Conform to the new standard for Left Atrial Appendage (LAA) Occlusion.

The TIGERPAW® System II with its unique Fastener technology is designed with soft silicone housing to minimize risk and damage to the friable LAA. Once implanted, the Fastener conforms to the shape and thickness of the patient’s appendage, resulting in 100% clinically proven occlusion.¹

- Easy and rapid application (60 seconds or less)¹
- Conforms to variable LAA size and thickness with pliable silicone housing
- Zero blood loss at device footprint

MAQUET — The Gold Standard.


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\(^{1}\) US IDE Trial. Endurant 2011 Clinical Update.
\(^{2}\) Data on file at Medtronic.
\(^{3}\) BOXI data as of March 16, 2012.
**Indications**
The Endurant® II Stent Graft System is indicated for the endovascular treatment of infrarenal abdominal aortic or aorto-iliac aneurysms in patients with the following characteristics:
- Adequate iliac/femoral access that is compatible with vascular access techniques, devices and/or accessories
- Proximal neck length of ≥ 10 mm
- Infrarenal neck angulation of ≤ 60°
- Distal fixation length of ≥ 15 mm
- Aortic neck diameters with a range of 19 to 32 mm
- Iliac diameters with a range of 8 to 25 mm
- Morphology suitable for aneurysm repair

**Contraindications**
The Endurant II Stent Graft System is contraindicated in:
- Patients who have a condition that threatens to infect the graft.
- Patients with sensitivities or allergies to the device materials.

**Warnings and Precautions**
- The long-term safety and effectiveness of the Endurant II Stent Graft System has not been established. All patients should be advised that endovascular treatment requires lifelong, regular follow-up to assess the health and the performance of the implanted endovascular stent graft. Patients with specific clinical findings (e.g., endoleaks, enlarging aneurysms or changes in the structure or position of the endovascular graft) should receive enhanced follow-up. Specific follow-up guidelines are described in the Instructions for Use.
- Patients experiencing reduced blood flow through the graft limb, aneurysm expansion, and persistent endoleaks may be required to undergo secondary interventions or surgical procedures.
- The Endurant II Stent Graft System is not recommended in patients unable to undergo or who will not be compliant with the necessary preoperative and postoperative imaging and implantation studies as described in the Instructions for Use.
- Renal complications may occur: 1) From an excess use of contrast agents. 2) As a result of emboli or a misplaced stent graft. The radiopaque marker along the edge of the stent graft should be aligned immediately below the lower-most renal arterial origin.
- Studies indicate that the danger of micro-embolization increases with increased duration of the procedure.
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Please reference product Instructions for Use for more information regarding indications, warnings, precautions, contraindications and adverse events.

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Abstract

**Background:** Seasonal variations of Stanford Type A dissections (STADs) have been previously described in the Northern Hemisphere (NH). This study sought to determine if these variations are mirrored in the Southern Hemisphere (SH).

**Methods:** Data from patients treated surgically for STADs were retrospectively obtained from existing administrative and clinical databases from NH and SH sites. Data points of interest included age, sex, date of dissection, and 30-day mortality. The dates of dissections (independent of year) were then organized by season.

**Results:** A total of 1418 patients were identified (729 NH and 689 SH) with complete data available for 1415; 896 patients were male with a mean age of 61 ± 14 years, and the overall 30-day mortality was 17.3%. Comparison of NH and SH on a month-to-month basis demonstrated a 6-month phase shift and a significant difference by season, with STADs occurring predominantly in the winter and least in the summer. Decomposition of the monthly incidence using Fourier analysis revealed the phase shift of the primary harmonic to be –21.9 and 169.8 degrees (days), respectively, for NH and SH. The resultant 191.7 day difference did not exactly correspond to the anticipated 6-month difference but was compatible with the original hypothesis.

**Conclusion:** Chronobiology plays a role in the occurrence of STADs with the highest occurrence in the winter months independent of the hemisphere. Season is not the predominant reason why aortas dissect, but for patients at risk, the increase in systemic vascular resistance during the winter months may account for the seasonal variations seen.

**Key Words**

Aortic dissection • Chronobiology

**Introduction**

There is a growing body of evidence to suggest that cardiovascular events display both a seasonal and circadian variation in occurrence. For acute Stanford Type A aortic dissections (STADs), it is widely
observed and appreciated that the winter months tend to be the time when these dissections most often occur [1-3], possibly due to the cold weather having an impact on the systemic vascular resistance [4]. Other factors occur during these same months, which might also have an impact including the increased “stress” of holidays and year-end business events [5]. Interestingly, all studies (including the International Registry of Acute Aortic Dissection, IRAD) have relied on patient data from Northern Hemisphere (NH) sites. We were interested in whether or not Southern Hemisphere (SH) occurrences would mirror those of the NH, thereby lending more credence to the weather theory rather than to the time of year. Our hypothesis was that the season would have more of an impact than the time of year.

