Open Repair of Chronic Thoracic And Thoracoabdominal Aortic Dissection Using Deep Hypothermia and Circulatory Arrest

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ABSTRACT:

Background: Chronic dissection of the thoracic and thoracoabdominal aorta as sequela of a prior type A or B dissection is a challenging problem that requires close radiographic surveillance and prompt operative intervention in the presence of symptoms or aneurysm formation. Open repair of chronic thoracic and thoracoabdominal aortic dissection using deep hypothermia has been our preferred method to treat this complex pathology. The advantages of this technique include organ and spinal cord protection, the flexibility to extend the repair proximally into the arch and the ability to limit ischemia to all vascular beds.

Methods: Open repair of arch by left thoracotomy, descending thoracic and thoracoabdominal aortic pathology using deep hypothermia was performed in 664 patients from 1995 to 2015. A subset of this cohort had chronic thoracoabdominal aortic dissection (n=196). All non-emergent patients had coronary angiography and echocardiography preoperatively. Significant coronary artery disease or severe aortic insufficiency was addressed prior to repair of the chronic dissection. In recent years, lumbar drains were placed preoperatively in the most extensive repairs extents II and III). Important intercostal arteries from T8 to L1 were revascularized with smaller diameter looped grafts. Multibranched grafts for the visceral segment have been preferred in recent years.

Results: Mean age was 58±14 years. Males comprised 74% of the cohort. Aortopathy was confirmed in 18% of the cohort. Prior thoracic aortic repair occurred in 57% and prior abdominal aortic repair in 14%. Prior type A aortic dissection occurred in 44% and prior type B in 56%. Operative mortality was 3.6%, permanent spinal cord ischemia occurred in 2.6%, permanent hemodialysis in 0% and permanent stroke in 1%. Reexploration for bleeding was 5.1% and respiratory failure requiring tracheostomy occurred in 2.6%. Postoperative length of stay was 11.9±9.7 days. Reintervention for pseudoaneurysm or growth of a distal aneurysm was 6.9%. One, five and ten year survival was 93%, 79% and 57%, respectively.

Conclusions: Open repair of chronic thoracic and thoracoabdominal aortic dissection using deep hypothermia and circulatory arrest has low morbidity and mortality. The need for reintervention is low and long-term survival is excellent. We believe that open repair continues to be the gold standard in patients who are suitable candidates for surgery.

Chronic dissection of the thoracic and thoracoabdominal aorta as sequela of a prior type A or B dissection is a challenging problem that requires close radiographic surveillance and prompt operative intervention in the presence of symptoms or aneurysm formation. Every thoracoabdominal aortic dissection is unique in its combination of primary entry tear, reentry tears, fenestrations, extent of aortic involvement, involvement of branch vessels, flow distribution, and interplay between the true and false lumen. This complex pathology typically does not lend itself to a simple surgical solution.

Open repair of chronic thoracic and thoracoabdominal aortic dissection using deep hypothermia has been our preferred method to treat this complex pathology. The advantages of this technique include organ and spinal cord protection, the flexibility to extend the repair proximally into the arch and the ability to limit ischemic injury to important vascular beds. The disadvantages include longer perfusion times and limited applicability to the ruptured aneurysm. The entire thoracoabdominal dissection can be addressed at once if the entire thoracoabdominal aorta is aneurysmal. Alternatively, only the aneurysmal portion need be replaced with fenestration of the dissection septum at the distal extent of the repair.

This study highlights our experience with open repair of arch by left thoracotomy, descending thoracic and thoracoabdominal aortic dissection using deep hypothermia and circulatory arrest.

Methods:

Institutional Review Board of Indiana University approval was obtained for the study. Individual patient consent was waived. From January 1995 to December 2015, 664 patients underwent open thoracic (by left thoracotomy) or thoracoabdominal aneurysm repair using deep hypothermia and circulatory arrest. The technique has been described elsewhere [1,2]. Briefly, anesthetic technique is composed of total intravenous anesthesia using propofol and remifentanil to allow preservation of motor-evoked and somatosensory-evoked potentials for spinal cord monitoring. Placement of a double-lumen endotracheal tube (Covidien Mallinckrodt Endobronchial tube, left, Medtronic, Inc., Minneapolis, MN)) or single lumen tube with a bronchial blocker (Arndt Endobronchial Blocker Set, Cook Medical Inc., Bloomington, IN) is used for selective airway control. Transcranial infrared oxygen sensors are placed on the left and right forehead (INVOS Cerebral/Somatic Oximeter, Somenetics Corporation, Troy, MI). Motor-evoked and somatosensory-evoked potentials (Cadwell Cascade stimulator-detector, disposable subdermal needle electrodes, Cadwell Laboratories, Inc, Kennewick, WA) are recorded after induction of anesthesia for baseline values and are assessed intraoperatively after separation from cardiopulmonary bypass.

