Mid-term Outcomes and Aortic Remodelling After Thoracic Endovascular Repair for Acute, Subacute, and Chronic Aortic Dissection: The VIRTUE Registry

The VIRTUE Registry Investigators *

WHAT THIS PAPER ADDS

The VIRTUE Registry, describes the mid-term clinical and morphological results of thoracic endovascular repair in patients with type B aortic dissection. Analysis of aortic morphology showed that patients with subacute dissection demonstrated a similar degree of aortic remodelling to patients with acute dissection. Retention of aortic plasticity in the subacute group lengthens the therapeutic window for the treatment of uncomplicated type B dissection.

Objective: The VIRTUE Registry describes the mid-term clinical and morphological results of thoracic endovascular repair (TEVR) in patients with type B aortic dissection.

Methods: This was a prospective cohort study. The VIRTUE Registry is a prospective, multicentre clinical trial that enrolled patients with complicated acute (<15 days), subacute (15–92 days), and chronic (>92 days) type B aortic dissections treated with the Valiant endograft. One hundred patients were enrolled and the clinical outcomes described at the 3-year follow-up. Analysis of the aortic area and false lumen thrombosis rates defined the morphological response to TEVR in the three clinical groups.

Results: Three-year all-cause mortality (18%, 4%, and 24%), dissection related mortality (12%, 4%, and 9%), aortic rupture (2%, 0%, and 4%), retrograde type A dissection (5%, 0%, and 0%), and aortic reintervention rates (20%, 22%, and 39%) were, respectively, defined for patients with acute (n = 50), subacute (n = 24), and chronic (n = 26) dissections. Analysis of aortic morphology observed that patients with subacute dissection demonstrated a similar degree of aortic remodelling to patients with acute dissection. Patients with acute and subacute dissection exhibited greater aortic plasticity than patients with chronic dissection.

Conclusions: The principle clinical findings suggest that TEVR is able to provide good protection from aortic-related death in the mid-term, but with a high rate of aortic reintervention. Analysis of aortic morphology suggested that aortic remodelling in subacute patients is similar to the acute group. Retention of aortic plasticity in the subacute group lengthens the therapeutic window for the treatment of uncomplicated type B dissection.

© 2014 European Society for Vascular Surgery. Published by Elsevier Ltd. All rights reserved.
Article history: Received 31 December 2013, Accepted 6 May 2014, Available online 18 June 2014
Keywords: Aortic dissection, Endovascular, Type B

INTRODUCTION

In recent years, the management algorithm for patients with type B aortic dissection has changed with the introduction of endovascular repair of the thoracic aorta. The introduction of thoracic endovascular repair (TEVR) for acute complicated type B aortic dissection has largely been predicated on early results, which are favourable in comparison with open surgery, but the mid- and long-term outcomes remain less well defined.

TEVR has also been advocated as a potential therapeutic modality in patients with chronic type B aortic dissection. Although early results have been generally satisfactory, the mid-term outcomes of TEVR for chronic dissection are heterogeneous, and it is uncertain whether TEVR can prevent late aortic-related complications or death. The uncertainty over the longer-term clinical outcome in chronic dissection is partly owing to the differences in aortic remodelling when acute and chronic aortic dissections are compared. Acute dissections demonstrate rapid expansion of the true lumen, collapse of the false lumen, and false lumen thrombosis. These changes are less marked in the chronic phase, and the lack of false lumen thrombosis may lead to adverse clinical outcomes.

The traditional 2-week distinction between the acute and chronic phases of aortic dissection relate to the timing of death in the era of open surgery, and may not be as useful...
in describing the aortic response to endovascular repair. There has been some suggestion that defining the response to aortic surgery in a subacute phase (15–92 days) of dissection might be useful in order to investigate when the aorta becomes less plastic in its ability to remodel and whether this has any implications on clinical outcomes.\(^7,^8\)

This study describes the mid-term results of the VIRTUE Registry. The registry enrolled patients with acute, subacute, and chronic type B aortic dissections treated with the Valiant endograft (Medtronic, Santa Rosa, CA, USA). The early outcomes of this registry have been previously described,\(^9\) and the current report details the later outcomes with reference to clinical results and aortic remodelling.

