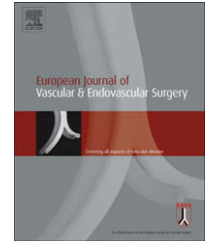




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# Aortic Morphology Following Endovascular Repair of Acute and Chronic Type B Aortic Dissection: Implications for Management

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## KEYWORDS

Aortic dissection;  
Endovascular;  
Rupture

**Abstract** *Objective:* The study aimed to define early clinical outcomes, and medium term morphological changes, following endovascular treatment of acute (AAD) and chronic (CAD) Type B aortic dissections.

*Main outcomes:* The cohort comprised 78 patients who underwent endovascular repair for AAD (38) and CAD (40). Early and late clinical outcomes were prospectively recorded. All patients underwent serial follow up with CT scanning. False lumen thrombosis rates, true, false and total aortic short axis diameter were recorded at the mid point of the endograft and below this level in the thoracic aorta. The total maximum aortic diameter in the thoracic, abdominal aorta was quantified.

*Results:* The 30-d mortality was 2.6% in AAD and 7.5% in CAD. The 30-d stroke and paraplegia rates were 5.3% and 0% in AAD. There were no cases of stroke or paraplegia in patients with CAD. At 30 months follow up, the cumulative survival for the two groups was 93% for AAD and 66.5% for CAD ( $P = 0.015$ , Kaplan Meier) and the cumulative re-intervention rate was 62% and 55% in AAD and CAD respectively ( $P = 0.961$ , Kaplan-Meier). False lumen thrombosis rates were equivalent in the two groups and were higher at the level of the endograft than below this level ( $P < 0.05$ ). Aortic remodelling was greater in AAD, whereas the aortic dimensions after treatment of CAD remained relatively static. Up to 20% of patients in both groups demonstrated enlargement of the thoracic aorta.

*Conclusions:* The data support the use of endovascular repair of the thoracic aorta in Type B aortic dissection. 30-d outcomes are acceptable. Patients with AAD demonstrate significant aortic remodelling whereas patients with CAD do not. This has significant implications for practice as patients with CAD must rely on maintenance of false lumen thrombosis to preserve the integrity of the endovascular repair.

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## Introduction

The management of Stanford Type B aortic dissection has undergone fundamental change over the past decade.<sup>1</sup> Conventional surgical intervention for acute Type B dissections was reserved for life threatening complications and was associated with a significant mortality, even in highly specialised centres.<sup>2</sup> The adoption of endovascular repair for acute Type B dissections has shown considerable promise, and registry outcomes have suggested that early mortality for comparable patient groups is significantly lower than the surgical alternatives.<sup>3,4</sup> Endovascular techniques have more recently been applied to the treatment of chronic dissections that have become complicated or which have progressed to aneurysmal degeneration. The results of these interventions are less well defined than for acute lesions,<sup>5,6</sup> and carefully reported series are required to determine the efficacy of endovascular therapy in the chronic phase of Type B dissection.

An important component of endovascular therapy for aortic dissections is the long-term changes that occur in the stented segment and distal aorta. Endovascular therapy in the acute phase relies on covering the primary entry tear to initiate false lumen thrombosis, expand the true lumen and stimulate aortic remodelling. In chronic dissections, covering the primary tear may not be sufficient to obliterate flow in the false lumen and preliminary studies have suggested that the potential for remodelling may be less.<sup>7</sup>

The aim of the present study was to document the outcome of a large cohort of patients undergoing endovascular treatment of Type B aortic dissection. In addition to clinical outcomes, the study documented the morphological changes in the aorta that occurred after the endovascular repair.

## Methods

A prospective database of all patients undergoing endovascular treatment for Stanford Type B aortic dissections, at a tertiary referral centre, was maintained from Feb 2000 to Feb 2007. The databases included clinical outcomes, secondary procedures and details of treatment.

Stanford Type B dissections were sub-classified according to the recommendations of the European Society of Cardiology.<sup>8</sup> Acute dissections (AAD) were defined by a period of 2 weeks from onset of symptoms and chronic dissections (CAD) by an elapsed time of greater than 2 weeks following initial dissection.<sup>1</sup> The reported cohort consisted of 78 patients; 38 with acute dissections and 40 with chronic dissections. The acute group comprised 4 patients with penetrating thoracic ulcers (European Task Force Type 4), 5 with intramural haematoma (Type 2), and 29 with classic dissections (Type 1). Patients with traumatic aortic transection were not considered in this investigation.

