

Elective Endovascular Stent-Graft Repair of Atherosclerotic Thoracic Aortic Aneurysms: Clinical Results and Midterm Follow-Up

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OBJECTIVE. The purpose of this study was to evaluate the clinical and midterm results after endovascular treatment of atherosclerotic thoracic aortic aneurysms.

MATERIALS AND METHODS. Twenty-eight consecutive patients who were 53–82 years old (mean age, 71.6 years) were treated with a commercially available endoprosthesis. Subclavian transposition or bypass surgery was performed before the procedure in eight patients. Size dynamics of the aneurysms were analyzed on the basis of diameter and thrombus volume measurements obtained on three-dimensional CT reconstructions before hospital discharge ($n = 22$) and at the 1-year ($n = 22$), 2-year ($n = 12$), and 3-year ($n = 5$) follow-ups.

RESULTS. The technical success rate was 100%. There was no 30-day mortality. None of the patients had symptoms due to spinal cord ischemia. The survival rate at 1, 2, and 3 years was 96.1%, 90.9%, and 80.2%, respectively. During the perioperative period, patients presented with leukocytosis (37%), fever (36%), elevated C-reactive protein value (92%), pleural effusion (50%), and periaortic atelectasis (41%). Three early type I endoleaks sealed spontaneously. Three early type II endoleaks persisted over time, and one late type II endoleak was detected. In patients with type II endoleaks, thrombus volume of the aneurysms was constant ($n = 2$) or increased ($n = 2$). In patients without endoleaks, mean thrombus volume decreased (-53.2 ± 56.8 mL, -40%) significantly ($p = 0.001$) during the first year. There was no significant interval decrease between the 1- and 2-year follow-ups (mean, -2.4 mL, $p = 0.92$) and between the 2- and 3-year follow-ups (mean, -0.4 mL, $p = 0.68$).

CONCLUSION. Endovascular treatment of atherosclerotic thoracic aortic aneurysms may result in a substantial reduction of the aneurysm sac in patients without endoleaks.

Untreated thoracic aortic aneurysms are likely to expand over time and rupture [1, 2]. Significant rates of expansion in aneurysms larger than 5 cm were reported in a series by Dapunt et al. [1]. Furthermore, analyses by Coady et al. [3] revealed a 43% probability for rupture in descending thoracic aortic aneurysms 7 cm or greater, and they recommended surgical repair when aneurysms reach a diameter of 6.5 cm.

Patients who require treatment are most commonly elderly and have additional substantial comorbidities. Despite remarkable improvements in the results of conventional open surgical repair, a combination of risk factors, including coronary artery disease, cerebrovascular disease, and poor pulmonary or renal reserve, may substantially increase the morbidity rate after surgical treatment [4–6].

Therefore, endovascular repair of thoracic aortic aneurysms as a less invasive alternative

to open surgical repair has become widely accepted for a selected population of patients. The initial results from several studies suggest that this new treatment modality may potentially reduce postoperative morbidity and mortality rates, as well as the duration of the hospital stay [7–13]. However, further mid- and long-term follow-ups are mandatory to determine the effectiveness of these stent-grafts in preventing dilatation of the aneurysm.

The purpose of our study was to evaluate the significance of volume and diameter measurements at midterm follow-up to determine changes in the size of the aneurysm after endovascular repair of atherosclerotic thoracic aortic aneurysms. Furthermore, the influence of endoleaks on aneurysm size during time intervals was evaluated, and changes in stent-graft diameters, as well as perioperative CT findings and clinical data, were assessed.

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Materials and Methods

Patient Population

From April 1998 to November 2001, 11 women and 17 men with atherosclerotic aneurysms of the descending thoracic aorta were electively treated with endovascular stent-graft repair. Patients were 53–82 years old (mean age, 71.6 years). The institutional review board approved the investigative protocol, and written informed consent was obtained from all patients.

Conjoint agreement on therapeutic interventions was obtained from cardiothoracic surgeons. Unsuitability for open surgical repair of 20 patients (71%) was based on either serious comorbidities, including hypertension ($n = 18$, 90%), coronary artery disease ($n = 8$, 40%), cerebral vascular disease ($n = 7$, 35%), renal impairment ($n = 5$, 25%), and poor pulmonary reserve ($n = 10$, 50%), or previous thoracic surgery ($n = 5$, 25%).

