Paraplegia after extensive thoracic and thoracoabdominal aortic aneurysm repair: Does critical spinal cord ischemia occur postoperatively?

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Objective: Spinal cord injury can occur not only during extensive thoracoabdominal aneurysm repair but also postoperatively, causing delayed-onset paraplegia.

Methods: A series of 858 thoracoabdominal aneurysm repairs (June 1990–June 2006) with an overall paraplegia rate of 2.7% was analyzed retrospectively. Serial segmental artery sacrifice was monitored by using somatosensory evoked potentials; segmental arteries were not reimplanted. Of a total of 20 cases of paraplegia, 3 occurred intraoperatively and 7 occurred late postoperatively; these will not be analyzed further. In 10 cases (the paraplegia group) spinal cord injury occurred within 48 hours after thoracoabdominal aneurysm repair, despite intact somatosensory evoked potentials at the end of the procedure. These patients with early postoperative delayed paraplegia were compared with 10 matched control subjects who recovered without spinal cord injury.

Results: In the paraplegia group a median of 9 segmental arteries (range, 5–12 segmental arteries) were sacrificed. There were 9 male subjects; median age was 63 years (range, 40–79 years), and 4 of 10 had cerebrospinal fluid drainage. A median of 9 segmental arteries (range, 2–12 segmental arteries) were also sacrificed in the matched recovery group. There were 4 male subjects; median age was 66 years (range, 40–78 years), and 8 of 10 had cerebrospinal fluid drainage. During the first 48 hours postoperatively, there were no significant differences in arterial and mixed venous oxygen saturation, partial arterial O₂ and CO₂ pressures, body temperature, glucose, hematocrit, or pH. The mean central venous pressures, however, were significantly higher in the paraplegic patients from 1 to 5 hours postoperatively ($P = .03$). In addition, although absolute mean aortic pressures did not differ between matched pairs postoperatively, when pressures were considered as a percentage of individual antecedent preoperative mean aortic pressure, paraplegic patients had significantly lower values during the first 5 hours postoperatively ($P = .03$).

Conclusions: This study suggests that paraplegia can result from inadequate postoperative spinal cord perfusion caused by relatively minor differences from control subjects in perfusion parameters. Delayed paraplegia can perhaps be prevented with better hemodynamic and fluid management.

Spinal cord injury is the most dreaded complication of repair of descending thoracic and thoracoabdominal aneurysms (TAA/A), whether by means of open surgical repair or endovascular strategies.¹⁻³ A number of adjuncts have been successfully used to counteract the consequences both of spinal cord ischemia during surgical intervention and a precarious spinal cord blood supply postoperatively, and the incidence of paraplegia and paraparesis at centers for aneurysm repair has been decreasing.⁴⁻¹² However, the occasional case of spinal cord injury still occurs, and it remains unclear which factors are most important in preventing either immediate or delayed paraplegia.

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Abbreviations and Acronyms

CSF	= cerebrospinal fluid
CVP	= central venous pressure
MEP	= motor evoked potential
SA	= segmental artery
SSEP	= somatosensory evoked potential
TAA/A	= thoracic and thoracoabdominal aortic aneurysm

Recent clinical and experimental studies suggest that ischemic spinal cord injury is not inevitable, even with extensive segmental artery (SA) sacrifice.¹³⁻¹⁵ With the aid of intraoperative electrophysiologic monitoring, it has become apparent that some patients do not sustain any spinal cord injury intraoperatively with extensive sacrifice of intercostal and lumbar arteries but nevertheless have paraplegia either immediately postoperatively or even weeks after the operation. This so-called delayed-onset paraplegia accounts for up to one third of cases of postoperative permanent spinal cord injury^{16,17} and in recent experience constitutes the majority of patients with spinal cord injury at our institution.

