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Prevention of Spinal Cord Injury After Thoracoabdominal Aortic Aneurysm Repair

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1. Introduction

Thoracoabdominal aortic aneurysm (TAAA) repair still remains a challenging operation since it necessitates extended exposure of the aorta and reimplantation of the vital aortic branches. Among possible postoperative complications, spinal cord injury (SCI) seems one of the most formidable morbidities. Even by experts who have championed in TAAA repair the incidence of SCI remained as high as 16% in the early 1990s (Svensson et al., 1993). Thanks to numerous experimental investigations and technical advances or modifications, it has improved up to 3.6%-4.6% in the worldwide largest centers with a contemporary strategy (i.e. aggressive use of distal aortic perfusion) (Safi et al., 2003, Coselli et al., 2007, Zoli et al., 2010). However, this incidence of SCI should vary according to the extent of aortic involvement. In 1986, Crawford and his colleagues proposed classification of TAAA into four groups according to the extent of aortic involvement and clearly showed that this classification could predict late death and neurological deficit (Crawford et al., 1986). Their classification is as following; group I involves most of the descending thoracic and upper abdominal aorta. Group II involves most of the descending thoracic aorta and most or all of the abdominal aorta. Group III involves the distal descending thoracic aorta and varying segments of abdominal aorta. Group IV involves most or all of the abdominal aorta including the segment from which the visceral vessels arise. Later, Safi and his co-workers advocated additional group V that involves below the sixth intercostal space to just above the renal arteries (Safi et al., 1998, 1999, 2003) (Figure 1). As shown later in Table 3, this classification correlates well with postoperative SCI still in contemporary era, thus it has been used widely to date. In the most extended type of TAAA (type II), SCI still occurs in 4.2-15.8% despite with experienced hands in the last decade (Safi et al., 2003, Jacobs et al., 2006, Coselli et al., 2007, Conrad et al., 2007, Acher et al., 2008, Zoli et al., 2010). Thus spinal cord protection during TAAA repair has emerged as great clinical importance. The primary aim of this review is to provide a better appreciation and understanding of the mechanisms and pathophysiology of SCI after TAAA repair.

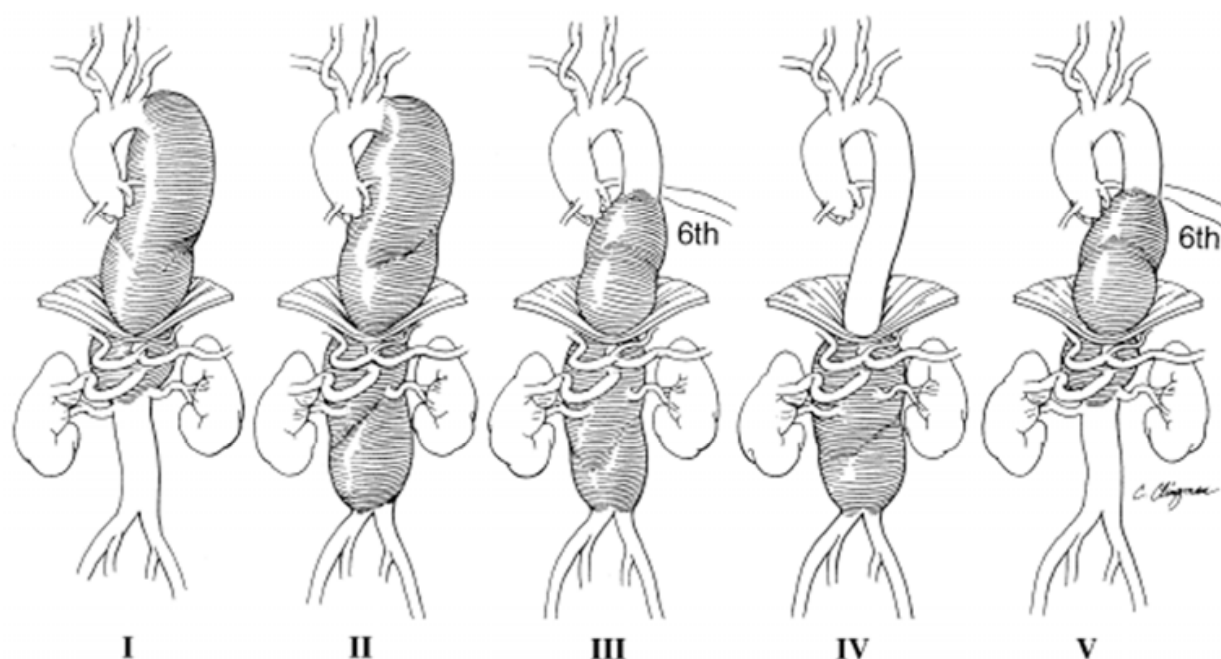


Fig. 1. Crawford/Safi's classification of the extent of aortic involvement (From; Safi, et al., 1999).

2. Mechanisms of spinal cord injury

Underlying mechanisms of SCI after TAAA repair are multifactorial, however, they can be primarily summarized into three key processes; 1) the duration and degree of ischemia, 2) failure to re-establish blood flow to the spinal cord after repair, and 3) a biochemically mediated reperfusion injury (Svensson 1997).

2.1 The duration and degree of ischemia

To reduce the duration and degree of spinal cord ischemia, either oxygen supply should be enhanced or oxygen demand attenuated. For these purposes, multi-segmental sequential clamping technique combined with mild hypothermic distal aortic perfusion has favorably been used in many institutions. When sequential clamping is not feasible, deep hypothermic circulatory arrest should be applied.

2.1.1 Distal aortic perfusion

Pioneers have used simple cross-clamping technique and reported acceptable outcomes (Crawford et al., 1986) (Schepens et al., 1994). Nowadays, majority of surgeons use distal aortic perfusion to reduce ischemia of the distal organs and cardiac afterload. Surprisingly, distal shunt was already used in a half century ago and has been applied with some modifications (Etheredge et al., 1955, Gott 1972, Cambria et al., 1998). Distal shunt has some advantages such as its simplicity and mild heparinization, but has also some disadvantages. Distal blood flow depends largely on proximal blood pressure and it is difficult to manage massive bleeding. On the other hand, left heart bypass using a centrifugal pump can provide flexible control of distal blood flow, thus it is currently used widely. Many experts have demonstrated that left heart bypass could significantly reduce the incidence of SCI

especially in extended TAAA (type II) repair (Safi et al., 1997) (Coselli et al., 1999, 2003). We have favorably used a partial cardiopulmonary bypass (CPB) through the femoral artery and vein. This alternative has some advantages in case with cardiopulmonary instability or massive bleeding. Some disadvantages such as acceleration of bleeding tendency has been resolved by reduction of heparin dosage using a fully heparin-coated closed-loop CPB system with a soft reservoir bag (Shiia et al., 2005, 2006).

2.1.2 Multi-segmental sequential clamping

Blood flow of the anterior spinal artery is primarily supplied by some radicular arteries. The radicular artery has a rich collateral network between adjacent intercostal/lumbar arteries. Therefore it may be beneficial to maintain this collateral blood flow during reattachment of the intercostals/lumbar arteries. Especially in patients with degenerative aortic aneurysm, occlusion or stenosis of the intercostals/lumbar arteries may alter and complicate this collateral pathway, which may emphasize the importance to preserve it. In addition, during reattachment of the intercostals/lumbar arteries, back bleeding from adjacent one may accelerate steal phenomenon (Christiansson et al., 2001). The Mount Sinai group emphasized importance of avoiding steal phenomenon. They sacrificed all involved intercostals/lumbar arteries outside of the aorta to avoid steal phenomenon in 100 cases and experienced only 2% SCI (Etz et al., 2006). Their findings have a great implication for extensive deployment of endovascular stentgraft.

