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Replacement of the descending aorta: Recent outcomes of open surgery performed with partial cardiopulmonary bypass

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Objective: Surgical replacement is our standard treatment for descending aortic aneurysm, despite the advent of thoracic endoprostheses. We retrospectively analyzed outcomes of descending aortic replacement performed with partial cardiopulmonary bypass.

Methods: Since 1994, a total of 113 patients in our institution (mean age 68 ± 12 years, n = 75 male) have undergone graft replacement of the descending aorta for nondissecting aneurysm. There were 16 emergency cases (14.2%). All operations were performed through left thoracotomy with partial cardiopulmonary bypass with segmental clamping. Since 1998, preoperative magnetic resonance angiography has been performed to detect the Adamkiewicz artery in elective cases. Motor evoked potentials are now measured intraoperatively.

Results: Early mortalities were 5.3% overall (6/113), 1.0% (1/97) in elective cases, and 31.3% (5/16) in emergency cases. Rates of spinal cord dysfunction were 2.7% overall (3/113), 1.0% (1/97) in elective cases, and 12.5% (2/16) in emergency cases. Stroke rates were 7.1% overall (8/113), 4.1% (4/97) in elective cases, and 25.0% (4/16) in emergency cases. Rates of respiratory failure were 9.7% overall (11/113), 9.2% (9/97) in elective cases, and 12.5% (2/16) in emergency cases. No patient underwent reoperation for the same lesion as a result of repair problems in the follow-up period. Kaplan–Meier overall survival estimates were 92.2% at 3 years, 90.6% at 5 years, and 70.2% at 10 years.

Conclusion: Although it is more invasive than stent graft repair, descending aorta replacement performed with partial cardiopulmonary bypass involves a risk comparable to that associated with thoracic endoprosthesis placement.

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Surgical treatment for a descending thoracic aneurysm (DTA) is changing drastically in response to the advent of endovascular treatment. Endoprostheses have been used for DTA, with generally favorable results.[1,2] Stent graft repair for thoracic aortic diseases is a therapeutic option even for high-risk patients who are not candidates for open surgery. Open surgical replacement, however, is still our current standard treatment for DTA. There are several operative strategies for DTA, such as the single-clamp technique,[3] distal perfusion with left heart bypass,[4] hypothermic circulatory arrest,[5] and partial cardiopulmonary bypass (PCPB).[6] We have usually used PCPB for DTA, with hypothermic circulatory arrest when there is no space for crossclamping. We retrospectively analyzed the outcomes for DTA repair performed with PCPB and compared them with those reported in the literature for endoprostheses.

Materials and Methods

Patients

From 1994 to 2004, a total of 113 patients (75 men, mean age 68 ± 12 years) underwent graft replacement of the descending aorta for nondissecting aneurysm. The cases that required open

Abbreviations and Acronyms
AKA = Adamkiewicz artery
DTA = descending thoracic aneurysm
MEP = motor evoked potential
PCPB = partial cardiopulmonary bypass

proximal anastomosis under circulatory arrest were excluded, and the patients in this study had sufficient space for crossclamping next to the left subclavian artery and celiac artery. There were 16 cases of emergency surgery (14.2%), all because of rupture of the aneurysm. Fifteen patients had undergone previous abdominal aortic replacement, 3 had undergone previous thoracoabdominal aortic replacement, and 11 had undergone previous thoracic aortic replacement. Since 1998, preoperative magnetic resonance angiography has been performed to detect the Adamkiewicz artery (AKA) in elective cases.[7] The AKA was preoperatively imaged by contrast magnetic resonance angiography with gadolinium dimeglumine (0.3 mmol/kg body weight). Early- and late-phase images were used to differentiate arteries from veins. Imaging volumes covered the levels between T6 and L3. The AKA and the anterior spinal artery were identified by at least two radiologists in 0.6-mm contiguous sections processed by multiplanar reconstruction. Our institution approved this retrospective study and did not require patient consent on the condition that patients not be identified.

Operative Techniques

The patients were anesthetized and intubated with a double-lumen endotracheal tube. The patients were then positioned in the right lateral decubitus position with the hips flexed 60°. An incision was made from the vertebral border of the scapula to the costal cartilage along the intercostal space. From the 4th to the 7th intercostal space, access to the left thorax was selected according to the location of the aneurysm. The left or right femoral artery and vein were dissected and looped with umbilical tape. A cannula was inserted in the femoral artery for perfusion inflow, and another cannula was inserted in the femoral vein for perfusion outflow. The tip of the venous cannula was placed at the opening of the inferior vena cava in the right atrium, with placement confirmed by transesophageal echocardiography. PCPB was initiated, and normal proximal aortic pressure was maintained; the flow rate was usually around 1.5 to 2.0 mL/(min · m^2). The pump circuit had an extracorporeal membrane oxygenator, including a heat exchanger. The bladder temperature was cooled to between 33°C and 34°C during PCPB.[8] The DTA was exposed and clamped after establishment of PCPB. The clamps were placed sequentially when the aneurysm involved a long segment. The aorta was opened longitudinally, and intercostal arteries were ligated or oversewn for hemostasis when they were considered to be unimportant. Intercostal arteries that had to be reattached or preserved were temporarily closed with a bulldog clamp or small balloon-tip catheters. The anastomosis was always performed with complete transection of the descending aorta. An appropriately sized Dacron polyester fabric graft was chosen, and the proximal anastomosis was performed first with running 3-0 or 4-0 polypropylene suture with a polytetrafluoroethylene felt strip. Intercostal arteries were reattached with a short, small-caliber graft. The distal anastomosis was then performed with running 3-0 or 4-0 polypropylene suture with a polytetrafluoroethylene felt strip. The flow of PCPB was reduced, and the aortic clamps were then gently released. The patient was weaned from PCPB once the bladder temperature reached 36.5°C.

We have been measuring motor evoked potentials (MEPs) during surgery since 1998 to detect spinal ischemia and have previously described the details.[9] With sufficient anesthesia maintained with low doses of fentanyl (0.02–4 mg/kg), propofol (4—6 mg/[kg · h]), and vecuronium (0.04 mg/[kg · h]), the motor cortex was activated by 600 V transcranial electrical stimulation. The action potentials conducted through the anterior horn motor neurons were recorded from the skin over the upper extremity muscles (as a control), the lower extremity muscles, and the thenar muscles. The signals of the MEPs are affected by femoral arterial cannulation; the probe was therefore always placed on the contralateral side from femoral cannulation. Monitoring of MEPs is also influenced by anesthesia, including neuromuscular blockade, only a low dose of vecuronium was therefore used during the operation. During crossclamping, MEP levels were determined every 2 to 5 minutes. A fall in MEP amplitude below 25% of the baseline was taken to indicate ischemia of the spinal cord. When critical reduction of MEP amplitude was observed, rapid revascularization of the spinal cord blood supply was performed. Additionally, the blood pressures of upper and lower body were increased with use of catecholamines, transfusion, and perfusion flow.

