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- 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
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- Occlusion of device or native vessel
- Pulmonary complications and subsequent attendant problems
- Renal complications and subsequent attendant problems (e.g., artery occlusion, contrast toxicity, insufficiency, failure)
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Thursday, October 9, 2014
7:00 AM – 4:30 PM

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The History of Deep Hypothermic Circulatory Arrest in Thoracic Aortic Surgery

Lara Rimmer, MBChB, Matthew Fok, MBChB, Mohamad Bashir, MD, MRCS*

Thoracic Aneurysm Service, Liverpool Heart and Chest Hospital, Liverpool, United Kingdom

Abstract
Depending on the extent of aortic disease and surgical repair required, thoracic aortic surgery often involves periods of reduced cerebral perfusion. Historically, this resulted in detrimental neurological dysfunction, and high risk of mortality and morbidity. Over the last half century, rapid improvements have revolutionized aortic surgery. Among these, deep hypothermic circulatory arrest (DHCA) has drastically reduced the risk of mortality and morbidity following surgery on the thoracic aorta. This progress was facilitated by experimental pioneers such as Bigelow, who studied reduced oxygen expenditure consequent on induction of hypothermia in dogs. These encouraging findings led to trials in human cardiac surgery by Lewis in 1952 and further made possible the first successful aortic arch replacement by Denton Cooley and Michael De Bakey. Modern day surgery has come a long way from the use of immersion of the patient in ice baths and other primitive techniques previously described. This paper explores the development of deep hypothermic circulatory arrest from its origins to the present.

Key Words
Deep hypothermic circulatory arrest · Aortic aneurysm · Thoracic aortic surgery · History · Cerebral protection

Introduction
“History Teaches Everything Including the Future”—Alphonse de Lamartine, 19th Century French Poet

The first reported use of hypothermia as a therapeutic intervention dates to the Hippocratic era, from the Hippocratic School of Medicine in the 4th century BD [1,2]. Denton A. Cooley, in his inaugural article in this journal, AORTA, on the history of aortic aneurysm surgery, described aortic disease as a fundamental problem of mankind [3]. Depending on the anatomic involvement of aortic disease, a surgical repair may mandate periods of reduced cerebral and visceral perfusion. Historically, such interventions were coupled with harrowing rates of death, and led to neurological complications and major visceral organ dysfunction. These facts led pioneering surgeons and researchers to dedicate their lives in the search for methods to reduce such outcomes. Among the advances is hypothermia, a technique that is now employed in a wide range of situations, including post cardiac arrest, aortic arch and thoracoabdominal surgery, and specialized cerebrovascular surgery. DHCA requires the use of cardiopulmonary bypass and cools the body to lower degrees than standard hypothermia alone [4]. Its modern day use resides mainly in cardiothoracic surgery (and neurosurgery) as a mechanism of cerebral protection. Its use has dramatically reduced the incidence of neurological damage following aortic surgery [5,6].

This review will elicit key milestones in the development of deep hypothermic circulatory arrest (Table 1).

Deep Hypothermic Circulatory Arrest

The first reported use of hypothermia as a therapeutic intervention dates to the Hippocratic era, from the Hippocratic School of Medicine, where it was described as a treatment for tetanus [1]. Hippocrates himself pro-
moted the use of snow and ice packed around the injured soldier to promote healing [7]. In 1812, Dominique Larrey, surgeon to famous military leader Napoleon, used ice to alleviate injured soldiers’ pain during amputations [8]. Despite a history of well over two thousand years, hypothermia did not gain popularity until the 21st century. In modern medicine, mild therapeutic hypothermia is widely used post cardiac arrest with return of spontaneous circulation in an effort to reduce the incidence of neurological damage [9]. DHCA is, however, reserved for aortic surgery and cerebral vascular surgery as a method of cerebral protection [2,5].

The birth of hypothermic cooling techniques for use in cardiac surgery began in earnest with the work of William Bigelow (Fig. 1) [10]. Today, Bigelow is famous for writing two books, including one called “Cold Hearts.” He is further recognized for his role in the development of the pacemaker. He was awarded the title of Officer of the Order of Canada, the second highest honor of merit awarded by the Queen to civilians, and he was inducted into the Canadian Medical Hall of Fame in 1997.

In 1950, a research team in Toronto led by Bigelow published their two years’ work on oxygen uptake and expenditure in canines at temperatures considered hypothermic [10]. They hypothesized that a reduced body oxygen requirement could be achieved through a reduced metabolic drive secondary to hypothermia. This in turn would enable the heart to be excluded from circulation and allow the possibility of cardiac surgery. Their work was published before the invention of cardiopulmonary bypass by John Gibbons, who began clinical application of his heart-lung machine in 1952 [11]. Bigelow’s experiments encompassed 176 dogs, who were cooled with the aid of muscle relaxants to control homeostatic temperature regulation resulting in severe shivering, venesection as a method of reducing pressure within the venous system, and phrenic nerve stimulation to induce artificial respiration. Bigelow was able to successfully exclude the

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Table 1. Key Innovators and Their Contributions to DHCA

<table>
<thead>
<tr>
<th>Year</th>
<th>Contributor</th>
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<tr>
<td>4th century BD</td>
<td>Hippocrates (Greece)</td>
<td>Use of snow to cause hypothermia to aid healing</td>
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<tr>
<td>1812</td>
<td>Larrey (France)</td>
<td>Use of ice on injured soldiers</td>
</tr>
<tr>
<td>1950</td>
<td>Bigelow (Canada)</td>
<td>Canine and Rhesus monkey experiments on hypothermia to reduce body oxygen requirements to allow exclusion of the heart from circulation</td>
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<tr>
<td>1950s</td>
<td>Gollan (Czechoslovakia/US)</td>
<td>Use of an oxygenator with a heat exchanger to cause hypothermia and rewarming in animals</td>
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<tr>
<td>1952</td>
<td>Lewis (US)</td>
<td>Performed the first successful human open-heart surgery to close an atrial septal defect using cooling blankets to induce hypothermia</td>
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<td>1953</td>
<td>Swan (US)</td>
<td>Experimented with hypothermia further, and used this knowledge to the success of his first open-heart surgery. Swan went on to use this on hundreds of patients, with low mortality</td>
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<tr>
<td>1955</td>
<td>Cooley (US)</td>
<td>First use of hypothermia for cerebral protection during first aortic arch aneurysm repair with a homograft</td>
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<tr>
<td>1955</td>
<td>Lillehei and Kirklin (US)</td>
<td>Noticed and published that better outcomes occurred when body temperature cooled spontaneously during oxygenation</td>
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<td>1959</td>
<td>Sealy (US)</td>
<td>Continued Lillehei and Kirklin’s development and added a heat exchanger to a DeWall oxygenator to use hypothermia alongside it</td>
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<tr>
<td>1959</td>
<td>Drew (England)</td>
<td>First employed his own technique to use the patient’s lungs instead of an oxygenator alongside hypothermia, and went on to apply this throughout his surgical career</td>
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<tr>
<td>1960s</td>
<td>Meshalkin (Russia)</td>
<td>Used ice and snow to operate without cardiopulmonary bypass</td>
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<td></td>
<td>Delorme (Scotland) and Boerema (Holland)</td>
<td>Developed methods of cooling in patients through experiments on dogs, passing the cannulated blood through an ice bath and returning it through a vein</td>
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<td>1959</td>
<td>Ross and Brock (England)</td>
<td>Popularized using hypothermia alongside cardiopulmonary bypass</td>
</tr>
<tr>
<td>1963</td>
<td>Barnard and Schrire (South Africa)</td>
<td>First used DHCA and CPB at the same time on an ascending and arch aortic aneurysm</td>
</tr>
<tr>
<td>1975</td>
<td>Greipp (US)</td>
<td>Used surface cooling with CPB to resect aortic arch aneurysms in four patients</td>
</tr>
</tbody>
</table>

DHCA, deep hypothermic circulatory arrest; CPB, cardiopulmonary bypass.
heart from circulation without arrest at a core temperature of 20°C in 39 of his dogs. Of these 39, 51% of dogs were successfully revived. Cardiac arrhythmias, particularly ventricular fibrillation, were the major hindrance in the dogs who were successfully revived [10].

In these early experiments, a common theme was to avoid ventricular fibrillation or at least to correct it as soon as it developed. We must remember this, as in the current era of cardiopulmonary bypass, we are immune to the impact of ventricular fibrillation, which is expected as part-and-parcel of deep hypothermia.

Despite these promising advances, the team continued to search for better methods of cooling. They knew that a hibernating mammal, such as the groundhog, could survive a temperature of 3°C. They wished to reduce the current limit of 20°C [10,12]. The team performed further research on Macacus Rhesus monkeys, once again using cooling blankets, this time to below 20°C [12]; 11 of 12 monkeys cooled to temperatures between 16 and 19°C survived between 15 and 24 minutes. Whereas in previous experiments on dogs, at which their respirations ceased around 24°C, monkeys continued to respire at 8 respirations per minute at 20°C. Similarly, Bigelow et al. used groundhogs cooled below 5°C (as in their natural hibernating state), operated, and successfully revived 5 of 6 animals [12].

A physiologist named Frank Gollan worked in the 1950s using hypothermia and an oxygenator of his own invention, and presented his work in 1955 [13–15]. Gollan made an important step in that his bubble oxygenator included a heat exchange device, whereby he could induce hypothermia as well as carry out rewarming [16]. He was able to achieve measured core temperatures of 4°C and published revival of the animals. Despite this, his research was not widely recognized and was largely ignored among the surgeons at the American Association for Thoracic Surgery [17]. In Sweden, Juvenelle and colleagues were also coming to conclusions similar to Gollan’s—specifically, that the use of a pump-oxygenator and hypothermia of 12°C would decrease oxygen requirements of the body to allow open cardiac operating times of up to two hours without adverse consequences [17,18]. However, Juvenelle’s method produced little in the way of long-term survival [18].

The first successful human operation utilizing a period of hypothermia was performed in 1952 at the University of Minnesota by Dr. John F. Lewis [19]. Armed with knowledge of William Bigelow’s experiments on hypothermia, and his own extensive experiments involving several hundred canines, he was successful in closing a secundum atrial septal defect in a 5-year-old girl [20]. For two hours he wrapped the anesthetized patient in refrigerated blankets until her rectal temperature had fallen to below 28°C. Lewis
describes the operation in his landmark paper [20]. “The chest is opened with a transverse, sternal splitting incision through the 4 interspaces, the heart is explored digitally through the right auricular appendage. Cardiac inflow and outflow are occluded and the right atrium is opened widely to allow repair. The left and right heart are filled with saline and atrium closed”. Following the operation, the patient was placed in hot water at 45°C to increase her rectal temperature to 36°C. This operation is heralded in cardiac history as the first ever successful operation within the open human heart under direct vision.

