# Continued expansion of aortic necks after endovascular repair of abdominal aortic aneurysms

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*Background*: Longitudinal studies have revealed that the aortic segment proximal to an infrarenal abdominal aortic aneurysm (AAA) is at risk for continued enlargement after a standard aneurysm repair. Similarly, preliminary reports have shown expansion of one or both aortic necks after endovascular repair. Although some investigators have suggested that this may be a transient effect, continued dilatation at the endograft attachment site could effect the overall device stability.

*Methods*: As part of a multi-institutional trial of endovascular grafting for the treatment of AAA, 59 patients were successfully implanted with straight endografts between February 1993 and January 1995. A morphometric analysis of aortic neck size was undertaken with serial review of computed tomography scans available through April 1997. The neck sizes at both graft attachment sites were measured, with investigators blinded to patient identity and date of scan. Changes in minor diameter were defined, annual interval expansion rates were calculated, and the data were correlated with endoleak, device migration, aneurysm size change, endograft diameter, attachment system fractures, and initial preimplant neck size.

*Results*: Significant aortic neck enlargement, particularly at the level of the distal neck, was observed for at least 24 months after AAA repair. The annual interval dilation rates of the proximal aortic neck were 0.7 ± 2.1 mm/y ($P$ = .023) and 0.9 ± 1.9 ($P$ = .008) mm/yr during the first and second years, respectively. Enlargement of the distal neck during the observation period was more marked, with corresponding annual expansion rates of 1.7 ± 2.9 mm/y ($P$ < .001) and 1.9 ± 2.5 ($P$ < .001) mm/year. In 5 patients (14%), the minor diameter of the distal neck was at least 6 mm larger than the preimplant diameter of the graft. Migration of the distal attachment system was observed in 3 of these 5 patients. Expansion rates did not have a statistically significant correlation with initial neck size, endograft dimensions, aneurysm size change, presence of endoleak, or attachment system fracture.

*Conclusions*: Aortic neck enlargement was observed for at least 2 years after endovascular grafting. Close patient follow-up remains mandatory in lieu of the potential risk of late failure as a result of continued aortic expansion. The relative contribution of device design to this phenomenon will need to be defined. (J Vasc Surg 1998;28:422-31.)

Endovascular grafting holds significant promise as a minimally invasive treatment for infrarenal abdominal aortic aneurysm (AAA), with the potential for associated reductions in hospital costs, perioperative morbidity and mortality rates, and duration necessary for complete recovery after surgery. Nonetheless, interim analysis in several prospective trials, which have included both tube and bifurcated endografts, has suggested that long-term limitations may exist in the application of this new technology. In particular, concerns have been raised regarding the observation of aortic dilatation both proximal and distal to the endograft and its effects on overall device stability.

Aortic neck dilation with subsequent pseudoaneurysm formation is an infrequent, but well-known, occurrence in the pararenal aorta craniad to standard

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infrarenal aneurysm repair. The underlying mechanisms that contribute to this phenomenon after standard aortic surgery are not well defined but likely represent ongoing degenerative changes in the host aorta. To date, most morphometric studies conducted after endovascular repair have found enlargement of one or both aortic necks during the initial follow-up period. Studies performed at Utrecht revealed distal neck enlargement within 6 months of endovascular grafting in 9 patients.[1] In a report from Mälmo, proximal neck dilation was documented during the first postoperative year.[2] By contrast, May et al[3] have suggested that the aortic neck size stabilizes after a short early period of dilation. Mean proximal and distal neck enlargement varied between 2.5 to 6.5 mm for patients with follow-up intervals from before surgery to 6, 12, and 18 months, and appeared to be a plateau phenomenon. Others have yet to observe aortic neck enlargement.[4] In a previous report from the North American EndoVascular Technologies, Inc (EVT, Menlo Park, Calif) clinical trial, statistically significant distal neck dilatation of 1.4 mm occurred in the first 12 months after endovascular grafting but not during the periprocedural period.[5] Proximal neck dilation was not statistically significant in this early study.

Continued aortic neck dilation is significant in that attachment-site stability may be compromised, which would lead to the development of an endoleak or catastrophic device failure. In this regard, case reports of late endoleaks associated with neck dilation have been reported.[6-9] The objectives of this investigation were to characterize the rate of neck size enlargement after AAA repair with a tube endograft system, the duration of this phenomenon, and its relationship to endoleak, endograft size, device migration, aneurysm size change, attachment system failure, and preoperative neck diameter.

## METHODS

Endovascular tube grafts for AAA were successfully placed in 59 patients at 13 institutions in the United States between February 10, 1993, and January 24, 1995, during the course of the Food and Drug Administration–approved phase I and II trials. Implants were halted in January 1995 because of the detection of unanticipated fractures in the metallic attachment system in a significant proportion of patients treated with this early endograft prototype. Informed consent for participation in the study was signed by each patient, with approval from local Institutional Review Boards. The primary investigators at each contributing institution, listed in Appendix A, and EVT provided unrestricted access for this study. A more comprehensive description of the clinical trial design, device configuration, implantation technique, and early clinical outcome can be found elsewhere.[10-12]

All available preoperative and postoperative abdominal computed tomography (CT) scans were reviewed through April 1997 at a central data collection site. The mean follow-up was 27 ± 8.6 months, with a range of 2 to 46 months (median, 27 months). Analysis was confined to 2-dimensional transverse images. *Proximal* and *distal* aortic necks were each defined as the most cephalad or caudal image containing a complete hook set (ie, where the device pins were actually implanted). These areas were chosen because of their direct relationship to attachment-site stability. Comparable images of these areas were selected on each CT data set by matching both the calcification pattern in the aortic wall and bony landmarks. Therefore in the event of device migration, the attachment site was determined on the basis of its position in the original postimplant scan. Other studies have chosen to measure aortic segments halfway in the neck or attachment system, at a site suitable for proximal stent implantation, and at 1 cm below the lowest renal artery and 1 cm above the aortic bifurcation.[1-3]

