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Abstract

**Background:** Surgery confers the best chance of survival following acute Type A dissection (ATAD), yet perioperative mortality remains high. Although perioperative risk factors for mortality have been described, information on the actual causes of death is sparse. In this study, we aimed to characterize the inciting events causing death during surgical repair of ATAD.

**Methods:** Nine centers participated in the study. We included all patients who died following surgical repair for ATAD between January 2007 and December 2013. An aortic surgeon at each site determined the primary cause of death from seven predetermined categories: cardiac, stroke, hemorrhage, other organ ischemia (peripheral, renal, or visceral), multiorgan failure, sepsis, or other causes. Additional characteristics and variables were analyzed to delineate potential modifiable factors for mortality.

**Results:** Of the 692 surgeries for ATAD, there were 123 deaths (17.8% mortality rate). Mean age at death was 66 years. Events contributing to death were: cardiac (25%), stroke (22%), hemorrhage (21%), multiorgan failure (12%), other organ ischemia (11%), sepsis (4%), and other causes (5%). Neurologic injury at presentation was a predictor of stroke as the inciting cause of death (p = 0.04). Peripheral, renal, or visceral ischemia at presentation was highly predictive of death due to these presenting ischemic conditions (p = 0.004). We found no associations between cardiogenic shock, tamponade, or cardiopulmonary bypass duration and cardiac death.

**Conclusion:** Operative mortality for ATAD remains high in Canada. Nearly 70% of deaths arise from cardiac failure, stroke, or hemorrhage. Therefore, novel surgical, hybrid, and endovascular strategies should target these three areas.

**Key Words:**
Aorta • Aortic dissection • Outcomes
Introduction

The diagnosis of an acute Type A dissection (ATAD) is associated with a high mortality rate, near 50% at 48 hours without surgical intervention [1]. Despite surgery being the best treatment option, rates of in-hospital mortality following surgical repair remain quite sobering. Recent data from the International Registry of Acute Aortic Dissection (IRAD) show a decrease in operative mortality from 31% to 22% over a 17-year time period [2]. The German Registry for Acute Aortic Dissection Type A (GERAADA) recently reported a 30-day mortality of 17% for surgically treated ATAD [3]. To improve upon these results, modifiable factors need to be sought out and defined. Although preoperative risk factors predicting operative mortality are well described for ATAD [4–6], the inciting cause of death is not well understood. Therefore, characterizing the cause of death may guide modifications in surgical approach to improve outcomes. This study aimed to characterize the cause of death following surgical repair of ATAD in the current era.

Materials and Methods

The Canadian Thoracic Aortic Collaborative (CTAC) is a group of cardiovascular clinicians across Canada with specific interest and expertise in the management of thoracic aortic disease. Nine Canadian cardiac surgery centers in the collaborative participated in this study. Institutional Review Board approval was obtained from each site. Patient informed consent was waived, as the cohort of interest was comprised solely of patients who were deceased. The primary outcome of interest was cause of death following surgical repair of ATAD. Patients who were diagnosed with an ATAD but did not undergo surgery were excluded. Cause of death was classified into one of seven predetermined categories: cardiac failure, stroke, hemorrhage, other organ ischemia (peripheral, renal, or visceral), multiorgan failure, sepsis, and other causes. Definitions of the seven predetermined categories are listed in Table 1. A single attending cardiac surgeon from each site reviewed the data and determined the primary cause of death for each case. In instances in which multiple categories may have contributed to the final outcome, the surgeon selected the single inciting event.

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
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<tr>
<td>Cardiac failure</td>
<td>Patient was in cardiogenic shock, requiring inotropes and/or intra-aortic balloon pump support with ventricular dysfunction noted in the operating room. Patient showed difficulties weaning from cardiopulmonary bypass, documented perioperative myocardial infarction, or low cardiac output state postoperatively resulting in death.</td>
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<tr>
<td>Stroke</td>
<td>Patient was clinically comatose or suffered profound neurological injury in the postoperative period with computer tomographic or magnetic resonance imaging to support diagnosis.</td>
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<td>Hemorrhage</td>
<td>Patient required massive blood transfusion in the face of active bleeding. Despite volume resuscitation efforts, unrecoverable hemodynamic instability persisted, leading to cardiogenic shock or multiorgan failure.</td>
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<tr>
<td>Other organ ischemia</td>
<td>Clinical and supportive laboratory data showed peripheral, renal, or visceral end organ dysfunction.</td>
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<tr>
<td>Multiorgan failure</td>
<td>Patient died of multi-organ failure, with no specific inciting event beyond multiorgan failure identified.</td>
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<tr>
<td>Sepsis</td>
<td>Patient showed clinical signs of septicemia leading to multiorgan failure despite maximal medical support, with blood, urine, or sputum cultures supporting unrelenting bacterial infection.</td>
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<tr>
<td>All other causes</td>
<td>Any death that did not fit into one of the six other categories.</td>
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felt most likely to be responsible for death. Surgical strategies and perioperative management at the time of aortic repair (e.g., arterial and venous access, myocardial and cerebral protection, anesthetic and intensive care unit protocols) were at the discretion of the operative surgeon and perioperative team. The conventional strategy of immediate operative repair and aortic reconstruction, removing aortic tissue at the site of injury to restore true lumen blood flow as expeditiously as possible, was used in all cases. Alternative treatment algorithms, such as delaying surgery for preferential percutaneous fenestrations or selective branch vessel stenting in the presence of malperfusion syndromes, were not utilized in this cohort.

Baseline characteristics, clinical status on presentation to the hospital, intraoperative variables, and postoperative complications were retrospectively reviewed from datasets at each hospital. In cases of missing data or dataset discrepancies, individual patient charts were reviewed to ensure accuracy. Descriptive statistics for continuous variables were expressed as mean ± standard deviation (SD) or median and range, as appropriate. Independent t-tests were used to evaluate differences in normally distributed continuous variables. For non-normally distributed continuous data, Wilcoxon Signed rank tests were used. Chi-square tests were used to assess the association between presenting clinical characteristics and a specific cause of death for categorical variables. When a cell count was less than five in a 2 × 2 table, Fisher’s exact tests were used. Statistical significance was determined by a two-sided \( p < 0.05 \). Statistical analyses were performed with SAS version 9.3 (SAS Institute, Inc., Cary, NC, USA).

Baseline characteristics for the cohort are shown in Table 2. The mean age at death was 65.9 ± 12.8 years. Women accounted for 50% of the cohort.

### Results

During the 7-year study period, 692 patients underwent surgical repair for ATAD at the nine participating institutions. There were 123 deaths, for a mortality rate of 17.8%. Datasets were compiled and charts reviewed for these 123 non-survivors.

The most common causes of death were cardiac failure (31 patients, 25.2%), stroke (27 patients, 22.0%), and uncontrollable hemorrhage (26 patients, 21.1%). Less common causes of death were multi-organ failure (15 patients, 12.2%), other organ ischemia (13 patients, 10.6%), sepsis (5 patients, 4.1%), and other causes (6 patients, 4.9%) (Figure 1).

Clinical status at the time of presentation to the treating hospitals is described in Table 3. Almost half of the patients were taken to the operating room with an emergent or salvage status. One-third of patients were in cardiogenic shock, and 31% either had a focal neurologic injury or were in a coma.

Operative characteristics are shown in Table 4. Adjunct antegrade cerebral perfusion was used for one-third of patients, and retrograde cerebral perfusion was used for 18% of patients. For patients without adjunct cerebral perfusion, median cerebral ischemic time was 22 min (range, 2–100 min). The median lowest systemic temperature for the cohort was 19°C (range, 13–34°C). Median cardiopulmonary bypass time was 3 h and 12 min. Of the 121 patients with data available regarding site of arterial cannulation, the femoral artery was used in just over half of patients. Right axillary cannulation was used in 37% of patients, the distal arch in 3% of
We analyzed the relationship between cause of death and patients’ preoperative clinical status and intraoperative surgical techniques. Patients presenting with focal neurologic injury or coma were more likely to have stroke as the inciting cause of death (34.3% vs. 17.2%, p = 0.04). Patients presenting with peripheral, renal, or visceral ischemia were more likely to have died from peripheral, renal, or visceral ischemia (28.6% vs. 6.9%, p = 0.004). There was a non-significant trend toward an association between peripheral, renal, or visceral ischemia at presentation and peripheral, renal, or visceral ischemia or multi-organ failure as the cause of death (38.1% vs. 19.8%, p = 0.07). Cardiac failure as the cause of death was not associated with a presenting status of tamponade (p = 0.37), cardiogenic shock (p = 0.33), or cardiopulmonary bypass time (p = 0.26).

We also analyzed potential associations between intraoperative protection strategies and stroke as the cause of death. No clear associations were identified. There was no association between circulatory arrest time and stroke as the cause of death. The median circulatory arrest time was 23.5 min (range, 1–65 min) for patients who died secondary to stroke and 25 min (range, 1–141 min) for patients who died from other causes (p = 0.93). Moreover, antegrade cerebral perfusion was patients, and alternative sites in 8% of patients. The extent of aortic replacement was conservative, with only 10% of patients receiving total arch replacement. Adjunct thoracic endovascular stent placement was used in only one patient.