The current retrospective study concerns 10 cases of paraplegia that developed within 48 hours after surgical intervention despite intact somatosensory evoked potentials (SSEPs) throughout the operation. In this study the patients with paraplegia are compared with 10 matched control patients operated on contemporaneously who recovered spinal cord function. All available intraoperative and postoperative physiologic measurements that might have had an influence on spinal cord function were compared between affected patients and their matched control subjects to try to pinpoint factors that might have contributed to the development of this early, postoperative, delayed-onset spinal cord injury.

Materials and Methods

A series of 858 TAA/A repairs (June 90–June 2006) in which permanent postoperative paraplegia or severe paraparesis developed in 20 (2.7%) patients was analyzed retrospectively. The institutional review board approved this research; additional patient consent was not required.

The overall hospital mortality in the entire series of patients (including emergencies and reoperations) was 9.7%.

Paraplegia Group

This report focuses on 10 cases of delayed-onset permanent paraplegia in which spinal cord injury occurred within 48 hours after TAA/A repair involving SA sacrifice, despite intact SSEPs at the end of the procedure: these are designated the paraplegia group, and their clinical characteristics are outlined in Table 1. These cases of early postoperative paraplegia despite intact SSEPs intraoperatively represent the largest subgroup among cases of paraplegia or paraparesis at our institution since the introduction of SSEP monitoring.¹⁸

All the patients with spinal cord injury in this series of patients with TAA/A are depicted in Figure 1. Although SSEP monitoring

TABLE 1. Clinical profile, aneurysm cause, aneurysm extent, and intraoperative data

	Recovery, n (%)	Paraplegia, n (%)
Demographics		
Mean $\pm$ SD age, y	64 $\pm$ 11	61 $\pm$ 12
Age >60 y	7	5
Male sex	5	9
Previous cardioaortic operations	4 (2)	2 (—)
Timing of surgical intervention		
Elective	7	5
Urgent/emergency	3	5
Risk factors		
History of neurologic dysfunction	2	1
History of hypertension	8	7
Coronary artery disease	2	2
Smoking	4	7
Diabetes	2	1
COPD	0	3
Cause (aorta)		
Degenerative	1	1
Marfan's syndrome	0	1
Atherosclerosis	8	7
Dissection	1	4
Other	—	1*
Extent of aortic replacement		
Crawford I	5	6
Crawford II	2	3
Crawford III	3	1
Extent of segmental artery sacrifice		
Segmental arteries sacrificed (mean $\pm$ SD)	9 $\pm$ 3	9 $\pm$ 3
Intraoperative findings		
Aneurysm diameter, mm (mean $\pm$ SD)	71 $\pm$ 9	71 $\pm$ 17
Intraluminal clot	1	4
Intramural hematoma	1	1
With ulcerated perforation	1	—
Contained rupture	1	3
Bypass technique		
CPB/DHCA	4	1
Femoral-femoral bypass	1	3
Distal aortic perfusion (Biomedicus circuit)	5	6
Open distal anastomosis	9	9
Postoperative management		
Cerebrospinal fluid drainage	4	8

SD, Standard deviation; COPD, chronic obstructive pulmonary disease; CPB, cardiopulmonary bypass; DHCA, deep hypothermic circulatory arrest. *Luteal aneurysm.

was not introduced until 1993 (or motor evoked potential [MEP] monitoring until 2002), the operative strategy and postoperative management of all the patients in this series was otherwise the same. We designated 3 patients with severe intraoperative ischemia with SSEP loss as having experienced immediate postoperative paraplegia. One of these recovered spinal cord function postoperatively. In addition to the 10 patients with early delayed paraplegia,

Figure 1. Permanent spinal cord injury (n = 20) after thoracic and thoracoabdominal aortic aneurysm (TAA/A) repair with segmental artery sacrifice and somatosensory evoked potential (SSEP) monitoring in 858 patients. Three additional patients had transient spinal cord ischemia (intraoperative = 1; late-delayed = 2), all of whom recovered with vasopressor therapy.

there were 7 patients who had onset of spinal cord injury several days to several weeks after the procedure. Five of these patients had paraplegia (one after cardiopulmonary resuscitation and another after a laparotomy), and 2 were left with paraparesis. No patient had paraplegia more than 3 weeks after SA sacrifice during TAA/A repair. Neither the patients with intraoperative nor those with late delayed-onset paraplegia will be considered further.