Definitions

Early mortality was defined as death during the hospital stay. Postoperative stroke was defined as newly developing neurologic deficit confirmed by computed tomography. Neurologic diagnoses were made by neurologists. Respiratory failure was defined as the need for intubation and ventilatory support longer than 72 hours.

Statistical Analysis

Values are the mean ± SD. Data were analyzed with Fisher exact tests for categorical variables.

Results

The early mortalities were 5.3% overall (6/113), 1.0% (1/97) in elective cases, and 31.3% (5/16) in emergency cases. The rates of spinal cord dysfunction were 2.7% overall (3/113), 1.0% (1/97) in elective cases, and 12.5% (2/16) in emergency cases. Spinal cord dysfunction occurred more frequently in patients older than 75 years and was not prevented by preoperative AKA detection (Table 1). The stroke rates were 7.1% overall (8/113), 4.1% (4/97) in elective cases, and 25.0% (4/16) in emergency cases. Stroke occurred most frequently in emergency cases, but it was not related to crossclamping adjacent to the aortic arch (Table 2). The rates of respiratory failure were 9.7% overall (11/113), 9.2% (9/97) in elective cases, and 12.5% (2/16) in emergency cases.

Thirty-two patients were older than 75 years, and 9 of these underwent emergency operations. The older patients' mortality was 6.3% (2/32), and the 2 patients who died had both undergone emergency operations. The rates of spinal

TABLE 1. Spinal cord dysfunction and variables

	Total	Spinal cord dysfunction	*P* value
All	113	3 (2.7%)	
Male	76	3 (3.9%)	.55
Age			
>70 y	56	3 (5.4%)	.12
>75 y	32	3 (9.4%)	.02
Partial cardiopulmonary bypass duration			
>60 min	83	3 (3.6%)	.99
>90 min	39	1 (2.5%)	.99
>120 min	15	1 (6.7%)	.36
Emergency operation	16	2 (12.5%)	.05
Preoperative Adamkiewicz artery detection	50	2 (4.0%)	.59

cord dysfunction in this age group were 9.3% overall (3/32), 4.3% (1/23) in elective cases, and 22.2% (2/9) in emergency cases. The stroke rates were 9.4% overall (3/32), 0% (0/23) in elective cases, and 33.3% (3/9) in emergency cases. The rates of respiratory failure were 12.5% overall (4/32), 13.0% (3/23) in elective cases, and 11.1% (1/9) in emergency cases.

Overall, the mean operative time was 291 ± 93 minutes, the mean PCPB time was 84.8 ± 32.1 minutes, the mean bleeding volume was 1187 ± 1432 mL, and the mean transfusion volume was 1335 ± 2642 mL, with 45.1% of the patients not requiring transfusion. In elective cases, the mean operative time was 280 ± 78 minutes, the mean PCPB time was 80.7 ± 27.7 minutes, the mean bleeding volume was 921 ± 845 mL, and the mean transfusion volume was 851 ± 1870 mL, with 51.5 % of the patients not requiring transfusion.

Magnetic resonance angiography was performed in 65 cases, and the AKA was detected in 50 patients (76.9%). Among these patients, 2 had paraplegia; the AKA had been detected in both. Three patients had paraplegia or paraparesis; 1 had undergone surgery without MEP monitoring, another showed MEP change, and the third patient showed no change in MEPs. MEPs were altered in 2 patients; 1 had paraplegia and the other had a postoperative stroke.

None of the patients underwent reoperation for the same lesion to repair problems in the follow-up period. Kaplan–Meier overall survival estimates were 92.2% at 3 years, 90.6% at 5 years, and 70.2% at 10 years (Figure 1).

Discussion

DTA repair is usually discussed in combination with thoracoabdominal aortic aneurysms. Reports focusing solely on surgical repair for DTA are relatively uncommon. Many DTAs will probably be repaired with endoprostheses, because a DTA has no visceral branches. The advent of endovascular treatment is believed to be a great innovation in treatment for aortic aneurysm. Endoprostheses have been used for abdominal aortic aneurysms, and some surgeons are now using them for DTA repair. Makaroun and colleagues[2] used the GORE TAG thoracic endoprosthesis in 139 patients with DTA. They reported that the procedure time was 150 minutes on average, blood loss was 506 mL on average, and that mortality, stroke, and spinal ischemia rates were 1.5%, 4% and 3%, respectively. Fattori and colleagues[10] used the Talent thoracic stent graft for DTA in 457 patients. They reported mortalities of 7.9% in acute cases and 4% in elective cases, a stroke rate of 3.7%, and a spinal ischemia rate of 1.7%.

The articles on endoprostheses refer to open repair of DTA, and they often point out that the mortality associated with open repair is greater than 10% and that the risk of spinal ischemia is 4% to 5%. On the other hand, the results of open repair are improving. Coselli and colleagues[11] reported a mortality of 4.4% and a paraplegia rate of 2.6% after open repair of DTA. Estrera and associates[12] reported a mortality of 8.8%

TABLE 2. Stroke and variables

	Total	Stroke	*P* value
All	113	8 (7.1%)	
Male	76	5 (6.6%)	.72
Age			
>70 y	56	4 (7.1%)	.99
>75 y	32	3 (9.4%)	.69
Partial cardiopulmonary bypass duration			
>60 min	83	8 (9.6%)	.20
>90 min	39	5 (12.8%)	.13
>120 min	15	3 (20.0%)	.08
Emergency operation	16	4 (25.0%)	.01
Crossclamp near arch	37	4 (10.8%)	.43

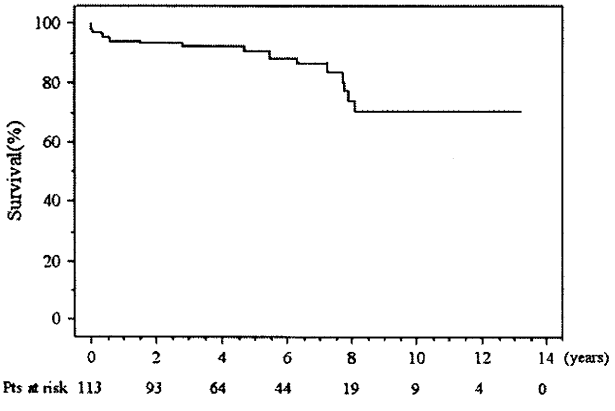

Figure 1. Kaplan–Meier cumulative actuarial survival curve.

