

LEARNING OBJECTIVES

- Describe the etiology, classification, and screening for thoracic aortic aneurysms and thoraco-abdominal aortic aneurysms, and their screening
- Discuss the risk factors for rupture of these aneurysms
- Review the surgical techniques and operative strategies used to treat these types of aneurysms

Indications and surgical strategy for thoracic aortic aneurysm repair

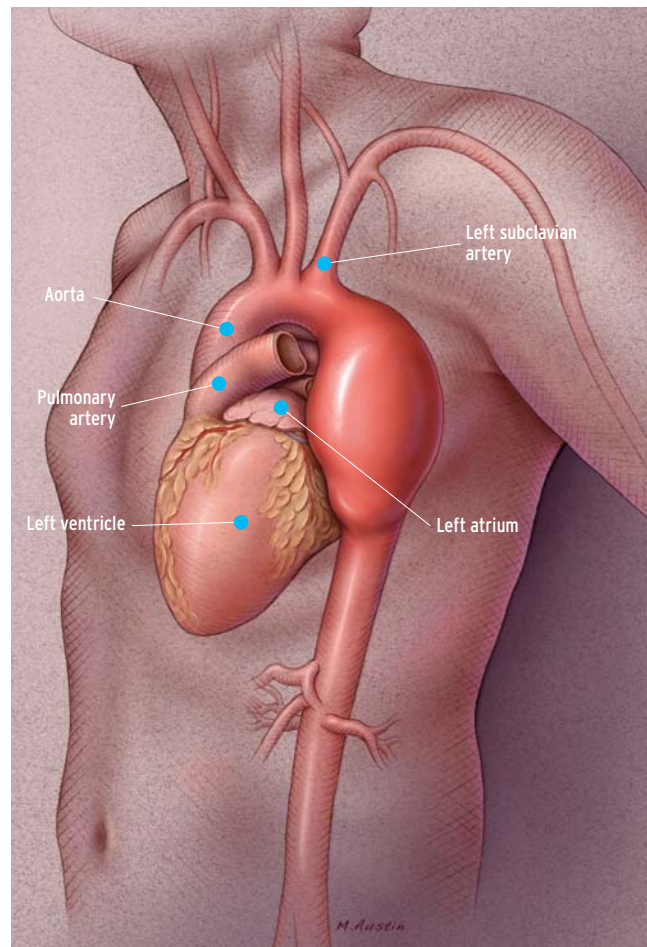
Thoracic and thoracoabdominal aortic aneurysms are a challenging clinical situation. Rupture is most often fatal, but a systematic approach to surgical repair can achieve a good outcome.

Debra Kleinschmidt, PhD, PA; Konstadinos Plestis, MD; Panagiotis Housits, MD

Surgical repair of thoracic aortic aneurysms (TAAs) and thoracoabdominal aortic aneurysms (TAAAs) are challenging procedures that require a systematic approach in order to prevent devastating complications, especially ischemic spinal cord injury. Advances in surgical technique and organ protection have improved the outcomes of these procedures. Standardized protective strategies, such as monitoring somatosensory evoked potentials (SSEPs) and motor evoked potentials (MEPs), mild hypothermia, distal aortic perfusion, CSF drainage, spinal cord perfusion monitoring, and selective implantation of segmental spinal cord arteries, also improve outcomes. This article describes a current approach to TAA and TAAA repair and analyzes the outcomes.

Classification of aneurysms Surgical repair of TAAs and TAAAs were first performed in the early 1950s. Historically, the procedures are associated with a high mortality rate and a significant risk of paraplegia and multiple organ complications.¹ E. Stanley Crawford, MD, and colleagues defined four classifications for TAAAs based on location and the extent of the aneurysm² (Figure 1). According to the Crawford classification, a **type I aneurysm** originates distal to the left subclavian artery and involves the visceral arteries. **Type II** involves the entire aorta distal to the left subclavian artery; **type III** involves the distal half of the descending thoracic aorta and the entire abdominal aorta; and **type IV** involves the infradiaphragmatic aorta. Types I and II are associated with the highest rates of mortality and paraplegia.²