**Methods**

Institutional Review Board exemption for de-identified patient outcome data analysis was obtained, and the requirement for written informed consent was waived. Patient information was retrospectively collected from six sites (Table 1) with 729 patients in the NH and 689 patients in the SH for a total of 1418 patients; three patients were excluded from the analysis because of missing data. Specific data points queried were age, sex, date of operation, and 30-day mortality. Patients were assigned a hemispheric location based on study site and results were aggregated based on hemisphere. An assumption was made that the date of operation was associated with the date of clinical presentation; that date was then converted to a season with the seasons in the NH being defined as Winter (December 21–March 20), Spring (March 21–June 20), Summer (June 21–September 20), and Fall (September 21–December 20). SH seasons were offset by 6 months. Nonparametric comparisons were performed using the Pearson Chi-square test (SPSS Version 22, IBM, Armonk, New York, USA) and temporal variation analyzed with Fourier analysis (Matlab R2010B, Mathworks, Natick, Massachusetts, USA).

**Results**

A total of 1418 patients were identified (729 NH and 689 SH) with complete data available for 1415; 896 patients were male (63%) with a mean age was 61 ± 14 years (range 18–92), and the overall 30-day mortality was 17.3%. Comparison of NH and SH on a month-to-month basis demonstrated a 6-month phase shift (Pearson-χ² 26.7, p=0.005) and a significant difference by season, with STADs occurring predominantly in the winter and least in the summer (Pearson-χ² 11.6, p=0.009) (Figure 1). Decomposition of the monthly incidence using Fourier analysis revealed the phase shift of the primary harmonic to be –21.9 and 169.8 degrees (days), respectively, for NH and SH. This corresponds to peak incidences in the NH in early December and in the SH in late May. The resultant 191.7-day difference did not exactly correspond to the anticipated 6-month difference but was compatible with the original hypothesis. All dissections (both hemispheres) were combined with the day of year calculated with respect to the associated winter solstice. Using this combined data set, when harmonic analysis was performed the phase shift of the primary harmonic occurred 20 days prior to the winter solstice. Thus the actual peak event occurs prior to the calendar winter season (i.e., late November in the NH) but the seasonal variation persists. Using the peak as the actual starting point and distributing

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<td>2000–2012</td>
</tr>
<tr>
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<td>Australia/New Zealand</td>
<td>Smith</td>
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</tr>
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<td>Buenos Aires</td>
<td>Argentina</td>
<td>Navia</td>
<td>106</td>
<td>2001–2011</td>
</tr>
</tbody>
</table>

*The Australian and New Zealand Society of Cardiac and Thoracic Surgeons National Cardiac Surgery Database.
the data in six month blocks, the mirror image results are strikingly seen (Figure 2).

Discussion

This study confirms the seasonal association of aortic dissection, with an increase in incidence during the winter months. The impact of weather on cardiovascular disease has been investigated previously. Verberkmoes et al. [6] looked at the incidence of STADs, acute myocardial infarctions (AMI), and abdominal aortic aneurysms in the Dutch population and found a positive correlation with temperature (and no correlation with atmospheric pressure) for STAD and AMI, but did not see a similar correlation with abdominal aortic aneurysms. As suggested by Edwin et al. [7], Verberkmoes proposed that the pathogenesis of STAD was a balance between three factors: an underlying abnormality of the aortic media (either genetic or acquired), intimal injury, and hemodynamic factors, which would propagate the dissection once the tear was initiated. In this framework it would appear that cold weather would impact the third of these factors due to an increase in systemic vascular resistance. The work of Benouaich et al. [8], like the Dutch study, correlated the incidence of STAD with atmospheric temperature; and like our study, they found the incidence of dissection to be higher in the winter months than in the summer (p=0.018). Using data from the French national meteorological office (MétéoFrance) the authors further showed that within the same season, days with aortic dissection were colder than those without (p=0.017).

Weather is not the primary reason aortas dissect, and as already noted there is interplay between at least three factors. Other hemodynamic factors would include underlying chronic hypertension, acute hypertensive episodes as well as other extrinsic events. Ryu et al. [9], in their small series of 166 patients with STADs, found that 72% of the STAD episodes were associated with either physical (53%) or mental (19%) activities. Hatzaras et al. [10] documented 31 patients with an acute STAD associated with intense physical exertion, predominantly weightlifting. Based on their analysis, they concluded that “…moderate aortic dilatation confers vulnerability to exertion-related aortic dissection” (mean aortic diameter in this series was 4.63 cm). Thus we could speculate that colder weather, in the context of moderate aortic dilatation, could increase the systemic vascular resistance, resulting in a higher risk for dissection. In addition, colder temperatures also have systemic effects on hematologic, hemostatic, and rheological factors which may produce local effects on the intima which may further increase the risk of dissection [11].

