Volume regression of abdominal aortic aneurysms and its relation to successful endoluminal exclusion

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Objectives: **Evaluating the success of endoluminal repair of abdominal aortic aneurysms (AAAs) is frequently based on diameter measurements and determining the presence of endoleaks. The use of three-dimensional volumetric data and observation of morphologic changes in the aneurysm and device have been proposed to be more appropriate for postdeployment surveillance. The purpose of this study was to analyze the long-term volumetric and morphologic data of 161 patients who underwent endovascular AAA exclusion and to assess the utility of volume measurements for determining successful AAA repair.**

Methods: **Patients with spiral computed tomography scans obtained preoperatively, within the first postoperative month, at 6 months, and annually thereafter, were included in this analysis. Computerized interactive three-dimensional reconstruction of each AAA scan was performed. Total aneurysm sac volume was measured at each time interval (mean preoperative volume 169.0 ± 78.5 mL), and the significance of volume changes was determined by mixed linear modeling, a form of repeated measures analysis, to account for longitudinal data clustered at the individual level. Sixty-two patients (38%) developed endoleaks at some time during follow-up—15 type I leaks, 45 type II leaks, and 2 type III leaks. The patients with type I and type III leaks were treated with cuffs, and the type II leaks were treated either with observation, side-branch embolization, or required open conversion.**

Results: **Aneurysm sac volume increased slightly at 1-month follow-up (+3.3%), and then decreased steadily to −12.9% at 5 years ($P < .0001$). This effect remained unchanged after controlling for the three device types used in our study population. Patients who did not exhibit an endoleak (n = 99) showed a significant decrease in aneurysm volume across the entire follow-up duration when compared with those who did exhibit an endoleak (n = 62) ($P < .0001$). The presence of a 10% or greater decrease in volume at 6 months demonstrated a sensitivity of 64%, a specificity of 95%, a positive predictive value of 95%, a negative predictive value of 62%, and an accuracy of 75% for predicting primary clinical success defined by successful deployment of the device; freedom from aneurysm- or procedure-related death; freedom from endoleak, rupture, migration, or device malfunction; or conversion to open repair.**

Conclusions: **Volumetric analysis may be used to predict successful endoluminal exclusion of AAAs. Volume regression appears to be device-independent and should be expected in most clinically successful cases. The presence of volume increases in the first 6 months is suspicious for an endoleak that is pressurizing the aneurysm sac and heralds the need for closer evaluation and possible intervention. A volume decrease of 10% or greater at 6 months and continuing regression over time is associated with successful endovascular repair. (J Vasc Surg 2003;38:1254-63.)**

The advent of endovascular techniques has revolutionized the way surgeons approach the treatment of abdominal aortic aneurysms (AAAs). The feasibility of endoluminal stent-graft placement with a multitude of devices has been well documented, and newer prostheses are rapidly being developed.[1-6] What remains unclear is the optimal mechanism for assessing long-term device performance and procedural success. As the main goal of endoluminal graft repair is to prevent aneurysm rupture and its high morbidity, accurate postoperative surveillance parameters need to be defined that predict successful outcomes and detect the complications unique to endovascular interventions.

Shrinkage of the excluded aneurysm sac combined with freedom from endoleak are generally felt to be indicative of depressurization and a successful repair, and most reported series focus on sac diameter changes and the presence or absence of endoleaks.[7-12] The use of maximal sac diameter to track postoperative changes, however, has been challenged by recent studies.[13-16] Volumetric analysis has been proposed to be more appropriate, more accurate, and more reliable for determining three-dimensional (3-D) morphologic changes after endovascular repair. This follow-up approach relies on precise imaging modalities and highlights the importance of postoperative surveillance of these patients.[17]

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Three-dimensional spiral computed tomography (CT) scanning has emerged as the gold standard and most efficient modality to provide the most pertinent and accurate information of AAA morphology.[17,18] Techniques to measure volumes from spiral CT data typically require dedicated workstations with complex algorithms and sometimes time-consuming and tedious manual measurements, but recently advanced imaging software has allowed for the automation of such calculations.[15,16]

The purpose of this study was to measure and analyze the long-term volumetric and morphologic data from a single center's series of patients undergoing endovascular AAA repair and to assess the utility of these measurements for determining successful AAA exclusion. Volume changes were specifically analyzed in the presence and absence of endoleaks, before and after secondary intervention, and in patients with device-related morbidity.

METHODS

Patients included in this study were involved in multiple prospective Food and Drug Administration–approved US trials performed at our institution between June 1996 and June 2001. These trials included the use of the AneuRx bifurcated prosthesis (Medtronic AVE, Santa Rosa, Calif), Talent bifurcated prosthesis (Medtronic World Medical, Sunrise, Fla), and Endologix bifurcated device (Endologix, Irvine, Calif). Patients were enrolled in a protocol approved by the Institutional Review Board of our medical center, and signed consents were obtained for the use of the investigational devices and the imaging surveillance protocols.