Postoperative complications are shown in Table 5. Nearly half of the patients had some form of cardiac complication, although only 25% ultimately died from a cardiac cause. Nearly one-third of patients had a postoperative stroke, with 22% eventually succumbing to this cerebrovascular event. One-fifth of patients required re-exploration for bleeding or experienced multi-organ failure, and over 25% of patients experienced respiratory failure or renal failure requiring dialysis.

Table 3. Clinical status on presentation to treatment hospital (n = 123).

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurological injury, focal</td>
<td>32 (26)</td>
</tr>
<tr>
<td>Neurological injury, coma</td>
<td>6 (5)</td>
</tr>
<tr>
<td>Limb ischemia</td>
<td>16 (13)</td>
</tr>
<tr>
<td>Visceral ischemia</td>
<td>8 (7)</td>
</tr>
<tr>
<td>Tamponade</td>
<td>33 (27)</td>
</tr>
<tr>
<td>Shock</td>
<td>41 (33)</td>
</tr>
<tr>
<td>Emergent salvage</td>
<td>59 (48)</td>
</tr>
</tbody>
</table>

Figure 1. Distribution of patient mortality depending on cause of death during surgical repair for acute Type A dissection at nine surgical centers across Canada between 2007 and 2013.
not more protective against stroke as a cause of death compared to other cerebral management strategies. Of the 77 patients who received deep hypothermic circulatory arrest or retrograde cerebral perfusion as a cerebral management strategy, 26% (20/77) died of stroke, whereas of the 38 patients who received antegrade cerebral perfusion for cerebral protection, 18% (7/38) died of stroke ($p = 0.36$). There were no association between temperature and stroke as the cause of death ($p = 0.54$).

**Discussion**

In this study, we aimed to identify the inciting
events causing death during surgical repair of ATAD. With a more formal characterization of the cause of death in this patient population, operative variables and their associations with death may be better assessed. The 30-day mortality observed in this cohort was 17.8%. This mortality rate is consistent with those in larger worldwide registries such as IRAD (mortality = 20%) and GERAADA (mortality = 17%). These results highlight the high mortality associated with surgical repair of ATAD and the need for the international aortic community to improve upon this outcome. As cardiac failure, stroke, and uncontrollable bleeding were the primary causes of death in almost 70% of cases in the present series, focused efforts to improve management strategies specific to these three areas are likely to have the highest yield in improving outcomes.

Stroke is a frequent and major complication associated with ATAD [7]. In the present series, 26% of non-surviving patients presented with a focal neurological deficit, and 30% were diagnosed with a new postoperative stroke. Whether these strokes were due to embolic phenomena, dissection of the arch or distal intracranial vessels, or hypoperfusion at the time of surgery is not known. The impact of postoperative neurologic injury is significant, as 17% of deaths were due to new strokes not detected preoperatively, suggesting that they may be directly related to operative strategy or progression of the pathology. This study also highlights the variance in cerebral perfusion strategies employed across centers for surgical repair of ATAD, with 49% of non-survivors receiving deep hypothermic circulatory arrest as the lone protection strategy. Although data continue to accrue in support of antegrade cerebral perfusion for cerebral protection for all forms of aortic surgery [8–10], within this cohort of non-survivors, antegrade cerebral perfusion did not decrease the risk of death due to stroke. Choice of cerebral management strategy needs to be taken in context, as it may have more to do with the emergent status of the patients (48% of patients presented in an emergent or salvage state) than surgeon preference for or against adjunct cerebral management strategies. Extent of surgical repair may also be related to neurologic complications. Whereas the most common approach to repairing ATAD is conservative, as was the case in the present series, some data suggest that identification of resid-
vival after surgical repair of ATAD is also highlighted by this study. Twenty percent of patients in this non-survivor series presented with limb or visceral ischemia. Although only 11% of patients were thought to subsequently die from peripheral, renal, or visceral ischemia, patients were four times more likely to die from malperfusion if they presented to the hospital with one of these malperfusing states. This is in line with a recent registry review reporting a significant increase in mortality, from 12.6% with no malperfusion at the time of surgery, up to 21.3% with one malperfused organ system, and up to 43.4% when three organ systems were affected [14]. Therefore, expeditious restitution of perfusion is absolutely paramount in such scenarios.

The timeframe from diagnosis to surgery also warrants discussion. Although treatment protocols call for immediate aortic reconstruction upon diagnosis, in the event that the diagnosis is identified at a smaller center without cardiac surgery services, time for transfer to a tertiary center could impact preoperative status and postoperative outcome. The Canadian health system is structured such that the vast majority of patients would have presented directly to a tertiary center, but for a small percentage of patients, transfer from smaller hospitals to more specialized centers would have been required. Many patients in this cohort presented in extremis conditions (33% were in shock at presentation). This could reasonably be attributed to the calamity of the aortic pathology, but the degree to which delayed intervention was also a contributing factor cannot be fully ascertained.

This study suggests that focused efforts to mitigate cardiac, cerebral, and bleeding complications may improve outcomes after surgical repair of ATAD. Patients presenting to the hospital in need of emergent life-saving aortic surgery bring forth unique challenges not encountered in elective scenarios. As the time delay from diagnosis to treatment is critical to survival, an organized algorithm of medical resuscitation, emergent investigations, and timely transfer to the operating room is necessary. The importance of timely transfer to the operating room negates the ability to modify preoperative risk factors prior to treatment. Therefore, quality initiatives to improve outcomes in the emergent setting need to focus on the betterment of intraoperative treatment strategies in addition to early recognition and correction of operative or postoperative complications. These become the only truly modifiable factors when faced with an emergent condition such as ATAD. With this in mind, novel surgical practices, hybrid techniques, and incoming endovascular devices should either focus on cerebral or myocardial protection or facilitate a reduction in intraoperative blood loss in an effort to improve results.

With respect to novel surgical practices, the idea of dedicated aortic surgeons and a defined aortic team within the greater cardiac surgery division is likely a worthwhile endeavor. A dedicated aortic team facilitates repeated exposure and much-needed caseload experience to develop more mastery of the operation. In this sense, the surgeon, or more precisely the surgeon’s experience, is the modifiable factor. Outcome data from cardiac centers committed to the aortic team philosophy demonstrate significant improvements to patient care with the implementation of such strategies [15–16]. Another surgical practice of likely benefit, as previously mentioned, is a more liberal use of total arch replacement in the face of cerebral malperfusion or a primary tear within the aortic arch [11]. Concerns about the potential increase in operative risk associated with this more aggressive approach could arguably be mitigated by greater expertise of the surgeon through an added caseload enabled by the aortic team concept.

Although not geared toward cerebral and myocardial protection, visceral malperfusion remains a lethal problem, and hybrid techniques to combat this issue with insertion of a frozen elephant trunk have shown early promise [17]. Alternatively, the delayed approach to surgery with initial endovascular interventions has also proved to be a useful strategy in the face of visceral malperfusion [18]. Each of these approaches has merit in select cases. Efforts to disseminate and more routinely implement such techniques should be promoted.

Building upon these techniques and projecting forward with this technology, it is conceivable that future technological advances in this area may spawn smaller devices for carotid artery deployment for cerebral protection. Such concepts and other “outside the box” research endeavors are needed if we are to make a sincere push toward improving outcomes of ATAD. This is an unchartered area in which future re-
search and development may have high yield.

Our study has some limitations. This is a retrospective review of 123 non-survivors from a larger cohort of 692 patients who underwent surgical repair of ATAD. This study focused on events that had significant impact on death, yet it did not capture similar events that may have occurred in the 569 surviving patients. Although a complete dataset on the total cohort of 692 patients would have been ideal, this was logistically unattainable. Recognizing this major limitation, due to the unique non-modifiable emergent status of ATAD patients, non-survivor data does, in and of itself, convey important information for the aortic team to discuss. Although it is possible that some findings within the non-survivor cohort would not hold true if assessed across the greater cohort, this does not lessen the importance of these findings to the surgeon and aortic team. As non-survivor cohorts highlight extremis conditions, they provide opportunities to develop strategies that mitigate such extremes. Moreover, because the data were captured retrospectively, they possess the inherent limitations of retrospective data. As it is possible that surgeons misclassified the cause of death, every effort was made to minimize such misclassifications by performing chart reviews in cases of discrepancy. Finally, this is a descriptive study with post-hoc analyses, and all observed associations should be considered exploratory.

Conclusion

Operative mortality following surgery for ATAD in the contemporary era remains high. Cardiac failure, postoperative stroke, and hemorrhage were the cause of death in nearly 70% of cases. In an effort to improve outcomes, emerging surgical, hybrid, and endovascular strategies should target these three areas.

Conflict of Interest

The authors have no conflict of interest relevant to this publication.

References


Impact of Thoracic Endovascular Repair on Pulsatile Aortic Strain in Acute Type B Aortic Dissection

Preliminary Results

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Abstract

**Background:** The impact of thoracic endovascular aortic repair (TEVAR) on pulsatile aortic strain remains undetermined in patients with Type B aortic dissection (TBAD). Therefore, we quantified pulsatile aortic strain in TBAD patients and control subjects.