Other chronobiological themes have been examined in STADs. Recently, Shuhaiiber et al. [12] examined the impact of the lunar cycle on mortality and length of stay following surgical repair of STAD. Season did not have a significant impact on survival, but the lunar cycle, specifically the Full moon, impacted...
Acknowledgments

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ANZSCTS DATA MANAGEMENT CENTRE, CCRE, Monash University: Prof. Chris Reid, Dr. Lavinia Tran, and Mrs. Angela Brennan.

ANZSCTS DATABASE PROGRAM STEERING COMMITTEE: Mr. Gil Shardey (Chair), Mr. Peter Skillington, Prof. Julian Smith, Mr. Andrew Newcomb, Mr. Siven Seevanayagam, Mr. Bo Zhang, Mr. Hugh Wolfenden, Mr. Adrian Pick, Mr. Jurgen Passage, A/Prof Rob Baker, Prof Chris Reid, Dr Lavinia Tran, and Mr. Andrew Clarke.

The following investigators, data managers and institutions participated in the ANZSCTS Database: Alfred Hospital: Pick A, Duncan J; Austin Hospital: Seevanayagam S, Shaw M; Cabrini Health: Shardey G; Geelong Hospital: Morteza M, Zhang B, Bright C; Flinders Medical Centre: Knight J, Baker R, Helm J, Canning N; Jessie McPherson Private Hospital: Smith J, Baxter H; Hospital: John Hunter Hospital: James A,

Limitations

As is common with retrospective database analyses, the data analyzed were heterogeneous in the time-frame of data collection between institutions. We attempted to maximize the number of patients included by pooling the site data without regard to time of collection with the underlying assumption that there would not be a historical difference in occurrence. Additionally, as noted previously, we assumed that the onset of the STADs coincided with the time of surgery, which would mean that chronic STADs are likely to be included in the analysis. Without censoring of chronic STADs, we analyzed the data under the assumption that each site had the same proportion of chronic STADs, and that the incidental finding of a chronic STAD would be randomly distributed through the calendar year. Likewise, since acute STADs are traditionally defined as occurring within 2 weeks of the onset of symptoms, the possibility that the actual acute event could straddle the defined cutoff of seasons might erroneously place the onset in the wrong season. Finally, this analysis was based only on patients undergoing surgical repair, and thus conclusions cannot be drawn regarding the effect of season on populations with dissection who expired without antemortem diagnosis or who were excluded from surgical consideration.

Conclusion

Our study confirms the seasonal variation associated with aortic dissection and presents the added finding that this variation is independent of hemisphere, that is, calendar month. As proposed by others, this variation is multifaceted and may reflect seasonal changes in sympathetic activity, including changes in blood pressure and heart rate in colder months. Season alone is not the primary driver for aortic dissection, but this and other studies promote the idea that for patients deemed at risk, attention to amelioration of co-factors (i.e., hypertension) becomes especially pertinent during the winter season.
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Conflict of Interest

The authors have no conflicts of interest relevant to this publication.
Quadricuspid Aortic Valve Combined with Moderate Ascending Aortic Dilatation
A Report of Four Cases

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Abstract
The quadricuspid aortic valve is a very uncommon malformation associated with aortic insufficiency, aortic stenosis, endocarditis, and ascending aortic dilatation. We report four cases of this aortic valve malformation. One patient with severe aortic regurgitation and moderate aortic dilatation required aortic valve replacement. Three patients had mild or moderate aortic insufficiency combined with moderate ascending aortic dilatation. These patients were referred to follow-up. The presented cases demonstrate that this aortic valve malformation may not be as rare as it appears and that attention must be paid to any quadricuspid findings during computed tomographic angiography and echocardiography.

Key Words
Aorta • Aortic valve disease • Bicuspid aortic valve • Dilatation • Quadricuspid ascending aneurysm

Introduction
The quadricuspid aortic valve (QAV) is a very uncommon malformation [1]. A much more common congenital aortic valve lesion is bicuspid aortic valve (BAV). Approximately 50% of patients with QAV develop severe aortic insufficiency (AI) [2], which may require aortic valve replacement [3]. Another condition common to QAV is ascending aortic dilatation. We present four cases of congenital QAV with varying degrees of aortic dilatation and insufficiency.

Case Report
Three asymptomatic patients (two males) underwent computed tomographic (CT) angiography, which revealed borderline ascending aortic dilatation (Figure 1A) and congenital QAV (Figure 1B). Echocardiography revealed mild or mild-to-moderate (between grades 1 and 2) AI in all patients, without significant aortic stenosis, and with normal to moderately dilated left ventricle (Table 1). The 2- and 3-dimensional echocardiography verified symmetric a 4-cusp valve and a 4-sinus aortic root. These patients were followed medically.