## Indications for endovascular treatment

In our centre, endovascular repair is the preferred option for the treatment of complicated Type B aortic dissection. Indications for intervention in acute aortic syndrome were

rupture (haemothorax, haemomediastinum), malperfusion (acute limb ischaemia, intestinal ischaemia, renal ischaemia), persistent pain for a 3 day period or hypertension refractory to treatment. In the acute patient cohort, 10 patients presented with aortic rupture, 4 had renal ischaemia and 4 mesenteric ischaemia. Chronic dissections were considered for intervention in the presence of complications (rupture, acute dissection, end organ ischaemia or pain), maximum short axis thoracic aortic diameter exceeding 5.5 cm, or rapid growth of the thoracic aorta (1 cm in 6 months – local protocol). Four of the 40 patients treated for CAD presented with aortic rupture and one with visceral ischaemia. The demographics of the patient cohort are illustrated in Table 1.

## Post procedural imaging

All patients were imaged with computerised tomography (CT) as the preferred modality. Patients were imaged before endovascular repair; prior to discharge and then at 3 months, 6 months and yearly thereafter. The short axis diameter of the true lumen, false lumen and total aorta was quantified for each CT scan at 7 levels (mid aortic arch, upper edge of the descending aorta, the carina, lower left atrium, coeliac axis, uppermost renal artery and the iliac bifurcation).<sup>7</sup> The patency of true and false lumens was determined at these levels and was also related to the level of the endograft (i.e. false lumen patency at the mid point of the endograft and below the endograft). The maximum aortic diameter of the ascending, descending thoracic and abdominal aorta was recorded.

Analysis of false lumen thrombosis, and true and false lumen diameters did not include those patients with penetrating ulcers or intramural haematomas as these did not have a defined false lumen that could be examined.

## Statistical analysis

Statistical analysis was performed using SPSS 14.0 (SPSS Inc, Chicago, IL) and Excel 2003. Continuous variables are presented as mean with 95% confidence intervals. Comparison between continuous variables utilised the student T test. Categorical variables were analysed by Chi squared and Fisher's exact tests. Analysis of variance was utilised to compare short axis, false lumen and true lumen diameters over the follow up period. Cumulative survival rates were calculated using Kaplan Meier analysis and presented as life tables.

## Results

### Procedural details of initial endovascular repair

Endovascular coverage of the primary entry tear was achieved in all cases. There were a total of 57 stents used in 38 acute cases for a mean of 1.5 endografts per case (24 Valiant endografts [Medtronic, Santa Rosa, CA], 19 Talent grafts [Medtronic], 11 Excluder grafts [WL Gore, Flagstaff, AZ], and 3 TX2 [Cook Medical, Limerick, Eire]). In the patients with CAD, 2.12 endografts per patient (85

**Table 1** displaying patient demographics for the cohorts with acute and chronic dissections. Risk factors are tabulated as the percentage of patients with each SVS/ISCVS score for eight domains. Type of surgery gives the proportion of patients who have had aortic surgery, cardiac surgery or previous endovascular intervention

	Acute (n = 38)				Chronic (n = 40)				P
Age	62.5 ± 15.1				66.6 ± 11.9				0.19
Sex	Male 26		Female 12		Male 26		Female 14		
<b>Aetiology of dissection</b>									
Atherosclerosis	17 (44.2%)				21 (52.5%)				0.5072
Hypertension	22 (57.2%)				27 (67.5%)				0.4831
Marfan	3 (7.8%)				7 (17.5%)				0.4238
Other	2 (5.2%)				0 (0%)				0.3118
									0.066
<b>SVS Score</b>									
	<b>0</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>0</b>	<b>1</b>	<b>2</b>	<b>3</b>	
Smoking	44.7%	5.3%							
Hypertension	21%	27.5%	31.6%	18.4%	47.5%	10%	15%	27.5%	0.306
Diabetes	89.5%	0%	26.5%	25%	17.5%	2.5%	17.5%	62.5%	<b>0.001</b>
Hyperlipidaemia	65.8%	0%	7.9%	2.6%	90%	2.5%	2.5%	5%	0.505
Cardiac Disease	84.2%	2.6%	10.5%	23.7%	42.5%	0%	2.5%	55%	<b>0.017</b>
Carotid Disease	89.5%	0%	7.9%	5.3%	70%	15%	12.5%	2.5%	0.202
Renal Impairment	89.4%	5.3%	7.9%	2.6%	87.5%	0%	7.5%	5%	0.297
Pulmonary Disease	81.5%	13.2%	5.3%	0%	62.5%	25%	5%	7.5%	<b>0.022</b>
Overall SVS	7.9%	10.5%	5.3%	0%	85%	2.5%	12.5%	0%	0.133
			23.7%	57.9%	2.5%	5%	7.5%	85%	0.066
<b>Medications</b>									
Antiplatelet	4 (10.4%)				19 (47.5%)				
Warfarin	1 (2.6%)				3 (7.5%)				<b>0.0004</b>
Statins	8 (20.8%)				26 (65%)				0.6156
B Blocker	11 (28.6%)				30 (75%)				<b>0.0001</b>
ACE Inhibitor	13 (33.8%)				20 (50%)				<b>0.0001</b>
A2RB	2 (5.2%)				5 (12.5%)				0.1766
Ca Channel Blocker	3 (7.8%)				22 (55%)				0.4321
Diuretic	9 (23.4%)				21 (52.5%)				<b>0.0001</b>
									<b>0.0111</b>
<b>Previous Vascular Reconstruction</b>									
Aortic Surgery	1 (2.6%)				11 (27.5%)				
Cardiac Surgery	1 (2.6%)				3 (7.5%)				<b>0.0033</b>
Endovascular	1 (2.6%)				1 (2.5%)				0.6156
									1.0000