**Methods:** We retrospectively analyzed two TBAD patients from our database with cardiac-gated computed tomography angiography imaging available before and after TEVAR and two control subjects (67- and 76-year-old males). Patient 1 (54-year-old female) presented with acute TBAD, and Patient 2 (55-year-old male) had Marfan syndrome and ruptured acute TBAD. Custom-developed software was used to compute aortic length, diameter, and area during the cardiac cycle. Pulsatile strain was calculated as systolic increments of length and circumference divided by corresponding diastolic values.

**Results:** Before TEVAR, pulsatile longitudinal strain of the thoracic aorta was lower in TBAD patients (1.4–1.7%) than in control subjects (2.1–4.5%). After TEVAR, pulsatile longitudinal strain increased proximal to the stent-graft by 65% in the arch of Patient 1 and by 70% in the ascending aorta of Patient 2. Pulsatile circumferential strain was elevated in false lumen patency (4.4–6.2%) compared with thrombosed false lumen (1.4–2.1%) or control subjects (0.9–3.3%). Following TEVAR, circumferential measurements within stented segments were deemed unreliable due to artifacts.

**Conclusions:** TEVAR led to a considerable increase of pulsatile longitudinal strain proximal to the stent-grafts, and TBAD was associated with longitudinally stiffer aortas, which may be part of the pathophysiology of TEVAR-related complications such as retrograde dissection and aneurysmal dilatation. These preliminary data call for larger prospective studies.

**Key Words**

Type B aortic dissection • Thoracic endovascular aortic repair • Aortic strain

Introduction

Thoracic endovascular aortic repair (TEVAR) has been successfully adopted to treat complicated Type B aortic dissection (TBAD) [1]. However, major
TEVAR-related complications are widely reported with retrograde or antegrade dissection, typically originating at the proximal or distal end of the device, with rupture being the most lethal [2–4]. Unfortunately, the mechanisms of these TEVAR-related complications are mostly unknown. Changes in aortic dynamics during the cardiac cycle, induced by a mismatch between the stent-graft and the aorta, are suggested to play a key role [2, 5]. However, little is known about deformations of the dissected aortic wall, and few studies have reported strain after endovascular repair [6].

Biomechanical studies show that current thoracic stent-grafts are about 125 times stiffer than the adult thoracic aorta (55.2 MPa vs. 0.44 MPa) [7, 8]. Implantation of such stiff devices may underlie reported cases of TEVAR-induced acute hypertension and cardiac remodeling [9, 10]. However, the exact nature of TEVAR-induced local changes in aortic wall strain remains to be determined. While most dynamic imaging studies have focused on aortic area or diameter changes during the cardiac cycle [11–13], the importance of longitudinal strain should be not underestimated, as it is predominantly the longitudinal axis that fails in the event of aortic dissection [14–16]. During each cardiac contraction, the heart pulls the aortic root downward, resulting in longitudinal strain of about 7–9% in the ascending aorta [15, 17]. Implantation of a rigid stent-graft in the proximal descending aorta might stiffen that segment, potentially forcing the ascending aorta and aortic arch to overstretch during each heartbeat, as we observed in a study of eight aneurysm patients managed with TEVAR (unpublished data). Such elevated wall stress has been associated with increased risk of aortic dissection [15] and aneurysm growth [5].

These considerations led us to quantify pulsatile aortic strain in both longitudinal and circumferential directions in acute TBAD patients before and after TEVAR as well as control subjects. For this purpose, we used a semi-automatic technique to post-process cardiac-gated computed tomography angiography (CTA) image data.

Materials and Methods

For this retrospective study, we included patients with acute TBAD enrolled in our imaging database who underwent cardiac-gated CTA before and after TEVAR at the University of Utrecht, The Netherlands, which resulted in a limited number of patients (n = 2). Two patients without thoracic aortic disease served as control subjects (Table 1). The local ethical review board evaluated the study protocol; formal approval was given, and informed consent was waived.

Patient Population

Patient 1 was a 54-year-old female with acute TBAD and thrombosed false lumen (Figure 1A). This patient underwent TEVAR using two Bolton Relay (Bolton Medical Inc, Sunrise, FL) stent-grafts (Figure 1B). The proximal landing zone was just distal to the left common carotid artery, covering the left subclavian artery (LSA), with distal extension 10 cm above the celiac bifurcation. Stent-graft oversizing was 8%. A post-operative cardiac-gated CTA was conducted 3 weeks later, which was used for this study. During 5 years of follow-up, no endoleaks or other stent-graft-related

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (Years), Gender</th>
<th>Aortic Disease</th>
<th>Dissection Extension</th>
<th>Stent-graft Types</th>
<th>Stent-graft Sizes (mm)</th>
<th>Stent-graft Length in situ (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>54, F</td>
<td>Acute TBAD</td>
<td>5 cm distal from the LSA to the aortic bifurcation</td>
<td>Bolton Relay</td>
<td>36-36-200</td>
<td>174</td>
</tr>
<tr>
<td>2</td>
<td>55, M</td>
<td>Ruptured acute TBAD, Marfan syndrome</td>
<td>3 cm distal from the LSA into right iliac artery</td>
<td>Medtronic Captivia</td>
<td>34-34-200</td>
<td>235</td>
</tr>
<tr>
<td>3</td>
<td>66, M</td>
<td>None</td>
<td></td>
<td></td>
<td>36-36-150</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>75, M</td>
<td>None</td>
<td></td>
<td></td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

LSA = left subclavian artery; TBAD = Type B aortic dissection.
Two Medtronic Captivia (Medtronic Vascular, Santa Rosa, CA) stent-grafts were implanted, with extension distal from the LSA to just proximal to the celiac bifurcation (Figure 1D). The stent-graft diameter was oversized by 40% considering the ruptured aorta and hypotension [18] found during examination as well as the unavailability of smaller devices in the acute setting in our hospital. The cardiac-gated CTA conducted at the second presentation and 3 days postoperatively were used for this study. After 1 month, CTA imaging revealed a pseudoaneurysm caused by aortic perforation of a proximal bare stent strut, as previously reported [19]. A proximal stent-graft extension was implanted (32-32-164 mm), landing just distal to the left common carotid artery and covering the origin of the LSA. No endoleaks or other stent-graft-related complications occurred during 5 years of follow-up, with complete thrombosis of the false lumen and subsequent positive remodeling of the aorta.

Patients 3 and 4 were both males (67- and 76-years-old) with no visible evidence of thoracic aortic disease and no medical history of aortic surgery or connective tissue disorder.

**Workflow from Data Collection to Strain Quantification**

The workflow for this study is illustrated in Figure 2 and described stepwise below.

1. Image acquisition and data collection: Basic input data was a dicom set of cardiac-gated CTA images obtained during eight phases of the cardiac cycle. These dynamic images were obtained with a 256-row multislice CT system (Philips Medical System, Best, The Netherlands) and imported into 3Mensio software (3Mensio Medical Imaging, Biltoven, The Netherlands) for analysis. A three-dimensional (3-D) scan volume was acquired during all eight phases, for which maximum (end-systolic) and minimum (end-diastolic) measurements were acquired. Patients were included in the study only if image acquisition was accomplished successfully and image quality was considered adequate. Pixel spacing and slice thickness were similar for all patients and are described in Supplemental Table 1 (see supplemental Table 1 at http://dx.doi.

![Figure 1. Panels A-D. Computed tomography angiography (CTA) imaging showing the acute type B aortic dissection (TBAD) of Patient 1 (Panel A) before and (Panel B) after thoracic endovascular aortic repair (TEVAR) and the ruptured acute TBAD of Patient 2 (Panel C) before and (Panel D) after TEVAR.](image-url)
Figure 2. Workflow. **Panel A.** Cardiac-gated computed tomography angiography imaging provided dicom imaging sets during eight phases of the cardiac cycle. **Panel B.** The thoracic aorta was segmented at each phase, and three-dimensional reconstructed models were computed. **Panel C.** Center lumen lines were computed. **Panels D and E.** Longitudinal and circumferential segments were identified using side branch origins as anatomical landmarks, resulting into (**Panel D**) longitudinal segments (ascending aorta (purple), aortic arch (green), and descending aorta (red)) and (**Panel E**) circumferential levels (sinotubular junction (STJ), just before the origin of the brachiocephalic trunk (BCT), left subclavian artery (LSA), 10 cm (LSA + 10 cm) and 20 cm (LSA + 20 cm) distal to the left subclavian artery, and celiac trunk (CT)). Lengths, areas, and diameters were then calculated by a custom-developed script [21].
LSA, 10 and 20 cm distal to the LSA, and celiac trunk (Figure 2E). Diameters were measured in 360° by calculating the distance between all points constituting the section of interest perpendicular to the center lumen line and then extracting the end-diastolic and end-systolic values, as previously described [12, 13]. Aortic areas were measured by triangulating the particular section and summing the area of all obtained triangles. Pulsatile circumferential strain was computed as the difference between end-systolic and end-diastolic circumference divided by end-diastolic circumference.

**Intra- and Inter-Observer Variability**

Length and area measurements were acquired twice by the same investigator (C.T.) and blindly repeated by another investigator (F.N.) to allow for intra- and inter-observer variability.

**Statistical Analysis**

Data were analyzed with SPSS 22.0 (SPSS, Chicago, IL). Continuous data are presented as mean ± standard deviation (SD) unless stated otherwise, and categorical data are given as counts (percentage). Intra- and inter-observer variability were calculated according to Bland and Altman. Data were considered normally distributed based on skewness and kurtosis Z-values between -1.96 and 1.96, a Shapiro-Wilk test p > 0.05, and visualization of approximately normally distributed data with histograms. p < 0.05 were considered statistically significant.