On the other hand, the anterior spinal artery has a narrow critical zone at around T4 level, where the spinal cord is susceptible to ischemia (Dommissie 1974, Svensson et al., 1986). Thus an ascending blood flow through the anterior spinal cord artery alone is not enough to feed this area. These mechanisms may explain why distal aortic perfusion alone was not beneficial to prevent SCI in the early era (Crawford et al., 1988). Therefore segmental sequential clamping seems advantageous to preserve collateral blood flow to the spinal cord during reattachment of the critical intercostals/lumbar arteries (Kuniyoshi et al., 2003). Trend has become toward clamping shorter segments (multi-segmental sequential clamping). We experienced no postoperative SCI when less than two pairs of intercostals/lumbar arteries were involved between segmental clamping. We identified that multi-segmental sequential clamping in combination with distal aortic perfusion was the only significant predictor for SCI after type I and II TAAA repair in multivariate analysis (Shiia et al., 2005). The fact that a previous abdominal aortic aneurysm repair is a significantly high risk for postoperative SCI may support this collateral network concept (Svensson 2005).

2.1.3 Hypothermia

It has been widely accepted that mild hypothermia is clinically effective to prevent SCI. A reduction in temperature of 3°C provides a 2-fold increase of the tolerable ischemic interval in rabbit (Vacanti et al., 1984) and a 5°C reduction does a 2.5-fold increase in pig (Strauch et al., 2004). Therefore almost all aortic surgeon currently use mild hypothermia (32-34°C) using heat-exchanger or permissively in combination of distal aortic perfusion. Whenever the aorta cannot be safely clamped, use of deep hypothermic circulatory arrest should be warranted. Clinically, moderate to profound hypothermia was proved to prolong tolerable ischemic interval, which enables secure reconstruction of the aorta for aortic surgeons (Svensson et al., 2003) (Fig. 2).

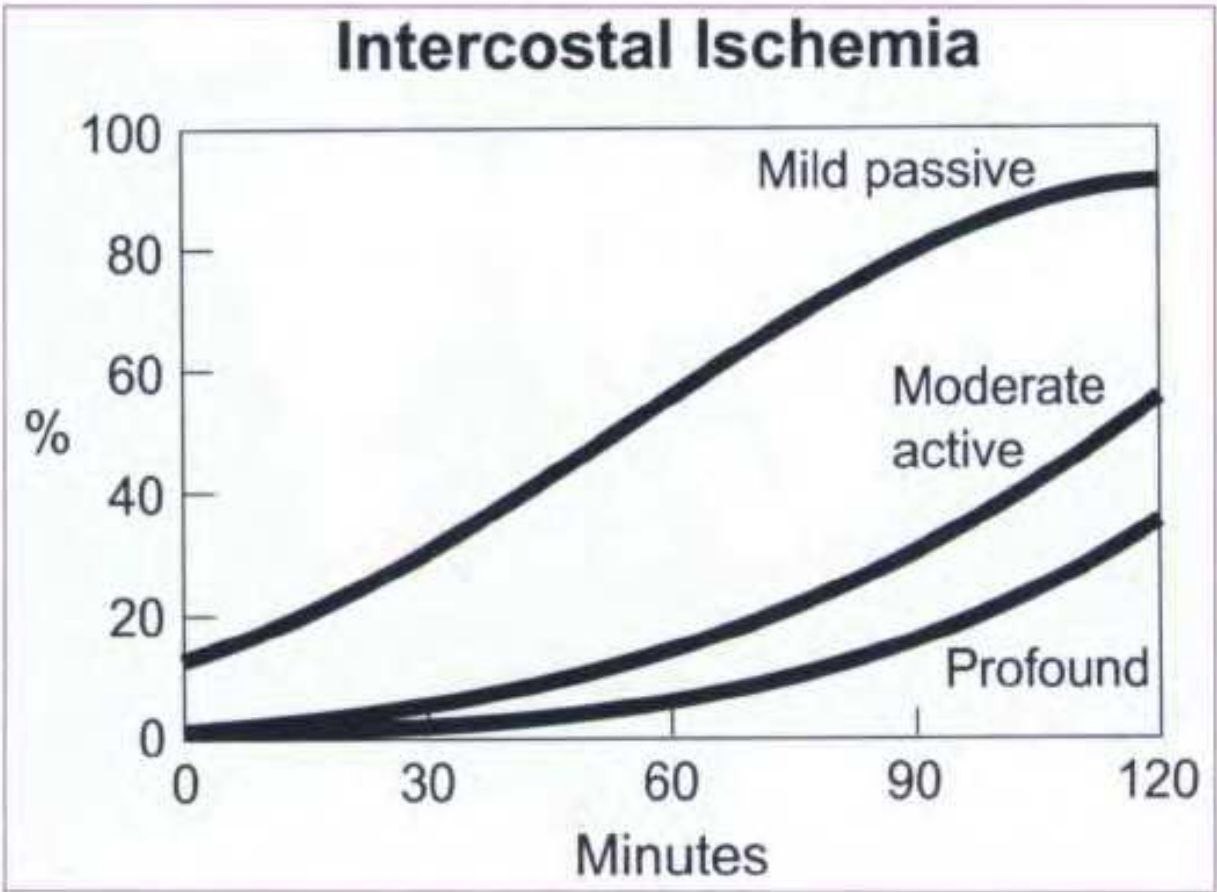


Fig. 2. Relationship between interval of spinal cord ischemia and risk of neurological deficit stratified by systemic temperature (Svensson 2005).

Kouchoukos and his colleagues have aggressively performed TAAA repair under profound hypothermia and reported its substantial protective effect against SCI (2.9% paraplegia) (Kouchoukos et al., 2003). Using deep hypothermic circulatory arrest Coselli and his co-workers could reduce the incidence of SCI up to 1%, however on the other hand, it increased morbidity and mortality rate (Coselli et al., 2008). Therefore this alternative is currently limited to selected cases (large or extensive aneurysm, friable aortic tissue, high risk of embolization, complex repair, redo cases), principally due to risk of coagulopathy, pulmonary dysfunction, and massive fluid shift. Accordingly some experts have proposed regional epidural or intrathecal cooling (Black et al., 2003, Shimizu et al., 2010). Although conceptually promising, this alternative currently lacks the support of prospective randomized data in larger cohort.

2.2 Failure to re-establish blood flow to the spinal cord after repair

2.2.1 Preoperative identification of critical intercostals artery

There are some surgeons who will blindly re-attach every intercostal/lumbar arteries to secure spinal cord protection (so-called onlay patch methods) (DeAnda et al., 2005). However, this technique seems unrealistic because longer ischemic time is definitively associated with higher risk of SCI (Safi et al., 1998). In addition, considerable extent of aneurismal aortic wall remains to exist. To re-establish blood flow to the spinal cord,

therefore, it is crucial to identify critical intercostals artery to the spinal cord. This is normally the largest radiculomedullary artery; so-called “the Adamkiewicz artery”. Preoperatively, it used to be identified by angiography, however, it appears rather invasive and difficult in case with large, thrombosed, or kinky aorta (Kiefer et al., 2002). Since we have visualized Adamkiewicz artery using multi-detector row computed tomography in 2003 (Maruyama et al., 2003), numerous reports have demonstrated that it is feasible in over 80% of cases by both computed tomography and magnetic resonance angiography (Yoshioka et al., 2006, Uotani et al., 2008) (Fig 3) (Table 1).

