Original Article

Evolving Strategy and Results of Spinal Cord Protection in Type I and II Thoracoabdominal Aortic Aneurysm Repair

Norihiko Shiiya, MD, Takashi Kunihara, MD, Kenji Matsuzaki, MD, and Keishu Yasuda, MD

Purpose: **We report our strategy and results of spinal cord protection in Crawford I and II thoracoabdominal aortic replacement.**
Methods: **Retrospective analysis of 43 elective operations. Before 1994, we reconstructed segmental arteries during a single period of blood flow interruption in 11 of 12 patients, using distal aortic perfusion and evoked spinal cord potential (ESCP) monitoring. Deep hypothermia was used in one. Since 1994, we used multi-segmental sequential repair, in which T8-L1 arteries were sequentially reconstructed irrespective of evoked potential change, in 20 of 31 patients. In the remaining 11, deep hypothermia was used. Cerebrospinal fluid drainage (CSFD) was introduced in 1996 (n=26), and continuous infusion of naloxone in 1999 (n=17).**
Results: **In patients undergoing distal aortic perfusion without multi-segmental sequential repair, six spinal cord injuries including two deaths occurred. Change in evoked potentials was observed in nine of 10 monitored patients. With multi-segmental sequential repair, only one spinal cord injury occurred, and three of 11 monitored patients showed evoked potential change. With deep hypothermia, no spinal cord injury occurred. Multivariate analysis identified operation without multi-segmental sequential repair as a risk factor for spinal cord injury (p=0.008).**
Conclusion: **Evolving strategy resulted in an improved outcome. Both multi-segmental sequential repair and deep hypothermia were more effective than our previous technique. (Ann Thorac Cardiovasc Surg 2005; 11: 178–85)**

Key words: spinal cord protection, thoracoabdominal aortic aneurysm, distal aortic perfusion, deep hypothermia, cerebrospinal fluid drainage

Introduction

Ischemic spinal cord injury remains a major morbidity after thoracoabdominal aortic operation in the patients with more extensive disease such as Crawford type I and II.[1-3] We have previously reported a spinal cord protection strategy, in which the segment of the aorta that includes critical intercostal arteries is determined by combined use of evoked spinal cord potential (ESCP) monitoring, segmental resection of the aorta, and distal aortic perfusion, and these arteries are "selectively" reconstructed.[4] In our hands, however, incidence of spinal cord injury was high despite this strategy, presumably because of the relatively long duration of ischemia that was required for reconstruction.

To overcome this problem, we have introduced new strategies that may reduce the severity of ischemia during reconstruction (the multi-segmental sequential repair technique[5] and cerebrospinal fluid drainage(CSFD),[3,6] may prolong the safe duration of ischemia (deep hypothermia),[7] or may reduce the severity of ischemia/reperfusion injury (naloxone infusion).[8] The purpose of this study is to report our results of spinal cord protection in the Crawford type I and II extent according to these evolving strategies.

From Department of Cardiovascular Surgery, Hokkaido University Hospital, Sapporo, Japan

Received November 10, 2004; accepted for publication January 13, 2005.
Address reprint requests to Norihiko Shiiya, MD: Department of Cardiovascular Surgery, Hokkaido University Hospital, N14W5, Kita-ku, Sapporo 060-8648, Japan.

Materials and Methods

From October 1991 through July 2004, 91 patients underwent replacement of the thoracoabdominal aorta in our hospital. Among these, 20 patients had Crawford type I lesion and 25 had type II lesion. Two patients with type I lesion who underwent emergent operation for rupture were excluded because the spinal cord protection strategy reported here was not applied and no intercostal reconstruction was performed. Records of the remaining 43 patients who underwent elective operation were retrospectively analyzed. The patient's age ranged from 26 to 77 (median 60). Twenty-four patients were men, and 27 had chronic aortic dissection including eight patients with Marfan's syndrome. Maximal diameter of the aneurysms was more than 60 mm, and that from the distal descending aorta to the upper abdominal aorta was more than 50 mm. History of aortic operation was present in 16 patients, which included aortic arch replacement in 10 (four with elephant trunk, three with Bentall operation, one with coronary artery bypass grafting), operation on the descending thoracic aorta in two, and replacement of the infrarenal abdominal aorta in seven. Chronic renal dysfunction (serum creatinine >2.0-mg/dL) was present in two. Coronary artery disease was present in three.

