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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.
- The safety and effectiveness of the Endurant II Stent Graft System has not been evaluated in some patient populations. Please refer to the product Instructions for Use for details.

MRI Safety and Compatibility
Non-clinical testing has demonstrated that the Endurant II Stent Graft is MR Conditional. It can be scanned safely in both 1.5 T & 3.0 T MR systems under certain conditions as described in the product Instructions for Use. For additional information regarding MRI please refer to the product Instructions for Use.

Adverse Events
Potential adverse events include (arranged in alphabetical order): Amputation; Anesthetic complications and subsequent attendant problems (e.g., aspiration); Aneurysm enlargement; Aneurysm rupture and death; Aortic damage, including perforation, dissection, bleeding, rupture and death; Arterial or venous thrombosis and/or pseudoaneurysm; Arteriovenous fistula; Bleeding, hematoma or coagulopathy; Bowel complications (e.g., ileus, transient ischemia, infarction, necrosis); Cardiac complications and subsequent attendant problems (e.g., arrhythmia, myocardial infarction, congestive heart failure, hypotension, hypertension); Claudication (e.g., buttock, lower limb); Death; Edema; Embolization (micro and macro) with transient or permanent ischemia or infarction; Endoleak; Fever and localized inflammation; Genitourinary complications and subsequent attendant problems (e.g., ischemia, erosion, fistula, incontinence, hematuria, infection); Hepatic failure; Impotence; Infection of the aneurysm, device access site, including abscess formation, transient fever and pain; Lymphatic complications and subsequent attendant problems (e.g., lymph fistula); Neurologic local or systemic complications and subsequent attendant problems (e.g., confusion, stroke, transient ischemic attack, paraplegia, paraparesis, paralysis); Occlusion of device or native vessel; Pulmonary complications and subsequent attendant problems; Renal complications and subsequent attendant problems (e.g., embolism, stroke, transient ischemic attack, paraplegia, perinephric; Vessel damage; Wound complications and subsequent attendant problems (e.g., dehiscence, infection, hematoma, seroma, cellulitis)

Please reference product Instructions for Use for more information regarding indications, warnings, precautions, contraindications and adverse events.

CAUTION: Federal (USA) law restricts this device to sale by or on the order of a physician.
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Prognostic Implications of Acute Renal Failure after Surgery for Type A Acute Aortic Dissection

Fabrizio Sansone, MD*, Alessandro Morgante, MD†, Fabrizio Ceresa, MD, Giovanni Salamone, MD, Francesco Patanè, MD
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† The first two authors have contributed equally to the paper

Abstract

Background: “Type A” acute aortic dissection (AAAD) is the most challenging among the emergency operations in cardiac surgery. The aim of this study was the evaluation of the role of acute renal failure (ARF) in postoperative survival of patients operated for AAAD. Methods: From February 2010 to April 2012, 37 consecutive patients were operated at our department for AAAD. We studied our population by subdividing the patients within groups according to the presence of ARF requiring continuous veno-venous hemofiltration (CVVH) and according to hypothermic circulatory arrest (HCA) times and degrees. Results: The overall 30-day mortality was 27% (50% group A with ARF, 13% group B no ARF). Acute renal failure requiring CVVH was 37.8%. Multivariate analysis revealed a significant association with 30-day mortality (odds ratio 6.6 and p = 0.020). Preoperative oliguria (urine output less than 30 ml/h (odds ratio 4.7 p = 0.039)), CPB greater than 180 minutes (odds ratio 6.5 p = 0.023) and postoperative bleeding requiring a surgical reopening (odds ratio 12.2 and p = 0.021) were the variables significantly associated with acute kidney injury. Conclusions: The data obtained from our analysis bring out the high incidence of renal injuries after surgery for AAAD, and indicate a negative impact on renal injuries of a preoperative oliguria, longer Cardiopulmonary bypass (CBP)/HCA times, and postoperative bleeding requiring a surgical revision. Our data also suggest a better 30-day survival and better renal outcomes in case of shorter HCA and lesser degree of hypothermia.

The option of lesser and shorter hypothermia may be very useful, especially for the elderly patients and octogenarians.

Key Words
Type A • Acute aortic dissection • Acute renal failure • Continuous veno-venous hemofiltration • Hypothermic circulatory arrest

Introduction

Nowadays “Type A” acute aortic dissection (AAAD) represents the most dramatic emergency in the clinical practice of cardiac surgery, because of high in-hospital mortality rates, estimated as about 26% from IRAD (International Registry of Acute Aortic Dissections) data, and because of several potential multiorgan complications after surgery. Preoperative clinical conditions are very often the best predictors of the final outcome, and deficient pre-operative coronary or visceral perfusion, pulseless shock, cardiopulmonary resuscitation or a coma or active stroke play a major prognostic role. Current surgical techniques of neuroprotection include the hypothermic circulatory arrest (HCA) alone or together with perfusion methods such as anterograde cerebral perfusion (ACP, Kazui technique, both unilateral and bilateral), permitting longer safe “HCA time” and shortening cooling and
rewarming times. The main aim of our study was to
determine the prognostic impact of acute renal fail-
ure (ARF) on postoperative survival. We also wished
to verify the potential associations of ARF with preop-
erative, perioperative and postoperative factors.

Material and Methods

Study population

From February 2010 to April 2012, 37 consecutive patients
were admitted at our division of Cardiac Surgery to undergo
cardiac surgery for AAAD. Our study population was charac-
terized by an age at surgery of 65±11 years (mean±SD), a male
sex 83.8% (31 men and 6 women), a BSA 1.86±0.2 (m²). All the
patients underwent surgery within 24 hours, outlining that
our patients very often arrive from peripheral hospitals, with
delay from diagnosis time.

Statistical analysis

Continuous variables are expressed as mean±SD and com-
pared by t-test, while categorical data are presented as per-
centages and compared by y² test and Fisher exact test, as
appropriate. Variables with prognostic impact on univariate
analysis entered the multivariate modality. A p value less than
0.05 was considered as statistically significant. All statistical
analysis was performed using SPSS 19.0 (SPSS Inc., Chicago,
United States) and Microsoft Office Excel. Primary end points
were 30-day mortality (30d-Mort) and postoperative ARF re-
quiring continuous veno-venous hemofiltration (CVVH). Renal
failure and injury were evaluated according to the RIFLE criteria;
other predictive biomarkers such as NGAL or IL-18 were not used
in our study. The association between primary end points and risk
factors was estimated through the calculation of odds ratio (OR).

Surgical methods

All operations were performed through a median sternoto-
mny. Cardio-pulmonary bypass (CPB) was established between
femoral (25 patients) or axillary artery (12 patients) and the
right atrium. After systemic anti-coagulation was reached by
300 UI/kg of heparin and after arterial cannulation, the median
sternotomy was performed; thus, the right atrium was cannu-
lated and CPB was started, reducing body temperature. In two
cases, CPB was established between femoral artery and femo-
ral vein during cardiopulmonary resuscitation for sudden rup-
ture of the aortic wall and pulseless shock. Left ventricular ven
was inserted via the right superior pulmonary vein. We usually
perform the proximal suture as a first step, while the cooling
phase. Thus, the distal suture was performed under deep hy-
pothemic circulatory arrest. In most cases we performed an
ascending aorta and hemiarch replacement (83.7%). A Bentall
procedure was performed in five patients (13.5%), and a Wheat
procedure with separate replacement of the ascending aorta
and aortic valve in one patient (2.4%). In three cases (7.3%) con-
comitant coronary artery bypass grafting was required as well.

During the cooling phase (1°C/3–5 minutes), follow-
ing the α-stat strategy, pump flow is adjusted to levels of
2.4–2.6 l/min/m² with a temperature gradient of about 6–7°C,
so that the natural alkaline shift related to hypothermia could
be offset resulting in better availability of oxygen for tissues.
We sometimes enhance cerebral cooling by packing ice
around the head. At the induction of the arrest we administer
Pentothal sodium, an ultra short-acting barbiturate, to reduce
cerebral metabolism. In conjunction with HCA, we usually
perform anterograde cerebral perfusion (ACP) by three can-
nulae inserted in the supra-aortic vessels (cannula into the left
sub-clavian artery was not always inserted for technical diffi-
culties), according to the Kazui technique. An average perfu-
sion pressure of approximately 65 mm Hg and a flow of about
760 mL/min were maintained. We did not use strategy of vis-
ceral protection during and after circulatory arrest, except for
hypothermia. Once the distal suture was completed, the flow
was slowly restored. In 10 patients of the 25 who received
femoral cannulation, the flow was restored through the side
branch of the prosthesis. In case of axillary cannulation, the
flow was restored through the same cannula.

Study groups

Patients were categorized according to the presence of ARF
requiring CVVH into two groups (Group A with ARF and Group
B without ARF). Furthermore, we subdivided our total study
population into other groups according to HCA time (HCA<15
min; HCA 15–30 min and HCA>30 min), and in relation to de-
grees of HCA (HCA<21°C, HCA 21–25°C, and HCA>25°C). We
evaluated the primary endpoints for each subset of patients.