**METHODS**

**Study design**

The study design has been described in detail previously.\(^9\) Briefly, the VIRTUE Registry is a prospective, non-randomised, multicentre European Clinical Registry that enrolled 100 patients with type B aortic dissection treated with the Valiant (Medtronic) thoracic stent-graft.\(^10\) Enrolment was performed and consent obtained before the procedure. The primary endpoint of the registry was all-cause mortality at 12 months after the procedure. Secondary endpoints included all-cause and dissection-related mortality, major complications, reintervention, and graft-related complications up to 3 years after the procedure.

Specific indications for inclusion were documented by the duration of the disease and were at the clinical discretion of the investigators:

- acute dissection (14 days from first dissection)—aortic rupture, malperfusion syndromes (visceral, renal, lower limb), impending rupture (persistent pain), refractory hypertension;
- subacute dissection (15–92 days)—complicated/symptomatic dissection, aortic expansion >5.5 cm, aortic diameter >4.0 cm with true and false lumens both patent;
- chronic dissection (>92 days)—complicated/symptomatic dissection, aortic diameter >5.5 cm or expanding >0.5 cm/year.

Patients were recruited from the centres detailed in Appendix 1.

As is common in postmarket registries, patient recruitment was not consecutive and there is no information available concerning how many patients were treated outside of the registry in the recruiting centres. The nonconsecutive enrolment is a limitation of this study with regard to the clinical results reported, but has little impact on the morphological analysis. The VIRTUE registry was designed and funded as a 3-year study. The study closed at 3 years and so longer-term data are not available for analysis.

**Analysis of aortic morphology**

Patients entered into the registry underwent cross-sectional imaging (computed tomography angiography or magnetic resonance angiography) preprocedure, prior to discharge, and at 6, 12, 24, and 36 months postprocedure. Computed tomography and magnetic resonance imaging were performed according to local protocols and were not standardized across the study. The cross-sectional imaging was analysed by a core laboratory (University of Utrecht) to investigate aortic remodelling. Cross-sectional areas were determined for the total aorta, the true lumen, and the false lumen at four reproducible locations (2 cm distal to the left subclavian artery [LSA], 10 cm distal to the LSA, at the coeliac axis, and at the largest point of the descending thoracic aorta [DTA]). The degree of aortic remodelling post-TEVR was defined as the absolute change in aortic area (mm\(^2\)) from the baseline value determined on the pre-procedure scan.

The rate of complete false lumen thrombosis was site determined at three locations (first half of the DTA [LSA to carina], second half of the DTA [carina to diaphragm], and between diaphragm and coeliac axis) at all time points. Data were presented as cumulative false lumen thrombosis rates for the three clinical groups. False lumen thrombosis was assessed by the treating physicians, as this aspect is often difficult to interpret and there were differing post-procedural imaging protocols in the study institutions.

**Statistical analysis**

Statistical analysis was performed with SAS version 9.2 (SAS Institute, Cary, NC, USA). Clinical outcomes and false lumen thrombosis rates were defined by Kaplan—Meier estimated survival curves, and the differences between the three clinical groups analysed using the log rank test. Any differences between the three clinical groups with respect to aortic remodelling was analysed using repeated measures analysis of variance with any individual subgroup relationships defined using one-way analysis of variance. Significance was assumed at 1:20.

**RESULTS**

**All-cause and dissection-related mortality**

The 30-day and in-hospital mortality has been previously described in detail.\(^7\) Briefly, there were six in-hospital deaths in the cohort with acute complicated type B dissection, but no early deaths in the patients with subacute or chronic aortic dissection. Two deaths in the acute cohort were from myocardial ischaemia, one due to paraplegia and one from ongoing intestinal ischaemia. Two were dissection related, but no specific causation was identified.

The mid-term, all-cause, and dissection-related mortality are illustrated in Figs. 1 and 2. There was no statistical difference between the three clinical groups for all-cause (\(p = .20\)) or dissection-related mortality (\(p = .48\)).

There were three mid-term deaths in the acute group. One patient died of a cerebral haemorrhage 251 days after
TEVR. Two of the mid-term deaths were dissection related, with one patient dying from an acute upper gastrointestinal bleed due to the formation of an aorto-oesophageal fistula. The third patient died suddenly out of hospital owing to a retrograde type A dissection that occurred 65 days after the endovascular procedure. There was one death in the sub-acute group due to an extension of the thoracic dissection to involve the abdominal aorta with development of visceral malperfusion. Interventional therapy was not possible and the patient died 84 days after the original surgery. There were six deaths in the chronic group, two from cardiac causes, two from respiratory causes, and one each from gastrointestinal ischaemia and pancreatic cancer.