Recovery Group

Ten matched control subjects who recovered without spinal cord injury (ie, with equivalent aortic disease and TAA/A repair [Figure 2] by using the same surgical technique) were selected. The control subjects, designated the recovery group, were chosen by taking the individuals who had undergone roughly equivalent operations closest in time to the paraplegic patients but had survived with intact

spinal cord function. The postoperative parameters analyzed were not included in the database from which the patients were selected and therefore could not have influenced the choice of control subjects. Patient characteristics in the recovery group are also shown in Table 1.

Comparability of Groups

Figure 2 shows the extent of the aneurysm by showing the exact anatomic localization of the proximal and distal margins of aneurysmal resection; this information is also given for the matched control subjects. We believe that this is a more precise designation than aneurysm extent, as defined by the traditional Crawford classification, but the latter is also shown for each group in Table 1.

The most common indication for TAA/A replacement (Table 1) was an atherosclerotic aortic aneurysm, which was noted in 7 of the paraplegic patients and 8 of the control subjects; a chronic dissection, however, was present in 4 of the patients in the paraplegia group and 1 of the control subjects. One patient in the paraplegia group had Marfan syndrome.

As might have been anticipated, hypertension was present in most of the patients in both groups: 8 in the control group and 7 in the paraplegia group. There was a comparable incidence of history of coronary artery disease ($P = 1.0$). Of factors thought to be associated with a generally less favorable outcome, age was marginally higher in the recovery group, which also contained more female patients, slightly more patients with diabetes, and more patients with previous abdominal aneurysm operations. Several other risk factors, however, were more prevalent in the paraplegia group: a higher proportion of smokers and patients with chronic obstructive pulmonary disease and situations requiring urgent and emergency operations.

Among intraoperative factors, the diameter of the aneurysm was the same in both the paraplegia and recovery groups, as was the extent of the aneurysm, reflected by the number of SAs that were sacrificed (Figure 2). The patients in the paraplegia group, however, had a somewhat higher incidence of intraluminal clot and of contained rupture.

Operative Management

All patients are placed in the standard thoracoabdominal position. A double-lumen endotracheal tube is used to isolate the left lung.

Figure 2. Extent of segmental artery (SA) sacrifice during thoracoabdominal aneurysm repair (TAASA) in paraplegic versus recovered patients precisely defining the proximal and distal extent of aneurysm resection. *n.s.*, Not significant.

A right radial arterial line, a right common femoral line, and a pulmonary artery catheter are inserted. Intraoperative transesophageal echocardiography is used in all patients. Since 1990, a spinal catheter has been placed whenever possible, and cerebrospinal fluid (CSF) pressure has been monitored during the operation and for the subsequent 72 hours: the CSF is drained at a maximum rate of 15 mL/h, as long as the CSF pressure remains greater than 10 mm Hg. Since 1993, SSEP monitoring has been used intraoperatively, with the addition of MEP monitoring since 2002. SSEP monitoring is continued for the first 12 hours postoperatively.^{4,19}

Operative Technique

The essential steps of our approach to the repair of descending TAA/As have been described previously. The aorta is accessed through a left thoracotomy or thoracoabdominal incision. The diaphragm is divided circumferentially. The infradiaphragmatic aorta is exposed through a retroperitoneal approach. Once the aneurysm has been fully exposed, the SAs are serially temporarily occluded, and if no change in the MEPs or SSEPs occurs, each one is subsequently ligated before the aneurysm is removed. All operations are carried out under moderate hypothermia (32°C). If needed, deep hypothermia is used, with circulatory arrest initiated at a bladder temperature of 15°C and a jugular bulb cerebral venous saturation of 95% or greater.

