

Thoracoabdominal Aneurysm Surgery and the Risk of Paraplegia: Contemporary Practice and Future Directions

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Abstract: Thoracoabdominal aneurysm surgery is associated with a high incidence of morbidity and mortality. Spinal cord ischemia and the risks of paraparesis or paraplegia remain devastating complications. The mechanisms of spinal cord injury involve both acute ischemic injury and delayed reperfusion injury. Blood flow to the spinal cord frequently arises in the segment of the aorta requiring aortic cross clamping. As such, there is an obligate period of blood flow disruption. Multiple strategies have evolved to reduce the ischemic interval and to provide adjunct interventions to reduce the impact of the ischemia. Despite a multidisciplinary approach, a spinal cord ischemia is present in approximately 4 to 16% of patients, depending on the type of aneurysm and other comorbid diseases. Cerebral spinal fluid drainage, distal perfusion techniques, intercostal artery anastomosis, hypothermia techniques, and mechanisms of ischemic preconditioning are interventions employed to reduce the

risk of paraplegia after thoracoabdominal aortic surgery. Surgeons, anesthesiologists, and perfusionist are intimately involved in the decision making as to which interventions will be employed in a given case. Although these adjuncts have been evaluated in multiple animal and human protocols, the efficacy of each intervention when looked at in isolation remains difficult to determine fully. This is attributable, in part, to the complex mechanisms of the patient injury, the inherent risks of the surgical procedure, and the confounding effects of comorbid disease states. Nonetheless, clinicians must have comprehensive understanding of these various interventions and their application. This review serves as an overview of these various interventions with special emphasis on outcome data. **Keywords:** thoracoabdominal aneurysm, surgery, paraplegia, spinal cord. *JECT. 2002; 34:11-17*

Surgical repair of thoracoabdominal aneurysm (TAAA) disease is associated with a high incidence of morbidity and mortality. In the largest reported series of patients, perioperative mortality is reported at 10%, renal failure 9%, stroke 3%, cardiac complications 12%, and paraplegia or paraparesis 16% (1). Despite these grave risks, surgical repair for patients with TAAA is recommended, given the poor natural history of uncorrected disease. The 2-year survival rate in patients with untreated thoracic and thoracoabdominal aneurysms is approximately 25% (2, 3). This is compared to an expected 2-year survival rate after surgery of 71% (4).

Paralysis or paraplegia is a particularly devastating complication after TAAA repair. The spinal cord is prone to ischemia during aortic cross clamping. Blood supply to the spinal cord is divided into posterior and anterior segments.

The posterior one-third of the cord receives its supply from the two posterior spinal arteries. These vessels receive their blood flow from the vertebral arteries and paired radicular arteries forming a continuous arterial supply running the entire length of the posterior spinal column. The single anterior spinal artery is a discontinuous artery that supplies blood to the anterior two-thirds of the cord. The superior segment receives blood from the vertebral arteries and three to five radicular arteries; whereas, the major supply to the midthoracic cord is via the arteria radicularis magna (ARM), also known as the Artery of Adamkiewicz. The origin of this vessel is between the ninth and twelfth thoracic segments in 75% of patients (5). Aortic cross clamping during TAAA resection often incorporates many of the intercostal vessels and the ARM, thereby interrupting blood flow to the anterior spinal artery and the anterior two thirds of the spinal cord. An anastomosing plexus of lumbar arteries supplies the lower cord.

Several factors are associated with increasing risk of spinal cord injury during TAAA repair. Aortic cross

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clamp time, extent of aneurysm, patient age, presence of hypotension or shock, urgency of the operation, aneurysm with presence of aortic dissection, diabetes, and patient age are all significant predictors of poor outcome (6, 7). The Crawford Classification best characterizes the extent of aortic disease. Type 1 TAAA extends from the proximal descending thoracic aorta to the upper abdominal aorta; type 2 extends from the proximal descending aorta to the below the renal arteries; type 3 extends from the distal half of the descending aorta to the abdominal aorta; and type 4 TAAA includes most or all of the abdominal aorta (Figure 1). The risk of spinal cord injury is directly related to aortic cross clamp time and extent of disease. This relationship is demonstrated in Figure 2.