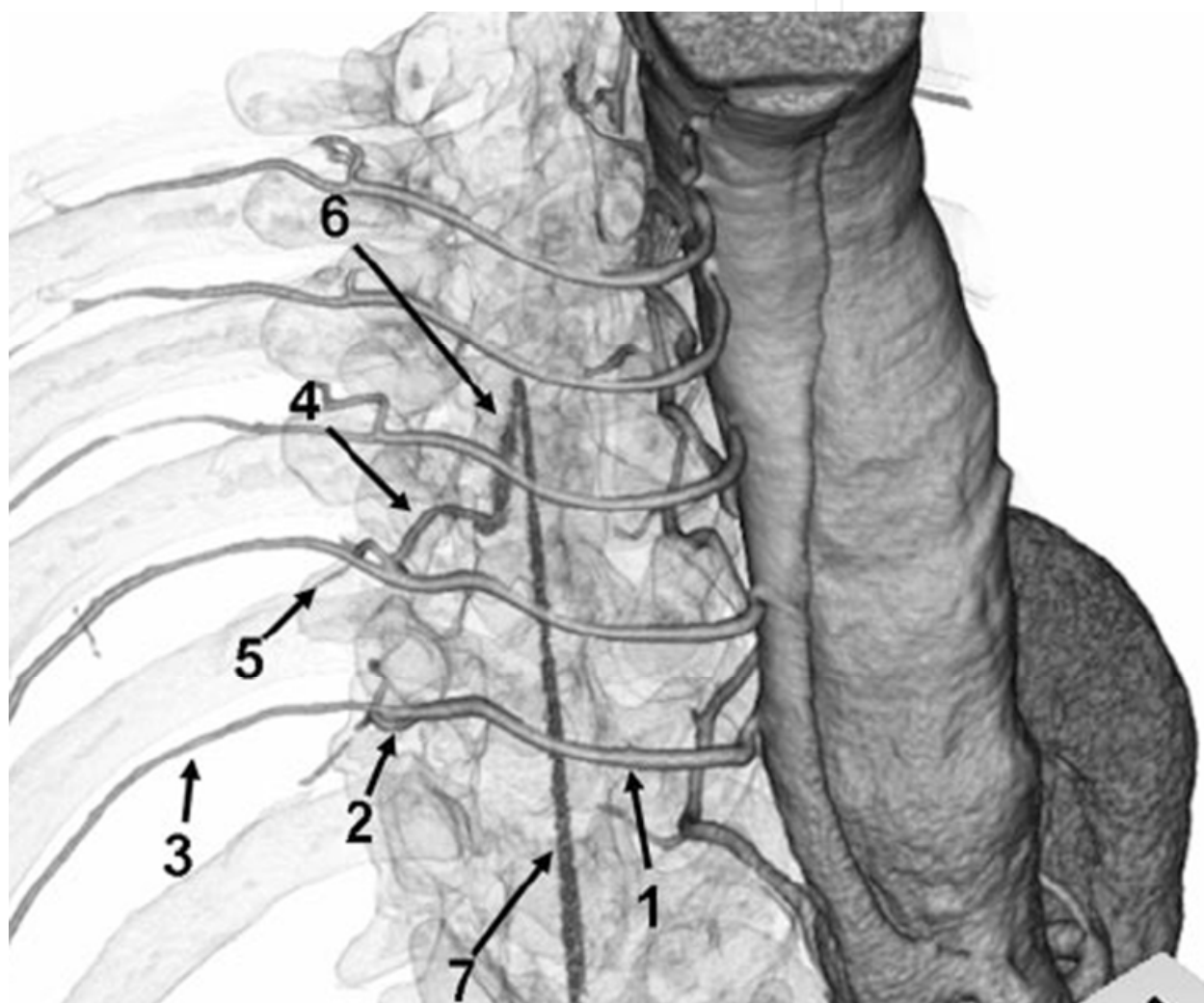


Fig. 3. Identification of the Adamkiewicz artery by 3D volume-rendered computer tomography. 1: Intercostal and lumbar arteries with their posterior (2) and anterior (3) branches, 4: Radiculomedullary artery and muscular branch (5), 6: the Adamkiewicz artery, 7: the anterior spinal artery. (Uotani et al., 2008)

The majority of the Adamkiewicz artery proved to originate from T8-L1 level with left dominance, which was consistent with angiographic findings and cadaver study (Koshino et al., 1999, Kiefer et al., 2002) (Table 2, Fig 4-5).

MRA		
Nijenhuis RJ.	J Vasc Surg. 2007;45:677.	97%
Yoshioka K.	Radiographics. 2006;26:S63-73.	93%
Mell MW.	J Surg Res. 2008 Jun 20.	85%
Hyodoh H.	J Magn Reson Imaging. 2007;26:359.	82.4%
Ogino H.	Ann Thorac Surg. 2006;82:592.	70.7%
CT		
Nojiri J.	Eur J Cardiothorac Surg. 2007;31:249.	100% (I.A.)
Boll DT.	Am J Roentgenol. 2006;187:1054.	100%
Ou P.	Am J Neuroradiol. 2007;28:216.	95%*
Uotani K.	Am J Neuroradiol. 2008;29:314.	94% (I.A.) 60% (I.V.)
Yoshioka K.	Radiographics. 2006;26:S63-73.	83%
Utsunomiya D.	Eur Radiol. 2008;18:2684.	80%
Takase K.	J Comput Assist Tomogr. 2006;30:716.	80%
Nijenhuis RJ.	J Vasc Surg. 2007;45:677.	71%
Kudo K.	Am J Neuroradiol. 2003;24:13.	68%
Von Tengg-Kobligk H.	J Endovasc Ther. 2007;14:639.	59%
Angiography		
Kieffer E.	J Vasc Surg 2002;35:262.	86%
Heinemann MK.	Ann Thorac Surg. 1998;65:346.	65%
Williams GM.	J Vasc Surg. 2004;39:314.	43%

Table 1. Preoperative identification rate of the Adamkiewicz artery using different modalities. *child cases (7.5 +/- 5 years old), I.A. or I.V.: intra arterial or venous injection of contrast materials, respectively.

	Rt	Lt	Total
Th7	-	1	1
Th8	2	3	5
Th9	1	4	5
Th10	1	6	7
Th11	2	1	3
TH12	3	4	7
L1	-	4	4
L2	-	2	2
L3	-	1	1
L4~	1	-	1
	10	27	37
	(27%)	(73%)	

Table 2. Our experience to detect the Adamkiewicz artery in 48 cases using a computer tomography between 2001 and 2004. Detection rate was 77% and 86% of the Adamkiewicz artery originated from T8-L1

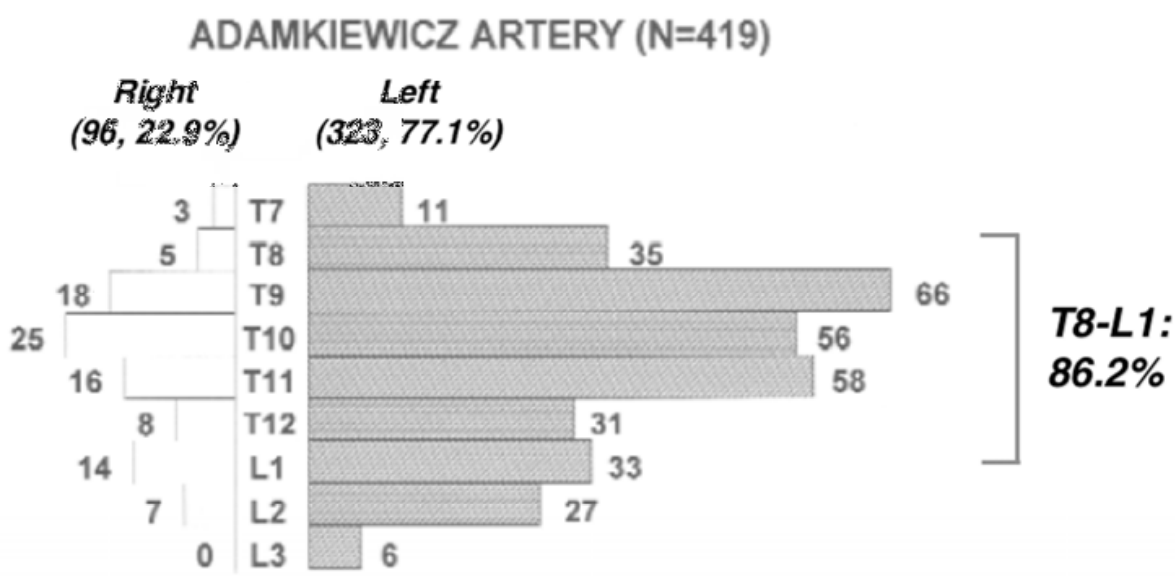


Fig. 4. Identification of the origin of the Adamkiewicz artery using an angiography (Kiefer et al., 2002 with modification)