Postoperative Management

Aggressive fluid administration for at least the first 24 postoperative hours is initiated, aiming for a mean aortic pressure of 80 to 90 mm Hg, with peripheral vasoconstrictors administered as necessary to maintain this pressure. Gentle diuresis is begun 48 to 72 hours after the operation. SSEPs, when used, are monitored until the patient awakens. Thereafter, hourly brief neurologic examinations are performed for 72 hours. CSF drainage (as previously described) is continued for 72 hours. Steroids are tapered over 48 to 72 hours.

Statistical Methods

All statistical analyses of these data were based on methods for matched pairs, although for the purpose of clinical interest, some outcomes are described as overall medians, means, or percentages for the paraplegic patients and the recovered control subjects. McNemar tests with exact *P* values were used for comparing categorical data. Wilcoxon signed-rank tests were used to compare continuous characteristics. The first 5 hourly repeated measures were compared in a random-effects mixed model. A similar model was used for comparing the patients and control subjects in terms of the average of their nonmissing measurements during the first 5 hours, hours 6 to 24, and hours 25 to 48.

Results: Comparability of Experimental Groups

Although some differences in preoperative characteristics were present (as noted above), the groups were comparable with regard to many of the most important known risk factors for mortality and paraplegia. There were no significant differences in aneurysm extent between matched pairs (Figure 2): Wilcoxon tests showed no significant differences with regard to the extent of SA sacrifice, with 9 SAs (range, 5–12 SAs) sacrificed in the paraplegia group and 9 SAs (range, 2–12

SAs) sacrificed in the recovery group. The diameter of the aneurysm, averaging 7.1 cm, was also the same in both groups. The median age was not significantly different: 63 years (range, 40–79 years) in the paraplegia group and 66 years (range, 40–78 years) in the recovery group.

Tests of case-control differences among 10 paraplegic patients and their matched nonparaplegic control subjects (McNemar tests with exact *P* values) revealed a slight preponderance of male subjects (*P* = .125) in the paraplegia group, as well as a history of smoking (*P* = .375), as shown in Table 1. Multiple other factors were also tested, but with the small numbers involved, there was no chance of finding significance because cases and control subjects were the same in 7 or more pairs: this was true of the presence of diabetes and chronic obstructive pulmonary disease.

Intraoperatively, the most important finding was that all the patients had intact SSEPs throughout the procedure. There were, however, as noted above, more patients in the paraplegia group with emergency or urgent procedures, clot noted during the course of the operation (*P* = .38), and contained rupture (*P* = .69). Although the mean temperatures were the same in both groups, there were more patients who underwent operations with deep hypothermic circulatory arrest in the recovery group.

During the first 48 hours postoperatively, there were no significant differences in mean arterial O₂ saturation, arterial partial O₂ and CO₂ pressures, body temperature during re-warming, glucose levels, hematocrit values, and pH. Mixed venous saturation, which was used to detect variability in cardiac output during the postoperative period, also did not significantly differ between the pairs in the paraplegia and recovery groups (Table 2).

The mean central venous pressures (CVPs; Figure 3), however, were significantly higher in the paraplegic patients from 1 through 5 hours postoperatively (overall *P* = .03); the most marked differences occurred at 2, 3, and 4 hours (*P* = .02, *P* < .005, and *P* = .03, respectively). Of note (see the Discussion section), 4 of 10 of the group with subsequent paraplegia had CSF drainage, in contrast with 8 of 10 patients who had recovery of function (*P* = .125, Table 1).

In addition, closer examination of the mean arterial pressure showed that although the absolute mean pressures were not different between the groups (Figure 4, A), if the pressures were considered in relation to each patient's antecedent baseline arterial pressure, which was obtained after placement of the arterial line at hospital admission or before surgical intervention, then the arterial pressures in the paraplegic patients were significantly less than those in the control group during the first 5 hours postoperatively (*P* = .027; Figure 4, B).

