arteriography, whenever CT or duplex ultrasound scanning was suggestive of an endoleak originating in the graft or at the attachment sites, we investigated and treated it. During follow-up, 13 patients underwent arteriography and treatment of an endoleak. One chronic endoleak was treated because of an increase in size.

In the absence of endoleak, aneurysm diameter decreased on XSCT at a mean rate of 0.50 ± 0.74 mm/mo and on 3DCT by 0.46 ± 0.84 mm/mo. The mean absolute decrease at 6 months was 3.4 ± 4.5 mm (XSCT) and 3.3 ± 5.9 mm (3DCT), and at 12 months it was 5.9 ± 5.7 mm (XSCT) and 5.4 ± 5.7 mm (3DCT). Among patients without an endoleak, the aneurysm diameter decreased by 5 mm or more in 37% (XSCT) and 38% (3DCT) (Tables I and II). No patient without an endoleak had an increase in aneurysm size.

There were 22 patients with a more than 3-month radiologic follow-up and a chronic endoleak. In four of these, the endoleak was shown to be Type 1; in 18 others the endoleak was probably unrelated to the graft or attachment sites. In the presence of chronic endoleak, both the axial diameter and 3-D orthonormal diameter remained unchanged (-0.04 ± 0.33 mm/mo and 0.12 ± 0.52 mm/mo, both not significant). This is in contrast to patients without endoleak who had a decrease in axial diameter ( $P < .001$ , 95% CI for difference of means 0.28-0.77 mm/mo) and 3-D orthonormal diameter ( $P < .05$ , 95% CI for difference of means 0.12-1.03 mm/mo)

**Table II.** Direction of change in orthonormal diameter (3DCT) in patients with radiologic follow-up of 3 months or longer

	Endoleak	No endoleak
Increase (≥ 5 mm)	6% (1)	0% (0)
No change (± 4 mm)	81% (13)	62% (21)
Decrease (≥ 5 mm)	12% (2)	38% (13)
Total	16	34

compared with patients with endoleak. The rate of change was unrelated to the initial size of the aneurysm or to the amount of preoperative thrombus within it. In most patients with an endoleak, the aneurysm diameter (91% XSCT, 81% 3DCT) remained within 4 mm of preoperative and initial postoperative diameter. In one patient (XSCT) and in two patients (3DCT), the aneurysm diameter decreased by 5 mm or more, and in one patient (XSCT and 3DCT) it increased in size by 6 mm compared with the initial postoperative scan.

Four patients in this series had a late endoleak: two were symptomatic, and two were identified on routine follow-up. The diameters of the two symptomatic patients decreased significantly (by 10 and 16 mm) before the onset on the endoleak. After its onset, expansion occurred by 16 mm over 20 days and by 10 mm over 10 days, and both returned to preoperative size. In one asymptomatic patient, expansion occurred by 10 mm over 6 months since the last examination to a size 9 mm greater than the preoperative one, and the fourth patient presented at the same size as the immediate postoperative test without documentation between these time points. The rate of increase under these circumstances may be between 1.7 mm/mo and 1 mm/d and is significantly higher than in patients with persistent endoleaks ( $P < .05$ ).

## DISCUSSION

The purpose of EVR of AAA is to modify the natural history of AAA and prevent rupture-associated mortality. Absence of an endoleak and decrease in aneurysm size are considered to be evidence of successful and effective repair. We have shown that after EVR, the aneurysm diameter decreases at a mean rate of 0.28 to 0.34 mm/mo, according to two different methods of measurement. Patients without evidence of an endoleak experience a decrease in aneurysm size, which is significantly greater than in patients with an endoleak.<sup>5-7</sup>