**Results**

Patient and procedural characteristics are shown in Table 1. The median interval from TEVAR to post-operative cardiac-gated CTA imaging was 0.4 months (range, 0.1–0.7).

**Aortic Length and Pulsatile Longitudinal Strain**

Aortic length and pulsatile longitudinal strain before and after TEVAR are shown in Table 2. In Patient 1, the end-systolic length of the descending aorta decreased following TEVAR, while the ascending aorta...
Aortic Diameter and Pulsatile Circumferential Strain

End-systolic diameter and circumferential strain are shown in Table 3. Overall, smaller diameters of the true lumen were observed in TBAD patients than in control subjects (from 10 cm distal to the LSA and celiac trunk). Figure 4 illustrates pulsatile circumferential strain before TEVAR, which ranged from 0.9% to 3.3% and was comparable among Patient 1 and the two control subjects. However, Patient 2, who presented with acute TBAD and patent false lumen, showed considerably higher circumferential strain at sections of the patent false lumen (4.4% at 20 cm distal to the LSA and 6.2% at the celiac trunk).

After TEVAR, the true lumen expanded in sections adjacent to the stent-graft (brachiocephalic trunk and celiac trunk). Pulsatile circumferential strain also increased in sections adjacent to the stent-grafts, except for the distal adjacent section in Patient 2 (celiac trunk with patent false lumen), which decreased considerably (6.2% before TEVAR vs. 2.5% after TEVAR). Diameter and area measurements within stented sections were deemed unreliable due to stent artifacts in the segmentations and were therefore excluded from this analysis.

Table 2. End-systolic aortic length and longitudinal strain for each patient.

<table>
<thead>
<tr>
<th>Pt</th>
<th>Location</th>
<th>Total Thoracic</th>
<th>Ascending</th>
<th>Arch</th>
<th>Descending</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>End-systolic length (mm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Pre-TEVAR</td>
<td>363.7 ± 1.8</td>
<td>80.7 ± 1.6</td>
<td>38.2 ± 1.0</td>
<td>246.3 ± 1.5</td>
</tr>
<tr>
<td></td>
<td>Post-TEVAR</td>
<td>361.1 ± 2.2</td>
<td>83.4 ± 1.4</td>
<td>42.6 ± 1.8</td>
<td>238.6 ± 1.8</td>
</tr>
<tr>
<td>2</td>
<td>Pre-TEVAR</td>
<td>391.2 ± 3.1</td>
<td>74.0 ± 2.5</td>
<td>42.3 ± 1.4</td>
<td>281.0 ± 4.2</td>
</tr>
<tr>
<td></td>
<td>Post-TEVAR</td>
<td>398.7 ± 5.4</td>
<td>80.6 ± 3.8</td>
<td>43.9 ± 1.4</td>
<td>275.1 ± 1.8</td>
</tr>
<tr>
<td>3</td>
<td>Control</td>
<td>373.3 ± 5.1</td>
<td>73.0 ± 3.5</td>
<td>55.1 ± 0.9</td>
<td>246.3 ± 1.4</td>
</tr>
<tr>
<td>4</td>
<td>Control</td>
<td>336.3 ± 2.3</td>
<td>52.0 ± 2.2</td>
<td>50.1 ± 0.8</td>
<td>235.2 ± 1.3</td>
</tr>
</tbody>
</table>

Longitudinal strain (%)

<table>
<thead>
<tr>
<th>Pt</th>
<th>Location</th>
<th>Total Thoracic</th>
<th>Ascending</th>
<th>Arch</th>
<th>Descending</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Pre-TEVAR</td>
<td>1.4</td>
<td>5.0</td>
<td>8.6</td>
<td>1.5</td>
</tr>
<tr>
<td></td>
<td>Post-TEVAR</td>
<td>1.7</td>
<td>5.3</td>
<td>14.2</td>
<td>2.4</td>
</tr>
<tr>
<td>2</td>
<td>Pre-TEVAR</td>
<td>2.4</td>
<td>9.9</td>
<td>9.6</td>
<td>4.9</td>
</tr>
<tr>
<td></td>
<td>Post-TEVAR</td>
<td>4.4</td>
<td>16.8</td>
<td>9.5</td>
<td>1.7</td>
</tr>
<tr>
<td>3</td>
<td>Control</td>
<td>4.5</td>
<td>14.0</td>
<td>3.9</td>
<td>1.7</td>
</tr>
<tr>
<td>4</td>
<td>Control</td>
<td>2.1</td>
<td>12.9</td>
<td>4.2</td>
<td>1.7</td>
</tr>
</tbody>
</table>

Values are shown as mean ± SD, where appropriate. TEVAR = thoracic endovascular aortic repair.

and arch elongated. Patient 2 showed similar shortening of the descending aorta with elongation of the ascending aorta and arch after TEVAR.

Pulsatile longitudinal strain before and after TEVAR in the two acute TBAD patients are illustrated in Figure 3. Before TEVAR, pulsatile longitudinal strain throughout the thoracic aorta ranged from 1.5% to 9.9% in the two TBAD patients and from 1.7% to 14.0% in the two control subjects, with mean pulsatile longitudinal strain of the total thoracic aorta of 1.9 ± 0.5% and 3.3 ± 1.2%, respectively. In three of the four patients, pulsatile longitudinal strain was highest in the ascending aorta (9.9–14.0%), decreasing downstream along the arch (3.9–9.6%) and descending aorta (1.7–4.9%).

After TEVAR, an increase of pulsatile longitudinal strain was observed proximal to the stent-graft in both TBAD patients (65% increase in the arch of Patient 1, and 70% increase in the ascending aorta of Patient 2). In the descending aorta, Patient 1, with a stent-graft length of 174 mm, showed increased longitudinal strain (+60%) following TEVAR. By contrast, Patient 2, with a longer stent-graft length of 235 mm, showed decreased longitudinal strain (-65%) in the descending aorta after TEVAR.
Figure 3. Pulsatile longitudinal strain before and after thoracic endovascular aortic repair (TEVAR) in Patient 1 with acute Type B aortic dissection (TBAD) (left panel) and Patient 2 with ruptured acute TBAD and Marfan syndrome (right panel). Pre- and post-TEVAR strain values are shown as percentages. Relative strain changes were computed as the percentage increase or decrease of post-TEVAR strain relative to pre-TEVAR strain.

Table 3. End-systolic diameter and circumferential strain.

<table>
<thead>
<tr>
<th>Location</th>
<th>STJ (mm)</th>
<th>BCT (mm)</th>
<th>LSA (mm)</th>
<th>LSA + 10 cm (mm)</th>
<th>LSA + 20 cm (mm)</th>
<th>CT (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Pre-TEVAR</td>
<td>33.6 ± 0.8</td>
<td>36.3 ± 0.4</td>
<td>30.2 ± 0.4</td>
<td>23.7 ± 0.3</td>
<td>22.8 ± 0.1</td>
<td>24.2 ± 0.3</td>
</tr>
<tr>
<td>Post-TEVAR</td>
<td>32.9 ± 0.8</td>
<td>32.2 ± 0.8</td>
<td>24.9 ± 0.6</td>
<td>Stent</td>
<td>Stent</td>
<td>21.6 ± 0.3</td>
</tr>
<tr>
<td>2 Pre-TEVAR</td>
<td>34.2 ± 0.7</td>
<td>36.6 ± 0.6</td>
<td>28.2 ± 0.5</td>
<td>28.0 ± 0.4</td>
<td>27.4 ± 0.2</td>
<td>27.0 ± 0.1</td>
</tr>
<tr>
<td>Control</td>
<td>34.3 ± 1.4</td>
<td>34.0 ± 0.9</td>
<td>31.4 ± 1.1</td>
<td>27.2 ± 1.1</td>
<td>26.5 ± 1.1</td>
<td>26.5 ± 1.0</td>
</tr>
<tr>
<td>3 Control</td>
<td>2.1</td>
<td>1.5</td>
<td>2.0</td>
<td>2.1</td>
<td>1.4</td>
<td>1.9</td>
</tr>
<tr>
<td>4 Control</td>
<td>2.2</td>
<td>1.6</td>
<td>Stent</td>
<td>2.8</td>
<td>4.4</td>
<td>6.2</td>
</tr>
</tbody>
</table>

Circumferential strain (%)

<table>
<thead>
<tr>
<th>Location</th>
<th>STJ</th>
<th>BCT</th>
<th>LSA</th>
<th>LSA + 10 cm</th>
<th>LSA + 20 cm</th>
<th>CT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Pre-TEVAR</td>
<td>2.4</td>
<td>1.5</td>
<td>1.9</td>
<td>2.8</td>
<td>4.4</td>
<td>6.2</td>
</tr>
<tr>
<td>Post-TEVAR</td>
<td>3.5</td>
<td>2.7</td>
<td>3.6</td>
<td>Stent</td>
<td>2.5</td>
<td>2.5</td>
</tr>
<tr>
<td>3 Control</td>
<td>3.3</td>
<td>2.1</td>
<td>1.8</td>
<td>1.7</td>
<td>2.2</td>
<td>2.0</td>
</tr>
<tr>
<td>4 Control</td>
<td>2.3</td>
<td>1.2</td>
<td>2.9</td>
<td>1.8</td>
<td>1.2</td>
<td>0.9</td>
</tr>
</tbody>
</table>