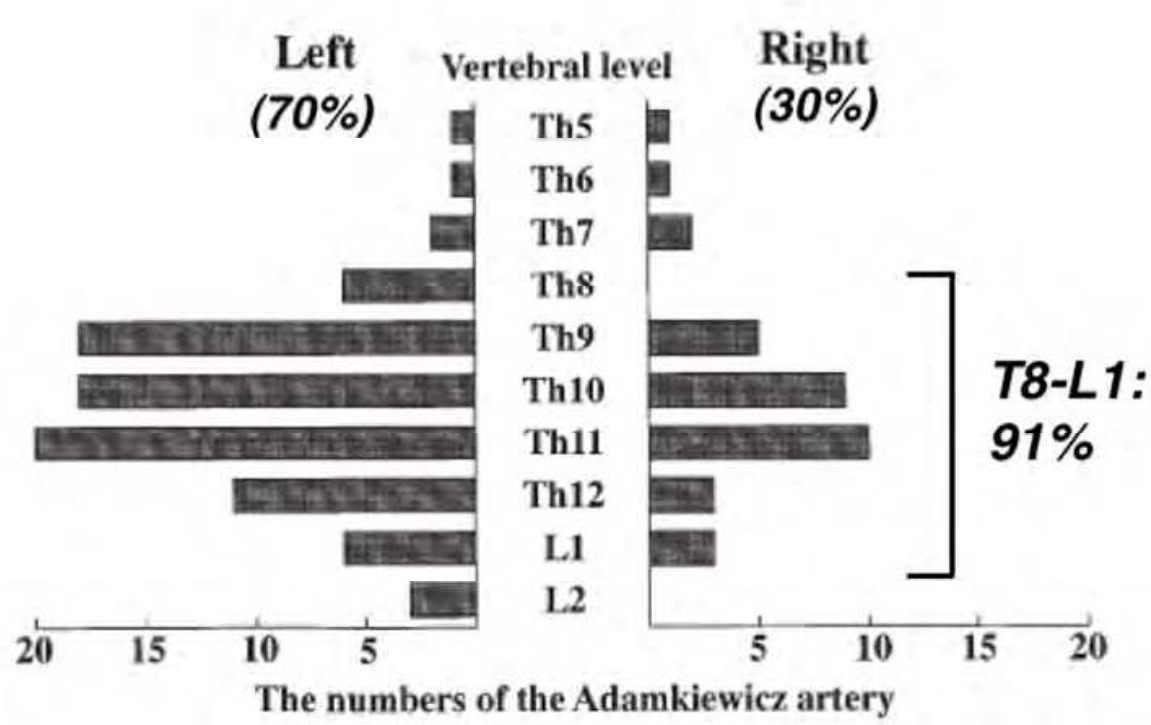


Fig. 5. Identification of the origin of the Adamkiewicz artery from 90 cadavers. (Koshino et al., 1999 with modification)

2.2.2 Intraoperative identification of critical intercostals artery

To identify the Adamkiewicz artery intraoperatively, traditionally we used to apply evoked spinal cord potential, which required two electrodes placed in the epidural space and

published its usefulness to prevent SCI (Matsui et al., 1994, Shiiya et al., 1995). However, this method is more or less invasive and technical demanding, and although rare, complications related to epidural catheter are catastrophic. Therefore majority of surgeon are currently using motor evoked potentials (Jakobs et al., 2006). Nonetheless, these electrophysiological examinations have some disadvantages such as time delay or effect of electric noise, anesthetic agents, peripheral ischemia, and hypothermia. Therefore we have tried to measure immediate information of spinal cord oxygenation noninvasively using transcutaneous or transesophageal near-infrared spectrophotometry (Kunihara et al., 1998, Kunihara et al., 2004). Further studies and technical advance will be essential toward its clinical use.

2.3 A biochemically mediated reperfusion injury

2.3.1 Clinical significance of cerebrospinal fluid drainage

Cerebrospinal fluid (CSF) drainage (CSFD) may be one of the most promising procedures to prevent SCI. Its usefulness has already advocated in 1960 (Miyamoto et al., 1960). Acher and associates have reported that combined use of CSFD and naloxone hydrochloride (naloxone), an opioid receptor antagonist, reduces the risk of SCI in patients undergoing TAAA repair in 1994 (Acher et al., 1994). In 2002, CSFD itself proved to be beneficial to reduce the risk of SCI after TAAA repair in prospective randomized clinical trial (Coselli et al., 2002). Meta-analysis of three randomized controlled trials and five cohort studies revealed beneficial effect of CSFD on paraplegia (Fig 6) (Cinà et al., 2004). Although rare (0.2%), CSFD-associated complications are catastrophic (subdural hematoma, intracranial bleeding, meningitis), so that CSFD should currently be reserved for extended aneurysm (type I and II) or patient at high risk (Cinà et al., 2004, Wynn et al., 2009).

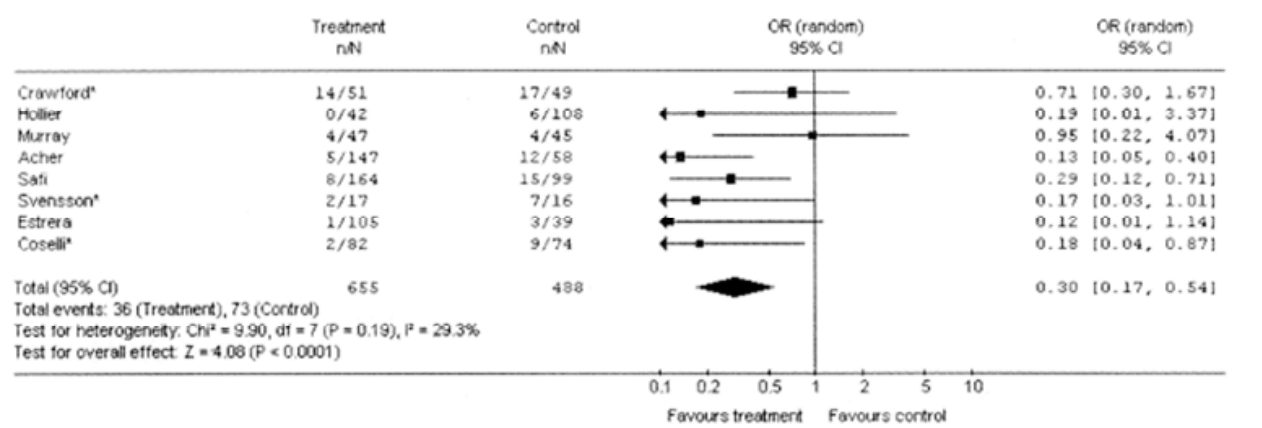


Fig. 6. Meta-analysis showing beneficial effect of cerebrospinal fluid drainage on paraplegia (Cinà et al., 2004).

2.3.2 Clinical significance of cerebrospinal fluid drainage

However, underlying mechanisms of CSFD to reduce SCI seems still unclear and it has not been investigated whether naloxone can alone attenuate the risk of SCI clinically like CSFD. Experimental studies have revealed that CSF pressure rises during aortic clamping, leading to decrease of spinal cord perfusion pressure (mean distal aortic pressure – CSF pressure) (Kaplan et al., 1995) (Fig. 7).

