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Progress and Future Challenges in Thoracoabdominal Aortic Aneurysm Management

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Abstract
Background Postoperative paraplegia and paraparesis have been the scourge of thoracoabdominal aortic repair since the inception of the procedure.
Methods and Results In our experience with more than 1,250 cases in 15 years, we have developed adjunctive strategies that have pushed neurologic deficit rates down from over 30% percent in the most extensive cases to less than 2% overall in the modern era. The dramatic reductions in risk reported by ourselves and others have led to recent complacency regarding the importance of this complication, and widening use of endovascular technologies raises the potential specter of a return to suboptimal results.
Conclusions Utmost care is required in the vetting of these new technologies to prevent regression of the hard-won excellent results obtainable by open repair. Postoperative renal failure, in stark contrast to neurologic deficit, has remained resistant to every treatment that has been described in the literature. The fact that we haven't learned to treat it means that we don't understand the mechanisms behind it, and this problem is worthy of a major focused discovery and management effort. We have recently begun a multi-pronged research program to discover basic mechanisms of renal injury so that appropriate and effective treatments can be discovered.

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A thoracoabdominal aortic aneurysm (TAAA) involves the segment of the aorta from the left subclavian artery and various segments of the abdominal aorta. The management of TAAA, since its inception, has been challenged by many operative complications. Among these are paraplegia, paraparesis, multisystem organ failure, and impairment of renal, respiratory, and pulmonary functions. Fortunately, methods of organ protection (particularly for the spinal cord) have been developed to reduce these risks. These techniques have reduced morbidity and mortality rates, resulting in a reasonable outcome for open surgical approaches. This article provides a brief history of the treatment of such aneurysms before discussing the rationale for the protective techniques. A review is given of our operative results, in addition to some of the emerging endovascular techniques becoming available for treatment of select patients.

History

In the late 19th century, the standard treatment for an aortic aneurysm was simply to ligate the aorta. Rupture was prevented, but disastrous consequences often resulted. The patient frequently suffered gangrene and a painful death. The first physician to perform thoracoabdominal aortic aneurysm surgery was Samuel N. Etheredge in San Francisco in 1954 [1]. The aneurysm was excised and a tube was used from the descending to the infrarenal abdominal aorta. In 1956, Michael E. DeBakey and colleagues devised

an ingenious method to treat this condition [2]. First, they used a homograft, and later developed the polyester tube graft for placement in the descending thoracic aorta. DeBakey sutured the Dacron graft to the infrarenal and the descending thoracic abdominal aorta, and sequentially bypassed the celiac axis, superior mesenteric, and both renal arteries. With this method, organ ischemia was limited to 10–15 min during performance of the operation. In 1965, E. Stanley Crawford established the current technique for repair of TAAAs, implementing three important innovations [3]. The first principle was the inclusion technique, as practiced by Matas [4] and Creech [5]. The second innovation involved reattachment of small arteries to larger arteries as a patch, as originally described by Alexis Carrell at the turn of the twentieth century [6] The third principle arose from the experimental canine work of Frank Spencer at Johns Hopkins, which involved the reattachment of the intercostal arteries into the graft in order to prevent paraplegia [7].

Several methods have since been developed to allow a longer cross-clamp time without multi-organ damage. During the clamp-and-sew era, aortic cross-clamp time was the most important predictor of immediate postoperative neurologic deficit during repairs of the thoracic and thoracoabdominal aorta. Analysis of Crawford's population of 1,509 patients demonstrated that a cross-clamp time longer than 60 min was associated with an overall incidence of paraplegia of 27%; incidence when clamp time was less than 30 min was still significant at 8% [8]. We reported similar findings in our own population when the clamp-and-go technique was used [9]. Results from the clamp-and-sew era were superior to those of earlier series, yet still troubling.