Results

Surgical outcomes

The main preoperative, intraoperative and post-
operative variables are schematized in Table 1. In
terms of preoperative risk factors, a “shock condition”
(systolic pressure less than 100 mm Hg, heart rate
greater than 100 bpm, low urine output less than
30 ml/h) occurred in 45.9% of cases (42.8% group A,
47.8% group B) and 21 patients (56.7%) developed
cardiac tamponade (50% group A, 60.8% group B),
but both shock and cardiac tamponade were not
significantly correlated with ARF. A preoperative
reduced or absent urine output before surgery, was
found in 56.7% of cases (78.5% group A, 43.4% group
B), with an OR for the onset of ARF of 4.7 (p = 0.039).

Analyzing operative times expressed in minutes
(mean±SD), we found a cardiopulmonary bypass (CPB)
time of 212±77 (242±77.3 group A, 193±72.2 group B),
a cross-clamp time of 87.2±51.5 (92.2±48.5 group A,
84.1±54 group B), and a HCA time of 37.2±21.4 (48±23.5
group A, 30.6±17.4 group B). The ACP following
the “Kazui” technique was 33.9±19.6 in the study

AORTA, June 2015

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Bleeding complications were manifest in 16.2% of cases (35.7% group A, 4.3% group B), resulting in a significant association with kidney injury (OR 12.2; p = 0.021). Acute renal failure requiring CVVH was found with a frequency of 37.8% (14 pts) and was on multivariate analysis significantly associated with 30-day mortality (p = 0.020) with OR of 6.6, confirming this as an independent risk factor for early mortality. The definition of an acute renal injury followed the criteria of RIFLE (risk, injury, failure, loss of population. CPB times greater than 180 minutes presented a higher incidence of ARF (52.1%, OR 6.5, p = 0.023) than CPB<180 minutes (14.2%, OR 0.1, p = 0.023). The overall 30-day mortality was 27% (50% group A, 13% group B, p < 0.05), Five patients (13.5%) had a mortality-corrected permanent neurological dysfunction (PNDmc, a parameter that defines the percentage of patients with neurological deficits who survived to 30 days or were discharged with neurological dysfunction).

Table 1. Preoperative, intraoperative, and postoperative variables in the total population: ARF group (1) and noARF group (2).

<table>
<thead>
<tr>
<th></th>
<th>Total Population</th>
<th>ARF Group</th>
<th>NoARF Group</th>
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</thead>
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<tr>
<td>No. of patients (%)</td>
<td>37</td>
<td>14 (37.8)</td>
<td>23 (62.2)</td>
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<tr>
<td>Age at surgery, y (mean±SD)</td>
<td>65±11</td>
<td>65±10</td>
<td>65±11</td>
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<tr>
<td>Male sex, %</td>
<td>83.8</td>
<td>92.8</td>
<td>78.2</td>
<td></td>
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<td><strong>Preoperative risk factors, % (no. of patients)</strong></td>
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<tr>
<td>Neurological deficit before surgery</td>
<td>8.1 (3)</td>
<td>14.2 (2)</td>
<td>4.3 (1)</td>
<td>p = ns</td>
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<tr>
<td>Shock condition</td>
<td>45.9 (17)</td>
<td>42.8 (6)</td>
<td>47.8 (11)</td>
<td>p = ns</td>
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<tr>
<td>(systolic pressure &lt; 100 mm Hg, heart rate &gt; 100 bpm, low urine output)</td>
<td></td>
<td></td>
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<tr>
<td>Pericardial tamponade</td>
<td>56.7 (21)</td>
<td>50 (7)</td>
<td>60.8 (14)</td>
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<td>Cardiopulmonary resuscitation</td>
<td>2.7 (1)</td>
<td>7.1 (1)</td>
<td>0</td>
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<tr>
<td>Intubated</td>
<td>45.9 (17)</td>
<td>64.2 (9)</td>
<td>34.7 (8)</td>
<td>p = ns</td>
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<td>Acute myocardial infarct</td>
<td>2.7 (1)</td>
<td>0</td>
<td>4.3 (1)</td>
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<tr>
<td>Oliguria(urine output &lt; 30 ml/h)</td>
<td>56.7 (21)</td>
<td>78.5 (11)</td>
<td>43.4 (10)</td>
<td>p = ns</td>
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<tr>
<td>Aortic regurgitation ≥ 2nd degree</td>
<td>27 (10)</td>
<td>35.7 (5)</td>
<td>21.7 (5)</td>
<td>p = ns</td>
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<td><strong>Postoperative risk factors, % (no. of patients)</strong></td>
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<td></td>
<td></td>
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<tr>
<td>PNDmc</td>
<td>13.5 (5)</td>
<td>14.2 (2)</td>
<td>13 (3)</td>
<td>p = ns</td>
</tr>
<tr>
<td>30-Day mortality</td>
<td>27 (10)</td>
<td>50 (7 pt)</td>
<td>13 (3pt)</td>
<td>p = ns</td>
</tr>
<tr>
<td>Reopening for bleeding</td>
<td>16.2 (6)</td>
<td>35.7 (5)</td>
<td>4.3 (1)</td>
<td>p = ns</td>
</tr>
<tr>
<td>Respiratory failure</td>
<td>35.1 (13)</td>
<td>50 (7)</td>
<td>23 (6)</td>
<td>p = ns</td>
</tr>
<tr>
<td><strong>Intraoperative and postoperative times, mean±SD</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiopulmonary bypass time, min</td>
<td>212±77</td>
<td>242±77.3</td>
<td>193±72.2</td>
<td>p = ns</td>
</tr>
<tr>
<td>Hypothermic circulatory arrest time, min</td>
<td>37.2±21.4</td>
<td>48±23.5</td>
<td>30.6±17.4</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>Intensive care unit stay, d</td>
<td>10.6±13.3</td>
<td>19±18.1</td>
<td>6±6.8</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>Hospital stay, d</td>
<td>17±13.1</td>
<td>17.3±16.8</td>
<td>13.3±8.4</td>
<td>p = ns</td>
</tr>
</tbody>
</table>

CPR = cardiopulmonary resuscitation; AMI = acute myocardial infarction; PNDmc = mortality-corrected permanent neurological dysfunction; Respiratory failure, respirator time > 48 hours or need of reintubation; CBP = cardiopulmonary bypass; HCA = hypothermic circulatory arrest; ICU = intensive care unit; ARF = acute renal failure.
function and end-stage renal disease) system classification, according to serum creatinine and glomerular filtration rate.

The flow chart we used to detect and treat acute renal failure was:

- Acute renal injury not requiring CVVH was defined as an increase in creatinine level more than 1.5 times the normal value or urine output less than 0.5 ml/kg/h for more than 6 h;
- Acute renal injury requiring CVVH was defined as an increase in creatinine level greater than 3 times the normal value or urine output less than 0.3 ml/kg/h for 12 hours;
- Anuria was always treated by CVVH within the 3 hours following the onset.

Furthermore, by HCA times we subdivided our population into various groups, studying the outcomes so obtained. In the Figure 1, we can observe a group of five patients with HCA less than 15 min (30-day mortality 0%, ARF 0%), another group (13 pat.) with HCA between 15-30 min (30-day mortality 15.3%, ARF 30.7%) and the last group (19 pat.) with HCA greater than 30 min (30-day mortality 42%, OR 5.8 p = 0.038; ARF 52.6% OR 2.8 p = 0.05). These data emphasize the benefits of as short as possible HCA in terms of a short-term survival and renal outcomes (Figure 1).

In addition according to degrees of HCA, as illustrated in Figure 2, we identified 12 patients who underwent HCA less than 21°C (30-day mortality 33.3%; ARF 50%, p = ns), another group of 23 patients in whom we performed HCA between 21–25°C (30-day mort 26%; ARF 34.7%, p = ns), and a last group of two patients with HCA greater than 25°C (30-day mortality 0%; ARF 0%).

These data seem to suggest a better 30-day survival and better renal outcomes with lesser hypothermia, an option especially useful for elderly patients. However, the statistical significance was not reached because of the small number of patients.

Discussion

Except for clinical instances in which both renal arteries arise from the false lumen of an aortic dissection, most cases of acute renal failure in acute Type A aortic dissection result from a functional (non-anatomic) mechanism with a multifactorial etiology. Renal damages can reflect medullary ischemia or reperfusion injury (HCA-mediated). The reduction of medullary blood flow with related hypoxia produces inability to regulate osmotic gradients involving active mechanisms of Na⁺ reabsorption; therefore, the subsequent cortical vasoconstriction and diminished glomerular filtration rates represent the true basis of an acute tubular necrosis [1-4].

Furthermore, after HCA during the reperfusion time an adequate hematocrit level is clearly useful for its redox and free-radical scavenging capacity, remembering also the potential negative influence on the coagulatory system by an excessive hemodilution. In addition, the same anemic status due to excessive postoperative bleeding can activate renal failure, as proven by our findings,
Chronic arterial hypertension, commonly accepted as the main risk factor leading to an acute dissection, plays an active role in generating a sclerosis process about the renal microcirculation, even if the serum creatinine values may be normal – creating a physiopathological basis for the development of acute kidney injury [5, 6].