Subsequently, Lewis used this technique on 29 more patients, with only three deaths. Without cardiopulmonary bypass, hypothermia still carried an inherent risk of ventricular fibrillation, which remained a significant danger of hypothermia, previously noted by Bigelow et al. and Lewis et al., who used cardiac massage, intracardiac adrenalin, and electrical shock successfully in more than 90% of patients for restoration to normal sinus rhythm [12,20]. The success of Lewis’s operations gained worldwide medical recognition, and they represent a major milestone in cardiac surgical history. However, with the introduction of the cardiopulmonary bypass machine, the sole use of hypothermia as a technique to allow intracardiac operations was short-lived, in view of the limited operating time this technique provided and the associated complications it carried.

In 1953 came Henry Swan, who had repeated the work of Bigelow to investigate impact on the variables of pH, serum sodium, chloride, potassium, phosphorus, plasma protein, and hematocrit under the influence of hypothermia, with particular interest in prevention of the well documented complication of ventricular fibrillation [21]. On February 19th of the same year, Swan carried out open-heart surgery, a pulmonary valvulectomy, using hypothermia for the first time [8,21]. Swan then applied these findings in a surgical setting, prior to the use of cardiopulmonary bypass, using 26-28°C hypothermia on hundreds of patients, with a low mortality rate [8]. As such, Swan was considered to have the most surgical experience using hypothermia.

The renowned surgeon Dr. Denton Cooley employed hypothermia for cerebral protection during his first attempt at total resection and replacement of the aortic arch in 1955 [22,23]. The 49-year-old gentleman was suffering from a syphilitic aneurysm involving the arch and a further aneurysm affecting the descending aorta. Cooley used surface cooling to achieve 33°C, and temporary shunts were placed to provide blood to the carotids and distal aorta. In this case, the patient went on to suffer a stroke and then death, although this was attributed to an 8 minute occlusion of the right carotid shunt. This case not only represented the first ever aortic arch resection and replacement, but exploited the use of hypothermia as an adjunct in aortic arch surgery, as it is still used today.

Further, important developments in 1955 were introduced by Lillehei and Kirklin, who used the pump-oxygenator for intracardiac surgery [24]. During their operations, it was noticed that body temperature would often cool spontaneously, as early oxygenators lacked heat exchangers. In this way it was noted that allowing spontaneous cooling alongside pump-oxygenators could produce better outcomes. Lillehei and Kirklin published their successful work, and their techniques became fashionable [17].

Sealy et al. fronted this development throughout the late 1950s and, in 1959, were the first to add a heat exchanger alongside a DeWall oxygenator [17,25]. This allowed rapid active cooling and rewarming of patients to a temperature of 32°C [25]. They confirmed the compatibility of using hypothermia alongside an oxygenator. Sealy reported this technique for 95 patients in a variety of open cardiac surgeries including tetralogy of Fallot, complete transposition, valvular disease, septal defects, and reported a mortality rate of 17% [26].

From 1959 onwards, Charles Drew, a surgeon at the London Westminster Hospital, began developing his own methods for intracardiac surgery after disappointing results using other popularized methods [17]. Drew first used his technique, developed through experimentation on dogs, on a 1-year-old child with Down’s syndrome in congestive heart failure from an endocardial cushion defect, although the child later died after recovery. He successfully repaired his following two patients, who underwent ventricular septal defect (VSD) closure, and they recovered without complications in 1959 [27]. His technique involved a circulatory support system to cool patients to 15°C. His cynicism toward oxygenators led to using the patient’s own lungs for oxygenation. This technique was gradually advanced to children and adults across a career of 22 years, with varying degrees of success. But, eventually this technique lost ground [17]. Drew’s work represented a cardinal contribution to today’s knowledge of hypothermia, and this was recognized in 1961, when Drew was invited to present the
renowned Hunterian lecture at the Royal College of Surgeons.

In Siberia, Professor E. N. Meshalkin, who is credited as the pioneer of Soviet cardiac surgery, used hypothermia during the 1960s on a variety of patients. He was notorious for operating on congenital defects without cardiopulmonary bypass, with only mild hypothermia [28]. It is documented that Prof. Meshalkin’s method of cooling was the utilization of the abundance of snow and ice available in Siberia for surface cooling [29].

Meanwhile, methods of cooling were being advanced by Delorme and Boerema via the insertion of a cannula into the femoral artery of canines, passing the blood through an extracorporeal coil immersed within an ice bath, and returning the cooled blood through the femoral vein [30,31]. They were both able to cool canines to 22 to 26°C (except in 1 dog) without causing fibrillation, a dreaded complication of surface cooling. armed with this knowledge, Delorme concluded that operating on a bloodless field would become possible in cardiac surgery. However, arteriovenous cooling was subject to complications, including fistula formation and thrombosis. Furthermore, the technique required initiation before surgery.

Mr. Donald Ross, well known for leading the team that performed the first cardiac transplant in the United Kingdom, is further credited with popularizing venovenous cooling, a method he had devised in canine experimentation by cannulating the external jugular vein for blood drainage and providing return through the superior vena cava [32,33]. He reported improved success using venovenous cooling over surface cooling due to the greater control over the stages of cooling, preventing core temperatures from dropping too low, or too rapidly, reducing the risk of cardiac irregularities. The recognized detrimental effect of ventricular fibrillation from rapid cooling was still a very real risk. Ross did not commence cooling until the chest was open and, thus, was able to observe the heart and deal with any irregularities.

In 1959, Ross and Sir Russel Brock, from Guy’s Hospital London, declared “deep hypothermia by means of a heart lung bypass machine or a differential cooling technique holds promise of longer safe periods of intra cardiac surgery in the future” [34]. Following this pronouncement, there was a wave of experiments in the use of cardiopulmonary bypass with hypothermia.

World renowned Dr. Christiaan Neethling Barnard and Velva Schrire in 1963 were the first to use deep hypothermic circulatory arrest and cardiopulmonary bypass simultaneously, on two patients with aortic aneurysm involving the ascending aorta and arch. They cooled the patients to a temperature of approximately 10°C measured in the esophagus [35]. They were successful in one of their patients. Following this, multiple renowned surgeons began reporting success with combinations of hypothermia and cardiopulmonary bypass, including Borst and Lilliehei.

In 1975, Professor Randall Griep published a series of four patient operations for aortic arch aneurysms using hypothermia via a combination of surface cooling and cardiopulmonary bypass [36]. He published successful resection of aneurysms in all four patients. Griep et al. would later (1991) report the limitations of using hypothermic circulatory arrest alone for cerebral protection, noting a relationship between duration of hypothermic arrest and mortality [37,38].

These concerns were echoed by Haldenwang et al. [39], who noted that temporary or permanent neurological dysfunction incidence rose when HCA exceeded 40 minutes and mortality rates increased above 60 minutes of HCA. It was this observation that led to the development of further techniques: antegrade cerebral perfusion (ACP) and retrograde cerebral perfusion (RCP), which are used in combination with DHCA today.

**Summary**

History surrounds cardiac surgery in the form of many successes and failures of pioneering individuals in the face of difficult challenges. The past fifty years have been monumental for the refinement of aortic surgery and decreasing the risk of patient mortality and neurological deficit. DHCA started from small laboratory experiments, to the use of ice baths, and is still used in aortic surgery today (Fig. 2). The relative infancy of deep hypothermic circulatory arrest means the near future is extremely promising and will surely hold many new exciting developments and innovations within this field.

**Conflict of Interest**

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


Hepatopancreaticobiliary Values after Thoracoabdominal Aneurysm Repair

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Abstract

Background: After thoracoabdominal aortic aneurysm (TAAA) repair, blood tests assessing hepatopancreaticobiliary (HPB) organs commonly have abnormal results. The clinical significance of such abnormalities is difficult to determine because the expected postoperative levels have not been characterized. Therefore, we sought to establish expected trends in HPB laboratory values after TAAA repair.

Methods: This 5-year study comprised 155 patients undergoing elective Crawford extent II TAAA repair. In accordance with a prospective study protocol, all repairs involved left-sided heart bypass, selective visceral perfusion, and cold renal perfusion. Blood levels of aspartate transaminase (AST), alanine transaminase (ALT), γ-glutamyl transpeptidase (GGT), lactate dehydrogenase (LDH), total bilirubin, amylase, and lipase were measured before TAAA repair and for 7 days afterward. Ratios between postoperative and baseline levels were compared for each time point with 95% confidence intervals.

Results: Temporal patterns for the laboratory values varied greatly. Amylase, lipase, and AST exhibited significant early increases before decreasing to preoperative levels. LDH increased immediately and remained significantly elevated, whereas ALT increased more gradually. GGT remained near baseline through postoperative day 4, and then increased to more than twice baseline. Total bilirubin never differed significantly from baseline. After adjusted analysis, the ischemic time predicted the maximum AST, lipase, GGT, and LDH values.

Conclusions: Although most HPB laboratory values increase significantly after elective TAAA repair, the temporal trends for different values vary substantially. The ischemic time predicts the maximum AST, lipase, GGT, and LDH levels. These trends should be considered when laboratory values are assessed after TAAA repair.

Key Words
Thoracoabdominal aortic aneurysm · Hepatopancreaticobiliary function · Laboratory values · Alanine transaminase · Aspartate transaminase

Introduction

Repair of thoracoabdominal aortic aneurysms (TAAAs) is a highly complex process that involves interruption of blood flow to vital organs. Despite adjunctive measures, organ ischemia is unavoidable. Consequently, elevation of hepatopancreaticobiliary (HPB) laboratory values is common after TAAA repair. It is not clear whether a certain degree of elevation should trigger further clinical investigation or could provide information about the degree of ischemic insult. To clarify the clinical significance of these changes, we elucidated the expected trends in HPB laboratory values after extensive TAAA repairs.
Table 1. Preoperative Exclusion Criteria

<table>
<thead>
<tr>
<th>Condition</th>
<th>Exclusion Criteria</th>
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<tbody>
<tr>
<td>Planned Crawford extent I or IV TAAA repair</td>
<td></td>
</tr>
<tr>
<td>Planned repair without left heart bypass</td>
<td></td>
</tr>
<tr>
<td>Hypothermic circulatory arrest</td>
<td></td>
</tr>
<tr>
<td>Previous TAAA repair</td>
<td></td>
</tr>
<tr>
<td>Pseudoaneurysm</td>
<td></td>
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<tr>
<td>Free aortic aneurysm rupture</td>
<td></td>
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<tr>
<td>Inability to monitor left kidney temperature</td>
<td></td>
</tr>
<tr>
<td>Impaired renal function (renal failure requiring dialysis, or serum creatinine ≥3 mg/dL)</td>
<td></td>
</tr>
<tr>
<td>Impaired left ventricular function (ejection fraction &lt; 20%)</td>
<td></td>
</tr>
<tr>
<td>Liver disease (conjugated bilirubin &gt; 0.3 mg/dL)</td>
<td></td>
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<tr>
<td>Age younger than 18 years</td>
<td></td>
</tr>
<tr>
<td>Inability to obtain consent</td>
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TAAA, thoracoabdominal aortic aneurysm.