The main issue with the clamp-and-sew approach was that this depended on expeditious surgery, precise intraoperative care with cardiovascular anesthesia, and comprehensive postoperative care in a cardiovascular intensive care unit. Ultimately deemed unsatisfactory in the late 1980s and early 1990s, the clamp-and-sew technique was abandoned in favor of adjuncts. In some cases, we used profound hypothermia through the left chest; other surgeons used medications, local hypothermia, and regional hypothermia to the spinal cord. Beginning in 1992, working in animal models, we started evaluating distal aortic perfusion, cerebrospinal fluid drainage, and moderate passive hypothermia. Beginning in 1992, we settled on this method of spinal cord protection in humans.

Natural history

Abdominal aortic aneurysm is among the 15 leading causes of death in the United States [10]. The incidence of thoracic aortic aneurysms is estimated to be 10.4 cases per 100,000 person-years [11]. Aortic aneurysms involve the infrarenal abdominal segment more commonly than any other arterial segment in the human body. Infrarenal abdominal aortic aneurysms occur 3–7 times more frequently than thoracic aortic aneurysms [11–14]. The estimated prevalence of abdominal aortic aneurysms varies between 2.3% and 10.7%, depending on the population studied and the size used to define an aneurysm [12, 14, 15]. The mean age of patients with thoracic aortic aneurysm is between 59 and 69 years, with a male to female preponderance of 2–4:1 [13].

Reports estimate that less than 39% of patients with untreated large thoracic aneurysms survive beyond 5 years, with the majority of deaths due to rupture [13, 16–18]. Thoracic aortic aneurysm studies have shown that rupture is more likely to occur when aneurysms exceed 5 cm, and that the rate of rupture rises with increasing aneurysm size [16, 19–22]. The median size at which a TAAA will rupture is around 7.0 cm [23, 24]. Aneurysms equal to or greater than 8 cm have an 80% risk of rupture within a year of diagnosis [25]. Untreated aortic aneurysms have a 75%–80% lifetime probability of rupture, but the size at which the aneurysm will rupture and the time that will elapse until rupture are not easily estimated.

Protective measures

We classify thoracoabdominal aortic aneurysms according to the modified Crawford classification (Fig. 1). Our promising results using cerebrospinal fluid drainage [9] and distal aortic perfusion for thoracic aortic aneurysm (TAA) repairs [26] led us to hypothesize that increased distal aortic perfusion pressure using left heart bypass in combination with decreased cerebrospinal fluid pressure with drainage might lead to improved spinal cord perfusion and, ultimately, better neurologic outcomes of thoracoabdominal aortic repair. Based on this possibility, over the last 15 years, we have settled on adjunctive procedures consisting of three elements: distal aortic perfusion, in which oxygenated blood is taken from the left atrium directly to the femoral artery via the left lower pulmonary vein, using the centrifugal pump; cerebrospinal fluid drainage; and moderate, passive hypothermia, in which core temperature is allowed to drift to 33°–34°C. Since 1992, we have employed this method to protect the spinal cord and the descending thoracic aorta, as well as the thoracoabdominal aorta. We chose this approach based on a study that showed a significant impact in protecting the spinal cord by means of these three modalities. We refer to the combination of distal aortic perfusion as described above, cerebrospinal drainage, and moderate hypothermia as a single adjunct