The current state of the art in neuroprotection considers hypothermic circulatory arrest as the main option, even if nowadays the optimal temperature and the “safe time” remain debated [7]. Among the surgical neuroprotective techniques, anterograde cerebral perfusion (ACP) allows a moderate grade of hypothermia (according to the Kazui technique). Axillary artery cannulation can help perfuse the innominate trunk and the ipsilateral vertebral district, making use of the connections of the circle of Willis for the left hemispheric perfusion [8-10].

It is commonly understood that prolonged CPB and HCA times contribute to acute kidney injury. Despite deep HCA having always been considered more protective towards the visceral organs and kidneys, new clinical trends advocate moderate hypothermia without compromise in renal function [11-13].

Experimental studies have brought out the role of recombinant atrial natriuretic peptide as a protective factor to combat HCA-related renal injury. Benefits rely on vasodilation of the renal medulla through cGMP-mediated mechanisms and interference with the angiotensin-aldosterone system, reducing more-over the myeloperoxidase activity.

**Conclusions**

The theme of renal protection is complex and remains a compelling challenge. Multifactorial pathophysiology plays an underlying prognostic role regarding the final outcome for this life-threatening complication. The data obtained from our analysis bring out the high incidence of renal injuries after surgery for AAAD, outlining the predictive role of preoperative low urine output, longer CPB/HCA times and postoperative bleedings requiring a surgical revision, moreover confirming that a postoperative acute
renal failure represents an independent risk factor for early mortality. Additionally, our data suggest a better survival within 30 days and renal outcomes in case of shorter HCA and lesser hypothermia, an option very useful especially for the elderly patients.

References


EDITOR’S QUESTIONS

1. Do you think the poorer outcomes with longer bypass and deeper HCA just represent the more technically challenging disease? What can a surgeon do about this? How can we use this observation?

We agree with you that longer bypass time and deeper HCA are significantly associated to poorer outcome because of a “bias” caused by the higher surgical risk of the population considered. Thus, longer bypass time and deeper HCA are required in case of extensive involvement of the aortic arch when a more radical resection and reconstruction are mandatory and this leads to a significant higher risk of death. However, we think that in the modern era of neurological protection by selective cerebral perfusion, it is advisable to avoid a “too deep” HCA (i.e., <20°C) because the “safe period” for brain is not exclusively related to the temperature rather than the selective cerebral perfusion.
According to David experience, we think that a moderate hypothermia associated to selective anterograde cerebral perfusion, is the “gold standard of care” in case of HCA.

2. How should we modify practice based on your study?

Thanks to the experience we have reported, we have modified some details of neurological protection in our department. At the present time, we usually perform HCA at 22-24°C associated to selective cerebral perfusion and we reach the 18°C exclusively in case of preoperative evidence of aortic tear into the aortic arch when a complete aortic arch replacement is required. We think that this approach should be considered by other surgeons and hope that our results will be discussed in further studies.
Analysis of Strengths, Weaknesses, Opportunities, and Threats as a Tool for Translating Evidence into Individualized Medical Strategies (I-SWOT)

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1 German Aorta Centre of Hamburg, Centre of Cardiology and Cardiovascular Surgery, University Medical Centre Hamburg-Eppendorf, Hamburg, Germany
2 Institute of Medical Genetics and Human Genetics, Charité Universitätsmedizin, Berlin, Germany

Abstract

Background: It is the physicians’ task to translate evidence and guidelines into medical strategies for individual patients. Until today, however, there is no formal tool that is instrumental to perform this translation.

Methods: We introduce the analysis of strengths (S) and weaknesses (W) related to therapy with opportunities (O) and threats (T) related to individual patients as a tool to establish an individualized (I) medical strategy (I-SWOT). The I-SWOT matrix identifies four fundamental types of strategy. These comprise “SO” maximizing strengths and opportunities, “WT” minimizing weaknesses and threats, “WO” minimizing weaknesses and maximizing opportunities, and “ST” maximizing strengths and minimizing threats. Each distinct type of strategy may be considered for individualized medical strategies.

Results: We describe four steps of I-SWOT to establish an individualized medical strategy to treat aortic disease. In the first step, we define the goal of therapy and identify all evidence-based therapeutic options. In a second step, we assess strengths and weaknesses of each therapeutic option in a SW matrix form. In a third step, we assess opportunities and threats related to the individual patient, and in a final step, we use the I-SWOT matrix to establish an individualized medical strategy through matching “SW” with “OT”. As an example we present two 30-year-old patients with Marfan syndrome with identical medical history and aortic pathology. As a result of I-SWOT analysis of their individual opportunities and threats, we identified two distinct medical strategies in these patients.

Conclusion: I-SWOT is a formal but easy to use tool to translate medical evidence into individualized medical strategies.

Key Words
Aorta • SWOT analysis • Medical decision-making • Evidence-based medicine • Strategy

Introduction

The German Aorta Center of Hamburg (DAZ-H) is run by a team of surgeons, interventionalists and geneticists with experience in treating aortic diseases. Our aortic diseases board holds weekly conferences for decision-making on patients with aortic disease. In this report we describe how our board uses I-SWOT to establish individualized medical strategies for our patients [1, 2]. Originally, SWOT analysis was designed to assess strengths (S) and weaknesses (W) as internal capabilities of an organization as opposed...
to opportunities (O) and threats (T) posed by the external environment [3]. Today, SWOT analysis is one of the world’s most widely used methods for strategic planning [4, 5]. We use this instrument to match strengths and weaknesses of therapy with opportunities and threats related to individual patients and to establish individualized medical strategies. Such strategies are important to systematically integrate both specific health conditions and needs, values and attitudes of patients.

**Translation of evidence into individualized medical strategies**

Evidence based medicine (EBM) is “the integration of the best available evidence with our clinical expertise and our patients’ unique values and circumstances” [6]. However, protagonists of EBM emphasize that “evidence, whether strong or weak, is never sufficient to make clinical decisions”, and that EBM “is far from a one-size-fits-all strategy” [7].

Similarly, guidelines state that “the final decisions concerning an individual patient must be made by the responsible health professional(s)” [8]. Hence, it remains the physicians’ task to translate evidence and guidelines into medical strategies for individual patients. Until today, however there is no formal tool available to perform this translation. Here we introduce I-SWOT as a simple and easy to use tool to accomplish this task [2].

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**The general SWOT matrix**

At its simplest, strategy is what one may use to reach goals. SWOT analysis regards as “internal capabilities” all those factors that a strategic planner contributes himself to reaching a goal. Depending on whether these capabilities support or jeopardize attaining goals, SWOT analysis terms these factors “strengths” or “weaknesses”. Conversely, SWOT analysis regards as “external possibilities” all factors that the strategic planner does not control directly. Depending on whether these possibilities support or jeopardize goals, SWOT analysis terms these factors “opportunities” or “threats”. In our SWOT analysis physicians take the role of the strategic planner, where we call “strengths” or “weaknesses” those factors of therapy that relate to both the efficacy of a medication or an intervention, and the physician’s capability to deliver such therapy. “Opportunities” or “threats” are those factors of therapy that mostly relate to patients, such as the disease that requires treatment, the health status of the patient, or the motivation or capability to support therapy.

Matching strengths and weaknesses with opportunities and threats identifies four distinct types of strategy [1] (Figure 1).

**SO strategy**

This strategy maximizes both internal strengths and external opportunities (“Maxi-maxi” strategy) [3]. The strategy might be chosen in a cheerful situation...
with abundant own strengths and auspicious external opportunities. For example, well-established standard procedures are available for surgery of aortic root aneurysm, but a skilled surgeon may perform a promising but not yet-established operation in a healthy and motivated patient [9]. This surgeon maximizes exploit of strengths (“S”) given by his skills to accomplish the operation where he takes opportunity (“O”) of motivation and relatively good health of his patient. However, in such situations doctors may overrate their prospects.

WT strategy

This strategy minimizes both weaknesses and threats (“Mini-mini” strategy) [3]. The strategy might be chosen in a precarious situation in which strengths are sparse and threats are mounting. For example, a doctor may resort to purely medical therapy of a patient with an acute Type A dissection in a hospital without heart surgery. This doctor minimizes the weakness of therapeutic options in his clinic through applying the easily available but hardly effective medical therapy of Type A dissection (“W”), and he minimizes threats (“T”) by avoiding transportation of an unstable patient to another hospital. Doctors may strive to escape precarious situations and seek for other strategies.

WO strategy

This opportunity-focused strategy minimizes weaknesses and maximizes opportunities (“Mini-maxi” strategy) [3]. The strategy may be chosen in a situation where therapeutic options are severely restricted (“W”) while external opportunities (“O”) are promising. For instance, the doctor mentioned above might order a helicopter to get his patient into a surgical center. This doctor minimizes weaknesses (“W”) of treating aortic dissection in his own clinic by maximizing opportunities (“O”) of patient survival in another clinic. In the WO position doctors may seek to reinforce their own strengths to get better control over therapy.