Materials and Methods

Study Enrollment

The population consisted of 155 patients who underwent Crawford extent II TAAA repair over a 5-year period and were enrolled in a randomized trial comparing cold crystalloid and cold blood renal perfusion for renal protection [1]. The Institutional Review Board at Baylor College of Medicine approved the randomized trial and also this retrospective analysis. In accordance with the study protocol, left heart bypass (LHB), selective visceral perfusion, and cold renal perfusion were used during all repairs. Patients with preoperative liver dysfunction (conjugated bilirubin level > 0.3 mg/dL) and preoperative left ventricular dysfunction (ejection fraction < 20%) were excluded. Table 1 lists additional exclusion criteria for the trial. Baseline laboratory values for aspartate transaminase (AST), alanine transaminase (ALT), γ-glutamyl transpeptidase (GGT), lactate dehydrogenase (LDH), total bilirubin, amylase, and lipase were measured preoperatively and for 7 days postoperatively. Table 2 shows the patients’ preoperative characteristics.

Surgical Technique

The surgical technique used for TAAA repairs in our practice has been described in detail elsewhere [1]. A standard protocol for selective visceral perfusion was used in all cases. While the thoracoabdominal aorta was exposed, the patient’s body temperature was allowed to drift to 32-34°C. Left heart bypass was initiated at a flow of 500 mL/min. After the proximal and mid-thoracic aortic cross-clamps were placed, LHB flow was increased to 2000 mL/min. The proximal anastomosis was then completed. Left heart bypass was discontinued, the mid-thoracic aortic clamp was removed, and the ostia of the visceral arteries were exposed. The celiac and superior mesenteric arteries were perfused via 2 9F Pruitt balloon perfusion catheters (Ideas for Medicine, St Petersburg, Florida), with isothermic blood from the LHB circuit at a total average flow rate of 400 mL/min. The intercostal arteries and then the visceral arteries were reattached to openings in the graft. In 54 patients (35%), a single patch containing the origins of all 4 visceral vessels was reattached to an opening in the side of the graft.

Table 2. Preoperative Patient Characteristics

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>62.1 ± 13.1</td>
</tr>
<tr>
<td>Male gender (n; %)</td>
<td>99 (64)</td>
</tr>
<tr>
<td>Hypertension (n; %)</td>
<td>135 (87)</td>
</tr>
<tr>
<td>Diabetes mellitus (n; %)</td>
<td>14 (9)</td>
</tr>
<tr>
<td>Smoking history (n; %)</td>
<td>123 (79)</td>
</tr>
<tr>
<td>Peptic ulcer disease (n; %)</td>
<td>13 (8)</td>
</tr>
<tr>
<td>Aortic dissection</td>
<td></td>
</tr>
<tr>
<td>Acute (n; %)</td>
<td>2 (1)</td>
</tr>
<tr>
<td>Subacute (n; %)</td>
<td>6 (4)</td>
</tr>
<tr>
<td>Chronic (n; %)</td>
<td>70 (45)</td>
</tr>
<tr>
<td>Acute and chronic dissection (n; %)</td>
<td>2 (1)</td>
</tr>
<tr>
<td>AST (IU/L)</td>
<td>23.5 ± 13.9</td>
</tr>
<tr>
<td>ALT (IU/L)</td>
<td>21.3 ± 17.7</td>
</tr>
<tr>
<td>GGT (IU/L)</td>
<td>43.9 ± 58.8</td>
</tr>
<tr>
<td>LDH (IU/L)</td>
<td>252.3 ± 136.5</td>
</tr>
<tr>
<td>Total bilirubin (mg/dL)</td>
<td>1.3 ± 4.8</td>
</tr>
<tr>
<td>Amylase (U/L)</td>
<td>59.5 ± 6.9</td>
</tr>
<tr>
<td>Lipase (U/L)</td>
<td>55.7 ± 92.7</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation. AST, aspartate transaminase; ALT, alanine transaminase; GGT, γ-glutamyl transpeptidase; LDH, lactate dehydrogenase.

Table 3. Intraoperative Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total ischemic time (min)</td>
<td>72.2 ± 22.9</td>
</tr>
<tr>
<td>Total celiac artery ischemic time (min)</td>
<td>63.1 ± 13.9</td>
</tr>
<tr>
<td>Total SMA ischemic time (min)</td>
<td>62.9 ± 13.8</td>
</tr>
<tr>
<td>Unprotected celiac artery ischemic time (min)</td>
<td>41.4 ± 11.3</td>
</tr>
<tr>
<td>Unprotected SMA ischemic time (min)</td>
<td>41.2 ± 11.2</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation. SMA, superior mesenteric artery.
(age, sex, preoperative laboratory value, smoking status, hypertension, diabetes, PRBC). All of the maximum laboratory test values were extremely skewed, violating the assumption of normality of residuals for linear regression. A log transformation was conducted for each of the maximum laboratory tests; this measure showed improved normality and was the variable modeled. All analyses were conducted with Statistical Analysis Software, Version 9.3 (SAS, Cary, NC). In multiple regression models testing for potential factors, backward selection was used with a threshold of $P = 0.20$ to enter the model and $P = 0.10$ to stay in the model.

**Results**

The temporal patterns of the laboratory values were highly variable (Fig. 1). The AST levels showed significant early increases, then decreased toward baseline (Fig. 2). The ALT levels continued to increase during the postoperative period (Fig. 2). The GGT levels remained near baseline through postoperative day 4, and then increased significantly to more than 2 times baseline (Fig. 2). Similarly, the LDH levels also increased immediately and remained significantly elevated throughout the week before trending downward (Fig. 2). The amylase levels increased initially but then trended downward toward baseline (Fig. 3). The lipase levels increased initially, returned to near baseline levels, and then slowly increased again (Fig. 3). The total bilirubin levels were highly variable and showed no definite trend.

In the adjusted analysis, the total ischemic times were not predictive of maximum laboratory values for amylase, ALT, or total bilirubin. Ischemic times were predictive of maximum AST, lipase, GGT, and LDH values. After adjusting for age, sex, and preoperative baseline AST levels, we found that smoking ($\beta = 0.347; \text{standard error (SE)} = 0.109; P = 0.0019$), PRBC ($\beta = 0.035; \text{SE} = 0.013; P = 0.0064$), and the total ischemic time ($\beta = 0.005; \text{SE} = 0.002; P = 0.0133$) were significantly associated with the maximum AST value. Therefore, an increase of 1 min in the total ischemic time was associated with an approximate increase of 0.005 unit in the log of the maximum AST value (10 min would raise the log of the maximum AST by 0.05, 100 min would raise the log of the maximum AST value by 0.5).

After adjusting for age, sex, and the preoperative baseline lipase level, we found that the total celiac artery ischemic time ($\beta = 0.027; \text{SE} = 0.008; P = 0.0011$) was significantly associated with the maximum lipase value. None of the other potential confounders were significant.

After adjusting for age, sex, and the preoperative baseline GGT level, we found that smoking ($\beta = 0.225; \text{SE} = 0.100; P = 0.0264$) and the total ischemic time ($\beta = 0.0055; \text{SE} = 0.0016; P = 0.0011$) were significantly associated with the maximum LDH value.

**Table 4. Postoperative Hepatopancreaticobiliary Values**

<table>
<thead>
<tr>
<th></th>
<th>Median ± SD</th>
<th>Normal values [2]</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST (IU/L)</td>
<td>86.5 ± 105</td>
<td>12–38</td>
</tr>
<tr>
<td>ALT (IU/L)</td>
<td>52 ± 94.5</td>
<td>7–41</td>
</tr>
<tr>
<td>GGT (IU/L)</td>
<td>72 ± 104.5</td>
<td>9–58</td>
</tr>
<tr>
<td>LDH (IU/L)</td>
<td>543 ± 634.7</td>
<td>115–221</td>
</tr>
<tr>
<td>Total bilirubin (mg/dL)</td>
<td>1.8 ± 1.51</td>
<td>0.3–1.3</td>
</tr>
<tr>
<td>Amylase (U/L)</td>
<td>169.5 ± 361.4</td>
<td>20–96</td>
</tr>
<tr>
<td>Lipase (U/L)</td>
<td>85 ± 591.2</td>
<td>3–43</td>
</tr>
</tbody>
</table>

AST, aspartate transaminase; ALT, alanine transaminase; GGT, $\gamma$-glutamyl transpeptidase; LDH, lactate dehydrogenase.

![Figure 1. Overall summary of changes in hepatopancreaticobiliary values after thoracoabdominal aortic aneurysm repair. AST, aspartate transaminase; LDH, lactate dehydrogenase; ALT, alanine transaminase; GGT, $\gamma$-glutamyl transpeptidase.](image-url)
Figure 2. Changes in aspartate transaminase (AST) values, alanine transaminase (ALT) values, γ-glutamyl transpeptidase (GGT) values, and lactate dehydrogenase (LDH) values after thoracoabdominal aortic aneurysm repair.

Figure 3. Changes in amylase values, lipase values, and total bilirubin values after thoracoabdominal aortic aneurysm repair.
Eight patients (5.2%) died within the first 30 days. Five patients (3.2%) developed paraplegia or paraparesis. No patients developed clinically significant hepatic, pancreatic, or gastrointestinal complications.

Discussion

Hepatosplanchnic hypoperfusion and ischemia are rare, but severe, complications after cardiac surgery, and even transient hepatosplanchnic hypoperfusion can lead to severe postoperative complications [3]. In TAAA repair, hepatosplanchnic ischemia is unavoidable, and patients undergoing repair of extent II TAAAs can have unprotected ischemic times of greater than 40 min. In the postoperative period, an alarming elevation of HPB enzymes is common, but the clinical significance of this elevation is not clear. We found that, in the absence of significant heart and liver dysfunction, patients undergoing elective repair of extent II TAAAs had substantial variations in their HPB laboratory values.