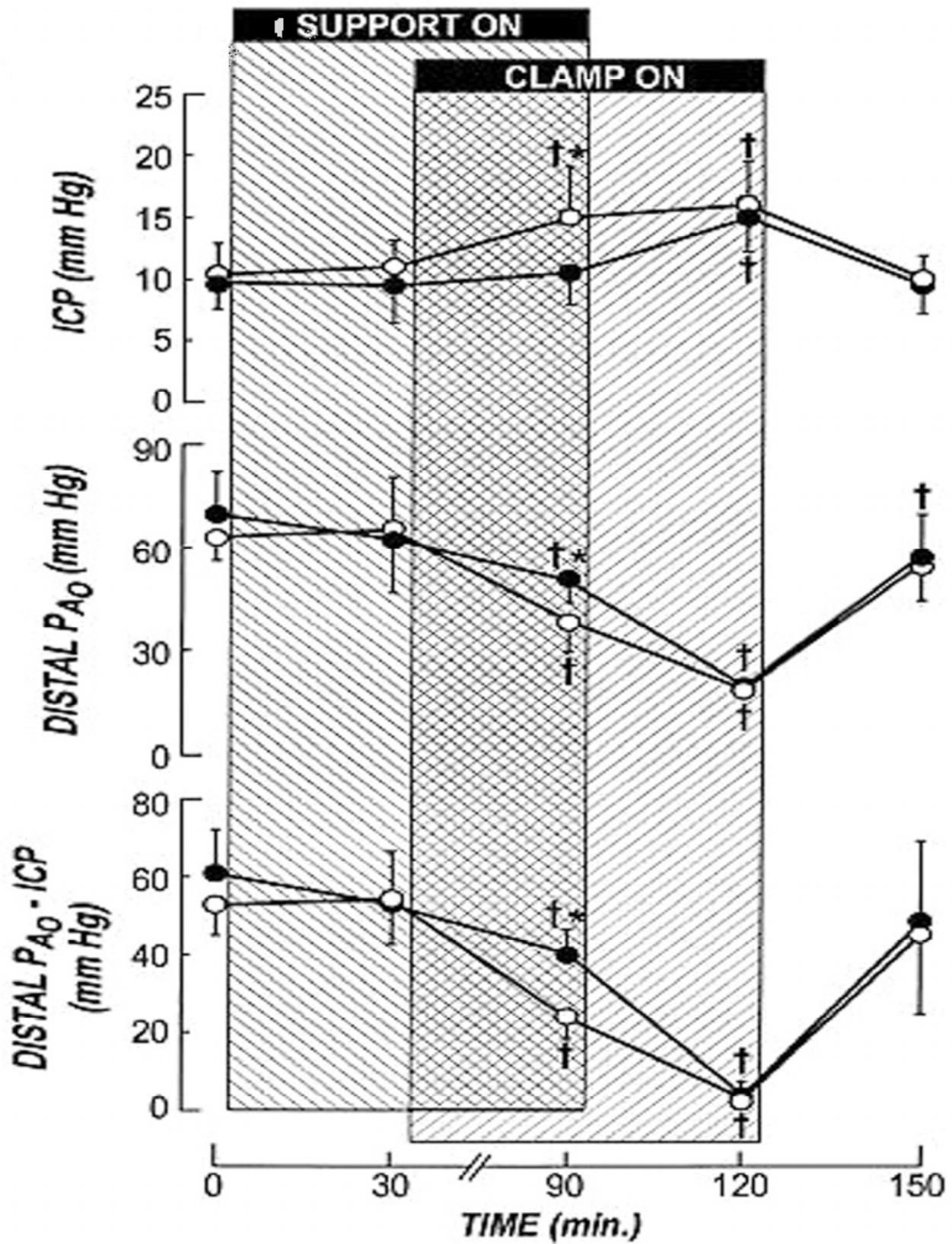


Fig. 7. Relationship between intracranial pressure (ICP), mean distal aortic pressure (DISTAL P_{AO}), and spinal cord perfusion gradient (DISTAL P_{AO} - ICP) during aortic clamping (Kaplan et al., 1995).

It has been believed that CSFD may restore spinal cord blood flow by decompressing spinal compartment syndrome caused by spinal cord edema in relation to ischemia-reperfusion injury (Safi et al., 1997) (Fig. 8). This speculation is supported by the clinical consequence that delayed-onset paraplegia could be successfully reversed by reducing CSF pressure (Cheung et al., 2002).

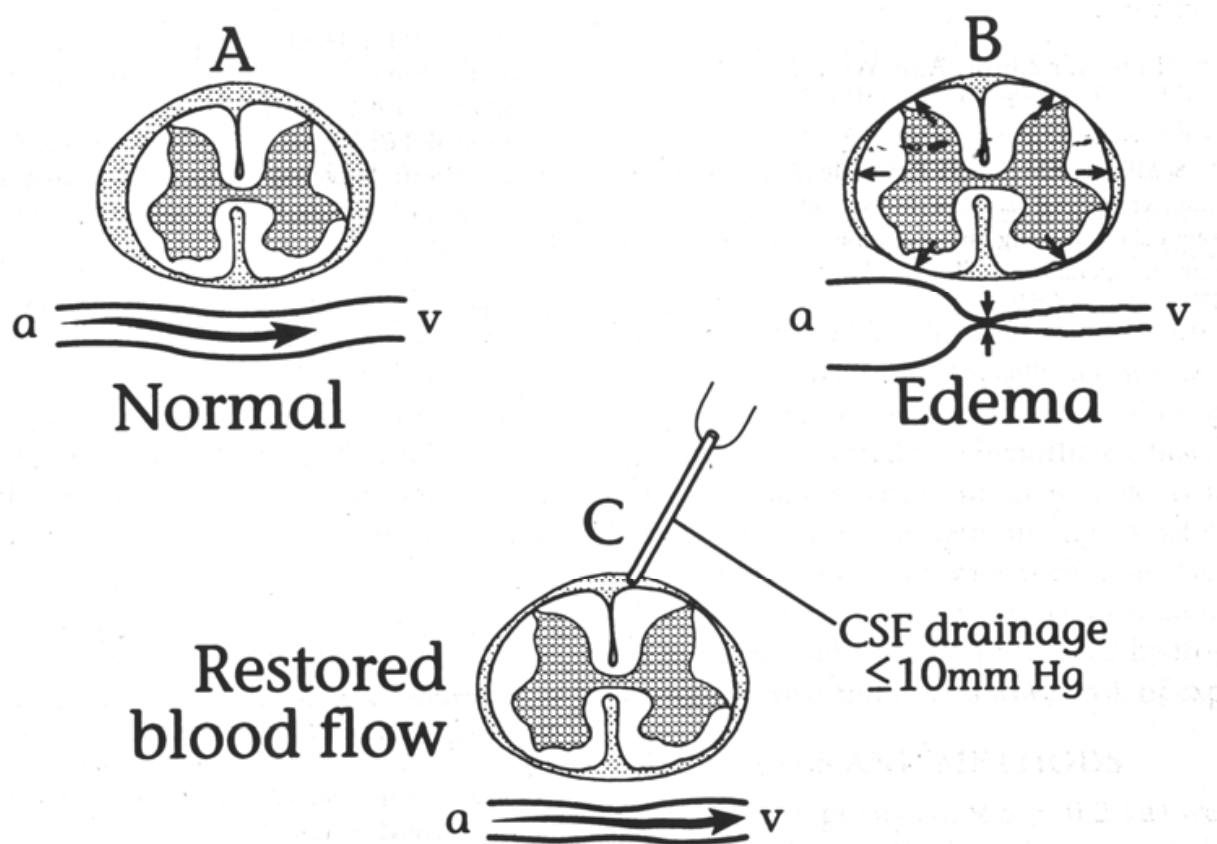


Fig. 8. Speculated mechanism of beneficial effect of cerebrospinal fluid drainage on spinal cord injury (Safi et al., 1997).

2.3.3 Role of cerebrospinal fluid drainage in ischemia-reperfusion injury

On the other hand, it has been well known that ischemia-reperfusion injury mediates some negative neurotrophic substances. Indeed, we showed that pro- and anti-inflammatory cytokines were generated in the rabbit spinal cord after ischemic SCI and they were clearly reversed by 21-aminosteroids (lazaroids) (Kunihara et al., 2000). Thus we have speculated that another key role of CSFD might be removal of negative neurotrophic substances. Actually, we revealed pro-inflammatory cytokines and S-100 β protein were predominantly generated in CSF than in serum after TAAA repair and their levels were more pronounced and prolonged in patients with SCI (Kunihara et al., 2001, Kuniharat et al., 2001) (Fig. 9, 10). Moreover, CSF levels of excitatory amino acids in patients with postoperative SCI was significantly higher than those in patients without SCI. These elevated CSF levels of excitatory amino acids could be significantly antagonized by intraoperative continuous intravenous administration of naloxone (Kunihara et al., 2004) (Fig. 11).