Of 269 consecutively treated patients available for review, 177 (66%) patients had complete data sets of spiral CT imaging at all predeployment intervals and at least one postdeployment interval. This cohort constituted our study population. Spiral CT data sets included preoperative scans obtained within 30 days of surgery (n = 177), and postoperative scans at 1 month (n = 169), 6 months (n = 161), 1 year (n = 152), 2 years (n = 119), 3 years (n = 77), 4 years (n = 38), and 5 years (n = 11). Patients enrolled in the trials who did not have available CT data at the designated time intervals were excluded in order to enhance the accuracy and comparability of the data.

All CT imaging was performed with a PQ 6000 single-detector spiral scanner (Philips Medical Systems, Best, The Netherlands). Parameters for the acquisitions were helical/spiral mode with settings of slice reconstruction at 2 or 3 mm, collimation 5, table speed 10 mm/s, pitch 1:1.5, and 120-kV, 230- to 250-mA tube current. Precontrast and postcontrast acquisitions were obtained with 140 mL of Omnipaque (VHA Plus, Princeton, NJ) solution (concentration 300-mg iodine/mL) injected at a flow rate of 3 mL/sec at 300 psi. The raw data generated from the spiral CT were then processed and reconstructed into a 3-D interactive model with Preview software (Version 2.0; Medical Media Systems, West Lebanon, NH).

Analyses of sequential 3-D images at each designated time interval for each patient were performed retrospectively in a blinded fashion without knowledge of individual outcomes by a panel of two surgeons (J.T.L and I.N.A) in conjunction with a radiologist (I.W.). The measurements generated were not available for the decision-making and postoperative care of the patients in the study population. All measurements were obtained by following standardized definitions established by the Lifeline Registry of Endovascular Aneurysm Repair.[19] These guidelines include 17 measurements for preoperative scans and 12 measurements for postoperative scans, which resulted in 11,733 measurements among the 177 patients included in this analysis. For the purposes of this study, we focused on total AAA volume, which was measured from the slice immediately below the lowest renal artery (D2a) to the slice at the aortic bifurcation (Fig 1). This volume is composed of luminal blood flow, thrombus, calcification, stent graft, and endoleak, if present. Aneurysm sac diameter was obtained after locating the maximum cross-sectional diameter, with all measurements made in a plane perpendicular to the centerline of the aortic lumen rather than in slices acquired perpendicular to the body of the patient, as is the case with conventional axial CT.[17]

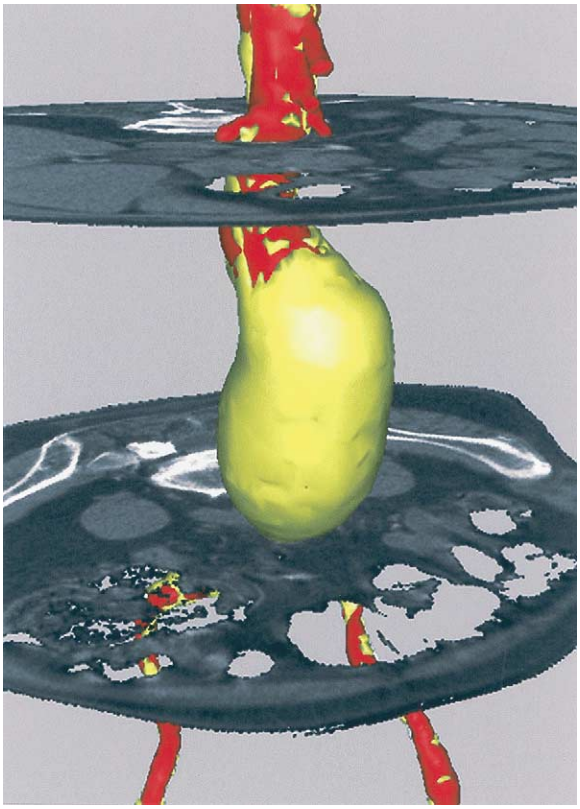

Fig 1. Interactive 3-D model based on spiral CT data demonstrates AAA volume measurement from the slice immediately below the lowest renal artery to the slice at the aortic bifurcation.

Repeated measures analysis was performed with a mixed linear model to account for longitudinal data clustered at the individual level.[20] This was done in order to

Table I. Patient demographics

Comorbid factors	*N = 177*	*%*
ASA ≥ 3	172	97
Male	154	87
Hypertension	134	76
Age > 70	127	72
Smoking > 10 pack-years	92	52
History of myocardial infarction	34	19
Diabetes mellitus	27	15
Chronic renal failure (creatinine > 1.4)	25	14
Congestive heart failure	24	14
Unstable angina	18	10

Table II. Preoperative AAA measurements

	All patients (n = 177)	*AneuRx (n = 103)*	*Talent (n = 44)*	*Endologix (n = 30)*
D2a (mm) Neck diameter	24.3 ± 3.6	22.9 ± 2.6*	27.8 ± 3.3*	23.4 ± 3.1*
H1 (mm) Neck length	30.5 ± 14.3	32.7 ± 14.8	26.1 ± 15.9	29.7 ± 13.5
D3 (cm) Max diameter	5.7 ± 1.0	5.7 ± 0.9	5.5 ± 0.8	6.1 ± 1.2
Angle (degrees)	37 ± 15	36 ± 14	37 ± 18	39 ± 17
Volume (mL)	169 ± 78	170 ± 73*	194 ± 98*	128 ± 38*

*Significant difference between devices ($P < .05$).