The patient is placed on full cardiopulmonary bypass typically through the left common femoral artery and vein. The arterial inflow temperature is reduced to achieve 15 degrees Centigrade. Once the heart fibrillates, decompression of the left ventricle is performed by placing a drainage catheter (Covidien Argyle, ventricular sump catheter, Medtronic, Inc., Minneapolis, MN) through the left ventricular apex to low active suction. Once the patient has been cooled for 30 minutes with an arterial blood temperature of less than 20 degrees Centigrade, circulatory arrest is performed. The left ventricular sump catheter is turned off, the venous line is clamped and arterial flow is stopped. A cross clamp is placed distal to the proposed proximal anastomosis on the descending thoracic aorta. Hypothermic low flow (1 to 1.5 L/min) is started to the lower body while the proximal anastomosis is performed. If the entire transverse arch is to be replaced, perfusion catheters (Gundry Silicone RCSP cannula, Medtronic, Inc., Minneapolis, MN) are placed in the innominate and left common carotid arteries for bilateral selective antegrade cerebral perfusion at 10 mL/kg/min. After the arch is reconstructed or the proximal anastomosis performed, perfusion is restored to the upper body (1 to 1.5L/min) and the repair progresses step-wise proximally to distally. Intercostal arteries, three to four levels, from T8 to L1 are revascularized. When the abdominal aortic segment is opened, perfusion catheters (9F Pruitt Irrigation Occlusion Catheter, LeMaitre Vascular, Inc. Burlington, MA) are placed into the orifices of the visceral vessels and low flow hypothermic blood perfusion is instituted at 200 to 300 mL/min. After reconstruction of the abdominal visceral segment is completed and the abdominal viscera reperfused, rewarming can commence with commensurate increases in cardiopulmonary bypass flow. Finally, the distal anastomosis is performed at the infrarenal aorta at the bifurcation or prior abdominal aortic graft. If the common iliac arteries are aneurysmal, iliac artery reconstructions are also performed.

As the patient is being rewarmed, the heart is defibrillated when the arterial inflow temperature is above 30 degrees Centigrade. Once rewarmed to tympanic and bladder temperatures above 35 degrees Centigrade, the patient is weaned from cardiopulmonary bypass. Vasopressor support is used to achieve a mean arterial pressure of 70-90 mmHg. If motor evoked potentials are not present in the bilateral lower extremities, the mean pressure is elevated to 90-110 mmHg and a lumbar drain is placed prior to leaving the operating room if it was not placed at the beginning of the procedure. Immediate postoperative hemoglobin is maintained above 8 mg/dL.

Recent technical changes in our surgical strategy for repair of all pathologies of thoracic and thoracoabdominal aneurysm has included the addition of arterial cannulation sites in the ascending aorta, distal aortic arch, proximal descending aorta, left or right common carotid arteries to effort to avoid retrograde flow in the thoracoabdominal aorta. However, the vast majority (188/196) of the patients with chronic dissection had common femoral arterial cannulation for cardiopulmonary bypass. We have also championed the use of branched surgical grafts or smaller diameter grafts to reconstruct the abdominal visceral arteries. Additionally, revascularization of intercostal arteries is now performed using a small diameter looped bypass graft [3]. It is our thought, that the use of smaller diameter grafts will reduce the likelihood of visceral or intercostal patch pseudoaneurysm.

Of 664 patients who had thoracic (by left thoracotomy) or thoracoabdominal aortic aneurysm repair, 196 patients, who comprise the current study, had chronic thoracoabdominal aortic dissection from prior type A or B dissection. Indications for repair included an overall maximal size of the aneurysmal aorta to be at least 50 to 55 mm in diameter or rapid growth of the aorta at a rate greater than or equal to 5 mm per year by computed tomographic imaging (and since 2012 with center-line reconstruction). Patients with a known or suspected connective tissue disorder or a known familial aortopathy were repaired at a thoracic aortic diameter of 50 mm, whereas those without connective tissue disorder or familial aortopathy were repaired at a thoracic aortic diameter of 55 mm. An abdominal aortic component of 50 mm in the setting of a chronic thoracoabdominal aortic dissection was also an indication for surgery. Other indications include persistent and difficult to control hypertension (one patient), chronic mesenteric ischemia (three patients), intractable pain (two patients) and a left common iliac artery aneurysm of 38 mm (one patient).