Evaluation for endovascular stent-graft repair was based on contrast-enhanced CT angiography using both single- (Somatom Plus 4; Siemens, Erlangen, Germany) and multidetector (Somatom Volume Zoom; Siemens) scanners. CT scanning ranged from the apex of the lung to the origin of the celiac trunk or the groin. To calculate the individual scan delay, volume, and flow rate for CT acquisitions, we initiated a bolus-optimized technique for the arterial phase, as described previously [14]. The calculated volume of contrast medium (iopamidol, Iopamiro 300; Bracco, Milan, Italy) varied between 70 and 120 mL (mean, 103 ± 17 mL) with biphasic flow rates of 3–12 mL/sec.

For the Somatom Plus 4 scanner, acquisition parameters were a slice collimation of 3 mm and a table increment of 6 mm/360° gantry rotation (gantry rotation time, 1 sec). Transverse sections were reconstructed at 2-mm intervals.

Acquisition parameters for the Somatom Volume Zoom scanner were a slice collimation of 4×1 mm and a table increment of 6 mm/360° gantry rotation (gantry rotation time, 0.5 sec). Transverse images of 3-mm section thickness were reconstructed at 2-mm intervals. CT scans were processed with three-dimensional reconstructions, which included multiplanar reconstruction, curved planar reconstruction, and maximum intensity projection. For evaluation of the iliac and common femoral arteries, we obtained an angiogram using a calibrated pigtail catheter or a helical CT angiogram.

Endovascular Prosthesis

All patients were treated with the Excluder endoprosthesis (W. L. Gore & Associates, Flagstaff, AZ). The endoprosthesis consists of a self-expanding nitinol stent, in which the inner surface is lined with expanded polytetrafluoroethylene. The stent-graft is constrained on the delivery catheter by a lacing fiber. Withdrawal of the lacing fiber releases the device from the middle portion to both ends. Depending on the diameter of the device, a 22- or 24-French vascular introducer sheath is required. The diameter of the stent-graft was calculated from the

largest diameter of the proximal or distal neck, and an oversizing factor of 10–20% was added.

Endovascular Procedure

In our institute, surgeons, anesthesiologists, and interventional radiologists take an active hand in endovascular stent-graft procedures. All procedures were performed in the angiography suite, which is equipped with digital subtraction angiography (Multistar T.O.P.; Siemens) with an overlay imaging technique. Treatment was performed with the patient under general anesthesia ($n = 17$, 61%) or epidural anesthesia ($n = 11$, 39%). Access sites were created by surgical cut-down of the femoral artery ($n = 16$, 57%) or the iliac arteries ($n = 12$, 43%). Before introduction of the stent-graft system, 3000–5000 IU of heparin sodium was administered IV, and 2 mg of ceftriaxone (Rocephin; Hoffman-LaRoche, Basel, Switzerland) was given prophylactically. The mean volume of the contrast agent given during the procedure was 232 ± 73 mL (range, 140–380 mL).

In patients who required close positioning of the stent-graft to the left subclavian or the left common carotid artery, a 5-French pigtail catheter was placed into the ascending thoracic aorta through a percutaneous brachial access. An angiogram was obtained immediately before stent-graft deployment. If close positioning of the stent-graft to the celiac artery was unavoidable, a sidewinder catheter (Simmons I; Cordis, Miami, FL) was positioned by a contralateral percutaneous femoral access into the origin of the celiac trunk to avoid overstenting. Stent-grafts were deployed to cover at least 1.5–2 cm of a relatively normal aortic segment proximally and distally to the aneurysm. To improve the expansion and the friction seal, we used a latex balloon (Thoracic Excluder balloon catheter; W. L. Gore & Associates) for modeling each end of the stent-graft to the aortic wall. Completion angiography was performed to confirm the correct position of the stent-graft, unimpaired aortic side branches, and absence of perigraft leakage.

Follow-Up

All patients were followed up clinically, and laboratory values of creatinine, C-reactive protein, and WBC on the first day after the procedure and before hospital discharge were compared with data obtained before the procedure.

To rule out small endoleaks, we added late acquisitions (scanning started with a 30-sec interscan delay) during follow-up CT. CT angiography was performed before hospital discharge in all patients except one who had also undergone MR angiography. Twenty-four patients underwent CT again once or twice during the first year. Furthermore, 22 patients were followed up at 1 year, 12 patients at 2 years, and five patients at 3 years.

A total of 234 CT examinations were reevaluated. The presence or absence of perigraft endoleaks, pleural effusion, and periaortic atelectasis was assessed on CT before hospital discharge and compared with subsequent CT examinations.