Values are shown as mean ± SD, where appropriate. BCT = brachiocephalic trunk; CT = celiac trunk; LSA = left subclavian artery; STJ = sinotubular junction; TEVAR = thoracic endovascular aortic repair.
Figure 4. Panels A and B. Pulsatile circumferential strain of (Panel A) Patient 1 with acute Type B aortic dissection (TBAD) and thrombosed false lumen and (Panel B) Patient 2 with acute ruptured TBAD, Marfan syndrome, and patent false lumen at the LSA + 20 cm and CT levels. Panel C. Comparison of pulsatile circumferential strain among all patients. BCT = brachiocephalic trunk; CT = celiac trunk; LSA = left subclavian artery; LSA + 10 cm = 10 cm distal to the LSA; LSA + 20 cm = 20 cm distal to the LSA; STJ = sinotubular junction.
**Intra- and Inter-Observer Variability**

For area changes, the intra-observer repeatability coefficient (RC) was 22.85 mm². Mean differences between pre- and post-TEVAR area changes were smaller than the RCs, and linear regression analysis was non-significant ($p = 0.31$). Inter-observer repeatability showed an RC of 17.88 mm², and mean differences between pre- and post-TEVAR area changes were smaller than the RCs. Linear regression analysis was non-significant ($p = 0.46$). These results indicate good intra- and inter-observer agreement.

For length changes, the RC for intra-observer repeatability was 2.78 mm, and mean differences between pre- and post-TEVAR length changes were smaller than the RCs. Linear regression analysis was non-significant ($p = 0.70$). The RC for inter-observer repeatability was 2.84 mm. Mean differences between pre- and post-TEVAR areas were smaller than the RCs, and linear regression analysis was non-significant ($p = 0.64$). Again, these results indicate good intra- and inter-observer agreement.

**Discussion**

In both TBAD patients, TEVAR was followed by elevated pulsatile longitudinal strain proximal to the stent-graft. Moreover, TBAD patients showed lower pulsatile longitudinal strain of the total thoracic aorta before TEVAR compared with control subjects. Regarding pulsatile circumferential strain, Patient 1, who presented with patent false lumen, showed higher strain of the true lumen when compared with Patient 2, who had a thrombosed false lumen. Pulsatile circumferential strain in control subjects ranged from 0.9% to 3.3% and was consistent along the thoracic aorta, which agrees with previous literature [22].

After TEVAR, we found increased pulsatile longitudinal wall strain proximal to the stent-grafts in both TBAD patients. Other authors suggest that increased pulsatile wall stress is associated with aortic wall fatigue, resulting in aneurysmal dilatation and increased risk of new entry tears at the proximal or distal end of the stent-graft or rupture [2–5]. Patients with fragile aortic walls, such as those with aortic dissection, are at particular risk for such sudden strain changes. In our study, Patient 2 developed aortic perforation due to a stent strut. In this patient, we observed a considerable increase in circumferential strain (1.9% vs. 3.6%) at the LSA after the initial TEVAR procedure. This elevated strain may have triggered the strut perforation. To better understand the pathophysiology of such complications, further studies are warranted to investigate pulsatile strain changes following TEVAR.

Pulsatile longitudinal strain was lower in TBAD patients than in older-aged control subjects (1.4–1.7% vs. 2.1–4.5%), even though aging is associated with aortic stiffening [8, 22]. This observation suggests that aortic stiffening is a risk factor for aortic dissection, although this remains to be confirmed by larger studies. Moreover, in Patient 2, we observed that stent-grafting of a longer segment was associated with a decrease in longitudinal strain, stiffening the descending aorta and potentially exerting adverse effects on cardiac function and geometry [10].

We noticed markedly high pulsatile circumferential strain at the level of patent false lumen in Patient 1. This was in contrast to the control subjects and Patient 2, who had a thrombosed false lumen. This observation emphasizes the potential role of variables such as dissection flap motion and thrombosis on prognosis. It also sheds light on differences between acute and chronic TBAD and underscores the importance of dynamic imaging to assess these risk factors and correctly size stent-grafts. Moreover, a substantial pulsatile circumferential strain may have implications for future device selection, as patients with more compliant aortas may benefit from more compliant stent-grafts to minimize mismatch.

Aortic stiffness is reportedly higher in Marfan patients [23]. We did not observe this in our study, most likely because our Marfan patient (Patient 2) presented with acute TBAD complicated by rupture. The movement of the dissection flap elevated the pulsatile circumferential strain before TEVAR. In addition, this patient was younger than the two control subjects, and younger age is associated with higher strain [22].

To continuously improve treatment outcomes of TBAD, it is important to obtain data on the biomechanical behavior of dissected aortas. In particular, promising advancements in computational fluid dynamics offer unique insights into complex vascular pathologies [8]. However, such computational techniques depend on clinically measured data to serve
as boundary conditions, which are only sparsely available for TBAD. Thus, this study provides preliminary data on dissection flap strain.

The main limitation of this study is the small cohort, as we were only able to include two TBAD patients and two control subjects. Therefore, our results cannot be generalized and should be considered descriptive. Moreover, we were unable to report circumferential strain within stented segments due to artifacts. More systematic studies are needed to elucidate the effects of aortic stent-grafts on the physiologic function of the aorta. Finally, with advancements in medical imaging, pixel spacing and the number of equidistant time steps during the cardiac cycle could be improved and should be further investigated.

In conclusion, this study represents an initial investigation on pulsatile aortic strain in patients with acute TBAD before and after TEVAR. We observed considerably higher pulsatile circumferential strain in a case of patent false lumen compared with thrombosed false lumen. After TEVAR, both TBAD patients showed increased pulsatile longitudinal strain proximal to the stent-graft, which is known to be associated with an increased risk of aortic dissection. These observations may motivate future research to assess the biomechanical impact of TEVAR in order to continuously improve treatment outcomes for patients with TBAD.

Acknowledgments

Prof. F. Auricchio and Dr. M. Conti acknowledge the support of Ministero dell’Istruzione, dell’Università e della Ricerca (No. 2010BFXRHS), ERC Starting Grant through the Project ISOBIO: Isogeometric Methods for Biomechanics (No. 259229), and the iCardiocloud project by Cariplo Foundation (No. 2013-1779) and Lumbarly Region (No. 42938382; No. 46554874).

Conflict of Interest

The authors have no conflicts of interest relevant to this publication.

References

EDITOR’S COMMENT

The provocative preliminary observations by Foeke and colleagues should stimulate our attention. We tend to focus on the stented zone after TEVAR, with perhaps less than optimal consideration for the non-stented zone. These investigators, in a very preliminary study, show that having a descending stent graft substantially increases the stretch of the ascending or arch zones with each heartbeat. They speculate, plausibly, that this increased stretch may be harmful and may contribute to early and late complications in the aorta outside the stent zone. This may be especially pertinent vis-a-vis the troublesome, and often lethal, occurrence of retrograde ascending aortic dissection following descending aortic stent grafting. While the number of patients studied is very small, the authors describe the technical methods of analysis clearly; hopefully, their posing these important questions and laying the requisite computational methods will stimulate more extensive studies of this important topic.
A Rare and Late Complication After Left Ventricular Assist Device Explantation

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2 Department of Cardiothoracic Transplantation and Mechanical Circulatory Support, Harefield Hospital, Royal Brompton and Harefield NHS Foundation Trust, Harefield, Middlesex, United Kingdom

Abstract
Late complications can arise after explantation of a left ventricular assist device. We report the case of a patient who presented at the age of 19 years with peri-partum cardiomyopathy and was initially managed with a biventricular support device, which was subsequently upgraded to an ambulatory left ventricular assist device. This was successfully explanted after myocardial recovery via a minimally invasive approach 7 months later. The patient re-presented 5 years following explantation with hemoptysis. At redo sternotomy, a 10-cm remnant of the outflow graft was found to be eroding the surface of the right lung. The conduit was excised and the stump oversewn. Eleven months later, she presented again with hemoptysis, and a pseudo-aneurysm was identified on the computed tomography scan. She underwent urgent open repair with peripheral cannulation and deep hypothermic total circulatory arrest. The Dacron stump was found to be partially dehisced, leaving a 2–3 cm defect in the ascending aorta. The defect was repaired with a bovine pericardial patch. The patient made a good recovery and was successfully discharged.

Introduction
Our center has previously reported successful explantation of left ventricular assist devices (LVADs) from patients who have been ‘bridged-to-recovery’ [1]. Here, we report a late complication of LVAD explantation in a woman who had been successfully bridged to recovery after developing a peri-partum cardiomyopathy.