Spinal cord injury may be either acute or delayed in onset. Acute spinal cord injury seen immediately after surgery is caused by an ischemic injury. Delayed spinal cord injury may occur anywhere from hours or days after surgery and represents a more complex lesion involving pathways of both ischemic cellular death and reperfusion injury. Ischemic injury results from adenosine triphosphate (ATP) depletion leading to cellular membrane disruption and death. Reperfusion injury involves one or more pathways, including polymorphonuclear leukocyte activation, platelet activation, inflammatory response mechanisms, edema formation, ischemia, and apoptosis. In a recent report of 170 patients by Cambria et al., paraplegia or paraparesis was immediate in four patients and delayed in eight patients for an over-all incidence of 7% (8). The delayed spinal cord injury ranged in onset from 24 hours to 2 weeks after surgery. Effective strategies to prevent spinal cord injury must address mechanisms for both acute and delayed injury.

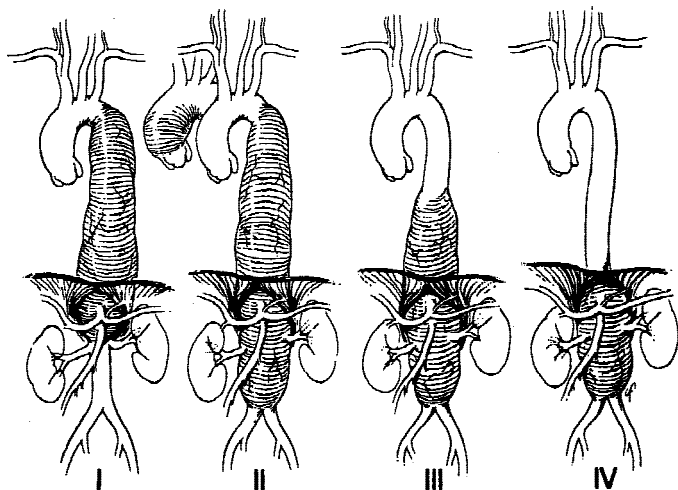


Figure 1. The Crawford classification of thoracoabdominal aortic aneurysms is defined by anatomic location and the extent of involvement. (Reproduced with permission from Crawford ES. Thoracoabdominal and suprarenal abdominal aortic aneurysm. In: Ernst CB, Stanley JC, eds. Current Therapy in Vascular Surgery. Philadelphia: Decker, 1987, p. 96.)

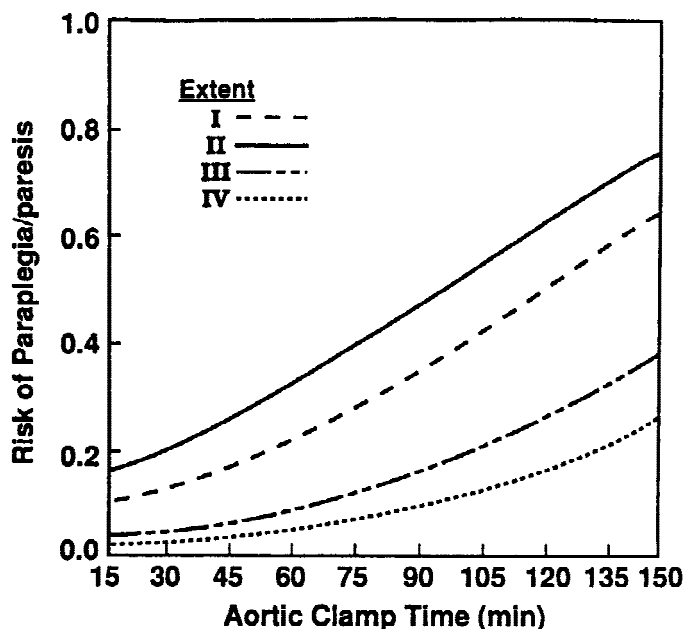


Figure 2. Multiple logistic regression analysis of risk according to extent and total aortic clamp. (Reproduced with permission from Svensson LG, et al. Experience with 1509 patients undergoing thoracoabdominal aortic operations. *J Vasc Surg.* 1993;17:357-70.)