Images were scanned at 300 dots per inch, cropped of identifying data, randomly coded, and measured with computerized planimeter (NIH Image, National Institutes of Health, Bethesda, Md). Aortic scans were enlarged, the external perimeter traced, and measurements taken on the basis of the accompanying calibrated scales. Minor diameter, the shortest diameter of the best fit ellipse approximating the aortic perimeter, was selected for analysis because of its relative independence from the potentially confounding effects of aortic tortuosity on calculating aortic dimensions derived strictly from vertical or transverse CT diameters. The blinding procedure, although tedious and time consuming, was necessary to obtain objective dimensions during the measurement process.

Both plain abdominal radiographs and fluoroscopy were used to detect fractures of the metallic attachment system and device migration (Fig. 1). Migration included “tipping” angulation of the attachment system, which occurred more commonly than the cranial-caudad movement of the entire system. CT scans were also examined for evidence of endoleak as defined with radiocontrast enhancement between graft and aneurysm wall. In addition, the extent of endograft mismatch (disparity between

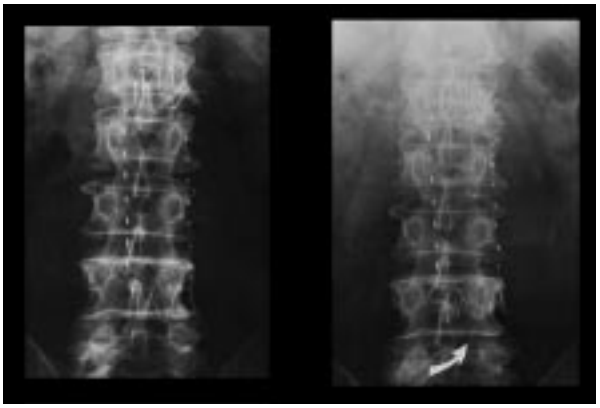

Fig. 1. Plain radiographs showing attachment system fracture at distal (inferior) end of graft. *Left panel* shows intact system at time of discharge. *Right panel* shows single-hook shank fracture *(white arrow)* with cephalad migration of distal end of prosthesis.

graft and aortic size) was characterized by subtracting the preimplant neck diameter from the initial endograft diameter, such that positive values were indicative of oversizing. At the time of this clinical study, significant endograft oversizing was not practiced, and mean endograft mismatch values were 0.4 ± 2.3 mm at the proximal and 0.8 ± 3.3 mm at the distal. Selection of CT images, measurements, and interpretation of radiographs was performed with the consensus of 2 observers. Codes were broken only after the completion of data collection.

Paired 2-tailed tests were used to compare interval size changes. A *P* value of less than .05 was selected for the determination of statistical significance. Descriptive statistics are given as mean ± standard deviation of the mean. Spearman rank correlation coefficients between continuous variables were calculated with a standard software package (SPSS Release 6.1.4, SPSS, Inc, Chicago, Ill).

## RESULTS

This study was prompted by the course of 2 patients who underwent endograft placement for treatment of aortic aneurysms. A 63-year-old man had a 4.6-cm aortic aneurysm treated with an endovascular tube graft in January 1995. The aneurysm was stable in size during the first postoperative year. Hook fracture at the distal attachment system was noted at 18 months (Fig. 1). Although the endoleak was initially undetected, later reanalysis of a concurrent CT scan revealed, in retrospect, a small distal endoleak in a single 1.5-mm slice.[13] The aneurysm enlarged to 5.3 cm at 24 months and ruptured soon thereafter. The patient underwent emergency open repair and recovered uneventfully. Fig. 2 illustrates progressive distal neck dilation with interval enlargement of 10.2 mm. Inspection of the preoperative scans showed that the distal aortic neck, although slightly ectatic, met size inclusion criteria and was without visible mural thrombus or significant calcification (Fig. 3).

A second patient also had a late endoleak that necessitated conversion to standard surgery. This 69-year-old man had an endograft placed in April 1994 for a 4.9-cm aneurysm. An endoleak was first detected 21 months after implantation. At 30 months, attachment system fracture and craniad migration of the distal end of the graft were observed and associated with a 2.8-mm dilation of the distal neck. The aneurysm had enlarged to 5.1 cm, and the patient underwent an open conversion without complication. The occurrence of attachment system fractures and late onset endoleaks in both cases limited our ability to determine if progressive aortic enlargement was caused by degeneration of the aortic wall, which contributed to device failure, or vice versa. Sufficient suspicion existed that this phenomenon was intrinsic to the host aorta to warrant the present investigation of neck size.