The overall hospital mortality, defined as death in the hospital or within 30 days postoperatively, was 5 of 10 in the paraplegia group. Furthermore, only 2 patients with paraplegia survived the first postoperative year. In contrast, there

TABLE 2. Postoperative data

Variable	Hours postoperatively	Mean*		Matched pair differences†
		Recovery	Paraplegia	
Arterial O ₂ saturation	1–5	97.7	97.8	–1.1
	6–24	97.5	97.1	–0.4
	25–48	95.8	96.5	0.7
Mixed venous O ₂ saturation	1–5	77.4	73.6	–4.1
	6–24	76.2	73.2	–3.2
	25–48	76.0	73.3	–2.7
Temperature	1–5	34.4	34.3	0.1
	6–24	37.0	37.1	0.2
	25–48	37.2	37.3	0.2
Hematocrit	1–5	35.9	38.0	1.5
	6–24	35.0	35.8	0.8
	25–48	33.5	33.6	0.1
P _{O₂}	1–5	172.3	163.7	–8.6
	6–24	105.6	111.4	5.7
	25–48	96.6	92.5	–4.2
P _{CO₂}	1–5	37.6	44.0	6.4
	6–24	35.8	36.8	1.0
	25–48	36.7	37.0	0.3
Glucose	1–5	253.8	226.5	–27.3
	6–24	213.2	185.2	–27.9
	25–48	163.8	155.9	–7.9
pH	1–5	7.4	7.4	–0.02
	6–24	7.4	7.4	0.01
	25–48	7.5	7.5	–

*Based on the average of each person's hourly values during the corresponding period. †Mean differences for matched pairs: Recovered control subjects–Paraplegic patients.

were no deaths among the control subjects either shortly after the operation or within 1 year postoperatively (Table 1).

Discussion

This review of our experience with paraplegia after TAA/A repair has yielded a number of interesting insights, although the small number of patients makes it difficult to draw firm conclusions. We interpret our observations in light of a somewhat unorthodox theory of the pathogenesis of spinal cord injury after TAA/A surgery and a surgical strategy that does not require reattachment of any SAs. Our theory is that the pathogenesis of spinal cord ischemic injury has 2 potential components: intraoperative and postoperative. In instances of paraplegia in which SSEPs are intact after SA sacrifice, as was true in our selected paraplegia group, we theorize that whatever intraoperative injury has occurred is not severe enough to cause functional impairment. Therefore, our objective in this review was to find differences in immediate postoperative management that would distinguish those patients who subsequently had paraplegia from those who recovered seemingly normal function. Although this is a retrospective report and involves only a small number of patients, our

Figure 3. Mean central venous pressure (mCVP) during postoperative intensive care unit stay after thoracic and thoracoabdominal aortic aneurysm replacement.

review of all patients with paraplegia at our institution after aneurysm repair has documented that this group, with early postoperative spinal cord injury, constitutes the largest single subgroup of patients who have paraplegia after surgical intervention and therefore seems to warrant close scrutiny.

After serial surgical sacrifice of SAs during aneurysm resection, the perfusion of the spinal cord depends on the stabilization of the collateral network of remaining SAs, fed from below by the hypogastric arteries and from above by the internal thoracic artery and other branches from the subclavian arteries. The pressure conducted through these vessels to the spinal cord, the spinal cord perfusion pressure, is a balance between the inflow and outflow pressures within the closed confines of the spinal canal. The inflow obviously depends principally on arterial pressure, which is largely determined by cardiac output, blood volume, and the competing demands of viscera and muscle tissue connected to the same collateral network. It is therefore not surprising to find that there is an effect of arterial pressure on the development of spinal cord injury. Every aneurysm surgeon has anecdotal cases in which patients have experienced delayed-onset paraplegia after dramatic instances of severe hypotension, even several weeks postoperatively. What is surprising in this study is that the hypotension that precipitated spinal cord injury within the first 48 hours after surgical intervention is quite subtle and depends on viewing appropriate postoperative blood pressure in terms of antecedent ambulatory pressures rather than absolute values. Our neurosurgical colleagues observe loss of intraoperative SSEPs quite often in patients with chronic hypertension if intraoperative blood pressures are not maintained at high normal levels, and MEP or SSEP loss intraoperatively in aneurysm operations, as well as spinal