Within 2 months, measurements for the evaluation of the thrombus volume on the first postoperative CT images and on the 1-, 2-, and 3-year follow-up CT scans were accomplished twice by one investigator with no knowledge of the previous values on a MagicView workstation (SIENET; Siemens). All images along the entire length of the aneurysm were selected from the CT study and processed with multiplanar reconstruction. A contour was hand-drawn on each slice along the outer border of the thrombus and segmented. In a second step, the vessel lumen and the stent-graft were segmented manually by drawing the contour along the outer border of the stent-graft. The thrombus volume of the aneurysm was obtained by subtracting the stent-graft volume from the total volume of the aneurysm. In addition, the maximal diameter of the aneurysm and the diameter at the proximal and distal end of the stent-graft were measured on multiplanar reconstructions perpendicular to the axis of the vessel. Distances from the proximal and the distal stent to the left subclavian artery and the celiac trunk, respectively, were assessed.

Definitions and Statistical Analysis

For statistical analysis, a minimum follow-up of 1 year (≥ 11 months) was required (range, 11–47 months; mean, 22.7 months). The dependent variables were the diameter and the thrombus volume of the aneurysm sac at each postinterventional control.

For the definition of a significant change in thrombus volume, the intraobserver variability was calculated by the difference of means analysis as described by Bland and Altman [15]. The repeatability coefficient calculated by this method was 13.4 mL for the thrombus volume measurements. Therefore, it can be expected that a difference between two measurements beyond ± 13.4 mL derives from a true change in thrombus volume rather than from measurement scatter.

For the aneurysm diameter measurements, intra- and interobserver repeatability coefficients of less than 4 mm were recorded in a series by Wever et al. [16]. Related to this result, changes in thoracic aortic aneurysm diameters were determined to be significant when there was a difference of 4 mm or greater compared with the postoperative CT.

Differences between the mean measurements were assessed for significance with the paired Student's *t* test. A *p* value of less than 0.05 was considered significant. All statistical calculations were performed using 10.0 software (SPSS, Chicago, IL). Continuous data are presented as mean values \pm standard deviations.

Results

Preinterventional Data

The mean maximal diameter of the aneurysm sacs was 65.6 ± 11.3 mm (range, 50–96 mm). The average of the distances between the left subclavian artery and the aneurysm was 53 ± 54.2 mm (range, 0–185 mm), with a length of the proximal neck of less than 15 mm in

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eight patients. To allow a sufficient proximal landing zone, we performed a subclavian-to-carotid transposition ($n = 6$), a left subclavian-to-carotid bypass ($n = 1$), or a bifurcated prosthesis from the ascending aorta to the left carotid and subclavian artery ($n = 1$) in eight patients (28.6%) as a separate procedure before stent-graft treatment. The mean distance from the aneurysm outlet to the celiac artery was 98 ± 62.2 mm (range, 18–243 mm). In addition, eight patients (28.6%) had an abdominal aortic aneurysm, with previous surgical repair in five.

Perioperative Findings

A total of 58 stent-grafts with a mean diameter of 37.1 mm (range, 31–40 mm) and a length between 10 and 20 cm were successfully deployed. The origin of the left subclavian artery was completely covered in the eight patients with a previous transposition or bypass surgery. In four additional patients, the orifice of the subclavian artery was partially covered without ischemic symptoms of the arm or the posterior circulation. In the remaining patients, the mean distance from the proximal attachment site to the subclavian artery was 47 ± 49.1 mm (range, 0–136 mm), and the covered aortic segment proximal to the aneurysm had a mean length of 34 ± 15.3 mm (range, 11–63 mm). The mean distance from the distal attachment site to the celiac artery was 36 ± 48.5 mm (range, 0–136 mm), and the covered aortic segment distal to the aneurysm had a mean length of 45 ± 23.1 mm (range, 15–105 mm).

After the procedure, patients were transferred to the recovery room where they had a mean stay of 9.5 ± 10.3 hr (range, 3–48 hr). The mean hospital stay was 9 ± 3.2 days (range, 4–20 days).

After stent-graft insertion, the mean elevation of the WBC value was $2.6 \pm 2.11 \times 10^9/L$, with newly developed leukocytosis ($WBC > 10.0 \times 10^9/L$) in 10 (37%) of 27 patients and fever ($>38^\circ C$) in 10 (36%) of 28 patients. These findings improved in all patients by the time of hospital discharge. An elevation of the C-reactive protein value (>1 mg/dL) occurred in 24 (92%) of 26 patients. Compared with preinterventional C-reactive protein values (mean, 1.4 ± 1.9 mg/dL), the acute phase proteins were elevated substantial after the procedure (mean, 7.2 ± 4.8 mg/dL) and had a further significant increase by hospital discharge (mean, 11.2 ± 7.9 mg/dL). The mean serum creatinine value at baseline was 1.29 ± 0.91 mg/dL (range, 0.72–5.41 mg/dL) and remained constant (mean, 1.28 ± 0.9 mg/dL; $p =$

0.88) 24 hr after the procedure. Before hospital discharge, the mean value of creatinine was 1.24 ± 0.95 mg/dL (range, 0.73–5.44 mg/dL).