grafts) were deployed (46 Valiant, 33 Talent, and 6 Excluder). The mean distance of the aorta covered was  $182 \pm 18$  mm in AAD and  $204 \pm 20$  mm in CAD. The proximal and distal stent diameters were  $30.8 \pm 1.7$  mm and  $28.4 \pm 1.4$  mm in acute and  $32.9 \pm 1.3$  mm and  $26.9 \pm 2.0$  mm in chronic dissections. The mean procedure time was very similar for the both groups being 138 mins and 133.5 mins for AAD and CAD respectively.

The proximal landing zones for the endovascular repair were classified according to the position with respect to the great vessels. The total cases covering the left common carotid ostium were 3 in AAD and 9 in CAD, covering the LSCA ostium (AAD 17, CAD15) and beyond the LSCA (AAD18, CAD15). The planned site of the proximal endograft necessitated a prior procedure in 4 cases for the acute dissections and 11 cases in the chronic dissections. These were 10 carotid-carotid-L subclavian bypasses and 5 carotid-L subclavian bypasses.

### Mortality, stroke and paraplegia

There was one 30-d death in the acute group (2.6%) due to ongoing haemorrhage from a ruptured dissection. There were three 30-d deaths in the chronic group (7.5%); two from rupture of the aneurysmal false lumen in the abdomen following an apparently successful procedure (after hospital discharge) and one from a retrograde Type A dissection. The 30-d stroke and paraplegia rates were 5.3% and 0% in AAD. There were no cases of stroke or paraplegia in patients with CAD. Mean ICU stay was 3.7 days and 2.7 days, while hospital stay was 18.7 days and 11.9 days ( $P = 0.02$ ) for AAD and CAD respectively.

Cumulative survival rates for patients with AAD and CAD are illustrated in Fig. 1. At 30 months, the cumulative survival for the two groups was 93% for AAD and 66.5% for CAD. Patients with AAD had significantly higher survival rates than patients with chronic disease ( $P = 0.015$ , Kaplan

Meier). This may be in part due to a higher prevalence of renal disease, hypertension and hyperlipidaemia in the patients with chronic dissections.

**Secondary intervention**

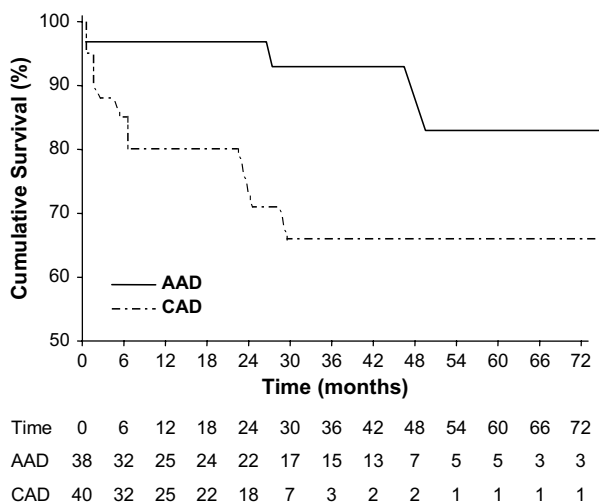
In patients treated for acute dissection, 8 required a secondary procedure for continuing problems with the thoracic aorta or endograft. There were 3 proximal procedures for continuing proximal perfusion due to type 1 endoleak, 2 subclavian artery embolisations for Type II endoleaks and three distal stent extensions for ongoing false lumen perfusion in the thorax associated with aortic expansion. None of these secondary procedures were required in patients with IMH or penetrating aortic ulcers. This cohort also required 6 procedures to treat other aortic pathologies, which included 5 procedures on abdominal aortic aneurysms (all in patients with classic dissection except for one with IMH, one requiring a retrograde visceral bypass and thoracoabdominal stenting), and one thoracotomy for a large haemothorax.