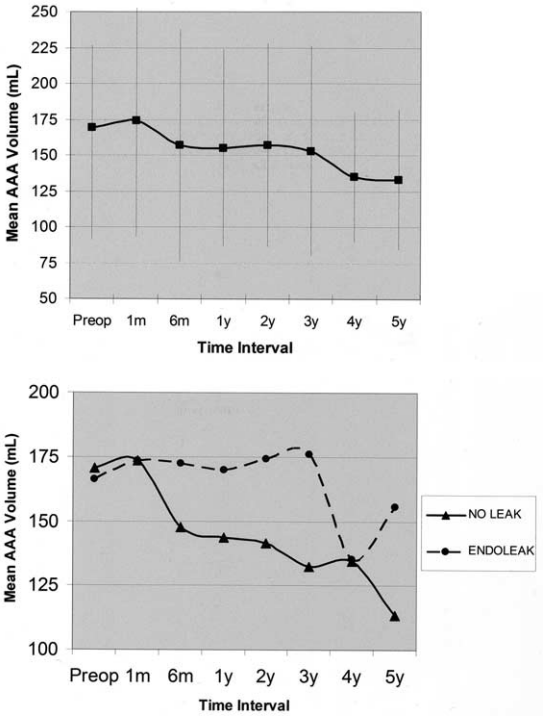

Fig 2. (**A**) AAA volume regression over each time interval for all 177 patients (lines reflect 95% confidence intervals). (**B**) Mean changes in AAA volume for 113 patients without endoleak and 64 patients with endoleak.

appropriately assess for change in AAA volume and to control for confounders, including the type of graft used during surgery, the occurrence of endoleak during the postoperative period, and, if an endoleak occurred, the type of secondary intervention. Endoleaks were identified on postoperative imaging surveillance with CT angiography and classified as type I (proximal or distal attachment zone), type II (collateral perfusion), or type III (graft disconnection).[21,22] The Wilcoxon rank sum test, Kruskal-Wallis test, or Fisher exact test was used to test for differences between groups, where appropriate. Measurements are presented as mean ± SD, and 95% confidence intervals (CIs) are reported when applicable. All data were entered into an electronic database (Microsoft Excel; Microsoft Corporation, Redding, Wash), and statistical analyses were performed with Systat 8.0 (SPSS Inc, Chicago, Ill) and SAS Version 8.2 (SAS Institute Inc, Cary, NC).

RESULTS

The medical records of the 177 patients from our prospectively maintained database were reviewed; patient demographics are shown in Table I. Of these, 154 (87%) were men, and the mean age was 74 (range, 46 to 96) years. Over a mean follow-up time of 39 (range, 1 to 68) months, 904 spiral CT scans were performed in the 177 patients and the 3-D models available for analysis, including 103 AneuRx, 44 Talent, and 30 Endologix patients. Stent grafts were successfully placed in all 177 patients with a 30-day mortality of 3% (95% CI 1% to 7%). Mean preoperative AAA volume was 169 ± 78 mL (range, 64 to 539), and sac diameter was 5.7 ± 0.9 cm (range, 4.1 to 9.7). Table II shows the breakdown of preoperative AAA measurements, and only the infrarenal neck diameter ($P = .02$) and AAA volume ($P = .0006$) were statistically different between the device cohorts.

Volume changes in all 177 patients are graphically represented in Fig 2. After an initial mean increase of 4.6 mL, there was an 11.5-mL decrease by 6-month follow-up (n = 161) and an annual mean decrease of 7.3 mL/year out to 5 years. When expressed more appropriately as percent change in AAA volume (Table III), comparisons during the follow-up intervals demonstrated significant differences in AAA volume change at each interval, including 1 month (+3.3%, $P < .0001$), 6 months (−2.6%, $P < .0001$), 1 year (−5.8%, $P < .0001$), and 5 years (−12.9%, $P < .0006$).