ST strategy

This strength-focused strategy maximizes own strengths and minimizes threats (“Maxi-mini” strategy). The strategy may be chosen in bail-out situations where maximizing own strengths may be the only way to overcome substantial threats. For instance, a surgeon may treat a Crawford Type II expanding aortic dissection through replacement of the entire descending thoracoabdominal aorta (10). This doctor overcomes the substantial threat given by an expanding Crawford Type II dissection (“T”) by using his own capabilities (“S”) to perform extensive surgery with exceptionally good results.

However, in such situations doctors may avoid to exaggerate reliance on their own capabilities.

Four steps of I-SWOT to establish an individualized medical strategy

At our center, we perform four steps to establish an individualized medical strategy to treat aortic disease (Figure 2).

1. Define the goal of therapy and identify all evidence-based therapeutic options. The patients discussed in our aortic diseases board present with decision problems that are related to aortic disease. In the following, we present our discussion of a 30-year old man with a disease-causing FBN1 mutation and clinical criteria of Marfan syndrome (MFS). His aortic root diameter had progressed from 4.3 cm to 4.6 within one year. The goal of therapy is to protect this patient with an aortic root aneurysm against dissection. We identified the following 5 options which were available according to the literature [11-13]:

- Medical treatment with beta-blockers (BAB) [14], or angiotensin II–receptor blockers (ARB) [15];
- Elective surgery of the aortic root including personalized external aortic root support [9, 16];
- Aortic-valve-sparing reimplantation technique according to David [17];
- Composite valve grafting according to Bentall with bio-aortic valve prosthesis [18]; or
- Composite valve grafting according to Bentall with a mechanical valve [19].

In addition, we define the prognosis of aortic root aneurysm and the need for timing of an intervention. With an aortic diameter of 4.6 cm most guidelines would consider surveillance and BAB
### Four steps of I-SWOT to establish an individualized medical strategy

<table>
<thead>
<tr>
<th>Step</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Define the goal of therapy and identify all evidence-based therapeutic options</td>
</tr>
<tr>
<td>2</td>
<td>Assess Strengths and Weaknesses of each therapeutic option (SW-matrix)</td>
</tr>
<tr>
<td>3</td>
<td>Assess Opportunities and Threats related to the individual patient (OT-matrix)</td>
</tr>
<tr>
<td>4</td>
<td>Use I-SWOT to establish an individualized medical strategy</td>
</tr>
</tbody>
</table>

**Figure 2.** Four steps to establish an individualized medical strategy.

### Example for a standardized I-SWOT matrix form

<table>
<thead>
<tr>
<th>Internal capabilities related to therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>I-SWOT matrix form</td>
</tr>
<tr>
<td>Aortic root aneurysm in Marfan syndrome</td>
</tr>
<tr>
<td>Patient name: ___________________________</td>
</tr>
<tr>
<td>Date of audit: __________________________</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>External possibilities related to patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opportunities</td>
</tr>
<tr>
<td>ST maxi-mini strategy</td>
</tr>
<tr>
<td>WO mini-mini strategy</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Strengths</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. No surgical trauma (S1)</td>
</tr>
<tr>
<td>2. Losartan may cure aorta (S2)</td>
</tr>
<tr>
<td>3. No anticoagulants (S3)</td>
</tr>
<tr>
<td>4. Avoidance of prosthesis-patient mismatch (S4)</td>
</tr>
<tr>
<td>5. Low risk for endocarditis (S5)</td>
</tr>
<tr>
<td>6. No bypass needed (S6)</td>
</tr>
<tr>
<td>7. Good results at DA2-H (S7)</td>
</tr>
<tr>
<td>8. Short clamping-time (S8)</td>
</tr>
<tr>
<td>9. Durability of AVR (S9)</td>
</tr>
<tr>
<td>10. Classical gold standard (S10)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Weaknesses</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Protection is not safe (W1)</td>
</tr>
<tr>
<td>2. Drug intolerance (W2)</td>
</tr>
<tr>
<td>3. Innovative therapy (W3)</td>
</tr>
<tr>
<td>4. Need of expertise (W4)</td>
</tr>
<tr>
<td>5. Operation not available at DA2-H (W5)</td>
</tr>
<tr>
<td>6. Long clamping time (W6)</td>
</tr>
<tr>
<td>7. Reoperation for AVD (W7)</td>
</tr>
<tr>
<td>8. Prosthesis mismatch (W8)</td>
</tr>
<tr>
<td>9. Oral anticoagulation (W9)</td>
</tr>
<tr>
<td>10. Risk for endocarditis (W10)</td>
</tr>
</tbody>
</table>

**Figure 3.** Example for a standardized I-SWOT matrix form.
medication with surgery indicated only when a diameter ≥ 5.0 cm is reached. However, the patient had exhibited progression of 0.3 cm of his aortic diameter within one year; evidence and guidelines yielded conflicting data as to whether this was a risk factor to initiate surgery already at ≥ 4.5 cm [8, 20-24]. Finally we concluded that based on a simple analysis of evidence all five therapeutic options remained acceptable options for treatment of our patient.

2. Assess strengths and weaknesses of each therapeutic option (SW-matrix). The first step of I-SWOT analysis is to assess strengths and weaknesses of each therapeutic option. We establish strength-weakness (SW) matrices for each option, where we integrate information from studies, case reports, guidelines, and from our own experience. **Table 1** shows the SW-matrix for treating aortic root aneurysm in MFS (**Table 1**).

3. Assess opportunities and threats related to the individual patient (OT-matrix). The core of an individualized treatment strategy is to adjust treatment plans to the individual patient [1, 2]. The patient may have physical, psychological or mental health conditions, individual wills, needs, beliefs, values, risk attitudes, and emotions that may speak in favor of or against specific therapeutic strategies. Again, we screen the literature and

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**Table 1.** Strengths-weaknesses matrix of specific options to treat aortic root aneurysm in Marfan syndrome.

<table>
<thead>
<tr>
<th>Treatment option</th>
<th>Strengths</th>
<th>Weaknesses</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Wait and medicate</td>
<td>Prevention of surgical trauma (S1); ARB may cure aortic disease in some patients (S2); Other strengths: S3–S6</td>
<td>Protection against aortic root rupture and aortic valve regurgitation is not safe especially at aortic diameters &gt; 5.0 cm (W1); Patient may be nonresponders to drugs or may have drug reaction (W2)</td>
</tr>
<tr>
<td>(2) Personalized external aortic root support</td>
<td>No oral anticoagulation (S3); Preservation of native aortic valve (S4); Low risk of endocarditis (S5); Avoidance of cardiopulmonary bypass (S6)</td>
<td>Innovative therapy with little data on long-term outcome, where intervention is performed at 4–4.5-cm diameters when surgery is usually not indicated (W3); High level of expertise (W4); Operation is not performed at DAZ-H (W5)</td>
</tr>
<tr>
<td>(3) AVS re-implantation technique according to David</td>
<td>No oral anticoagulation (S3); Avoidance of prosthesis-patient mismatch (S4); Low risk of endocarditis (S5); Good results at DAZ-H (S7)</td>
<td>High level of expertise required (W4); Long clamping time (W6); Reoperation for AVD (W7)</td>
</tr>
<tr>
<td>(4) CVG according to Bentall with Bio-AVR</td>
<td>No oral anticoagulation (S3); Shorter clamping-time and easier to perform than AVS (S8)</td>
<td>Reoperation for AVD (W7); Patient-prosthesis mismatch (W8)</td>
</tr>
<tr>
<td>(5) CVG according to Bentall with mechanical AVR</td>
<td>Life-long durability of AVR (S9); Classic gold standard procedure (S10)</td>
<td>Patient-prosthesis mismatch (W8); Oral anticoagulation (W9); High risk of endocarditis (W10); Noise of mechanical valve (W11)</td>
</tr>
</tbody>
</table>

ARB = angiotensin-II-receptor blockers; AVD = aortic valve dysfunction (either of the native valve or of a bio-prosthesis); AVS = aortic-valve-sparing operation; AVR = aortic valve replacement; CVG = composite valve grafting; and SW = strength and weaknesses.
discuss our personal experience to comprehensively assess patient conditions which may interfere with outcomes of therapy. Table 2 displays our opportunities-and-threats matrix (OT matrix) of assessing adult MFS patients for elective surgery.

4. Use I-SWOT to establish an individualized medical strategy. The final step to an individualized medical strategy is to match strengths and weaknesses of therapeutic options with opportunities and threats related to the individual patient.

