Evaluation of volumetric measurements in patients with acute type B aortic dissection – thoracic endovascular aortic repair (TEVAR) vs conservative

Iris Eva Chemelli-Steingruber, MD,[a] Andreas Chemelli, MD,[a] Alexander Strasak, MD,[b] Beate Hugl, MD,[c] Renate Hiemetzberger, MD,[d] and **Benedikt V. Czermak, MD,[a]** *Innsbruck, Austria*

Objective: **The aim of this retrospective study was to evaluate aortic volume changes in patients with acute type B aortic dissection (TBD), treated either by thoracic endovascular aortic repair (TEVAR) or conservatively.**

Materials and Methods: **From July 1996 through March 2008, 76 patients presenting with acute TBD were referred to our department. To ensure a follow-up of at least 24 months, only 64 of them were included in the present study, with the cut-off for inclusion being March 2006. Twenty-nine of these patients underwent TEVAR and 35 patients underwent conservative treatment. Indications for TEVAR were life-threatening symptoms. Follow-up was performed postinterventionally in patients after TEVAR and at 3, 6, and 12 months, and yearly thereafter in both groups. It included clinical examinations, computed tomography (CT) scans, analysis of volume changes in true thoracic lumen (TTL), false thoracic lumen (FTL), thoracic lumen (TL), abdominal lumen (AL), and aortic diameter measurements. In addition, the extent of thrombosis and its influence on volume changes were assessed.**

Results: **Mean follow-up was 41 months after TEVAR and 46 months in the conservatively-treated patients. At 60 months, cumulative rates of freedom from dissection-related death and rupture-free survival were 82.6% and 93.1% in the TEVAR group, respectively. They were 74.9% and 88.5% in the conservatively-treated group, respectively. In the conservatively-treated patients, 3 patients died of late aortic rupture, 4 were converted to open surgery, and 2 to TEVAR. Evaluation of volume changes showed better results in the TEVAR group within 24 months. However, within 60 months the difference between the two groups was no longer relevant. Relating to thrombosis of the FTL, analyses showed slightly better overall results and promotion of thrombus formation after TEVAR. However, at 60 months the results showed a tendency towards approximation between the two groups.**

Conclusion: **Our data suggest that TEVAR seems to delay the natural course of the disease but not to stop it. (J Vasc Surg 2009;49:20-8.)**

Acute type B aortic dissection (TBD) is defined as any nontraumatic dissection involving the descending aorta that appears within 14 days after the onset of symptoms.[1] The treatment of choice for acute TBD remains a matter of debate in the scientific community, but there is widespread consensus that in-patients with uncomplicated acute TBD conservative therapy is superior to open surgery.[2-4] However, persistent pain, aortic dilatation, drug-resistant hypertension, or dissection-related complications, such as rupture, impending rupture, and end-organ ischemia are clear indications for immediate intervention. As surgery continues to result in high mortality rates,[5-8] depending on the complexity of the aortic dissection, thoracic endovascular aortic repair (TEVAR) has been emerging as a less invasive and safe alternative to conventional surgery in patients with aortic disease. For more than a decade now, many authors have documented their experience with TEVAR.[5,9-13]

From the Department of Radiology,[a] Department of Medical Statistics, Informatics, and Health Economics,[b] Department of Vascular Surgery,[c] Department of Cardiology,[d] Innsbruck Medical University.

Competition of interest: none.

Correspondence: Benedikt V. Czermak, MD, University Hospital Innsbruck, Department of Radiology, Anichstr. 35, A-6020 Innsbruck, Austria (e-mail: benedikt.czermak@i-med.ac.at).

0741-5214/$36.00

doi:10.1016/j.jvs.2008.08.062

It is well known that an increase in aortic diameter raises the risk of rupture, which is the most common cause of disease-related mortality.[14,15] Therefore, diameter and volume changes may be an important predictor for the outcome of different therapy strategies and for the risk of rupture. Most authors have used diameter measurements only in the follow-up of TBD.[16-18] We used both diametric and volumetric measurements.

In a previous study, we reported on our results in patients with acute TBD treated by TEVAR. This study included volumetric measurements and their correlation with the length of stent-graft-coverage and the number of re-entry points, but the focus was placed on clinical aspects.[19] The aim of the present retrospective study was to evaluate volume changes in patients with acute TBD, who underwent either TEVAR or conservative treatment, including volumetric measurements of the true thoracic lumen (TTL), the false thoracic lumen (FTL), the thoracic lumen (TL), and the abdominal lumen (AL) in both patient groups. In addition, thrombosis of the false lumen was assessed in both patient groups and the impact of thrombosis on aortic lumen changes was analyzed.

MATERIALS AND METHODS

Patients. From July 1996 through March 2008, a total of 76 patients were referred to our department with acute TBD. To ensure a follow-up of at least 24 months,

only 64 of them were included in the present study, with the cut-off for inclusion being March 2006. Thirty-five of them with uncomplicated TBD, including 11 women and 24 men (age range, 42-86 years; mean age, 66 years) received conservative treatment and 29 patients including 8 women and 21 men (age range, 35-89 years; mean age, 64 years) underwent TEVAR. The decision for TEVAR was made by a team of vascular and cardiovascular surgeons, anesthesiologists, and interventional radiologists. Indications for TEVAR were intractable pain (n = 12, 41.4%), aortic branch compromise resulting in end-organ ischemia (n = 11, 37.9%), rapid aortic dilatation (n = 4, 13.8%), and rupture (n = 2, 6.9%).

Intractable pain was defined as ongoing chest pain despite conservative therapy[18] and rapid aortic dilatation as a diameter increase of more than 10% within 14 days after the onset of symptoms. Endovascular treatment was performed between 1 and 336 hours (mean, 50 hours) after admission.