All patients with elective operations had coronary angiography and echocardiography prior to aortic repair. Significant flow-limiting coronary stenosis was addressed prior to aneurysm repair with percutaneous coronary intervention or coronary artery bypass (sometimes performed concomitantly with aneurysm repair, n=6/196). Severe aortic valve insufficiency required either aortic valve repair or replacement prior to repair of the chronic dissection.

Finally in 2013, we began preoperatively placing cerebrospinal fluid drains in our most extensive repairs, Crawford extents II and III, and in those patients who will have thoracoabdominal repair joining with a prior abdominal aortic graft or thoracic aortic graft. The reason for this change in technique was the increased number of paraplegia and paraparesis in patients with de novo extensive repairs (extents II or III) or completion thoracoabdominal replacement after prior aortic repair. A lumbar drain is not placed preoperatively in patients taking non-aspirin antiplatelet agents or oral anticoagulation or if there is a mycotic aneurysm or systemic infection or if the operation is emergent.

Statistical Analysis

The study is retrospective. Continuous variables are represented as the mean with the standard deviation and/or or median with interquartile range as necessary. Categorical variables are represented as the number and percentage of the cohort. The Social Security Death Index, the Indiana Health Information Exchange and the electronic medical record of our institution were used to identify patients who have died during the study period. Follow up data were retrospectively collected and the last known follow up was recorded. Analysis of survival was performed using the method of Kaplan and Meier. The Kaplan-Meier estimates of survivor function and corresponding 95% confidence interval were calculated and plotted. The statistical software package used was called R: A Language and Environment for Statistical Computing, R Foundation for Statistical Computing, Vienna Austria. (http://www.R-project.org/).

Results:

Preoperative patient characteristics are listed in Table 1. The mean age was 58±14 years. Males comprised 74% of the cohort. Patients with known connective tissue disorder (confirmed Marfan syndrome, Loeys-Dietz syndrome or familial aortopathy) comprised 18% of the cohort. Other comorbid disease is found in Table 1. Chronic thoracoabdominal aortic dissection after prior repair of a Type A aortic dissection occurred in 44%. Chronic thoracoabdominal aortic dissection as a result of a type B aortic dissection occurred in 56%. Prior thoracic aortic procedures occurred in 57% of patients, prior abdominal aortic procedures in 14%.

The preoperative size of the thoracic or thoracoabdominal aorta was 57.2±11.0 mm (median 55 mm, interquartile range 10 mm). Twenty percent of patients had rapid growth (greater than 5 mm increase in size within a 12 month period with an aortic diameter less than 50 mm) as an indication for repair.

The extent of aortic replacement is found in Table 2. Aortic replacement including total arch (proximal anastomosis to the ascending aorta) or partial arch (proximal anastomosis between the innominate and left subclavian arteries) in combination with the descending thoracic aorta comprised 13% of the cohort. Descending thoracic aortic replacement alone (proximal anastomosis distal to the left subclavian artery) occurred in 9%. A Crawford extent I thoracoabdominal aortic aneurysm repair occurred in 31%. Repair that extended through the visceral abdominal aorta was needed in nearly 50% of patients; 35% had extent II repair, 9% extent III and 3% extent IV. Aorto-iliac or aorto-femoral reconstruction was performed in 7%. The mean circulatory arrest time to the cerebrum was 24.7 ± 9.1 minutes and the mean cardiopulmonary bypass time was 290.5 ± 94.6 minutes.

The operative outcomes are listed in Table 3. Overall operative mortality was 3.6%. Neurological deficit occurred in 3%, but only 1% had permanent deficits. Spinal cord ischemia occurred in 11 patients, but only 5 patients (2.6%) suffered permanent paraplegia or paraparesis.