and a paraplegia rate of 2.7% after open repair of DTA with cerebrospinal fluid drainage and distal perfusion. Even with hypothermic circulatory arrest, Patel and coworkers[13] reported a mortality of 6.0%, a stroke rate of 6.8%, and a spinal ischemia rate of 4.5%. Our results were comparable with or even better than those reported for open repair and for endoprostheses. Open repair of DTA has several merits relative to repair with an endoprosthesis, especially long-term durability. Moreover, there are no anatomic limitations such as interfere with the applicability of an endoprosthesis, including short or wide proximal or distal landing zones, severe neck angulations, and tortuous or stenotic access arteries.[14]

Stroke is a devastating complication after aortic surgery. The incidence and etiology of stroke related to DTA repair have not been frequently described. Attention is generally paid to spinal ischemia as a primary neurologic complication of DTA repair. Actually, DTA repair with PCPB involves a certain risk of stroke, as indicated in this study. Goldstein and colleagues[15] reported a stroke rate of 8.1 % in DTA repair and also noted that stroke was a significant predictor of postoperative death. Patel and coworkers[13] reported a stroke rate of 6.8% in DTA repair with hypothermic circulatory arrest. The retrograde flow of PCPB from femoral cannulation when normal proximal aortic pressure is not maintained could be a reason for the stroke risk. Moreover, crossclamping adjacent to the aortic arch has also been mentioned as a cause of stroke.[16] In our study, however, some patients without crossclamping adjacent to the aortic arch still had stroke occur under normal proximal aortic pressure. Crossclamping adjacent to the aortic arch was not a statistically significant risk factor of stroke in our study.

The preoperative detection of AKA by magnetic resonance angiography is, we believe, useful in preventing spinal cord injury during DTA repair. The utility of the detection of AKA has already been described elsewhere, and the effects were reflected in the lower rate of spinal ischemia. Although the spinal blood supply is not completely understood, we consider that reimplantation or preservation of the intercostal arteries, which connect the AKA, contributes to improved results. In this study, however, 2 patients showed spinal ischemia despite detection of the AKA. This implies that preservation of the AKA per se is not enough to prevent spinal ischemia. MEPs have been reported to be a rapid indicator of spinal cord injury during thoracoabdominal aortic repair.[17] We also believe that MEP monitoring contributes to prevention of spinal cord injury, even during DTA repair, but such an effect was not clear in this study.

Advanced age is supposed to be among the risks for DTA repair. Huynh and colleagues[18] reported a stroke rate of 9% in their series of descending and thoracoabdominal aortic replacements in patients of advanced age. In this study, the frequencies of stroke in patients older than 70 years and in those older than 75 years old were comparable. No deaths and no postoperative strokes were seen among elective cases. The rate of respiratory failure, however, was high even in elective cases, as expected.

In conclusion, outcomes of traditional open DTA repair are improving. The long-term result of this technique is in clear contrast to that of endoprosthesis. Even in patients older than 75 years, open DTA repair can be performed with acceptable risk. Although open DTA repair is by definition more invasive and should be further improved, the risks involved in replacement of the descending aorta under PCPB were comparable to those associated with thoracic endoprosthesis placement at this time.

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EDITORIAL

Is hypothermia a reliable adjunct for spinal cord protection in descending and thoracoabdominal aortic repair with regional or systemic cooling?

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Since DeBakey first carried out thoracoabdominal aortic repair with an aortic homograft using a temporary arterial shunt in 1956,[1] multidisciplinary approaches have been directed toward minimizing spinal cord ischemic injury during surgery of the descending thoracic and thoracoabdominal aorta. The principal modality has been distal aortic perfusion during the aortic cross-clamping, which was passive in the past with a temporary shunt tube or bypass and that currently has been active with a left heart bypass or a cardiopulmonary bypass (CPB). Other approaches include the following: intraoperative monitoring of spinal cord ischemia with somatosensory evoked potentials and transcranial motor evoked potentials; reattaching the responsible intercostal artery; increasing collateral flow by controlling the back-bleeding of the patent intercostal arteries with high cardiac output and arterial pressures with cerebrospinal fluid (CSF) drainage; increasing ischemic tolerance with pharmacological adjuncts including naloxane, steroid, barbiturates, and with hypothermia; reducing excitotocixity from neuronal ischemia with hypothermia, naloxane, and steroid; and attenuating reperfusion injury with steroid, hypothermia, and free radical scavengers. Particularly, in Japan, with recent great advances in diagnostic imaging modalities, preoperative demonstration of the arteria radicularis magna (Adamkiewicz artery) by magnetic resonance imaging (MRI) or computed tomography (CT) scans has been highlighted as a reliable guide for reattachment or preservation of the responsible intercostal arteries including the collaterals.

Since the 1950s hypothermia has been proven effective for protecting organs, including the central nervous system, in the cardiovascular surgical field.[2–5] When focusing on milestones of aortic surgery, systemic profound hypothermia at 12°–15°C was applied to aortic arch surgery for brain protection in 1975.[6] Thereafter, during the 1980s, based on systemic hypothermia, more sophisticated antegrade or retrograde cerebral perfusion has been established for more definitive cerebral safety.[7,8] For aortic surgery through a left thoracotomy, Kouchoukos et al. employed systemic deep hypothermia to protect the spinal cord as well as the brain during descending thoracic and thoracoabdominal aortic repair in 1995.[9] In most, an open aortic anastomotic technique was used with circulatory arrest, avoiding aortic cross-clamping. On the other hand, mild hypothermia around 32°C with distal perfusion of a partial CPB has been our routine, as in most of Japan.[10] During the surgery, transcranial motor evoked potentials are used to monitor spinal cord ischemia continuously. A couple of the intercostal arteries responsible for the spinal cord ischemia are aggressively reattached according to the preoperative demonstration of the Adamkiewicz artery by MRI and CT, while controlling back-bleeding from the other intercostal arteries without delay. In selected "high-risk" patients having an extent I and II thoracoabdominal aortic aneurysm, however, our tactics have lately been shifted to deep hypothermic surgery (around 20°C) with total CPB for more rigorous spinal safety. Even in this setting, motor evoked potentials can be recorded above

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25°C with adjusted anesthetics. Early rewarming after the proximal anastomosis has been another refinement for minimizing some of the adverse effects of systemic hypothermia by reducing the time of ventricular fibrillation and CPB.