Hepatic dysfunction is difficult to measure in the postoperative period because commonly performed laboratory measurements reflect only gross functional abnormalities and are more indicative of cell damage than of dysfunction [4,5]. This is particularly evident in the liver, which has regional variations in the number of hepatocytes and in the ability of different hepatic cell types to withstand hypoxia. We know from metabolic studies of visceral organs protected by adjunctive measures that the resulting flow is not physiologic [6]. Although normal liver blood flow may resume as early as 4-6 h after surgery, the change in hepatocyte metabolism resulting from diminished flow may persist for longer periods [1]. Increases in liver enzyme levels can indicate only hepatocyte damage, not regional perfusion or functional defects. Nevertheless, we chose to use the results of commonly performed liver function tests as markers for liver injury and not liver dysfunction.

Because cytoplasmic enzymes are present in liver parenchymal cells, AST and ALT are indicators of parenchymal injury; however, cardiac, skeletal muscle, and hematologic disorders can also cause elevation of these enzymes. In our series, we found that, despite a continued increase in ALT values throughout the 7-day postoperative period, maximal ALT values were not associated with the total ischemic time, even after adjusted analysis. In contrast, after adjusting for age, sex, and preoperative baseline values, we found that maximal AST values were associated with a history of tobacco use, PRBC transfusion, and the total ischemic time. Because ALT is more sensitive than AST as an indicator of liver damage, elevations of AST may reflect systemic damage resulting from ischemia rather than ischemic damage to the liver. For instance, smoking is known to increase oxidative stress in the body, and differences between ALT and AST after adjusted analysis may relate to differences in the susceptibility of multiple organs to ischemic damage [7]. Elevation of ALT and AST levels has also been correlated with changes in the iron concentration after transfusion [8]. Transfusion of blood products leading to changes in the chelatable iron pool may exceed the hepatocellular iron-chelating capacity and lead to a greater increase in AST than in ALT levels. Because we did not measure the preoperative and postoperative iron levels, we cannot comment further about this association.

GGT and LDH are also widely distributed in the liver and other tissues. In this study, we found that both enzymes became increasingly elevated with time and also were associated with the total ischemic time. However, elevations of these enzymes can be caused by other disorders in the absence of liver disease or dysfunction and are not specific indicators of liver ischemia or injury. Similarly, elevations of the total bilirubin level can be caused by factors other than liver injury. Cholestasis secondary to impaired bile flow may be due to intrahepatic causes (hepatocellular dysfunction resulting from ischemia) or extrahepatic causes, such as biliary obstruction. In this series, we did not find any pattern for changes in the total bilirubin level.

Although the synthetic function of the liver is best assessed by analyzing coagulation factors, we did not do this because (1) there was little variation in these laboratory values, (2) patients often received blood products in the immediate postoperative period to correct coagulopathy, and (3) because of the blood’s interaction with the Dacron graft, patients were often in a mild state of disseminated intravascular coagulation after surgery. Moreover, albumin levels depend on a number of factors, including nutritional status and renal dysfunction, and because of its long half-life, albumin is not a marker of acute hepatic dysfunction. Therefore, the interpretation of abnormal laboratory values depends on the type of abnormality that predominates: hepatocellular damage, abnormal synthetic function, or cholestasis. In this instance, patients with underlying liver dysfunction (e.g., hepatitis C), as...
Elevations in amylase and lipase levels in the postoperative period are indicative of pancreatic ischemia. However, in this study, we found that only a postoperative increase in lipase was associated with the total ischemic time. This insult was manifested by the immediate elevation of pancreatic enzymes on postoperative day 1, followed by a later increase in lipase levels during the ensuing postoperative period. However, depending on the extent of dissection and bowel manipulation, it is common practice to resume enteral nutrition after the return of bowel function, usually around postoperative day 4. The resulting increase in lipase levels may reflect an exacerbation of pancreatic damage induced by the surgery. All patients in our series with elevation of amylase or lipase levels tolerated feeding without abdominal pain or other sequelae, indicating that, despite evidence of ischemic pancreatitis, total metabolic function might not have changed.

To assess the degree of ischemic insult, we compared the ratios between postoperative values and baseline laboratory results for each postoperative day. We did not perform a frequency analysis to further stratify risk factors associated with specific organ dysfunction. Analyzing these risk factors would have been inconsequential because patients with preoperative liver or renal dysfunction were excluded, few patients had diabetes (9%), and most patients smoked (79%). Instead, we used a multiple linear regression model with generalized estimating equations, which allowed us to adjust for the correlated nature of the laboratory values measured. Because the study’s main goal was to establish generalized trends in the postoperative period, we did not perform a statistical analysis examining all the different variables that correlated with extreme postoperative laboratory values.

The primary determinant of whether a patient will develop postoperative multiple organ dysfunction (MOD) is visceral ischemia lasting for longer than 40 min [9,10]. In our series, the mean total ischemic time was 72.2 min, with a mean unprotected celiac artery ischemic time of 41.4 min and mean superior mesenteric artery ischemic time of 41.2 min. However, none of our patients developed MOD. Use of adjunctive measures such as LHB significantly reduces the duration of visceral ischemia. Although our patients had a mean ischemic time of >40 min, selective visceral perfusion may have offered a protective effect against MOD, even in the presence of elevated postoperative HPB laboratory values.

Despite improved surgical techniques, spinal cord ischemia and renal failure remain the most devastating complications associated with repair of TAAA. The rate of paraplegia in our series was 3.2%. There was no correlation between significant elevations in HPB laboratory values and the development of either paraplegia or renal failure. However, in paraplegia patients, this may have been due to a small sample size. Most importantly, none of the patients in our series developed clinically significant postoperative HPB dysfunction. This may have been due to the fact that patients had relatively normal preoperative liver function and moderate-to-good left ventricular function, and most often underwent elective TAAA repair. Safi et al. [11] have shown that a history of hepatitis, extent II aortic aneurysm, ruptured aortic aneurysm, and emergency presentation are significant predictors of elevated postoperative liver function values. Therefore, patients with borderline liver function or prolonged ischemic times may be pushed into liver failure and perhaps even multisystem organ failure.

Limitations

This study has a number of limitations. First, although data were collected prospectively in accordance with a randomized clinical trial protocol, the secondary analysis of HPB enzyme levels was retrospective and, thus, consequently can provide only a descriptive picture of the metabolic changes that occur in patients who undergo elective repair of extent II TAAAs. Second, use of a longitudinal analysis model over a 7-day period limits the amount of information that can be gained. Third, we acknowledge that providing 400 mL/min through balloon-perfusion catheters may not provide optimal visceral flow. The 9F catheters have been used in our practice in part to facilitate safe catheter placement in vessels that often have small ostia and atherosclerotic plaques and to avoid vessel injury and dislodgement of atherosclerotic debris. Although we have been satisfied with the clinical results achieved when using these catheters (an extremely low incidence of overt hepatic, pancreatic, and gastrointestinal ischemic complications), the current study suggests that substantial subclinical organ injury occurs and that use of larger catheters warrants consideration. The study leaves several significant questions unanswered, such as (1) whether there is a relationship between the duration of visceral ischemia and

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injury to the liver or other organs, (2) whether any of the abovementioned preoperative HPB laboratory values are more or less specific in predicting organ dysfunction postoperatively, (3) whether there is a relationship between HPB injury and dysfunction of other organs (as indicated by elevated laboratory values) or development of multisystem organ failure, and (4) whether adjunctive measures are significantly protective in patients who have preoperative hepatic dysfunction or cirrhosis to allow safe repair of TAAAs. Moreover, in some instances proximal aortic clamping is not possible; the trends revealed by this study cannot be applied to patients in whom circulatory arrest is used.

**Conclusion**

In patients undergoing TAAA repair, HPB enzyme levels are expected to be elevated postoperatively in the absence of liver dysfunction or multisystem organ dysfunction. In this study, we established the normal expected patterns for HPB laboratory values in the postoperative period after TAAA repair. In some cases, the degree of elevation correlated with the duration of ischemia. We hope that our findings may be useful for evaluating laboratory results in similar cases and for interpreting the results of future studies related to visceral protection.

**Acknowledgments**

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**Conflict of Interest**

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

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EDITOR’S COMMENT

We are indebted to Dr. LeMaire and colleagues for documenting this important information. We always wondered, but never knew, how to interpret these LFT abnormalities.
Acute Type A Dissection Repair in an Achondroplastic Dwarf
Anesthetic, Perfusion, and Surgical Concerns

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Abstract
In this report we present a 43-year-old male with achondroplastic dwarfism who presented with acute Type A aortic dissection with aortic insufficiency. The patient underwent successful Bentall and hemiarch repair. Anesthetic, perfusion-related, and surgical planning and execution are presented.

Key Words
Dwarfism · Dissection

Introduction
Achondroplasia is the most common type of chondrodysplasia with an incidence of 1 in 25,000 to 40,000 births [1]. It occurs due to a mutation in fibroblast growth factor receptor 3 (FGFR3). To our knowledge, there is no association between achondroplastic dwarfism and aortic dissection.

To the best of our knowledge, this case represents the fifth case report of open heart surgery in a dwarf patient and the first one of aortic dissection repair [2–5]. Informed consent was obtained from the patient for publication of this case report with the accompanying images.

Case Presentation
A 43-year-old man with achondroplastic dwarfism and known hypertension presented with severe chest pain migrating to his back. Computed tomography angiography (CTA) showed acute Type A aortic dissection, and the patient was brought to our institution for surgical management.

Past medical history was significant for achondroplastic dwarfism [height, 103 cm; weight, 37 kg; body surface area (BSA), 1.03 m²], hypertension, smoking, and chronic obstructive pulmonary disease. Specific complications of achondroplasia included thoracic kyphosis, spinal stenosis, and a surgically fused cervical spine.

Chest X-ray showed a wide mediastinum (Fig. 1) and CTA demonstrated an aortic root dissection flap that extended through the ascending aorta and aortic arch to the iliac bifurcation (Fig. 2A and 2B). The great vessels of the aortic arch communicated with the true lumen, as did the major abdominal branches.

Intraoperative Management
The patient was taken to the operating room for a Bentall procedure and replacement of the ascending aorta and hemiarch. General anesthesia was induced and intubation was performed by video laryngoscopy. Right internal jugular vein and left femoral artery lines were placed with ultrasound guidance. Intraoperative transthoracic echocardiogram (TEE) demonstrated a dissection flap in a dilated (52 mm) aortic root with dissection extending into the descending thoracic
aorta (Fig. 3). There was severe aortic insufficiency due to a failure of central coaptation.