The number of patients who had segmental artery reimplantation was 104 (53%). The need for segmental artery reimplantation was related to the extent of repair: extent I 39 of 61 patients (64%), extent II 53 of 69 patients (77%), extent III 8 of 17 patients (47%), extent IV 2 of 5 patients (40%), and descending with or without arch replacement 2 of 44 patients (5%). The number of patients with spinal cord ischemia who had intercostal reimplantation was 8 (7.7%). The number of patients with spinal cord ischemia who did not have intercostal artery reimplantation was 3 (3.3%, p=0.22 vs. patients with intercostal reimplantation, Fisher's exact test).

We placed lumbar drains preoperatively in only 15 patients (since 2013 when our use of preoperative spinal drains began for those with the highest risk of spinal cord ischemia). Of these, only 2 had transient paralysis or paraparesis and no patient had permanent motor deficit. Lumbar drains were placed in a total of 36 patients (18.4%) either pre or postoperatively and up to 30 days postoperatively. Ten of 36 experienced transient (6) or permanent (4) spinal cord ischemia. Only one patient (0.6%) who did not receive a spinal drain had permanent paralysis.

Acute renal failure was a postoperative complication in 5.1%, any hemodialysis was needed in 4.1%. However, no patients who survived the procedure required permanent hemodialysis. Postoperative pneumonia occurred in 15% of patients. Only 2.6% of patients required tracheostomy. Analysis of major complications and mortality according to the extent of aortic repair is found in Table 3A.

Reexploration for postoperative hemorrhage was needed in 10 patients (5%). Mean postoperative length of stay of survivors (n=189/196) was 11.9±9.7 days. Blood product utilization for the index hospitalization is listed in Table 4. The median blood product administration for the hospitalization was 4 units (interquartile range 13 units).

Follow up after surgery for distal chronic aortic dissection occurs at one month after discharge from the hospital and at 12 months postoperatively with computed tomographic imaging. Subsequent imaging occurs at 30 months and 54 months postoperatively. Imaging can occur at more frequent intervals depending upon aneurysm change or growth or the development of symptoms. Of the operative survivors, 186 out of 189 (98%) had at least one follow up visit. Mean follow up for our series was 46.9±47.4 months (median follow up 30.9 months, interquartile range 59 months). Reintervention was uncommon. Two patients required reoperation for infected aortic grafts. Pseudoaneurysm of either a visceral or intercostal island anastomosis occurred in 6 patients (3.1%). Reoperation for growth of a distal aneurysm was performed in 7 patients (3.6%).

Figure 1 shows the long-term survival of the cohort using Kaplan-Meier analysis including 95% confidence intervals and number of patients at risk. The one, three, five and ten year survival was 93%, 86%, 79% and 57%, respectively. Median survival was 10.7 years.

Discussion:

Open repair of chronic distal thoracic and thoracoabdominal aortic dissection has very good operative outcomes and long-term survival [4-8]. The technique of deep hypothermia and circulatory arrest has low mortality and a low complication rate in thoracic and thoracoabdominal aneurysm repair as seen in our series and other series by Kouchoukos [1,2,9-11]. In the current study, mortality for operative repair of chronic distal aortic dissection was *not* associated with a greater extent of repair or the need for arch replacement. The reason for this is not clear, however hypothermia is imperative in arch replacement and is likely protective against mortality in other extensive repairs. There was more spinal cord ischemia and renal failure in the type II repair cohort, as would be expected in these patients with the most extensive repair, however there was no statistically significant difference compared to lesser extents of repair. Deep hypothermia may be able to equalize mortality across all extents of repair, including arch repair. Deep hypothermia may ameliorate major complications from the most extensive and complex thoracoabdominal repairs, however there were too few events in this series to make meaningful statistical comparisons. Our clinical impression is that the most extensive repairs (extent II and III thoracoabdominal repairs) carry the highest risk of major complication including spinal cord ischemia and renal failure.

One reported potentially catastrophic disadvantage of the technique of deep hypothermia for thoracic and thoracoabdominal aneurysm repair is parenchymal lung hemorrhage requiring lung resection. In separate series by Coselli et al and Safi et al, they reported high mortality rates and hemorrhagic lung complications and advocated against routine use of deep hypothermia [12,13]. We had one patient who required lung resection for intractable parenchymal hemorrhage and this patient did not survive. Adequate decompression of the left ventricle and pulmonary vascular bed during the period of hypothermic fibrillation and minimizing the manipulation of the lung during the period of anticoagulation help to avoid this potentially fatal complication. Although mild parenchymal contusion is not infrequent, it did not prolong ventilation times nor increase the risk of respiratory failure requiring tracheostomy in our series since we are vigilant in ensuring that the left ventricle is adequately decompressed.