In the chronic cohort, 6 patients required secondary procedures related to the dissection (one patient had a subclavian embolisation for a Type II endoleak, one required proximal stenting following a carotid-carotid bypass for a proximal endoleak, one had a distal extension due to persistent chest pain, one had a retrograde visceral bypass and thoracoabdominal stenting, one had a sternotomy, ascending aortic bypass to the innominate and left common carotid artery, followed by a proximal stent extension for a persistently perfused false lumen of a previous type A dissection and one patient required treatment of a type A aneurysm). A further 8 had treatment for other aortic pathology, which include 5 abdominal

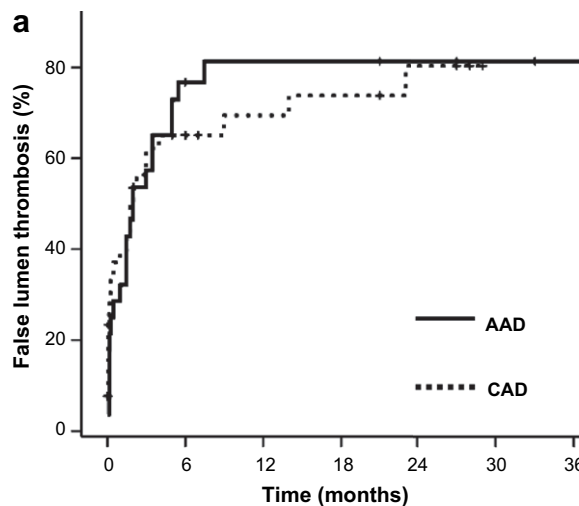
aneurysms and one ilio-femoral bypass. At 30 months the cumulative freedom from re-intervention rate was 62% and 55% in CAD and AAD respectively ( $P = 0.961$ , Kaplan-Meier).

**False lumen thrombosis**

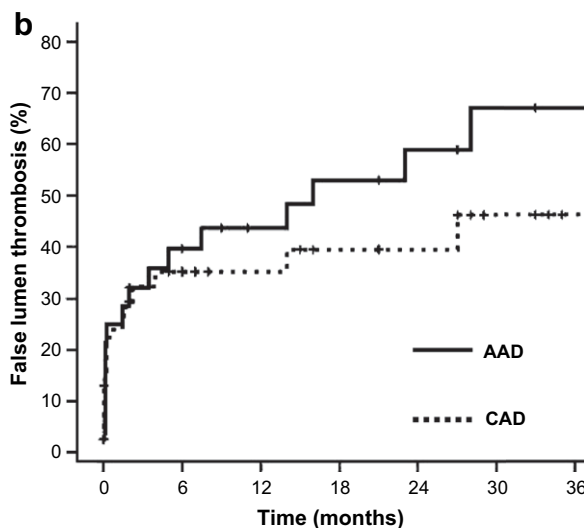
The false lumen thrombosis rates were quantified at 7 aortic levels and were also related to the position of the endograft. When considering the false lumen thrombosis rates at



**Figure 1** Life table plotting cumulative survival (%) against time (months), in patients having undergone endovascular repair for acute (AAD) or chronic aortic dissection (CAD). The numbers of patients at risk at each time point are given in tabular form. The cumulative survival for patients with CAD is significantly worse than for patients with AAD ( $P = 0.015$ , Kaplan Meyer).



Time	0	6	12	18	24	30	36
AAD	28	6	4	4	3	2	1
CAD	40	11	7	6	3	0	0



Time	0	6	12	18	24	30	36
AAD	28	16	12	10	7	4	3
CAD	40	21	15	12	9	4	1

**Figure 2** Life table plotting cumulative false lumen thrombosis rate at the mid point of the endovascular stent (a) and below the level of the endograft (b) against time (months), in patients undergoing endovascular repair for acute (AAD) or chronic aortic dissection (CAD). The numbers of patients at risk at each time point are given in tabular form.

different levels, the rates were lower the more distal the measurement (data not shown). Fig. 2 illustrates the cumulative false lumen thrombosis rates at the mid point of the thoracic aortic endograft, and the thrombosis rate in the thoracic aorta immediately below the endograft.