A 6-mm graft was anastomosed to the right axillary artery using a subclavicular approach. Median sternotomy was performed, the patient was heparinized, and arterial and venous cannulation (28/30F, Medtronic, Minneapolis, MN, USA) were established. We utilized a standard-sized cardiopulmonary bypass (CPB) circuit. Retro-priming was used to remove 400 cc of prime volume. Flow rates maintained a cardiac index of 1.8-2.5 L/min/m². Hemo-concentration was performed throughout the procedure.

Cooling was commenced to a target temperature of 18°C. At this point, we were not sure whether we would be able to use antegrade selective cerebral perfusion (ASCP) due to the patient’s small-caliber vessels. The aorta was cross-clamped and 1 L of cold retrograde cardioplegic solution was administered. The aortic aneurysm was opened and the dissection flap was identified at the right coronary sinus. The aorta was resected, the coronary buttons were detached, and the valve was excised. A composite graft constructed from a 26-mm conduit and a 23-mm mechanical bileaflet valve was inserted using 13 sutures of 2-0 Tycron reinforced with pledgets. At 18°C, the innominate artery was clamped and circulatory arrest was initiated with ASCP (12-15 cc/kg/min; mean pressure 39 mm Hg). Once we established adequate ASCP, we started to rewarm the patient. The aorta was transected just proximal to the innominate artery and a 22-mm Dacron graft was anastomosed end-to-end. The nondissected aortic tissue in the aortic arch was robust with no obvious signs of aortopathy. The graft was clamped and CPB was reinitiated. The remaining proximal anastomosis and reimplantation of the coronary buttons were completed.

TEE confirmed a normal-functioning prosthetic valve. Global ventricular function was preserved and the patient was weaned from bypass without difficulty. With satisfactory hemostasis achieved, the sternum, pre-sternal fascia, and skin were closed in routine fashion. Total CPB, cross-clamp, and circulatory arrest times were 154, 102, and 19 min, respectively. No blood products were given. Total pump balance including cardioplegia was +850 mL.

The patient’s overall hemodynamic status was stable, and he remained neurologically intact. Postoperative TTE
demonstrated normal biventricular function, and a well seated #23 mechanical aortic valve with a 6 mm Hg mean gradient and a 2.2 cm² effective orifice area. The patient was discharged to a rehabilitation facility on postoperative day 6. By six weeks postoperatively, he had near complete functional recovery (Fig. 4).

Discussion

Although still relatively rare, achondroplastic dwarfism is the most common type of dwarfism. To our knowledge, there is no association between achondroplastic dwarfism and aortic dissection. This, to the best of our knowledge, is the first report of an acute Type A aortic dissection in an achondroplastic dwarf. There are only four other accounts of cardiac surgeries and dwarfism [2–5]. For a coronary artery bypass graft procedure, the major concern is limited saphenous vein reserve, but otherwise, the procedure can be performed routinely [3]. For aortic valve replacement, aortic root enlargement was required for a very small aortic annulus [4].

Anesthetic Considerations

Achondroplasia can present some unique challenges for anesthetic management [6–8]. Difficult airway management may occur, possibly related to short kyphotic cervical spine, maxillary hypoplasia, and megalencephaly [6]; however, larynx visualization is usually uncomplicated. There may be stenosis of foramen magnum and hydrocephaly. Our patient had short thyromental distance and remote history of surgical cervical spine intervention with spinal stenosis. Despite preparations for difficult airway, the trachea was intubated uneventfully with neutral neck position and a video laryngoscope.

Restrictive lung disease from thoracic kyphosis and rib hypoplasia may be present and can lead to pulmonary hypertension and cor pulmonale in advanced cases [6,8]. Our patient had a history of kyphosis but did not have any preexisting symptoms of cardiopulmonary function.

Finally, vascular access and positioning may be difficult because of reduced joint mobility, thoracic kyphosis, and small limb size [8]. Careful patient positioning using modified padding was required due to marked kyphosis and reduced extremity mobility. Ultrasound guidance was critical for central venous and femoral arterial vascular access.

Perfusion Considerations

The overall perfusion strategy employed was that of a pediatric patient. Every effort was taken to mini-
mize the static and dynamic prime volumes. We used retrograde autologous priming. Also, venous tubing was downsized from 1/2 inch to 3/8 inch and arterial axillary cannulation was accomplished with a 6 French graft without flow or drainage issues. Scafuri et al. [4] reported the use of pediatric cannulae both for the aorta (20F) and for the superior and inferior venae cavae (28F), but their patient was considerably smaller than ours (height 100 cm, weight 27 kg, BSA 0.87 m²). However, because ours is an adult cardiac center, pediatric cannulae were not readily available on such short notice. The CPB circuitry was optimized to reduce the number of shunts, surface area, and connectors. Although we did not have access to the small adult oxygenator with integrated filter, this would have further reduced our dynamic prime volume. Hemo-concentration was utilized throughout to minimize hemodilution. A hemo-concentrator can also be utilized for modified ultrafiltration post-CPB; however, this technique was not employed in our patient. Overall, the patient responded well to CPB.

**Surgical Considerations**

To our surprise and relief, the surgical management was rather uncomplicated as the patient’s thorax was of relatively normal size. The right axillary cut-down was performed with the usual depth and anatomical landmarks. Due to small lower extremities, axillary cannulation is preferred because it avoids cannulation of hypoplastic femoral arteries. The sternum was moderately kyphoid, but sternotomy was uncomplicated. The aortic root was dilated, and although we did not have preoperative echocardiographic imaging, the CTA showed quite a large left ventricular outflow tract. Because the BSA was only 1.03 m², patient–prosthesis mismatch was unlikely to be a concern. We planned to implant either a 19- or a 21-mm valve but were pleased to find that a 23-mm valve could be implanted without difficulty. In the only other reported case of aortic valve replacement in a patient with dwarfism, a small aortic root required a Manougian-type patch aortic root enlargement to fit a 16-mm mechanical Carbomedics valve (Sorin Group Company, Austin, TX, USA) in a 56-year-old female with a very small, 10-mm, aortic annulus [4].

**Conclusion**

This report, to the best of our knowledge, is the first to describe successful aortic dissection repair in a patient with achondroplastic dwarfism. The procedure was performed without the need for specialized cannulation equipment or aortic root enlargement procedures to facilitate a suitable valve prosthesis. This report adds to the experience of successful acquired and congenital cardiac surgery in this unique patient population.

**Conflict of Interest**

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

**References**


Development of Mega-Aorta Following Incompletely Treated Giant Cell Arteritis

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Abstract
An 82-year-old male presented with a 9.3 cm ascending aorta and arch aneurysm with additional aneurysms of the innominate, right subclavian, and left common carotid arteries. The patient had a history of temporal arteritis that was only briefly treated in 1989 and a 6 cm ascending aortic aneurysm that was repaired in 1993. Our operative strategy was to construct a temporary parallel cerebrovascular circuit for cerebral protection during the redo-sternotomy and aortic arch reconstruction, with the added benefit of permanently excluding the branch arch vessel aneurysms. Pathological analysis of the aortic specimen at the first operation may have identified giant cell arteritis, prompting medical therapy against further disease progression.

Key Words
Aortic aneurysm · Inflammation · Subclavian artery · Carotid artery

Case Presentation
An 82-year-old male of Hungarian descent presented with dilated neck veins and pulsatility in his neck. Preoperative imaging (Fig. 1) identified a 9.3 cm ascending aortic (AscAo) and aortic arch aneurysm, as well as a 4.0 cm innominate artery (InomA), a 2.5 cm right subclavian artery (RScA), and a 2.6 cm left common carotid artery (LCCA). The RScA was at risk of rupturing. Both common carotid arteries were redundant proximally. The maximal diameters of the descending and visceral aorta were 5.8 and 5.0 cm, respectively.

In 1989 the patient presented with malaise, extremity joint pain, and fatigue. A biopsy conducted at that time indicated temporal arteritis. The patient self-reported a 1- to 2-month course of prednisone after that admission. In 1993 a 6 cm proximal AscAo aneurysm was resected. The AscAo aneurysm diameter distal to the repair grew to 7.4 cm in 2004, 8.2 cm in 2007, and 9.3 cm in 2011. The patient did not have a history of tobacco abuse, hypertension, or emphysema. He had no family history of aneurysms, although his aunt had temporal arteritis. Three weeks prior to surgery, a percutaneous transluminal coronary angioplasty was performed and a bare metal stent was placed in the left anterior descending artery.

Preoperative imaging showed extremely tortuous left and right carotid arteries (Fig. 2), as well as apposition of the aneurysm to the previous sternotomy with compression of the vena cava and innominate vein (Fig. 3). Our operative strategy was to (1) maintain continuous cerebrovascular perfusion during initial exclusion of the InomA, RScA, and left common carotid artery, (2) conduct a redo-sternotomy to replace the AscAo and arch aneurysms, and (3) replace the aortic valve. To these ends, a temporary extra-anatomic circuit was constructed. The first step was to perfuse the right and left common carotid arteries, and the right axillary
artery (RAx) using the left subclavian artery (LScA). Bilateral supraclavicular incisions were used to expose the arch branch vessels. The left common carotid artery was divided distally to its aneurysmal portion and transposed onto the apex of the LScA.

A three-limbed graft was constructed from a bifurcated 16X8 mm Gore-Tex® (Flagstaff, Arizona) aortic graft by dividing one of the 8 mm limbs and sewing it to the 16 mm main body. This graft was left external to the loban™ drape (3M, St. Paul, Minnesota) that had been placed across the operative field. The three 8 mm side-arm limbs were sewn to the circulation in the following order: end-to-side to the mid-left common carotid artery, end-to-end to the right common carotid artery after resecting the redundant proximal portion, and end-to-side to the RAx (Fig. 4). The 16 mm limb of the graft was then attached end-to-end to the cardiopulmonary bypass (CPB) circuit for arterial inflow. A 25F Bio-Medicus Multi-Stage Femoral Venous Catheter (Medtronic, Santa Rosa, California) was placed in the right common femoral vein for venous drainage into the CPB circuit.

Flow into the RScA was excluded with a staple line. Visualization of the RScA origin was possible only after the right common carotid artery had been transected, permitting anterior retraction of the aneurysmal InomA. The RScA branches were clipped in continuity and the proximal RAx was ligated just below the clavicle, excluding retrograde flow into the RScA. Intraoperative imaging showed no central neurological changes.