Figure 4. A, Mean aortic pressure (MAP) during postoperative intensive care unit stay after thoracic and thoracoabdominal aortic aneurysm replacement. B, Postoperative mean aortic pressure relative to antecedent baseline pressure (rMAP) before surgical intervention (A, radialis; no vasoactive drugs added to the patient's medication).

surgery, is often correctable by raising the blood pressure. The findings of this study thus support a policy of maintaining blood pressures at high levels not only intraoperatively, which has even become the practice with endovascular repair, but also for at least 48 hours postoperatively.^{20,21} This should especially be emphasized in patients with antecedent hypertension.

The finding that a high CVP is also associated with spinal cord injury is somewhat more of a surprise but is quite consistent with the idea that spinal cord perfusion after aneurysm surgery is very precarious. Outflow from the spinal canal depends directly on CSF and venous pressures²² and whether spinal cord edema is present. Tobinick and Vega²³ describe

the human vertebral venous system as a unique, large-capacity, valveless venous network in which flow is bidirectional and includes the vertebral venous plexuses, which course along the entire length of the spinal cord and anastomose with the intracranial veins in the suboccipital region. Caudally, the vertebral venous system communicates freely with the sacral and pelvic veins and with the prostatic venous plexus. The cerebrospinal venous system plays an important role in the regulation of intracranial pressure and venous outflow from the brain,²³ and cerebral venous outflow pathways have been shown to depend on CVP.²⁴ An increased CVP is therefore likely to be reflected by increased pressure in the extensive vertebral venous plexuses²⁵ and would thereby impair spinal cord outflow.

From direct measurements from collateral vessels feeding the spinal cord in pigs and in human subjects, we know that spinal cord perfusion pressures are well below aortic pressures, even at baseline, and that after SA sacrifice, these pressures decrease to a level as low as 20 mm Hg several hours postoperatively.⁴ At such low values of inflow pressure, it is easy to imagine that a high venous pressure could significantly impede spinal cord perfusion. An appreciation of the vascular anatomy within the spinal canal in pigs shows that branches of the SAs directly supplying the anterior spinal artery have to cross the extensive venous plexuses surrounding the spinal cord, which is likely to be distended with an increased CVP, and therefore a high CVP could also mechanically impede arterial inflow in addition to its effect on outflow.

At these low perfusion pressures, it is easy to imagine that CSF drainage is also important, although the numbers in this study, in which not all patients had spinal cord drainage, are too small to confirm its effect. The effectiveness of spinal cord drainage in reducing the incidence of paraplegia and paraparesis, however, has been firmly established by other investigators.^{5,26} In 1991, Grum and Svensson²² described a strong positive correlation between intraoperative CSF pressure and CVP ($r = 0.9$) before aortic crossclamping. In a recent series of 29 patients, Eide and colleagues²⁷ confirmed this finding during and after TAA/A repair ($r = 0.8$) and observed that the occurrence of neurologic deficits was related to the intraoperative level of CSF pressure, which was greater than 10 mm Hg in the majority of injured patients.

The high early postoperative and 1-year mortality among patients with paraplegia (but not among control subjects) is also not surprising and has been noted in other studies.^{28,29} We acknowledge that the number of patients in this study is quite small and that the matching for preoperative and intraoperative characteristics is far from perfect. Some factors more common among the control subjects, such as more advanced age and diabetes, would seem to predict a worse prognosis, but others that are more prevalent in the paraplegia group might have contributed to their risk of an adverse outcome, among them chronic obstructive pulmonary disease,

emergency operation, and intraoperative clot. Nevertheless, this review of our experience suggests that some cases of paraplegia, with its attendant serious morbidity and high mortality, might be preventable with more meticulous attention to postoperative hemodynamics and fluid management, which is sometimes complicated by concurrent renal failure. The need for a high postoperative blood pressure but a low CVP suggests that use of inotropes is likely to be important in optimizing early postoperative hemodynamics.