The interval minor diameter changes for proximal and distal necks are presented in Table I. Aortic neck enlargement occurs for at least 24 months after endovascular treatment of AAA with this trend continuing during the third year of follow-up. Mean proximal neck dilation was 0.7 mm and 0.9 mm in the first and second years, respectively. During the same time period, distal neck size increased at an even greater rate, averaging 1.7 mm/y and 1.9 mm/y. In patients with complete 2-year follow-up, distal neck diameter was more than 6 mm larger than initial endograft diameter in 5 patients (14%). The perioperative interval is between the last preoperative and first postoperative CT scans and averaged 76 ± 48 days. Therefore changes during this period may be caused by the following: (1) iatrogenic enlargement after the device or balloon inflations, (2) natural dilation during the interval between preoperative and first postoperative imaging, (3) observer bias as a result of the unblinded nature of preoperative scans, (4) systematic bias in measurement caused by artifact from the metal attachment system, or (5) thickening of the aortic wall as a result of a local inflammatory response in the immediate postprocedure period. Because of the widely varying perioperative interval and these other enumerated factors, the use of these values is limited to assessing whether significant

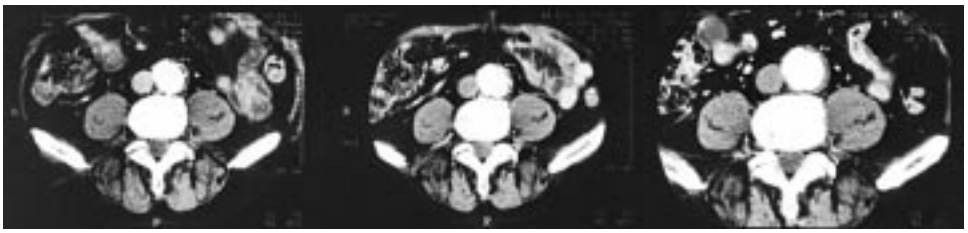

Fig. 2. Same patient is shown as in Fig. 1. This is a composite of computed tomography slices at the distal neck: *left* at 18 months, *center* at 21 months, and *right* at 24 months after placement of endograft. Note different scales visible adjacent to the images. Calibrated measurements shown over 10-mm dilation of minor diameter. It is also evident that internal diameter cannot be assessed with this method.

Table I. Interval expansion rates of proximal and distal aortic necks (mm / year, mean ± standard deviation of the mean)

| | *Perioperative (n)* | *1st year (n)* | *2nd year (n)* | *3rd year (n)* |
|---|---|---|---|---|
| Proximal | 0.5 ± 2.4 (53)<br>*P* = .115 | 0.7 ± 2.1 (51)<br>*P* = .023 | 0.9 ± 1.9 (35)<br>*P* = .008 | 0.5 ± 1.4 (6)<br>*P* = .446 |
| Distal | 0.4 ± 2.6 (51)<br>*P* = .298 | 1.7 ± 2.9 (48)<br>*P* < .001 | 1.9 ± 2.5 (36)<br>*P* < .001 | 1.4 ± 2.2 (8)<br>*P* = .130 |

Table II. Correlations of neck diameter changes with initial neck size, endograft size, endograft size mismatch (endograft diameter minus initial neck diameter), and aneurysm size change. Third year data are omitted because of too few data points. Data are expressed as correlation coefficients *(r)*.

| *Spearman correlations* | *1st year* | *2nd year* |
|---|---|---|
| Proximal neck change vs initial neck size | 0.175 | –0.122 |
| Proximal neck change vs endograft size | 0.191 | –0.178 |
| Proximal neck change vs (endograft – neck size) | 0.131 | –0.088 |
| Proximal neck change vs aneurysm size change | 0.234 | –0.158 |
| Distal neck change vs initial neck size | –0.018 | 0.169 |
| Distal neck change vs endograft size | 0.076 | 0.189 |
| Distal neck change vs (endograft – neck size) | 0.074 | –0.044 |
| Distal neck change vs aneurysm size change | 0.090 | –0.278 |

Table III. Interval diameter changes of aortic necks in patients with and without device migration (mm / year, mean ± standard deviation of the mean). *P* value refers to *t* test in comparison with no migration.

| *Location* | *Status* | *1st year (n)* | *2nd year (n)* | *3rd year (n)* |
|---|---|---|---|---|
| Proximal | No migration | 0.6 ± 1.9 (44) | 1.0 ± 1.9 (29) | 0.3 ± 1.5 (5) |
| Proximal | Migration | 1.5 ± 3.0 (7)<br>*P* = .274 | 0.5 ± 1.5 (6)<br>*P* = .593 | 1.2 (1) |
| Distal | No migration | 1.5 ± 2.8 (41) | 1.7 ± 2.4 (30) | 1.6 ± 2.2 (7) |
| Distal | Migration | 3.2 ± 3.5 (7)<br>*P* = .162 | 3.0 ± 3.2 (6)<br>*P* = .252 | 0.7 (1) |

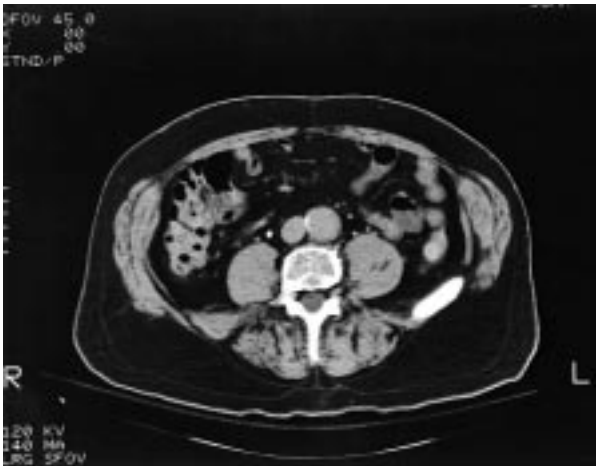

Fig. 3. Preoperative computed tomography scan of distal neck is shown. No significant calcification or mural thrombus is visualized, but shape is slightly ectatic. Newer scanning techniques reconstruct images with thin sections, which permits better assessment of neck characteristics.[13]

changes occurred as a result of periprocedural events, and subsequent attention in this study is focused on annual interval changes beginning with the first postoperative (ie, discharge) CT scan.