Patients treated with AneuRx devices showed a 9.4% volume decrease at 6 months, those treated with Endologix devices showed an 8.1% decrease, and those treated with Talent devices showed a 5.7% decrease (Fig 3). No significant difference in volume change was found between the devices after controlling for them in the analysis ($P = .2$). Patients without endoleak had volume regression at

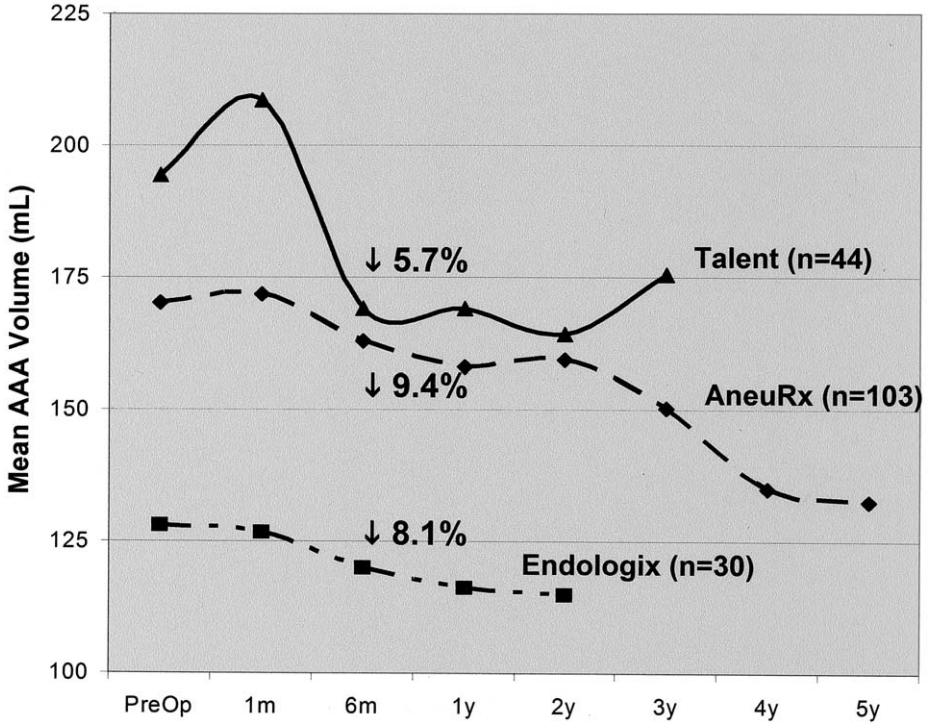

Fig 3. AAA volume regression separated into patients receiving AneuRx (n = 103), Talent (n = 44), and Endologix (n = 30) bifurcated stent grafts.

Table III. Mean percent change of aneurysm sac volume compared to preoperative measurement

	1 month (n = 169)	*6 month (n = 161)*	*1 year (n = 152)*	*2 year (n = 119)*	*3 year (n = 72)*	*4 year (n = 38)*	*5 year (n = 11)*
All (n = 177)	+3.3%	−2.6%	−5.8%	−6.7%	−7.3%	−7.1%	−12.9%
No leak (n = 115)	+2.3%	−7.6%	−12.9%	−16.7%	−19.0%	−16.8%	−24.9%
Endoleak (n = 62)	+4.9%	+4.9%	+4.0%	+4.9%	+5.3%	+4.9%	+1.6%

6-month follow-up of −7.6% and continued annual regression from −12.9% to −24.9% out to 5 years. This subset of patients (n = 99) showed a significant decrease in volume change across the entire follow-up duration when compared with those who exhibited an endoleak or required secondary intervention ($P < .0001$). When the type of device and the occurrence of endoleak were simultaneously controlled for, these findings did not change.

Type I endoleaks occurred in 15 patients (9%) and were repaired with 14 proximal cuffs and 1 distal cuff (Table IV). Seven patients had their endoleaks identified and secondary procedures performed within the first 6 months (mean = 4 months). The other eight type I endoleak patients presented much later at a mean postoperative time of 35 (range, 15 to 50) months. Volume changes for early and late type I endoleak patients are shown in Fig 4, *A*, which demonstrates volume increases of +7.3% and +15.6%, respectively, up to the time of the secondary intervention. After the appropriate cuff was placed, the early type I endoleak patients exhibited volume regression of −5.5% 2 years later, and the late type I endoleak patients showed a −14.7% volume decrease 1 year later (Table V).

Type II endoleaks were found in 45 patients (25%) and, more frequently, in patients treated with AneuRx devices (32%) versus Talent (18%) and Endologix (13%) ($P = .03$). Eight patients with type II endoleaks were treated with inferior mesenteric artery (IMA) or lumbar embolization during the follow-up period (range, 4 to 32 months). One of these patients underwent multiple embolizations but had persistent aneurysm enlargement and required open conversion at 21 months.[23] Volumetric analysis of the remaining patients who underwent embolization (n = 7) revealed a volume increase of +14.2% at 6 months. Postembolization volume changes were small (+0.9% when compared with pre-embolization volume), even 2 years after the intervention (Table V). The patients treated with embolization had a higher incidence of IMA-lumbar type II leaks (75%).