Another patient, a 64-year-old male, was admitted to our clinic, with dyspnea on exertion and chest discomfort. Echocardiography revealed QAV with severe AI (Figure 2A) and moderate ascending aortic dilatation. The left ventricle was dilated with normal ejection fraction (Table 1). CT angiography confirmed these findings. Coronary angiography revealed normal coronary arteries. The patient underwent open heart surgery (Figure 2B). The aortic valve was replaced with a 23-mm mechanical bi-leaflet.
prosthesis. Histological examination revealed fibrosis of the aortic cusps. The post-operative period was uneventful. Echocardiography before discharge showed normally functioning aortic valve prosthesis and reduced volume of the left ventricle without any depression of its contractility.

Discussion

The congenital QAV is a rare abnormality of the aortic valve, diagnosed in 0.04–0.008% of autopsies as well as echocardiography studies [1, 2]. Like BAV, congenital QAV is associated with the development of AI, aortic stenosis and ascending aortic dilatation [2, 3]. In all our presented cases, the ascending aorta was moderately dilated with the presence of some AI. In one case aortic valve replacement was required due to severe AI. In addition, QAV can be associated with other cardiac abnormalities: ruptured sinus of Valsalva, subaortic stenosis, hypertrophic cardiomyopathy, coronary artery abnormalities, patent ductus arteriosus and ventricular septal defects [4].

Hurwitz and Roberts [1] described seven types of congenital QAV based on valve leaflet size. Due to the risk of aortic-associated severe complications (e.g., acute ascending aortic dissection) and progression of aortic regurgitation, early diagnosis and follow-up in patients with congenital QAV is very important. In all our presented cases, the diagnosis was made by CT angiography.

In QAV, a question arises regarding the timing of surgery. Two factors should be noted: the degree of ascending aortic dilatation and the AI grade. In current guidelines for the management of patients with valvular heart disease there is no data concerning surgery in patients with QAV. We feel that recommendations regarding BAV may reasonably be used also for QAV.

We conclude, that QAV may not be quite as rare as we previously thought. QAV is associated with the development of aortic regurgitation and ascending

<p>| Table 1. |</p>
<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age, years</th>
<th>BSA, m²</th>
<th>QAV type [1]</th>
<th>Aortic root diameter, mm</th>
<th>Ascending aortic diameter, mm</th>
<th>LV EDV, ml</th>
<th>LV EF, %</th>
<th>AV peak gradient, mm Hg</th>
<th>AI, grade</th>
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<td>B</td>
<td>41</td>
<td>44</td>
<td>291</td>
<td>51</td>
<td>19.2</td>
<td>3</td>
<td>Surgery</td>
</tr>
<tr>
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<td>63</td>
<td>1.89</td>
<td>B</td>
<td>40</td>
<td>43</td>
<td>142</td>
<td>55</td>
<td>9.3</td>
<td>1</td>
<td>Follow-up</td>
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<tr>
<td>3</td>
<td>61</td>
<td>2.21</td>
<td>D</td>
<td>44</td>
<td>46</td>
<td>224</td>
<td>56</td>
<td>21.0</td>
<td>2</td>
<td>Follow-up</td>
</tr>
<tr>
<td>4</td>
<td>70</td>
<td>1.82</td>
<td>A</td>
<td>37</td>
<td>45</td>
<td>173</td>
<td>65</td>
<td>17.6</td>
<td>2</td>
<td>Follow-up</td>
</tr>
</tbody>
</table>

AI = aortic insufficiency; BSA = body square area; EDV = end-diastolic diameter; EF = ejection fraction; LV = left ventricle; QAV = quadricuspid aortic valve.

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EDITOR’S COMMENTS

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Quadricuspid Aortic Valve: Rare But Real

With interest I read the report of Uspenskiy and associates on quadricuspid aortic valve (QAV) with ascending aortic dilatation.

QAV is believed to be a very rare congenital anomaly, but the incidence is probably underestimated. Although the incidence was reported to be between 0.008-0.033% in early years, the incidence was 0.043% in an echo series [1]. In patients undergoing aortic valve replacement, the incidence ranges from 0.55% to 1.46% [2].

In 2001, Nakamura and colleagues [3] proposed a classification according to the position of the accessory cusp based on a review of 42 patients with QAV in Japan. It is composed of 4 types and simpler than the classification originally described by Hurwitz and Roberts in 1973 [4].

Recent literature suggests that an abnormal number of cusps is caused by developmental changes in the early stages of truncal separation. The abnormal aortic valve can be uni-, bi-, quadri- and penta-cuspid [5]. BAVs tend to be stenotic while QAVs tend to regurgitate. In a recent large series of 31 patients with QAVs from the Cleveland Clinic, moderate to severe aortic regurgitation was seen as the predominant hemodynamic lesion in 68% of the patients [6]. Specific criteria for aortic intervention in patients with QAV do not exist, due to the rarity of this anomaly.
References


Principles for Management of Intraoperative Acute Type A Aortic Dissection

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Abstract
Intraoperative Type A aortic dissection is a rare pathology with incidence of 0.06–0.32%. It is associated with a high mortality between 30–50%. Some associated risk factors, including hypertension, enlarged aorta, peripheral vascular disease, advanced age, atheroma, and high arterial pressure on cardiopulmonary bypass, have been identified. Modification of these risk factors could reduce the incidence of this event. Prompt diagnosis and management, with the aid of intraoperative trans-esophageal echocardiography and/or epi-aortic ultrasound has been shown to reduce the mortality to 17%. We illustrate the principles of management of this pathology with the case of a 62-year-old female who developed acute Type A aortic dissection while undergoing minimally invasive mitral valve repair.