Extent of the dissections. In the patient group who underwent TEVAR, the dissection extended into the iliac arteries in 17 patients, into the abdominal aorta in 7 patients, and in 5 patients it was limited to the thoracic aorta. In the conservatively-treated patients, the dissection extended into the iliac arteries in 17 patients, into the abdominal aorta in 17 patients, and in 1 patient it was limited to the thoracic aorta.

Diagnostic work-up. The diagnosis of dissection was established on the basis of computed tomography (CT) scans and/or angiography and clinical examination. In each patient, contrast-enhanced helical CT with three-dimensional (3D) vascular reconstruction from the apex of the thorax down to the groin was obtained immediately after admission and diagnostic angiography at the time of stent-graft insertion. These studies provided the needed information on length and diameter of the aortic lesion and anchoring sites and about involvement of important thoracic and abdominal branches, as well as the anatomy of the vessels used for access.

Up to May 1999, CT examinations were performed using a single detector computed tomography (SDCT) scanner and from May 1999 to June 2006 using a four-row multi-slice scanner. Since June 2006, data have been acquired from a 64-row multidetector computed tomography (MDCT) scanner (VCT, GE Medical Systems, Milwaukee, Wis) using a slice thickness of 0.625 mm with pitch 0.98 in the standard reconstruction kernel. Scans were obtained using 120-150 mL of a nonionic contrast agent (Ultravist, Schering, Berlin, Germany) administered at a concentration of 300-370 mg I/mL and a flow rate of 4 mL/second. The raw data were transferred to an independent workstation (Sun Ultra 60, Sun Microsystems, Mountain View, Calif) running the Advantage Windows software (AW 4.0, GE Medical systems, Milwaukee, Wis) for calculating 3D reconstructions.

Follow-up protocol. Conservatively-treated patients were closely monitored by multiple CT scanning during hospitalization. Patients treated by TEVAR were scanned postinterventionally. After discharge, the follow-up protocol included clinical examination and enhanced spiral CT scans at 3, 6, and 12 months, and yearly thereafter in both groups. Additional scans and/or digital subtraction angiography (DSA) were performed as needed to address specific problems. The 3D volume rendering reconstructions of the graft and the aorta were performed in order to detect possible aneurismal degeneration of the aorta or distortion and/or migration of the grafts. For the evaluation of volume changes, the follow-up studies were compared to the postinterventional studies in patients after TEVAR and to the studies performed on admission in the conservatively-treated group, respectively.

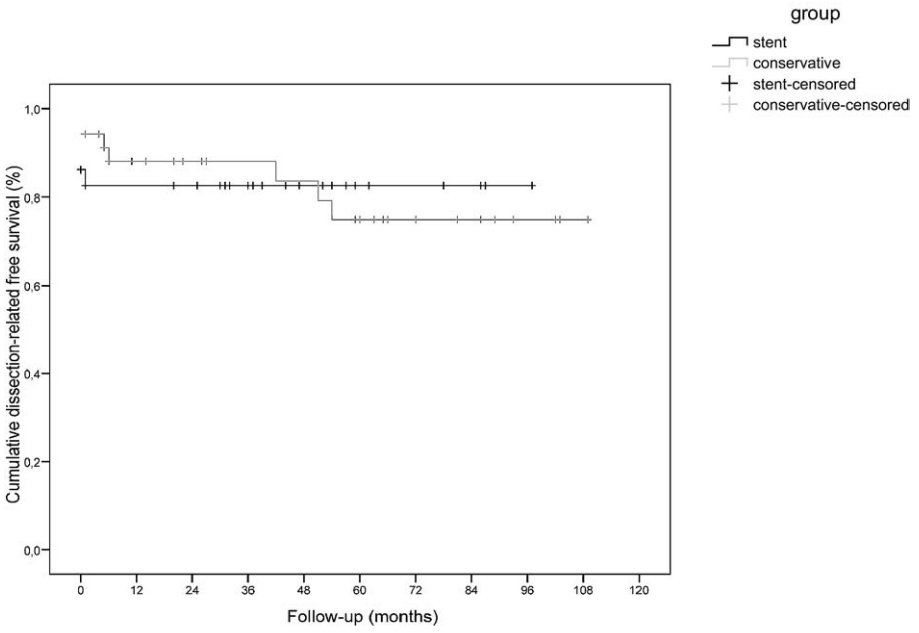
Diametric and volumetric measurements. The maximal aortic diameter was measured by hand on axial CT slices. To avoid overestimation of the aortic diameter due to tortuosity, diametric measurements were obtained perpendicular to the maximal diameter of the aorta.[20]

For volumetric measurements, which take about 20 minutes for each CT examination, the data obtained from the CT scans were transferred to a dedicated workstation (Sun Ultra 60, Sun Microsystems, Mountain View, Calif) running the Advantage Windows software (AW 4.0, GE Medical systems, Milwaukee, Wis) and using digital imaging and communications in medicine (DICOM)-coded data sets. The volumes of interest were outlined on each CT slice manually with the cursor of the workstation. After this procedure, volumes were assessed using the summation of area technique. Further technical details of measurements were described previously.[21] Volumes were expressed in milliliters. The TTL, the FTL, and the TL were measured between the origin of the left subclavian artery and the origin of the celiac trunk. The AL was measured between the origin of the celiac trunk and the aortic bifurcation.

In addition to the mean volume changes, we also documented the number of patients who showed volume increase, volume decrease, or volume stagnation of the TL, respectively. The cut-off was 10%.

Aneurismal dilatation of the aorta in the area immediately adjacent to the SG was defined as marked circumscribed dilatation of the aorta (volume >25% compared to the segments proximal and distal to the dilated segment).