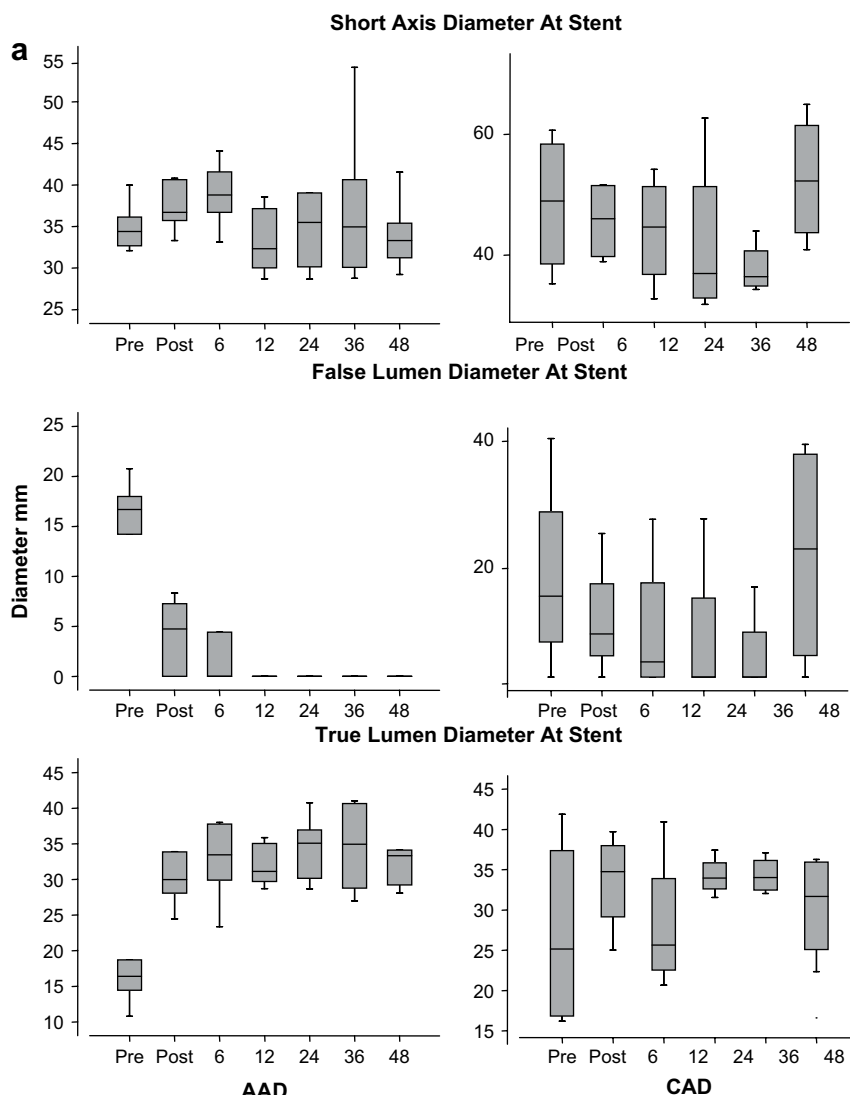
In general, the false lumen thrombosis rate at the level of the endograft was similar for both acute and chronic cases. Thrombosis rates for both AAD and CAD were lower in the section of aorta immediately distal to the endograft than at the mid-point of the endograft ( $P = 0.005$  AAD;  $P = 0.002$  CAD). The false lumen thrombosis rate was reduced for CAD in comparison to AAD when the thoracic aorta below the level of the endograft was analysed. This difference did not reach statistical significances ( $P = 0.62$ ).

### Aortic dimensions following endovascular repair

The maximum short axis diameter of the thoracic aorta, false lumen and true lumen diameters were measured at 7

points in the aorta and were also related to the level of the endograft. These measurements are illustrated in Fig. 3 for diameters at the mid point of the endograft and immediately below the endograft in the thoracic aorta. Diameter changes are illustrated for both AAD and CAD. At the level of the endograft, endovascular repair of AAD resulted in a significant increase in true lumen diameter over time ( $P = 0.001$ , ANOVA) and a significant decrease in false lumen diameter ( $P = 0.002$ , ANOVA). The short axis diameter of the aorta in AAD was not significantly altered in AAD. These changes were similar, although not as profound, in the aorta below the level of the endograft.