The perfusion strategy using deep hypothermia in the repair of chronic distal aortic dissection may have the best surgical outcomes compared to other perfusion strategies [2,6]. The advantages of full cardiopulmonary bypass and deep hypothermia include organ protection including the spinal cord and kidneys, the ability to extend the repair proximally into the arch as needed and the limitation of ischemia to important vascular beds [9-11]. Protection of the spinal cord with the combination of hypothermia and aggressive intercostal reimplantation provides low rates of permanent paralysis. We believe that the addition of preoperative spinal drains in our most extensive repairs can lower our paraplegia rate even further as seen in our most recent extensive repairs after 2013 with no permanent spinal cord deficit.

Perfusion times are certainly longer with the technique of deep hypothermia, but we have not encountered significant postoperative bleeding and coagulopathy as evidenced by our low rate of reexplorations for bleeding and low need for blood product transfusion. Other surgical techniques using left heart bypass cannot address arch pathology proximal to the left subclavian artery. Organ protection can be suboptimal with periods of warm ischemia or hypoperfusion to various vascular beds. Historical series of open repair of chronic distal aortic dissection using normothermic techniques report mortality of 10-15% [14,15]. Mortality using left heart bypass techniques is 8.6% in a contemporary series, however in this series, repair was limited to the descending thoracic aorta [8]. Other contemporary series describe mortality 5.8% to 9.6% using a variety of perfusion techniques, including deep hypothermia and circulatory arrest in a minority of patients [4-6]. Although it is difficult to directly compare perfusion strategies, our series compares favorably to these while including complex arch and aorto-iliac repairs. Deep hypothermia and circulatory arrest has been our preferred technique to repair thoracoabdominal aortic aneurysms of all extents and in particular chronic thoracic and thoracoabdominal aortic dissection.

Open repair of chronic thoracic and thoracoabdominal aortic dissection using deep hypothermia for the suitable operative candidate has low morbidity and mortality. The average age of our cohort was 58 years. This young age reflects our patient population with denovo type B aortic dissection, prior type A dissection, familial aortopathy and connective tissue disorders. Those with connective tissue disorders and familial aortopathy comprise nearly 20% of our cohort. The mean age of those without known connective tissue disorder or known familial aortopathy was 61±12 years, reflecting the relatively young patient with aortic dissection in Indiana.

We intervened on distal chronic aortic dissection when the thoracic aorta was 55 mm in patients without known aortopathy and 50 mm with known or suspected aortopathy. We also intervened on chronic thoracoabdominal dissection when the abdominal aorta was 50 mm. Kim et al stated that the risk of an aortic event of an unrepaired descending or thoracoabdominal aneurysm ranges from 5.5% to 8.0% in aortic diameters as small as 50 mm, 7.2% to 11.2% at aortic diameter 55 mm and 9.3% to 15.6% at aortic diameter 60 mm [16]. We believe that size-for-size, chronic dissection is a more lethal problem than degenerative atherosclerotic aneurysm and have chosen to be more aggressive in repairing them especially given our excellent surgical outcomes. Although repairing a smaller aneurysm may be technically easier and therefore may have a decreased risk of mortality, the size of the aneurysm did not correlate with an increased risk of mortality in our series. In fact, there was no mortality in those who had aneurysms 65 mm or greater (19% of the cohort).

Long-term survival is very good in our study. The ten-year survival is nearly 60%. The need for reintervention is low; very low for infected surgical grafts and surprisingly low for aneurysm formation of the distal aorta. Although it may be possible that our patients were cared for at other institutions for complications of the repair or for distal aneurysm formation, we believe this number to be low since we are the only institution in Indiana who performs these procedures to a great extent.

The limitations of this study are those that are inherent to an observational, single center retrospective review. The outcomes in this study may not be generalizable as the operations described are predominantly performed by 2 surgeons (JW and JC) at one tertiary care hospital. Our analysis of survival using the institutional medical record, the Indiana Health Information Exchange data set and the Social Security Death Index (which is an incomplete data set) is limited by the frequency and completeness of follow up of our patient population. The last known follow up date or date of death was used in the survival analysis with the consequence of a significant number of censored patients.