Patient selection for statistical analysis of diametric and volumetric measurements. Of the 64 patients who were included in the study, 6 were lost to follow-up, 12 died within 0-407 days, 2 were converted to TEVAR, and 3 had to be converted to open surgery 1, 20, and 22 months after admission. The remaining 41 patients, including 20 patients treated by TEVAR (age range, 46-83 years; mean age, 65 years) and 21 conservatively-treated patients (age range, 42-80 years; mean age, 63 years) had a complete and uninterrupted follow-up of 24 months. In 29 of them, including 11 patients treated by TEVAR (group A, age range, 46-81 years; mean age, 62 years) and 18 conservatively-treated patients (group B, age range, 42-75 years; mean age, 63 years) complete and uninterrupted follow-up



Months	0	12	24	36	48	60	72	84	96	108	120
Patients at risk (stent)	29	23	21	16	12	8	5	4	2	0	0
Patients at risk (conservative)	35	26	22	20	19	16	9	8	4	2	0

Fig 1. Kaplan-Meier estimates of cumulative rates of freedom from dissection-related death in patients treated for acute type B dissection, either by thoracic endovascular aortic repair or conservatively.

was 60 months. Only groups A and B are shown in the statistical analysis of volumetric/diametric measurements and thrombus assessment.

Thrombus assessment. In both groups, changes in the degree of thrombosis were evaluated by comparing the first available CT scan (the preinterventional scan in group A and the scan obtained on admission in the conservatively-treated patients, respectively) to that at 60 months. In addition, the preinterventional CT scans were compared to the postinterventional scans to show the impact of TEVAR on the degree of thrombosis in group A. Missing thrombosis was identified by opacification of the FTL by contrast agent, partial thrombosis by the presence of contrast and thrombus in the FTL, and complete thrombosis by the absence of contrast and complete occlusion of the FTL by a thrombus.

Statistics. Continuous data were expressed using means ± standard deviation (SD) and medians; categorical data were shown as frequencies. To assess normal distribution in volumetric and diametric measurements, Kolmogorov-Smirnov Test was applied. As the data showed significant departure from Gaussian distribution, nonparametric tests were used throughout the analyses. Friedman-Test and Wilcoxon-Test were applied to investigate longitudinal differences in volumetric and diametric measurements within groups. Cumulative rates of rupture-free survival and freedom from dissection-related death were evaluated by means of Kaplan-Meier estimates. The χ^2 tests were used to compare the influence of the degree of thrombosis on volume changes in the TL (volume decrease of more than 10%, volume stagnation, and volume increase of more than 10%). Two-sided P values of $< .05$ were considered statistically significant. All statistical analyses were conducted using SPSS 15.00 (SPSS Inc, Chicago, Ill) statistical software.

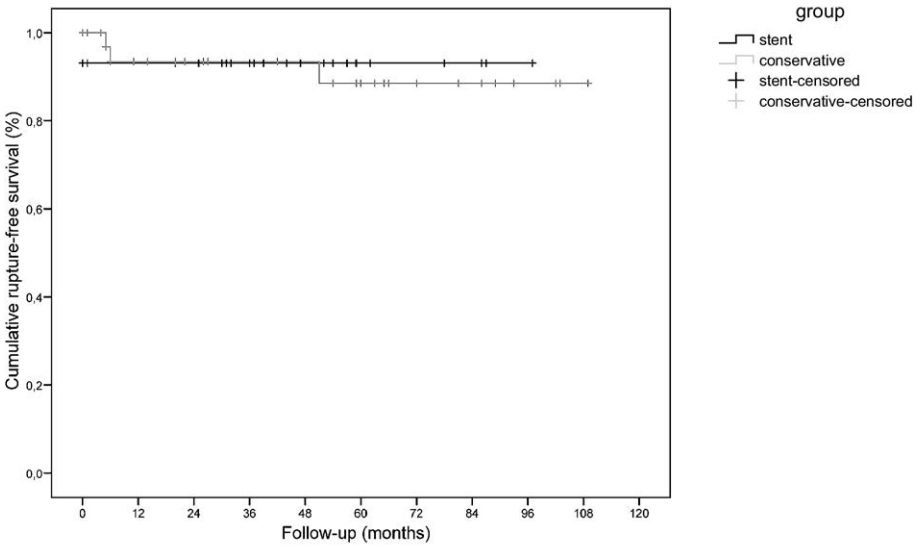
The first available volumetric result, that means the postinterventional result after TEVAR and the result obtained on admission in the conservatively-treated patients, respectively, were defined to be 100%. All following measurements were expressed in percentages and referred to 100%. The same method was applied to diametric measurements.

RESULTS

Mean follow-up was 41 months (range, 0-97 months) in patients after TEVAR and 46 months (range, 0-109 months) in the conservatively-treated patients.

Freedom from dissection-related death and rupture-free survival. In the patients treated by TEVAR, freedom from late dissection-related death was 82.6% (standard error [SE] 7.1%) at 1, 2, and 5 years (Fig 1) and rupture-free survival (Fig 2) was 93.1% (SE 4.7%) at 1, 2, and 5 years. There was no late rupture in this group.

In the conservatively-treated patients, freedom from late dissection-related death was 88.1 % (SE 5.6%) at 1 year and at 2 years and 74.9% (SE 8.5%) at 5 years (Fig 1) and



Months	0	12	24	36	48	60	72	84	96	108	120
Patients at risk (stent)	29	23	21	16	12	8	5	4	2	0	0
Patients at risk (conservative)	35	26	22	20	19	16	9	8	4	2	0

Fig 2. Kaplan-Meier estimates of cumulative rates of rupture-free survival in patients treated for acute type B dissection, either by thoracic endovascular aortic repair or conservatively.

rupture-free survival was 93.4% (SE 4.5%) at 1 year and at 2 years and 88.5% (SE 6.4%) at 5 years (Fig 2). There were three late ruptures in this group.