Key Words
Aortic dissection • Aortic operation • Surgical complications • Practice guidelines (aorta) • Perioperative care

Introduction
Intraoperative or iatrogenic Type A aortic dissection is a very rare but important pathology with an incidence of 0.06 to 0.32% [1, 2]. It has a high mortality of between 30 to 50% [3]. Early detection and prompt treatment has been shown to reduce its mortality to about 17% [1]. It is associated with high morbidity and often requires further surgery. Its risk factors are known. All cardiovascular surgeons should be aware of its risk factors and have immediately in mind a strategy for prompt identification and management. We illustrate the management principles of this pathology with the case of a 62-year-old female admitted for mitral valve repair.

Case Presentation
A 62-year-old female presented with sudden onset, shortness of breath, and chest pain. Further investigation showed severe mitral regurgitation due to chordae rupture with a dilated aortic root measuring 4.0 cm and trivial aortic regurgitation with a trileaflet aortic valve. Her past medical history was remarkable for paroxysmal atrial fibrillation and hypertension but no history of steroid use or connective tissue disorder. She had unobstructed coronary arteries and was referred for urgent mitral valve repair.

She had an uneventful general anesthesia for minimally invasive mitral valve repair. There was fluctuation in her blood pressure during femoral artery cannulation for the initiation of cardiopulmonary bypass and trans-esophageal echocardiography showed features of Type A acute aortic dissection.

The operation was converted to full sternotomy. Ascending aortic cannulation was secured and the arterial line transferred from the femoral site. The hemodynamic was stable with blood pressure of 50–60 mm Hg, after cooling to 18°C, the pump was stopped...
and cannulation site changed. Via aorto-bicaval cannulation, she underwent uneventful mitral valve repair with Capentier Edward’s 38-mm mitral annuloplasty ring and two neo-chodea to P2 with Gortex sutures via inter-atrial sulcus approach; the ascending aorta was replaced up to the hemi-arch with 30-mm Hemashield graft. A period of deep hypothermic circulatory arrest at 18°C was required for the aortic anastomosis. There was difficulty in weaning off bypass with some ischemic changes on the inferior ECG leads. A saphenous vein coronary artery bypass graft to right coronary artery facilitated weaning off cardiopulmonary bypass and resolution of ischemia. She made a good and uneventful recovery. Histology of aorta showed cystic medial necrosis. Postoperative CT aortogram on day 2 (Figure 1) showed residual aortic dissection extending from the proximal aortic arch down to and involving both iliac arteries. There was no compromise of any of the aortic branches. She was followed up with serial echocardiogram and CT aortogram. Three years postoperatively, there was a significant increase in the diameter of the aortic arch to 5.5 cm, extending to the proximal descending thoracic aorta. The residual aortic dissection was electively repaired by replacement of the aortic arch with a 30-mm Hemashield graft using the elephant trunk technique under deep hypothermic circulatory arrest (DHCA) at 18°C. A 4-mm tear was found at the junction of the distal aortic arch and descending aorta probably from the previous dissection. There was a false lumen in the native aorta partially covering the innominate origin and extending into the distal arch. The innominate and left carotid arteries were re-implanted to the graft on an island of tissue, while the subclavian artery was left in situ. The innominate and left carotid arteries were cannulated with a cardioplegia cannula and used for antegrade cerebral perfusion at 10 ml/kg. The distal arch was divided between the left carotid and subclavian arteries circumferentially. The intimal tear distal to this site was closed with two pledged horizontal mattress sutures. At the completion of the graft to island anastomosis, the neck vessels cannulae were removed carefully, de-aired, and then the circulation through the aortic arch was commenced to the entire body except the heart. The proximal end of the graft tube was sutured to the previous tube graft. Complete circulation was recommenced, including perfusion of the heart. She again made a good and uneventful recovery from the operation. The residual stable dissection in the distal descending thoracic aorta is being managed conservatively and monitored with serial CT scans (Figure 2) [2].
Management of intraoperative iatrogenic aortic dissection

Discussion

Iatrogenic Type A aortic dissection is a very rare but important pathology with an incidence of 0.06 to 0.32% [3, 4]. It has a high mortality of between 30 to 50% [5]. Early detection and prompt treatment has been shown to reduce its mortality to about 17% [1]. Proper anesthetic strategy of hypotension and DHCA have also been shown to enhance outcome [1, 6].