There is a great interest in using endovascular techniques in the treatment of chronic distal aortic dissection. Endovascular repair of chronic thoracoabdominal aortic dissection can be accomplished with low morbidity and mortality and usually is able to affect false lumen thrombosis and aortic remodeling at the level of the endograft in the thoracic aorta [17-20]. However, aortic remodeling is uncommon in the abdominal aorta and reintervention *is* common [20]. The currently available thoracic endografts are limited in its application to complex thoracoabdominal aortic dissection with multiple visceral arteries coming off the false lumen, multiple fenestrations and severely compressed true lumen. Additionally, the use of thoracic endografts in connective tissue disorders may not be prudent. The long-term outcomes and durability of thoracic endograft repair for chronic distal aortic dissection are not known. In open repair, aortic remodeling is never in question, reintervention is rare, and success is not limited by complex anatomy. The durability of open repair is excellent. The mortality and morbidity of this series compares admirably with that of endograft series of chronic distal aortic dissection, but with markedly less reintervention and proven long-term survival.

Open and endovascular repair should not be competing modalities. Rather, they should be complementary. For the young or otherwise suitable operative candidate, open repair should remain the gold standard. Ideally, all patients with connective tissue disorders should have open repair as well. However, for the patient with advanced age and multiple comorbidities making them a poor surgical candidate, endovascular repair may be the procedure of choice if the anatomy of the aorta is favorable. As the technology for endovascular devices advances and branched and/or fenestrated thoracoabdominal systems become widely available, the paradigm for open versus endovascular repair will likely change.

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References:

1. Fehrenbacher JW, Siderys H, Terry C, et al. Early and late results of descending thoracic and thoracoabdominal aneurysm open repair using deep hypothermia and circulatory arrest. J Thorac Cardiovasc Surg 2010;140:S154-60.
2. Corvera JS, Fehrenbacher JW. Open repair of chronic aortic dissection using deep hypothermia and circulatory arrest. Ann Thorac Surg 2012;94:78-83.
3. Woo EY, Mcgarvey M, Jackson BM, et al. Spinal cord ischemia may be reduced via a novel technique of intercostal artery revascularization during open thoracoabdominal aneurysm repair. J Vasc Surg 2007;46:421-6.
4. Zoli S, Etz CD, Roder F, et al. Long-Term survival after open repair of chronic distal aortic dissection. Ann Thorac Surg 2010;89:1458-66.
5. Pujara AC, Roselli EE, Hernandez AV, et al. Open repair of chronic distal aortic dissection in the endovascular era: Implications for disease management. J Thorac Cardiovasc Surg 2012;144:866-73.
6. Conway AM, Sadek M, Lugo J, et al. Outcomes of open surgical repair for chronic type B aortic dissections. J Vasc Surg 2014;59:1217-23.
7. Van Bogerijen GHW, Patel HJ, Williams DM, et al. Propensity adjusted analysis of open and endovascular thoracic aortic repair for chronic type B dissection: A twenty-year evaluation. Ann Thorac Surg 2015;99:1260-66.
8. Estrera AL, Jan A, Sandhu H, et al. Outcomes of open repair for chronic descending thoracic aortic dissection. Ann Thorac Surg 2015;99:786-94.

9. Kulik A, Castner CF, Kouchoukos NT. Outcomes after thoracoabdominal aortic aneurysm repair with hypothermic circulatory arrest. J Thorac Cardiovasc Surg 2011;141(4):953-60

10. Kouchoukos NT, Masetti P, Murphy SF. Hypothermic cardiopulmonary bypass and circulatory arrest in the management of extensive thoracic and thoracoabdominal aortic aneurysms. Semin Thorac Cardiovasc Surg 2003;15:333-9.

11. Fehrenbacher JW, Hart DW, Huddleston E, et al. Optimal end-organ protection for thoracic and thoracoabdominal aneurysm repair using deep hypothermic circulatory arrest. Ann Thorac Surg 2007;83:1041-46.

12. Safi HJ, Miller CC, Subramaniam MH et al. Thoracic and thoracoabdominal aneurysm repair using cardiopulmonary bypass, profound hypothermia and circulatory arrest via left side of chest incision. J Vasc Surg 1998;28:591-8.

13. Coselli JS, Bozinovski J, Cheung C. Hypothermic Circulatory Arrest: Safety and Efficacy in the Operative Treatment of Descending Thoracic and Thoracoabdominal Aortic Aneurysms. Ann Thorac Surg 2008;85:956-64.