Pleural effusion and periaortic atelectasis were evaluated in relation to the stent-graft procedure if patients were free of them before implantation and changes occurred only unilaterally. On the first CT examination before hospital discharge, a newly developed pleural effusion was observed in 12 (50%) of 24 patients and a periaortic atelectasis in nine (41%) of 22 patients. Pleural effusions were completely resolved on follow-up CT in 11 (92%) of 12 patients and atelectasis in seven (78%) of nine patients.

Complications

None of the patients had symptoms due to spinal cord ischemia. Minor complications occurred in three (11%) of 28 patients. One patient who had normal serum creatinine findings before the procedure (≤ 1.3 mg/dL) presented with a temporarily slight increase in the creatinine value without the need for hemodialysis. Two patients had complications related to the access site, with a prolonged duration of healing. Two patients (7%) had major complications. One patient who had a previous subclavian-to-carotid transposition sustained a posterior circulatory stroke and improved completely over time. Related to the access site, the second patient experienced postoperative bleeding that required blood transfusion.

Survival

None of our patients died within 30 days after the procedure. During the follow-up period, three patients died. One patient's death resulted from hepatic failure due to hepatitis 42 weeks after the procedure. The second patient's death was caused by cardiac failure 16 months after endovascular repair. The third patient's death was caused by metastatic carcinoma of the colon 34 months after stent-graft treatment. Cumulative survival was $96.1\% \pm 3.8\%$ at 1 year. At 2

and 3 years, the actuarial survival was $90.9\% \pm 6.2\%$ and $80.2\% \pm 11.5\%$, respectively.

Endoleaks

Completion angiograms revealed exclusion of the aneurysm in all patients except three (11%). Of these three intraoperatively detected type I endoleaks, two were successfully treated either with rebalancing of the proximal graft or an extension at the distal attachment site in a second procedure. One proximal type I endoleak had already sealed before the first CT examination after the intervention. Type II endoleaks were not seen on the completion angiogram.

CT performed before hospital discharge revealed five early endoleaks (18%). In two of these patients, a type I endoleak was present (2/28 patients, 7%). These sealed spontaneously during the first month. The three early type II endoleaks (3/28 patients, 11%) persisted at the 1-year follow-up. In addition, one newly occurring type II endoleak, not evident on CT angiography but resulting in a remarkable increase in the aneurysm, was shown on MR angiography at the 2-year follow-up. This patient was successfully treated with embolization of the intercostal arteries using *N*-butyl-2-cyanoacrylate (Glubran; GEM, Viareggio, Italy) mixed with ethiodized oil (Lipiodol Ultra-Fluide; Guerbet, Zurich, Switzerland).

Follow-Up Diameter Measurements

During the follow-up of patients with either an early or late detected type II endoleak, the diameter measurements revealed an increase in the aneurysm in one patient and a constant size in three patients (Table 1).

In patients without an apparent endoleak at any time, the mean diameter decrease in the aneurysm was 6.6 ± 6.4 mm (-10.4% , $p = 0.001$) at the 1-year follow-up (Table 2 and Fig. 1). During the first year, the aneurysm diameter decreased in 12 (67%) of 18 patients, was unchanged in four (22%), and increased in two (11%). There was no significant interval de-

TABLE 1 Changes in Diameter and Thrombus Volume in Four Patients with Type II Endoleaks at Follow-Up

Patient No.	Changes in Diameter (mm) Between Intervals		Changes in Thrombus Volume (mL) Between Intervals		Changes in Size (mL)
	0–1 yr	0–2 yr	0–1 yr	0–2 yr	
	1	5 (5.3)	16 (17)	30 (10.5)	
2	2 (3.3)	—	2 (3.7)	—	Unchanged (± 13.4)
3	-1 (-1.9)	2 (3.7)	2 (5.4)	2 (5.4)	Unchanged (± 13.4)
4	-2 (-2.9)	3 (4.4)	1 (1.1)	15 (16.5)	Increase (>13.4)

Note.—Data in parentheses for diameter and thrombus volume are percentages. Dash (—) indicates data are not available.