Our observations also add weight to the notion that endovascular therapy of these extensive aneurysms might be an achievable goal if accompanied by careful monitoring of spinal cord function and sophisticated hemodynamic management in the immediate postoperative interval, including use of CSF drainage. Direct monitoring of spinal cord perfusion pressure would enable postoperative management specifically addressing the needs of the spinal cord after serial SA sacrifice during open repair or occlusion during endovascular repair.

We recognize that this retrospective study has limitations related to the very small numbers of patients with paraplegia and the difficulty of finding perfectly matched control subjects. Our control subjects were picked blindly and in an effort to allow for changes in technique and experience over time, and all statistical comparisons were carried out in matched pairs. Nonetheless, we cannot be absolutely certain that some unrecognized bias in control patient selection did not occur. Consequently, our findings must be considered suggestive rather than conclusive. But in light of the grave consequences of paraplegia, we believe that even imperfect observations regarding the importance of hemodynamic management during the first 48 hours after TAA/A surgery are worth documenting in the hope that they will add to our understanding of this tragic and possibly avoidable complication.

References

- Griep RB, Griep EB. Spinal cord perfusion and protection during descending thoracic and thoracoabdominal aortic surgery: the collateral network concept. *Ann Thorac Surg.* 2007;83(suppl):S865-92.
- Conrad MF, Crawford RS, Davison JK, Cambria RP. Thoracoabdominal aneurysm repair: a 20-year perspective. *Ann Thorac Surg.* 2007;83(suppl):S856-61; S890-2.
- Roselli EE, Greenberg RK, Pfaff K, Francis C, Svensson LG, Lytle BW. Endovascular treatment of thoracoabdominal aortic aneurysms. *J Thorac Cardiovasc Surg.* 2007;133:1474-82.
- Etz CD, Halstead JC, Spielvogel D, Shahani R, Lazala R, Homann TM, et al. Thoracic and thoracoabdominal aneurysm repair: is reimplantation of spinal cord arteries a waste of time? *Ann Thorac Surg.* 2006;82:1670-7.
- Coselli JS, Lemaire SA, Koksoy C, Schmittling ZC, Curling PE. Cerebrospinal fluid drainage reduces paraplegia after thoracoabdominal aortic aneurysm repair: results of a randomized clinical trial. *J Vasc Surg.* 2002;35:631-9.
- Kouchoukos NT, Masetti P, Rokkas CK, Murphy SF. Hypothermic cardiopulmonary bypass and circulatory arrest for operations on the descending thoracic and thoracoabdominal aorta. *Ann Thorac Surg.* 2002;74(suppl):S1885-7; S1892-8.
- Safi HJ, Miller CC 3rd. Spinal cord protection in descending thoracic and thoracoabdominal aortic repair. *Ann Thorac Surg.* 1999;67:1937-9; 1953-8.
- Cambria RP, Davison JK, Carter C, Brewster DC, Chang Y, Clark KA, et al. Epidural cooling for spinal cord protection during thoracoabdominal aneurysm repair: A five-year experience. *J Vasc Surg.* 2000;31:1093-102.
- Estrera AL, Miller CC 3rd, Huynh TT, Porat E, Safi HJ. Neurologic outcome after thoracic and thoracoabdominal aortic aneurysm repair. *Ann Thorac Surg.* 2001;72:1225-31.
- Schepens M, Dossche K, Morshuis W, Heijmen R, van Dongen E, Ter Beek H, et al. Introduction of adjuncts and their influence on changing results in 402 consecutive thoracoabdominal aortic aneurysm repairs. *Eur J Cardiothorac Surg.* 2004;25:701-7.