Case Presentation
The patient first presented to our center in October 2009 at the age of 19 with peri-partum cardiomyopathy. She had severe globally impaired left ventricular function, with left ventricular ejection fraction (LVEF) estimated at 15%. Supportive measures, including intra-aortic balloon pump, maximal anti-heart failure medication, and steroids proved inadequate. She remained ventilator-dependent and, approximately 2 months after her initial presentation, biventricular support was instituted with a Levitronix system (Levitronix®). After another 2 months, the right-sided support was successfully weaned, and the left ventricular support was upgraded to an ambulatory HeartMate II device (Thoratec®). One month later, the patient was successfully weaned from the ventilator and discharged home. In September 2010, her left ventricular function had recovered (LVEF = 67%), and...
she was admitted for explantation of the HeartMate device using the limited combined right anterior and left anterolateral thoracotomy approach previously described by our center [2]. The procedure was accomplished uneventfully, but due to dense adhesions, a length of the Dacron aortic outflow graft was not retrieved. The patient was discharged and continued cardiac rehabilitation with stable LVEF of 50–60% on follow-up echocardiography.

In January 2015, the patient presented to the emergency room with frank hemoptysis (estimated at around 400 mL). Computed tomography (CT)-pulmonary angiography demonstrated an out-pouching (2.1 × 1.7 cm) arising from the ascending aorta, which was surrounded by soft tissue density consistent with a pseudo-aneurysm (Figure 1A). Furthermore, a large opacity surrounded by ground glass changes was noted in the right upper pulmonary lobe. However, comparison of the mediastinal mass with previous images from a 2011 CT scan showed no significant changes in the appearance of the soft tissue around the ascending aorta (Figure 1B). Bronchoscopy showed no evidence of active bleeding from the right upper lobe, but a large amount of old blood was noted in the right lower lobe. The patient was taken to theater, and a 10-cm length of Dacron tubing was identified as arising from the ascending aorta, representing the old outflow graft of the LVAD that was not retrieved when the device was explanted. Although there was no active bleeding from the graft, its tied-off distal end abutted against and had slowly eroded the right upper lobe surface, causing hemoptysis. The graft was mobilized and excised with oversewing of the stump arising from the

Figure 1. Panels A and B. Computed tomography (CT) scans showing the outflow graft stump one year after ventricular assist device explantation (Panel A) and before the redo sternotomy in January 2015 (Panel B). Panels C and D. CT scans in December 2015 showing the pseudo-aneurysm increase within two days.
ascending aorta. A wedge of the chronically inflamed right upper lobe was also excised.

The patient was discharged and remained well on follow-up until July 2015, when a deep sternal wound infection became apparent. Sternal wires were removed, and a vacuum-assisted closure (VAC) dressing was inserted in November 2015. One week after the VAC dressing removal in December 2015, she again presented to the emergency room with recurrent hemoptysis. A CT scan showed a clear pseudo-aneurysm of the ascending aorta (Figure 1C). A repeat CT scan 2 days later showed the aneurysm to be enlarging (Figure 1D). The patient was therefore scheduled for urgent open repair. Cardiopulmonary bypass was instituted via right femoral venous and right axillary arterial cannulation. The apex of the left ventricle was vented via the old left anterior thoracotomy, and the patient was core-cooled to 18°C. The sternum was divided and the pseudo-aneurysm entered. The dehiscence of the remaining graft stump was noted, which created a 2–3-cm defect in the ascending aorta (Figure 2). During a 31-min period of total circulatory arrest, the defect was repaired with a patch of bovine pericardium using pledgeted interrupted 3/0 Prolene mattress sutures. The patient recovered well but required temporary renal dialysis.

**Discussion**

No clear consensus exists regarding the surgical approach for explanting ventricular assist devices. Although a minimally invasive approach should, at least in theory, be beneficial for the patient and the recovered myocardium, there is always a risk of leaving prosthetic material behind due to dense adhesions. The unusual presentation in this case was caused by an erosion of the lung surface caused by the residual stump of the outflow graft. Although previously reported after LVAD explantation, pseudo-aneurysms of the ascending aorta are rare, and the cause is usually infectious [3]. However, in this case, all intra-operative samples collected after repair of the pseudo-aneurysm were negative for bacteria or fungi. Thus, the cause of the dehiscence of the outflow graft stump and subsequent pseudo-aneurysm formation remains unclear.

Endovascular stenting has been reported as an option in similar cases [4] but requires custom-designed endografts. In our case, given the urgency and unsuitable anatomy, open repair was preferred. Furthermore, the relatively small size of the aortic defect and the absence of clinical features of infection mandated a simple patch repair as opposed to replacement of the entire ascending aorta. Thus, our clear preference in complex redo surgery for pseudo-aneurysms is peripheral arterial and venous cannulation with left ventricular apex venting through a limited left anterior thoracotomy and circulatory arrest during sternotomy and initial dissection of the heart.

**Conflict of Interest**

The authors have no conflicts of interest relevant to this publication.

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Figure 2. Intraoperative image showing the dehisced graft stump and defect in the ascending aorta.
References


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Aorto-Cutaneous Fistula and False Aneurysm of the Ascending Aorta Five Years after its Prosthetic Replacement for Stanford Type A Acute Aortic Dissection

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Department of Thoracic and Cardiovascular Surgery, Pierre et Marie Curie University, Assistance Publique Hôpitaux de Paris, La Pitié Salpêtrière Hospital, Paris, France

† These authors contributed equally to this work

Abstract

Aorto-cutaneous fistula and false aneurysm of the ascending aorta in patients who previously underwent Stanford Type A acute aortic dissection are rare and severe complications. Surgical correction remains a demanding challenge. In a case of false aneurysm rupture during redo sternotomy, selective cannulation of the right axillary and left carotid arteries allowed an efficient method of cerebral perfusion.

Key Words:
Aortic dissection • Pseudo-aneurysm • Aorto-cutaneous fistula • Cerebral protection

Introduction

False aneurysm (FA) occurring after replacement of the ascending aorta by a vascular prosthesis, which is defined as total or partial dehiscence of the prosthesis from the aortic wall, is a rare complication of this type of surgery. As with other aneurysms, it is notable for a marked increase in diameter and the occurrence of life-threatening complications such as rupture and fistula formation [1]. Despite advances in endovascular techniques, treatment in the majority of cases remains surgical. The procedure is complex, has a high mortality [2, 3], and represents a surgical challenge. Aorto-cutaneous fistula and FA of the ascending aorta is even more exceptional and challenging. The purpose of this case report is to describe our surgical strategy for aorto-cutaneous fistula associated with FA of the ascending aorta 5 years after its prosthetic replacement for Stanford Type A acute aortic dissection.

Case Presentation

An 82-year-old woman underwent ascending aortic replacement in 2010 for a Stanford Type A acute aortic dissection. She remained well for 5 years before exhibiting fresh, oozing blood from the upper part of the sternum (Figure 1). Computed chest tomography revealed anterior and posterior extensions of a FA of the ascending aorta with a communication with the skin through the sternum (Figures 2, 3, and 4).

Due to the risk of aortic rupture during sternotomy and the aortic dissection history, our surgical approach consisted of cannulating the right axillary and left carotid arteries through right axillary and left stern-
cleidomastoid cervical incisions by cardiopulmonary bypass (CPB) and cerebral protection for instituting hypothermic partial circulatory arrest before reentry into the chest. Cerebral protection was monitored with near-infrared spectroscopy. CPB and cooling were instituted before skin incision and stopped when the FA rupture occurred during the sternotomy. Cerebral protection was achieved by cooling the patient to 24°C, occluding the proximal left carotid and brachiocephalic arteries, and reducing bypass flow transiently to 800 mL/min. Mediastinal dissection and distal anastomosis between the hemi-arch and Dacron graft (Gelweave Vascutek Terumo, 32 mm) for hemi-arch replacement were performed under partial hypothermic circulatory arrest. The total duration of partial hypothermic circulatory arrest was 41 min. The distal Dacron graft was then cross-clamped, and normothermic full flow cardiopulmonary bypass (3900 mL/min) was restarted through the right axillary artery. Redo aortic root replacement (modified Bentall procedure) was performed using a biologic aortic valve (Trifecta, St. Jude Medical, 25 mm) and Dacron graft (Gelweave Vascutek Terumo, 28 mm). Finally, graft-to-graft anastomosis was performed. Distal and proximal graft anastomoses were performed using 4.0 monofilament running sutures at the aortic wall juncture and interrupted sutures at the annular level. Teflon reinforcement was used. Cross clamp duration was 80 min, and the total duration of CPB was 200 min. No intraoperative finding alluding to the etiology of the FA was identified. A diagnosis of prosthetic infection was excluded microbiologically. After debridement of the aorto-cutaneous fistula area, the sternum was rewired and the skin closed. The patient had an uncomplicated post-operative course and was transferred for cardiac rehabilitation 10 days after surgery.

Discussion
FA is a rare but serious and complex complication of ascending aorta and/or aortic root prosthetic replacement. Its incidence, risk factors, and natural history are unknown. Bachet et al. [4] reported three FAs in 143 patients initially undergoing operation for acute dissection of the ascending aorta with prosthetic replacement over a 10-year period. Also, Mohammadi et al. published the Pitie Salpetriere experience regarding 29 operations for FAs of the ascending aorta after prosthetic replacement, demonstrating that a well-planned operative strategy guided by preoperative imaging results in low operative mortality [5].

Figure 3. Computed tomography angiography reconstruction of the chest showing the anterior and posterior extensions (arrows) of false aneurysm of the ascending aorta after its prosthetic replacement.

In conclusion, selective cannulation of the right axillary and left carotid arteries allowed an efficient method of cerebral perfusion and protection in this case of FA rupture during sternotomy. A well-planned operative strategy guided by preoperative imaging permitted careful analysis of the lesions, resulting in a good outcome for our patient.