Risk factors for intraoperative Type A aortic dissection have been identified (Table 1). These include hypertension, peripheral vascular disease, advanced age (>65 years), large diameter of the aorta, use of steroid, pre-existing aortic pathology, femoral arterial cannulation, and high cardiopulmonary bypass pressures greater than 120 mm Hg [3, 5, 7].

Intraoperative Type A aortic dissection is potentially fatal and thus essential that all cardiovascular surgeons are aware of this entity, including its risk factors, strategy for prompt identification, and management options.

Our case demonstrates a patient with identifiable risk factors for intraoperative Type A aortic dissection, namely hypertension and enlarged aorta. Also, intra-operatively, she had a period of hypertension during femoral cannulation. The immediate availability of intraoperative TOE facilitated a prompt diagnosis of the pathology [8]. Prompt strategy of immediate repair of the dissection via sternotomy and alternative arterial cannulation site with deep hypothermic circulatory arrest facilitated a good outcome for the patient [9].

Intraoperative Type A aortic dissection has been reported in all types of cardiac surgery including off pump coronary artery bypass surgery. All arterial cannulation sites have been identified as possible initiation sites [1]. Precipitating factors include arterial cannulation, aortic cross clamp application, and removal of the arterial cannula [7].

Table 1. Risk factors for iatrogenic Type A aortic dissection.

<table>
<thead>
<tr>
<th>Preoperative steroids</th>
<th>Femoral cannulation</th>
<th>Enlarged ascending aorta (≥ 4.0 cm)</th>
<th>Hypertension</th>
<th>Peripheral vascular disease</th>
<th>Advance age (≥70 years)</th>
<th>Asian race</th>
<th>Female gender</th>
<th>High cardiopulmonary bypass pressure (&gt;120 mm Hg)</th>
<th>Connective tissue disease</th>
<th>Aortic pathology (aortitis)</th>
<th>Arteriosclerosis</th>
</tr>
</thead>
</table>

Figure 2. Computerized Tomography (CT) aorta showing staged replacement of ascending aorta and arch (Panel B) with residual stable descending aorta dissection. (Panel A)
Intraoperative trans-esophageal echocardiography (TEE) and/or epi-aortic ultrasound are the gold standards for prompt diagnosis of intraoperative aortic dissection, although clinical observation is also essential [1, 10]. Dissection is easily identified by blue discoloration and expansion of the ascending aorta, intractable bleeding from aortic sites, and high perfusion line pressures with associated systemic hypertension. Once dissection is identified, separation from cardiopulmonary bypass and pursuit of an alternative cannulation site with repair of the aortic dissection under deep hypothermic circulatory arrest is the optimal treatment strategy. Blood pressure of less than 50 mm Hg during change of cannulation site is associated with poor outcome [4]. Transapical aortic cannulation is a reasonable option in this situation [11].

Risk factor modifications include avoiding cannulation of significantly diseased arterial sites, avoiding clamping of a highly pressurized aorta, torqueing of partial occlusion clamp, and minimal or gentle handling of the aorta may discourage the occurrence of this pathology.

We suggest that all patients with identifiable risk factors for intraoperative Type A aortic dissection should have intraoperative TEE monitoring and risk factor modification strategy during any cardiac surgery. This will facilitate prompt identification and treatment with significant reduction in mortality [1].

Intraoperative Type A aortic dissection is a rare but important pathology with high morbidity and mortality. Identifiable risk factors can be managed to discourage its occurrence. Prompt identification of intraoperative aortic dissection by intraoperative TEE has been shown to enhance outcomes.

**Conflict of Interest**

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

References


Case Report

Complex Reoperation for Late Complications After Acute Type A Aortic Dissection Surgery

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Abstract

The correct management of acute Type A dissection continues to be a challenge. The primary goal is to save the patient’s life. However, the decision regarding the surgical approach determines possible later complications. We present the case of a 59-year-old female patient with a past history of emergent surgery for acute Type A dissection treated by supra-coronary ascending and aortic valve replacement 19 years previously. Later, in a second endovascular approach, the descending aorta was treated by a thoracic endoprosthesis. During follow-up a dilated aortic root and a Type I endoleak were observed, and complex reoperation was required. We performed a total aortic arch replacement with a 4-branched graft and a complete aortic root replacement using the Cabrol technique for the reinsertion of the coronary arteries. The mechanical aortic normally functioning valve was preserved. The patient was discharged 30 days postoperatively.

Key Words
Aortic root aneurysm • Reoperation after Type A dissection • Complication Type A dissection

Introduction

It is well known that in acute Type A aortic dissection preventing mortality determines the emergency surgical tactic. The aim is perioperative patient survival. The decision regarding the technique to be used depends on the center and the surgeon’s experience, as well as on the patient’s clinical condition.