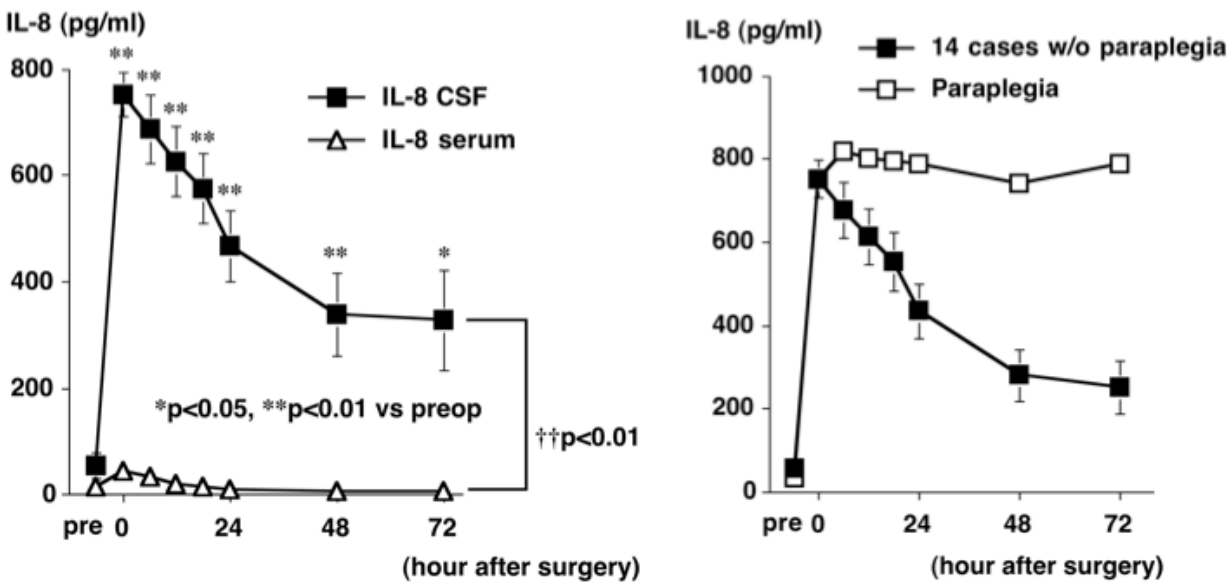


Fig. 9. Changes in serum and cerebrospinal fluid (CSF) interleukin-8 (IL-8) levels in overall patients (left). Changes in CSF IL-8 levels in patients with or without (w/o) paraplegia (right). (Kunihara et al., 2001)

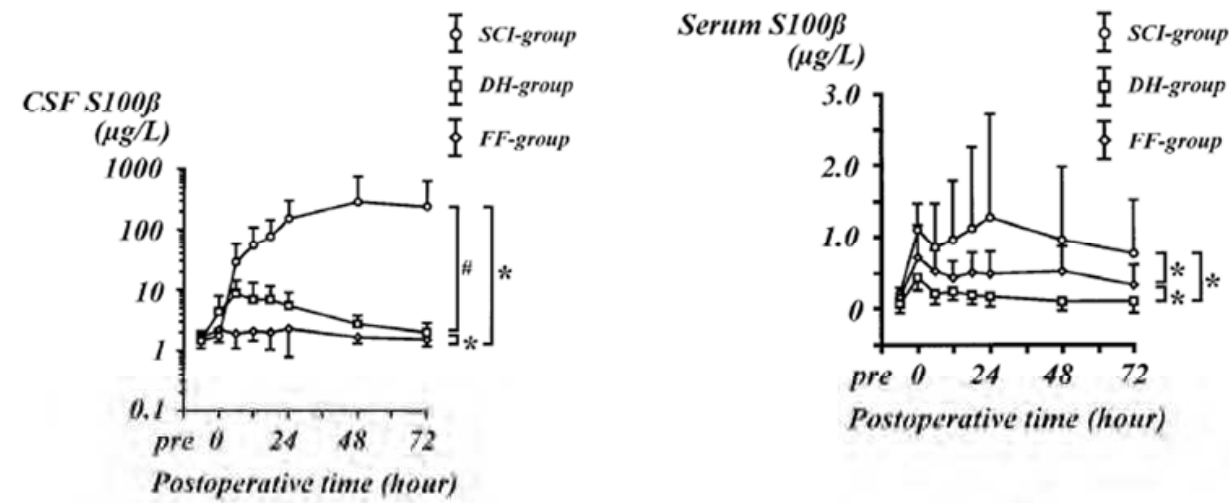


Fig. 10. Postoperative time course of S-100β protein levels in the serum and CSF (Kunihara et al., 2001).

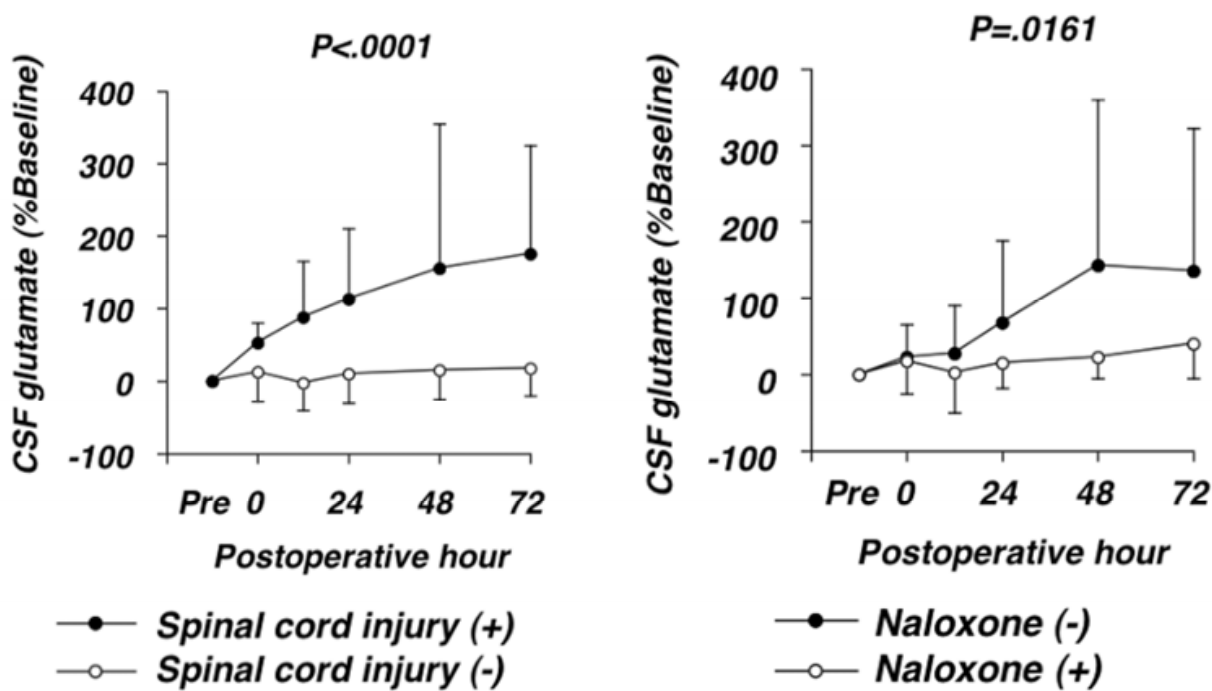


Fig. 11. Postoperative time course of CSF glutamate levels (Kunihara et al., 2004).