In patients without endoleaks, the rate of

decrease in the mean aneurysm diameter was 0.46 to 0.50 mm/mo, similar to the rate of 0.41 to 0.44 mm/mo observed by the Utrecht and the Malmö groups.<sup>6,9</sup> This rate was unrelated to the amount of preoperative thrombus, as reported by Matsumura et al<sup>10</sup>; was unrelated to initial aneurysm size; and did not appear to stop after 18 months as suggested by Malina et al.<sup>11</sup> However, the rates were individually variable, and more than half the aneurysms did not decrease in size at all. The reasons for this variability after EVR are unclear. This is reminiscent of the variable expansion rate of untreated AAA, where the mean expansion rate is well documented but individual aneurysm behavior is quite unpredictable, and 24% or more of aneurysms do not expand at all for prolonged periods of time.<sup>4</sup> Although it has been shown that an absence of endoleak is associated with reduction in the pulsatile wall motion of the aneurysm sac,<sup>11</sup> experimental data from dogs showed that closure of an endoleak by coil embolization and thrombosis was not associated with a reduction of pressure within the aneurysm sac.<sup>12</sup> Whether this individual variability after endovascular exclusion is related to characteristics of the aneurysm or to the degree of pressure reduction within the aneurysm sac remains to be evaluated.

In patients with endoleaks, the mean cross-sectional diameter (XSCT) and the mean orthonormal diameter (3DCT) did not change significantly. This was significantly different from those without an endoleak but also clearly different from the enlargement one observes in untreated aneurysms. In this respect it is clear that even in patients with an endoleak, aneurysm behavior, on average, has been modified, as observed also by Matsumura and Moore.<sup>13</sup> However, the follow-up period in this study is short, and additional data will be acquired with time.

Different types of endoleaks may vary in clinical significance. Although we did not investigate every endoleak, our impression is that most chronic endoleaks (70%-80%) were related to branch vessel retrograde perfusion (Type 2). In patients with endoleaks, individual aneurysm behavior varied considerably. Most of these aneurysms remained unchanged, whereas a minority decreased or increased in size. Although it appears that aneurysms with endoleaks that decrease in size are protected and those that increase in size require treatment, the dilemma lies with those aneurysms with an endoleak that remains stationary. How do these aneurysms differ from untreated ones? How do they differ from the large proportion of aneurysms after EVR without an endoleak that do not decrease in size? Until

direct assessment of pressure within the aneurysm sac becomes feasible, these issues will remain conjectural. Currently, aneurysm size remains the best guide for identifying those patients after EVR who need evaluation and consideration of further therapeutic intervention.

Late onset Type 1 endoleaks may induce very rapid aneurysm expansion. When this occurs after a significant decrease in aneurysm size, reexpansion to baseline size takes place. This implies that true remodeling of the aneurysm sac does not occur and that the structural components of the aneurysm wall retain their original size.

In this study we compared two different methodologies, the manual measurement from cross-sectional CT film (XSCT) and orthonormal measurement from 3-D reconstruction (3DCT). Although the orthonormal measurement has a significant theoretical advantage because it is less affected by changes in aortic tortuosity and angulation, it is technician dependent. Software has been developed to calculate orthonormal diameter automatically, free of observer interaction, but currently, it is not widely available.<sup>14</sup> We found here that the manually measured and 3-D derived values were roughly equivalent. The small but significant difference between the two may be related to obliquity in some of the manual measurements or to differences in defining the outer edge of the aortic wall.

We conclude that after EVR of AAA, aneurysms decrease in diameter at an overall rate of 4 mm/y. Aneurysms in patients without evidence of endoleak decrease at a rate of 6 mm/y, and most aneurysms in patients with chronic Type 2 endoleaks do not change in size. However, individual aneurysm behavior is unpredictable, and the presence or absence of an endoleak is not consistently reliable in predicting changes in diameter. Monitoring changes in aneurysm size may be more important.