We present results from the systematic audit of strengths and weaknesses in the top row of the SWOT matrix, where we use standardized forms which allow us to prepare SWOT matrices for various aortic disease entities. Since most strengths and weaknesses relate to more than one therapeutic option, we list strengths and weaknesses in the final I-SWOT matrix regardless of specific therapeutic options. For example, in the final I-SWOT matrix we list “no oral anticoagulation” as strength “S3”, which relates to therapeutic options 1-4, or “patient-prosthesis mismatch” as weakness “W8”, which relates to therapeutic options 4 and 5 (Table 1; Figure 3). Conversely, in the left column of the I-SWOT matrix we list individualized results from the audit of patient-related opportunities and threats. For instance, good health without comorbidity may be entered into the I-SWOT matrix as an opportunity for treatment in the “health status domain”, which corresponds to domain 4 of our OT matrix (“O4”; Table 2).

I-SWOT exemplified

As the basic example we use the Marfan patient mentioned above. Imagine him to be an active cyclist in good health (O4) who had undergone surgical closure of a ventricular septal defect in childhood (T4; Figure 4). Accordingly, an SO strategy might be to perform an aortic-valve-sparing reimplantation operation according to David to maximize outcome through advanced surgical techniques (S3-5, S7), and promote the patient’s good health and participation in sports activities (O4), whereas a WT strategy might be medical treatment to minimize surgical trauma (W3-8) and minimize the likelihood of a third heart operation (T4).

<table>
<thead>
<tr>
<th>Domain</th>
<th>Opportunity or threat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Will (O1/T1)</td>
<td>Is the patients’ will clear, strong and stable or unclear, weak and fluctuating? Does the patient want to follow best medical advice or does he refuse or request specific measures?</td>
</tr>
<tr>
<td>Needs (O2/T2)</td>
<td>Does the patient have needs in his private life, profession, or spare-time activities that support or threaten therapy?</td>
</tr>
<tr>
<td>Beliefs, values, risk attitude (O3/T3)</td>
<td>Are there religious beliefs (e.g., Jehovah’s Witness), values (e.g., refusal of surgery before finishing his master degree), or risk attitudes (e.g., being particularly anxious or frivolous)?</td>
</tr>
<tr>
<td>Health status (O4/T4)</td>
<td>Is there diagnostic uncertainty about the aortic pathology or etiology of disease? Are there allergies or contraindications against BAB, ARB, ACE-inhibitors, or anticoagulants? Do certain conditions jeopardize surgical or interventional success (e.g., bleeding disorders, aortic and cardiac anatomy)? Is the patient in good health, of young age, does he have endurance, other organ diseases, or multi-morbidity?</td>
</tr>
<tr>
<td>Intellect (O5/T5)</td>
<td>Does the patient have a high or low level of education, breadth of understanding? Is he well informed, or able to understand complex courses of argument or action? Does he have restrictions of intellect such as dementia?</td>
</tr>
<tr>
<td>Psychiatric status (O6/T6)</td>
<td>Does the patient have a stable psychological status? Does he have restrictions in his behavior imposed through diseases such as depression or schizophrenia?</td>
</tr>
<tr>
<td>Sociology (O7/T8)</td>
<td>Does the patient have supportive or rather complicating social resources (health insurance, housing conditions, and economic conditions) and environment (e.g., family, friends, or profession)?</td>
</tr>
<tr>
<td>Emotions (O9/T9)</td>
<td>Does the patient have high or low trust in health professionals? Does the patient have high or low confidence in medical success and in his future prospects? Does he have an optimistic or rather a pessimistic attitude? Does the patient have high or low motivation for intervention?</td>
</tr>
</tbody>
</table>

A WO strategy might be to perform a composite valve grafting according to Bentall with a bio-aortic valve prosthesis to minimize surgical risk (W4, W6),

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but to maximize the patient’s sports opportunities by avoiding anticoagulation (O4). Finally, a ST strategy might be to perform a composite valve grafting according to Bentall with a mechanical valve to maximize therapeutic durability (S9, S10), and to minimize the likelihood of a third heart operation (T4).

We present four examples with variations in the individual attitude and character of this patient. We show how these variations influence I-SWOT based decisions, where each example relates to one of the four distinct types of strategy.

**Patient 1.** In our first example, the above mentioned patient is a highly accurate (O3) and risk-avoiding bureaucrat (T3), who might at best be convinced to undergo a mechanical Bentall operation and then to personally self-control INR values throughout his life (ST type of strategy) (Figure 4).

**Patient 2.** Imagine, the same patient to be a professional rock drummer living a “no risk, no fun” lifestyle. He might feel perfect about taking a David operation once and then get rid of major health troubles for the next couple of years (SO type of strategy).

**Patient 3.** Alternatively, the above mentioned patient is a doctor of anthropology, who cannot be dissuaded from maximizing his professional career by living with natives in a tropical moist forest for the next five years (O3). For him, a bio-Bentall minimizes the

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**Figure 4.** The use I-SWOT to establish an individualized medical strategy in a 30-year old male Marfan patient with an aortic root aneurysm (“Patient 1” as discussed in the text).
We identify specific advantages of SWOT analysis to translate evidence into individual medical strategies. SWOT analysis is a formal way to support the systematic integration of the patient and their individual issues into medical strategies. With our technique of standardizing evidence-based SW matrices of therapies, I-SWOT saves rather than costs time in the decision-making process. I-SWOT reminds us to consider four basic options of strategy rather than just a single one, which might be, for instance, only to seek exploiting the maximum of strengths and opportunities [3]. Practice guidelines may provide SW matrices of treatment options to provide standardized support for individualized medical decisions.

Moreover, I-SWOT may be used in case studies as an instrument to teach individualized medical decision-making. Finally, we believe that I-SWOT is a simple approach to holism in medical strategy, which encourages to integrate attitudes and values of both doctors and patients.

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

Conclusions

We identify specific advantages of SWOT analysis to translate evidence into individual medical strategies. SWOT analysis is a formal way to support the systematic integration of the patient and their individual issues into medical strategies. With our technique of standardizing evidence-based SW matrices of therapies, I-SWOT saves rather than costs time in the decision-making process. I-SWOT reminds us to consider four basic options of strategy rather than just a single one, which might be, for instance, only to seek exploiting the maximum of strengths and opportunities [3]. Practice guidelines may provide SW matrices of treatment options to provide standardized support for individualized medical decisions.

Moreover, I-SWOT may be used in case studies as an instrument to teach individualized medical decision-making. Finally, we believe that I-SWOT is a simple approach to holism in medical strategy, which encourages to integrate attitudes and values of both doctors and patients.

Acknowledgment
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References


Comment on this Article or Ask a Question
EDITOR’S QUESTIONS

1. How is I-SWOT different from the decision-making that a physician or team makes automatically in caring for any patient?

I-SWOT may indeed describe what thoughtful clinicians actually do during the process of decision-making. Nonetheless, we are not aware of reports on SWOT analysis for individualized medical decision-making.
I-SWOT adds important aspects to tacit or intuitive SWOT-ing: First, it provides an explicit and rational method that explains how evidence actually translates into individualized strategies; future treatment guidelines may take advantage of I-SWOT and establish SW matrices for specific goals of treatment. Second, I-SWOT allows physicians to make transparent, to discuss, to criticize and to teach where, why and how they make specific choices of strategy. Third, whereas simple-minded protagonists of EBM tend to understand variation of medical practice exclusively as a sign of malpractice, I-SWOT shows why such variation may reflect a wise physicians’ respect of his patient as an individual with an own personality. Fourth, especially for decisions debated in groups it may be helpful to agree on a common method of how to make decisions. Finally, the formalization of I-SWOT can vary: one may do it quickly and tacitly, one may use standardized I-SWOT matrix forms as shown in Figure 3 and Figure 4, and another one may even weigh all different strengths, weaknesses, opportunities and threats using score points.

2. Can you describe for us how the process of SWOT analysis proceeds in your Aortic Board meetings? Does one individual prepare an analysis before the meeting? Is the analysis prepared after the meeting? Does the team go to the literature for each case, or do they know the literature and just input their knowledge? Please help us to understand the process.

A physician presents their patient including history, medical findings, comorbidities, CT-images and so on. Then we formulate the goal of therapy and identify the options for treatment. We start establishing firm SW matrices for different goals of aortic therapy (Table 1). However, for “standard” goals of aortic therapy we do not use these matrices. In a next step, we discuss specific opportunities and threats related to the patient, where we ask questions about all domains as outlined in the OT matrix (Table 2). This works well for assessing individual medical and social issues of a patient (O4/T4; O6/T6; O7/T8). However, the conference setting imposes serious limits to the audit of complex and subjective domains of the OT matrix. Domains such as will (O1/T1), needs (O2/T2), values (O3/T3), intellect (O5/T5), and emotions (O9/T9) require an intense dialogue with the patient. Such dialogue is difficult to delegate to the physician who presents the patient. In a final step, we formulate an individualized medical strategy and designate which of our Board members actually performs therapy. This person discusses our suggestion of strategy with the patient and re-assesses the patients’ will, values and attitudes. It happens in a considerable quantity of individuals that we change strategies after intense dialogue with the patient. Then we adjust strategies and another colleague may take over the further management.

3. Do you know of other teams that use I-SWOT actively in clinical care in the aortic arena or in any other disease entity?