The primary end point for this investigation was designated a priori as the minor diameter change from discharge to 2-year follow-up. This 2-year difference in diameter was 1.9 ± 2.3 mm at the proximal neck and 3.7 ± 3.6 mm at the distal neck. Both changes were statistically significant at $P < .0001$. These interval changes are slightly different from the simple sum of the first and second year intervals because they include only the patients with complete follow-up for both years. In the present study, all CT images were masked and remeasured, and so these values are also slightly different from previously reported values with a smaller sample size and shorter follow-up.[5,12] These previous studies did not detect significant proximal neck size change, most likely because of a lack of statistical power. This diameter increase is small and only reliably detected with a large sample size, multi-year follow-up, and blinded, computerized planimetry.

The following parameters' relationships to neck changes were evaluated: endoleak, device migration, preimplant neck size, endograft size mismatch, aneurysm size change, endograft diameter, and attachment system fractures. Spearman rank correlation analysis was performed between change in neck dimension and initial neck size, endograft diameter, endograft sizing, and aneurysm size change. These data are summarized in Table II. No statistically significant relationship was observed between any of these factors and neck expansion at either proximal or distal attachment site.

It is noteworthy that a statistically significant difference in the rate of change of neck diameter was not observed in patients with or without device migration (Table III). Nonetheless, in 5 patients, external distal neck expansion progressed to a diameter 6 mm greater than the endograft diameter. Device migration occurred in 3 of these 5 patients. A total of 9 patients had associated device migration, 8 involving the distal attachment site, and all but 1 underwent complete explantation, revision, or redo endovascular grafting. The patient remaining declined further intervention. Similarly, significant differences were not observed between patients exhibiting the presence or absence of either an endoleak (Table IV) or attachment system fracture (Table V). Admittedly, the statistical power of these tests may be limited by the small number of patients in each of the subgroups.

## DISCUSSION

Persistent aortic neck enlargement of approximately 1 mm / y to 2 mm / y was observed for at least 24 months after endovascular treatment of AAAs with straight tube endografts. This phenomenon was most marked at the distal attachment site, typically located in the region immediately proximal to the aortic bifurcation. Aneurysmal degeneration in the juxtarenal and suprarenal aorta subsequent to operative repair of an AAA has been well described. For example, in a 25-year review of 920 patients having infrarenal aneurysm repair, 8% of subsequent deaths were attributed to rupture of a second aneurysm.[14] Though not all secondary aneurysms in that investigation were located in the abdominal aorta, other studies, which have included serial sonographic, angiographic, and CT surveillance, have reported a similar proportion of late para-anastomotic dilation or juxtarenal aneurysms in the range of 6% to 8%.[15-17] A population-based study from Olmstead County has highlighted that the overall incidence rate of gradual aortic enlargement after surgery, although not always clinically significant, may be significantly higher than noted even in these reports—in the range of 13%.[18] Clearly, a history of aneurysmal disease is associated with risk of a secondary aneurysm formation. Furthermore, studies of healthy adults, veterans, and patients with nonvascular disease, have also verified that aortic size generally increases with advancing age.[19-21]

Table IV. Interval diameter changes of aortic necks in patients with and without endoleak (mm / year, mean ± standard deviation of the mean). *P* value refers to *t* test in comparison with no endoleak.

| *Location* | *Status* | *1st year (n)* | *2nd year (n)* | *3rd year (n)* |
|---|---|---|---|---|
| Proximal | No endoleak | 0.5 ± 2.1 (26) | 1.5 ± 2.2 (18) | –0.1 ± 1.3 (4) |
| Proximal | Endoleak | 0.8 ± 2.1 (25) | 0.3 ± 1.2 (17) | 1.7 ± 0.7 (2) |
| | | *P* = .609 | *P* = .056 | *P* = .146 |
| Distal | No endoleak | 1.5 ± 2.5 (25) | 1.5 ± 2.6 (19) | 2.0 ± 2.3 (6) |
| Distal | Endoleak | 2.0 ± 3.3 (23) | 2.4 ± 2.5 (17) | –0.5 ± 0.3 (2) |
| | | *P* = .508 | *P* = .306 | *P* = .193 |

Table V. Interval diameter changes of aortic necks in patients with and without attachment system fractures (mm / year, mean ± standard deviation of the mean). *P* value refers to *t* test in comparison with no fractures.

| *Location* | *Status* | *1st year (n)* | *2nd year (n)* | *3rd year (n)* |
|---|---|---|---|---|
| Proximal | No fractures | 1.0 ± 1.7 (28) | 0.9 ± 2.1 (21) | –0.1 ± 1.3 (4) |
| Proximal | Fractures | 0.3 ± 2.4 (23) | 0.8 ± 1.5 (14) | 1.7 ± 0.7 (2) |
| | | *P* = .200 | *P* = .854 | *P* = .146 |
| Distal | No fractures | 1.7 ± 2.8 (27) | 2.2 ± 2.9 (21) | 2.3 ± 2.3 (5) |
| Distal | Fractures | 1.8 ± 3.1 (21) | 1.5 ± 2.0 (15) | –0.3 ± 0.5 (3) |
| | | *P* = .864 | *P* = .437 | *P* = .115 |