Methods for spinal cord protection can be arbitrarily divided into mechanical and pharmacologic interventions (Table 1). As can be seen, numerous strategies have been tried in both animal models and human subjects. Such a list suggests that there is no single answer to spinal cord ischemia during TAAA. Clinicians and researchers interested in reducing spinal cord injury after aortic surgery face several challenges. For example, evaluation of any intervention is difficult, because no intervention can be studied in isolation. In addition, contemporary surgical care of this disease is highly variable, rendering comparisons in outcome difficult. Finally, outside a very few centers, most institutions do not perform a large number of these procedures, thereby making systematic study and analysis of data difficult, if not impossible. Nonetheless,

Table 1. Spinal cord protection.

Mechanical	Pharmacological
1) Clamp and run	1) Intrathecal vasodilators
2) Shunt placement	2) Excitatory amino acid antagonists, riluzole
3) Cardiopulmonary bypass	3) Calcium channel blockers
4) Hypothermic circulatory arrest	4) Magnesium
5) Selective cooling of the spinal cord	5) O ₂ scavengers
6) CSF drainage	6) Anesthetics, Ketamine, Naloxone
7) Intercostal anastomosis	7) Steroids
8) Ischemic preconditioning	8) Adenosine
9) Partial bypass techniques	9) K _{ATP} channel agonists
10) Spinal cord evoked potential monitoring	

there are data to support the benefit of various surgical techniques and perioperative management.

A comprehensive review of all the interventions found in Table 1 could fill several volumes. For example, spinal cord evoked potential monitoring is a technique highly valued by many clinicians. The place of evoked potential monitoring in clinical care remains hotly debated (9). Similarly, a review of all pharmacologic agents either evaluated, or in use, for spinal cord protection remains beyond the scope of this text. This review focuses on several areas of special interest to the clinician. These include the techniques of cerebral spinal fluid drainage, distal perfusion techniques, intercostal artery anastomosis, hypothermic protection, and ischemic preconditioning.

CEREBRAL SPINAL FLUID (CSF) DRAINAGE

Spinal cord perfusion pressure is equal to distal aortic pressure minus CSF pressure. If CSF pressure can be lowered by CSF drainage, then perfusion pressure increases (Figure 3) (10). Although numerous authors have demonstrated experimental evidence of protective benefit in animal models from lowered CSF pressure, human data remain inconclusive. CSF drainage is reported to reverse delayed onset paraplegia after thoracic aortic surgery (11, 12). Strong evidence with randomized, controlled studies, however, is scant. Ling and Arellano reviewed world literature on CSF drainage in a recent publication (13). Results from this review are shown in Table 2. As can be seen, the majority of the trials are poorly controlled, and confounding variables complicate all but one trial. Unfortunately, in this one blinded randomized clinical trial not plagued by confounding variables, the study design was poorly constructed, thereby rendering the conclusions of little clinical relevance (14). In their review, Ling and Arellano conclude that CSF drainage remains to be sufficiently studied in humans to provide a definitive answer. Regardless, many clinicians continue to add CSF drainage as a component of spinal cord protection during TAAA surgery. Should a clinician employ CSF drainage, certain principles are generally accepted. These would include maintaining a CSF pressure at 10–12 mmHg and continuing the CSF drainage for 24–72 hours postoperatively.

DISTAL PERFUSION TECHNIQUES

The presumed underlying mechanism behind all spinal cord injury, whether acute or delayed in onset, is disruption of nutritive blood flow during the period of aortic cross clamping. Distal perfusion techniques aim to resolve this problem by reducing the time and degree of spinal cord ischemia by routing blood to the aorta distal to the inferior cross clamp. Several techniques, including simple passive shunting from the proximal to distal aorta, femoral