No. No other team uses I-SWOT with clear reference to this method. We wrote the article to inform and encourage colleagues to try out this tool: Guidelines may provide SW matrices for specific treatment goals, as mentioned. Clinical decision-makers may assess OT matrices of each patient with complex decisions where they really assess the patients’ will, needs, values, intellect, and emotions. Intense dialogue with patients is important to make best use of the potential benefits of I-SWOT. So, who uses I-SWOT “unknowingly” already and who may probably use I-SWOT in the future? Any physician who strives for medical decisions and respects the patient as a unique individual. That is why we are confident that I-SWOT will find friends outside the Hamburg Aorta Centre.
Dissecting the Dissection
Towards More Comprehensive Decision-Making Methodology for Thoracic Aortic Disease

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Abstract
Aortic dissection remains one of the most devastating diseases. Current practice guidelines provide diagnostic and therapeutic interventions based primarily on the aortic diameter. The level of evidence supporting these recommendations is Level C or “Expert Opinion.” Since aortic dissection is a catastrophic structural failure, its investigation along the guidelines of accident investigation may offer a useful alternative, utilizing process mapping and root-cause analysis methodology. Since the objective of practice guidelines is to address the risk of serious events, on the utilization of a probabilistic predictive modeling methodology, using bioinformatics tools, may offer a more comprehensive risk assessment.

Key Words
Aortic • Dissection • Bioinformatics • Predictive modeling • Clinical guidelines

Introduction
Thoracic aortic dissection remains a significant health risk. Affecting about 3–4 in 100,000 or nearly 30 cases per million per year, aortic dissection is always associated with a high risk of mortality and morbidity [1]. In light of these risks, the professional organizations involved in the regulation of cardiovascular disease care have emphasized the importance of early intervention in high-risk situations, in an effort to reduce or mitigate the adverse effects of such a devastating condition. The current clinical practice guidelines, issued jointly by these organizations in 2010, attempt to establish some basic predictive rules for patients perceived to be at an elevated risk for aortic dissection. However, the high-level (Class I) recommendations in these guidelines are based on low-level evidence (Level C or expert opinion) in most cases [2]. Furthermore, the guidelines are primarily based on a single criterion: namely, the aortic diameter. This threshold for intervention is presented as a raw data unit or as indexed to the body surface area. The language of the introductory part of the guidelines seems to suggest that aortic dilatation up to or beyond a specific diameter is associated with a higher risk for aortic dissection. This may be related to the description of the “Dissecting Aneurysm” in the literature. Notably, data from the international Registry of Aortic Dissection (IRAD) demonstrate that almost half (~46%) of cases dissect with an aortic diameter less than 5.5 cm [3].

Historical Background
The first recorded report of an acute aortic dissection was on October 1760, when the British monarch
King George II collapsed in the bathroom shortly after having his usual morning chocolate drink. Resuscitation attempts by Mr. Andrews, surgeon to the royal household, failed. Dr. Nicolls, physician to the late king, was called upon for “embalming” the body. In his postmortem examination, he noted a “transverse fissure in the ascending aorta measuring about 3.75 cm. In 1802, Maunoir described blood “dissecting throughout the circumference of the aorta” . In 1809, a necropsy report by Burns on a 56 year old male again demonstrated a tear 1.25-cm long in the ascending aorta, with an “aneurysmal sac” described as located “between the proper and cellular layers” (most likely referring to the false lumen). Similar findings were reported by Hodgson in 1815 in a 70-year-old female [4]. The first clinician to coin the term “anevrisme dis-séquant” or “dissecting aneurysm” was Laennec in 1826 [5]. This term may have influenced an assumption that all dissections are the result of dilatation of the aorta up to or beyond a critical diameter (6.0 cm);

**Structural and Functional Considerations**

The aorta is composed of multiple layers arranged in a concentric pattern, the innermost being the single-layer endothelium, resting against the basement membrane. Beyond that, the tunica media layer comprises a complex array of extracellular matrix components: collagen fibers arranged in longitudinal, circumferential and radial fashion, describing a helical pattern; elastin fibers arranged in criss-cross patterns starting at the basement membrane and enveloping the vascular smooth muscle cells with intimate contact. Elastin fibres describe two distinct circular layers: the internal and external elastic lamellae at the inner and outer boundaries of the tunica media. This layer is responsible for both structural integrity (by virtue of its “stiff” or “rigid” collagen component which can sustain considerable energy loads without stretching) as well as elasticity property of the vessel (because of its elastin content, which deforms easily in response to mechanical stress and quickly resumes its original shape as the stress decreases). In addition, there is a “fibrous skeleton” within the vascular wall, incorporating the smooth muscle cells, and is crucial for transmission of the energy load across the thickness of the wall (Figure 1). The structure, composition and configuration of the tunica media components is dictated and regulated by numerous factors, including genetic, transcriptional, enzymatic, metabolic, hormonal and humoral mechanisms that have a highly complex and interdependent pattern of influence [6, 7]. Although the vascular smooth muscle cells play a central role [8] in the regulation of the extracellular matrix and its components, these cells are themselves under the influence of numerous other factors derived from the vascular wall as well systemic components [9, 10].

One factor exerting a major influence on the vascular wall homeostasis is the process of mechano-transduction, where the cyclic changes in the intraluminal pressure with each heartbeat cause a cyclic stretch of the aortic wall. This process causes an energy load to be transmitted across the entire thickness of the aortic wall, mediated through the endothelial layers at first but more significantly through the tunica media and its elastin-derived components. The elastic property of the aortic wall, allowing it to deform then resume its original dimensions, combined with the structural strength of its predominately collagen-containing extracellular matrix, are crucial for preserving the integrity of the vascular wall in the face of the force applied to it with each pulsation. Unlike other rigid materials, the aortic wall’s extensibility is not proportional to the force applied. Rather, the modulus of elasticity (the tangential slope of the stress-strain curve) changes little at the normal ranges of pressure, but increases...
rapidly with the increasing pressure—up to a physiologic limit. It remains elevated and changes very little with intraluminal pressure above 100 and up to 200 mmHg [11-14].

The peculiar helical vorticeal pattern of flow inside the aorta causes this energy load to be exerted in three axes in the vascular wall: longitudinal, circumferential and radial. In addition, this pattern of streamlined helical flow leads to low shear stress at the endothelial level, causing the release of endothelial nitric oxide (eNO), which exerts positive remodeling effects on the vascular wall, promoting repair of the extracellular matrix components and their further maturation and organization. The impact of flow pattern on the wall stress and shear stress is crucial in understanding the genesis of different aortic disease processes. Most of the existing literature (and even textbooks) is based on older fluid dynamics studies rooted in the Windkessel model or the Poiseuille equation, where intraluminal flow is assumed to be linear, parallel coaxial flow along the longitudinal axis of the vessel, with a parabolic wavefront, with the highest velocity being at the center. Recent evidence from imaging and simulation studies has established the two-phase helical, vorticeal flow, where a vortex travels inside the vessel in a spiral fashion, with the direction of the twist reversed during diastole [15-17].

The end result of this flow-mediated homeostasis of the aortic wall is increased structural integrity and improved “flexibility” (deformability or compliance). In other words, the aorta becomes more able to withstand the energy load, distend in response to its application with each pulsation, and recoil back to its baseline dimensions afterwards without significant damage to its components [18].

Changes in any of the factors involved in these homeostatic mechanisms in the aortic wall (e.g., flow pattern, turbulence, endothelial dysfunction, genetic predisposition, signaling or transcriptional pathway anomalies, receptor disorders, extracellular matrix components, structural, or functional disorders, metabolic or toxic agents, etc.) (Table 1) will cause a shift of these mechanisms towards another pathway rather than the normal reparative one. Two alternate pathways are described:

1. A pathway where there is increased destruction, disorganization, weakening or malfunction of the tunica medica components without endothelial dysfunction, atherosclerotic or atherothrombotic processes. This results in decrease in the structural integrity and compliance of the vessel. Such aortas have a greater tendency to dilate, since their elastic recoil properties are decreased. As seen from the effects of lathyrism on animals, these vessels also are at a higher risk for spontaneous rupture (i.e., dissection) regardless of diameter.

2. Another pathway is mediated primarily by the endothelium, and the pathology is a pronounced chronic inflammatory process centered around the intima-media region, involving oxidative stress, lipid deposition and ultimately calcification. This is the classic atherothrombotic pathway (Figure 2).

Therefore, preservation of the structural integrity and elastic properties (i.e., wall stiffness and wall compliance) of the tunica media is far more significant, in terms of mechanical performance, than just the diameter. Time and again, surgeons have encountered dilated segments of the aorta without any evidence of structural wear or defect, while having to operate emergently on normal-sized aortas that have disintegrated because of their fragility. Several animal and human studies have documented dissection of normal-sized aortas upon exposure to certain substances (sometimes known as lathyrogens) [19].

**Dissection as a catastrophic structural failure:**

The aorta, in engineering terms, is designed to sustain a certain mechanical load in order to transfer a specific volume of blood per unit of time, while maintaining the blood volume inside the vessel at all times. A useful parallel of this process is the function of a fluid pipe, which must withstand the pressure of the fluid inside to safeguard its flow to its destination.