Treatment decisions are based on balancing the risk of death from aortic rupture with the risk of mortality and com-



Thoracic aortic aneurysm distal to the left subclavian artery

© Mike Austin

plications from the procedure. Surgical results have improved significantly over the past 10 years. Rupture of these aneurysms is invariably fatal.^{3,4} Prediction of the probability of rupture may improve selection of patients for elective surgery, as rupture rates in patients not treated surgically are high, ranging from 21% to 74%.^{5,6}

Specific risk factors that increase the likelihood of rupture have been identified from studies of the history of TAAs and TAAAs. An individualized yearly risk of rupture can now be calculated based on specific dimensional and nondimensional variables. Selected patients with an operative risk that is significantly lower than the calculated risk of rupture are candidates for elective surgery.⁷ Of note, the Crawford classification of aneurysms is commonly mistaken for the DeBakey or Stanford aortic dissection classifications.

Primary care and screening Typically, aneurysms are an incidental finding; a widening of the aorta is noticed on plain radiography of the chest during a workup for something else. The presenting symptoms can be vague, especially in the case of a TAA. However, a history of Marfan syndrome or Ehlers-Danlos syndrome is a reason to suspect an aneurysm because the symptoms can be vague and confused with chest, back, or abdominal pain; therefore, aortic aneurysm should be included in the differential diagnosis. A comprehensive history and astute physical examination should assist in determining the likelihood of an aneurysm. Once aneurysm is diagnosed or suspected, CT angiography should be ordered to establish a definitive diagnosis and a baseline for comparison over time. In addition, referral to an aortic-thoracic surgeon is recommended.

RISK FACTORS FOR RUPTURE

Size and expansion rate The size of an aneurysm is a major predictor of rupture. A review of the history of TAAs found that rupture is much more likely to occur when an aneurysm exceeds 5 cm in diameter, and risk of rupture increases as the aneurysm expands. The probability of TAAA rupture is 40% when the diameter is more than 7 cm, and 46% of patients with large aneurysms will die within 5 years. Among patients with an aneurysm larger than 6 cm, the annual rate for rupture or dissection is 6.9% and for death is 15.6%. The rate of aneurysm expansion is also a

significant variable. A fast rate of growth—more than 0.5 cm per year—is considered to be an independent risk factor for rupture. Elective surgery eliminates the risk of rupture and restores survival to near normal.⁸

Smoking and chronic obstructive pulmonary disease (COPD) In our multivariate analysis of nondissecting TAA and TAAA, a history of COPD increased the odds of rupture by a factor of 3.6 ($P = .04$). A history of smoking is a risk factor for rupture; however, COPD has eclipsed smoking in

“Surgery is recommended for a degenerative aneurysm if the risk of rupture within 1 year exceeds estimated surgical risks.”

several studies in which the two were reviewed in the same multivariate analysis.^{6,7} Aneurysm growth was significantly more rapid in smokers, justifying a recommendation for smoking cessation to all patients with aneurysmal disease.⁹

Age The risk for aneurysm rupture increases with age.¹ TAAs are more prevalent in males; however, the proportion of women with TAAs increases with age, and nearly equals that of males in the elderly. The relative risk of rupture increased by a factor of 2.6 for every decade of life ($P = .02$).⁶

Pain The presence of pain in patients with TAA or TAAA, even if it is vague and uncharacteristic, was associated with subsequent rupture. Our multivariate analysis showed an odds ratio of 2.3 ($P = .04$).⁶

Hypertension A history of hypertension, specifically diastolic hypertension, correlates highly with the initial development of an aneurysm.⁷ Treatment with beta-adrenergic blocking agents and antihypertensive agents is recommended to minimize the progression of aneurysmal disease.¹⁰

Renal failure Renal failure is a risk factor for rupture of any aortic aneurysm—thoracic, thoracoabdominal, or abdominal.^{2,5,11} In our own study, however, renal failure did not emerge as a significant risk factor for aneurysm rupture.