Evolution of spinal cord protection strategy

Among the 12 patients who underwent operation before August 1994, only one patient underwent deep hypothermic operation for simultaneous proximal aortic reconstruction. The remaining 11 patients underwent operation under distal aortic perfusion, with a proximal clamp placed between the arch vessels in eight patients. In these 11, our previous strategy of "selective" intercostal reconstruction was applied. We used partial cardiopulmonary bypass as distal aortic perfusion and determined the critical arteries by combined use of ESCPs monitoring and segmental aortic resection. Namely, when ESCP showed change during resection of an aortic segment, arteries in this segment were considered critical and reconstructed.[4] For ESCPs monitoring, two bipolar electrodes were placed in the epidural space at the level of T12-L1 for electrical stimulation and C7-T1 for recording. Square-wave pulses were applied at a rate of 10 to 20/sec, and the evoked potentials were averaged five to 20 times to eliminate electrical noise.[4] In this strategy, descending thoracic aorta was resected in two or three large segments, so that each segment contained three or more intercostal arteries. They were usually reconstructed as a group during a single period of blood flow interruption. The number of reconstructed arteries was 3.3±2.7 (range 0 to 8).

Since September 1994, we have introduced the multi-segmental sequential repair technique, in which the aorta at the level of T8-L1 was resected in three or more segments and arteries in this area were sequentially reconstructed (Fig. 1). Reconstructed arteries were reperfused before the next aortic segment was opened, so that blood flow interruption was limited to only one or two pairs of arteries that were being reconstructed. After the proximal anastomosis was completed using double clamping, the distal clamp was moved to the mid-descending thoracic aorta, and one pair of intercostal arteries from the proximal descending thoracic aorta was usually reconstructed. Then, using the multi-segmental sequential repair technique, we usually reconstructed three pairs of arteries from the T8-L1 area. The distal clamp was usually placed twice on the distal descending thoracic aorta and once on the supra-celiac abdominal aorta. In the post-dissection aneurysms application of multiple clamps was usually possible with a big clamp. In the non-dissecting aneurysms we selected clamping sites where thrombus formation and calcification were not severe and the aorta showed narrowing. The separate graft technique was used in all but one patient with the multi-segmental sequential repair because the distal clamp was present in the proximity of the arteries. 10-mm tube grafts were most frequently used as side-arm grafts. Although one pair of arteries was connected to one side-arm graft in most cases, two pairs may also be connected in some cases when the next pair was closely located. The number of reconstructed arteries was 4.3±1.7 (range 2 to 8). Intercostal reconstruction was "non-selectively" performed irrespective of ESCP change. Therefore ESCP monitoring was no more an integral part of the strategy, and was not used in every patient. This technique was applied in 20 patients, with ESCP monitored in only 11 patients.

In the remaining 11 patients who underwent operation after September 1994, deep hypothermia was used during intercostal reconstruction. This means that we became more aggressive for the use of deep hypothermia than before (1/12 before August 1994 vs. 11/31 after September 1994). Besides spinal cord protection, indication for deep hypothermic operation was the use of open proximal aortic anastomosis in five patients (no patient underwent operation under distal aortic perfusion with a clamp between the arch vessels during this period) and simultaneous reconstruction of the proximal aorta in one. One other patient who had three-channel dissection underwent