- Jacobs MJ, Mess W, Mochtar B, Nijenhuis RJ, Statius van Eps RG, Schurink GW. The value of motor evoked potentials in reducing paraplegia during thoracoabdominal aneurysm repair. *J Vasc Surg.* 2006;43:239-46.
- Svensson LG, Hess KR, D'Agostino RS, Entrup MH, Hreib K, Kimmel WA, et al. Reduction of neurologic injury after high-risk thoracoabdominal aortic operation. *Ann Thorac Surg.* 1998;66:132-8.
- Etz CD, Homann TM, Plestis KA, Zhang N, Luehr M, Weisz DJ, et al. Spinal cord perfusion after extensive segmental artery sacrifice: can paraplegia be prevented? *Eur J Cardiothorac Surg.* 2007;31:643-8.
- Acher CW, Wynn MM. Technique of thoracoabdominal aneurysm repair. *Ann Vasc Surg.* 1995;9:585-95.
- Biglioli P, Spirito R, Porqueddu M, Agrifoglio M, Pompilio G, Parolari A, et al. Quick, simple clamping technique in descending thoracic aortic aneurysm repair. *Ann Thorac Surg.* 1999;67:1038-44.
- Wong DR, Coselli JS, Amerman K, Bozinovski J, Carter SA, Vaughn WK, et al. Delayed spinal cord deficits after thoracoabdominal aortic aneurysm repair. *Ann Thorac Surg.* 2007;83:1345-55.
- Huynh TT, Miller CC 3rd, Safi HJ. Delayed onset of neurologic deficit: significance and management. *Semin Vasc Surg.* 2000;13:340-4.
- Griep RB, Ergin MA, Galla JD, Lansman S, Khan N, Quintana C, et al. Looking for the artery of Adamkiewicz: a quest to minimize paraplegia after operations for aneurysms of the descending thoracic and thoracoabdominal aorta. *J Thorac Cardiovasc Surg.* 1996;112:1202-15.
- Galla JD, Ergin MA, Lansman SL, McCullough JN, Nguyen KH, Spielvogel D, et al. Use of somatosensory evoked potentials for thoracic and thoracoabdominal aortic resections. *Ann Thorac Surg.* 1999;67:1947-58.
- Jacobs MJ, Meylaerts SA, de Haan P, de Mol BA, Kalkman CJ. Strategies to prevent neurologic deficit based on motor-evoked potentials in type I and II thoracoabdominal aortic aneurysm repair. *J Vasc Surg.* 1999;29:48-59.
- Strauch JT, Lauten A, Zhang N, Wahlers T, Griep RB. Anatomy of spinal cord blood supply in the pig. *Ann Thorac Surg.* 2007;83:2130-4.
- Grum DF, Svensson LG. Changes in cerebrospinal fluid pressure and spinal cord perfusion pressure prior to cross-clamping of the thoracic aorta in humans. *J Cardiothorac Vasc Anesth.* 1991;5:331-6.
- Tobinick E, Vega CP. The cerebrospinal venous system: anatomy, physiology, and clinical implications. *MedGenMed.* 2006;8:53.
- Gisolf J, van Lieshout JJ, van Heusden K, Pott F, Stok WJ, Karemaker JM. Human cerebral venous outflow pathway depends on posture and central venous pressure. *J Physiol.* 2004;560:317-27.
- Pearce JM. The craniospinal venous system. *Eur Neurol.* 2006;56:136-8.
- Acher CW, Wynn MM, Hoch JR, Popic P, Archibald J, Turnipseed WD. Combined use of cerebral spinal fluid drainage and naloxone reduces the risk of paraplegia in thoracoabdominal aneurysm repair. *J Vasc Surg.* 1994;19:236-48.
- Eide TO, Romundstad P, Stenseth R, Aadahl P, Myhre HO. Spinal fluid dynamics during thoracic- and thoracoabdominal aortic surgery. *Int Angiol.* 2006;25:46-51.
- Cambria RP, Clouse WD, Davison JK, Dunn PF, Corey M, Dorer D. Thoracoabdominal aneurysm repair: results with 337 operations performed over a 15-year interval. *Ann Surg.* 2002;236:471-9.
- Jacobs MJ, Mommertz G, Koeppel TA, Langer S, Nijenhuis RJ, Mess WH, et al. Surgical repair of thoracoabdominal aortic aneurysms. *J Cardiovasc Surg (Torino).* 2007;48:49-58.