14. Fann JI, Smith JA, Miller DC, et al. Surgical management of aortic dissection during a 30-year period. Circulation 1995;92:II113-21.

15. Safi HJ, Miller CC, 3rd, Reardon MJ, et al. Operation for acute and chronic aortic dissection: recent outcome with regard to neurologic deficit and early death. Ann Thorac Surg 1998;66:402-11.

16. Kim JB, Kim K, Lindsay ME et al. Risk of Rupture or Dissection in Descending Thoracic Aortic Aneurysm. Circulation 2015; 132(17):1620-9.

17. The VIRTUE registry investigators. The VIRTUE registry of type B thoracic dissections – study design and early results. Eur J Vasc Endovasc Surg 2001;41:159-66.
18. Parsa CJ, Williams JB, Bhattacharya SD, et al. Midterm results with thoracic endovascular aortic repair for chronic type B aortic dissection with associated aneurysm. J Thorac Cardiovasc Surg 2011;141:322-7.
19. Xu SD, Huang FJ, Yang JF et al. Early and midterm results of thoracic endovascular aortic repair of chronic type B aortic dissection. J Thorac Cardiovasc Surg 2010;139:1548-53.
20. Leshnower BG, Szeto WY, Pochettino A, et al. Thoracic endografting reduces morbidity and remodels the thoracic aorta in DeBakey III aneurysms. Ann Thorac Surg 2013;95:914-21.

Legends:

Central Picture Legend:

Completed repair of a chronic thoracoabdominal aortic dissection and aneurysm

Figure Legend:

Figure 1. Kaplan-Meier Survivor Function with 95% confidence intervals and number at risk at 3 year intervals.

Video Legend:

Open Repair of a Thoracoabdominal Aortic Aneurysm Using Deep Hypothermia and Circulatory Arrest

	n=196	
Age (years)	57.6±13.8	
Male	145	(74.0)
Marfan syndrome	23	(11.7)
Loeys-Dietz syndrome	2	(1.0)
Familial aortopathy	10	(5.1)
Prior CABG/PCI	44	(22.4)
Hypertension	174	(88.8)
Chronic Obstructive Pulmonary Disease	27	(13.8)
Hyperlipidemia	90	(45.9)
Diabetes Mellitus	23	(11.7)
Chronic Kidney Disease	41	(20.9)
Prior Abdominal Aortic Procedure	28	(14.3)
Prior Thoracic Aortic Procedure	112	(57.1)
Prior Type A Aortic dissection	87	(44.4)
Prior Type B Aortic dissection	109	(55.6)

Table 1. Patient Characteristics. Age is represented as mean ± standard deviation. Values in parenthesis represent percentages.

	n=196	
Descending Thoracic Aorta	44	(22.4)
Descending only	18	(9.2)
Partial arch + descending	15	(7.7)
Total arch + descending	11	(5.6)
Crawford Extent I	61	(31.1)
Crawford Extent II	69	(35.2)
Crawford Extent III	17	(8.7)
Crawford Extent IV	5	(2.6)

Table 2. Extent of Aortic Replacement. Values in parenthesis represent percentages.

	n=196	
Operative Mortality	7	(3.6)
Permanent Stroke	2	(1.0)
Transient Neurological Deficit	4	(2.0)
Permanent Paraplegia/Paraparesis	5	(2.6)
Transient Spinal Cord Ischemia	6	(3.1)
Acute Renal Failure (STS definition)	10	(5.1)
Any Hemodialysis	8	(4.1)
Permanent Hemodialysis	0	(0)
Respiratory Failure, Tracheostomy	5	(2.6)
Pneumonia	29	(14.8)
Reexploration for Bleeding	10	(5.1)
Postoperative Length of Stay (days)	11.9±9.7	