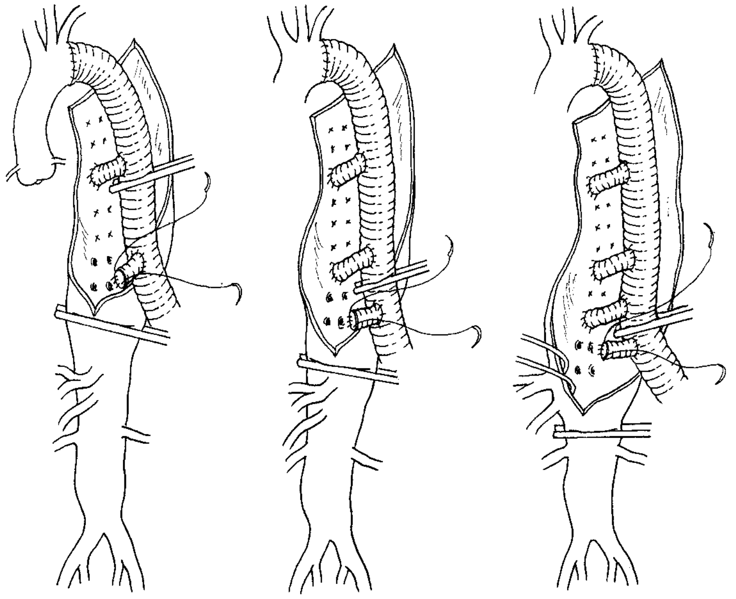

Fig. 1. Schematic drawing of the multi-segmental sequential technique. In this illustration the distal descending thoracic aorta is clamped once.

deep hypothermic operation because of the risk of malperfusion associated with retrograde distal aortic perfusion. In the remaining four patients, spinal cord protection was the only reason for deep hypothermic operation, and both the proximal and distal perfusion were maintained during reconstruction. In these patients, application of multiple clamps was judged not suitable because of the aneurysm size and thickness of mural thrombus. The patients with brain circulatory arrest were cooled down to achieve a urinary bladder temperature below 18°C, while target urinary bladder temperature was set at 22°C in those without brain circulatory arrest. The number of reconstructed arteries in the patients undergoing deep hypothermic operation (n=12, one before August 1994 and 11 after September 1994) was 3.8±1.8 (range 1 to 8).

Since August 1996, we have introduced CSFD. We used a 16G-indwelling catheter, which was inserted through the L4/5 intervertebral space after induction of the anesthesia. Cerebrospinal fluid was allowed to freely drain if the pressure exceeded 13 cmH_2O. CSFD was discontinued from 12 hours to 72 hours after surgery according to the patients' condition. This was used in 26 patients, 18 with the multi-segmental sequential repair technique and eight with deep hypothermic operation. In the patients undergoing deep hypothermic operation, however, we have recently abandoned the use of CSFD because of the fear of subdural hematoma reported by others.[9] Although this complication is reported to be caused by excessive drainage[9] and we, like Cheung and colleagues,[10] have not so far experienced this in our patients, concern of anticoagulation and postoperative coagulopathy prompted us to abandon it.

Continuous intravenous infusion of naloxone at a dose of 1 μg/kg/h[8] has been used since April 1999 in 17 patients, 11 with the multi-segmental sequential repair technique and six with deep hypothermic operation.

Data analysis

To determine factors associated with spinal cord injury, the following variables were entered into the analysis; adjuncts and reconstruction method (deep hypothermia, distal aortic perfusion with or without multi-segmental sequential repair), CSFD, naloxone, patients' age, gender, extent of repair (Crawford I or II), etiology (dissection or not), chronic renal insufficiency, coronary artery disease, chronic obstructive pulmonary disease (COPD), history of previous proximal and abdominal aortic operation, redo operation, number of involved segmental arteries, number of reconstructed segmental arteries, proximal clamp site (between the arch vessels, descending aorta, or open anastomosis), aortic clamp time. To clarify the role of the multi-segmental sequential repair technique, the same variables were also evaluated in the patients undergoing operation with distal aortic perfusion (n=31).

Data are presented as mean ± standard deviation. Statistical analyses were performed using the SPSS 11.0 for Windows software (SPSS Inc, Chicago, IL). Univariate analysis was performed with the chi-square test and Fisher's exact test for categorical variables and analysis of variance test for continuous variables. Multivariate analysis was performed with stepwise logistic regression analysis. All variables with a p-value less than 0.1 on univariate analysis were entered into the multivariate analysis, in which backward elimination with the likelihood-ratio statistic was used.

Results

Clinical outcome

There were two hospital deaths (5%). Both patients underwent operation under distal aortic perfusion without multi-segmental sequential repair, had Crawford I extent, and developed postoperative spinal cord injury. One had liver cirrhosis and chronic renal dysfunction before operation. The causes of death in these two patients were multiple organ failure.