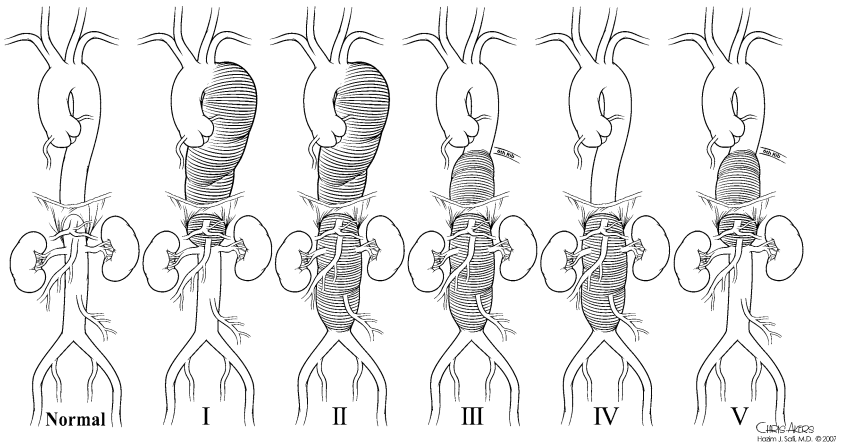

Fig. 1 Modified Crawford Classification of thoracoabdominal aortic aneurysms (TAAA). The classifications of TAAA are as follows: (1) extent I, from left subclavian to above the renal artery; (2) extent II, from the left subclavian to below the renal artery; (3) extent III, from the 6th intercostal space below the renal arteries; (4) extent IV, from T12 to below the renal arteries; (5) extent V, from the 6th intercostal space to above the renals

because the emergent protective effect of these three elements when used together is greater than their individual efficacies.

Procedure

The patient is brought to the operating room and is placed in the supine position on the table. After intubation, a double-lumen tube is placed to block the left lung. A balloon-tip catheter is inserted into the jugular vein. Intravenous lines are inserted in the upper and lower limbs in preparation for blood transfusion (Fig. 2). The patient is then placed in the right lateral decubitus position and the anesthesiologist inserts a catheter between L3 or L4 and advances it 5–10 cm before stabilization. In perioperative CSF drainage, the CSF pressure is maintained at 10 mmHg and below intraoperatively and for 3 days postoperatively (Fig. 3). In distal aortic perfusion, the left atrium is a source of oxygenated blood, and then the femoral artery is perfused. If this artery is not usable, then the distal abdominal aorta or distal thoracic aorta is used. The body temperature is allowed to drift passively to the range of 33°–34°C.

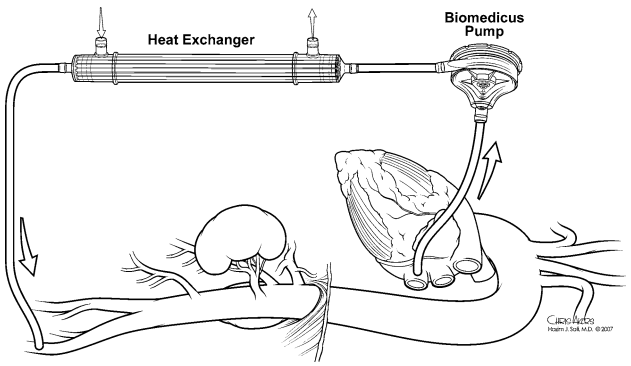

Fig. 2 Blood transfusion circuit

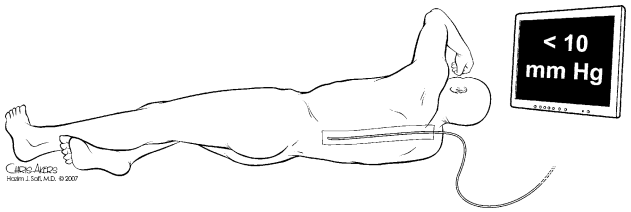

Fig. 3 Patient position for cerebrospinal fluid drainage

Following that, the patient is placed in the thoracoabdominal position with the shoulder blades at right angles to the edge of the table and the hip tilted 45° for access to both groins. The incision is made and is tailored to the extent of the aneurysm. With regard to extent II, III, or IV, the skin incision begins just above the symphysis pubis and extends from the umbilicus to the costal cartilage as shown in the graphic. It is extended 2 cm below the angle of the scapula parallel to the vertebral border of the scapula. The latissimus dorsi and the serratus anterior muscles are cut. The 6th rib is usually removed. Once the pleural cavity is entered, the costal cartilage is excised in wedge fashion and the muscular portion of the diaphragm is cut. Medial rotation of the viscera—the spleen, kidneys, and intestine—exposes the crus of the diaphragm as well as the abdominal aorta, including the orifices of the celiac axis, superior mesenteric artery, and both renal arteries. With the patient exposed, and with a self-retaining retractor in place, the crus of the diaphragm is cut around the aorta allowing the passage of the graft. The lower pulmonary vein is exposed using 3–0 Prolene suture. A cannula is inserted into the left atrium or left lower pulmonary vein directly. This is connected to a centrifugal pump and in-line heat exchanger. The femoral artery is cannulated after exposing it through a longitudinal groin incision. This area is secured. Then, in cephalad fashion, we dissect from the hilum at the wall of the aorta and parallel to vagus, and that leads into the atretic ligamentous. Care is taken to protect the recurrent laryngeal nerve. If the neck is distal to the left