Conflict of Interest

The authors have no conflicts of interest relevant to this publication.
References


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Complete Shrinkage of the Obliterated False Lumen After Open and Endovascular Chronic Aortic Dissection Stanford Type A Repair

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Abstract

A 48-year-old man was admitted to our hospital with chronic aortic dissection Stanford Type A. His diagnosis was confirmed by chest multi-detector computed tomography (CT). The patient underwent combined (i.e., hybrid) open and endovascular repair (frozen elephant trunk) in a one-stage operation with moderate hypothermic circulatory arrest and antegrade cerebral perfusion. His postoperative course was uneventful, and he was discharged home on postoperative day 9. At 2-year follow-up, chest CT angiography revealed complete shrinkage of the obliterated false lumen in the distal aortic arch and descending thoracic aorta.

Key Words:
Aortic dissection • Chronic aortic dissection • Hypothermic circulatory arrest

We present a case of a 48-year-old man who underwent elective combined (i.e., hybrid) open and endovascular repair of chronic aortic dissection Type A (frozen elephant trunk with endovascular stent) in a one-stage operation with moderate hypothermic circulatory arrest and antegrade cerebral perfusion (ACP). His past medical history included a 7-month-old known aortic dissection Type A, hypertension, and smoking. Preoperative transthoracic echocardiography showed left ventricular ejection fraction of 58% with dissection of the ascending aorta above the sinotubular junction extending to the aortic arch. Preoperative computed tomography (CT) angiography of the aorta revealed aortic dissection of the ascending aorta extending to the descending thoracic and abdominal aorta (Figures 1IA, 1IB, 1IC, 1ID and Figure 2A). The dimensions of the aortic root (sinus of Valsalva level), ascending aorta, aortic arch, and descending aorta were 40 mm, 50 mm, 52 mm, and 40 mm, respectively. Aortic arch vessels and all abdominal branches originated from the true lumen.

Replacement of the thoracic aorta was performed under moderate hypothermic circulatory arrest and ACP. Cardiopulmonary bypass (CPB) was established via a graft to the right axillary artery and right atrium. Cardioplegic arrest was achieved using retrograde cold Custodiol solution. When bladder temperature reached 22.5°C and circulatory arrest was established, ACP was started via the right axillary artery and left common carotid artery. The ascending aorta, aortic arch, and proximal descending aorta were resected, and debranching of the aortic arch vessels was performed.

A 24-mm endovascular stent with a 50-mm length graft and 130-mm stent graft (E-vita OPEN PLUS graft, JOTEC, Schweiz, Germany) was implanted in the proximal descending aorta. Arch vessels were selectively implanted on the graft. The proximal ascending aorta and the rest of the aortic arch graft was sutured, and circulation was reestablished. The patient was weaned from...
Figure 1. Computed tomography (CT) angiography depicting the evolution of thoracic aorta remodeling during the follow-up period at four different levels (Panel A. axial cut pulmonary level, Panel B. axial cut distal graft level, Panel C. coronal view distal arch level, Panel D. sagittal view pulmonary level and distal graft level). Panel I. Preoperatively. Panel II. Four days after the operation. Panel III. Two years after the operation.

Figure 2. CT angiography showing evolution during the follow-up period. Panel A. Preoperatively. Panel B. Four days after the operation. Panel C. Two years after the operation.
CPB and transferred to the intensive care unit. Circulatory arrest, aortic cross clamp, and CPB durations were 93 min, 146 min, and 292 min, respectively. Four days after the operation, CT angiography revealed thrombosis of the false lumen in the descending thoracic aorta (Figures 1IIA, 1IIB, 1IIC, and 1IIDD). The patient’s postoperative course was uneventful, and he was discharged home on postoperative day 9. At 24-month follow-up, CT angiography of the aorta showed complete obliteration and shrinkage of the false lumen of the descending thoracic aorta (Figures 1IIIA, 1IIIB, 1IIIC, and 1IIID) and thrombosis of the false lumen of the abdominal aorta (Figures 2B and 2C).

The descending thoracic aorta is amenable to remodeling by extending the stent graft into the true lumen with subsequent obliteration of the false lumen. The latter procedure is achieved at all levels of the stent graft. A similar procedure has been considered successful when complete thrombosis of the false lumen is achieved at 3-month follow-up, both in cases of acute and chronic Stanford Type A aortic dissection [1–3]. Gorlitzer et al. [1] described shrinkage of the false lumen in 64% of patients who underwent this treatment modality across a follow-up period of 12 months. In our case, complete shrinkage of the false lumen was present at 24-month follow-up.

**Conflict of Interest**

The authors have no conflict of interest relevant to this publication.

**References**


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Samanidis, G. et al. Endovascular Repair of Chronic Aortic Dissection
Abstract

Iatrogenic simultaneous inferior vena cava (IVC) and iliac vessel injury is a rare entity. Ligation of the IVC in a life-threatening situation is well reported in the literature. Our case demonstrates that such a clinical situation requires optimization of fluid volume and management of sequelae such as deep vein thrombosis.

Key Words:
Inferior vena cava injury • Psoas abscess • Deep vein thrombosis

Introduction

Combined inferior vena cava (IVC) and iliac vessel injury after interventional procedures is a rare entity. Ligation of the IVC in a life-threatening situation is well reported in the literature. Our case demonstrates that such a clinical situation requires replacement of fluid volume and management of sequelae such as deep vein thrombosis (DVT).

Case Presentation

A 50-year-old man presented to the emergency room with a history of insertion of a 16 F pigtail catheter for drainage of psoas abscess in a peripheral hospital (Figure 2). The patient had a gush of blood from the catheter, for which he underwent a computed tomography scan (Figure 1) in the referring hospital, which was suggestive of the catheter piercing the IVC. In our hospital, a catheter angiogram was advised, which revealed a leak from the sideholes of the catheter into the right common iliac artery. To delineate the exact location of the catheter, the patient underwent a conventional angiogram through left groin access. The catheter was confirmed to have traversed the IVC and passed through the right common iliac artery, with the tip of the catheter communicating with the retroperitoneal free space.

For this iatrogenic injury of major vascular structures, with the catheter in situ, we decided to proceed with open surgery. The patient was clinically stable with palpable distal pulses. Under general anesthesia, a midline abdominal incision was made and infra-renal abdominal aorta control achieved. Right colon mobilization was performed to track the pathway of the pigtail catheter. The catheter was found to have crossed the psoas abscess cavity and to have pierced the confluence of the IVC and right common iliac artery (Figure 3). The catheter was cut between the aorta and IVC and removed with repair of the right common iliac artery with a 4-0 prolene suture. While mobilizing the catheter from the IVC, the rent extended into the left common iliac vein. Despite applying proximal and distal venous compression followed by Satinsky clamps, there was further extension of the injury with massive exsanguination. In a life-saving
approach, the IVC was plicated at its confluence with the iliac vein. Hemostasis was achieved. Drains were placed in the psoas abscess cavity and peritoneum. The abdominal cavity was closed. The patient was managed with inotropic support and transfusion of blood and blood products in the perioperative period. He was administered unfractionated heparin considering the high probability of DVT.

The patient developed swelling of the right lower limb, for which intermittent pneumatic compression was performed and a crepe bandage applied. Both lower limb pulses were palpable. Unfractionated heparin was initiated for suspected DVT, which was confirmed by Duplex scan of the venous system. The patient had an uneventful post-operative course and was discharged on oral anticoagulation for 3 months. He was also advised to wear elastic compression stockings for 3 months. When the patient was assessed in the follow-up clinic, he exhibited normal gait and contour of both lower limbs.

Discussion

The earliest reported IVC ligations date back to Kocher in 1883 and Billroth in 1885 [1]. The first survivors of vena caval transection were reported by Bottini for infrarenal and Detrie for suprarenal ligation [1]. Yet, documented cases of caval ligation are rare. Ivy et al. described 23 case reports of infrarenal caval ligation and six case reports of suprarenal ligation in addition to their patient [2]. Also, a literature search revealed trauma or IVC tumor resection as
the antecedent cause of IVC ligation [3–7]. Whereas infrarenal caval ligation has been performed more frequently, there is reluctance among surgeons to ligate the suprarenal cava [8–11].

Hypotension and renal failure following ligation of an infrarenal cava has been documented by Gazzaniga et al. [1]. If this acute dramatic problem can be controlled, collateral circulation soon develops via the retroperitoneal and vertebral plexuses, ascending lumbar veins, and paravertebral veins, which drain into the azygos and hemiazygos systems. Testicular and ovarian veins may also contribute as accessory pathways [1].

The left renal vein and collaterals are the principal draining channels following suprarenal ligation of the IVC. The left adrenal vein and left spermatic/ovarian vein normally join the left renal vein. In addition, the left renal vein has lumbar and hemiazygos connections in 70% of patients [12]. Lumbar, ascending lumbar, and vertebral veins also contribute to collateral drainage. In 35% of patients, transient edema of the extremities develops, which becomes permanent in 2% of patients [13]. Abdominal compartment syndrome with extensive pedal edema following suprarenal ligation of the IVC has also been reported [2]. Our patient had edema of both lower limbs lasting 1 month. Doppler ultrasound and strain gauge plethysmography have been used to study the venous pattern following caval ligation [14]. Transient defects in renal function manifesting as oliguria/anuria and hematuria with rising creatinine levels have also been observed following caval ligation [2, 9, 10].