Prolonged ventilator support (>48-h) was required in six patients (55%) without multi-segmental sequential repair and four patients (33%) undergoing deep hypothermic operation. Of the latter four patients, two underwent simultaneous replacement of the ascending aorta and aortic arch. No patient undergoing operation under distal aortic perfusion with multi-segmental sequential repair required prolonged ventilator support.

Postoperative hemodialysis was required in the two patients who died during hospitalization. Temporary increase in serum creatinine level to more than 3.0-mg/dL was observed in two other patients, one without multi-segmental sequential repair with abnormal preoperative renal function and another undergoing deep hypothermic operation for simultaneous replacement of the ascending aorta and aortic arch. The overall incidence of postoperative renal insufficiency was thus 9% (4/43).

Spinal cord injury

Spinal cord injury occurred in seven patients (16%), two with paraplegia and five with paraparesis. Six of these seven patients underwent distal aortic perfusion without multi-segmental sequential repair, and two of them died. The remaining one patient underwent distal aortic perfusion with multi-segmental sequential repair. No spinal cord injury occurred in those patients undergoing deep hypothermic operation. Two of the five patients with paraparesis, one without multi-segmental sequential repair and another with it, eventually recovered and became able to walk without assistance. Therefore only three patients without multi-segmental sequential repair had persisting disabling symptoms at discharge.

In the univariate analysis for the entire cohort (Table 1), use of distal aortic perfusion without multi-segmental sequential repair (p=0.0005), proximal clamp between the arch vessels (p=0.0023), no use of CSFD (p=0.0106), chronic renal dysfunction (p=0.0233), and no use of naloxone (p=0.0310) were identified to have significant association with spinal cord injury. In the multivariate analysis, the use of distal aortic perfusion without multi-segmental sequential repair (p=0.008, odds ratio: 24.8) was identified as a significant independent risk factor for spinal cord injury. Results of the analysis for the patients with distal aortic perfusion were similar. In the univariate analysis, operation without multi-segmental sequential repair (p=0.0036), proximal clamp between the arch vessels (p=0.0056), no use of CSFD (p=0.0124), chronic renal dysfunction (p=0.0452), and no use of naloxone (p=0.0331) were identified to have significant association with spinal cord injury (Table 2). In the multivariate analysis, operation without multi-segmental sequential repair (p=0.026, odds ratio: 15.2) was identified as a significant independent risk factor for spinal cord injury.

ESCP change

In the patients undergoing operation with distal aortic perfusion and ESCP monitoring (n=21), intraoperative ESCP change suggesting spinal cord ischemia was observed in nine out of 10 patients without multi-segmental sequential repair, while three out of 11 patients with multi-segmental sequential repair showed ischemic ESCP change (p=0.0075). Among the 12 patients with intraoperative ESCP change six patients developed spinal cord injury, while no patient had a deficit in the absence of ESCP change.

Patency of intercostals reconstructed by separate tube grafts

Patency of intercostals reconstructed by separate tube grafts was evaluated by postoperative computed tomography and aortography when available. Arteries were considered patent when both the side-arm graft and the intercostal arteries were continuously visualized. The patency could be studied in 27 patients (18 with multi-segmental sequential repair and nine with deep hypothermia) in whom the separate tube graft technique was used either exclusively or in combination with other techniques. Among the 76 grafts thus studied, 32 (42%) were judged patent.

Discussion

The present results suggest that both the multi-segmental sequential repair technique and deep hypothermia are