Rupture. In the conservatively-treated group, 3 patients died of aortic rupture, 5, 6, and 51 months after admission, respectively. In none of these patients did the CT scans performed during hospitalization show any aortic volume changes. In the first of them, the 3-month follow-up CT scan showed that aortic volumes had not changed compared to those seen on admission, that the maximal aortic diameter did not exceed 40 mm and that the FTL was not thrombosed. Aortic rupture occurred at the level of the kidneys a few days prior to the 6-month follow-up.

In the second patient, the scan on admission showed that, apart from the dissection, the aorta was enlarged along its entire course with the maximal aortic diameter measuring 80 mm and that the FTL was not thrombosed. Surgical treatment was not possible, as the patient was in severe medical condition and TEVAR could not be performed, because none of the SGs available has a diameter of more than 46 mm. The patient missed the 3-month follow-up and aortic rupture occurred within the descending aorta also a few days prior to the 6-month follow-up.

The patient who died of aortic rupture 51 months after admission was followed as mentioned above. The CT scans at the 48-month follow-up revealed volume increase of up to 122% in the TTL, of up to 262% in the FTL, and of up to 181% in the TL. The maximal aortic diameter had increased from 35 mm on admission to 47 mm at the 48-month follow-up and the FTL was partially thrombosed. Although, the patient was regularly informed about the necessity of surgical or endovascular treatment, he declined any further intervention.

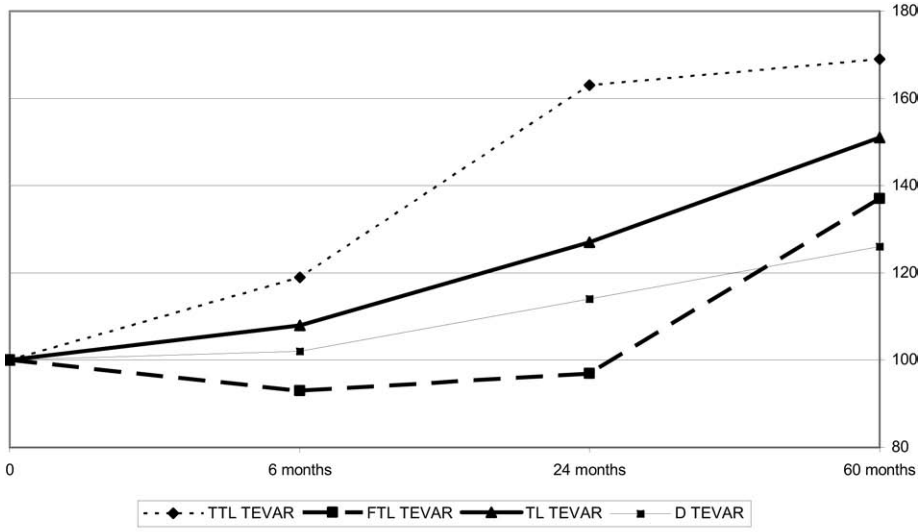
Conversions to open surgery or to TEVAR due to aortic dilatation. In the group treated by TEVAR, there were no conversions to open surgery. As all patients in whom aortic dilatation indicated failure of TEVAR were in reduced medical condition, we preferred conservative management to surgical repair, because conversion from endovascular aortic repair to open surgery is associated with a high mortality.[22]

In the conservatively-treated patient group, 4 had to be converted to open surgery, 1, 20, 22, and 51 months, respectively, after the imaging performed on admission.

In the first 3 of them, CT scans were performed because of sudden onset of intractable pain. The maximal aortic diameter was 49, 48, and 42 mm, respectively. However, as these patients presented in good clinical condition and as we agree with Lopera et al,[15] who stated that endovascular treatment of chronic TBD is very difficult, they were referred to vascular surgery.

The patient who was converted to open surgery 51 months after his first CT has been described above. His aortic diameter was 47 mm at 48 months. Three months later, he presented with aortic rupture and, unfortunately, surgical therapy was not successful and he died.

Two of the conservatively-treated patients had to be converted to TEVAR due to rapid aortic dilatation, one of



TEVAR	Months	0	6	24	60
TTL	Mean	100	118	150	155
	SD	0	10	31	19
	Median	100	117	151	162
FTL	Mean	100	90	87	133
	SD	0	21	39	79
	Median	100	93	97	137
TL	Mean	100	107	122	149
	SD	0	12	24	39
	Median	100	108	127	151
D	Mean	100	102	109	124
	SD	0	7	17	34
	Median	100	102	114	126

Fig 3. Volume courses of true thoracic lumen, false thoracic lumen, thoracic lumen and diameter in patients treated by thoracic endovascular aortic repair. Mean values ± SD and medians of the different lumina and of the diameter in percentages are shown. *TTL,* True thoracic lumen; *FTL,* false thoracic lumen; *TL,* thoracic lumen; *D,* diameter; *SD,* standard deviation; *TEVAR,* thoracic endovascular aortic repair.

them at 4 months and the other one at 11 months after the first imaging.

One of them received 3 SGs, which were implanted in a telescope fashion, the other one received 1 SG. Total graft length was 205 mm (range, 160-250 mm). As the patients were in severe clinical condition, we preferred TEVAR to open surgery.