TABLE 2 Changes in Diameter of Aneurysm in Patients Without Visible Endoleak at Follow-Up

Variable	Total Diameter of Aneurysm				Changes in Diameter Between Intervals		
	0 yr ^a	1 yr	2 yr	3 yr	0–1 yr	0–2 yr	0–3 yr
No. of patients	18	18	9	5	18	9	5
Mean ± SD (mm)	63.9 ± 8.3	57.3 ± 10.1	54.9 ± 11.1	54.0 ± 14.9	-6.6 ± 6.4 ^b	-9.2 ± 6.9 ^b	-9.8 ± 7.6 ^b
Mean ± SD (%)					-10.4 ± 9.8	-14.8 ± 10.5	-16.7 ± 13.0

^aFirst postoperative measurement.^b $p < 0.5$.

crease in the diameters between the 1- and 2-year follow-ups (mean, 0.1 ± 3 mm, $p = 0.7$) and the 2- and 3-year follow-ups (mean, -0.8 ± 3.7 , $p = 0.91$). During the second year, the diameter of the aneurysm was constant in eight (89%) of nine patients and increased in one (11%). Measurements at the 3-year follow-up showed a constant diameter in three (60%) of five patients, a decrease in diameter in one (20%), and an increase in diameter in one (20%).

The 1-year follow-up revealed a significant increase in the stent-graft diameters at the proximal end (mean, 1.9 ± 1.6 mm) and the distal end (mean, 1.3 ± 1.6 mm). No significant increase in stent-graft diameter occurred during the second and third years.

Follow-Up Volume Measurements

With regard to the four patients who presented with an early or late type II endoleak, the volume of the aneurysm sac was unchanged in two patients and increased in two (Table 1).

In 18 patients without an apparent endoleak (Fig. 2), the mean decrease in thrombus volume was 53.2 ± 56.8 mL (-40% , $p = 0.001$) at the 1-year follow-up (Table 3 and Fig. 3). During the first year, the volume decreased in 13 (72%) of 18 patients, was unchanged in four (22%), and increased in one (6%). There was no significant interval decrease of the thrombus volume between the 1- and 2-year follow-ups (mean, -2.4 ± 17.1 mL, $p = 0.92$) and between the 2- and 3-year follow-ups (mean, -0.4 ± 6.5 mL, $p = 0.68$). During the second year, the volume decreased in two (22%) of nine patients, was constant in six (67%), and increased in one (11%). Measurements obtained at the 3-year follow-up showed a constant thrombus volume in five of five patients.

When changes in thrombus volume and maximal diameter were compared during time intervals in all 38 follow-up measurements, discordant results were found in seven instances (18%).

Discussion

Since the first report of endovascular repair of a thoracic aortic aneurysm appeared in 1994 [17], this technique has become a generally accepted alternative therapy to open surgical repair. Although there is considerable interest in this new treatment modality, to our knowledge, the short-term clinical and radiologic findings after stent-graft therapy for thoracic aortic aneurysms and the long-term effects have not, as yet, been clearly depicted.

After elective endovascular treatment of thoracic aortic aneurysms and dissections, a 30-day mortality rate of 0–13%, similar to perioperative mortality rates after surgical repair, has been reported [8, 11–13, 18]. However, the incidence of serious comorbid events, including pulmonary and renal insufficiency, is distinctly reduced for endovascular treatment compared with surgical repair [4–6]. In our series, we had no perioperative deaths, and none of our patients had pulmo-