With regard to neurological complications, there was one mid-term cerebellar stroke in the acute group at 2 years. The patient originally had TEVR for a ruptured dissection

![Figure 1](image1.png)

**Figure 1.** Freedom from all-cause mortality by clinical group. There was no overall difference in survival between the three groups \(p = .200\). Numbers at risk at each time point (days) are tabulated below the figure. *Note.* TEVR = thoracic endovascular repair; Post TEVR = number of patients at risk immediately after TEVR.

<table>
<thead>
<tr>
<th>Group</th>
<th>30</th>
<th>90</th>
<th>180</th>
<th>270</th>
<th>365</th>
<th>730</th>
<th>1095</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute</td>
<td>49</td>
<td>45</td>
<td>42</td>
<td>41</td>
<td>39</td>
<td>38</td>
<td>37</td>
</tr>
<tr>
<td>Chronic</td>
<td>26</td>
<td>26</td>
<td>25</td>
<td>25</td>
<td>25</td>
<td>25</td>
<td>21</td>
</tr>
<tr>
<td>Sub-acute</td>
<td>24</td>
<td>24</td>
<td>23</td>
<td>23</td>
<td>23</td>
<td>23</td>
<td>19</td>
</tr>
</tbody>
</table>

![Figure 2](image2.png)

**Figure 2.** Freedom from dissection-related mortality by clinical group. There was no overall difference in survival between the three groups \(p = .480\). Numbers at risk at each time point (days) are tabulated below the figure. *Note.* TEVR = thoracic endovascular repair; Post TEVR = number of patients at risk immediately after TEVR.
with partial LSA coverage and no LSA revascularisation. There was one episode of transient spinal cord ischaemia in a patient originally treated for a chronic aortic dissection that occurred 1 year after the index operation.

Aortic related reinterventions, endoleak, aortic rupture, and retrograde type A dissection

The freedom from aortic-related interventions is illustrated in Fig. 3 and the nature of the most common interventions is given in Table 1. Although there was no significant difference in the three patient groups with respect to overall aortic reintervention (\( p = .19 \)), the need for additional thoracic endografts was greater in the patients with chronic dissection, with >30% of these patients requiring further TEVR. In the majority of cases, the additional endografts were required for distal aneurysmal degeneration of the chronic dissection below the primary endografts.

In total, there were four proximal type 1 endoleaks reported that required intervention. Two patients originally treated for subacute dissections required proximal endografts (one needing arch debranching). Similarly, two patients with chronic dissections needed revisional surgery for proximal endoleak, one requiring TEVR and the other surgical conversion. One patient required intervention for a type III endoleak. Seven patients with chronic dissection required distal extension owing to a distal type 1 endoleak or persistent perfusion of the false lumen with aortic expansion.

There were two cases of aortic rupture in the study. There was one early rupture in a patient with an acute dissection and one rupture in the mid-term due to persistent perfusion of the false lumen in a patient treated for a chronic dissection. The latter patient was successfully treated by distal endograft placement.

There were no cases of retrograde type A dissection (RTAD) in the subacute or chronic groups. There were two patients with RTAD in the acute group. One patient had a fatal event on day 65 and has been described above. The other patient developed chest pain 20 months after the index procedure and was diagnosed with RTAD and aortic valve regurgitation. Surgical repair was undertaken with a good clinical result.

Aortic morphology after TEVR

The luminal areas were defined for three anatomical aortic locations, 2 cm distal to the LSA, 10 cm distal to the LSA, and at the level of the coeliac axis. The luminal areas were also analysed at the point of maximum thoracic aortic diameter. The mean change in true lumen and false lumen area compared with preprocedural imaging is illustrated in Figs. 4 and 5 for the three clinical groups, at 10 cm distal to the LSA. Changes for all aortic locations are illustrated in the Supplementary Figs. S1 and S2.