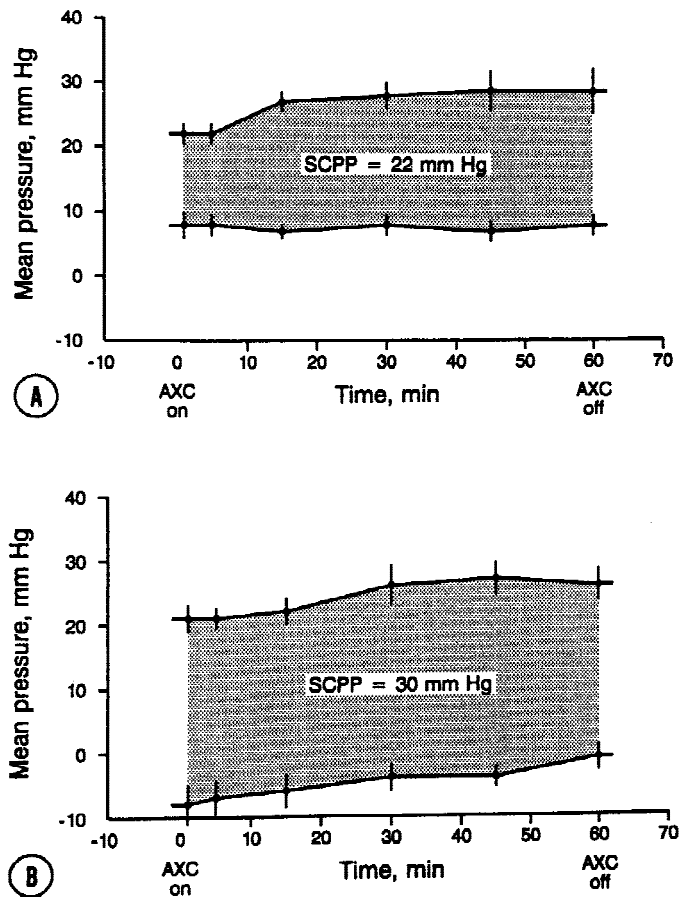


Figure 3. Spinal cord perfusion pressure (SCPP) (distal aortic pressure, upper curve; cerebrospinal fluid [CSF] pressure, lower curve) was lower in the control group (A) than in the CSF drainage group (B). (Reproduced with permission from McCullough JL, et al. Paraplegia after thoracic aortic occlusion: influence of cerebrospinal fluid drainage. *J Vasc Surg.* 1988;7:153–60.)

vein to femoral artery bypass, and left atrial to femoral artery bypass, are reported in the literature with varying degrees of success (15, 16). All techniques are aimed at incorporating the ARM artery in the distal flow thereby preserving spinal cord blood flow during surgical repair. Coselli and LeMaire report experience with 710 patients over a 12-year period with type I or type II TAAAs (15). Left heart bypass was used in 312 (43.9%) of patients, and this group was compared to 398 (56.1%) of patients without left heart bypass. In operations with left heart bypass, the perfusion circuit consisted of an outflow cannula in the left atrium, an inflow cannula in either the femoral artery (122 cases, 39.1%) or distal aorta (190 cases, 60.9%), 3/8-inch polyvinylchloride tubing, and a centrifugal pump. For selective visceral and renal perfusion, balloon perfusion catheters were connected to the outflow limb using a three-way stopcock. No blood reservoir, heat exchanger, or oxygenator was incorporated in the circuit. The 30-day survival rate was 94.8%, and paraplegia or paraparesis was seen in 6.0%. Left heart bypass significantly reduced spi-

Table 2. Outcome data according to level of evidence.

Reference	Design	CSFD	Confounders	Crawford Type I/II/III/IV/TA	Outcome Blinded	Advocate CSFD
Crawford et al., 1990 (14)	RCT	<50 mL	No	19/27/0/0/0 (C) 25/27/0/0/0	Yes	No
Svensson et al., 1998 (24)	RCT	>10 mmHg + Papaverine	Yes	13/4/0/0/0 (C) 11/5/0/0/0	Yes	Yes
Svensson et al., 1988 (30)	OCS	5–15 mmHg + Papaverine	Yes	—	—	No
Acher et al., 1994 (31)	OCS	<10 mmHg	Yes	10/14/6/7/0 (C) 9/19/6/15/0	—	Yes
Acher et al., 1998 (32)	OCS	? + Naloxone	Yes	—	—	Yes
Acher et al., 1990 (33)	NRHC	<14 mmHg	Yes	5/8/5/3/2 (C) 4/8/5/3/4	—	Yes
(subset of Acher, 19994) (31)		+ Naloxone				
Murray et al., 1993 (34)	NRHC	<15 mmHg	Yes	8/14/17/0/11 (C) 15/4/6/0/23	—	No
Hollier et al., 1992 (35)	NRHC	<10 mmHg	Yes	7/13/16/6/0 (C) 22/16/23/47	—	Yes
Safi et al., 1998 (36)	NRHC	<10 mmHg	Yes	—	—	Yes
Safi et al., 1994 (37)	Case	<15 mmHg	Yes	14/31/0/0/0	Yes	Yes
Svensson et al., 1990 (38)	Case	5–15 mmHg	Yes	5-Type I or II 6-TA	—	No
Safi et al., 1996 (39)	Case	<50 mL + Papaverine	Yes	31/63/0/0/0	Yes	Yes
(45 patients from Safi 1994) (37)						
Cambria et al., 1989 (40)	Case	Unspecified	Yes	—	—	No