Continued on page 46

KEY POINTS

- Surgical repair of thoracic aortic aneurysms and thoracoabdominal aortic aneurysms require a systematic approach in order to prevent devastating complications, especially ischemic spinal cord injury.
- Treatment decisions are based on balancing the risk of death from aortic rupture with the risk of mortality and complications from the procedure.
- Some persons with peripheral vascular disease and persons who are at high risk for complications from cardiopulmonary bypass are possible candidates for endovascular repair.
- Thrombotic or atheromatous debris dislodging into segmental vessels may result in embolization to small end vessels within the spinal cord. This possible mechanism of injury to the spinal cord offsets the benefits of an endovascular repair. Perceived benefits of this procedure are the absence of any aortic occlusion and less hemodynamic instability.

CHRONIC DISSECTIONS VERSUS DEGENERATIVE ANEURYSMS

Aneurysms in patients with known connective tissue disorders, particularly Marfan syndrome and Ehlers-Danlos syndrome, are more likely to rupture than are aneurysms in patients who do not have connective tissue disorders. Patients with a family history of dissection and/or rupture may be particularly at risk.

Evaluations of patients who experienced rupture revealed that those with chronic Stanford type B dissection had smaller maximal diameters in the descending aorta before rupture than patients with a degenerative aneurysm. Aortic diameter in patients with chronic dissection was a median of 5.4 cm versus 5.8 cm in patients with degenerative aneurysms ($P = .05$).^{6,11}

An equation for estimating the probability of rupture within 1 year that incorporates the patient's age, history of COPD, presence of pain, and the maximal thoracic and abdominal aneurysm diameters was developed^{6,7,12} (Figure 2). Surgery is recommended for patients with degenerative aneurysms if the risk of rupture within 1 year exceeds estimated surgical risks.

SURGICAL TECHNIQUE

The surgical technique utilized in our study was as follows. The patient is placed in the standard thoracoabdominal position. A double-lumen endotracheal tube is placed to isolate the left lung. A right radial arterial line, a right common femoral line, and a pulmonary artery catheter are established for continuous monitoring of BP and heart function during the procedure. Intraoperative transesophageal echocardiography (TEE) provides excellent imaging of the left ventricle for monitoring the ejection function. TEE is also used to assess heart function

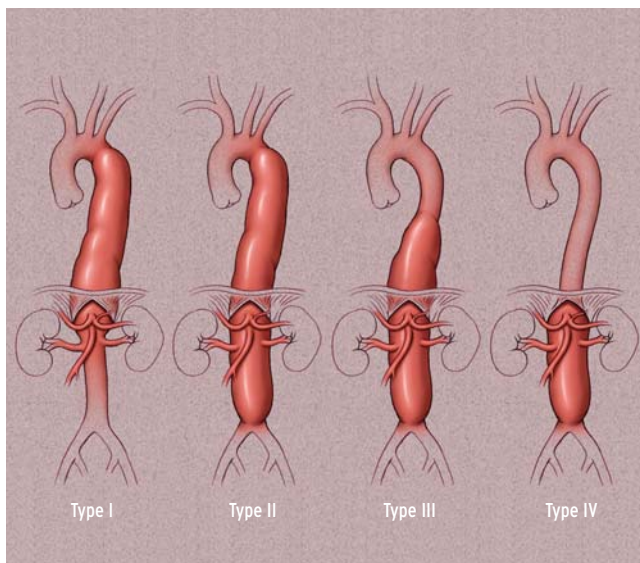


FIGURE 1. Crawford classifications of thoracoabdominal aortic aneurysm

“Careful attention to perfusion variables is important to maintain adequate blood flow to the spinal cord via the collateral network.”

before stopping and after rewarming/restarting. A spinal catheter is placed to monitor CSF pressure during the procedure; monitoring is continued for 72 hours after surgery. Intraoperative SSEP/MEP monitoring is utilized to monitor perfusion. SSEP monitoring is continued for 12 hours after surgery. Distal aortic perfusion is used in most patients; however, full cardiopulmonary bypass and deep hypothermic circulatory arrest are required when the proximal clamp cannot be placed.