Analysis of true lumen change demonstrated that, across all three clinical groups, the true lumen area increased after TEVR at all anatomic locations, and that the extent of true lumen expansion increased with time (\( p < .001 \)). There was no significant difference between the three clinical groups with regard to the expansion of the true lumen after TEVR. The majority of the aortic remodelling was complete within 6 months of TEVR.

The change in false lumen area was more heterogenous across the three clinical groups. At all three anatomical locations, the false lumen area decreased in the acute and

Figure 3. Freedom from aortic-related reinterventions by clinical group. There was no overall difference in reinvention between the three groups (\( p = .190 \)). Numbers at risk at each time point (days) are tabulated below the figure. Note. TEVR = thoracic endovascular repair; Post TEVR = number of patients at risk immediately after TEVR.
Table 1. The most common aortic reinterventions required in the three clinical cohorts. Some patients required multiple aortic reintervention.

<table>
<thead>
<tr>
<th></th>
<th>Acute (n = 50)</th>
<th>Subacute (n = 24)</th>
<th>Chronic (n = 26)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TEVR extension</td>
<td>4 (8%)</td>
<td>4 (17%)</td>
<td>8 (31%)</td>
</tr>
<tr>
<td>Open AAA repair</td>
<td>2 (4%)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Reballooning of graft</td>
<td>1 (2%)</td>
<td>0</td>
<td>1 (4%)</td>
</tr>
<tr>
<td>LSA embolisation</td>
<td>1 (2%)</td>
<td>1 (4%)</td>
<td>0</td>
</tr>
</tbody>
</table>

Note. TEVR = thoracic endovascular repair; AAA, abdominal aortic aneurysm; LSA = left subclavian artery.

Subacute clinical groups, with the extent of false lumen area reduction being significantly greater with time after TEVR ($p < .001$). In contrast, the false lumen area change was less consistent in the patients with chronic dissection, with both reduction in false lumen area and an increase in false lumen area being observed at different time points and different locations. At all three anatomical locations there was a significant difference in false lumen area change between the clinical groups ($p < .001$, $p < .001$, $p < .001$ for anatomic locations 2 cm distal LSA, and 10 cm distal LSA and coeliac axis, respectively), with the acute and subacute group demonstrating a greater reduction in false lumen area than the chronic clinical group. The differences between the clinical groups in terms of false lumen remodelling are illustrated by the analysis of the aorta at the largest point. This analysis demonstrated that the acute and subacute clinical groups demonstrated a reduction in false lumen area at all time points, which is in contrast to the chronic group where an overall increase in false lumen area was observed. There was a significant difference in false lumen area change between the acute and chronic group ($p < .001$) and the subacute and chronic groups ($p = .004$). There was no difference in false lumen area change between the acute and subacute groups.

The cumulative occurrence of false lumen thrombosis in the three clinical groups is illustrated in Figs. 6 and 7 for two anatomical regions (distal half of the DTA and between diaphragm and coeliac axis). The cumulative occurrence of false lumen thrombosis increased with time after TEVR, and was greater the more proximal the anatomical location. There was no difference in false lumen thrombosis rates between the three clinical groups in the proximal ($p = .330$) and distal DTA ($p = .520$). Analysis of false lumen thrombosis rates between the diaphragm and coeliac axis demonstrated that patients with chronic dissection had significantly lower false lumen thrombosis than patients with subacute or acute dissection ($p = .035$).

**DISCUSSION**

Patients with type B aortic dissection form a heterogenous group with regard to endovascular intervention. Endovascular repair of the thoracic aorta has been enthusiastically adopted for treatment of acute complicated type B dissection, and this technique has now been used for more chronic lesions. The indications for intervention in acute and chronic type B aortic dissections differ considerably and the aim of therapy often differs, with treatment in the acute group concentrating on relief of life threatening complications, whereas therapy in the chronic group must focus on the long-term prevention of aortic-related death. Given the inherent heterogenicity of the aortic pathology, outcome data must be stratified by the temporal classification of the aortic pathology.
dissection. The present study reports the mid-term clinical outcomes and aortic morphology after TEVR for acute, subacute, and chronic aortic dissection.