In chronic dissections, endovascular repair did not significantly alter the aortic dimensions either at the level of the endograft or below this point. Again, this may reflect the relative mobility of the dissection flap in acute and chronic cases, but also suggests that different end points of success might be defined for AAD and CAD.



**Figure 3** Bar graph plotting mean short axis diameter of the thoracic aorta, false lumen and true lumen at the level of the endograft (a) and below the level of the endograft (b) against time (months after procedure; Pre-diameter before endovascular repair, Post-immediately after repair). Values given are median with interquartile range and range. The separate graphs represent values for acute aortic dissection (AAD) and chronic aortic dissection (CAD). The numbers at each time point are equivalent to Fig. 2.

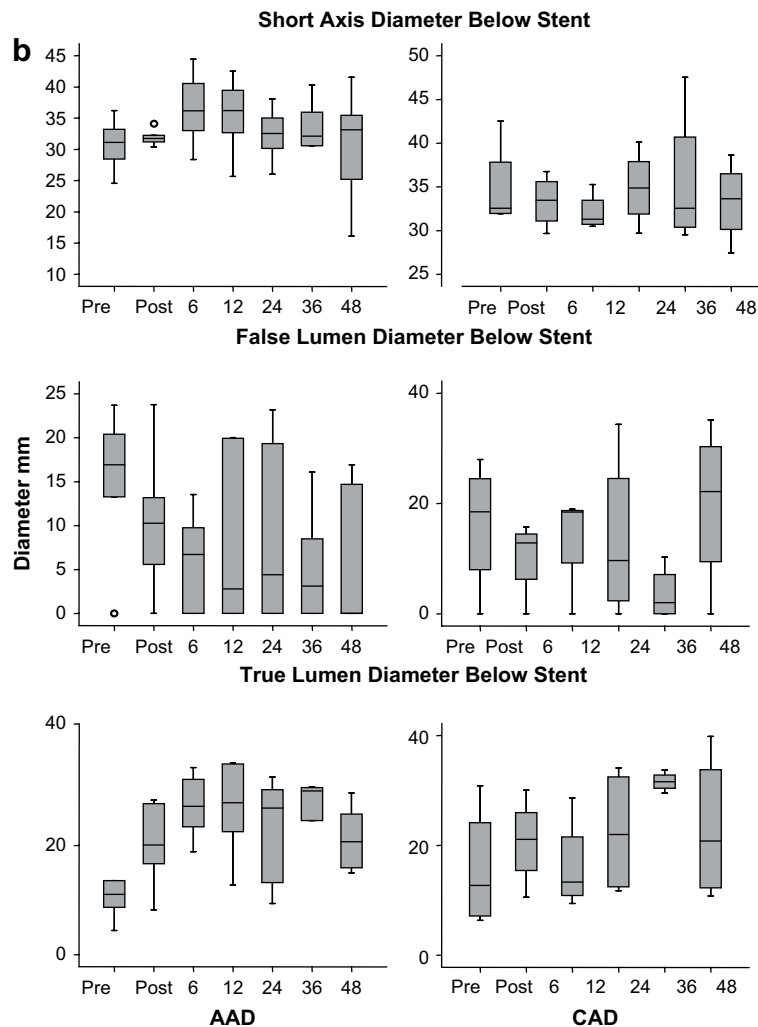


Figure 3 Continued

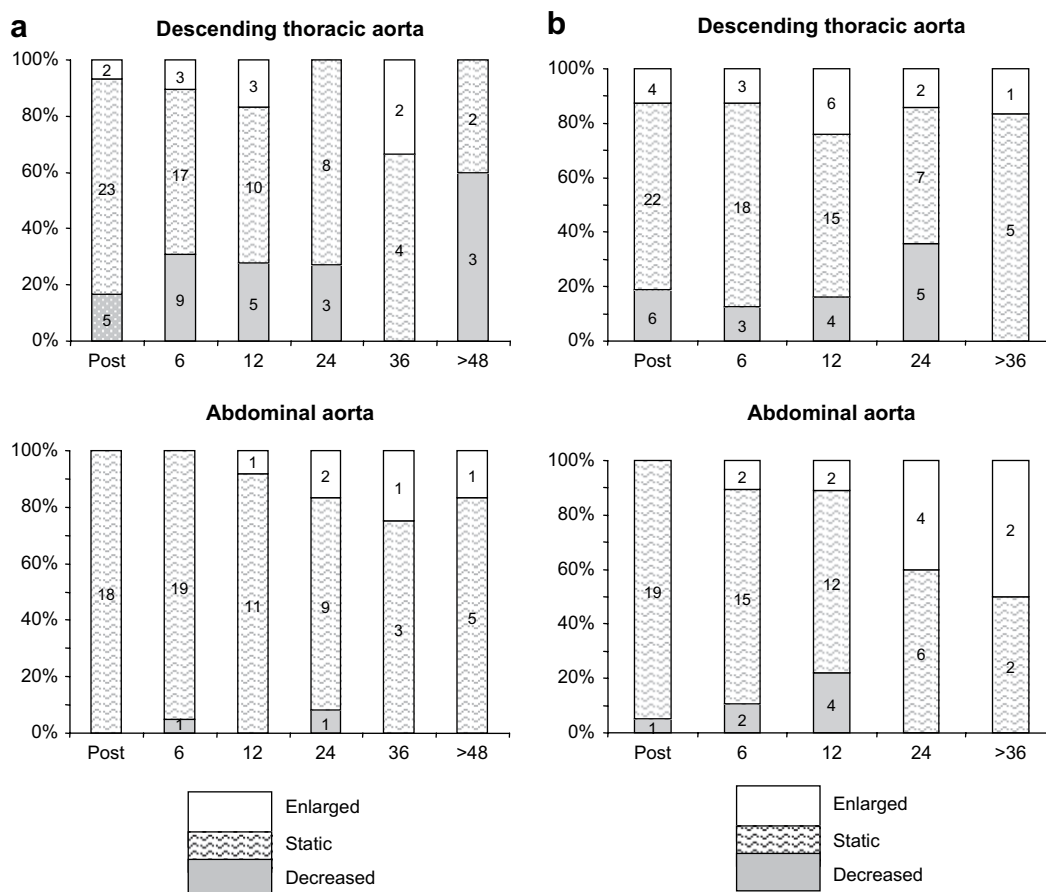
The changes in the maximum short axis diameter of both descending thoracic and abdominal aorta for both AAD and CAD are illustrated in Fig. 4. Interestingly, following repair of AAD, there are some apparent treatment failures with up to 20% of patients experiencing expansion of the descending thoracic aorta, although this was defined by a 5 mm increase in the present study. In spite of this, the abdominal aorta remained relatively static. Conversely, following repair of CAD, whilst a similar proportion of patients to those with AAD experience dilatation of the thoracic aorta, a much higher percentage appear to demonstrate dilatation of the abdominal aorta.

## Discussion

Open surgery for aortic dissection has reported mortality rates exceeding 30%,<sup>10–12</sup> in comparison to the significantly lower mortality rates for endovascular repair.<sup>4,13</sup> The present study supports the use of endovascular techniques for AAD with an observed mortality rate of 2.6%, a stroke rate of 5.3% and no cases of spinal cord ischaemia. All of the acute cases in this investigation had robust indications

for treatment and 20 of the 38 necessitated coverage of the left subclavian ostium.