subclavian artery, this is the area that will be clamped. If it is proximal to the left subclavian artery, then we have to prepare the distal arch for clamping. The patient is anticoagulated using 1 mg of heparin per kg of body weight. The clamp is applied either proximal or distal to the left subclavian artery and the mid-descending thoracic aorta. The area in between is opened and the walls are retracted with #2 self-retracting sutures. The proximal descending thoracic aorta distal to the left subclavian artery is excised and lifted away from the esophagus to prevent esophageal aortic fistula. A graft that is appropriate to the size of the aorta is selected and sutured with 3–0 polypropylene suture. When this is done, the mid-descending thoracic aortic clamp is repositioned on the abdominal aorta at the level of the celiac axis and the remainder of the aorta is opened. The aortic hiatus is retracted using a skinny retractor. The lower intercostal arteries from T8 to T12 will be preserved if they are patent. We cut a side-hole into the graft and the graft is sutured around the lower intercostal artery with 3–0 polypropylene sutures. Following completion of the intercostal anastomosis, we move the clamp from the proximal to the distal left subclavian artery on the graft and the flow is restored to the intercostal arteries. Next, we apply the clamp to the infrarenal abdominal aorta if possible. If this is not possible, then the iliac artery is clamped and we open the remainder of the abdominal aorta. We identify the celiac axis, superior mesenteric artery, and both renal arteries. A balloon-tip catheter is used to perfuse the celiac axis, superior mesenteric artery, and both renal arteries. Two separate pumps are used. We also monitor the left renal artery blood pressure, distal femoral artery pressures, and the amount of blood excreted during the clamp. We then retrieve the other end of the graft in the abdominal portion of the aorta by cutting an elliptical hole opposite the visceral vessels. We anastomose the side-hole into the vessels with 3–0 polypropylene sutures. At this point, we clamp the visceral flow, remove the cannula, and finish the anastomosis. The patient is placed in the head-down position and the graft is flushed to remove all air and debris. We then restore pulsatile flow to the viscera and clamp distal to the visceral patch and anastomosis. Following that the graft will be cut to an appropriate length and sutured to the infrarenal abdominal aorta above the aortic bifurcation with running 3–0 polypropylene sutures. When that is finished, the clamp is released and pulsatile flow is restored to the viscera and lower extremities.

Rewarming is begun until the patient's nasopharyngeal temperature is 36°C. Once that is achieved and all of the bleeding sites are secured and the patient is hemodynamically stable, the pump is stopped. The cannulae are removed from the superior pulmonary vein and femoral artery. The suture on the pulmonary vein is secured and the femoral artery is repaired using a 5–0 polypropylene suture. The hole in the superior pulmonary vein is secured and removed from the femoral artery and the femoral artery is repaired using 3–0, 4–0 polypropylene sutures.

The closing is achieved in the usual fashion. We like to insert 3 #36 Argyll chest tubes. The pericostal space will be approximated with 2–0 Ticron. The muscular layers are approximated with #1 PDS. The diaphragm and the linea alba are closed with #1 Prolene. The skin will be approximated with interrupted skin staples. Once this is done and the patient is hemodynamically stable, the patient is placed in the supine position, and the double-lumen tube is exchanged for a single-lumen tube. The patient is then transferred to intensive care for 3 days of postoperative care.