## REFERENCES

1. Bernstein EF, Dillely RB, Goldberger LE, Gosink BB, Leopold GR. Growth rates of small abdominal aortic aneurysms. *Surgery* 1976;80:765-73.
2. Cronenwett JL, Sargent SK, Wall MH, Hawkes ML, Freeman DH, Dain BJ, et al. Variables that affect the expansion rate and outcome of small abdominal aortic aneurysms. *J Vasc Surg* 1990;11:260-9.
3. Limet R, Sakalihassan N, Albert A. Determination of the expansion rate and incidence of rupture of abdominal aortic aneurysms. *J Vasc Surg* 1991;14:540-8.
4. Wolf YG, Thomas WS, Brennan FJ, Goff WG, Sise MJ, Bernstein EF. Computed tomography scanning findings associated with rapid expansion of abdominal aortic aneurysms. *J Vasc Surg* 1994;20:529-38.

5. Armon MP, Yusuf SW, Whitaker SC, Gregson RH, Wenham PW, Hopkinson BR. Thrombus distribution and changes in aneurysm size following endovascular aortic aneurysm repair. *Eur J Vasc Endovasc Surg* 1998;16:472-6.
6. Resch T, Ivancev K, Lindh M, Nyman U, Brunkwall J, Malina 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;28:242-9.
7. May J, White GH, Yu W, Waugh RC, Stephen MS, Harris JP. A prospective study of changes in morphology and dimensions of abdominal aortic aneurysms following endoluminal repair: a preliminary report. *J Endovasc Surg* 1995;2:343-7.
8. Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986;1:307-10.
9. Broeders IA, Blankensteijn JD, Gvakharia A, May J, Bell PR, Swedenborg J, 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.
10. Matsumura JS, Pearce WH. Preexisting thrombus and aortic aneurysm size change after endovascular repair. *J Surg Res* 1999;81:11-4.
11. Malina M, Länne T, Ivancev K, Lindblad B, Brunkwall J. Reduced pulsatile wall motion of abdominal aortic aneurysms after endovascular repair. *J Vasc Surg* 1998;27:624-31.
12. Marty B, Sanchez LA, Ohki T, Wain RA, Faries PL, Cynamon J, et al. Endoleak after endovascular graft repair of experimental aortic aneurysms: does coil embolization with angiographic "seal" lower intraaneurysmal pressure? *J Vasc Surg* 1998;27:454-62.
13. Matsumura JS, Moore WS. Clinical consequences of periprosthetic leak after endovascular repair of abdominal aortic aneurysm. *Endovascular Technologies Investigators. J Vasc Surg* 1998;27:606-13.
14. Rubin GD, Paik DS, Johnston PC, Napel S. Measurement of the aorta and its branches with helical CT. *Radiology* 1998;206:823-9.

Submitted Sep 28, 1999; accepted Feb 15, 2000.

---

## DISCUSSION

**Dr Victor Bernhard** (Palisade, Colo). The change in aneurysm morphology after endovascular repair is an issue of major importance for the development of appropriate follow-up regimens for patients managed by this form of therapy. Dr Wolf and his colleagues have clearly demonstrated that progressive enlargement of the aneurysm is halted after deployment of an endograft, that diameter reduction is greater and occurs more often if the AAA is effectively excluded from the circulation, and that failure to achieve exclusion is associated with either no change or an increase in cross section measurements. They have further demonstrated that these trends are not absolutely consistent. Reduction in diameter does not always occur and on occasion may increase when postoperative imaging appears to indicate effective exclusion. Persistent endoleak is not always associated with an increase in diameter, and the diameter in some instances may decrease in the presence of an endoleak. These findings confirm the investigations of others and provide additional information that supports our understanding or misunderstanding of the natural history of AAA after endograft therapy. The data presented in this study also emphasize the shortcomings of current postoperative imaging techniques employed to demonstrate the presence and source of endoleaks and strongly suggest that we cannot rely entirely on a search for endoleaks as the primary method for judging the success or failure of endograft deployment in a given patient. This problem assumes greater importance as we become aware of an increasing number of reports of progressive AAA enlargement when repeated imaging studies including angiography fail to demonstrate any evidence of endoleak. Therefore, I concur with the essayists that protection against rupture may be more reliably determined by serial measurements of changes in AAA size over time.