(C) = control group; Case = case series; CSFD = cerebrospinal fluid drainage; NRHC = nonrandomized historical cohort; OCS = observational cohort study; RCT = randomized controlled trial; TA = thoracic aneurysm. (Reproduced with permission from Anesthesiology 2000;93:1115–22.)

nal cord injury in patients with type II TAAA from 13.1% in controls to 4.8% in left heart bypass patients, $p = .007$. In addition to spinal cord perfusion, significant benefit is gained with continued visceral and renal perfusion. Safi et al. reported a technique employing left atrial inflow, femoral artery outflow, and multiple perfusion cannula to the celiac axis, superior mesenteric artery, and renal arteries (17). In their report, postoperative liver function is preserved in patients with type II TAAAs.

INTERCOSTAL ARTERY (ICA) ANASTOMOSIS.

Because most of spinal cord blood flow comes via the intercostal arteries, it seems intuitive that anastomosis of intercostal arteries would afford the spinal cord better perfusion. Similarly, ligation of intercostal arteries might lead to cord ischemia. Unfortunately, data regarding ICA reanastomosis is, as with other areas of TAAA intervention, conflicting. Much published work detailing strategies of spinal cord protection include re-implantation of the intercostal arteries (7, 15, 16). Although most clinicians incorporate vital intercostal arteries as a button graft, there are reports of selective intercostal perfusion and graft re-implantation (18, 19). Data from Safi et al. support selective ICA reanastomosis (20). In a study of 342 patients, intercostal arteries were rated as either patent or obstructed. The ICAs were then either ligated or re-implanted and spinal cord function was evaluated (Figure 4). The patients at highest risk for spinal cord injury included those patients who have patent ICAs at the lower thoracic segments that were not re-implanted. In contrast

to these techniques, Dr. Griep and colleagues report good results with intercostal sacrifice and somatosensory evoked potential monitoring (21). The decision to include ICAs remains a surgical one and must be based on several factors weighing the risk of additional ischemic time during re-implantation versus the benefit of ICA flow inclusion.

HYPOTHERMIC TECHNIQUES

Hypothermia is known to protect organ viability during ischemia. Hypothermic techniques are routinely employed

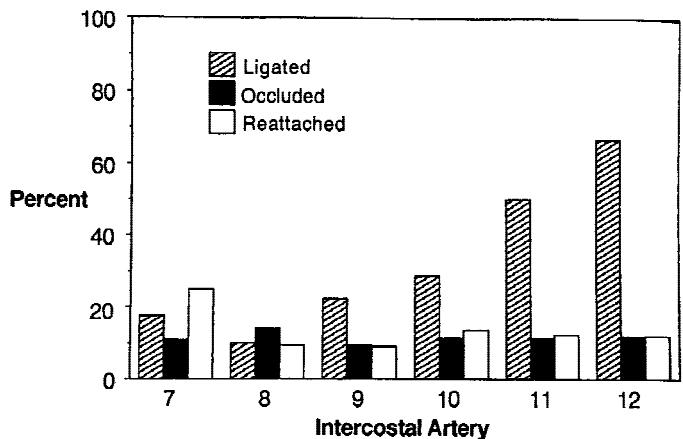


Figure 4. Percentage of patients with neurologic deficit by intercostal artery and artery status. The lower the position of the artery on the spine, the higher the risk of deficit if the artery is ligated. (Reproduced with permission from Safi HJ, et al. Importance of intercostal artery reattachment during thoracoabdominal aortic aneurysm repair. J Vasc Surg. 1998;27:58–68.)