The preponderance of subsequent aneurysm formation in the region proximal to a surgically implanted graft continues to be an intriguing observation. The mechanisms responsible for localizing secondary aneurysms to this area have not been fully defined but are probably related to the more advanced state of aortic wall degeneration in this region and to other biomechanical determinants. These factors remain pertinent, regardless of the chosen mode of aneurysm repair. For example, Illig et al[22] characterized the dilation rate of the infrarenal aortic cuff in 33 patients after standard aneurysm repair, measuring a mean increase of 4.3 mm at an average of 89 months after operation. The calculated expansion rate of 0.6 mm / year is nearly identical to that observed in our report. This suggests that continued aortic enlargement at the proximal neck after surgery or endovascular repair is inherently related to the intrinsic characteristics of the host aorta. Further, it is unlikely to be affected by the choice of either tubular or bifurcated prosthesis. In a subgroup analysis, Illig et al[22] have commented that dilation rates were significantly greater among those patients in which the preoperative neck diameter was 28 mm or greater in size. Current protocols for the use of EVT endografts exclude patients who fall in this anatomic category. A cautious approach may be warranted for the use of sutureless endoprostheses in this particular patient population.

The observation in this and other reports of distal neck expansion, endoleak formation, and device migration after endovascular AAA repair prompts 2 critical questions. Does endovascular grafting potentiate the process of neck expansion, particularly in the distal aorta? Are these prostheses less robust in their ability to compensate for anticipated regional aortic expansion? Persistent flow within the aneurysm sac after endovascular grafting may be related to an inadequate seal at the proximal or distal segments of the endoprosthesis or to branch flow through a patent inferior mesenteric or lumbar arteries. Although the former appears to be of diminishing significance with continued improvements in overall design, branch flow will likely remain a potential problem associated with all forms of exclusion, not just endovascular devices.[23] If the flow is sufficient to produce aneurysm dilation, it will likely contribute to expansion of both proximal and distal aortic necks. We did not observe a significant correlation between endoleak and associated change in neck dimensions, nor did we observe greater expansion rates in neck size during the first postimplant year during which the proportion of endoleaks is usually greatest. We also did not detect a significant correlation with aneurysm size change, regardless of diagnosis of endoleak. Nonetheless, we continue to speculate that the expansion of the distal aortic cuff, which appears uniquely associated with the use of endoprostheses, is related to this phenomenon. Our inability to detect this relationship may be caused by sample size or by the relatively short period of follow-up. However, the most

important limitation in the analysis of this factor may be the limited sensitivity of all current imaging methods in detecting small endoleaks.

As previously noted, fractures of the metallic attachment system developed in a significant proportion of patients after implantation of this early prototype. However, their occurrence did not inevitably lead to clinically significant complications, such as endoleak formation or device migration, and were not statistically associated with neck expansion. Therefore we do not believe that the failure of this prototype was uniquely related to an inability to compensate for neck dilation. Furthermore, a significant relationship was not detected between cuff dilation rates and absolute endograft size or size mismatch. Oversizing does not appear to be a contributory factor in neck expansion and may well be an important consideration in choosing a prosthesis that will respond to continued aortic enlargement. Apart from deliberate oversizing of the endograft, the capacity of the prosthesis to self-expand or the use of a durable mechanical attachment, such as hooks that completely penetrate the aortic wall, are all potentially valid solutions. Ideally, a mechanism to arrest neck dilation should be developed. In principle, increased use of bifurcated prostheses, with complete avoidance of distal attachment in the aorta proper, would also negate the effect of the distal cuff expansion. However, the potential of the iliac system to dilate over time has yet to be fully defined.

The clinical significance of continued aortic expansion is evident from prior reports of patients treated by standard operative approaches but was not conclusively shown in our investigation. In compiling several series, 37 cases of late endoleak noted at both proximal and distal necks have been reported, and, in at least 6 of these patients, endoleaks were associated with neck dilation.[6,7,9,24,25] Apart from continued aneurysm enlargement and risk of rupture,[12,26-28] a 6% proximal migration rate with a unitary bifurcated device reported in a recent abstract emphasizes the potential significance of aortic expansion.[29] Thus the risk of aortic expansion is real and mandates continued evaluation of all endoprostheses to determine with greater precision the susceptibility of a given device to failure and the probable time frame for this occurrence.

## CONCLUSIONS

Aortic neck diameter continuously enlarges at both proximal and distal attachment sites for at least 2 years after endovascular tube graft repair of AAA. This enlargement did not correlate with preoperative neck diameter, endograft sizing, presence of endoleak, aneurysm size change, and attachment system fractures. These data imply that neck dilation is not unique to device design. Further, aortic dilation in adults without aneurysms and after standard surgery supports the hypothesis that the aorta has a natural tendency to enlarge, which is likely exacerbated by the presence of underlying degenerative disease and by the potential of persistent branch flow in the unique case of endovascular repair. The absolute rate of dilation is small, but the 2 cases described herein and the other reports in the literature lead one to suspect that the risk of significant complications is genuine.