Several previous reports have documented that mortality following the treatment of acute dissections, exceeds that for chronic cases.<sup>4,14</sup> That was not observed in the present study with a mortality rate for chronic dissections of 7.5%. This may partly reflect the increased complexity of the chronic lesions that required a high prevalence of great vessel bypasses to create an adequate landing zone; and required a greater number of stents. All the deaths in this group may be attributed to the aortic intervention, with one retrograde Type A dissection and two patients with rupture of the false lumen in the abdomen. The two patients with ruptured false lumens provide a cause for concern. Previously our policy with chronic dissecting aneurysms affecting the throacoabdominal aorta was to cover the primary entry tear, wait for several months and only consider further intervention if the false lumen was not thrombosing. Clearly this policy needs to be considered following experience of false lumen rupture. In patients with a significantly sized abdominal component we would now advocate treatment of the entire throacoabdominal component at the same time; our



**Figure 4** Graph illustrating the percentage of patients having undergone endovascular repair of an acute aortic dissection (a) and chronic aortic dissection (b) that demonstrated enlargement, reduction or stasis in the maximum short axis diameter of the descending thoracic or abdominal aorta at varying time intervals after treatment. Enlargement and reduction were defined by a change of more than 5 mm from the pre-treatment CT. Numbers of patients in each category are given.

preference is to use retrograde visceral debranching and endovascular repair. The case of retrograde Type A dissection followed treatment of a chronic dissecting aneurysm in the mid descending thoracic aorta. Although a graft with bare proximal attachment springs was placed, this was not ballooned. Retrograde Type A dissection is now accepted as a recognised complication of endovascular thoracic procedures.<sup>15</sup> However, the aetiology remains largely undefined and further registry data are required to determine robust associations with this complication (current registry data collected at [www.tevarcomplications.com](http://www.tevarcomplications.com)).