The incidence of neurological deficit (paraparesis and paraplegia) was approximately 15% in patients with extent I, 31% in extent II, 7% in extent III, and 4% in extent IV. If we look at the survival rate in patients with and without neurological deficit, those with neurologic deficit do not live long. As stated above, we settled on use of the combination of CSF drainage, distal aortic perfusion, and passive moderate hypothermia (33°–34°C). The rationale is that during aortic clamping distal to the left subclavian artery, spinal cord perfusion stops as distal aortic perfusion drops below the perfusion pressure and CSF pressure goes up. For that reason, we hypothesized that CSF drainage as used with the distal aortic perfusion would rectify that imbalance [27].

Results in open approach

Between January 1991 and August 2004, we did 1,100 cases of thoracoabdominal or descending thoracic aortic repair. The median age of our patients was 64 years and 33% were women. The preoperative characteristics included hypertension in 73% of patients; coronary artery disease in 27%; renal disease in 19%; acute and chronic dissection in about 30%. Intraoperative variables included intercostal artery reattachment in 39%, mean aortic clamp time of 46 min, and adjunct usage in 74%. In looking at the evolution of thoracoabdominal surgery in time-period quartiles, divided from the first quartile, between 1991 to 1995, the overall incidence of neurologic deficit was 3.3%; without adjuncts, it was 5.7%, and with adjuncts it was 2.4% ($p = 0.008$). In all of the aneurysms, there was a drop in the first quartile from 7.5% to 1.1% (Fig. 4). With regard to extent II, there was a drop from 21% to 4% ($p = 0.0001$) (Fig. 5). Using multilogistic regression analysis, the odds ratios for risk of neurologic deficits were: 6.4 for extent II ($p = 0.0001$), 2.28 for renal dysfunction ($p = 0.03$), 0.26 for use of the adjunct ($p = 0 .0004$), and 1.01 for the duration of clamping ($p = 0.11$). This demonstrated for the

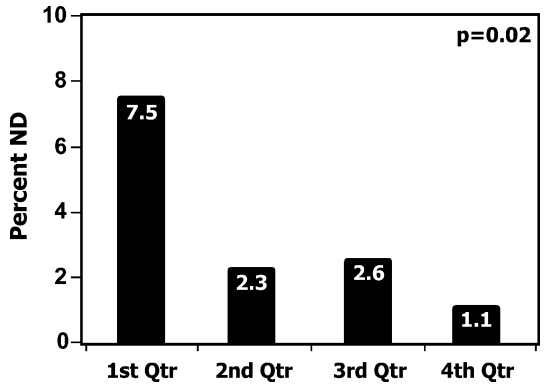

Fig. 4 Incidence (percent) of neurologic deficit (ND) for the entire cohort per quartile

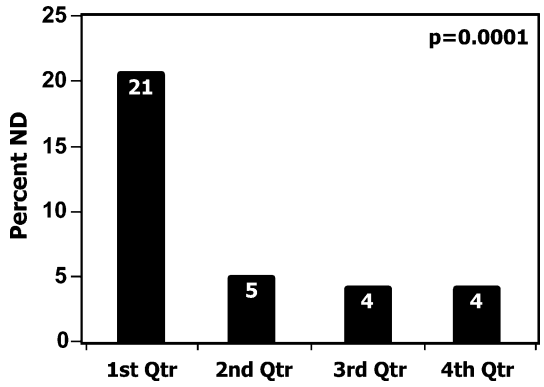

Fig. 5 Incidence (percent) of ND for TAAA extent II cases per quartile

first time that clamping time was no longer a risk factor for neurologic deficit in our group. Based on pre-adjunct-era risk factor analysis of odds ratios, one could classify thoracoabdominal aneurysms into extent II and non-extent II categories, with extent II presenting a less positive outcome. Figures 6 and 7 show the effect of cross-clamp time before and after adjunct use: while extent II continues to be associated with more neurologic deficits, use of adjunct has reduced this problem overall [28, 29].