Aortic dissection is, therefore, a failure of the aortic wall to sustain the energy load that is applied to it with each heartbeat, allowing the blood to escape outside its lumen and—of course—impair its transport function. It is, thus, similar to the rupture of a water pipe, which is defined in civil engineering terms as “gross movement of major components of a
Table 1. Factors influencing the composition and structural integrity of the aortic wall.

<table>
<thead>
<tr>
<th>(a) Aortic wall factors</th>
<th>(b) Blood Flow-Aortic Wall /interactions</th>
<th>(c) Patient Factors:</th>
</tr>
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<tbody>
<tr>
<td>Endothelial Function (intima)</td>
<td>Diameter (surface area)</td>
<td>Age</td>
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<tr>
<td>Integrity</td>
<td>Resting intraluminal pressure</td>
<td>Growth phase</td>
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<td>Trauma</td>
<td>Cyclic pressure change; total energy load; energy loss</td>
<td>Growth hormone replacement</td>
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<tr>
<td>Adhesion molecules (vCAM, VAP-1)</td>
<td>Heart rate (frequency of energy load)</td>
<td>Pro-apoptotic gene expression</td>
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<td>Endothelial nitric oxide</td>
<td>Ventricular systolic function and ejection (initial force generation)</td>
<td>VAP-1</td>
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<td>Subintimal Extra-Cellular Matrix</td>
<td>Aortic valve morphology and competence (flow wave morphology)</td>
<td>Gender</td>
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<td>Gene mutation, expression and transcription</td>
<td>Blood volume</td>
<td>Sex-specific conditions</td>
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<td>Collagen content</td>
<td>Blood viscosity</td>
<td>Gonadal function (endogenous sex steroids)</td>
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<td>Collagen organization</td>
<td>Humorally active blood components: platelets, circulating macrophages</td>
<td>Hormonal replacement therapy</td>
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<td>Elastic Lamellae</td>
<td>Integrity of elastin-fibrillin-smooth muscle cell &quot;scaffolding&quot; structure</td>
<td>Estrogen and progesterone</td>
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<td>Gene mutation, expression, and transcription</td>
<td>Aortic stiffness (&quot;Young's modulus&quot;)</td>
<td>Oral contraceptives</td>
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<td>Elastin content</td>
<td>Extent of mechano-transduction:</td>
<td>Pregnancy and peripartum period</td>
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<td>Elastin fiber organization and maturation</td>
<td>Signaling pathways</td>
<td>Cardiovascular Risk Factors</td>
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<td>Integrity of fibrillin &quot;bridges&quot;</td>
<td>Downstream gene expression</td>
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<td>Vascular Smooth Muscle Cells</td>
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<td>Differentiation</td>
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<td>Gene mutation, expression and transcription</td>
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<td>Extracellular matrix regulatory function</td>
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<td>Migration across elastic lamellae</td>
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<td>Interaction with macrophages</td>
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<td>Integrity of actin bridges</td>
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<td>Myosin heavy chain (MYH) integrity</td>
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<td>Contractile function</td>
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including manufacturing, industry, aviation and recently healthcare, this methodology aims at identifying the factor(s) directly responsible for causing the deviation from normal performance. In other words, identifying what caused the error to occur [23].

The fundamental first step in root cause analysis is a comprehensive understanding of "what should happen". This describes the Process Mapping; a procedure where the expected course and sequence of events is plotted on a central timeline from beginning to end, with every factor that is influencing this process plotted as a side arrow intersecting the process line central pathway at an angle. The process map has gained considerable popularity, as it provides a simple, systematic graphic representation of all the factors influencing the pathway and end-result of any given process. Graphically plotting the different factors involved in effecting a certain result permits us to more adequately evaluate the specific point where this anticipated effect did not take place, and for which reason. For instance, a missing or malfunctioning component in an assembled product can be traced to the point of its insertion and/or testing, structural system that renders them incapable of supporting the intended loading."

Since aortic dissection is a catastrophic structural failure, utilizing the principles of accident investigation may be a helpful approach towards understanding its genesis and, therefore, predicting the risk of its occurrence.

The first principle of any accident investigation is the definition of failure. The American Society of Civil Engineers’ Council on Forensic Engineering [20] provides this simple yet comprehensive definition: “An unacceptable difference between the expected and observed performance.” In other words, failure is a serious deviation from “what should happen” to “what should not happen.” This echoes the often cited definition of an error by James Reason: “Occasion in which a planned sequence...fails to achieve its intended outcome” [21, 22].

The process to better understand the reason why and how failure occurs, leads us to the second principle of accident investigation: Root Cause analysis. Now established as a standard methodology in investigating accidents and errors in numerous fields, including manufacturing, industry, aviation and recently healthcare, this methodology aims at identifying the factor(s) directly responsible for causing the deviation from normal performance. In other words, identifying what caused the error to occur [23].

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As described earlier, aortic dissection is a grave imbalance between two opposing forces: structural strength and energy load. These describe the two main processes impacting the aortic wall. These are akin to the tension and compression forces impacting the safety of a bridge. The energy load applied to the aortic wall with pulsatile flow is counteracted by the tension forces generated in the three axes within the aortic wall.

Thus, the process map for aortic dissection may be graphically represented by a central pathway impacted thereby identifying the “root cause” of the failure of that specific component. One well-publicized example of this has been the investigation of the O-rings in the space shuttle disaster by NASA.

The resulting Ishikawa diagram, after who first described it, is also commonly known as the “fishbone”, “herringbone” or even “fishikawa” diagram and has become one of the Seven Basic Tools of Quality in any field (Figure 3). Because of its adaptability and room for expansion, it can be applied to simple as well as complex processes, making it a very versatile and widely applied tool [24-27]. One of the most important features of the Ishikawa model for process mapping is the ability to identify all factors impacting any given process, no matter how “insignificant” they might seem. Lessons from the NASA “Threat and Error Model” [28] and the “Swiss Cheese” model spotlight the importance of small deviations at points further away from the central process that precipitate an “unintended state” affecting the next several steps in the process. If the effect of these unintended states is not reversed or counterbalanced at earlier points in the process, the deviation is transmitted farther downstream, creating an “unintended state” which is further amplified by the other factors involved and ultimately causing a major deviation from the expected result. The failure to identify the seemingly insignificant detail of the temperature range change on the performance of the O-ring—for instance—has “snowballed” into the catastrophic structural failure in the space shuttle.
by two side arrows, one representing the structural integrity of the vessel wall, while the other represents the energy load applied to the aorta. This produces two intersecting and inter-dependent Ishikawa diagrams, because each of these two processes itself is dependent on multiple upstream factors (Figure 4).

Since each result in this diagram is dependent on a number of other conditions “upstream,” the occurrence of one event (e.g., proper elastin organization) is dependent on the likelihood of the presence of some other factors (e.g., gene mutation, ligand activity, pharmacologic agents, etc.) This pattern of dependence describes an “Influence Diagram” of conditional probability. In such arrangement, the probability of one event is dependent on the presence of a number of antecedent events; each—in turn—is dependent on the presence of another set of prerequisites. The resulting arrangement is usually referred to as a Bayesian decision tree, where every event is dependent on the interaction of two or more preceding factors and/or events. Generally speaking, representing the probability of each condition is done either by a Boolean assignment of value in terms of 1/0 (i.e., true or false) or in terms of its probability of being true at the time of examination, that is, fractions of one or percentages.

Guidelines and Decision-Making Methodology

Over the centuries, the practice of medicine has evolved from a personality-based decision making model to an evidence-based, data-driven decision making model. Because of the increasing emphasis on patient safety and error prevention, practice guidelines have emerged in recent decades as decision support tools to ensure the identification of high-risk situations and the prompt and appropriate implementation of interventions to reduce the risk of death or harm to patients [30]. Consequently, and by definition, practice guidelines are predictive models based on risk stratification. Since risk is defined as the probability of an error or an accident, the implementation of a probabilistic approach to decision-making becomes more attractive. Drawing on principles
of medical triage, the inclusion of a prognostic approach, stratifying the possible outcomes according to their expected severity and impact on survival and quality of life, becomes a much desirable feature of any comprehensive set of practice guidelines.