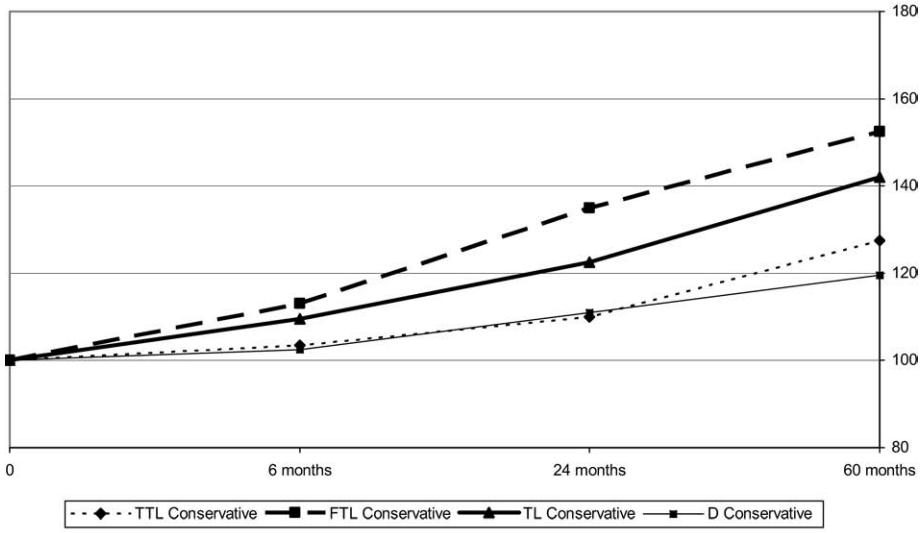
Volumetric analysis. Results of volumetric measurements are given in Figs 3-5 for both patient groups.

In group A, we observed a continuous and significant increase in TTL volume during the entire observation period ($P = .003$ from 0-6 months, $P = .003$ from 0-24 months and $P = .003$ from 0-60 months) and a non-significant decrease in FTL volume during the first 2 years ($P = .131$ from 0-6 months and $P = .477$ from 0-24 months), followed by a non-significant volume increase for the remaining observation period ($P = .286$ from 0-60 months). The TL volume increased continuously and significantly during the entire follow-up period ($P = .016$ from 0-24 months and $P = .008$ from 0-60 months) (Fig 3).

In group B, we observed continuous increase in TTL volume. However, this increase was significant only with regard to the entire follow-up period ($P = .001$ from 0-60 months), but not during the first 24 months ($P = .395$ from 0-6 months and $P = .058$ from 0-24 months). The FTL and the TL increased continuously and significantly during the entire follow-up period (FTL: $P = .006$ from 0-6 months, $P = .004$ from 0-24 months and $P = .001$ from 0-60 months, TL: $P = .002$ from 0-6 months, $P = .001$ from 0-24 months and $P < .0001$ from 0-60 months) (Fig 4).

The AL increased slightly in both groups. However, volume increase was more pronounced in group A than in group B (Fig 5).

In group A, TL volume increased by more than 10% in 9 patients (81.8%), it remained unchanged in 1 patient (9.1%) and in 1 patient (9.1%) it decreased.



Conservative	Months	0	6	24	60
TTL	Mean	100	102	110	138
	SD	0	14	18	41
	Median	100	104	110	128
FTL	Mean	100	117	136	167
	SD	0	23	46	63
	Median	100	113	135	153
TL	Mean	100	112	128	154
	SD	0	16	28	38
	Median	100	110	123	142
D	Mean	100	106	113	117
	SD	0	11	12	13
	Median	100	103	111	120

Fig 4. Volume courses of true thoracic lumen, false thoracic lumen, thoracic lumen and diameter in conservatively treated patients. Mean values ± SD and medians of the different lumina and of the diameter in percentages are shown. *TTL,* True thoracic lumen; *FTL,* false thoracic lumen; *TL,* thoracic lumen; *D,* xxx; *SD,* standard deviation.

In group B, TL volume increased by more than 10% in 16 patients (83.3%), it remained unchanged in 2 patients (27.3%) and it decreased in none of the patients.

Circumscribed aneurismal degeneration of the aorta immediately adjacent to the prosthesis was observed in 5 patients (13.5%).

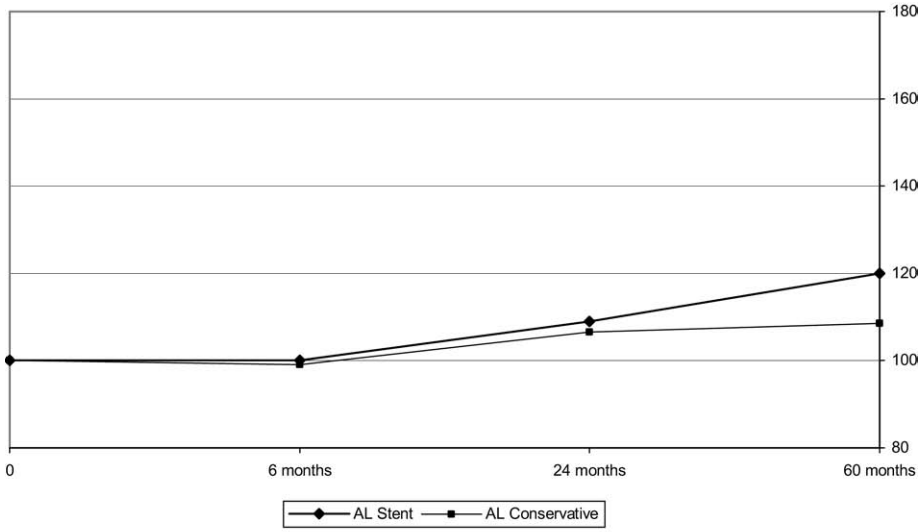
Diametric measurements. Mean values ± SD and medians are given in Table I. Diameters in percentages are shown in Figs 3-5.

Thrombosis. Thrombosis, if evident, was observed in the thoracic region only. The results are given in Table II. In most patients in group A, CT scans showed missing thrombosis pre-interventionally and partial thrombosis postinterventionally, but no relevant changes at 60 months. In most patients in group B, CT scans showed missing thrombosis on admission and partial thrombosis at 60 months. In none of the two groups was the association between the degree of thrombosis and volume outcome statistically significant.