The early clinical results from the VIRTUE Registry have been reported previously, and demonstrated a 12% decrease in hospital mortality in the acute group, with no deaths in the patients with subacute or chronic dissections. Analysis of the mid-term results in the present study demonstrated that patients in the acute and subacute group had relatively low all-cause mortality following the index admission. In contrast, patients with chronic dissection appeared to show an increased (although non

Figure 5. Change in false lumen area plotted for the three clinical groups over the course of the study. The data are given as the mean change in true lumen area from baseline preoperative scans at 10 cm distal to the left subclavian artery. Numbers analysed at each time point (months) are tabulated below the figure. Note. TEVR = thoracic endovascular repair; Post TEVR = number of patients at risk immediately after TEVR.

Figure 6. Cumulative incidence of false lumen thrombosis plotted for the three clinical groups in the distal half of the descending thoracic aorta. Numbers analysed at each time point (days) are tabulated below the figure. Note. TEVR = thoracic endovascular repair; Post TEVR = number of patients at risk immediately after TEVR.
statistically significant) all-cause mortality from nonaortic pathology. Analysis of patient demographics did not reveal any obvious explanation for this appearance, with similar risk factors reported in all three groups.

While there are numerous reports of the early clinical outcomes of TEVR for aortic dissection, the mid- and long-term outcomes are more sparsely described. In particular, it will be important to ascertain whether endovascular techniques offer the same degree of protection from late aortic-related mortality that is described for open surgical repair, although the results from open surgery are inconsistent.11,12 In the present study, life table analysis demonstrated low rates of dissection-related mortality in the mid-term, which is concordant with other studies.13,14 There was no difference in mid-term clinical outcomes between the three clinical groups, although the small size of the groups makes this analysis subject to potential error.

The dissection-related deaths that occurred were due to RTAD, development of an aorto-oesophageal fistula, and an extension of previous dissection. In all, there were two cases of RTAD, both in patients with acute dissections. There were no cases of RTAD in the subacute or chronic group, which supports previous observations that RTAD may be associated with the fragile aortic intima in the acute setting.15,16 The lack of RTAD in this study is encouraging given that the endograft used in the study would, today, be regarded as an early design without tip capture.

The present study reported a very low rate of aortic rupture in the mid-term, which is similar to previous publications and suggests reasonable mid-term outcomes.17 However, the protection from aortic-related death comes at a price of a high reintervention rate in all three groups. The reintervention rate in the present study was similar to that described in previous work,18 and remains a cause of concern. In particular, there was a high requirement (seven of 26) for a distal extension to the primary endograft in patients with chronic dissection. This may be related to the poor aortic remodelling observed in patients with chronic dissection who subsequently developed distal aneurysmal degeneration below the primary endograft. The length of aorta covered by the endograft in the acute, subacute, and chronic groups was, respectively, 151, 166, and 164 mm, which suggests a similar operative strategy in all three clinical groups with regard to the extent of aortic coverage. The clinical data revealed in the present study suggest that limited coverage of the DTA in patients with chronic dissection leads to a high requirement for later distal extension. Given the lack of aortic remodelling in patients with chronic dissection and the requirement to achieve false lumen thrombosis, these data suggest that extensive aortic coverage should be considered in the primary procedure. More extensive aortic coverage in chronic dissections would also be supported by recent data that demonstrate that false lumen thrombosis is not reliably achieved below the level of the stent,19 and that complete false lumen thrombosis leads to better clinical outcome.5 Extending the length of coverage has theoretical risks in increasing paraplegia rates and so further data will be required to clarify this risk, especially as spinal ischaemia may be mitigated by continued false lumen perfusion.

Figure 7. Cumulative incidence of false lumen thrombosis plotted for the three clinical groups at the level of the coeliac axis. Numbers analysed at each time point (days) are tabulated below the figure. Note. TEVR = thoracic endovascular repair; Post TEVR = number of patients at risk immediately after TEVR.
Patients treated for chronic dissection generally have lower spinal cord ischaemia rates than those treated for degenerative aneurysms.\textsuperscript{27}

A unique aspect of the present study was that dissections were classified into three temporal clinical groups, and included a cohort of patients with subacute dissections. This allowed remodelling of the aorta, in response to TEVR, to be defined for different temporal classifications, in contrast to the traditional acute and chronic groups. The traditional classification has some limitations when defining aortic remodelling as the chronic group contains dissections from 2 weeks to many years in existence. The present study confirmed previous findings that aortic remodelling (true lumen expansion and false lumen regression) after TEVR is a continuous process,\textsuperscript{20} that acute dissections remodel more rapidly than chronic dissections,\textsuperscript{21} and that remodelling is greater in more proximal aortic segments.\textsuperscript{4}