Valvular replacement or resuspension associated with replacement of the ascending aorta (AAR) alone has been demonstrated to be a safe and fast strategy, but there is no doubt that leaving the dissected aortic root may lead to numerous complications [1, 2]. The incidence of reoperation after repair of Type A aortic dissection is 5.4 to 18% [3]. The combination of valve and aortic root replacement eradicates proximal reoperations when it is made in the first instance.

Several mechanisms may be involved in late complications: progressive aortic valve regurgitation, increased dilation of the preserved aortic root [3], dilation of non-resected distal aorta, aneurysms of the false lumen, and pseudoaneurysm formation. We present the case of a patient who underwent first an aortic valve replacement (AVR) and an AAR due to acute Type A aortic dissection in 1992. Eleven years later an endograft was placed in the descending thoracic aorta due to chronic dissection. During patient follow-up, dilation of the aortic root and Type I endoleak were also observed. Our final approach was total aortic arch replacement with reimplantation of the supra-aortic vessels and an aortic root replacement with a Cabrol procedure preserving the mechanical aortic valve.

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Case Presentation

A 59-year-old female was admitted in 2011 to our institution for chronic back pain. The patient had a history of aortic valve and supracoronary aorta replacement with a mechanical prosthesis N° 23 (Car- bomedics®, Sorin Group, Milan, Italy) and a Dacron graft N° 28 in 1992, elsewhere. In 2003, because of refractory back pain, a computed tomography scan (CT scan) was done, and a progression of the dissection in the descending aorta was diagnosed. A thoracic endoprosthesis (Talent® 30 × 130 mm, Medtronic, Minnesota, USA) with occlusion of the left subclavian artery was implanted. In a CT control in 2005, a 1-cm distal migration of the endoprosthesis was observed leading to a Type I endoleak. A Talent® proximal cuff of 34 mm plus a distal extension of 32x150 mm were placed to deal with it. During later follow up, the aortic root showed a stable 50-mm diameter for 5 years. In 2010, the root was 6.05 cm, the thoracic aneurysm reached 6.4 cm, and the Type I endoleak was still persistent (Figure 1) and the patient continued with pain. We planned a reoperation in April 2011.

The procedure was performed through a median resternotomy under cardiopulmonary bypass (CBP). This was instituted through right axillary and right femoral arterial cannulation, and right femoral vein cannulation with a long cannula (Edwards Lifesciences, Irvine, California, USA). Left heart venting through right superior pulmonary vein was added. The ascending aorta graft was clamped and antegrade cold crystalloid Bretschneider cardioplegia was first

![Figure 1. Preoperative CT scan. Panel A. Aortic arch with type Ia endoleak (arrow). Panel B. Another view of the endoleak (arrow). Panel C. Dilated aortic root (60.5 mm) and descending aorta endoprosthesis with evidence of Type 1A proximal leak (arrow).](image1)

![Figure 2. Completed procedure. Four-branched prosthesis: 1. brachiocephalic trunk; 2. left carotid; 3. Dacron No. 8 (Cabrol technique); 4. distal anastomosis; and 5. perfusion limb, tied. *Note: The side-to-side Cabrol anastomosis was done with the third limb of the graft.](image2)
given, followed by cardioplegia via the coronary ostia after opening the Dacron graft. The graft was excised and the aortic root resected. When the temperature reached 20°C, the aortic clamp was removed and low flow brain perfusion begun through the axillary artery, clamping the brachiocephalic trunk (BCFT) proximally and adding selective perfusion through the left carotid artery. Carbon dioxide flooding was used. The aortic arch was transected between the left carotid (LCA) and left subclavian artery (LSA). The proximal bare wires of the thoracic endostent were cut and the arch reconstructed suturing a 4-branched Dacron graft (30 mm, Hemashield®, Maquet, Germany) with the endoprosthesis using 3.0 Prolene (Visi-black Prolene®, Ethicon, New Jersey, USA), reinforced with a band of Teflon felt from outside. The new prosthesis was then clamped and lower body flow was restored. Both LCA and the BCFT were transected at their origins and anastomosed to the 8-mm branches in an end-to-end fashion. The proximal aortic anastomosis was performed by suturing the proximal end of the 4-branched graft directly to the old mechanical valve ring in a continuous fashion using 3.0 Visi-black Prolene. The coronary reimplantation was done with a modified Cabrol technique, by using the proximal limb of the graft, cut at 3 mm of its origin for the side-to-side anastomosis (Figure 2). The perfusion limb of the graft was cut and oversewn. A needle vent was inserted for deairing and reperfusion was made. After weaning from CBP, protamine was administered, and the procedure was completed in a routine fashion. Aortic clamp time was 196 minutes, the total CPB time was 241 minutes, and the low flow perfusion time was 47 minutes. 