The aorta is accessed via a left thoracotomy or thoracoabdominal incision. The diaphragm is divided circumferentially, and the infradiaphragmatic aorta is exposed via a retroperitoneal approach. The diseased aorta is replaced with a Dacron hemashield graft using a sequential clamping technique. If needed, the visceral arteries are reimplanted using a separate trifurcation graft.

SPINAL CORD PROTECTION

The most devastating complication of surgical intervention for TAA and TAAA is paraplegia. In 1993, Svensson and colleagues reported on a series of 1,509 patients who underwent surgical resection of TAA and TAAA; the overall incidence of paraplegia or paraparesis was 16% (234).¹³ Significant predictors ($P < .05$) of paraplegia or paraparesis were total aortic clamp time, extent of aorta repaired, aortic rupture, patient age, proximal aortic aneurysm, and a history of renal dysfunction.

Major improvements have been made in mortality and paraplegia rates. Our clinical approach to TAAA resection is dictated by the hypothesis that spinal cord perfusion depends on an integrated collateral network that supplies input from the subclavian, hypogastric, and intercostal arteries, rather than the presence of a single major spinal artery.

The influence of **hypothermia** in decreasing the metabolic demands of the spinal cord has been assessed. In a pig, extensive ligation of segmental arteries can be achieved when using spinal cord monitoring and a moderate degree of hypothermia. Careful attention to **perfusion variables** is important to maintain adequate blood flow to the spinal cord via the collateral network. In addition, maintaining an intrathecal pressure of less than 10 cm H₂O with CSF drains allows for optimal reduction of flow resistance within the spinal cord.¹⁴

Moderate systemic hypothermia, distal aortic perfusion, CSF drainage, SSEP/MEP monitoring, guided segmental artery ligation, and continuous measurements of spinal cord perfusion pressure are key factors of spinal cord protection.

Intraoperative SSEP/MEP monitoring allows for immediate interventions if perioperative changes in perfusion occur.

Distal aortic perfusion During cross-clamping of the TAA, blood can be directed in the distal aorta with the use of a bio-pump system (Bio-Medicus Perfusion System; Medtronic, Minneapolis, Minnesota). Inflow into the bypass circuit is usually via the right atrium, outflow into the distal aorta is achieved most frequently via the left common femoral artery. Distal aortic perfusion is necessary because the hypogastric arteries are an important source of collateral blood supply to the spinal cord. The femoral artery pressure is monitored in order to maintain optimal distal aortic perfusion, hence spinal cord perfusion. Generally, flow rates of 2 to 3 L/min are necessary to achieve a distal mean arterial pressure of 60 to 80 mm Hg. The efficacy of distal perfusion in reducing paraplegia following extensive TAAA resection has been documented. Simultaneous maintenance of selective perfusion of the visceral arteries may have a beneficial effect in preventing renal insufficiency as well as liver and intestinal injury, which can potentially lead to severe coagulopathy and multisystem organ failure.

Pharmacologic agents Almost all resection techniques for TAAs and TAAAs involve some unavoidable intervals of cord ischemia. Perioperative administration of corticosteroids can increase spinal cord tolerance to ischemia. Mannitol (Osmitrol, generics) and naloxone may have beneficial effects.¹⁵

Passive hypothermia Moderate hypothermia (32°C) has been shown to decrease the metabolic demands of the spinal cord, thereby increasing tolerance to spinal cord ischemia from 20 to 50 minutes in pigs.¹⁶ Longer ischemic intervals can be tolerated at lower temperatures, as evidenced by the protective effect of profound hypothermia on the spinal cord during repair of extensive TAAA.^{17,18}

CSF drainage The titration of CSF drainage is proven to decrease postoperative spinal cord ischemia. However, excessive CSF drainage can create significant CNS complications. Invariably, CSF is drained at a rate of 10 cc/h to keep the pressure below 10 mm Hg.

Spinal cord monitoring SSEP and MEP changes can be a sensitive indicator of spinal cord ischemia during TAA and TAAA repair.¹⁹ Continuous monitoring of spinal cord function during these procedures and in the immediate postoperative period is essential to reduce the risk of neurologic injury.²⁰ In most cases, when cord ischemia is detected by SSEP or MEP monitoring during the surgical procedure, it can be corrected by raising the arterial pressure or decreasing the CSF pressure. Reimplantation of intercostal arteries may be necessary in select patients. Monitoring the spinal cord perfusion pressure by direct cannulation of the L1 segmental artery allows for continuous perioperative assessment of collateral network adequacy.