CPB was then instituted at 6 L/min, reversing flow in the Gore-Tex® external circuit into the RAx, the right and left common carotid arteries, and down the LScA into the aorta. The left ventricle (LV) was monitored for distention with transesophageal echocardiography. At 25°C the left common carotid artery was clamped between the Gore-Tex® external circuit and the LScA. This arrested arterial flow into the aorta and the rest of the cooled body, while preserving antegrade cerebral perfusion (ACP) at 2 L/min via the external Gore-Tex® circuit into the right and left common carotid arteries and the RAx.

Sternotomy followed without entry into the depressurized aneurysm and adjacent venous system. Via an intrapleural right pulmonary venotomy, a vent was placed across the mitral valve for LV decompression. The giant aneurysm was opened and the heart was arrested by giving cardioplegia directly into the left and right coronary arteries. A 30 mm Vascutek Gelweave 4 Plexus interposition graft with three arch branches and a side-arm (Teurmo, Ann Arbor, Michigan) was sewn end-to-end into the distal arch at the level of the origin of the LScA. Then the graft’s side-arm conduit was attached as a second arterial inflow from the pump so that CPB could be restored to the body at 6 L/min.

While the patient was rewarmed, an aortic valve replacement was performed using a 23 mm Edwards Magna Bioprosthetic Aortic Valve (Edwards Life-sciences, Irvine, California). The proximal end of the Dacron graft was then sewn end-to-end to the remnant of the old proximal AscAo Dacron graft. The ACP and aortic cross-clamp times were 23 and 98 min.
respectively. A pulse was regained, and the Gore-Tex® circuit was then excised while weaning from CPB. Its attachment to the left common carotid artery was disconnected and the artery repaired. The attachment to the right common carotid artery was excised and the artery was immediately sewn end-to-end to one of the three Dacron side-arms on the arch graft. The Gore-Tex® limb to the RAx was divided and sewn end-to-end to the second Dacron side-arm on the arch graft. The third side-arm of the Dacron arch graft was not needed.

No focal neurological deficits developed postoperatively, and the patient was extubated on postoperative day 3. The postoperative course was complicated by pneumonia, resulting in reintubation on postoperative day 7. The patient was discharged to a rehabilitation facility, neurologically intact, and with no evidence of infarcts on his follow-up brain CT scan. Cardiac function remained excellent and renal function was normal.

**Discussion**

This case report highlights a serious complication of temporal arteritis: aortic aneurysm. The incidence of giant cell arteritis (GCA) in the US has been reported as high as 27 cases per 100,000 persons over 50 years old. Patients with Northern European ancestry have a higher incidence [1]. The aortic root is involved in 9-18% of cases [2]. In a population study of Olmsted County, Minnesota, the incidence of GCA was 17.8 per 100,000 persons over 50 years old [3]. The incidence of thoracic aneurysm, and of isolated abdominal aortic aneurysm, was 17 times [95% CI (confidence interval), 8-33], and 2.4 times (95% CI, 0.8-5.5) higher, respectively, compared to patients without GCA. Thoracic aneurysms developed as far out as 15 years after treatment of GCA. This case report also identifies a modality to safely reenter a chest when an aneurysm is adjacent to the wired sternotomy closure, and illus-
trates how a combination of extra-anatomic strategies and CPB is feasible in an elderly patient with extensive aortic arch, aortic valve, and branch artery pathology.

There was no evidence of granulomatous inflammation or giant cells, hallmarks of GCA, in this patient’s aorta at either operation. There was, however, patchy extensive destruction of the transmural lamellar architecture (disappearance of the elastic fibers, fibrosis of the adventitia, thickening of the intima) and prominent penetrating vasa vasora, which are features of the disease. There is one other report of mega-aorta following diagnosis of GCA [4]. However, in our case, the absence of hypertension, tobacco use, emphysema, and hyperlipidemia in this patient suggests that GCA played a significant role in the evolution of his large aneurysms. The patient was only briefly treated with steroids after a positive temporal artery biopsy for GCA in 1989. Whether this patient’s aneurysms were preventable if he had had more aggressive medical management of his arteritis is unknown, since medical management of temporal arteritis is still evolving, and there are isolated reports of aneurysm development even after prolonged immunosuppression. Notably, his first 6 cm proximal AscAo aneurysm was treated four years after diagnosis of temporal arteritis.

Perhaps the progression of the aneurysm disease could have been avoided if pathological analysis had been conducted at the first operation in order to yield a diagnosis and commence aggressive medical management. Genetic analysis could also have been conducted as individuals homozygous for HLA-DR4 and the FCGR2A-131RR allele have a 6-fold greater risk of developing temporal arteritis. Further, both the HLA-DR1, HLA-DR3, and HLA-DR5 genes and polymorphisms in immune-modulating proteins such as interleukin-1 receptor antagonist, tumor necrosis factor-α, and intracellular adhesion molecule-1 are associated with a greater incidence of temporal arteritis.

Although the axial growth of this patient’s aneurysms was remarkable, the longitudinal growth was equally impressive. The InomA extended significantly into the patient’s neck. Both common carotid arteries were so redundant they each developed two nearly 180 degree turns. Arteriogenesis, the maturation of collaterals around an occluded large artery, also generates (on a much smaller scale) angiographically visible “corkscrew” collaterals due to both longitudinal and radial growth. While they both generate “corkscrew” vessels on a macroscopic level, GCA and arteriogenesis also have similar biochemical mechanisms. In both processes, endothelial cells express adhesion molecules which bind circulating inflammatory cells that are then brought subendothelially. There, they secrete membrane metalloproteinases and nonspecific serine proteases that digest the underlying supportive protein matrix, allowing for growth of the artery. Further elucidation of the mechanism of arterial growth in GCA may be dually beneficial. Insight into the mechanism of arterial growth may help design therapeutic strategies for arterial collateral growth in chronic severe limb-threatening ischemia, but also therapeutic inhibition of aneurysm expansion.

Regarding the operative approach, a temporary parallel cerebrovascular circuit was constructed rather than performing a simple axillary cannulation for two reasons. First, given the patient’s multiple aneurysms in the great vessels, extensive replacement of the great vessels was not indicated. Second, the temporary parallel circuit provided the added benefit of increased cerebral protection. We believed that there was a significant likelihood that we would enter the aorta on sternotomy. Given the patient’s age and the complexity of the procedure, we wanted to ensure cerebral protection. The patient was cooled before opening; however, due to the patient’s significant aortic valve insufficiency, there was concern that the ventricle would dilate and the patient would fibrillate, without being sufficiently cool for adequate cerebral protection.

In summary, this case report presents an 82-year-old patient who developed progressive aneurysmal aortic degeneration due to giant cell arteritis. The patient underwent initial ascending aortic aneurysm repair, and presented to our center over 15 years later with multiple, large aneurysms. The patient required a high-risk redo-reconstruction that necessitated the use of a temporary parallel cerebrovascular circuit. The operation was successful, and the patient was discharged to a rehabilitation facility on postoperative day 7, neurologically intact, with no evidence of infarcts on his follow-up brain CT scan.

Conflict of Interest

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


Establishment of Extracorporeal Circulation under Local Anesthesia in a Patient with an Acute Type A Aortic Dissection Complicated by Cardiac Tamponade

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Abstract
We report the case of an 82-year-old female who presented in a hemodynamically unstable condition to the emergency department of our institution. Transthoracic echo showed a hemodynamically relevant pericardial effusion and the suspicion of an intimal flap in the ascending aorta. The subsequent computed tomography scan revealed a Type A dissection that was limited to the ascending aorta. To prevent hemodynamic deterioration the patient was prepped and draped awake and underwent femoral cannulation for extracorporeal circulation under local anesthesia. After commencing extracorporeal circulation the patient was anesthetized and intubated. During this whole time period no relevant drop in mean arterial pressure was observed. The patient underwent routine replacement of the ascending aorta and was extubated the day after surgery without any neurologic sequelae. Awake cannulation and inception of extracorporeal circulation can prevent the hemodynamic deterioration and cardiac arrest often seen during induction of anesthesia in patients with cardiac tamponade.

Key Words
Type A aortic dissection · Pericardial tamponade · Extracorporeal circulation

Introduction
Cardiac tamponade is a life-threatening complication in acute Type A aortic dissection and is an important predictor of death in these patients [1]. Further hemodynamic deterioration and cardiac arrest during induction of anesthesia is a common complication of cardiac tamponade [2].

Case Report
An 82-year-old woman was admitted to the emergency department of our institution in hemodynamic instability. Transthoracic echo revealed a hemodynamically relevant pericardial effusion. Noninvasive blood pressure at this time was between 80 and 90 mm Hg systolic and the patient was conscious. An immediately performed computed tomography (CT) scan showed an acute Type A dissection limited to the ascending aorta, beginning above the sinotubular junction and ending before the brachiocephalic trunk.
(Figs. 1 and 2). During preparation of the operating theater the patient received an arterial line and a central venous catheter in the emergency department. On arrival in the operating theater the patient required increasing vasopressor support with norepinephrine and suprarenin to maintain a systolic blood pressure between 60 and 70 mm Hg. The patient was prepped and draped awake and underwent exposure and dissection of the right femoral vessels awake under local anesthesia. A purse string suture was placed on the femoral artery and vein and the patient was heparinized with 300 IU/kg. After measurement of the activated clotting time the venous cannula was placed in the right atrium and the arterial cannula in the iliac artery. Then extracorporeal circulation was established, anesthesia was induced, and the patient was intubated. Retrospective examination of the automatic anesthesia protocol showed no drop in mean arterial pressure below 55 mm Hg between the beginning of the monitoring in the operating room and the beginning of extracorporeal circulation. Then sternotomy was performed and the patient underwent replacement of the ascending aorta. No cooling and hypothermic arrest was needed as the dissection only involved the ascending aorta and clamping of the aorta was possible proximal to the brachiocephalic trunk in nondissected tissue. This was verified with a brief circulatory arrest, declamping of the aorta, and visual inspection. The aorta was replaced with a supra-coronary 28 mm Dacron® graft. The rest of the procedure was uneventful. The patient was extubated the following day and did not show any neurologic complications. A postoperative CT scan showed a good surgical result and the patient was discharged on postoperative day 10.

**Discussion**

Several prior reports describe emergency establishment of cardiopulmonary bypass before induction of general anesthesia in a variety of threatening
circumstances [3–5]. Acute Type A dissection with cardiac tamponade is associated with an increased risk of mortality [1]. Surgical or interventional sub-xiphoidal drainage is risky in an acute Type A dissection, as this may lead to exsanguination of the patient due to free rupture of the aorta. Also, due to clot formation, sufficient decompression of the heart may not be achieved. An interesting technique to deal with this situation was described by Hayashi et al. [6]. They percutaneously inserted a pigtail catheter in the pericardium and controlled the drainage volume by intermittent withdrawal to keep the systolic blood pressure around 90 mm Hg [6].