Endovascular repair of thoracic dissections appears to offer an attractive solution to the treatment of complicated aortic dissections. However, although the early outcomes with regard to operative mortality and morbidity are promising, the longer-term outcomes remain under-reported. Of significance in the present study was the high rate of reintervention in both acute and chronic groups. Interestingly, the majority of interventions were related to synchronous problems with the abdominal aorta, which highlights the importance of total aortic surveillance in patients with aortic dissection. Additionally the difference between mid-term survival was significant with the chronic group faring poorly. The differences in survival may be

attributable to a higher incidence of renal disease and hypertension in the patients with CAD. The present sample size is insufficient to perform a regression analysis to determine whether survival is a function of the chronicity of the dissection or other factors.

One of the primary aims in the endovascular treatment of thoracic dissections is to achieve false lumen thrombosis and/or aortic remodelling. In the present study, false lumen thrombosis was similar in acute and chronic cases. False lumen thrombosis was also greater at the level of the endograft than distal to this. False lumen thrombosis rates have not been extensively reported in the literature for AAD and CAD separately. Kusagawa *et al.*<sup>7</sup> observed that false lumen thrombosis was complete in 76% of patients with AAD after 2 years compared to just 36% of patients with CAD. This rate appears inferior to that reported in the present study, which may reflect differing definitions and levels of analysis. In a cohort combining 129 acute and chronic dissections, Resch *et al.*<sup>16</sup> reported a false lumen thrombosis rate at the level of the endograft of 80% with 50% of cases exhibiting distal perfusion, which would be similar to the present study.

The most significant difference between the acute and chronic cases in this study was related to the extent of aortic remodelling. In the acute setting there was a rapid

expansion of the true lumen and collapse of the false lumen around the stent within a year of the procedure. These are similar findings to those reported by Schoder *et al.*<sup>17</sup> In chronic dissections the true and false lumen diameters do not change to the same degree, which suggests that the capacity for aortic remodelling in CAD is less. Distally, in AAD, there appears to be some remodelling in the aorta below the stent, but again, in CAD the changes in diameter are minimal.

In acute dissections, up to 20% of the patients show aortic dilatation in the thoracic segment 1 year after endovascular repair, which appears to be similar to the figure in chronic dissections observed in the present study. Therefore, although the patients treated for AAD appear to have a greater capacity for remodelling, the chance of thoracic aortic dilatation with present treatment algorithms remains. Late dilatation and aortic rupture have previously been reported to a similar degree in other studies.<sup>7,18</sup> This possibility of enlargement suggests that any technique which accelerates the true and false lumen remodelling over the entire length of the thoracic aorta may be beneficial. The concept of a bare metal stent covering the thoracic aorta distal to the covered entry site has been proposed,<sup>19</sup> which has the theoretical advantages of accelerating remodelling but not raising the incidence of paraplegia from an increased length aorta covered with a traditional endograft.<sup>20</sup>

One further significant finding was the enlargement of the abdominal aorta observed following endovascular repair of both acute and chronic dissections. In AAD and CAD, a substantial proportion of cases exhibited enlargement of the abdominal aorta and there were a number of patients who required repair of an abdominal aortic aneurysm. This finding emphasises the importance of long term surveillance following endovascular treatment of thoracic dissections and that the distal arterial tree should be included in the imaging.

As with all new innovations, there are some unresolved issues with endovascular repair of Type B thoracic dissections. One technical issue involves the length of thoracic aorta that should be covered by the endograft in both AAD and CAD. Current practice encompasses a spectrum of opinion that ranges from those who believe that only the entry tear should be covered, to those who believe that the best results are obtained by covering the whole of the thoracic aorta. The present study provides evidence to support the assertion that different techniques should be applied to the acute and chronic conditions. In patients with AAD, the capacity for aortic remodelling appears to be high and even the distal unstented aorta demonstrates diameter changes with false lumen collapse and true lumen expansion. This suggests that coverage of the primary entry tear may be sufficient to treat AAD, as long as careful surveillance of the thoracic and abdominal aorta is maintained. In contrast to the situation in AAD, patients with chronic dissections demonstrate relatively little ability to remodel the aorta, and therefore the integrity of the repair is defined by achieving complete false lumen thrombosis. As the false lumen thrombosis rates distal to the endograft are particularly poor, a long length of aortic coverage is required to achieve the best chance of success.

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