## REFERENCES

1. Balm R, Kaatee R, Blankensteijn JD, Mali WPTM, Eikelboom BC. CT-angiography of abdominal aortic aneurysms after transfemoral endovascular aneurysm management. Eur J Vasc Endovasc Surg 1996;12:182-8.
2. Malina M, Ivancev K, Chuter TAM, Lindh M, Lanne T, Lindblad B, et al. Changing aneurysmal morphology after endovascular grafting: relation to leakage or persistent perfusion. J Endovasc Surg 1997;4:23-30.
3. May J, White GH, Yu W, Waugh RC, Stephen MS, Harris JP. Prospective study of anatomico-pathological changes in abdominal aortic aneurysms following endoluminal repair: is the aneurysmal process reversed? Eur J Vasc Endovasc Surg 1996;12:11-7.
4. Blum U, Beyersdorf F. Correspondence. N Engl J Med 1997;24:1756-7.
5. Matsumura JS, Pearce WH. Fate of aortic size before and after Endovascular Technologies tube grafting: augmented data. In: Yao JST, Pearce WH, editors. Progress in vascular surgery. Stamford CT: Appleton & Lange; 1997. p. 159-70.
6. Nasim A, Thompson MM, Sayers RD, Boyle JR, Bolia A, Bell PRF. Late failure of endoluminal abdominal aortic aneurysm repair due to continued neck expansion. Br J Surg 1996;83:810-1.
7. Ivancev K, Malina M, Lindblad B, Chuter TAM, Brunkwall J, Lindh M, et al. Abdominal aortic aneurysms: experience with the Ivancev-Malmo endovascular system for aortomonoiliac stent-grafts. J Endovasc Surg 1997;4:242-51.
8. Parodi JC, Ferreira LM. Different outcomes after endovascular treatment of small, medium, and large aneurysms: 7-year experience. J Endovasc Surg 1998;5:I-24.
9. Kato N, Semba CP, Dake MD. Embolization of perigraft leaks after endovascular stent-graft treatment of aortic aneurysms. JVIR 1996;7:805-11.
10. Moore WS, Rutherford RB. Transfemoral endovascular repair of abdominal aortic aneurysm: results of the North American EVT phase 1 trial. J Vasc Surg 1996;23:543-53.
11. Edwards WH Jr, Naslund TC, Edwards WH Sr, Jenkins JM, McPherson K. Endovascular grafting of abdominal aortic aneurysms: a preliminary study. Ann Surg 1996;223:568-75.
12. Matsumura JS, Pearce WH, McCarthy WJ, Yao JST. Reduction in aortic aneurysm size: early results after endovascular graft placement. J Vasc Surg 1997;25:113-23.

13. Beebe HG, Bernhard VM, Parodi JC, White GH. Endovascular forum: leaks after endovascular therapy for aneurysm: detection and classification. J Endovasc Surg 1996;3:445-8.
14. Crawford ES, Saleh SA, Babb JW III, Glaeser DH, Vaccaro PS, Silvers A. Infrarenal abdominal aortic aneurysm: factors influencing survival after operation performed over a 25-year period. Ann Surg 1981;193:699-709.
15. Edwards JM, Teefey SA, Zierler RE, Kohler TR. Intraabdominal paraanastomotic aneurysms after aortic bypass grafting. J Vasc Surg 1992;15:344-53.
16. Lipski DA, Ernst CB. Natural history of the residual infrarenal aorta after infrarenal abdominal aortic aneurysm repair. J Vasc Surg 1998;27:805-12.
17. Berman SS, Hunter GC, Smyth SH, Erdoes LS, McIntyre KE, Bernhard VM. Application of computed tomography for surveillance of aortic grafts. Surgery 1995;118:8-15.
18. Hallett JW Jr, Marshall DM, Petterson TM, Gray DT, Bower TC, Cherry KJ, et al. Graft-related complications after abdominal aortic aneurysm repair: reassurance from a 36-year population-based experience. J Vasc Surg 1997;25:277-86.
19. Sonesson B, Lanne T, Hansen F, Sandgren T. Infrarenal aortic diameter in the healthy person. Eur J Vasc Surg 1994; 8:89-95.
20. Lederle FA, Johnson GR, Wilson SE, Gordon IL, Chute EP, Littooy FN, et al. Relationship of age, gender, race, and body size to infrarenal aortic diameter. J Vasc Surg 1997;26: 595-601.
21. Pearce WH, Slaughter MS, LeMaire S, Salyapongse AN, Feinglass J, McCarthy WJ, et al. Aortic diameter as a function of age, gender, and body surface area. Surgery 1993; 114:691-7.
22. Illig KA, Green RM, Ouriel K, Riggs P, Bartos S, DeWeese JA. Fate of the proximal aortic cuff: implications for endovascular aneurysm repair. J Vasc Surg 1997;26:492-501.
23. Resnikoff M, Darling RC III, Chang BB, Lloyd WE, Paty PSK, Leather RP, et al. Fate of the excluded abdominal aortic aneurysm sac: long-term follow-up of 831 patients. J Vasc Surg 1996;24:851-5.
24. Parodi JC. Endovascular repair of abdominal aortic aneurysms and other arterial lesions. J Vasc Surg 1995; 21:549-57.
25. Heilberger P, Schunn C, Ritter W, Weber S, Raithel D. Postoperative color flow duplex scanning in aortic endografting. J Endovasc Surg 1997;4:262-71.
26. Matsumura JS, Moore WS, for the EVT Investigators. Clinical consquences of periprosthetic leak after endovascular repair of abdominal aortic aneurysm. J Vasc Surg 1998; 27:606-13.
27. Gilling-Smith GL, Cuypers P, Buth J, Harris PL, for the EUROSTAR Collaborators. The significance of endoleaks after endovascular AAA repair: results of a large European multicenter study. J Endovasc Surg 1998;5:I-12.
28. Lumsden AB, Allen RC, Chaikof EL, Resnikoff M, Moritz MW, Gerhard H, et al. Delayed rupture of aortic aneurysms following endovascular stent grafting. Am J Surg 1995; 170:174-8.
29. Yusuf SW, Armon MP, Whitaker SC, MacSweeney ST, Tennant WG, Wenham PW, et al. Intermediate-term results of endovascular aortic aneurysm repair. Br J Surg 1997; 84:1575-6.