The risk of hemodynamic deterioration during induction of anesthesia was very high in this patient as she already required vasopressor support and, under this support, had a systolic blood pressure between 60 and 70 mm Hg. Emergency sternotomy in the case of acute hemodynamic deterioration during induction of anesthesia and cannulation of the aorta would have been extremely risky due to the aortic dissection. In a case report published in 1989, Norman et al. [7] described two patients with aortic dissection and cardiac tamponade. In one patient they induced anesthesia and performed sternotomy as usual. This patient died due to aortic rupture and exsanguination after opening of the pericardium. In the other patient the first surgical procedure performed after induction of anesthesia was femoral cannulation and establishing of extracorporeal circulation to prevent a sudden increase of cardiac output and blood pressure during opening of the pericardium [7]. This patient survived surgery and was discharged from the hospital.

We chose to cannulate the femoral vessels under local anesthesia and initiate extracorporeal circulation while the patient was awake to minimize the risk of complications associated with induction of anesthesia. Additionally, as the dissection was limited to the ascending aorta, we did not have to fear the potential risks of femoral cannulation in patients with an acute Type A dissection [8].

In summary, our approach avoids the risk of hemodynamic deterioration during induction of anesthesia.
Also, our approach avoids the sudden increase of cardiac output and blood pressure after opening of the pericardium, which can produce free rupture and exsanguination. Finally, our approach preserves circulation in patients presenting with cardiac tamponade resulting from an acute Type A aortic dissection.

Conflict of Interest

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

References


EDITOR’S COMMENT

This is a valuable technique to have in the armamentarium. Another tool we use in such circumstances is to anesthetize the patient, perform sternotomy, and make a small pericardial opening—letting fluid out slowly, to avoid rebound hypertension and free rupture. The anesthesiologist controls the blood pressure with medications as hemodynamics improves during gradual relief of tamponade.

Concurrently, we have exposed the femoral artery for cannulation, which we use routinely in Type A dissection [1].

EDITORS’ COMMENT

Dr. Scott A. LeMaire, Professor, Baylor College of Medicine, Houston, Texas, USA.

The authors are to be congratulated for successfully managing a challenging emergency situation involving an elderly patient with acute dissection. Although the concept of establishing cardiopulmonary bypass via the peripheral vessels during local anesthesia in patients with severe cardiopulmonary instability is not new, the current report serves to remind readers that this can be a very useful approach in patients with acute dissection (particularly DeBakey type II dissection) and severe tamponade.
Hybrid Approach to Repair Type A Aortic Dissection
Combined Endovascular Superior Mesenteric Artery Stenting and Bentall Procedure

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Abstract
A Stanford Type A aortic dissection is a life-threatening surgical emergency that requires emergent surgery. The mortality after repair is high especially if the aortic dissection is complicated by visceral or peripheral malperfusion. We describe a case of a male patient who presented with an aortic dissection involving the ascending aorta, aortic arch, descending thoracic aorta, and the abdominal aorta down to the iliac bifurcation. The dissection also involved the visceral and renal arteries with evidence of superior mesenteric artery (SMA) occlusion. Successful outcome was achieved by endovascular stenting of the patient’s SMA, followed by a Bentall procedure. To the best of our knowledge this is the first case report in the English literature of SMA stenting followed by a Bentall procedure to treat acute Type A aortic dissection complicated by SMA occlusion.

Key Words
Type A aortic dissection · Bentall procedure · Superior mesenteric artery · Stent graft

Introduction
A Stanford Type A aortic dissection is a life-threatening surgical emergency that requires emergent surgery. The mortality after repair has been reported as high as 30% [1], although most recent series demonstrate mortality in the 10-16% range [2,3] after surgical repair. Approximately 25% of aortic dissections have evidence of peripheral malperfusion at presentation [4]. In cases of visceral malperfusion syndrome, particularly involving the superior mesenteric artery (SMA), the operative mortality is significantly increased [5]. Without intervention in acute Type A aortic dissection, early death occurs in 30% of patients by 24 hours and 93% at 1 month as a result of malperfusion syndromes, or cardiac complications or rupture [6]. Type A aortic dissection is complicated by visceral malperfusion in 16-33% of cases [4–7].

Case Report
A 55-year-old gentleman who is diabetic, hypertensive, and a heavy smoker presented to the emergency department of his local hospital with sudden severe neck pain that started 3 h earlier. The pain radiated to his back and was associated with sweating, nausea, and vomiting. A computed tomography (CT) scan of the chest and abdomen was performed which showed an aortic dissection involving the ascending aorta, aortic arch, descending thoracic aorta, and the abdominal aorta down to the iliac bifurcation (Fig. 1A and 1B). The dissection also involved the visceral and renal arteries with evidence of SMA occlusion (Fig. 1C).
The patient was transferred to our institution for further management. On arrival to our coronary care unit (CCU) he was hypertensive at 180/110 mm Hg and still in pain. He was also complaining of pain and tenderness involving his upper abdomen. There were absent pulsations in his left upper limb and left femoral arteries. Cardiac auscultation revealed a soft early diastolic murmur at the aortic area. He received intravenous nitroglycerin and beta-blockers to control his blood pressure. He underwent a transesophageal echocardiogram (TEE) which confirmed dissection of the aorta from the level of the aortic root and involving the right coronary artery, and extending into the aortic arch and descending aorta. The TEE also showed an intimal tear in the ascending aorta above the sino-tubular junction. The aortic root was dilated at 4.5 cm and the ascending aorta was 5 cm in diameter. Due to the nature of the static obstruction of the SMA and the onset of abdominal pain and slight abdominal tenderness, it was decided to begin by opening the SMA with a stent before performing the Bentall procedure to prevent the occurrence of bowel ischemia with its high mortality. The SMA was successfully opened in the catheterization laboratory by a vascular surgeon through a right femoral artery access under general anesthesia. The patient received a covered 6 × 40 mm self-expandable stent (Fig. 2A and 2C), a Nitinol stent from Cordis Corp. (Bridgewater, New Jersey). The time between patient arrival to our hospital and stenting the SMA was 3 h. Then, the patient was transferred directly to the operating room for repair of the aortic dissection by a Bentall procedure (Carbomedics size 23 mm valve, graft diameter 26 mm, model cp-023, Sorin Medica, Milan, Italy; Fig. 2B). The distal extent of resection was proximal to the innominate artery. The operation was successful and the patient had a smooth postoperative course. All pulses in both upper and lower extremities were present postoperatively. The patient was discharged home after 1 week in a good general condition. The fate of the false lumen was still present and not fully thrombosed. The patient was followed at the outpatient clinic and was doing well 1 year after this operation.

**Discussion**

Acute Type A aortic dissection is a life-threatening condition. The primary goal of early surgery is to minimize morbidity and mortality by preventing or reversing end-organ malperfusion. However, when systemic or visceral malperfusion is already occurring and complicates the condition, a strategy of address-
The possible persistence of residual false lumen in the untouch distal aorta after repair can increase the risk of death [9]. Ascending aortic graft, followed by endovascular stenting of the arch and of the descending and thoracic aortic segments [9] or elephant trunk technique [10] are two therapeutic options to deal with the false lumen and hence open dynamic peripheral and visceral arterial occlusions.

Two major issues should be considered in acute dissections associated with peripheral ischemia. One is deciding which lesion should be treated first (i.e., the proximal aorta or the visceral organ), and the other is the mechanism of occlusion of peripheral arteries. A primary aortic surgery has an increased risk of bowel infarction, which frequently leads to patient mortality, and a primary visceral revascularization might delay the proximal aortic repair.

Percutaneous management of ischemic complications in patients with Type B aortic dissection is well established. Vedantham's et al. [11] retrospective review of Washington University's interventional radiology database identified 11 patients with acute Type B aortic dissection who underwent 13 endovascular procedures to attempt revascularization of 23 ischemic vascular territories (4 mesenteric, 11 renal, 8 lower extremities). Percutaneous interventions included balloon fenestration (4 patients), aortic true lumen stent placement (3 patients), and branch vessel stent placement (8 patients).

Slonim et al. [12], at Stanford, reviewed 22 patients who underwent percutaneous treatment for peripheral ischemic complications of 12 Type A (five acute, seven chronic) and 10 Type B (nine acute, one chronic) aortic dissections. All five patients with an acute dissection underwent surgical repair of the ascending aorta before undergoing the endovascular procedure. In four of these five patients the endovascular procedure was performed within 24 h of the operation, and in the fifth patient it was performed 10 days after the operation.

In our case, after diagnosis and stabilization of the patient, management was done by stenting the superior mesenteric artery first, followed by the Bentall operation. This choice was taken because the patient was stable and also because of his abdominal pain and tenderness with impending bowel ischemia and because of the nature of the SMA occlusion, which was static, with lower chance of reopening after proximal aorta repair.

Figure 2. A and B. Computed tomography taken 10 days post-repair (A) and showing ascending aorta replacement with valved conduit (B). Note the stent in superior mesenteric artery (SMA). C. Axial view showing the stent in SMA (arrow).
Conclusion

We describe a case of successful management of an acute Type A aortic dissection and SMA occlusion and beginning bowel ischemia treated with endovascular stenting of the SMA, followed by a Bentall procedure.

References


EDITOR’S QUESTIONS

1. Why did you sample only the outer layer in your dissection patients? We know the dissection occurs in mid-media. Why not sample and examine the inner layer as well?

We are primarily interested in what drives aortic dilatation after aortic dissection has occurred. We focused on the outer wall of the false lumen because this is the region primarily responsible for aneurysm expansion and rupture in patients with dissection, and the region that would be the target for pharmacologic treatment designed to prevent dilatation after dissection. Changes involving the inner dissecting membrane (or dissection “flap”) would have limited clinical relevance in chronic dissection.

In our case, after diagnosis and stabilization of the patient, management was done by stenting of the superior mesenteric artery first, followed by a Bentall operation. This choice was taken because the patient was stable and also because of his abdominal pain and tenderness with impending bowel ischemia and because of the nature of the SMA occlusion, which was static with a lower chance of reopening after proximal aorta repair.

During aortic dissection, the true lumen can be compromised in several ways, including compression, resulting in malperfusion syndrome. Other examples include dynamic obstruction by intima invagination, or static collapse of the aortic true lumen, and dynamic or static occlusion of one or more vital side branches.