DISCUSSION

Many studies have shown that TEVAR is a valuable tool in the therapy of complicated TBD with very encouraging short-term results. Life-threatening symptoms can be stabilized by TEVAR. However, it is of great importance, to evaluate mid- and long-term results. As aortic lumen changes are valuable prognostic factors for the outcome of the disease,[7,15,18,20,23] it is of great interest whether the volume course in patients treated by TEVAR is different from that in conservatively-treated patients.

Several authors have evaluated the applicability of volumetric measurements to the follow-up after endovascular repair.[21,24] It has been shown that intra- and interobserver testing for CT-guided volume measurements has a reproducibility coefficient of 5-10%[21] and that volumetric measurements are superior to simple diameter measurement in the follow-up of patients treated for aortic aneurysms by



AL	Months	0	6	24	60
Stent	Mean	100	103	112	118
	SD	0	15	32	42
	Median	100	100	109	120

AL	Months	0	6	24	60
Conservative	Mean	100	98	107	106
	SD	0	16	21	36
	Median	100	99	107	109

Fig 5. Volume courses of the abdominal lumen in patients treated for acute type B dissection, either by thoracic endovascular aortic repair or conservatively. Mean values ± SD and medians of the different lumina and of the diameter in percentages are shown. *AL,* Abdominal lumen; *SD,* standard deviation.

Table I. Maximal diameter in mm of groups A and B

	Months	*0*	*6*	*24*	*60*
TEVAR	Mean	43,7	44,6	47,6	53,7
n = 11 (group A)	SD	3,3	3,2	7,5	13,7
	Median	44,0	44,5	49,6	54,0
Conservative	Mean	40,8	43,4	46,1	47,2
n = 18 (group B)	SD	7,4	8,8	8,9	8,4
	Median	40,5	41,3	45,2	46,5

TEVAR, Thoracic endovascular aortic repair; *SD,* standard deviation.
Mean values ± SD and medians of the maximal aortic diameter in mm are shown.

means of endovascular repair.[25,26] However, volumetric measurements are time consuming and, therefore, difficult to practice in clinical routine. This may be the reason why most centers prefer diameter measurements.[3,4,17,18,27,28] We performed both volume and diameter measurements. The results showed similar courses, which indicates that diametric measurements are sufficient in the CT follow-up of patients with TBD.

In our series, TEVAR resulted in the stabilization of the aorta with significant increase in TTL volume and non-significant decrease in FTL volume within 24 months. Our results are in agreement with those of Kusagawa et al.[27] Their study included 17 patients with acute TBD and diameter measurements, which were performed at 1, 6, 12, and 24 months after TEVAR at different levels of the descending aorta, showed increase in TTL, and decrease in FTL diameters. Our analyses of the different aortic lumina also showed that the TL volume increase observed in both groups within the first 24 months was mainly due to a significant increase in TTL volume in group A, whereas in group B it was due to a significant increase in FTL volume. These results are of concern, as it is well known that an increase in FL volume raises the risk of aortic rupture[15,29] and that an FL aneurysm is prone to rupture because of its thin outer aortic wall.[30]

However, within 60 months, our results showed significant volume increase in all lumina in both patient groups, except for FTL volumes in patients treated by TEVAR, which showed non-significant increase. Within 60 months the difference between the two groups became less obvious. This tendency was also observed when analyzing the number of patients with volume increase or decrease of more than 10%, as documented in the RESULTS section. The approximation of the results indicates that TEVAR may delay but not stop the natural course of the disease.

Table II. Degree of thrombosis in the false thoracic lumen as seen on computed tomography scans on admission, postinterventionally and at the 60 months follow-up in patients treated by thoracic endovascular aortic repair and degree of thrombosis in the false thoracic lumen as seen on computed tomography scans on admission and at the 60 months follow-up in conservatively treated patients

Patients	*Degree of thrombosis on the first CT*	*%*	*Patients*	*Degree of thrombosis on the first CT after TEVAR*	*%*	*Patients*	*Degree of thrombosis at the 5 year follow-up*	*%*
TEVAR (n = 11)			TEVAR (n = 11)			TEVAR (n = 11)		
0	complete	0,0	1	complete	9,1	2	complete	18,2
3	partial	23,3	9	partial	81,8	9	partial	81,8
8	missing	72,7	1	missing	9,1	0	missing	0,0
Conservative (n = 18)						Conservative (n = 18)		
0	complete	0,0				2	complete	11,2
5	partial	27,8				10	partial	55,5
13	missing	72,2				6	missing	33,3

TEVAR, Thoracic endovascular aortic repair; *CT,* computed tomography.

Continuous follow-up of the patients and results of other study groups will be of great interest.

The AL remained almost stable in both groups within 24 months. After that time, it increased slightly in group A, whereas it remained nearly stable in group B. As none of the patients showed thrombosis of the abdominal FL, we agree with other authors who think that patency of the abdominal FL is responsible for a lack of remodeling of the abdominal aorta despite adequate sealing of the proximal tears.[15]

Aneurismal dilatation immediately adjacent to the SG has been observed by several authors.[15,31,32] Although it can be treated successfully in most cases by placing an additional SG, problems arise when it occurs in the distal part of the descending aorta involving the origin of the celiac trunk and the superior mesenteric artery. In their study, Won et al[32] stated that the sharp stent tip, the rigidity of the SG, and/or the pulsatile blood flow may have induced this phenomenon. Lopera et al[15] suggested that new intimal tears caused by the SG may have triggered it. In our study, aneurismal dilatation of the aorta in the area immediately adjacent to the SG was observed in 5 patients.