As described above, systemic hypothermia has some drawbacks, such as coagulopathy, pulmonary dysfunction, cardiac arrhythmia leading to cardiac arrest or ventricular fibrillation, systemic edema due to fluid shift, in part due to prolonged CPB. To eliminate these shortcomings, the usefulness of regional cooling of the spinal cord was addressed in surgery for the descending thoracic and thoracoabdominal aorta. In 1975, Hansebout et al. first highlighted the effect of local hypothermia and steroid on recovery from experimental spinal cord compression injury.[11] Between 1992 and 1994, several impressive reports were published on regional cooling of the spinal cord in animal models.[12–14] In our country, Tabayashi and colleagues also addressed the impact of epidural cooling on the spinal cord protection in an animal experiment.[15] The rationale for this technique is to increase the ischemic tolerance of the spinal cord during the critical periods of aortic cross-clamping associated with the reduction of the spinal cord perfusion pressure. Around the same time, Davison, Cambria, and colleagues (Massachusetts General Hospital, or MGH, group) first adopted this technique clinically in eight patients undergoing descending thoracic and thoracoabdominal aortic resection.[16] With their method, at least 30 min (average 50 min) before the aortic cross-clamping, 4°C saline solution was infused into the epidural catheter until the CSF temperature decreased to 25°C. Another subarachnoidal catheter was also used to measure CSF pressure and, if necessary, to drain the CSF. They have continued ongoing efforts for the development of this technique. In the latest report of 240 patients requiring extent I to III thoracoabdominal aneurysmal repairs, the incidence of spinal cord injury of any severity was 12.1%, including the results of emergency cases.[17]

In this volume, Tabayashi and colleagues (Tohoku University group) reported their 10-year experience of epidural perfusion cooling with outstanding outcomes—spinal cord injury in 3.9% and hospital deaths in 5.9%—for 102 patients undergoing descending thoracic and thoracoabdominal aortic repair. This regional cooling has been applied for the spinal cord only by them in Japan,[18,19] as the MGH group has advocated over the last decades.[20,21] Between them, the techniques of regional epidural cooling are similar in conjunction with the CSF drainage and measurement of CSF temperature. The outcome of the Tohoku University group was more favorable than that of the MGH group. One reason might be employment of distal perfusion with CPB or left heart bypass with mild systemic hypothermia at 31°–32°C by the Tohoku University group, which differed from the fundamental "clamp-and-saw" technique with normothermia without any assisted circulation by the MGH group. Meticulous surgical techniques of antegrade or retrograde segmental sequential repair for reduced spinal cord ischemia by the Tohoku group might be another factor for more favorable spinal safety, in conjunction with aggressive reattachment of the responsible intercostal arteries based on preoperative CT or MRI findings. Although the statistical analysis did not reveal any relevant factors for spinal cord injury, they noted that the main cause of spinal cord injury was likely due to unstable hemodynamics caused by massive bleeding. However, the causes of spinal cord injury are supposed to be multifactorial. Even with efficient epidural cooling, the spinal tolerance should be limited owing to relatively lower perfusion pressure of the spinal cord. Rushed reattachment of the responsible intercostal artery has technical difficulties. Given real-time monitoring of spinal cord ischemia, such as with transcranial motor evoked potentials, it would have been feasible to clarify the cause of the paraplegia.[10]

Some drawbacks of this novel epidural cooling have been recognized. One is a sharp rise in CSF pressure coincident with epidural cooling in some instances, which might be a risk factor for spinal cord ischemia.[22,23] The MHG group also warned about this increase in CSF pressure. The two groups tolerated this "modest and transient" increase because it can be controlled well with further CSF drainage or epidural cooling fluid drainage and because the risk of spinal cord ischemia is thought to be outweighed by the potential neuroprotective effect of epidural cooling.[20,21] From this point of view, it is important to maintain an arbitrary 30–40 mmHg gradient between the mean arterial pressure and the CSF pressure before aortic cross-clamping, with some caution. Otherwise, given a newly designed cooling catheter system, for example, with a closed countercurrent lumen, it is theoretically feasible to avoid this CSF pressure elevation.[23] On a related issue, one patient suffered an unexplained cerebrovascular accident associated with the highest CSF pressure. Interestingly, the MGH group experienced a similar case and recognized the difficulty of excluding the possibility of the increased CSF pressure contributing to the highly unusual but devastating lower cervical or upper thoracic spinal cord injury. Another technical question is about the temperature measurement of the spinal cord. The both groups measured the temperature of CSF—not that of the spinal cord—based on the correlation of the temperature between the spinal cord and the CSF. The practical con-

sideration of the degree of hypothermia required for spinal safety has been answered though a variety of animal experiments, which suggested 25°–27°C for uniform protection.[12–14] However, homogeneous cooling of the spinal cord might be uncertain. In this context, the local cooling times are all different and prolonged to an average of 50 min as reported by the MGH group, which is a defect of this method.[17]

Hypothermia is still the principle for organ protection, particularly for cerebral and spinal safety. Regional perfusion cooling into the epidural space is a unique and reliable method for spinal protection, minimizing the adverse effects of systemic hypothermia. Tabayashi and colleagues are to be congratulated for their outstanding outcomes and ongoing efforts in the development of epidural cooling. However, the unsolved complexity of this novel technique for spinal safety, including some drawbacks, seems to keep it from being in routine use.[23]

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Multidisciplinary Approach to Prevent Spinal Cord Ischemia After Thoracic Endovascular Aneurysm Repair for Distal Descending Aorta

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Background. This article discusses the multidisciplinary approach to prevent spinal cord ischemia (SCI) with reference to the incidence of SCI after thoracic endovascular aneurysm repair (TEVAR) associated with closure of the intercostal-lumbar artery that supplies the Adamkiewicz artery (ICA-AKA).

Methods. We reviewed 60 patients [49 men, 57 to 89 years old] who underwent TEVAR (TAG [W. L. Gore & Associates, Flagstaff, AZ] 42; the Matsui-Kitamura (Kanazawa, Japan) 10; Talent [Medtronic Inc, Santa Rosa, CA] 5; TAG and Talent 3) for part of or the entire distal descending aorta between T7 and L2. These patients had frequently undergone aortic surgeries: ascending aorta (4), aortic arch (25), descending aorta (4), thoracoabdominal aorta (3), and abdominal aorta (19). The multidisciplinary approach consists of identification of the ICA-AKA by magnetic resonance angiography or computed tomographic angiography to avoid unnecessary coverage of the ICA-AKA, in combination with monitoring of motor evoked potentials, augmentation of mean arterial pressure (> 80 mm Hg), and cerebrospinal fluid drainage.