Table 1. Analysis of the factors associated with spinal cord injury

Variables	SCI (n=7)	no SCI (n=36)	univariate P	multivariate P	odds ratio	95% confidence interval
Adjuncts and methods			0.0003			
Deep hypothermia*	0	12	0.1630			
DAP with MSSR*	1	19	0.1003			
DAP without MSSR	6	5	0.0005	0.008	24.8	2.28-270
CSFD*	1	25	0.0106			
Naloxone*	0	17	0.0310			
Age (Y)	58±6	59±14	0.9893			
Gender (Male)	5	19	0.4370			
Extent (I:II)	4:3	14:22	0.4274			
Etiology (Dissection)	5	22	0.6950			
Chronic renal dysfunction	2	0	0.0233			
Coronary artery disease	0	3	>0.9999			
COPD	3	9	0.3778			
History of proximal aortic op.	1	9	>0.9999			
History of abdominal aortic op.	0	7	0.5769			
Redo op.	1	1	0.3023			
Involved segmental arteries	11.7±2.2	11.1±2.3	0.4864			
Reconstructed segmental arteries	3.3±2.1	4.0±2.0	0.3749			
Proximal clamp			0.0022			
Between arch vessels	5	4	0.0023			
Descending aorta*	2	26	0.0398			
Open anastomosis	0	6	0.5671			
Aortic clamp time (min)	218±135	157±63	0.0672			

SCI: spinal cord injury, DAP: distal aortic perfusion, MSSR: multi-segmental sequential repair, CSFD: cerebrospinal fluid drainage, COPD: chronic obstructive pulmonary disease, op.: operation, *: protective

more effective than our previous technique for spinal cord protection. Deep hypothermic operation was associated with more pulmonary complication than operation under distal aortic perfusion with multi-segmental sequential repair, a finding consistent with previous reports.[11] Therefore our preference is to use the multi-segmental sequential repair technique, and deep hypothermic operation is preserved for those who have specific indication such as a need for open proximal aortic anastomosis or those who are not suitable for the multi-segmental sequential repair technique. The multi-segmental sequential repair technique seems to reduce the severity of ischemia during reconstruction because ischemic ESCP change was less prevalent with this technique.

Griepp and colleagues have shown that ligation of the entire intercostal arteries before aortic crossclamping never resulted in spinal cord ischemia as detected by somatosensory evoked potentials.[12] When 11 or more intercostals were sacrificed, however, this resulted in permanent ischemic dysfunction of the cord in 9.5% of patients and additional 9.5% suffered from temporary dysfunction of the cord.[12] In addition, half of them were delayed-onset injuries. Similarly, Acher and colleagues, rapidly oversewing all intercostal arteries, have reported 20% incidence of spinal cord injury in Crawford type II aneurysms.[8] They reported that this incidence became even lower when CSFD and naloxone were used. These results suggest that in the absence of steal phenomenon, blood flow interruption to the intercostal arteries alone does not produce critical spinal cord ischemia in more than 80% of patients, but will make the cord blood flow precarious depending on the collateral circulation through multiple other feeders. Although reconstruction of the intercostal arteries may be effective to avoid this precarious situation after operation, it carries the a risk of exacerbating ischemia during reconstruction, either through the additional period of aortic crossclamping or through the steal phenomenon.

The multi-segmental sequential repair technique may reduce the risk of exacerbating ischemia during reconstruction through two mechanisms. The first is the maintenance of collateral blood flow during reconstruction of the intercostals. It is well known that a rich collateral network is present between the adjacent intercostal arteries.[5] During operation, we frequently noticed more back bleeding from an intercostal artery, once the adjacent ones were reperfused. It is therefore suggested that even if a spinal cord feeding artery is included in a resected seg-

Table 2. Univariate analysis of the factors associated with spinal cord injury in the patients undergoing distal aortic perfusion

Variables	SCI (n=7)	no SCI (n=24)	univariate P	multivariate P	odds ratio	95% confidence interval
DAP without MSSR	6	5	0.0036	0.026	15.2	1.38–168
CSFD*	1	17	0.0124			
Naloxone*	0	11	0.0331			
Age (Y)	58±6	58±15	0.8860			
Gender (Male)	5	10	0.2200			
Extent (I:II)	4:3	10:14	0.6705			
Etiology (Dissection)	5	13	0.6672			
Chronic renal dysfunction	2	0	0.0452			
Coronary artery disease	0	1	>0.9999			
COPD	3	7	0.6518			
History of proximal aortic op.	1	5	>0.9999			
History of abdominal aortic op.	0	3	>0.9999			
Redo op.	1	1	0.4065			
Involved segmental arteries	11.7±2.2	10.9±2.1	0.3970			
Reconstructed segmental arteries	3.3±2.1	4.1±2.1	0.3602			
Clamp between arch vessels	5	3	0.0056			
Aortic clamp time (min)	218±135	169±63	0.1812			