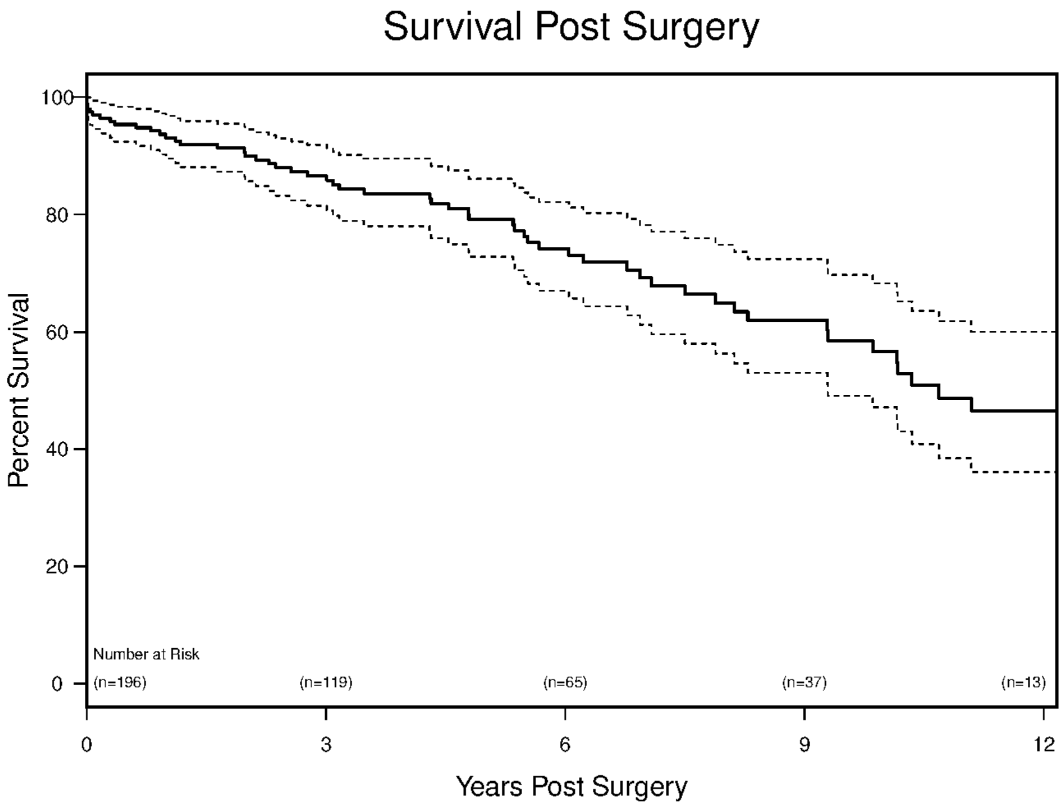
Table 3. Operative Results. Postoperative length of stay is represented as mean ± standard deviation. Values in parenthesis represent percentages.

	Desc only (n=18)	Partial arch + desc (n=15)	Total arch + desc (n=11)	Extent I (n=61)	Extent II (n=69)	Extent III (n=17)	Extent IV (n=5)
Mortality	2 (11.1)	1 (6.7)	0 (0)	1 (1.6)	2 (2.9)	1 (5.9)	0 (0)
Paralysis/paraparesis	1 (5.6)	0 (0)	0 (0)	1 (1.6)	7 (10.1)	2 (11.8)	0 (0)
Permanent	1 (5.6)	0 (0)	0 (0)	0 (0)	2 (2.9)	2 (11.8)	0 (0)
Temporary	0 (0)	0 (0)	0 (0)	1 (1.6)	5 (7.2)	0 (0)	0 (0)
Acute renal failure	2 (11.1)	0 (0)	0 (0)	0 (0)	7 (10.1)	1 (5.9)	0 (0)
Any hemodialysis	2 (11.1)	0 (0)	0 (0)	0 (0)	5 (7.2)	1 (5.9)	0 (0)
Stroke	0 (0)	0 (0)	0 (0)	0 (0)	2 (2.9)	0 (0)	0 (0)
Tracheostomy	1 (5.6)	1 (6.7)	1 (9.1)	0 (0)	2 (2.9)	0 (0)	0 (0)

Table 3A. Major complications and mortality by extent of aortic repair. Values in parenthesis represent percentages.

	Mean	Median	25th %ile	75th %ile
Packed Red Blood Cells (units)	4.8±6.1	3	1	6
Fresh Frozen Plasma (units)	2.1±3.0	0	0	4
Platelet Pheresis (units)	1.2±1.5	1	0	2
Cryoprecipitate (10 units)	0.9±1.6	0	0	2

Table 4. Blood product utilization for the hospitalization.


Survival Post Surgery
Percent Survival
100
80
60
40
20
0
Number at Risk
(n=196)
(n=119)
(n=65)
(n=37)
(n=13)
0
3
6
9
12
Years Post Surgery