Table IV. Endoleaks, secondary procedures, and mortality

Complication (%)	*Device*	*No. of patients (%)*	*Treatment/Outcome*
Early Type I endoleak (4%)	AneuRx	n = 1 (1%)	Proximal cuff 6 m
	Talent	n = 3 (7%)	Proximal cuff 1 w, 3 m, 7 m
	Endologix	n = 3 (10%)	Proximal cuff 2 m, 2 m, 7 m
Late Type I endoleak (5%)	AneuRx	n = 6 (6%)	Distal cuff 24 m, Proximal cuff 31 m, 40 m, 46 m, 48 m, 50 m
	Talent	n = 1 (2%)	Proximal cuff 28 m
	Endologix	n = 1 (3%)	Proximal cuff 15 m
Type II endoleak (25%)	AneuRx	n = 33 (32%)	Observation (n = 23) Embolization (n = 6) Rupture/conversion (n = 2) Open conversion (n = 2)
	Talent	n = 8 (18%)	Observation (n = 7) Embolization (n = 1)
	Endologix	n = 4 (13%)	Observation (n = 4)
Type III endoleak (1%)	AneuRx	n = 2 (2%)	Modular dislocation (cuff 16 m, 24 m)
Secondary procedure without endoleak (2%)	AneuRx	n = 1 (1%)	Rupture (26 m)
	Talent	n = 2 (5%)	Migration (cuff 25 m) Wireform fracture (cuff 19 m)
30-day mortality (3%)	AneuRx	n = 1 (1%)	Rupture
	Talent	n = 3 (7%)	Ischemic bowel (n = 2) Myocardial infarction
	Endologix	n = 1 (3%)	Myocardial infarction

Thirty-four patients with type II endoleaks were followed until spontaneous resolution, which occurred by 6 months in 23 patients (68%). These patients typically had lumbar-lumbar or small single-channel leaks (91%), as opposed to IMA-lumbar leaks. Of all observed patients, there was an increase in volume of +4.1% at the 6-month scan, and regression of −7.1% 2 years later, without any clinical sequelae.

Two patients with type II endoleaks experienced ruptures in the postoperative period (20 m and 24 m), including one who refused to follow-up and another who refused intervention to treat a patent accessory renal artery that was not excluded by the stent graft.[24] The four patients who had ruptures or required open conversions had an increasing AAA volume of +22.9% at 6-month follow-up and +44.7% immediately before their interventions. Figure 4, *B* summarizes the volume changes for the three groups of type II endoleaks. The volumes for those patients who had ruptures increased significantly, whereas those who either were observed or underwent embolization remained relatively unchanged. There was no statistically significant difference between those who were observed and those who underwent embolization ($P = .1$ at 6 months).

Two type III endoleaks were discovered at 16 and 24 months, occurring when there was modular disconnection of the main body from an iliac limb, both in patients treated with AneuRx devices. Their stent grafts were relined with cuffs immediately after the diagnosis was made. Interestingly, they showed a −30% volume decrease at 6 months when compared with baseline, but they had a +7.1% interval increase in the ensuing year, and then they showed a −22.3% regression after the secondary intervention.

Finally, one patient who suffered AAA rupture and two patients who required secondary procedures did not exhibit endoleaks. Cuffs were placed in a patient with significant stent-graft migration and one with a wireform fracture.[25] These patients exhibited modest volume regression, and their complications were not predictable from volumetric analysis. In the patient who experienced a rupture without a demonstrable endoleak, which occurred very early in our experience, AAA volume had increased at each interval by large amounts (+10% at 6 months and +15% at 1 year), with only modest increases in sac diameter (+1.2 mm and +2.3 mm, respectively).

Analysis of sac diameter measurements for the entire study population revealed a mean shrinkage of 0.7 mm at 6 months, 2.4 mm at 1 year, 3.3 mm at 2 years, 3.9 mm at 3 years, 4.4 mm at 4 years, and 7.4 mm at 5 years ($P < .00001$) (Fig 5). No statistically significant difference was observed between patients with and without endoleaks after controlling for these groups ($P = .2$) Out of the 64 patients that either had endoleaks or required secondary intervention, 7 patients (11%) had sac shrinkage >5 mm by diameter, with 58 patients (89%) showing sac diameter increases or stability, 4 patients demonstrating sac shrinkage of at least 5% by volume (6%), and 61 patients showing sac increases or stability by volume (94%).

A 10% reduction in volume at 6 months identified primary clinical success (defined as successful deployment of the endovascular device and freedom from procedure- or device-related death, endoleak, rupture, conversion, migration, device failure, or need for secondary intervention[26]) with a sensitivity of 64%, a specificity of 95%, a positive predictive value of 95%, and an accuracy of 75%. Similar analyses were performed with two-dimensional diameter decreases of >5 mm at both 6-month and 1-year postoperative intervals, and the results are summarized in Table VI.