Results. Spinal cord ischemia occurred in 4 patients and patent ICA-AKAs were covered in 3 of them. The overall incidence of SCI was 6.7% and 9.4% in the group of 32 patients whose patent ICA-AKAs were covered by TEVAR. After treatment for SCI, 3 patients regained full ambulatory ability. Significant risk factors were identified as the artificial graft at the proximal landing zone, the number of covered zones (>8), the length of aortic coverage (>250 or >300 mm), and the length of the uncovered distal aorta (<60 mm).

Conclusions. A multidisciplinary approach is essential to prevent SCI after TEVAR for the distal descending aorta. This approach includes the preservation of patent ICA-AKAs after their identification, early diagnosis of SCI during TEVAR by monitoring motor evoked potentials, and prophylaxis and treatment of SCI by increasing mean arterial pressure to at least 80 mm Hg and performing cerebrospinal fluid drainage.

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Thoracotomy, aortic clamp, and distal perfusion under extracorporeal circulation become unnecessary when thoracic endovascular aortic repair (TEVAR) is used. As perfusion of the spinal cord is maintained during the procedure and the hemodynamics remain stable, the spinal cord is not at risk of reperfusion injury. Thoracic endovascular aortic repair has thus been considered as less invasive for the spine than open repair [1].

However, TEVAR requires covering of the intercostal-lumbar arteries (ICAs) to extend the landing zones and the optimal method for revascularization of ICAs after TEVAR has not been established. Moreover, the incidence of spinal cord ischemia (SCI) is not insignificant after TEVAR for the distal descending aorta, from which the intercostal-lumbar artery that supplies the Adamkiewicz artery (ICA-AKA) usually branches off [2].

In the case of open repair, various postoperative measures for spinal protection have been reported [3, 4]. In this retrospective study we discuss our multidisciplinary approach to prevent SCI after TEVAR with reference to the incidence of SCI associated with closure of the ICA-AKA and the significant risk factors for SCI after TEVAR.

Patients and Methods

Of the 112 patients operated on during the preceding 32 months at our department, we reviewed the clinical records of 60 who underwent TEVAR for part of or for the entire distal descending aorta after the ICA-AKA was identified by magnetic resonance angiography (MRA) or computed tomographic angiography (CTA). Our institution approved this retrospective study and waived patient consent requirements if patients could not be identified. The distal descending aorta was defined as the segment between T7 and L2 [5]. Patients who developed an aortic dissection or

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pseudoaneurysm after aortic replacement were not included, but two patients who developed a thin and thrombosed aortic dissection on a degenerative aneurysm were included. Three patients whose spinal cord function could not be evaluated due to, respectively, intraoperative death, cervical spondylotic myelopathy, and cerebral infarction were also excluded.

There were 11 women and 49 men (57 to 89 [77] years old), 53 of whom were 70-years old or older. According to the American Society of Anesthesiologists physical status classification, 17 patients were class 2, 28 class 3, 14 class 4, and 1 class 5. They were also characterized by a history of frequent aortic surgeries: ascending aorta (4 patients), aortic arch (25; TEVAR 1), descending aorta (4; TEVAR 1), thoracoabdominal aorta (3), abdominal aortic aneurysm (AAA) replacement (18), and EVAR (1).

Identification of the ICA-AKA

When the stent graft was expected to cover the distal descending aorta, the ICA-AKA was first identified by MRA or CTA. Contrast MRA and CTA were performed as previously reported in detail [2, 6]. In principle, we preferred to use MRA (52 patients) in order to avoid influence of the spine and to accurately differentiate the AKA from the anterior radicular vein [7]. However, as MRA was not always possible for emergency diagnosis of ICA-AKA, CTA was used instead for 8 patients.

The ICA-AKA was patent in 48 patients, occluded at its origin in 8, and double "patent" ICA-AKAs were identified in 4 patients. In total, 60 ICA-AKAs were confirmed (Table 1). In 4 patients, the ICA-AKA could not be confirmed by MRA and was considered to be "absent."

The MRA also revealed that the vertebral level of the orifice of the ICA-AKA did not always coincide with the vertebral number of the ICA-AKA. When they differed, the orifice of the ICA-AKA was usually located at the vertebral level below; for example, the Th10 ICA-AKA would branch at the Th11 vertebral level of the descending aorta. Closure of the ICA-AKA was defined by the placement of the stent graft at the origin of the ICA-AKA, and in patients with double ICA-AKAs it was defined by closure of one or both ICA-AKAs.

TEVAR

When planning the TEVAR, a sufficiently long landing zone was considered to be the first priority and preservation of the ICA-AKA patency the second. Coverage of the patent ICA-AKA was unavoidable in 32 patients.

Table 1. Adamkiewicz Artery Distribution

	Right	Left	Total
Th7		1	1 (1.6%)
Th8	2	6	8 (13.3%)
Th9	1	19	20 (33.3%)
Th10	2	13	15 (25.0%)
Th11	1	5	6 (10.0%)
Th12	3	3	6 (10.0%)
L1	1	2	3 (5.0%)
L2		1	1 (1.6%)
Total	10 (16.7%)	50 (83.3%)	

The thoracic stent grafts used were the Gore TAG (W. L. Gore & Associates, Flagstaff, AZ) for 42 patients, the Matsui-Kitamura stent graft (Kanazawa, Japan) [8] for 10 patients, the Talent (Medtronic Inc, Santa Rosa, CA) for 5, and both Gore TAG and Talent for 3 patients.

To create suitable landing zones, the following aortic branches were closed: left carotid artery (2 patients), left subclavian artery (4), celiac axis (5), and mesenteric and renal arteries (2). All left carotid, left subclavian (LSCA), mesenteric, and renal arteries were reconstructed with bypass surgery. Except for one patient who underwent bypass surgery after division of the celiac axis (CA), the CA was closed by means of coil embolization using the balloon occlusion test of the celiac artery [9]. Bypass surgery to prepare for the closure of major aortic branch(es) was usually performed one week before TEVAR and supraaortic bypass was performed concomitant with TEVAR for 2 patients.

Intervention for AAA was concurrently performed for 5 patients: AAA replacement for 3 and endovascular aneurysm repair for 2. In 17 patients whose external iliac and (or) femoral arteries were not large enough, an 8 or 10 mm conduit was connected to the common or external iliac artery.