SCI: spinal cord injury, DAP: distal aortic perfusion, MSSR: multi-segmental sequential repair, CSFD: cerebrospinal fluid drainage, COPD: chronic obstructive pulmonary disease, op.: operation, *:protective

ment of the aorta, we can expect a considerable amount of collateral blood flow to this artery as far as blood flow to the neighboring arteries is maintained. In our previous segmental aortic resection technique, arteries in the critical segment are reconstructed as a group, while blood flow to all of these is interrupted. Therefore no collateral blood flow is expected from the neighboring arteries during reconstruction. In the multi-segmental sequential repair technique, by contrast, blood flow to the neighboring intercostal arteries can be maintained if blood flow to the reconstructed arteries is reestablished before resection of the next aortic segment and distal aortic perfusion is adequate.

The second mechanism is reduction of the risk of steal phenomenon. In this technique, only one or two pairs of intercostal arteries are usually included in a single resected segment. Therefore less blood is stolen from the arteries being reconstructed, even if back bleeding is not completely controlled by either balloon occlusion or application of bulldog clamps. In addition to these two mechanisms, combined use of CSFD may have also contributed to augment collateral blood flow. A recent randomized clinical trial by Coselli and colleagues[6] has shown benefits of CSFD in type I and II repair.

Reconstruction of one proximal intercostal artery is performed for two reasons. One is to provide potential source for collateral blood flow during subsequent reconstruction of the arteries in the T8-L1 area. Another is the result of angiographic studies that spinal cord feeding arteries may be present in the proximal descending thoracic aorta.[13,14] We[4] and others[14] have reported experiences in which arteries in these area was critical for spinal cord blood supply.

The drawbacks of the multi-segmental sequential repair technique may include prolonged total aortic crossclamp time. In reported series, aortic crossclamp time is identified as a risk factor for spinal cord injury,[1,8,15] although it is not clear that the influence of crossclamp time remains the same when the distal aorta is continuously perfused. In the current series, the aortic clamp time was 196±115 minutes in our previous technique and 172±64 minutes with the multi-segmental sequential repair (p=0.4559). In addition, it was not identified as a risk factor for spinal cord injury, as expected, in the patients undergoing operation with distal aortic perfusion. Therefore in the multi-segmental sequential repair technique the benefit of reducing severity of ischemia during reconstruction seems to exceed the risk of prolonged aortic crossclamping.

Another concern of our technique is the patency rate of the intercostals reconstructed with a separate tube graft, which was 42% at the time of discharge in the current series. From this point of view, it is not a good technique. We prefer it because of the technical reason, which is related to the use of multi-segmental sequential repair technique. Therefore it may be better if other techniques such as direct vessel reattachment are feasible with the use of multi-segmental sequential repair.

Despite the low patency rate at the time of discharge, however, incidence of spinal cord injury was low with this technique. In our series there was only one case of immediate paraparesis and no delayed-onset injury occurred among the 29 patients who underwent intercostal reconstruction with the separate graft technique together with the multi-segmental sequential repair or deep hypothermia. This suggests that long-term patency of the occluded arteries was not necessary to maintain spinal cord blood flow except in the one patient with paraparesis. We usually reconstruct three pairs of intercostals in the T8-L1 segment, because we do not know which artery is supplying the spinal cord. Results of anatomical studies showed that only one spinal cord feeding artery is usually present in this area.[16] We expect that, by reconstructing three pairs out of six, one of these three is directly supplying the cord or, if it is not the case, three pairs are enough to maintain cord blood flow through the collateral network. Therefore patency of all these three is not always necessary. In addition, results of Griepp and colleagues[12] and Acher and associates[8] that closure of all the intercostal and lumbar arteries did not result in ischemic spinal cord injury in more than 80% suggest that long-term patency of the reconstructed arteries is not necessary in more than 80% of patients.