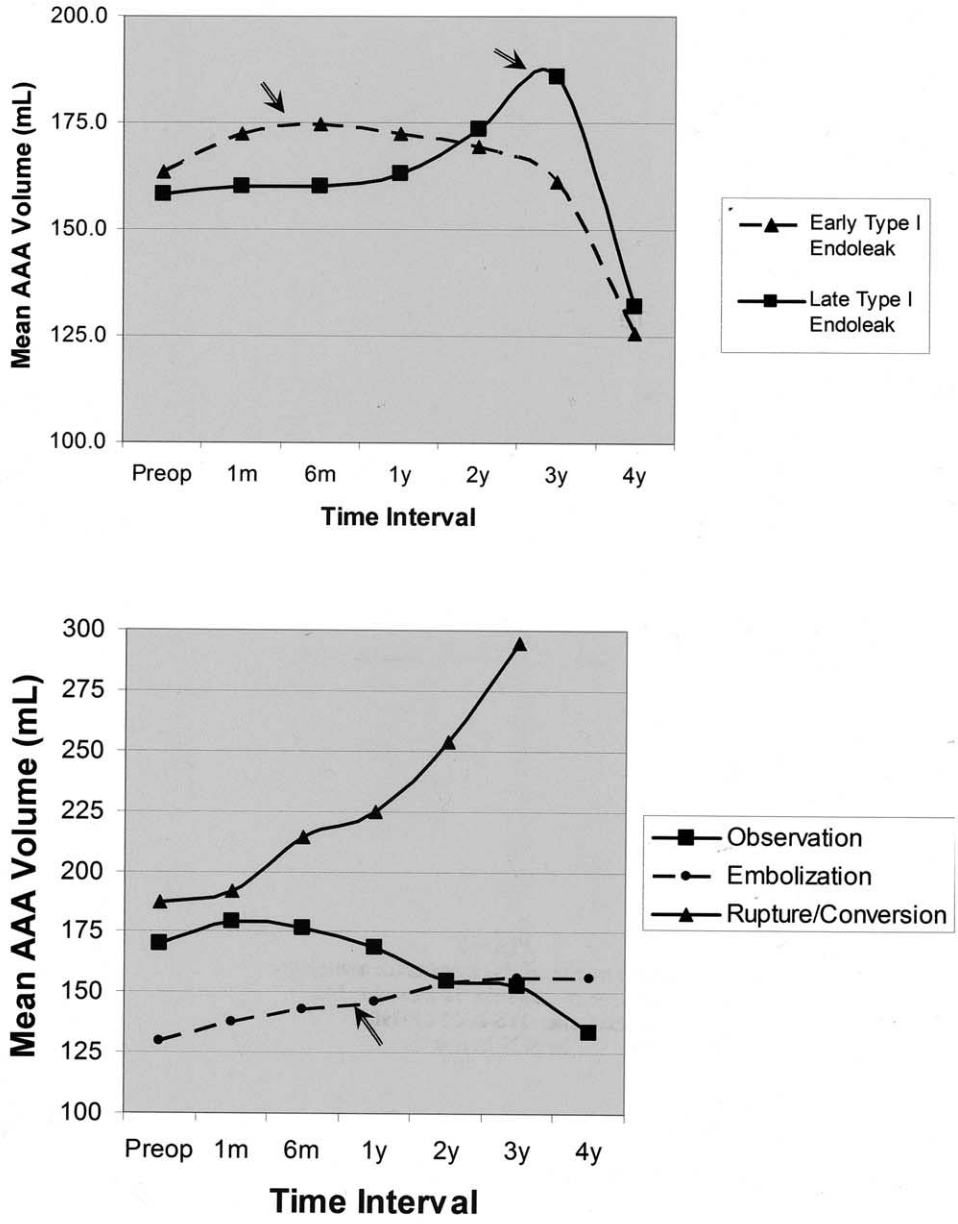

Fig 4. **A**, Changes in total AAA volume over time of seven patients early Type I endoleak (mean postoperative time = 4 m) and eight patients with late Type II endoleak (mean postoperative time = 35 m). All patients were treated with the appropriate cuff (shown as *arrows*). **B**, Changes in total AAA volume over time of Type II endoleak patients treated with observation (n = 33), embolization (n = 7), or open conversion/rupture (n = 5). Embolization occurred at mean postoperative time of 8 months (shown by *arrow*).

DISCUSSION

As more experience is gained with endovascular techniques, the technical aspects of endoluminal AAA repair are being refined to afford patients better outcomes. The high "success" rate of many series in the literature point out that AAA exclusion with a particular device is able to be performed usually in over 90% of patients it is attempted in, and with certain advantages in high-risk patients when compared with conventional open repair.[27,28] We now should shift the focus to long-term surveillance and define objective parameters that measure the efficacy of endovascular interventions. This need highlights the renewed interest in studying aneurysm morphology and the development of sophisticated software to analyze data generated by

Table V. Volume regression patterns for endoleak types

Endoleak	*Time course/ treatment*	*n = 64*	*Pre-intervention**	*Post-intervention†*
Type I	Early	7	+7.3%	−5.5%
	Late	8	+14.6%	−14.7%
Type II	Embolization	7	+19.5%	+0.9%
	Observation	34	+4.1%	−7.1%
	Rupture/ conversion	4	+44.7%	n/a
Type III	Late	2	+7.1%	−22.3%

*Compares the preoperative scan to the scan immediately before the intervention.
†Compares the scan immediately before the intervention to the scan 2 years postintervention (or sooner if not available).