Thoracic endovascular aortic repair was carried out under general anesthesia. The duration of the operation varied (51 to 406 [130] minutes) due to the need for concomitant performance of the procedures mentioned above. Twenty patients required blood transfusion. An artificial graft was used for the landing zone for endografting in 24 patients in view of their history of aortic surgeries. An artificial graft was used at the proximal landing zone for 20 patients after total arch replacement (TAR) with or without elephant trunk installation and for 3 patients after TEVAR for aortic arch or proximal descending aorta. For 11 of the patients subjected to TAR, TEVAR was performed as the second-stage surgery for extensive aneurysm in 7 patients or multiple aneurysms in 4. As for the distal landing zone, 5 patients underwent thoracoabdominal replacement and 2 AAA replacements in conjunction with debranching bypass for the visceral arteries. Artificial grafts were used for both landing zones in 4 patients.

Monitoring of Motor Evoked Potentials and Cerebrospinal Fluid Drainage

Transcranial motor evoked potentials of the anterior tibialis and thenar muscles were monitored in all patients every five minutes after stent grafting under general anesthesia [10, 11].

A cerebrospinal fluid drainage (CSFD) tube [12] was positioned in 41 patients prior to TEVAR. The CSFD tube could not be inserted due to exigency in 2 patients, to bleeding tendency in 5, and to preservation of ICA-AKA patency in 12. In 1 patient, who developed paraplegia the day after TEVAR due to rupture, a CSFD tube was inserted immediately after the onset of paraplegia.

Table 2. Risk Factors for Spinal Cord Ischemia (χ^2 Test)

Variables	SCI (−) (n = 56)	SCI (+) (n = 4)	*p* Value
75 years old or older	22 (39.3%)	3 (75.0%)	0.5734
Male gender	44 (78.6%)	2 (50.0%)	0.1337
History of total arch replacement	22 (39.3%)	3 (75.0%)	0.1611
History of AAA surgery	24 (42.9%)	0 (0.0%)	0.0747
Patent left internal iliac artery[a]	49 (87.5%)	4 (100%)	0.3103
Operation time ≥ 240 minutes	6 (10.7%)	2 (50.0%)	0.6360
Blood transfusion	16 (28.6%)	4 (100%)	0.0022
Emergency	2 (3.6%)	1 (25.0%)	0.1506
Concomitant surgery[b]	25 (44.6%)	3 (75.0%)	0.2328
Artificial graft at proximal landing zone	24 (33.9%)	4 (100%)	0.0043
Artificial graft at distal landing zone	5 (8.9%)	0 (0.0%)	0.3956
Number of covered zones ≥ 8	31 (55.4%)	4 (100%)	0.0336
Aortic coverage ≥ 250 mm	10 (17.6%)	4 (100%)	0.0004
Aortic coverage ≥ 300 mm	1 (1.8%)	3 (75.0%)	0.0001
Distal uncovered aorta ≤ 60 mm	28 (50.0%)	4 (100.0%)	0.0234
Coverage of ICA-AKA	29 (51.8%)	3 (75.0%)	0.3562
Reexploration for bleeding	1 (1.8%)	2 (50.0%)	0.0041
Prophylactic CSFD	9 (16.1%)	0 (0.0%)	0.2453

[a] Left subclavian and right internal iliac arteries were patent in all patients. [b] Concomitant surgery includes supraaortic bypass, AAA replacement, and iliac conduit.

AAA = abdominal aortic aneurysm; CSFD = cerebrospinal fluid drainage; ICA-AKA = the intercostal-lumbar artery that supplies the Adamkiewicz artery; SCI = spinal cord ischemia.

Treatment and Prophylaxis of SCI

Immediately after stent grafting, the mean arterial pressure (MAP) was raised to above 80 mm Hg with the aggressive use of catecholamines. This increase in MAP was maintained for two days.

When the amplitude of motor evoked potentials declined under general anesthesia, or when symptoms and signs of SCI were noted during the postoperative period, the MAP was raised to above 90 mm Hg and CSFD was started at 12 cm H_2O. The drainage gradient was adjusted between 10 and 15 cm H_2O to control the drainage volume at less than 20 mL per hour and 300 mL per day.

After the onset of SCI, methylprednisolone (30 mg/kg bolus and 5.4 mg/kg/hour for 23 hours followed by 2.7 mg/kg/hour for 2 days) and naloxone (1,200 μg/day) were infused [13]. This intensive spinal care, together with the augmentation of MAP, CSFD, methylprednisolone, and naloxone was discontinued 24 hours after full recovery or continued for 72 hours if the symptoms did not resolve.

During the early stages, CSFD was not indicated as a prophylactic measure. However, after the clinical data of this study had been accumulated, the indication of CSFD was modified. In the 9 most recent patients whose ICA-AKA had been patent and covered by TEVAR, prophylactic CSFD was started when the patient was transferred to the intensive care unit and continued for more than 12 hours.

Measurement of the Aortic Length

The length of the proximal uncovered aorta (from the LSCA to the stent graft), of aortic coverage by the stent graft, and of the distal uncovered aorta (from the stent graft to CA) was measured at the centerline of the aorta on CTA in 59 patients [2]. After TAR with a multibranch graft, the length of aortic coverage was measured from the distal anastomosis, which coincided with the origin of the elephant trunk. After replacement of the descending aorta or the thoracoabdominal aorta, the position of LSCA and (or) CA served as the point of reference for the measurement.

Statistical Analysis

Values show the mean ± SD. Data were analyzed with the χ^2 test for categoric variables and continuous variables with analysis of variance. The level of statistical significance was set at a *p* value less than 0.05.

Results

Spinal cord ischemia occurred in 4 patients and patent ICA-AKAs were covered in 3 of them. The overall incidence of SCI was 6.7% and 9.4% in the group of 32 patients whose patent ICA-AKAs were covered by using TEVAR, 2 of whom needed reoperation for hemostasis of a retroperitoneal hematoma and bleeding at the puncture site. In another patient, whose patent ICA-AKA was left uncovered, TEVAR was performed while the patient was in shock due to rupture, and the anastomosis side of the iliac conduit became occluded and it had to be reanastomosed 6 hours after TEVAR. One patient developed SCI as evidenced by a drop in motor evoked potential amplitude during TEVAR and the symptom was confirmed when the patient awoke from general anesthesia. The incidence of SCI, which was confirmed by a drop in motor evoked potential amplitude was 1.7% for all patients. Three other patients developed

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SCI 10 hours (1 patient) and 24 hours (2 patients) after TEVAR [14].

After treatment for SCI, 3 patients regained full ambulatory ability. The incidence of permanent SCI was 1.7% after TEVAR for the distal descending aorta and 3.1% for patients whose patent ICA-AKAs were covered.

There were 2 hospital mortalities. One was a patient who died of methicillin-resistant *Staphylococcus aureus* mediastinitis 4 months after undergoing TEVAR subsequent to TAR, the other a patient who had undergone TAR and thoracoabdominal aortic grafting but died 2 months after TEVAR due to infective endocarditis.