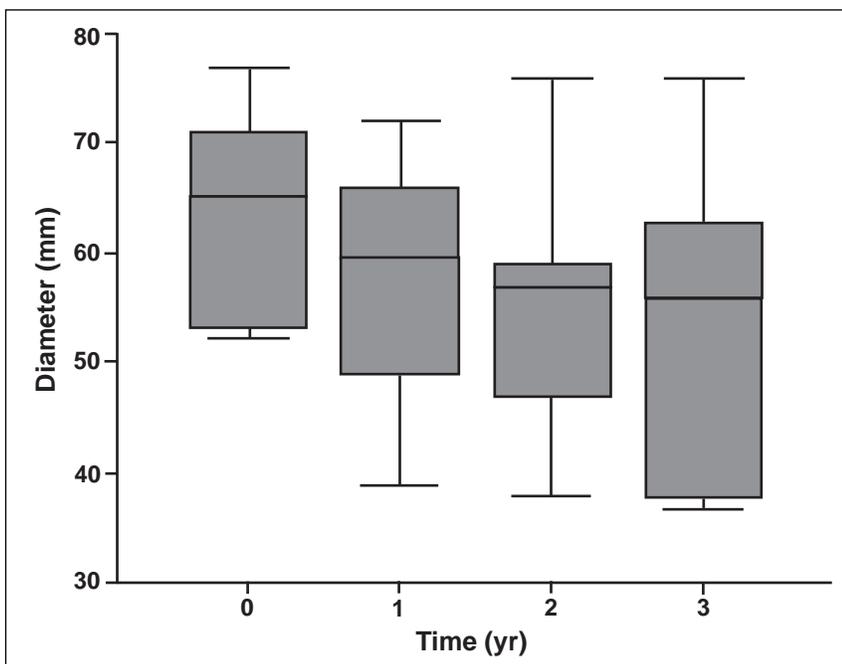


Fig. 1.—Box-and-whisker plot shows diameters of aneurysms measured before hospital discharge (0 year) and at 1-, 2-, and 3-year follow-ups. Center lines in boxes represent mean values; upper and lower ends of boxes represent 25th and 75th percentile values, respectively. Ends of error bars show extreme values.

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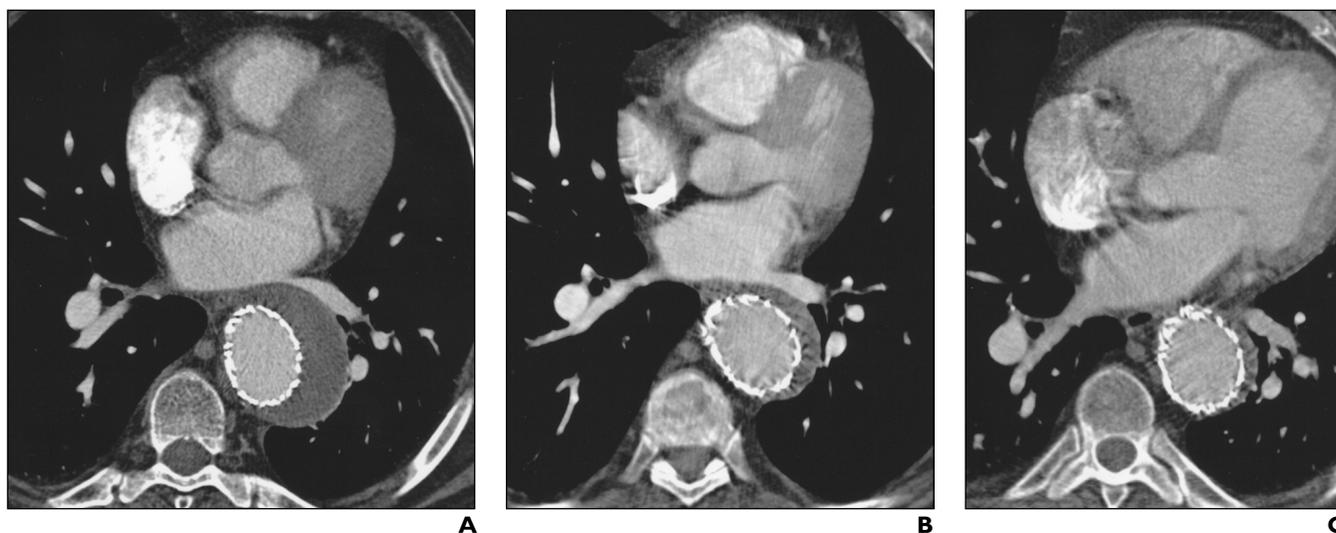


Fig. 2.—66-year-old man who presented with descending thoracic aortic aneurysm of 6.1 cm at maximal diameter. **A**, Contrast-enhanced axial CT scan obtained after stent-graft repair shows initial thrombus volume before hospital discharge to be 164 mL. **B** and **C**, Contrast-enhanced axial CT scans obtained at 1-year (**B**) and 2-year (**C**) follow-ups show thrombus volume to have decreased to 39 and 31 mL, respectively.

nary failure. Carlson et al. [19] reported renal dysfunction in 51% of their patients after surgical treatment of the descending aorta. Besides patient age, intraoperative hypotension and the use of simple aortic cross-clamping were significant predictors of renal dysfunction. The lack of these technical risk factors during endovascular aneurysm repair may explain the lower rate of postprocedural renal impairment. In our study, the one patient with a transient increase in the serum creatinine level had normal baseline serum creatinine findings. The five patients with preexisting renal impairment had no increase in their postprocedural serum creatinine levels. Postoperatively, most of our patients stayed in the recovery room for only a few hours. A prolonged hospital stay of more than 1 week was procedure-related in two patients.