With surgical repair of the dissected thoracic aorta, nearly 90% of peripheral pulse deficits can be reversed. However, patients with mesenteric or renal ischemia do not fare as well. The mortality rate of patients with renal ischemia is 50% to 70% and as high as 87% in mesenteric ischemia [1–3].
There appears to be a role for stent graft placement in treatment of static or dynamic obstruction of aortic branch arteries. Static obstruction of a branch can be overcome by placing endovascular stents across the vessel origin, and dynamic obstruction can be relieved by stents in the aortic true lumen with or without additional balloon fenestration [4].

Due to the nature of the static obstruction of SMA, and the start of abdominal pain and the slight abdominal tenderness, it was decided to begin with opening the SMA by a stent before performing the Bentall procedure to prevent the occurrence of bowel ischemia with its high mortality.

References

A Novel Tool to Facilitate Crimping Suture Placement for a Modified David V/Miller Aortic Root Replacement

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Abstract
Surgical treatment of aortic root and ascending aorta aneurysms with aortic insufficiency is still controversial. A valve-sparing operation is the procedure of choice for such patients, and the reimplantation technique is preferable. We describe a simple technique for aortic root reconstruction that has been successfully performed for patients with aneurysms of aortic root and ascending aorta with aortic insufficiency.

Key Words
Aneurysm · Aortic valve · Aortic root · Dissection

Introduction
Surgical treatment of aortic root and ascending aorta aneurysms with aortic insufficiency is still controversial. A valve-sparing operation is the procedure of choice for such patients, and the reimplantation technique is preferable [1]. Nowadays, multiple modifications of the reimplantation technique have been introduced. We describe a simple technique for aortic root reconstruction that has been successfully performed for patients with aneurysms of aortic root and ascending aorta with aortic insufficiency.

After median sternotomy, cardiopulmonary bypass is begun, using the classic scheme “aorta–right atrium” with moderate hypothermia (to 32°C). After completion of cardioplegia, the aneurysm is resected, leaving approximately 4-5 mm of the attached aortic wall, and the coronary arteries are excised with buttons. Then, the fibrous annulus is measured and a vascular graft is prepared for reimplantation. For those manipulations we created a new, simple, and convenient device (Fig. 1). There is a circular stop on the opposite side of the device. Cylinders are changeable and are of different diameters: 21, 23, 25, 27, or 29 mm. The working part of each cylinder has streamlined circular grooves 0.2 mm deep at a distance of 2 mm from each other. The fibrous annulus is measured by inserting the device into the left ventricle through the annulus of the aortic valve (Fig. 2).

We then prepare the vascular graft for reimplantation. An oversized vascular graft (annulus diameter = device chosen size + 8 mm) is necked down proximally to fit an appropriate device using 2-0 horizontal interrupted polyester plication sutures. The assistant introduces the device into the selected prosthesis to the circular stop. The surgeon performs the first proximal suture line. The first line of the suture is located 1-2 mm from the proximal edge of the prosthesis. The suture is tightened so that the graft is partially fixed on one of the device’s circular grooves. The second line is located 3-4 mm above the first line. The ribs of sutures are located in a chess order, thereby resulting in a border of approximately 5 mm (Figs. 3 and 4). The proximal suture line is performed by using 2-0 polyester sutures with pledgets that are passed from inside the left ventricular outflow tract to the outside.
under the aorta cusps. Then the pledgeted sutures pass through the prosthesis base edge as follows: 1-2 mm lower, between, and 1-2 mm higher than the lines formed by two horizontal sutures. Then, the graft is anchored in the aortic root by tying the suture. Thus, a zigzag line of fixation is created along the whole circular length (Fig. 5). Once the graft is anchored in

the aortic root, the commissures are trimmed by placing the stay sutures at the appropriate height inside the vascular graft. The correct position of the commissures inside the graft is identified by pulling on both the commissure and the vascular graft before stitching the sutures through the graft. The graft should extend by half or two thirds of its maximum length at this
segment. When the commissures are trimmed, the tissue remnants of the partially resected sinuses of Valsalva are reimplanted into the vascular graft using 4-0 monofilament running sutures. The aortic root is finally formed by 2-0 polyester-interrupted or plication sutures at the sinotubular ridge level (Fig. 6).

**Clinical Experience**

Between 2011 and 2013, a total of 39 patients (mean age 49 ± 17 years, range 14-70 years; 30 men) with aneurysms of the aortic root and ascending aorta and aortic insufficiency underwent aortic root reconstruction. All patients had aortic regurgitation; 24 pa-

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**Figure 3.** An oversized vascular graft is necked down proximally to fit an appropriate size of the new device using 2-0 interrupted polyester plication sutures. The first line of the suture is located 1-2 mm from the proximal edge of the prosthesis; the second line is located 3-4 mm above the first line.

**Figure 4.** A border of the proximal part of the graft.
tients had 3+ insufficiency. Before the procedure, the end-diastolic diameter of the left ventricle (EDDLV) was $55.3 \pm 6.9$ mm (44-69 mm), and the ejection fraction (EF) was $53.5 \pm 6.8\%$ (34-65 mm). There were no complications related to the aortic valve and root reconstruction observed. All patients survived the surgery and were discharged from the hospital (average, 12 days after surgery). The mean gradient across the valve was $7.8 \pm 2.6$ mm Hg. After the procedure, EDDLV was $52.8 \pm 6.7$ mm (43-65 mm), and EF was $49.1 \pm 8\%$ (30-59 mm). The follow-up extended to 24 months after the operation, and echocardiography results showed no significant aortic valve regurgitation in 34 patients, and a moderate level (grade 2) in 5 patients.

**Figure 5.** The zigzag line of fixation.

**Figure 6.** Echocardiography scan of the aortic root, the coaptation zone (arrow), and the bracing ring (stars).
Additional Surgical Procedures

A total of 11 patients underwent “hemiarch” replacement of the aortic arch, 8 had aortic valve leaflet reconstruction, 4 had concomitant coronary artery bypass grafting, and 1 had reconstruction of the mitral valve.

Discussion

Creation of novel sinuses of Valsalva is the key point of any reimplantation technique for long-term functioning of the new aortic root. Despite the fact that some clinics apply the reimplantation technique (David I) with satisfactory results [2], the overwhelming majority of surgeons prefer to construct novel sinuses in a novel aortic root. The importance of the mechanism of opening and closing of the aortic valve was depicted by Leonardo da Vinci in the 15th century. Risk of damage to the leaflets in a narrow prosthesis without sinuses makes surgeons strive to recreate the original anatomy. The reimplantation technique requires very careful selection of graft size. This topic is the subject of many publications, paying tribute to the operation’s complexity. The Seattle modification partially solved the problem of creation of new sinuses [3], but the Miller modification [4] simplified reimplantation of the aortic valve.

At the beginning of our experience we used a one-line plication suture for narrowing the graft’s proximal part, as described by Dr. Miller. Purposely, we used a graft 8 mm bigger than the native aortic annulus. With this approach, manipulations inside the prosthesis become free. However, the graft receives a significant proximal corrugation. We concluded that one line of plication sutures is unreliable for the anastomosis to hold pressure subsequently. Its strength properties are not sufficient to maintain the shape of the aortic root and to prevent bleeding despite the fact that there are fixing sutures with pledgets going from the left ventricular outflow tract outward. Thus, using two suture lines allows creation of a border of approximately 5 mm. The Miller technique supposes fixation of the graft at the horizontal line. We offer a zigzag line of fixation.

There are several reasons to use the zigzag technique and our new device. First, this technique allows one to minimize risk of bleeding. In our experience we have had one complication that led to gushing bleeding from the anastomosis zone after reestablishment of cardiac function. We managed to fix this problem despite some technical complexity. After this, we started to use our zigzag technique and did not encounter subsequent similar complications. Second, our narrowing of the proximal part of the graft creates crimping like the Miller technique does. The zigzag technique helps us to construct a thick line of contacting surface between the prosthesis and the left ventricular outflow tract. In addition, the zigzag technique helps to reduce excessive plication of fibrous annulus. From the point of view of physics, the advantage of our approach can be justified as follows. Every horizontal filament at the proximal graft has ribs of 5-7 mm lying in turn on the front and back sides. Every rib after its final fixation cements the surface area coaptation. Third, the bracing ring prevents dilatation of the aortic annulus. Fourth, in the beginning of our experience we used valve sizers, but we encountered some problems with sliding of the graft along the plastic sizer during narrowing. We introduced the new device (Patent Pending # 2013107839) and achieved the following advantages: a circular stop helps to fix the graft, and shallow circular grooves prevent the graft from sliding when fixing filaments.

Conclusion

Our approach helps to reconstruct a natural analog of the aortic root, to stabilize the aortic annulus, and to minimize risk of bleeding. This technique is quick, simple, and reproducible with multiple grafts.

Acknowledgments

This work was presented at the 63th International Congress of The European Society for Cardiovascular and Endovascular Surgery (ESCVS), Nice, France, 24-27 April 2014.

Conflict of Interest

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

Comment on this Article or Ask a Question
References


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List of Upcoming Meetings

August 2014

1. 8th Annual Australian and New Zealand Endovascular Therapies Meeting (ANZET14)
   August 20–22, 2014
   Melbourne, Australia

September 2014

1. 2014 American Association for Thoracic Surgery Cardiovascular Valve Symposium
   September 4–6, 2014
   Istanbul, Turkey
   Meeting information available at: www.aats.org/valveistanbul/

2. 24th World Congress of Cardiothoracic Surgeons
   September 6–10, 2014
   Geneva, Switzerland
   Meeting information available at: http://www.wsccts2014.ch

3. 4th International Meeting on Aortic Diseases:
   New insights into an old problem
   September 11–13, 2014
   Liege, Belgium
   Meeting information available at: www.chuliege-imaa.be

4. Mid-Atlantic Aortic Symposium - the regional meeting for the International Society of Endovascular Specialists
   September 27, 2014
   Philadelphia, PA, USA
   Meeting information available at: http://isesonline.org/ises-mid-atlantic.html

October 2014

1. Aortovascular Summit 2014: Cutting Edge Management of Aortic and Vascular Disease from Root to Toe
   October 9, 2014
   New York, New York, USA
   Meeting information available at: http://columbiasurgerycme.org/event_aortic_20141009.html

2. 28th European Association for Cardiothoracic Surgery Annual Meeting
   October 11–15, 2014
   Milan, Italy
   Meeting information available at: www.eacts.org/annual-meeting/

3. Eurovalve 2014
   October 24–25, 2014
   Rome, Italy
   Meeting information available at: www.eurovalvecongress.com

4. American College of Surgeons Clinical Conference
   October 26–30, 2014
   San Francisco, California, USA
   Meeting information available at: www.facs.org