Other intraoperative complications and postoperative comorbidities, one per patient, were iliac artery injury, femoral artery thrombosis, and tracheostomy for pneumonia. Spinal bleeding occurred in a patient who started walking before removal of the inserted CSFD tube.

During the follow-up period, 5 patients underwent a secondary intervention: repeated TEVAR for type I endoleak in 2 patients, for type III endoleak and for endotension in 1 patient each, and the ligation of intercostal arteries for type II endoleak in 1 patient.

The common risk factors for SCI were analyzed with the χ^2 test (Table 2). Four patients with SCI had no history of AAA surgery, but even with a history of frequent TAR and (or) AAA surgery, the LSCA and right internal iliac artery were patent and the patency of the left internal iliac was not a significant risk factor for SCI. Due to the small number of patients, parameters showing an obvious difference between the patients with and without SCI, such as long operation time (>4 hours), emergency TEVAR, concomitant surgeries including supraaortic bypass, and utilization of an iliac conduit were not significant risk factors for SCI in this study. On the other hand, artificial graft at the proximal landing zone, number of covered zones (>8), length of aortic coverage (>250 or >300 mm) and length of the uncovered distal aorta (<60 mm) were significant risk factors. Of the 4 patients who developed SCI, the ICA-AKA was covered in 3 patients and preserved in 1 who underwent emergency TEVAR while in shock after rupture of the aneurysm.

Comment

The risk for SCI is considered to be lower after TEVAR than with open aortic repair [1], but in case of the former, additional ICAs must be sacrificed for the landing zones and revascularization of the ICAs is impossible after TEVAR. We have been using a multidisciplinary approach to prevent SCI after TEVAR that consists of preoperative identification of the ICA-AKA and planning to avoid its coverage, early diagnosis of SCI during TEVAR through motor evoked potential monitoring, and prophylaxis and treatment of SCI by increasing MAP and performing CSFD.

In this retrospective study, we reviewed the clinical records of patients who underwent TEVAR for part of or for the entire distal descending aorta. Coverage of eight or more spinal zones, longer (>250 mm or >300 mm) aortic coverage, and the shorter (<60 mm) length of uncovered distal aorta were identified as significant risk factors for SCI. All these factors involve the sacrifice of a large number of ICAs [15–18]. Another significant risk for SCI was the artificial graft at the proximal landing zone; that is, the elephant trunk with TAR and the proximal descending graft or stent graft. The artificial graft at the proximal landing zone may be in relation with other factors because it also results in the sacrifice of a large number of ICAs. Of these factors leading to the closure of ICAs, aortic coverage with a length of greater than 250 mm was the most sensitive, aortic coverage of greater than 300 mm showed the lowest p value, and the number of covered zones could be counted most easily in a clinical situation. However, the most appropriate parameter to show the number of sacrificed ICAs remains to be determined. Nevertheless, the risk involved in long coverage of the descending aorta must be emphasized.

The LSCA and internal iliac arteries are usually considered to constitute possible collateral sources of spinal blood supply. In our study, these arteries proved not to be important risk factors for SCI because of the high percentage of patency, 100% of LSCA and right internal iliac artery, and 88% of the left internal iliac. Lumbar arteries are the other collateral source of spinal blood supply. Open and endoluminal AAA surgeries, which involve the sacrifice of several pairs of lumbar arteries, have thus been described as significant risks for SCI after TEVAR [1, 19]. However, a history of or concomitant performance of AAA surgery including three EVARs did not constitute a risk factor for SCI in our study because none of the patients who experienced SCI had a history of AAA surgery. Aguiar Lucas and colleagues [20] reported that multilevel aortic disease was not associated with an increased incidence of SCI, possibly due to the avoidance of thoracic aortic cross-clamping resulting in no reperfusion injury and maintenance of adequate spinal perfusion during the perioperative period [21–24].

After open repair of the thoracoabdominal aorta, perioperative hemodynamic stability is essential for the prevention of SCI. In particular, the duration of hypotension after termination of the partial cardiopulmonary bypass has proved to be an independent risk factor for paraplegia [25]. Bleeding was recognized as a risk factor for SCI in our study, so to achieve stable perioperative hemodynamics after TEVAR, bleeding and subsequent hypotension should be avoided. Two of our patients showed a tendency to bleed during TEVAR in the form of difficulties with hemostasis at the anastomosis of the conduit and bleeding from the puncture site. In these patients, extensive TEVAR from the elephant trunk to the subtotal descending aorta was indicated. Moreover, consumption coagulopathy due to massive coagulation inside an extended aneurysm may be indirectly related to SCI after TEVAR.

After closure of several pairs of ICAs, collateral flow was supplemented by increasing MAP. We initially maintained MAP at 80 mm Hg and further increased it to 90 mm Hg after the onset of SCI. This, together with concomitant CSFD and pharmacologic intervention, resolved SCI in 3 of 4 patients. Our current strategy is to maintain MAP above 90 mm Hg with the aggressive use of catecholamines for 48 hours to prevent SCI, including delayed-onset SCI [20, 26].

To break the vicious cycle of spinal ischemia and edema, perioperative CSFD has been confirmed to reduce paraplegia after open repair of extent I and II thoracoabdominal aneurysms [27, 28]. We could successfully perform CSFD after TEVAR, except for one patient who started walking before the CSFD tube had been removed and developed spinal bleeding.

In the early stages, a CSFD tube was positioned in all patients who underwent TEVAR for the distal descending aorta and CSFD was started only after the onset of SCI. However, due to the low sensitivity for SCI (9.7%) among the patients whose patent ICA-AKA was covered and high specificity (96.6%) among those whose patent ICA-AKA was left uncovered, no CSFD tube was inserted when the ICA-AKA remained patent. On the other hand, when coverage of the patent ICA-AKA is required, prophylactic CSFD was performed after TEVAR in the most recent nine cases. However, since the usefulness of prophylactic CSFD has not been demonstrated, its indication remains to be evaluated.

Coverage of the ICA-AKA was not a significant risk factor for SCI. This may be erroneously interpreted as meaning that identification of ICA-AKA is unnecessary provided MAP is increased to at least 80 mm Hg and prophylactic CSFD is executed. However, in our patients the aim of every preoperative and intraoperative attempt to plan and perform TEVAR for distal descending aorta was to avoid coverage of the ICA-AKA, although the effect of unnecessary or unexpected closure of ICA-AKA remains unknown.