Segmental vessel reimplantation Reimplanting the segmental vessels as a means of reducing the incidence of spinal cord injury appears to be physiologically sensible. However, in a recent series of 100 TAA and TAAA resec-

tions, the incidence of paraplegia was only 2% when the segmental vessel originating from the aneurysm sac was ligated before opening the aorta.¹⁶ Perioperative SSEP/MEP monitoring can identify those patients in whom spinal cord blood flow relies on the segmental arteries. In these cases, a separate graft to reimplant T8-L1 segmental arteries is recommended.

Our recent experimental data shows that metabolic and hemodynamic manipulation should enable routine sacrifice of all segmental arteries without injuring the spinal cord.²¹ This becomes significant when a stent graft is used to treat a TAA.

Postoperative hemodynamic stability Large volumes of fluid are routinely administered during TAA or TAAA repair in order to maintain an adequate preload and avert possible spinal cord ischemia.²² High perfusion pressures enhance spinal cord perfusion through the collateral network; therefore, maintaining a mean arterial pressure of 85 to 95 mm Hg during the immediate postoperative period is recommended. Maintaining normal hematocrit levels and careful attention to ventilation and oxygenation maximizes oxygen delivery.

Spinal cord injury can also occur during the postoperative period. Systemic hypotension can cause inadequate spinal cord perfusion. Delayed paraplegia can be prevented by maintaining systemic BP, keeping the mean arterial pressure at 85 to 95 mm Hg, prompt CSF drainage via the established spinal catheter, and fluid management.

ENDOVASCULAR REPAIR

Endovascular prostheses received FDA approval as an alternative to open aneurysm repair in 1999. Some persons with peripheral vascular disease and persons who are at high risk for complications from cardiopulmonary bypass are possible candidates for endovascular repair. Each surgical institution has its own criteria for patient selection. Criteria for determining the medical necessity for an endovascular stent in the descending aorta include adequate iliac and femoral access, an aortic diameter of 23 to 37 mm, and at least 2 cm of nonaneurysmal aorta on both ends of the aneurysm. Many TAAs and TAAAs are not anatomically suitable for endovascular repair. The final decision is based on an individualized balance of the risks and benefits.

Continued on page 48

$$\ln \lambda = -21.055 + (0.0093 \times \text{age}) + (0.841 \times \text{pain}) + (1.282 \times \text{COPD}) + (0.643 \times \text{desc dia}) + (0.405 \times \text{abd dia})$$

$$\text{Probability of rupture within 1 year} = 1 - e^{-\lambda(365)}$$

FIGURE 2. Formula for calculating the probability of rupture of a degenerative thoracic aortic aneurysm^{7,12}

Key: Abd dia, maximal abdominal aortic diameter in cm; age, in years; COPD, chronic obstructive pulmonary disease (0=absent, 1=present); desc dia, maximal descending aortic diameter in cm; and pain, 0=absent, 1=present.

Endovascular treatment of TAAs and TAAAs usually precludes segmental vessels within the stented segment from contributing to spinal cord blood supply, resulting in sudden complete occlusion of a large number of segmental vessels at normothermic temperatures. Thrombotic or atheromatous debris dislodging into segmental vessels may result in embolization to small end vessels within the spinal cord. This possible mechanism of injury to the spinal cord offsets the benefits of an endovascular repair. The perceived benefits of endovascular repair are the absence of any aortic occlusion and less hemodynamic instability. A recent review found that the incidence of spinal cord injury after endovascular repair of either all or a portion of the descending thoracic aorta is 2.7%, which is equivalent to the current outcomes of open aneurysm resection.²³