The pre-hospital mortality rate for IVC injuries is 36%, and among those who reach the hospital alive, mortality ranges from 21 to 57% [1]. Mortality also varies with the level of IVC injury. Trauma to the infrarenal cava is associated with a mortality of 25%, whereas injuries between the renal and hepatic veins carry a mortality of 41–55%. The mortality rates for caval injuries at or above the level of the hepatic veins exceed 80% [2]. Mortality following IVC injury is attributed to persistent hemorrhage and associated hypotension, hypothermia, coagulopathy, and acidosis [2]. Navsaria et al. retrospectively evaluated risk factors by comparing survivors and non-survivors after IVC injuries. They found that the site of injury and type of surgical management (ligation vs. repair) were not predictors of survival [7]. Thus, it may be concluded that the mortality associated with IVC injuries is related to the associated trauma and surrounding organ injury.

Elective ligation in a controlled setting allows collaterals to develop and results in a good outcome. When IVC ligation is performed, intensive monitoring to correct hypovolemic shock and possible abdominal compartment syndrome is required, particularly when the ligation is performed in the acute setting. In the context of trauma, with caval injuries, ligation of the IVC has been suggested as an acceptable and life-saving option [7].

In conclusion, management of IVC injury has long been a challenge. The key to management of IVC injury lies in the decision for repair or ligation. Repair should be given preference when possible. In an unavoidable circumstance, timely ligation of the IVC is rewarding and compatible with life.

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Conflict of Interest

The authors have no conflicts of interest relevant to this publication.

References

EDITOR’S COMMENTS

Initially, one may wonder why a venous paper is being included in AORTA. As Dr. Stansel taught years ago, iatrogenic operative venous injuries, incurred during arterial surgery, can be much more difficult to control and pose a greater risk to life than arterial injuries. I was not aware that inferior vena caval ligation was tolerable. Perhaps other readers will benefit from knowing that this option is available when dealing with massive hemorrhage from an iliac vein or the vena cava itself, at the infra- or supra-renal levels.
The following pages summarize and review this issue’s articles for an audience without a background in medicine or research.

**Original Research Articles**

McClure RS et al.: “Cause of Death Following Surgery for Acute Type A Dissection: Evidence from the Canadian Thoracic Aortic Collaborative”

In acute Type A aortic dissection, a tear occurs in the wall of the body’s main artery, the aorta. Emergent surgery offers by far the best chances for survival. However, even after surgery mortality is high. The authors of the article determined causes of death in 123 patients who died following surgical repair of acute Type A dissection. They identified heart failure, stroke (bleeding or lack of perfusion of vessels in the brain) and bleeding as major causes of death. Aortic dissection can impair perfusion of organs such as the brain, kidneys, limbs or bowels before surgery, which is a major risk factor for complications and death after surgery. New surgical techniques should address these major causes of death after surgery for in acute Type A dissection.

Foeke J.H. Nauta et al.: “Impact of Thoracic Endovascular Repair in Acute Type B Aortic Dissection: Preliminary Results”

In aortic dissection, a tear occurs within the wall of the aorta, the body’s main vessel. In Type B dissection, this occurs in the part of the vessel in the chest that leads downwards to the abdomen. It is often treated with a stent graft, a prosthesis that is inserted into the vessel to stabilize it. However, dissection and dilation of the aorta in the segment which is not stabilized can be a potential complication. Foeke et al. conducted a study to see if this might be related to the stretching of the vessel wall as the heart pumps blood through it and its impairment by the insertion of a stiff stent graft. By analyzing images of the aorta in 4 patients, they could show that after insertion of the stent graft, the unstented part of the aorta was subject to more stretching than before. They concluded that this might be causative of ruptures and tears in this segment. However, the study group is extremely small and the evidence only indirect, therefore this study can only be regarded as a pilot to guide further research.

**Case Reports**

George Gradinariu et al.: “A Rare and Late Complication after Left Ventricular Assist 1 Device Explication”

The authors present a case of a patient who developed severe impairment of her heart’s function after giving birth. Her heart was temporarily supported with a mechanical assist device, which could later be successfully explanted. The assist device pumps blood through a graft prosthesis into the aorta, the body’s main artery. A part of this graft was left in place after explantation and started leaking, causing the patient to cough blood. She underwent ur-
gent surgery and the leak-ing aor-ta was repaired with a patch. The patient recovered without further complications.

Pierre Demondion et al.: “Aor-to-Cutaneous Fistula and False An-eyrusm of the Ascending Aorta Five Years After Its Prosthetic Replace-ment for Stanford Type A Acute Aor-tic Dissection”

A rare complication of a repair or replacement of the aorta, the bod-ies main artery, is a leakage from the suture line between aorta and prosthesis. The leakage leads to the creation of a blood-filled space called “pseudo-aneurysm” which can cave its way through the tissues e.g. even through the skin (“called aorto-cutaneous fistula”). This complication is life-threaten-ing, and the surgical repair associ-ated with high mortality. Surgery in these cases is challenging, espe-cially because the false aneurysm can open up and bleed out when the chest is opened. To ensure continuous perfusion of the body and brain, the safest option is to connect the patient to the heart-lung machine via vessels in the axilla and/or neck before opening the chest. The authors describe a case of a 82-year-old patient who developed this complication, but recovered well from redo surgery.

Images in Aortic Disease

George Samanidis et al.: “Com-plete Shrinkage of the Obliterated False Lumen After Open and Endo-vascular Chronic Aortic Dissection Stanford Type A Repair”

The authors describe a case of the patient who had a Type A aor-tic dissection, a tear in the wall of the body’s main vessel. Blood was flowing through a false channel within the wall of the aorta. Blood was flowing through a false channel within the wall of the aorta. He underwent surgery, in which a part of the vessel was replaced and another part of the vessel stabilized with a stent graft prosthesis, a graft that is inserted into the vessel without replacing it. Since only a part of the aorta is replaced, in many cases, the false channel remains open and perfused in the parts of the aorta that are not replaced. This can cause dilation and rupture during follow-up. In this patient, imaging 2 years after surgery showed that the entire false channel was closed and had shrunk.

What I Did

Sandeep Mahapatra et al.: “Man-agement of Iatrogenic Injury to Infe-rior Vena Cava and Right Common Iliac Artery for Drainage of Psoas Abscess”

Mahapatra et al. describe a case of a patient who had an infection in a muscle in his lower trunk. The infection was treated with a drain-age, but during this procedure, two major vessels in the groin and abdomen were injured, causing life threatening bleeding. During surgery, the inferior vena cava, the main vessel that returns blood from the body back the heart, had to be closed off with a suture to stop the bleeding and save the pa-tient’s life. Closing the vena cava impairs the flow of blood back to the heart, but the body can potentially use other vessels instead. However, the impairment of venous blood flow can cause swell-ing e.g. of the lower limbs and thrombosis, but with appropriate management, the patient in the presented case recovered well.
Upcoming Meetings

AORTA, April 2017, Volume 5, Issue 2:70
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List of Upcoming Meetings

May 2017

1. 2017 Vascular Research Initiatives Conference
   Vascular Research Initiatives Conference
   Minneapolis, Minnesota
   May 3, 2017
   Minneapolis, Minnesota, USA
   Meeting information available at:
   www.vascular.org/meetings/2017-vascular-research-initiatives-conference

2. Southwest Valve Summit 2017
   May 5-7, 2017
   San Antonio, TX, United States
   Meeting information available at:
   events.houstonmethodist.org/swvalve

3. 66th International Congress of the European Society for Cardiovascular and Endovascular Surgery (ESCVS)
   May 11-14, 2017
   Thessaloniki, Greece
   Meeting information available at:
   www.escvs2017.org

4. Critical Issues in Aortic Endografting 2017
   May 19-20, 2017
   Nuremberg, Germany
   Meeting information available at:
   www.critical-issues-congress.com

5. 2017 Vascular Annual Meeting
   May 31-Jun. 3, 2017
   San Diego, California
   Meeting information available at:
   www.vascular.org/meetings/2017-vascular-annual-meeting

June 2017

1. Multidisciplinary European Endovascular Therapy Congress 2017
   June 1-2, 2017
   Nice, France
   Meeting information available at:
   www.meetcongress.com

2. Magna Græcia AORTic Interventional Project® (MAORI) 5th Symposium Complex Diseases of Thoracic and Thoraco-Abdominal Aorta
   June 20-21, 2017
   Catanzaro, Italy
   Meeting information available at:
   www.maori.unicz.it

3. Multidisciplinary Aortic Dissection (MAD) Symposium
   June 23-24, 2017
   Portland, Oregon
   Meeting information available at:
   www.vascular.org/meetings/multidisciplinary-aortic-dissection-mad-symposium

4. 2017 Aortic Valve Repair Summit
   June 23-25, 2017
   Ottawa, Ontario, Canada
   Meeting information available at:
   www.valvesymposium.com

5. Liverpool Aortic Symposium VII
   June 30-July 1, 2017
   Liverpool, United Kingdom
   Meeting information available at:
   www.aorticaneurysm.org.uk