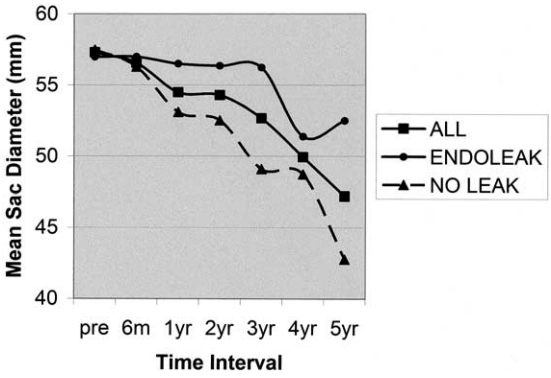

Fig 5. Maximal sac diameter changes for all 177 patients; separated by the presence or absence of endoleak.

newer imaging methods such as spiral CT, magnetic resonance 3-D imaging, and 3-D ultrasound scanning.[17,18,29]

Numerous early reports on aneurysm morphology after endovascular repair consisted of studies focusing on sac diameter regression in patients without demonstrable perigraft flow and on an increase in size in those patients with endoleak.[8-12] There have been, however, reports of aneurysm shrinkage in the face of endoleaks and aneurysm expansion in the absence of endoleaks, all based on diameter measurements.[30] The concept of simple two-dimensional data to predict AAA wall stress and subsequent risk of rupture has been recently questioned, and more detailed 3-D modeling and measurements have been proposed to be more predictive.[31,32]

Balm et al[33] first suggested that changes in volume might be more appropriate to discriminate successful from failed exclusion in a small report of nine patients with AAA sac and volume regression occurring in seven successfully excluded aneurysms at 6-month follow-up. Diameter measurements were found by Wever et al[13] not to reflect actual size changes when compared with volume measurements in 37% of cases, and they proposed volume criteria to more reliably discern subtle morphologic changes at an earlier time. Our study substantiates this finding, because discordance was found between sac diameter measurements and volume changes at the 6-month interval in 31% of cases. Other recent reports have documented or attempted to classify aneurysm sac volumetric changes in small series (30 to 50 patients) after stent-graft repair, usually with mixed aortouniiliac and bifurcated devices, as well as thoracic grafts.[14-16]

This current study includes a significant number of patients at longer follow-up intervals with comparable CT data and evaluates the usefulness of volume criteria in the long-term surveillance after endovascular repair. In our study, significant mean volume regression was found at 6 months, with continuing volume decreases out to 5 years. Annual regression rates were 7.3 mL/year for the entire cohort and 11.5 mL/year for those without demonstrable endoleak, and these findings agree with others' experiences.[14-16,34,35] Because the three devices used in this study population all had different mean preoperative volumes, percent volume changes were the focus of this analysis.

The effect of device type on volume regression was found not to be significant across the entire follow-up period ($P = .2$) and after controlling for the presence or absence of endoleak ($P = .3$). This implies that volume regression was device-independent in this series. Because we preferentially use self-expanding, fully stent-lined devices, these findings cannot be translated to stent grafts that use other means of fixation or have unsupported components. The volume regression patterns reported here need to be compared with other series where different devices were used.

The definition of successful endovascular repair is also a topic of debate. Acute parameters are those that led to the initial approval of this technology, namely, comparable mortality, decreased morbidity, and shorter hospital stay and disability, and they have continued to be observed over time.[36] We currently base clinical success on successful technical deployment; freedom from procedure- or device-related death, rupture, endoleak, migration, conversion, and secondary intervention; and aneurysm shrinkage by volumetric measurements.[26] In our study population, 64 patients had either endoleaks or needed a secondary intervention, and only 2 showed significant volume regression on 6-month CT. This finding allowed us to establish the criterion of 10% or greater volume regression at 6 months as a useful predictor for long-term clinical success. Comparisons with sac diameter changes (Table VI) confirm that volume criteria are more sensitive and accurate in predicting primary clinical success at an equivalent or earlier time interval.

Lack of volume regression is caused by continued pressurization within the sac, most often due to endoleaks. Type I endoleaks interestingly occurred at two peaks, within the first 6 months and at 35 months. In retrospect, all 15 patients with type I endoleaks were identified by gradually increasing volumes, and in the 8 patients with type I endoleaks that presented late, suspicion should have been raised because of lack of volume regression. After the appropriate cuff was placed, there were significant volume decreases of −5.5% to −14.7% in the year after the second

Table VI. Predictive accuracies of volume change (at 6 months) versus sac diameter changes (at 6 months and 1 year) for primary clinical success (with 95% confidence intervals)

	Sensitivity	*Specificity*	*Accuracy*	*Positive predictive value*	*Negative predictive value*
Volume regression > 10% at 6m	64% (49%–70%)	95% (87%–99%)	75% (67%–81%)	95% (86%–99%)	62% (52%–72%)
Sac regression > 5mm at 6m	9% (4%–16%)	91% (81%–97%)	41% (33%–49%)	60% (32%–84%)	39% (31%–47%)
Sac regression >5mm at 1 y	37% (27%–48%)	83% (72%–91%)	56% (48%–64%)	75% (60%–87%)	49% (39%–58%)

intervention, which indicates assisted primary clinical success. The occurrence of late type I endoleaks further highlights the need for long-term follow-up for all endograft patients.