2.3.4 Pharmacological intervention

2.3.4.1 Naloxone hydrochloride

Numerous pharmacological interventions have been tried to attenuate postoperative SCI, however, no compound has become popular for a wide standardized clinical use. Above all, naloxone, the opiate antagonist, has gained increased attention since a report by Acher and associates was published in 1994, as described before (Acher et al., 1994). In general, excessive synaptic accumulation of excitatory amino acids during ischemia is associated with neuronal cell damage, which is mediated through overactivation of their receptors (i.e. N-methyl-D-aspartate receptor) followed by excessive influx of calcium. It has been shown that high concentration of naloxone attenuates N-methyl-D-aspartate-mediated neurotoxicity in an animal model (Kim et al., 1987). Clinically, Acher and his colleagues disclosed that the addition of naloxone resulted in a 30% reduction in the SCI rate (Acher et al., 2010). The important findings of our study that CSF excitatory amino acids were significantly suppressed by naloxone up to 72 hours after TAAA repair may have great implication because neurotoxicity mediated by excitatory amino acids is supposed to be a late-onset process (Nakamura et al., 1994) (Fig. 11). Therefore Acher and his colleagues recommended intravenous continuous administration of naloxone up to 48 hours postoperatively (Acher et al., 2010). We have experienced no side effect of naloxone at a rate of 1µg/kg/h even at postoperative period.

2.3.4.2 Other glutamate inhibitors

With the same mechanism, beneficial effects of other glutamate antagonists have been reported. Rokkas and his colleagues reported that Dextrorphan, a noncompetitive N-methyl-D-aspartate receptor antagonist, inhibited the release of excitatory amino acids in the spinal cord during ischemia (Rokkas et al., 1994). However, this agent is no longer used

clinically because of an unfavorable tolerance profile (Albers et al., 1995, Rokkas & Kouchoukos, 2001). Instead, other antiglutamate compounds such as Riluzole (Lang-Lazdunski et al., 2000), Memantine (Ehrlich et al., 1999), MK-801 (Cho et al., 2005) or Magnesium sulfate (Lang-Lazdunski et al., 2000) have been evaluated to have a neuroprotective effect after ischemia reperfusion of the spinal cord, although further evaluation should be necessary for their wide clinical use.

2.3.4.3 Steroid

In the early 1980's, beneficial effects of intravenous methylprednisolone (30 mg/kg) on experimental SCI have been confirmed (Hall & Braughler, 1982, Braughler & Hall, 1983, Laschinger et al., 1984). It has been believed that steroid act as cellular and lysosomal membrane stabilizers. Since the Second National Acute Spinal Cord Injury Study reported in 1990 that methylprednisolone improved neurologic recovery after acute SCI when it was given in the first eight hours, methylprednisolone has become a clinical mainstay in the treatment of acute SCI (Bracken et al. 1990). In the field of aortic surgery, methylprednisolone has been given in many institutions with various dosages (7 mg/kg: Kouchoukos et al., 2003, 30 mg/kg: Hollier et al., 1992, Acher C. 2010) although its effect is still controversial. We have routinely administered 1g methylprednisolone intravenously prior to aortic cross-clamping and another 1g into CPB circuit (Kunihara et al., 2004). A drawback of steroid use such as postoperative hyperglycemia, disadvantageous to spinal cord protection, should be aggressively normalized. Recently, a new series of 21-aminosteroids (i.e. Lazaroids) have emerged as remarkable protective compounds against various ischemia-reperfusion injury model through the inhibition of irondependent lipid peroxidation (Sasaki et al., 1996, Holzgrefe et al., 1990, Johnson & Lefer, 1990). As was written above, these agents have also been shown beneficial effects of attenuating postischemic SCI (Fowl et al., 1990, Kunihara et al., 2000). Further examination will be required for clinical application.

2.3.4.4 Vasodilators

The other beneficial mechanisms against postischemic SCI include enhancing collateral blood flow to the jeopardized spinal cord. Systemic administration of vasodilators (i.e. nitroprusside) has been believed to decrease spinal cord perfusion pressure by reducing distal aortic pressure and increasing cerebrospinal fluid pressure, thus it should be avoided or combination with CSFD is recommended (Shine & Nugent, 1990, Marini et al., 1997). On the other hand, local use (i.e. intrathecal) of vasodilators can avoid reducing distal aortic pressure, thus may be beneficial for spinal cord protection. Svensson and his colleagues administered papaverine intrathecally and showed significant increase in spinal cord blood flow (Svensson 2005) and significant reduction of SCI clinically (Svensson et al., 1998) (Figure 12).

Prostaglandin E1 (PGE1) has also emerged as one of the promising compounds to enhance collateral blood flow to the spinal cord (Grabitz et al., 1993), however, it has also a dilemma to reduce distal aortic pressure. To avoid PGE1-induced hypotension in clinical use, Alprostadil, PGE1 incorporated in lipid microspheres to minimize metabolism and inactivation in the lung (Yone et al., 1999), has been examined to have the beneficial effect on ischemic SCI. In an experimental spinal cord ischemia model, we detected beneficial pharmacological effect of intravenous Alprostadil on the spinal cord oxygenation using

near-infrared spectrophotometry, which was not detected by spinal cord evoked potentials (Figure 13) (Kunihara et al., 2008). Thus Alprostadil can be given systemically, which may facilitate intraoperative management.

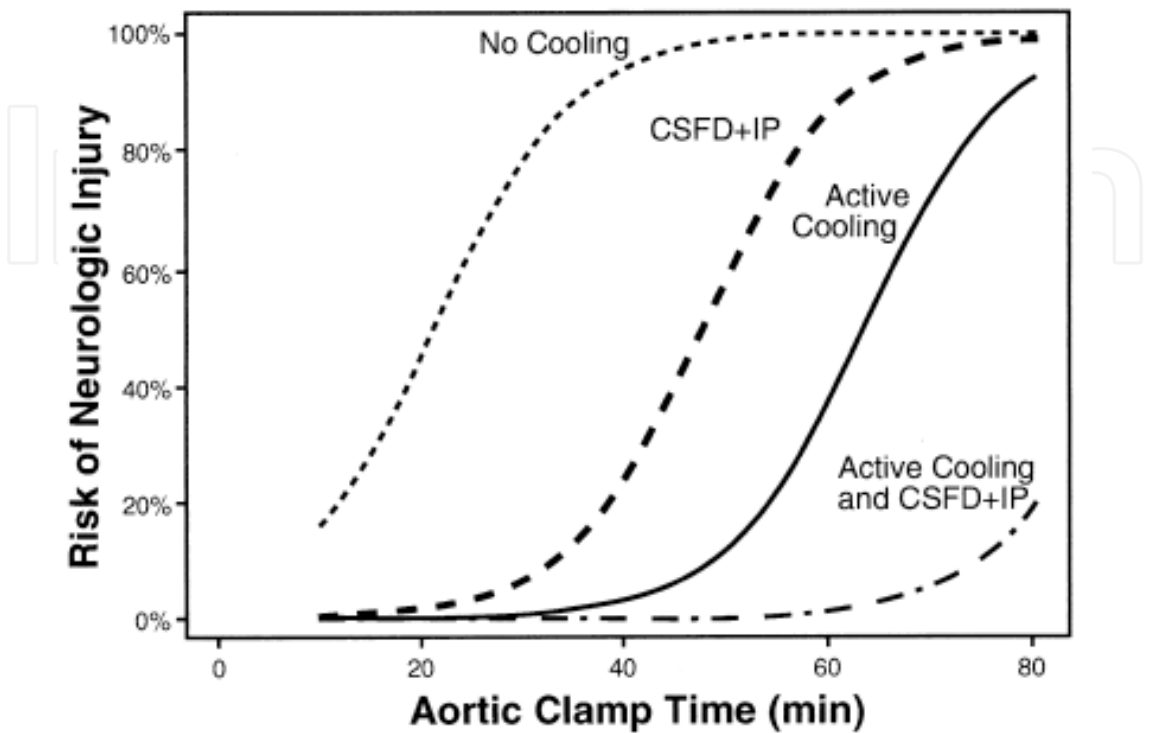


Fig. 12. Risk of spinal cord injury according to aortic cross-clamp time with no cooling or papaverine, cerebrospinal fluid drainage with intrathecal papaverine (CSFD+IP), active cooling with the combination. (Svensson et al., 1998)