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## APPENDIX A

We are indebted to the participants and to the United States investigators, coinvestigators, and radiologists:

Emory University Hospital. Primary investigator: Elliott Chaikof, MD, PhD. Coinvestigators: Alan Lumsden, MD, Thomas Dodson, MD, Atef Salam, MD, Robert B. Smith III, MD. Radiologists: Alan Zuckerman, MD, Stephen Kaufman, MD, Louis Martin, MD.

Henry Ford Hospital. Primary investigator: Calvin B. Ernst, MD. Coinvestigators: Daniel Reddy, MD, Joseph Elliott, MD, Alexander Shepard, MD. Radiologist: P. C. Shetty, MD.

Massachusetts General Hospital. Primary investigator: David C. Brewster, MD. Coinvestigators: William M. Abbott, MD, Richard Cambria, MD. Radiologists: Stuart Geller, MD, John Kaufman, MD.

Montefiore Medical Center. Primary investigator: Frank J. Veith, MD. Coinvestigator: Michael Marin, MD. Radiologist: Jacob Cynamon, MD.

Miami Vascular Institute. Primary investigator: Barry Katzen, MD, Orlando Puente, MD. Coinvestigators: Jose Alvarez Jr, MD, Steven Kanter, MD. Radiologists: Barry Katzen, MD, James Benenati, MD, Gerald Zemel, MD, Gary Becker, MD.

Northwestern Memorial Hospital. Primary investigator: James S. T. Yao, MD, PhD. Coinvestigators: William H. Pearce, MD, Walter J. McCarthy III, MD. Radiologists: Albert Nemcek, MD, Robert Vogelzang, MD.

New York University Medical Center. Primary investigator: Thomas S. Riles, MD. Coinvestigators: Patrick Lamparello, MD, Mark A. Adelman, MD, Gary Giangola, MD. Radiologist: Robert Rosen, MD.

St. Thomas / Vanderbilt University Medical Center. Primary investigator: William H. Edwards Sr, MD. Coinvestigators: William H. Edwards Jr, MD, Thomas A. Naslund, MD.

Stanford University Medical Center. Primary investigator: R. Scott Mitchell, MD. Coinvestigators: Christopher K. Zarins, MD, Edmund Harris Jr, MD. Radiologist: Charles Semba, MD.

University of Colorado. Primary investigator: Robert B. Rutherford, MD. Coinvestigators: William C. Krupski, MD, Darrell Jones, PhD. Radiologists: David Kumpe, MD, Janette Durham, MD.

University of California, Los Angeles. Primary investigator: Wesley S. Moore, MD. Coinvestigators: Samuel S. Ahn, MD, J. Dennis Baker, MD, William J. Quinones-Baldrich, MD, Hugh A. Gelabert, MD,

Herbert I. Machleder, MD, Richard W. Bock, MD, Rhoda Leichter, MD. Radiologist: Antionette S. Gomes, MD.

University of California, San Francisco. Primary investigator: Jerry Goldstone, MD. Coinvestigator: Susan Wall, MD. Radiologist: Ernest Ring, MD.

University of Texas Southwestern Medical Center. Primary investigator: G. Patrick Clagett, MD. Coinvestigators: Arun Chervu, MD, R. James Valentine, MD, Stuart Myers, MD. Radiologists: George Miller, MD, Rebhi Awad, MD, Margaret Hansen, MD, Helen Redman, MD, Jorge Lopez, MD.

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## DISCUSSION

**Dr William H. Edwards, Sr** (Nashville, Tenn). I am pleased to rise to discuss this paper because I was an investigator in this series.

The 59 patients on whom Drs Matsumura and Chaikof reported were indeed pioneers, if you will, in endovascular aneurysmal repair. Guidelines were set and approved by the Food and Drug Administration, but nobody knew whether they were going to be valid or whether they would stand the test of time.

I borrow from a 1960s musical icon who is having a resurgence in the nineties: "the times they are a-changing." Since the first successful resection of an abdominal aneurysm in 1951, we focused on the diameter of the aneurysm. Never mind the proximal or distal neck. Give me a 6-cm aneurysm, and I can resect it. Dr Matsumura's presentation points at the importance of a better understanding of the changes that may affect the site of attachment of endovascular grafts. As the pathogenesis and the morphology of aneurysms are better understood, more precise cuff measurements would improve endovascular technology. We will begin to solve the issues of cuff dilatation. The concept is sound, but we may learn that the anatomy does not lend itself to tube grafts. This is confirmed somewhat in his study in which the distal cuff had the more significant dilatation. As you move up on this learning curve, there probably will also be other issues that will need solving.

The incidence rate of abdominal aneurysm continues to increase, but the exact pathogenesis is still conjectural. The most common theory is the aortic wall degeneration by the atherosclerotic process. A possible genetic basis alone or a genetic process leading to musculoelastic destruction of the aortic wall may be the problem.

Dr Zarins has stated that the experimental models have supported all of the hypotheses proposed in the pathogenesis of abdominal aortic aneurysms. He feels that plaque deposition and the subsequent atrophy of the media leads to localized destruction of the wall matrix. The successful obliteration of flow into the aneurysmal sac demands that the prosthesis be firmly anchored in the aortic wall, which is then not at risk for further aneurysmal dilatation.

Dr Matsumura cited the study that Illig and his colleagues from the University of Rochester presented at this meeting last year. It was a retrospective review of 33 patients in which they looked at the aortic cuffs for up to 7 years after open operation. The message that they left and that I got from this was that those with a proximal cuff of less than 27 millimeters at the time of operation did not exceed 30 mm in that follow-up. The distal cuff was at a much greater risk for dilatation.