After the operation, the patient had atrial fibrillation without hemodynamic repercussion, and later bilateral pneumonia requiring 14 days of antibiotic treatment. The patient was discharged 30 days postoperatively. Before discharge, a new CT scan was performed (Figure 3).

**Discussion**

Acute Type A aortic dissection optimal treatment remains controversial. Different surgical approaches determine the short and long-term patient survival. The choice of the correct surgical strategy is generally determined by the individual risk of each patient and the surgeon’s experience [4].

Halstead and Rylski [3, 5] rethink indications after finding a higher incidence of reoperation after Type A dissection in patients undergoing valve replacement separately from the ascending aorta against the group who underwent Bentall procedure, who did not require reintervention. The average time between the first surgery and the development of a new aortic pathology was 4 years. In our case, it was 19 years. For the latter [5], the main causes of reoperation were incomplete resection of the tear, failure of obliteration of the false lumen, and severe aortic regurgitation. Pseudoaneurysm development and dehiscence of the suture line were also important causes of reoperation.

In our case, we decided to perform a Cabrol tech-
nique to treat the root due to the distance between coronary ostia [6]. We also showed, like Leacche [7], the advantage of avoiding the explantation of a well-functioning valve prosthesis, reducing the magnitude of the operation. We finally demonstrated the use of a Dacron 4-branched graft with the endoprosthesis incorporated into the distal anastomosis.

References


Conflict of Interest

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

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Abstract
An 81-year-old male patient with a history of aortic valve replacement presented to our hospital with right iliac fossa pain, chronic constipation, and fever. Hemocultures revealed the presence of anaerobic, Gram-positive bacteria identified as Clostridium septicum [1]. An initial thoraco-abdominal computed tomography (CT) scan demonstrated an abscessed tumor of the cecum with enlarged iliac lymph nodes. The aortic arch was moderately calcified but not dilated (Figure 1A).

After 1 day of triple antibiotic therapy (cefotaxim, gentacillin, and metromidazol), a right hemicolecotomy with double enterostomy was performed for an occlusive syndrome. Antibiotics were switched to penicillin plus clavulanic acid as suggested by sensibility tests.

Two weeks postoperatively, the patient presented with a new septic syndrome, and hemocultures revealed the presence of the same strain of C. septicum. A thoraco-abdominal CT scan found no deep abdominal collection but showed the beginning of dilatation at the distal part of the aortic arch with intra-luminal thrombotic material (Figure 1B).

Fifty days postoperatively, the patient presented with chest pain but no fever. A thoracic CT scan showed an sacciform distal aortic arch aneurysm with intramural air bubbles (Figure 1C and D) suggestive of anaerobic infection [2]. Considering the advanced age of the patient, endovascular temporary exclusion [3] or surgery was declined, and only palliative antibiotics were proposed (penicillin + clavulanic acid in addition to metromidazol). The patient subsequently died of aortic arch rupture.

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Conflict of Interest
The authors have no conflicts of interest relevant to this publication.
Figure 1. Thoracic CT scan. Axial images (Panel A and Panel C same level; Panel B and Panel D same level). Panel A. Day 0, showing a moderately calcified aortic arch. Panel B. Day 14, showing the beginning of dilatation of the distal part of the aortic arch with intraluminal hypo-dense material suggestive of thrombus. (Panel C and Panel D) Day 50, showing a sacciform distal aortic arch aneurysm with air within the aortic wall suggestive of anaerobic infection.

References


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List of Upcoming Meetings

March 2016

1. 12th International Congress of Update in Cardiology and Cardiovascular Surgery
   March 10-13, 2016
   Antalya, Turkey
   Meeting information available at: www.uccvs2016.org

2. Society for Cardiothoracic Surgery in Great Britain & Ireland 80th Annual Meeting
   Birmingham, United Kingdom
   Meeting information available at: www.scts.org

3. 22nd Annual Conference of the Egyptian Society of Cardiothoracic Surgery
   April 20-22, 2016
   Cairo, Egypt
   Meeting information available at: escts2016.com

4. 65th International Congress of the European Society for Cardiovascular and Endovascular Surgery
   April 21-24, 2016
   Belgrade, Serbia
   Meeting information available at: www.escvs2016.org

April 2016

1. Robotics Symposium - Fourth Biennial
   April 7-9, 2016
   Miami Beach, Florida
   Meeting information available at: MiamiRobotics.BaptistHealth.net

2. The 63rd Annual Conference of the Israel Heart Society
   April 12-13, 2016
   Tel-Aviv, Israel
   Meeting information available at: 2016.en.israelheart.com

May 2016

1. American Association for Thoracic Surgery
   Aortic Symposium 2016
   May 12-13, 2016
   New York, New York
   Meeting information available at: www.aats.org/aortic

2. American Association for Thoracic Surgery
   96th Annual Meeting
   May 14-18, 2016
   Baltimore, Maryland
   Meeting information available at: www.aats.org/annualmeeting