The benefit of multi-segmental sequential repair technique can not be expected when many intercostals are occluded before operation. In this situation the cord blood supply may be significantly altered, and contribution of arteries outside the T8-L1 area may be important. In addition, if there is only one patent artery in the T8-L1 area, we can not expect collateral blood flow from neighboring arteries. The multi-segmental sequential repair technique is thus more suitable for post-dissection aneurysms, in which the number of occluded intercostals is much less than that in the atherosclerotic aneurysms. In addition, post-dissection aneurysms usually do not have severe atheromatous disease, which may be a source for atheroembolism when multiple clamps are applied. Fortunately, 63% of our patients had post-dissection aneurysms. Recent publications have shown that chronic dissection is not a risk factor for spinal cord injury when distal aortic perfusion is used.[15,17,18] This is also true in our own experience.

We used ESCP change as an indication of spinal cord ischemia, because it correlates well with the neurologic outcome in our experiences. We have not so far experienced immediate spinal cord injury in the absence of ESCP change or in patients with complete return of the change.[19,20] In the patients undergoing operation with our previous technique (distal aortic perfusion without multi-segmental sequential repair), we have detected spinal cord ischemia during operation in all but one patient. Jacobs and colleagues,[21] using myogenic motor evoked potentials (MEPs) and the segmental resection technique that is similar to our previous technique, have reported that exclusion of the aortic segment between T5 and L1 in type II lesion resulted in abnormal MEP levels in 41%. They also reported that exclusion between L1 and L5 or stopping iliac perfusion resulted in abnormal MEP levels in 20%. Ischemic change during resection in their report thus seems less prevalent than that in our patients without multi-segmental sequential repair.

This may be explained by the difference in the monitoring technique and patient population. We used any reproducible change in the amplitude of ESCP as an indication of ischemia, because ESCP is elicited and recorded directly from the cord between two epidural electrodes and is therefore not influenced by neuromuscular blockade or peripheral nerve ischemia. When myogenic MEP is used, however, amplitude that decreased to less than 25[21] to 50%[22] of baseline is usually used as an indication of " critical" spinal cord ischemia. This is because smaller changes in amplitude can not be distinguished from variation due to the influence of anesthetics. Therefore less severe ischemia may not be detected when 25% criterion is used, although myogenic MEP is motor-specific and very sensitive to ischemia. In addition, although post-dissection aneurysms were prevalent in our series, Jacobs' series included patients with atherosclerotic aneurysms only, which usually have less patent intercostals and thereby less steal phenomenon during resection.

Protective effects of deep hypothermia on the spinal cord are well established.[23] Kouchoukos and colleagues have reported 2.4% (1/42) incidence of spinal cord injury in type II repair.[7] Nevertheless, only a few surgeons are routinely using it during thoracoabdominal aortic operations because of adverse effects of deep hypothermia on the respiratory or coagulatory systems. Safi and colleagues have reported 29% mortality and 67% respiratory complication rate in a series of 21 patients, and recommend the judicious application of this method in rare instances with no other option.[11] In our experience the results have so far been satisfactory. Except for the two patients who underwent simultaneous proximal aortic replacement, in only two patients prolonged ventilator support was required and in no patient reexploration for bleeding was required. These results may be thanks to the recent advances in the patient management and cardiopulmonary bypass equipment.

Limitation of the study

This is a retrospective analysis and the number of patients is small. There was a patient selection bias for each technique. The study includes the elective operations only, and the results are not applicable for the acute cases. Therefore further study with larger number of patients is required to validate our observation.