Paraplegia or paraparesis was reported in up to 4% of patients in one study [20]. These researchers have suggested that patients who

require a long stent-graft to cover intercostal arteries at the T10 level are at higher risk. Although 14 of our patients fell into this risk category, with covered intercostals at T10 or a lower level, none of our patients experienced complications due to spinal cord ischemia.

In our study, newly developed leukocytosis, fever, and a significant elevation of C-reactive protein value after stent-graft repair was apparent in 37%, 36%, and 92% of patients, respectively. However, a definitive cause of infection for these findings could not be observed in any patient. Other authors have reported similar data after endovascular repair of abdominal and thoracic aortic aneurysms [9, 21–23]. We suggest that this syndrome may be attributable to a nonspecific systemic inflammatory reaction to the procedure and implant.

Furthermore, unilateral pleural effusion and periaortic atelectasis is a finding that is difficult to explain. When maximal diameters before stent-graft therapy and shortly thereafter are

compared, a slight increase in aneurysm diameter in 30% of the patients without evidence of an endoleak has been reported [9]. Therefore, we speculate that either a slight expansion of the aneurysm immediately after stent-graft implantation or the reactive inflammatory processes may result in a reactive pleural effusion and periaortic atelectasis in some instances [9].

Shrinkage of the aneurysm sac in the absence of an endoleak is considered indicative of a successful endovascular aneurysm repair. Stent-graft-related endoleaks (types I and III) are thought to cause a substantial increase in aneurysm size [7, 24–27]. Aneurysms with such endoleaks are at continued risk for rupture. These endoleaks are clearly detectable on CT [28, 29]. There is general agreement that, in these cases, early reintervention is necessary [8–10, 18, 25]. However, because of the ongoing tendency of the stent-graft to expand, small type I endoleaks may seal spontaneously. Thus, two of our patients with

TABLE 3 Changes in Thrombus Volume in Patients Without Visible Endoleak at Follow-Up

Variable	Thrombus Volume of Aneurysm				Changes in Thrombus Volume Between Intervals		
	0 yr ^a	1 yr	2 yr	3 yr	0–1 yr	0–2 yr	0–3 yr
No. of patients	18	18	9	5	18	9	5
Mean ± SD (mL)	128.6 ± 62.8	75.4 ± 49.9	49.6 ± 42.2	49.6 ± 45.8	−53.2 ± 56.8 ^b	−55.0 ± 46.6 ^b	−49.2 ± 45.0 ^b
Mean ± SD (%)					−40.2 ± 36.9	−52.6 ± 45.1	−51.3 ± 54.2

^aFirst postoperative measurement.

^b $p < 0.5$.

delayed sealing of an attachment leak had a significant decrease of the thrombus volume at the 1-year follow-up.

There are no final recommendations about the utility of follow-up measurements in the assessment of aneurysm sizes after endovascular repair. In almost all series, changes in aneurysm size are usually assessed by diameter measurements [18, 21, 24]. However, thoracic aortic aneurysms can be fusiform and irregularly shaped, saccular with bizarre configurations, and in some instances, angulation of the stent-graft may result in changes in aneurysm morphology [12, 24] during the postinterventional course. Therefore, measurement of cross-sectional diameters may not take into account all the changes in the shape of the aneurysm sac.

In recent studies [25, 30–32], volumetric analyses appear to be more accurate in the assessment of the natural history after endovascular repair of abdominal aortic aneurysms. However, little is known about the role of volume measurements for follow-up examinations after stent-graft repair of thoracic aortic aneurysms [12, 25].

Endoleaks related to branch vessel retrograde perfusion of the aneurysm sac (type II endoleak) after repair of abdominal aortic aneurysms observed at any time after intervention have been reported in 8–35% of cases [27, 33]. Most abdominal aortic aneurysms with persistent collateral perfusion of the sac remained unchanged in size [33] but

carry the risk of rapid aneurysm expansion and rupture [27].

In contrast, after endovascular repair of descending thoracic aortic aneurysms, type II endoleaks seem to occur less frequently in almost all reported series [8, 12, 13, 24]. In our study population, four patients presented with a type II endoleak, of which one finally was seen with MR angiography, including late MR acquisitions. Occasionally, retrograde perfusion of the aneurysm sac by branch vessels is hard to see. A possible reason for the apparently higher rate of type II endoleaks in this series was the fact that late CT acquisitions were routinely performed, which yielded a higher detection rate for endoleaks [34]. Furthermore, contrast-enhanced MR angiography might even increase the detection rates of type II endoleaks, possibly because of the higher contrast-to-noise ratio of gadolinium compared with the iodinated contrast medium used for CT angiography [28, 35].