Aortic rupture is fatal in most patients with TBD. In our study, aortic rupture occurred in 3 of the conservatively-treated patients but in none of the patients treated by TEVAR. Several authors suggest that TEVAR can prevent late aortic rupture.[10,16,28,33-35] Eggebrecht et al[12] reported that aortic rupture occurred in 2.3% of 561 patients over a mean follow-up of 19.5 months and that even in the presence of a thrombosed false lumen, the distal thoracic or abdominal aorta may enlarge over time. Therefore, possible rupture has to be taken into consideration in case of sudden onset of pain after TEVAR.

Thrombosis of the FL has been reported to be a good indicator of a favorable long-term prognosis[36] and Akutsu et al[37] stated that thrombosis of the FL lowered the risk of aortic rupture. In their long-term study comparing 48 patients with patent FL to 62 with thrombosed FL, aortic rupture resulting in death occurred in 10 patients with patent FL compared to 2 patients with thrombosed FL. Bernard et al[38] observed that FL patency in the descending aorta is a predictive factor for late mortality. This is in agreement with our results. Indeed, two of the conservatively-treated patients who died of rupture, showed no thrombosis of the FTL. Interestingly, in another patient who had partial thrombosis of the FTL, autopsy showed that rupture had occurred exactly in a circumscribed bulged area, where thrombosis was missing. However, no statistically significant correlation between the degree of thrombosis and TL volume course was observed. Our evaluation of thrombosis of the FTL, showed slightly better overall results in group A. In this group, we observed pronounced changes between the pre- and the postinterventional CT scans, but no relevant changes at 60 months, and our analyses showed also a tendency towards approximation between groups A and B within 5 years.

In conclusion, our analyses show that within 24 months volume courses are more favorable in patients after TEVAR, than those in conservatively-treated patients and that TEVAR promotes thrombus formation, which may reduce the risk of rupture. However, within 60 months we observed a tendency towards approximation between the two patient groups, relating to both aortic volume courses and thrombosis of the FTL. Therefore, we conclude that TEVAR delays but does not stop the natural course of the disease.

AUTHOR CONTRIBUTIONS

Conception and design: IS, BC
Analysis and interpretation: IS, AC, BC
Data collection: IS, AC, BH, RH
Writing the article: IS
Critical revision of the article: AC, BC
Final approval of the article: BC
Statistical analysis: AS
Obtained funding: IS, AC, BC
Overall responsibility: IS