We found that aortic cross-clamp times have increased significantly (34 s/year) since 1991. However, neurologic deficit rates have declined for the period of surgery in the same period (Figs. 4 and 5). The use of the adjunct has increased the aortic cross-clamp time by a mean of 12 min, but it is associated with a significant protective effect against neurologic deficit. Although other risk factors remain, cross-clamp time is no longer a significant predictor of neurologic deficit in our series (Fig. 7) [28].

In summary, despite the increase in aortic clamp times, adjuncts have reduced the overall risk of neurologic deficit and have blunted the effect of extended aortic cross-clamping, allowing surgeons top operate with less time pressure.

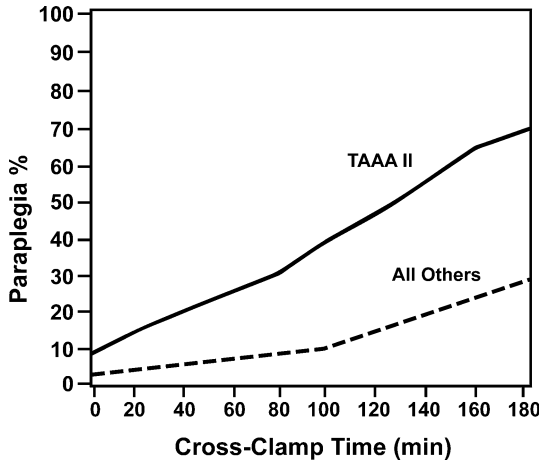

Fig. 6 Multiple logistic regression analysis according to risk of ND and aortic cross-clamp time without adjunct use. Solid line represents extent II TAAA and the dashed line represents all other extents

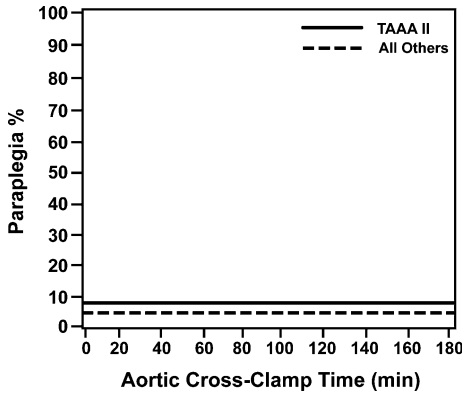

Fig. 7 Multiple logistic regression analysis according to risk of ND and aortic cross-clamp time with adjunct use. Solid line represents extent II TAAA and the dashed line represents all other extents

Advances in endovascular repair

In the last decade and a half, the introduction of endovascular techniques has made revolutionary changes in the way we think about surgical options for complex aortic surgery. Endovascular repair has been widely adopted for the treatment of infrarenal abdominal aortic aneurysms. Today, patients with aneurysms of the descending thoracic aorta can be offered this minimally invasive repair. Hybrid procedures are performed to extend the reach of this technique to aneurysms that include major vessels in the chest or abdomen. The durability of such repairs, however, remains to be determined.

In our practice, high-risk patients with thoracoabdominal aortic aneurysms are considered for a hybrid procedure. High risk is defined by the presence of medical co-morbidities including an ejection fractions less than 30%,

glomerular filtration rate less than 50 ml/min, or forced expiratory volume in less than 0.8 l/s [30].

The hybrid technique involves an open mesenteric revascularization followed by endovascular exclusion of the aneurysm. We bypass the celiac, superior mesenteric, and bilateral renal arteries, either from the distal abdominal aorta or the right or left common iliac arteries. This can be performed via a retroperitoneal or transabdominal approach. Following mesenteric revascularization, we exclude the aneurysmal segment with commercially available thoracic stent grafts. This procedure is applicable to patients with type II, III, and IV thoracoabdominal aortic aneurysms.

Minimally invasive techniques can reduce the morbidity and mortality associated with open aortic surgery, and hybrid procedures extend the application of this approach to a wider group of patients. Further refinement of endovascular techniques, including the introduction of branched and fenestrated technology, holds great promise for the future [31].

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