Since virtually all biologic systems are complex environments, involving numerous inter-dependent factors (many of which are unknown or poorly understood) exerting their influence in a complex, interconnected and highly variable pattern, the use of classic rule-based decision-making tools becomes inappropriate [31]. Rule-based decision making (e.g., IF X THEN Y) assumes that probability of dissection is entirely dependent on the probability of size; that is, \( p(D) = p(s) \):

- A high degree of predictability, where \( X \) will always be true all the time
- A high level of causality, dependence and linear relationship between \( X \) and \( Y \)
- A high degree of certainty about the effect of \( Y \) on \( X \)
- A high degree of certainty that only \( X \) is responsible for the issue being examined

The previous discussion is illustrative that these assumptions may be inappropriate in a complex environment such as the aorta [32]. Therefore, the simplistic rule-based decision-making dictating that "IF diameter is 6 cm THEN operate" does not take into account the influence of other factors contributing to the structural integrity of the vascular wall, such as collagen gene expression, ATR-1 receptor function and its impact on the vascular smooth muscle cell regulation of the extracellular matrix, the degree of collagen maturation and organization within the aortic media. Nor do they reflect the impact of the different flow patterns in trileaflet versus bicuspid aortic valves, for instance, on the location, degree and effect of energy load on the aortic wall. Similarly, there remain countless other unrecognized or poorly understood factors (such as chromosome X gene functions, vascular adhesion protein-1 function, impact of growth hormone and its interaction with other components of the vascular wall, specific properties of each segment of the aorta, etc.) which seem to have some degree of influence on the structural integrity of the vascular wall, based on observational and/or anecdotal reports.

Given the complex, variable inter-dependent patterns and high degree of unpredictability of these factors, the central question in developing a decision-making algorithm becomes "what is the probability of aortic dissection \( p(D) \) given factor 1, and factor 2, and factor 3, etc.?" In other words, what is the probability of aortic dissection \( D \) given condition \( C_1, C_2, C_3 \ldots C_n \).

Mathematically speaking, this is a composite function where the probability of \( D \) is dependent on the probabilities of \( C_1, C_2, C_3 \ldots C_n \) etc. [33-37]. The formula can be expressed as follows:

\[
p[D] = p[C_1] \times p[C_2] \times p[C_3] \times \ldots p[C_n]
\]  

(1)

The goal, then, becomes to be able to adequately solve this equation and properly assess the probability (i.e., risk) of aortic dissection in a specific patient. In other words, 'tailoring' the recommendations for management of aortic disease towards developing patient-specific guidelines, based on the calculated risk of the disease event versus the risk of surgery.

Expressed in clinical terms, the question becomes: What is the predicted likelihood (i.e., risk) of aortic dissection occurring in a 32-year-old infertile, diabetic female of a specific ethnic background, of normal height, with no history of smoking or illicit drug abuse, but has a history of pre-hypertension, mild skeletal problems with normal skin elasticity, bicuspid aortic valve and an aortic diameter of 4.7 at the mid-ascending segment, etc.?

**Conclusions**

The mission of the governing and regulatory bodies in medicine remains the promotion of scientifically sound practices to reduce harm from disease and injury as well as from medical practice. Based on this, practice guidelines are developed, circulated and their implementation encouraged toward these goals of promoting the safety of the public and the prevention of serious events in the course of their lives.

The primary objective for the guidelines for management of thoracic aortic disease appropriately remains the prevention of catastrophic events; namely aortic dissection or rupture. The assignment of a high-level recommendation requires a comparatively
high level of confidence (i.e., a high index of suspicion) about the probability of an event to occur, as well as the severity of its impact. In some cases, these guidelines have relied on a limited set of data (the so-called Expert Opinion) which has been derived from anecdotal evidence, limited observational studies or “personal experience” to develop high-level recommendations for clinicians. Given the gravity of the event and its serious impact on the survival, quality of life and healthcare resource allocations, the current discrepancy between the level of evidence and the level of recommendation calls for an improvement of the derivation of the clinical guidelines and an opportunity to better quantify “clinical experience” or “clinical sense.”

Because of their suitability to offer a more comprehensive prediction of the conditional probability of a specific event, and also having the capability for developing a hierarchy of prognostic triage levels based on the expected outcomes, probability-based bioinformatics tools, rooted in the Bayesian principle [37-38], can offer a useful, dynamic and adaptable platform for developing such improved guidelines.

The challenge in designing and implementing such predictive models remains the categorization and classification of data sets; optimally national, longitudinal disease-specific data sets. Transdisciplinary collaboration is crucial for acquisition of such large, diverse and comprehensive data sets. Given the nature of the clinical problem, randomized controlled studies are not feasible; and standard statistical methodology is severely limited. However, careful analysis of observational data can yield important insight into the probability of such catastrophic events.

The development of a specific, dedicated inter-societal task force for a more comprehensive study of the outcomes of different strategies for atrial fibrillation ablation has established a most commendable example. Establishing a national registry incorporating all miniature details of the patient characteristics, disease course characteristics as well as procedural details has provided us with an invaluable and huge data set for robust analysis.

Large data repositories such as national registries, society registries, disease-specific registries and electronic medical records do hold a considerable promise for robust data gathering, which will be conducive to a more comprehensive predictive analysis and modeling. This holds the promise of providing a more adequate prediction of the risk for aortic dissection and hence a better tailoring of the diagnostic and therapeutic recommendations for such patients.

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Conflict of Interest

The author holds a full United States Patent for a Total Aortic Arch Reconstruction Graft. There are no financial disclosures.

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Bioinformatics for Aortic Dissection Risk

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Rare Case of Vaginal Delivery in Giant Aortic Aneurysm

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Abstract

A 33-year-old woman underwent successful vaginal delivery despite previously unsuspected 8-cm ascending and 6-cm descending aortic aneurysms. These were repaired immediately after delivery.

It is well known that aortic dissection is a serious problem during pregnancy due to increased risk of maternal death [1]. In some congenital and genetic disorders (Marfan syndrome, bicuspid aortic valve, aortic coarctation), the natural history of the disease is well-known. However, there are a number of congenital heart diseases with aortic dilatation that are not well characterized. We present here a rare case of successful vaginal delivery in a patient with large ascending and descending aortic aneurysms, previously unsuspected.

A 33-year-old woman was admitted to the hospital 20 days after her first vaginal delivery from an outside hospital. She complained of chest pain during and after delivery. The patient had no family history of aortic disease or known connective tissue disorders.

On examination, there were no signs of genetic disorders such as Marfan syndrome, or Ehlers-Danlos syndrome. On echocardiography an aneurysm of the ascending aorta was found, with severe aortic regurgitation. The aortic valve was tricuspid. On computed tomography (CT) scan, the aneurysms of ascending and descending aorta were identified, with maximum size of the ascending aorta of 81 mm and maximum size of the descending aorta of 61 mm. There were no signs of aortic dissection (Figure 1).

The patient underwent a Bentall-De Bono procedure with the Kouchoukos modification with hemiarch reconstruction. Histology of aorta showed cystic medial degeneration with pseudocyst formation in the media, accompanied by extensive loss of elastic lamina. On control CT before the second stage of operation which was performed in 6 months time, enlargement of descending aorta and aortic dissection were found (Figure 2). The dissection of the descending aorta developed after Stage 1 of the operation and there were no signs of dissection on initial scans. The size of the aorta enlarged dramatically from 61 mm to 67 mm. Endovascular treatment of the descending aorta with Valiant Thoracic Captiva stent graft was performed. The stent graft did not cover...
any of the great vessels. On control CT performed 6 months after the procedure there were no signs of deterioration (Figure 3).

This is a unique case of vaginal delivery in a woman with giant aortic aneurysm that was not complicated by dissection during delivery. It is well known that aortic aneurysms in young patients are usually associated with connective tissue disorders such as Marfan syndrome, or bicuspid aortic valve. We presume that aortic dissection in this patient was part of the natural history of non-syndromic familial thoracic aortic aneurysm. In the 2014 European Society of Cardiology Guidelines on the diagnosis and treatment of aortic diseases, a new section on non-syndromic familial thoracic aortic aneurysm and dissection was included [2]. Numerous etiologic mutations have been identified. Routine echocardiography is not recommended...
for all pregnant women without previous cardiac medical history. That is most likely the reason why in this patient the aortic enlargement was undetected due to lack of family history of aortic disease.

**Conflict of Interest**

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

**Figure 3.** After Stage 2, a CT image shows a well-positioned stent-graft in the descending aorta.

**References**


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Upcoming Meetings

AORTA, June 2015, Volume 3, Issue 3: 121
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List of Upcoming Meetings

January 2016

1. Controversies and Updates in Vascular Surgery
   January 21-23, 2016
   Paris, France
   Meeting information available at:
   www.cacvs.org

2. Society of Thoracic Surgeons 52nd Annual Meeting and STS/AATS Tech-Con 2016
   January 23-27, 2016
   Phoenix, Arizona
   Meeting information available at:
   www.sts.org/annualmeeting

February 2016

1. Aortic Valve Repair: A Step by Step Approach
   L’Institut Mutualiste Montsouris
   February 4-5, 2016
   Paris, France
   Meeting information available at:
   www.caviaar.com

2. 62nd Annual Meeting of Indian Association of Cardiovascular and Thoracic Surgeons
   February 18-21, 2016
   Lucknow, UP, India
   Meeting information available at:
   www.iactsconlucknow2016.org

3. University of Virginia 8th Annual Cardiac Symposium: Debates and Controversies in Cardiovascular Disease
   Wintergreen, VA
   Meeting information available at:
   www.cmevillage.com

4. 34th Cardiovascular Surgical Symposium (CSS)
   February 27-March 5, 2016
   Zurs, Austria
   Meeting information available at:
   www.surgery-zurs.at

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