Dake et al. [8] found that, in the absence of a detectable endoleak, the size of thoracic aneurysms decreased in 48% of patients, were unchanged in 22%, and increased in 22% at a mean follow-up of 1.1 years. Our measurements obtained at the 1-year follow-up of patients without visible endoleaks revealed a substantial decrease in aneurysm diameter and thrombus volume of the aneurysm sac in 67% and 72%, respectively. With regard to both types of measurements, a constant size was found in four patients (22%), and an in-

crease in the aneurysm sac occurred in two patients (11%).

Comparing all our measurements obtained during the follow-up period, discordant results between diameter changes and thrombus volume changes were observed in 18%. This result is less substantial than the discordance of 37% between both types of measurements reported by Wever et al. [31]. However, the measurement technique must be reproducible to assess the rate of aneurysm expansion with or without endoleaks on sequential studies. It has been shown that volume measurements are the most sensitive measure of aneurysm size and have less variability compared with maximal diameter measurements [16, 30]. However, there are substantial drawbacks to this approach. Volume measurements are time-consuming and require accurate segmentation of the aneurysm sac and the stent-graft on each cross section. Further, according to the law of Laplace, the tangential stress on the aneurysm wall is directly proportional to the diameter of the aneurysm, and evaluations of aneurysm expansion with the risk for rupture were primarily based on traditional transverse diameter measurements [1, 36].

Our midterm findings after endovascular treatment of atherosclerotic aortic aneurysms show promising results and a decrease in size in two thirds of patients who were free of endoleaks. However, the small number of patients in this series does not allow the identification of independent risk factors for the development of

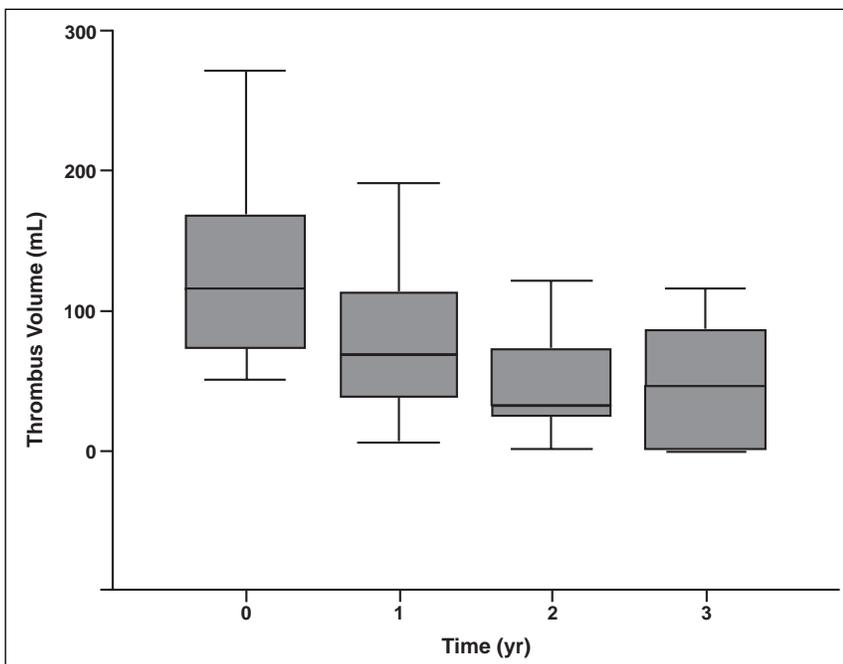


Fig. 3.—Box-and-whisker plot shows thrombus volume of aneurysms measured before hospital discharge (0 year) and at 1-, 2-, and 3-year follow-ups. Center lines in boxes represent mean values; upper and lower ends of boxes represent 25th and 75th percentile values, respectively. Ends of error bars show extreme values.

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endoleaks or the factors that influence size dynamics of the aneurysm sac.

In conclusion, endovascular stent-graft therapy of atherosclerotic thoracic aneurysms is technically feasible, with low mortality and morbidity. Patients with serious comorbidities and a high operative risk may benefit the most from this less invasive treatment modality. However, the occurrence of aneurysm expansion with or without a visible endoleak remains a cause for concern. Further evaluation of this new technique is indicated to determine the appropriate algorithm for follow-up measurements and the proper time for reintervention.

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