A recent prospective analysis of 66 patients for successive infrarenal abdominal aneurysm with angiographic 3-dimensional with spiral computed tomography has been reported from the Netherlands. The anatomical neck was cylindrical in only 33% of the patients and had less than a 10-mm length in 17%. That selection criteria for an endovascular graft on the basis of the protocol set up by Endovascular Technologies, Inc, revealed that only 18% or 27% of the 66 patients would have qualified for an endovascular graft. All of these would have required a bifurcated graft. In addition, only 1 of 11 small abdominal aneurysms, which is less than 5 cm, had a more favorable anatomy than the larger aneurysms. This might confirm my theory of once a neck always a neck. Aortic necks may indeed enlarge over time either after operation or after endograph repair.

In my 35 years of vascular practice, proximal cuff dilatation not para-anastomotic aneurysms were rarely encountered. They were seen most frequently when we did not remove all of the diseased infrarenal abdominal aorta. And in those early years, as some in this audience recall, it was very uncommon for us to clamp above the renal arteries or to dissect at that level because our skills and our backup were not quite that good. In his text, Dr Matsumura cites Dr Crawford's data of 920 abdominal aortic aneurysms followed up to 7 years, but this was reported in 1981, and there was a significant change in the necks and a significant mortality rate. However, we must remember that these statistics in these patients are from the 1950s, 1960s, and early 1970s.

The ideal endovascular prosthesis should be low profile, thin walled, and have a fairly immediate hemostasis, a flexible attachment system, and a rigid body. This will allow for an enlargement necessary to maintain the fixation with the aorta and, at the same time, maintain the rigidity in the mid-component of the graft.

In the text, Dr Matsumura also mentions branch flow

within the aortic sac as a potential problem for continued enlargement. It would seem that with the current techniques available for embolization and other forms of obliteration, this will probably not be a major long-term source of problems for aortic enlargement.

I have these questions for Dr Matsumura. First, would you comment on computed tomography scan versus a 3-dimensional helical computed tomography scan, both on visualization for eligibility and follow-up? Secondly, with the problems in the distal cuff of tube grafts, should 2 grafts continue to be used or will the technology and precision of deployment improve to allow that continued use later? Would you comment as to whether you use kissing balloons now in these tube grafts? We found early on that this was probably going to be necessary to maintain the stability of that distal graft.

I enjoyed Dr Matsumura's presentation. I congratulate him on bringing this important information to us, and look forward to other reports as this technology continues to improve.

Dr Jon S. Matsumura. Thank you, Dr Edwards, for your comments and your questions. I think that they are both very much related, and we have much agreement on many of those points. I definitely would agree with you that the prosthesis must be firmly anchored, just as it is in an open repair—ideally, permanently anchored to the neck in some way. And it is important to note, which I did not in the presentation, that this trial excluded neck sizes that were over 28 mm, which, as you mentioned, were correlated with greater neck growth in the study reported from last year.

In reference to your first question regarding computed tomography scans versus the helicocomputed tomography scan, I am adamantly convinced that there is additional information to be gained from the 3-dimensional helicocomputed tomography scans, and I think that is probably the way that most centers are headed. The thinly cut sections with dynamic contrasts allow a good analysis of the branch patency and of the shape that you alluded to of the necks and also allows more precise measurement and sizing and allows characterization of some of those qualitative things, such as thrombus within the neck or heavy calcification, which often subjectively should deter you from placing an endograft. I think that branch flow will probably never go away completely, but I think it will get very small. So, there is still about a 2% to 3% reperfusion rate from those side branches even when you cut across the aorta, and I think a lot of those occur late. To get back to this issue of helicocomputed tomography scan, I think that not only do you have to do the helicocomputed tomography with thin cuts, but in your later follow-up, it is important to do delayed views after the dynamic infusion at 5, 10, or 15 minutes later, because it is in the late follow-up when those collateral vessels through the illio lumbars will dilate and reprofuse the aneurysm. Those will only be seen on a delayed computed tomography scan. There are obviously other ways to get at that with an ultrasound scan, possibly pulsatile wall motion, and magnetic resonance angiography. So, I think that, in summary, the helicocomputed tomography scans are a tremendous advance.

In reference to tube grafts, that is quite a question. I do not think that in the data in this study we have enough information to say that tube grafts should no longer be used or are still viable. We have experience at Northwestern University with both modular and a 1-piece bifurcated system, and I have to say that they have their own set of complications that accompany them. The tube graft is much simpler and has that virtue, and if we could use it we would. With that disclaimer, I think there is an ideal aneurysm that you would use for a tube graft, the so-called apple on a string, where the proximal and distal necks are cylindrical, long, and do not appear to contain any disease, which as you said, is important.

In reference to bifurcation grafts, there is an abstract in the British Journal of Surgery that cites a 6% incidence rate of late proximal migration in an older series. I think that we are going to start seeing more problems at the proximal point; it is just going to take longer. And obviously, a bifurcation graft is not going to be safe from that problem, unless you are planting at the renal vessels.

Finally, I do not think that the iliac arteries have ever been studied in as detailed a manner as this in regards to what happens distally at the iliac vessels in a bifurcated graft. And certainly that would have to be answered before we give up on tube grafts.

The last question was in reference to the kissing balloon technique. I think that certainly this is an advance for tube graft deployment at the distal point. Ideally, we would like to develop a balloon that has a square bottom or, if you want to be imaginative, a figure-8 bottom that would fit the iliac vessels. The problem is, as you have said with the kissing balloon, that you get poor pressure applied to the contralateral side than with just a single aortic balloon used from unilateral approach. So, I think that the kissing balloons is an improvement, but I think we have a long way to go in that technology as well.