OUR RESULTS

Our study included 219 patients who underwent TAA repair (n = 79; 36%; 23 procedures were elephant trunk completions) and TAAA repair (n = 140; 64%) between June 2002 and June 2005 at Mt Sinai Hospital (130 procedures) and Montefiore Hospital (89 procedures), both in New York, New York. The mean age of the patients was 63.8 years (range, 18-88 years). Preoperative risk factors included a history of hypertension (212 patients), a history of smoking (110), COPD (53), a history of cerebrovascular accident (34), chronic renal insufficiency (27, 8 of whom required preoperative hemodialysis), and insulin-dependent diabetes mellitus (24). In all, 144 patients underwent elective surgery because of enlargement of a known aneurysm, 34 patients had urgent surgery, and 41 patients underwent emergent repairs because of rupture. Fifty-two percent of the TAAAs were classified as Crawford I, 10% were Crawford II, 11% were Crawford III, and 7% were Crawford IV.

Four patients developed postoperative paraplegia. Postoperative MI occurred in five patients, and 13 patients suffered a postoperative stroke. Sixty patients experienced respiratory complications with prolonged postoperative ventilation (longer than 48 hours); 24 patients required a tracheostomy. Eight patients had acute renal failure requiring postoperative dialysis; **Table: Postoperative complications and hospital stay, stratified by institution** (in the online version of this article) summarizes our results. Mortality rates were 5.1% (4 of 79) for TAA repair and 6.4% (9 of 140) for TAAA repair.

CONCLUSION

Surgical repair of TAAs and TAAAs can achieve acceptable mortality and morbidity outcomes when a multidisciplinary

approach to surgery and postoperative care is used. Organ-specific protective measures should be used to prevent postoperative complications. In addition, using specific variables to calculate the risk of rupture can identify those patients who will derive the most benefit from undergoing extensive repair procedures. **JAAPA**

Debra Kleinschmidt works at New York Presbyterian Hospital, Weill Cornell Department of Transplant Surgery in New York, New York. She practiced in cardiothoracic surgery at Lenox Hill Hospital at the time this article was written. **Konstadinos Plestis** is a cardiothoracic surgeon at Lenox Hill Hospital, New York, New York. **Panagiotis Housits** was a fellow in cardiothoracic surgery at Mt Sinai Hospital, New York, New York at the time this article was written. The authors have indicated no relationships to disclose relating to the content of this article.