REFERENCES

1. Tsai TT, Fattori R, Trimarchi S, Isselbacher E, Myrmel T, Evangelista A, et al. Long-term survival in patients presenting with type B acute aortic dissection: insights from the International Registry of Acute Aortic Dissection. Circulation 2006;114:2226-31.
2. Estrera AL, Miller CC, Goodrick J, Porat EE, Achouh PE, Dhareshwar J, et al. Update on outcomes of acute type B aortic dissection. Ann Thorac Surg 2007;83:S842-5.
3. Hata M, Shiono M, Inoue T, Sezai A, Niino T, Negishi N, et al. Optimal treatment of type B acute aortic dissection: long-term medical follow-up results. Ann Thorac Surg 2003;75:1781-4.
4. Winnerkvist A, Brorsson B, Radegran K. Quality of life in patients with chronic type B aortic dissection. Eur J Vasc Endovasc Surg 2006;32: 34-7.
5. Duebener LF, Lorenzen P, Richardt G, Misfeld M, Notzold A, Hartmann F, et al. Emergency endovascular stent-grafting for life-threatening acute type B aortic dissections. Ann Thorac Surg 2004;78:1261-6.
6. MacKenzie KS, LeGuillan MP, Steinmetz OK, Montreuil B. Management trends and early mortality rates for acute type B aortic dissection: a 10-year single-institution experience. Ann Vasc Surg 2004;18: 158-66.
7. Suzuki T, Mehta RH, Ince H, Nagai R, Sakomura Y, Weber F, et al. Clinical profiles and outcomes of acute type B aortic dissection in the current era: lessons from the International Registry of Aortic Dissection (IRAD). Circulation 2003;108(Suppl 1):II312-7.
8. Trimarchi S, Nienaber CA, Rampoldi V, Myrmel T, Suzuki T, Bossone E, et al. Role and results of surgery in acute type B aortic dissection: insights from the International Registry of Acute Aortic Dissection (IRAD). Circulation 2006;114(1 Suppl):I357-64.
9. Czermak BV, Waldenberger P, Fraedrich G, Dessl AH, Roberts KE, Bale RJ, et al. Treatment of Stanford type B aortic dissection with stent-grafts: preliminary results. Radiology 2000;217:544-50.
10. Dake MD, Kato N, Mitchell RS, Semba CP, Razavi MK, Shimono T, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. N Engl J Med 1999;340:1546-52.
11. Eggebrecht H, Lonn L, Herold U, Breuckmann F, Leyh R, Jakob HG, et al. Endovascular stent-graft placement for complications of acute type B aortic dissection. Curr Opin Cardiol 2005;20:477-83.
12. Eggebrecht H, Nienaber CA, Neuhauser M, Baumgart D, Kische S, Schmermund A, et al. Endovascular stent-graft placement in aortic dissection: a meta-analysis. Eur Heart J 2006;27:489-98.
13. Nienaber CA, Fattori R, Lund G, Dieckmann C, Wolf W, von Kodolitsch Y, et al. Nonsurgical reconstruction of thoracic aortic dissection by stent-graft placement. N Engl J Med 1999;340:1539-45.
14. Gysi J, Schaffner T, Mohacsi P, Aeschbacher B, Althaus U, Carrel T. Early and late outcome of operated and non-operated acute dissection of the descending aorta. Eur J Cardiothorac Surg 1997;11:1163-9.
15. Lopera J, Patino JH, Urbina C, Garcia G, Alvarez LG, Upegui L, et al. Endovascular treatment of complicated type-B aortic dissection with stent-grafts: midterm results. J Vasc Interv Radiol 2003;14(2 Pt 1):195-203.
16. Lonn L, Delle M, Falkenberg M, Lepore V, Klingenstierna H, Radberg G, et al. Endovascular treatment of type B thoracic aortic dissections. J Card Surg 2003;18:539-44.
17. Schoder M, Czerny M, Cejna M, Rand T, Stadler A, Sodeck GH, et al. Endovascular repair of acute type B aortic dissection: long-term follow-up of true and false lumen diameter changes. Ann Thorac Surg 2007;83:1059-66.
18. Song TK, Donayre CE, Walot I, Kopchok GE, Litwinski RA, Lippmann M, et al. Endograft exclusion of acute and chronic descending thoracic aortic dissections. J Vasc Surg 2006;43:247-58.
19. Steingruber IE, Chemelli A, Glodny B, Hugl B, Bonatti J, Hiemetzberger R, et al. Endovascular repair of acute type B aortic dissection: midterm results. J Endovasc Ther 2008;15:150-60.
20. Resch TA, Delle M, Falkenberg M, Ivancev K, Konrad P, Larzon T, et al. Remodeling of the thoracic aorta after stent grafting of type B dissection: a Swedish multicenter study. J Cardiovasc Surg (Torino) 2006;47:503-8.
21. Czermak BV, Fraedrich G, Schocke MF, Steingruber IE, Waldenberger P, Perkmann R, et al. Serial CT volume measurements after endovascular aortic aneurysm repair. J Endovasc Ther 2001;8:380-9.
22. Vallabhaneni SR, Harris PL. Lessons learnt from the EUROSTAR registry on endovascular repair of abdominal aortic aneurysm repair. Eur J Radiol 2001;39:34-41.
23. Marui A, Mochizuki T, Mitsui N, Koyama T, Kimura F, Horibe M. Toward the best treatment for uncomplicated patients with type B acute aortic dissection: a consideration for sound surgical indication. Circulation 1999;100(19 Suppl):II275-80.
24. Lee JT, Aziz IN, Lee JT, Haukoos JS, Donayre CE, Walot I, et al. Volume regression of abdominal aortic aneurysms and its relation to successful endoluminal exclusion. J Vasc Surg 2003;38:1254-63.
25. Armon MP, Yusuf SW, Whitaker SC, Gregson RH, Wenham PW, Hopkinson BR. Thrombus distribution and changes in aneurysm size following endovascular aortic aneurysm repair. Eur J Vasc Endovasc Surg 1998;16:472-6.
26. Wever JJ, Blankensteijn JD, Broeders IA, Eikelboom BC. Length measurements of the aorta after endovascular abdominal aortic aneurysm repair. Eur J Vasc Endovasc Surg 1999;18:481-6.
27. Kusagawa H, Shimono T, Ishida M, Suzuki T, Yasuda F, Yuasa U, et al. Changes in false lumen after transluminal stent-graft placement in aortic dissections: six years' experience. Circulation 2005;111:2951-7.
28. Xu SD, Huang FJ, Yang JF, Li ZZ, Wang XY, Zhang ZG, et al. Endovascular repair of acute type B aortic dissection: early and midterm results. J Vasc Surg 2006;43:1090-5.
29. Juvonen T, Ergin MA, Galla JD, Lansman SL, McCullough JN, Nguyen K, et al. Risk factors for rupture of chronic type B dissections. J Thorac Cardiovasc Surg 1999;117:776-86.
30. Prendergast BD, Boon NA, Buckenham T. Aortic dissection: advances in imaging and endoluminal repair. Cardiovasc Intervent Radiol 2002; 25:85-97.
31. Kato N, Shimono T, Hirano T, Suzuki T, Ishida M, Sakuma H, et al. Midterm results of stent-graft repair of acute and chronic aortic dissection with descending tear: the complication-specific approach. J Thorac Cardiovasc Surg 2002;124:306-12.
32. Won JY, Suh SH, Ko HK, Lee KH, Shim WH, Chang BC, et al. Problems encountered during and after stent-graft treatment of aortic dissection. J Vasc Interv Radiol 2006;17(2 Pt 1):271-81.
33. Herold U, Piotrowski J, Baumgart D, Eggebrecht H, Erbel R, Jakob H. Endoluminal stent graft repair for acute and chronic type B aortic dissection and atherosclerotic aneurysm of the thoracic aorta: an interdisciplinary task. Eur J Cardiothorac Surg 2002;22:891-7.
34. Lee KH, Won JY, Lee DY, Choi D, Shim WH, Chang BC. Elective stent-graft treatment of aortic dissections. J Endovasc Ther 2004;11: 667-75.
35. Nathanson DR, Rodriguez-Lopez JA, Ramaiah VG, Williams J, Olsen DM, Wheatley GH, et al. Endoluminal stent-graft stabilization for thoracic aortic dissection. J Endovasc Ther 2005;12:354-9.
36. Erbel R, Alfonso F, Boileau C, Dirsch O, Eber B, Haverich A, et al. Diagnosis and management of aortic dissection. Eur Heart J 2001;22: 1642-81.
37. Akutsu K, Nejima J, Kiuchi K, Sasaki K, Ochi M, Tanaka K, et al. Effects of the patent false lumen on the long-term outcome of type B acute aortic dissection. Eur J Cardiothorac Surg 2004;26:359-66.
38. Bernard Y, Zimmermann H, Chocron S, Litzler JF, Kastler B, Etievent JP, et al. False lumen patency as a predictor of late outcome in aortic dissection. Am J Cardiol 2001;87:1378-82.

Submitted May 18, 2008; accepted Aug 20, 2008.