in cardiothoracic and transplant surgery. In cardiac surgery, hypothermia is used during periods of myocardial ischemia to preserve organ viability during ischemia. In transplant surgery, harvested organs are transported and stored under hypothermic conditions to preserve viability during ischemia. Marsala and colleagues demonstrated spinal cord protection in an animal model during ischemia with graded temperatures of 28 to 34°C (22). Rokkas et al. demonstrated reduced neurotransmitter amino acid release with hypothermia (23). In this swine model of spinal cord protection, seven excitatory neurotransmitter amino acids were assayed by in vivo microdialysis techniques. Hypothermia uniformly prevented the release of amino acids in the extracellular space following an ischemic-reperfusion injury of the spinal cord via 60-minute aortic cross clamping. Significantly, the excitatory neurotransmitter glutamate, and the inhibitory neurotransmitter glycine, both associated with release following ischemia and reperfusion, were markedly reduced with hypothermia. Encouraging results from the use of hypothermic techniques in humans have yielded proponents of active and passive moderate systemic hypothermia, profound hypothermia involving deep hypothermic circulatory arrest (DHCA), and regional hypothermia of the spinal cord.

Coselli and LeMaire advocate permissive moderate hypothermia as a multifaceted approach to spinal cord protection (15). In another report, active systemic cooling to a bladder temperature of 29–31°C is associated with reduced spinal cord ischemic injury (24). There is little risk from moderate hypothermia in terms of coagulopathy and myocardial rhythm disturbances. Changes in anesthetic pharmacology and postoperative recovery must obviously be considered with passive or active moderate hypothermia.

Deep hypothermic circulatory arrest (DHCA) provides the surgeon with, perhaps, the best operative conditions for repair. DHCA also protects the spinal cord well, because it is known that organ ischemia is well tolerated for upward of 45–60 min at temperatures of 18°C. Several authors report success with DHCA for TAAA repair. Kouchoukos found in a study of 51 patients that DHCA had an over-all mortality rate of 9.8%, and paraplegia/paraparesis a rate of 6.5% (25). Many clinicians, however, avoid DHCA techniques unless surgically necessary before of concerns over postoperative coagulopathy, the re-

quirement for full cardiopulmonary bypass and heparinization, and extended operative time.

Regional hypothermia of the spinal cord offers an exciting new technique by providing the benefit of hypothermia to the spinal cord and avoiding the detriment of total body cooling. Cambria and Davidson report results in 170 patients of regional spinal cord cooling (26). In a technique involving epidural injection of iced saline and concurrent pressure monitoring and drainage of the CSF, the authors found spinal cord ischemia in 7% of patients, as compared to a predicted incidence of 18.5%, $p = .003$. The over-all operative mortality in this group of patients was 9.5%, and was associated with spinal cord injury, postoperative cardiac and renal complications. Further analysis reveals that half of the spinal cord deficits were minor with good functional recovery; devastating paraplegia occurred in only three patients (2.0%). Intraoperative epidural cooling data are shown in Table 3. In contrast to other spinal cord protective strategies lowering CSF pressure, this technique raises CSF pressure during ischemia. To counteract the negative effects of elevated CSF pressure, a gradient of 30–40 mmHg between the mean arterial and CSF pressures was maintained during infusion of the cold saline.

ISCHEMIC PRECONDITIONING

Although there are several areas of active research in the field of spinal cord protection during TAAA surgery, two related areas deserve special comment. Ischemic preconditioning is the natural physiologic process whereby short, sublethal events of ischemia to an organ initiate a protective cellular process by which the organ is protected from a subsequent, usually lethal ischemic insult. The mechanism of ischemic preconditioning in these organs remains unclear; however, pathways involving adenosine receptors, potassium-dependent ATP channels, alpha agonists, and heat shock proteins have all been implicated.

Ischemic preconditioning is present in the spinal cord. In a report by Zvara et al., spinal cord protection was demonstrated with a 3-minute ischemic preconditioning event 30 minutes before a 12-minute ischemic injury (Figure 5) (27). In other models of delayed ischemic preconditioning (i.e., preconditioning events occurring 24 to 48

Table 3. Intraoperative epidural cooling data.