A significant finding from the present study was that patients with subacute dissections showed aortic remodelling that was analogous to the acute group rather than the patients with chronic dissection. Patients in the subacute cohort demonstrated significantly greater false lumen area regression than patients with chronic dissection, while there was no difference between false lumen regression in the subacute and acute group. These findings are important and suggest that the aorta retains its plasticity to remodel in response to TEVR for at least 92 days after the index dissection.

In addition to changes in true lumen and false lumen area, aortic remodelling after TEVR aims to initiate complete false lumen thrombosis, which is an indicator of clinical success,\textsuperscript{22} and may influence long-term outcome.\textsuperscript{5} The present study confirmed that false lumen thrombosis is a continuous process, with a higher rate in the proximal aorta. The present study observed that the extent of false lumen thrombosis at the diaphragm was greater in the acute and subacute groups than in patients with chronic dissection. Again, this suggests similar aortic remodelling after TEVR in the acute and subacute groups. Interestingly, the timing of false lumen thrombosis appeared different to that of the aortic remodelling. While much of the aortic remodelling was complete at 1 year, false lumen thrombosis rates at the diaphragm continued rising to 3 years. The explanation for this is unclear, but has been reported in previous studies.\textsuperscript{4}

The aortic plasticity exhibited by the subacute group has relevance when considering therapy for patients who present with uncomplicated acute dissection. The INSTEAD trial has demonstrated a late survival benefit for patients with uncomplicated chronic dissection treated with TEVR as compared with standard medical management.\textsuperscript{23,24} This survival benefit appeared to be associated with a high rate of false lumen thrombosis and aortic remodelling; interestingly, the majority of patients in this trial were randomised in the early chronic phase of dissection.\textsuperscript{25} Given the low rate of complications for the endovascular treatment of patients with subacute dissection reported in the present study and elsewhere,\textsuperscript{8} the aortic plasticity observed in this phase of the disease opens a potentially attractive therapeutic window. Further clinical data are required to define exactly which groups of patients with uncomplicated dissection require intervention.

The present study has some limitations with regard to the analysis of aortic morphology and clinical outcomes. The clinical groups are insufficiently large to allow detailed analysis of the factors, aside from the defined clinical groups, that predispose to remodelling and false lumen thrombosis. In particular, detailed information regarding the exact location of primary and secondary tears is not available. The clinical outcomes may have been affected and potentially biased, as the patients in the registry were not enrolled consecutively.

The VIRTUE study has provided detailed clinical and morphological data regarding endovascular treatment of type B aortic dissection. The principle clinical findings suggest that TEVR is able to provide good protection from aortic-related death in the mid-term, but with a high rate of aortic reintervention. The present study has identified that technical refinements to the operative procedure might be able to reduce the rate of reintervention in the chronic groups with greater aortic coverage in the primary procedure. Analysis of aortic morphology has suggested that aortic remodelling in the subacute patients is similar to the acute group. Retention of aortic plasticity in the subacute group lengthens the therapeutic window for the treatment of uncomplicated type B dissection.

**FUNDING**

The VIRTUE study was a postmarket registry funded by Medtronic.

**CONFLICT OF INTEREST**

All institutions/investigators received payment from Medtronic for patient recruitment.

**APPENDIX A. SUPPLEMENTARY DATA**

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.ejvs.2014.05.007.

**APPENDIX 1. CONTRIBUTING CENTRES AND INVESTIGATORS WITH LEAD INVESTIGATOR AND NUMBER OF PATIENTS RECRUITED**

St. Antonius Ziekenhuis, Nieuwegein (R. Heijmen, 20 patients); Ospedale Sant’Orsola Malpighi, Bologna (R. Fattori, 19 patients); St George’s Vascular Institute (M. Thompson, 16 patients); Universitätsklinikum Freiburg (H. Roos, two patients); Azienda Osp “Santa Maria della Misericordia”,
REFERENCES


9 The VIRTUE registry of type B thoracic dissections—study design and early results. Eur J Vasc Endovasc Surg 2011;41:159—66.