The techniques used for aortic measurement must be consistent and reproducible if they are to be employed for routine follow-up. We would all agree that currently the most accurate method for determining AAA diameter is measurement of the slice perpendicular to the flow line at the widest portion of the aneurysm obtained from a multi-planar reconstruction of a spiral CT. Since this methodology is somewhat more arduous than simple measurements from axial projections, it is nice to know that these two techniques were comparable in your hands. Nevertheless, the difference between the two techniques was greater than 5 mm in 17% of the subjects in their study. For the individual patient this may have significant implications regarding selection of an appropriate follow-up management strategy. Could you provide us with the details of how measurements were actually obtained? Although this may appear to be nitpicking, precise methodologies need to be prescribed if they are to become standardized techniques that can be reliably employed by others. For instance, were your manual axial cross section measurements performed with calipers possibly with optical enhancement, the quick and dirty use of a marked cardboard held against the CT scale at the side of the image, or by electronic calibration from a digitized image? Did you measure the largest or the smallest diameter at the widest point of the aneurysm? Since, as you noted, measurements are technician dependent, did you make repeat measurements to determine the standard error for intraobserver and interobserver differences? What size change did you require to classify aneurysms as enlarging or diminishing, and what degree of change would you recommend for selecting the most appropriate follow-up management strategy?

Did you consider other methods such as changes in aneurysm volume for evaluating aneurysm size? The group from Utrecht, under the guidance of Bert Eikelboom, recently presented their findings comparing diameter with volume determination. They found a lack of correlation between diameter and volume changes in 37% of their patients and recommended volume as the gold standard. It is quite laborious to measure volume from a spiral CT image. However, with advances in computerized imaging technology, especially noteworthy from your institution, volume determinations may become a practical reality and replace diameter measurements.

How many of the endoleaks identified in your patients were Type I or Type II, and were you able to demonstrate a difference in the rate and extent of change in diameter between Type I and Type II endoleaks?

Finally, the rapid expansion of aneurysm diameter in two of your patients with late onset endoleaks is disturbing. Were these Type I or Type II? Could you speculate, or do you have data regarding changes in the aneurysm wall after endograft exclusion that might account for this phenomenon?

The authors have presented important information regarding the natural history of abdominal aortic aneurysm after endograft therapy. Their study emphasizes the need to define appropriate end points as the basis for a lifelong surveillance regimen for all aortic endografts. Their investigation is clearly presented, and I recommend it for your thoughtful consideration.

I would like to thank the program committee for offering me the opportunity to discuss this excellent and thought-provoking manuscript.

**Dr Yehuda Wolf.** Dr Bernhard, thank you for your comments and your questions. I will try to answer the questions in order.

First of all, I would like to mention that three-dimensional measurement of diameter was meant to corroborate the findings regarding changes in aneurysm size, and actual comparison and validation of the three-dimensional measurements versus the manual or traditional way of axial measurement would require a separate study paper dedicated specifically to this end. Therefore, there are aspects that were not discussed here.

Regarding the actual method of manual measurement, we usually used calipers. We did not use optic magnification. The axial diameter was measured at the largest aneurysm cross section, and both maximal and minimal diameter at that level was measured and recorded.

The method of measurement in the 3-D laboratory is also important. The radiology technicians scroll up and down the median centerline and decide where the largest cross section is by examining it on the screen. They also measure the largest and smallest diameter.

When we correlated the diameters, we ran correlations of the smallest and largest diameters, and the best correlation was still the largest diameter on the axial measurement versus the largest diameter on the three-dimensional image even though you could intuitively think otherwise. The problem with the axial measurement is that occasionally,

with the large diameter, you are measuring obliquity, but sometimes aneurysms are truly oval, and the differentiation is very hard.