	Baseline	During Cross Clamp	End of Operation
Core temperature (°C)			
Mean ± SD (range)	36.0 ± 0.5 (34.6 to 37.3)	35.0 ± 1.0 (27.5 to 37.5)	34.6 ± 0.8 (32.3 to 36.8)
CSF temperature (°C)			
Mean ± SD (range)	36.0 ± 0.9 (28.0 to 38.0)	26.4 ± 3.0 (21.1 to 37.4)	33.9 ± 1.2 (28.0 to 36.8)
CSF pressure (mmHg)			
Mean ± SD (range)	13.3 ± 4.5 (4.0 to 30.0)	35.1 ± 13.0 (7.0 to 90.0)	11.9 ± 5.2 (2.0 to 28.0)

(Reproduced with permission from J Vasc Surg. 1998;27:58–68.)

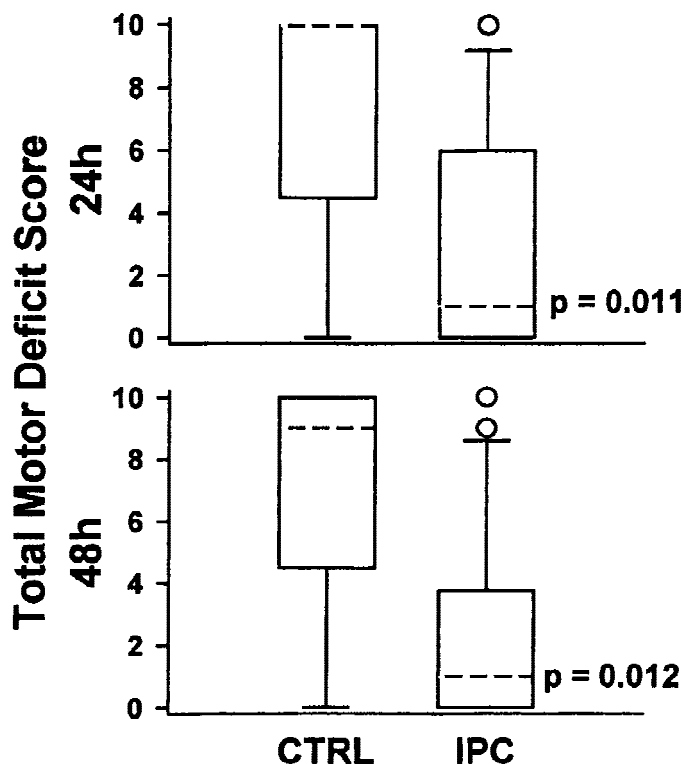


Figure 5. Data from the four motor neurologic tests (Tarlov scale, righting, placing, and inclined plane) were combined for a composite neurologic score and are presented as a box plot. Mann-Whitney rank sum test demonstrated significant difference between groups at both time points. (CTRL = control group; IPC = ischemic preconditioning group; boxed area = 25 to 75th percentile; — = 10th and 90th percentiles; ○ = individual animals above or below 10th and 90th percentiles; - - - = median value for group.) (Reproduced with permission from Zvara DA, et al. Ischemic preconditioning reduced neurologic injury in a rat model of spinal cord ischemia. *Ann Thorac Surg.* 1999;68:874–80.)

hours before the ischemic insult), other authors have demonstrated significant spinal cord protection (28). The models of delayed ischemic preconditioning are associated with production of heat shock proteins (29). Although ischemic preconditioning is an unlikely adjuvant in the surgical population, given the time demands of surgery and the usual presence of aortic atheromatous disease, and the inherent risks of intermittent cross clamping, the underlying mechanisms of ischemic preconditioning may lead to the development of other therapies that may invoke these protective pathways. For example, if an agent can be developed that stimulates the natural physiologic process of ischemic preconditioning or heat shock protein protection, then patients receiving these drugs may reap the benefit of this mode of organ protection without the risk of mechanical clamping and unclamping of the aorta to induce ischemic preconditioning.

In conclusion, TAAA repair is associated with a very high morbidity and mortality. Spinal cord ischemia and resultant spinal cord injury remains a significant and devastating complication in this patient population. Cur-

rently, no single technique provides adequate protection. Contemporary practice, although diverse, generally employs several combined techniques including, but not limited to, CSF drainage, distal perfusion, intercostal artery inclusion, and some degree of hypothermia. Future strategies for spinal cord protection may include adjuncts involving principles of ischemic preconditioning.

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