REFERENCES

1. Etheridge SN, Yee J, Smith JV, et al. Successful resection of a large aneurysm of the upper abdominal aorta and replacement with homograft. *Surgery*. 1955;38(6):1071-1081.
2. Crawford ES, Crawford JL, Safi HJ, et al. Thoracoabdominal aortic aneurysms: preoperative and intraoperative factors determining immediate and long-term results of operations in 605 patients. *J Vasc Surg*. 1986;3(3):389-404.
3. Bickerstaff LK, Pairolera PC, Hollier LH, et al. Thoracic aortic aneurysms: a population-based study. *Surgery*. 1982;92(6):1103-1108.
4. Johansson G, Markström U, Swedenborg J. Ruptured thoracic aortic aneurysms: a study of incidence and mortality rates. *J Vasc Surg*. 1995;21(6):985-988.
5. Pressler V, McNamara JJ. Thoracic aortic aneurysm: natural history and treatment. *J Thorac Cardiovasc Surg*. 1980;79(4):489-498.
6. Clouse WD, Hallett JW Jr, Schaff HV, et al. Improved prognosis of thoracic aortic aneurysms: a population-based study. *JAMA*. 1998;280(22):1926-1929.
7. Griep RB, Ergin MA, Galla JD, et al. Natural history of descending thoracic and thoracoabdominal aneurysms. *Ann Thorac Surg*. 1999;67(6):1927-1930.
8. Davies RR, Goldstein LJ, Coady MA, et al. Yearly rupture or dissection rates for thoracic aortic aneurysms: simple prediction based on size. *Ann Thorac Surg*. 2002;73(1):17-27.
9. MacSweeney ST, Ellis M, Worrell PC, et al. Smoking and growth of small abdominal aortic aneurysms. *Lancet*. 1994;344(8923):651-652.
10. Juvonen T, Ergin MA, Galla JD, et al. Risk factors for rupture of chronic type B dissections. *J Thorac Cardiovasc Surg*. 1999;117(4):776-786.
11. Masuda Y, Takashi K, Takasu J. Expansion rate of thoracic aneurysms and influencing factors. *Chest*. 1992;102(2):461-466.
12. Juvonen T, Ergin MA, Galla JD, et al. Prospective study of the natural history of thoracic aortic aneurysms. *Ann Thorac Surg*. 1997;63:1533-1544. <http://ats.ctsnetjournals.org/cgi/content/full/63/6/1533>. Accessed January 8, 2010.
13. Svensson LG, Crawford ES, Hess KR, et al. Experience with 1509 patients undergoing thoracoabdominal aortic operations. *J Vasc Surg*. 1993;17(2):357-368.
14. Cinà CS, Abouzahr L, Arena GO, et al. Cerebrospinal fluid drainage to prevent paraplegia during thoracic and thoracoabdominal aortic aneurysm surgery: a systematic review and meta-analysis. *J Vasc Surg*. 2004;40(1):36-44.
15. Laschinger JC, Cunningham JN Jr, Cooper MM, et al. Prevention of ischemic spinal cord injury following aortic cross-clamping: use of corticosteroids. *Ann Thorac Surg*. 1984;38(5):500-507.
16. Strauch JT, Lauten A, Spielvogel D, et al. Mild hypothermia protects the spinal cord from ischemic injury in a chronic porcine model. *Eur J Cardiothorac Surg*. 2004;25(5):708-715.
17. Spielvogel D, Halstead JC, Meier M, et al. Aortic arch replacement using a trifurcated graft: simple, versatile, and safe. *Ann Thorac Surg*. 2005;80(1):90-95.
18. 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(4):333-339.
19. Jacobs MJ, Mess W, Mochtar B, et al. The value of motor evoked potentials in reducing paraplegia during thoracoabdominal aneurysm repair. *J Vasc Surg*. 2006;43(2):239-246.
20. Cunningham JN Jr, Laschinger JC, Merkin HA, et al. Measurement of spinal cord ischemia during operations upon the thoracic aorta: initial clinical experience. *Ann Surg*. 1982;196(3):285-296.
21. Halstead JC, Wum M, Etz C, et al. Preservation of spinal cord function after extensive segmental artery sacrifice: regional variations in perfusion. *Ann Thorac Surg*. 2007;84(3):789-794.
22. Etz CD, Leuhr M, Kari FA, et al. Paraplegia after extensive thoracic and thoracoabdominal aortic aneurysm repair: does critical spinal cord ischemia occur postoperatively? *J Thorac Cardiovasc Surg*. 2008;135(2):324-330.
23. Cheung AT, Pochettino A, McGarvey ML, et al. Strategies to manage paraplegia risk after endovascular stent repair of descending thoracic aortic aneurysms. *Ann Thorac Surg*. 2005;80(4):1280-1288.



ON THE WEB

- **Table: Postoperative complications and hospital stay, stratified by institution**

Please see the online version of this article at www.jaapa.com for this enhancement.

TABLE. Postoperative complications and hospital stay, stratified by institution

Postoperative complication	Percentage of study population		
	Total (n = 219)	Mt Sinai (n = 130)	Montefiore (n = 89)
MI	2%	1%	4%
NEUROLOGIC DYSFUNCTION			
• Paraplegia	2%	2%	2%
• Stroke	6%	5%	8%
RENAL INSUFFICIENCY			
• Creatinine >2.5 mg/dL	9%	7%	11%
• Temporary dialysis needed	4%	4%	4%
HEMODYNAMIC STABILITY			
• Repeat thoracotomy needed	8%	8%	8%
RESPIRATORY			
• ARDS	2%	2%	2%
• Pneumonia	14%	7%	24%
• Reintubation	15%	7%	27%
• Tracheostomy	11%	11%	11%
• Ventilation >48 h	27%	23%	33%
• Vocal cord paralysis	11%	5%	19%
Length of hospital stay in days (range)	11 (6-35)	11 (6-29)	11 (5-40)
Key: ARDS, adult respiratory distress syndrome.			