The optimal treatment for type II endoleaks, when there is retrograde flow into the aneurysm sac through lumbar, IMA, or small side branch vessels, is unclear.[37] In our experience, there were three distinct patterns of volume regression based on treatment of the leak (Fig 4, *A*). Of the 34 patients that were observed without long-term sequelae, there was an increase in volume at 6-month follow-up, but then regression of −7.1% by 2 years. This suggests that type II endoleaks with AAA volume regression after 6 months are probably of insignificant origin, and they can be safely observed. Most of these patients had single-channel or lumbar-lumbar endoleaks (91%), as opposed to the more significant IMA-lumbar leaks.

A more substantial volume increase (19.5%) was seen in the seven patients who underwent successful embolization at 6-month follow-up, and this rate of expansion was slowed 2 years after embolization (+0.9%). Significant volume regression was therefore not seen postintervention. This finding does not agree with the series of seven successfully treated type II endoleaks with embolization, where significant sac regression was noted as measured by maximal aneurysm cross-sectional area.[38] This again might point out the inadequacies of two-dimensional criteria for the follow-up of patients. Another explanation is that our seven patients who underwent embolization are still under the influences of endotension that is retarding aneurysm volume regression. Still another possibility is that the embolization is not the optimal treatment for these patients or that the intervention was not performed early enough. Again, continued postoperative surveillance is mandatory to fully understand the consequences of all types of secondary interventions.

The most substantial volumetric changes occurred in the four patients with type II endoleaks who suffered ruptures or open conversion. Figure 4, *B* shows the large volume increases (+44.7% at time of rupture) up until the time of complication. The outcomes were predictable because of continued volume expansion, which occurred in two patients who refused treatment or follow-up and in two patients treated early in our experience prior to having volume measurements available for clinical decision making.

We had only two type III endoleaks, both occurring late, and after significant volume regression at 6-month (−30.4%) and 1-year (−27.4%) follow-up. The discovery of the modular disconnection was correlated with a modest increase in volume in the interval leading up to the secondary intervention (+7.1%), and then after the cuff was placed, there was continued volume regression 1 year later (−22.3%). This pattern of a late increase after initial significant regression is in agreement with Pollock et al,[16] who found only one type III endoleak in their series of 50 patients. One possibility is that a rapid volume decrease in the first 6 months led to morphologic changes and forces that caused graft instability and subsequent modular disconnection. As more bifurcated grafts are placed, we will undoubtedly be faced with discovering the optimal treatment and identification of type III endoleaks.

This assessment of the value and accuracy of volumetric analysis has modified our clinical practice. We aggressively identify and treat any type I or type III endoleaks with cuffs. Type II endoleaks when identified can be safely observed if the leak is from a single-channel or lumbar-lumbar connection and if the AAA volume is not significantly increasing. If there is an increase in AAA volume or the leak consists of an IMA-lumbar connection, then embolization is attempted. If successful, we expect no further AAA enlargement. In the face of an expanding AAA volume without demonstrable endoleak, we have adopted the policy of provocative tests to search out an endoleak or cause, including obtaining more frequent spiral CT scans, angiography, magnetic resonance angiograms, or intravascular ultrasound scans.

Our study is limited by its retrospective design. Although review of the imaging data was performed retrospectively, we attempted to minimize misclassification bias by blinding the reviewers to patient outcome. Selection bias was minimized because we used a mixed linear model to analyze volume changes over time. This form of analysis is designed, in part, to account for the unbalanced data. Thus, no missing longitudinal measurements (ie, CT scans) were excluded.

The issue of intraobserver and interobserver variability has also been raised when evaluating spiral CT data. Intraobserver variability has been found to be smaller than interobserver variability, implying that measurements and trends in the postoperative follow-up will be more accurate if performed by one observer or panel.[39] Another study pointed out that differences between manual measurements with calipers and computerized measurements exceed 2 to 3 mm, and that automated observer-independent measuring techniques should be used.[40] For those reasons, we used a computerized 3-D reconstruction and made our

measurements as a group with both vascular surgeons and radiologists. We have noted intraobserver variability of approximately 2% to 3%, which is in agreement with other studies.[41,42]

In conclusion, we believe that volumetric analysis is a necessary and accurate adjunct in the follow-up after endoluminal exclusion of AAAs and can be used to assess primary clinical success. AAA sac volume increases occur with perigraft flow and an aneurysm that is still at risk for rupture, which heralds the need for closer evaluation and surveillance and possible intervention. Volume changes are more predictive than are traditional sac diameter measurements for determining successful exclusion and detecting potential problems. A volume decrease of 10% or greater at 6 months and continuing regression over time is associated with successful endovascular repair and protection from rupture.

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