Regarding size change, we reported here on the threshold for size change as 3 mm. We also included the 5 mm difference in the manuscript.

With regard to volume measurements, this topic is still open, and we are currently in the process of evaluating the importance of volume changes, which are reported regularly by the 3-D laboratory monitoring for evidence of aneurysm progression.

Regarding the type of endoleaks, two of the 22 endoleaks in the last slide with longer follow-up appeared to be Type I. They were not regularly investigated by angiography, but this appeared to be the case on duplex ultrasound and CT.

The late onset endoleaks were all major Type I endoleaks. All of these were repaired with a reintervention and insertion of an extender cuff. Consequently we do not have any histology of the aneurysm wall.

**Dr Robert Rutherford** (Silverthorne, Colo). I have a couple of questions. The first one has to do with the practical application of this type of data.

Can you take the data on cases without endoleak and, having plotted them out against time with a 95% confidence limit, develop something that serves as "trigger" point for intervention, so that if a case falls outside of those limits, it serves as an indication for intensive search for an endoleak, and intervening to control it?

Have these data helped you with your "trigger," or what is your trigger? Is it a certain diameter increase, or would the lack of a decrease after a certain length of time serve in the same way?

Secondly, in terms of the data presented, I would like a little additional background data. What percent of the total cases had an increase in diameter, and what percent had no decrease over time, and do these correlate with the incidence of Type I endoleak? And what was the percent of initial and late Type I endoleaks?

**Dr Wolf.** First of all, I would like to say that we are still limited in this type of evaluation by the length of follow-up and the number of cases. Clearly this is an early study, and with time things may declare themselves better.

As the data stand now, it appears that patients without endoleak either remain at the same size or decrease in size, and as far as I can tell, nobody can figure out who does what.

As indicated in the manuscript, we evaluated thrombus size by crude grading, and change in size not related to the thrombus content of aneurysm. This was also shown by Matsumura et al. So we know that aneurysms can either stay the same for a long period, up to 2½ years, or decrease in size, and so far, we have no predictor for this difference.

As for defining outliers, it is pretty clear that an aneurysm without an endoleak that increases in size requires some type of attention. Increases in size by more than 5 mm, I would guess, are significant.

**Dr Gregory Moneta** (Portland, Ore). What happens to the iliac arteries in patients with a tube stent graft?

Do the iliac arteries remain the same, decrease, or increase in caliber?

**Dr Wolf.** There were no tube grafts in this series. All of these are bifurcated grafts.

**Dr Moneta.** In other series, do you have any idea when they did use tube grafts?

**Dr Wolf.** I cannot really comment about that.

**Dr Wesley Moore** (Los Angeles, Calif). Dr Wolf, that was a beautiful presentation. Since your experience and results very much parallel our own, I agree with your conclusions.

The reason that I rise is to ask if you have seen in your own series a phenomenon that is now being recognized more frequently, particularly in some of the reports from Europe; that is, aneurysm sac enlargement in the absence of demonstrable endoleak.

I have a couple of examples of these in my own series. We used to believe that there must be an underlying

endoleak present, but we failed to image it. In fact, there has now been an experience in explanting some of these in the European experience in which they find no endoleak and still mysteriously the aneurysm has enlarged. This has led to a new term called "endotension." Investigators were able to measure an increase or a maintenance of systemic pressure in what is seemingly a thrombosed aneurysm sac. The reason for this is not clear. I wonder if you have seen this in your own series and can speculate on the mechanism.

**Dr Wolf.** No, in this series we have not. I am aware of reports by the Malmö group who measured the pulsatility of aneurysm wall with endografts, and there are differences even if they do not have an endoleak. Also, the Montefiore group showed that even after embolization of endoleaks in dogs, you can still have a pressurized thrombus, and this conceivably can cause increase in aneurysm diameter. In our own series, thus far, we have not seen aneurysm enlargement without an endoleak.