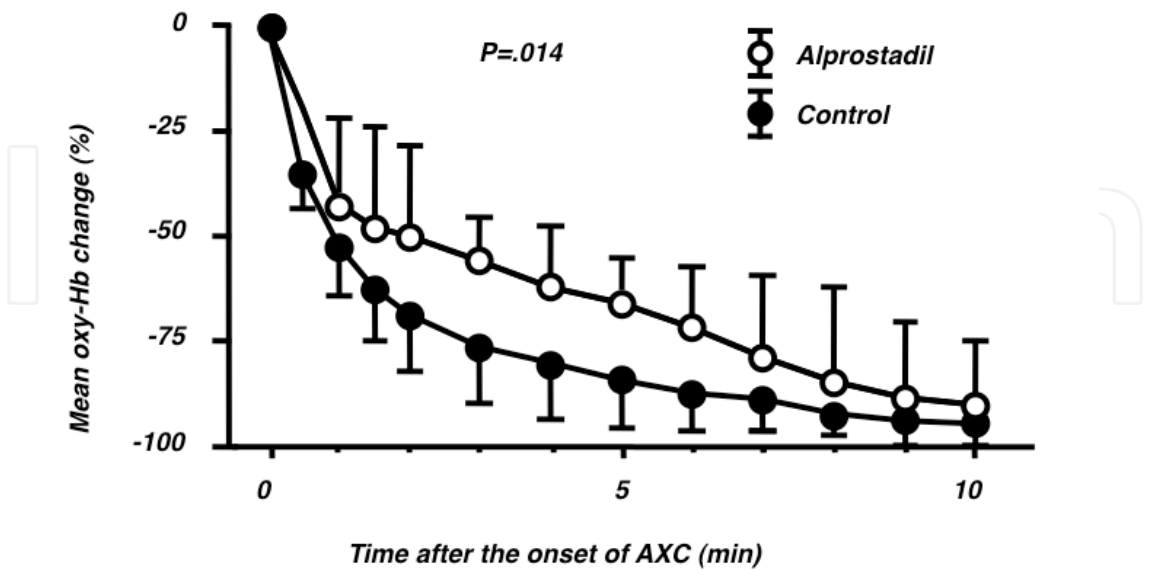


Fig. 13. Time course of mean %change of oxygenated hemoglobin (oxy-Hb) in the spinal cord measured by near-infrared spectrophotometry. AXC: aortic cross-clamping. (Kunihara et al., 2008)

2.3.4.5 Other drugs

Other numerous promising pharmacological interventions such as superoxide dismutase (Agee et al., 1991), allopurinol (Qayumi et al., 1994), calcium-channel blockers (Schitteck et al., 1992), Adenosine (Seibel t al., 1993) or Adenosine A2A agonist (ATL-146e) (Reece et al., 2004) have been tested. We have seriously published beneficial effects of nicorandil (an adenosine triphosphate-sensitive potassium channel opener) (Wakamatsu et al., 2001), immunophilin ligands FK506 or cyclosporine A (Tachibana et al., 2005) on transient spinal cord ischemia in rabbits. However, their protective effects on SCI seem still controversial and may require further evaluations.

3. Clinical results

With a better appreciation and understanding of the mechanisms and pathophysiology of SCI and with many clinical evidences the neurological outcomes after TAAA repair have dramatically improved. Table 3 illustrated postoperative SCI stratified by the extent of aortic involvement reported from institutions with large case volume.

Author	Year	Cases	Crawford/Safi’s classification					Total	DAP
			I	II	III	IV	V		
Svensson	1993	1509	15%	31%	7%	4%		16%	17%
Safi	2003	1004	0.5%	10.7%	6.0%	0.7%	2.0%	3.6%	73.8%
Jacobs	2006	112	0%	4.2%					100%
Coselli	2007	2286	3.3%	6.3%	2.6%	1.4%		3.8%	39.8%
Conrad/Cambria*	2007	455	9.1%	14.5%	11.6%	2.9%		9.5%	8%
Acher	2008	516	6.9%	15.8%	3.8%	0%		6.8%	no
Zoli/Griepp	2010	305	2.5%	11.5%	3.9%	2.2%		4.6%	95%

Table 3. Spinal cord injury after thoracoabdominal aortic aneurysm repair stratified by the extent of aortic involvement reported from institutions with large case volume. DAP: distal aortic perfusion. Conrad/Cambria*: only major paraplegia is demonstrated.

In spite of a contemporary and multidisciplinary approach with large experience, type II TAAA is still associated with high incidence of SCI as shown in table 3. Using a multivariate analysis, many authors have also emphasized that type II TAAA emerges as an independent predictor for postoperative SCI (Safi et al., 2005, Conrad et al., 2007, Schepens et al., 2009, Acher et al., 2010). Other preoperative independent predictors for postoperative SCI have been reported such as an emergent setting (Conrad et al., 2007, Acher 2010), aortic dissection (Schepens et al., 2009, Acher 2010), age >75 years old (Schepens et al., 2009), and renal dysfunction (Safi et al., 2005, 2008).

4. Conclusions

The underlying mechanisms and pathophysiology of SCI after TAAA repair may be multifactorial. We have thus reviewed the latest information with regard to this issue in this

manuscript briefly. From these considerable experimental and clinical evidences, we have evolved our strategy for TAAA repair over time as shown in Table 4 (Shiia et al., 2005). We believe this may be the best and reasonable approach to prevent SCI after TAAA repair at the present moment. Nonetheless, Jacobs and his colleagues described; “despite all available measures, complete prevention of paraplegia in type II aneurysms seems to be unrealistic” (Jacobs et al. 2006). Our multidisciplinary effort to prevent SCI after extensive TAAA repair may be never ending

Mild hypothermic distal aortic perfusion or deep hypothermic circulatory arrest
Multi-segmental sequential clamping
Preoperative identification of the Adamkiewicz artery and reimplantation of responsible intercostal arteries
Without information, reconstruction of the intercostal arteries at Th8-L1 left
Avoidance of steal phenomenon during reconstruction of the intercostal arteries
Electrophysiological monitoring of the spinal cord function
Cerebrospinal fluid drainage
Intravenous administration of naloxone and methylprednisolone.

Table 4. Our strategy to prevent spinal cord injury.

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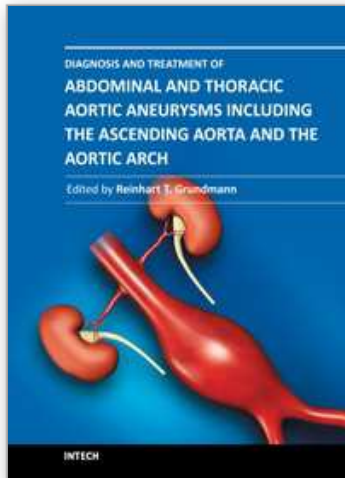
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This book considers diagnosis and treatment of abdominal and thoracic aortic aneurysms. It addresses vascular and cardiothoracic surgeons and interventional radiologists, but also anyone engaged in vascular medicine. The book focuses amongst other things on operations in the ascending aorta and the aortic arch. Surgical procedures in this area have received increasing attention in the last few years and have been subjected to several modifications. Especially the development of interventional radiological endovascular techniques that reduce the invasive nature of surgery as well as complication rates led to rapid advancements. Thoracoabdominal aortic aneurysm (TAAA) repair still remains a challenging operation since it necessitates extended exposure of the aorta and reimplantation of the vital aortic branches. Among possible postoperative complications, spinal cord injury (SCI) seems one of the most formidable morbidities. Strategies for TAAA repair and the best and most reasonable approach to prevent SCI after TAAA repair are presented.

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