References

1. 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.
2. Coselli JS. The use of left heart bypass in the repair of thoracoabdominal aortic aneurysms: current techniques and results. *Semin Thorac Cardiovasc Surg* 2003; **15**: 326–32.
3. Safi HJ, Miller CC 3rd, Huynh TT, et al. Distal aortic perfusion and cerebrospinal fluid drainage for thoracoabdominal and descending thoracic aortic repair: ten years of organ protection. *Ann Surg* 2003; **238**: 372–81.
4. Shiiya N, Yasuda K, Matsui Y, Sakuma M, Sasaki S. Spinal cord protection during thoracoabdominal aortic aneurysm repair: results of selective reconstruction of the critical segmental arteries guided by evoked spinal cord potential monitoring. *J Vasc Surg* 1995; **21**: 970–5.
5. Kuniyoshi Y, Koja K, Miyagi K, et al. Prevention of postoperative paraplegia during thoracoabdominal aortic surgery. *Ann Thorac Surg* 2003; **76**: 1477–84.
6. 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.
7. 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**: S1885–8.
8. Acher CW, Wynn MM, Hoch JR, Kranner PW. Cardiac function is a risk factor for paralysis in thoracoabdominal aortic replacement. *J Vasc Surg* 1998; **27**: 821–30.
9. Dardik A, Perler BA, Roseborough GS, Williams GM. Subdural hematoma after thoracoabdominal aortic aneurysm repair: an underreported complication of spinal fluid drainage? *J Vasc Surg* 2002; **36**: 47–50.
10. Cheung AT, Pochettino A, Guvakov DV, Weiss SJ, Shanmugan S, Bavaria JE. Safety of lumbar drains in thoracic aortic operations performed with extracorporeal circulation. *Ann Thorac Surg* 2003; **76**: 1190–7.
11. Safi HJ, Miller CC 3rd, Subramaniam MH, et al. Thoracic and thoracoabdominal aortic aneurysm repair using cardiopulmonary bypass, profound hypothermia, and circulatory arrest via left side of the chest incision. *J Vasc Surg* 1998; **28**: 591–8.
12. Griepp RB, Ergin MA, Galla JD, 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.
13. Kieffer E, Richard T, Chiras J, Godet G, Cormier E. Preoperative spinal cord arteriography in aneurysmal disease of the descending thoracic and thoracoabdominal aorta: preliminary results in 45 patients. *Ann Vasc Surg* 1989; **3**: 34–46.
14. Williams GM, Perler BA, Burdick JF, et al. Angiographic localization of spinal cord blood supply and its relationship to postoperative paraplegia. *J Vasc Surg* 1991; **13**: 23–35.
15. Coselli JS, LeMaire SA, de Figueiredo LP, Kirby RP. Paraplegia after thoracoabdominal aortic aneurysm repair: is dissection a risk factor? *Ann Thorac Surg* 1997; **63**: 28–36.
16. Biglioli P, Roberto M, Cannata A, et al. Upper and lower spinal cord blood supply: the continuity of the anterior spinal artery and the relevance of the lumbar arteries. *J Thorac Cardiovasc Surg* 2004; **127**: 1188–92.
17. Safi HJ, Miller CC 3rd, Estrera AL, et al. Chronic aortic dissection not a risk factor for neurologic deficit in thoracoabdominal aortic aneurysm repair. *Eur J Vasc Endovasc Surg* 2002; **23**: 244–50.
18. Schepens MA, Vermeulen FE, Morshuis WJ, et al. Impact of left heart bypass on the results of thoracoabdominal aortic aneurysm repair. *Ann Thorac Surg* 1999; **67**: 1963–80.
19. Matsui Y, Goh K, Shiiya N, et al. Clinical application of evoked spinal cord potentials elicited by direct stimulation of the cord during temporary occlusion of the thoracic aorta. *J Thorac Cardiovasc Surg* 1994; **107**: 1519–27.
20. Matsui Y, Shiiya N, Ishii K, et al. The reliability of evoked spinal cord potentials elicited by direct stimulation of the cord as a monitor of spinal cord ischemia during temporary occlusion of the thoracic aorta. *Panminerva Med* 1997; **39**: 78–84.
21. Jacobs MJ, de Mol BA, Elenbaas T, et al. Spinal cord blood supply in patients with thoracoabdominal aortic aneurysms. *J Vasc Surg* 2002; **35**: 30–7.
22. van Dongen EP, Schepens MA, Morshuis WJ, et al. Thoracic and thoracoabdominal aortic aneurysm repair: use of evoked potential monitoring in 118 patients. *J Vasc Surg* 2001; **34**: 1035–40.
23. Svensson LG, Khitin L, Nadolny EM, Kimmel WA. Systemic temperature and paralysis after thoracoabdominal and descending aortic operations. *Arch Surg* 2003; **138**: 175–80.