In conclusion, a multidisciplinary approach is essential to prevent SCI after TEVAR for the distal descending aorta. This approach includes the preservation of patent ICA-AKAs after their identification, early diagnosis of SCI during TEVAR by monitoring motor evoked potentials, and prophylaxis and treatment of SCI by increasing MAP to at least 80 mm Hg and performing CSFD.

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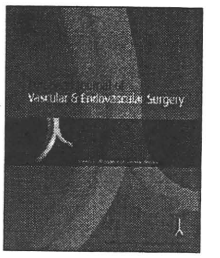

Spinal Cord Injury is Not Negligible after TEVAR for Lower Descending Aorta☆

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Thoracic aortic aneurysm;
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Abstract *Objectives:* To clarify the incidence of spinal cord injury (SCI) after thoracic endovascular aneurysm repair (TEVAR), we investigate the intercostal/lumbar arteries that supply the Adamkiewicz artery (ICA-AKA).
Patients: Among 81 patients subjected to TEVAR, we retrospectively reviewed the clinical records of 50 patients (range: 57–86 (median age: 77) years, 41 males) who underwent TEVAR for part of or the whole distal descending aorta (T7 to L2) after identification of ICA-AKA by magnetic resonance angiography (MRA) or computed tomography angiography (CTA).
Results: The 50 patients were classified into group A: 17 patients whose patent ICA-AKA was not covered, group B: 24 patients whose ICA-AKA was covered and group C: nine patients in whom no patent ICA-AKA was identified. Only three patients in group B suffered paraplegia and of them two recovered full ambulation. The estimated incidence of permanent and transient paraplegia was 3.7% in all TEVAR patients, 6.0% when part of or the entire distal aorta was covered and 12.5% when the patent ICA-AKA was covered. The length of aortic coverage in patients with paraplegia was >300 mm.
Conclusions: Paraplegia after TEVAR occurred in one of eight patients in whom the stent graft covered ICA-AKA. Long coverage of the aorta including the ICA-AKA was critical. To prevent this serious complication, identification of the ICA-AKA is crucial.

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The incidence of spinal cord injury (SCI) after thoracic endovascular aneurysm repair (TEVAR) has been reported to vary according to the demographics of the patients.[1–20] Whether the integrity of the Adamkiewicz artery (AKA) is essential for spinal cord function is still to be investigated.[21] However, after reattachment of the intercostal/lumbar arteries, which supply AKA (ICA-AKA), or of the adjacent intercostal/lumbar arteries during thoracoabdominal aortic replacement, motor-evoked potentials (MEPs) recover.[22] TEVAR has been reported to reduce SCI.[12] In principle, the longer the length of the aorta including both landing zones that is covered by TEVAR, the larger the number of ICAs that will be sacrificed and whose revascularisation will be impossible.[23]

To clarify the incidence and cause of SCI after TEVAR, we have investigated the patency of ICA-AKA in relation to other factors which may cause SCI.

Materials/Methods

Patient demographics

In the past 27 months, of 81 patients, we performed TEVAR with Gore TAG (W. L. Gore & Associates, Flagstaff, AZ, USA) in 47 patients, Talent thoracic stent graft (Medtronic, Inc., Santa Rosa, CA, USA) in five, both TAG and Talent in one and Matsui-Kitamura (MK) stent graft in 28 patients.[24] In this study, we included 50 patients who underwent TEVAR for part of or the whole distal descending aorta after ICA-AKA was identified by magnetic resonance angiography (MRA) or computed tomography angiography (CTA). The distal descending aorta was defined as the segment between T7 and L2.[25] Fifteen patients who underwent TEVAR above T6 and 16 patients who had not undergone MRA or CTA to identify ICA-AKA were not included in this investigation.

In general, the patients were senescent, debilitated and presented co-morbidities. (Table 1) Thirty-seven patients were ≥75 years old and the median age was 77. Thirty-seven patients were in ASA class 3 or 4, and 32 patients had a history of aortic surgery (48 surgeries in total).

Of the 18 patients who had undergone AAA repair, TEVAR had been indicated more than 1 year later in 11 patients, scheduled within 3 months in five and performed simultaneously in two. Emergency TEVAR was performed in three for haemoptysis, acute aneurysm dissection and persistent back pain. They were haemodynamically stable and could undergo CTA for ICA-AKA.

In all patients, another CTA was carried out to precisely measure the aneurysm and access. CTA also revealed the patency of the left subclavian (LSCA) and bilateral internal iliac arteries (IIA). Occlusion of left IIA (LIIA) was confirmed in three patients but LSCA and right IIA (RIIA) were patent in all the patients regardless of whether total arch replacement (TAR) or AAA repair was performed.

Identification of ICA-AKA

ICA-AKA was identified by MRA in 39 and by CTA in 11 patients.

The details of contrast MRA were previously reported by Yamada et al.[26] For the CTA, an Aquilion 16 multi-detector row CT scanner (Toshiba, Tokyo, Japan) was used. To detect AKA, the reconstruction field of view was set to the area around the aorta and spine. The images were processed in a workstation (Ziostation; Amin, Tokyo, Japan). Volume-rendered images of the entire aorta were routinely generated. Multiplanar reformation (MPR) images, including oblique coronal images with craniocaudal angulations and curved planar reformation images, were reconstructed to investigate the side and level of the origin of AKA.

Diagnostic criteria for the anterior spinal artery and AKA were as previously reported.[26] We preferred MRA as CTA is disadvantageous due to the influence of the spine and lack of accurate differentiation of the AKA from the anterior radicular vein.[22] However, the selection of MRA or CTA

Table 1 Patient demographics.

Number of Patients	50			
Age	57–86 [median 77] year-old			
Gender	41 male			
ASA class	Class 2: 13, Class 3: 19, Class 4: 18			
History of aortic surgery	Root to Ascending	3		
	Arch	21	Total arch replacement	20
			TEVAR after debranch	1
	Descending		Replacement	3
			TEVAR	1
	Thoraco-abdominal	2		
	AAA	18	Replacement	17
			EVAR	1
Aortic pathology	Degenerative aneurysm	39		
	Chronic dissection	3		
	Acute dissection on aneurysm	2		
	Penetrating atherosclerotic ulcer	3		
	Anastomotic false aneurysm	3		

Table 2 Distribution of ICA-AKA.

	Right	Left	(Occlusion at origin)
Th7	0	1	
Th8	1	6	(2)
Th9	0	18	(1)
Th10	1	10	
Th11	0	7	(1)
Th12	2	4	(2)
L1	0	1	
L2	0	0	
Total	4	47	(6)

ICA-AKA: intercostal/lumbar arteries which supplies Adamkiewicz artery.

depended on the availability of the equipment. CTA was used in all three emergency cases.

When AKA was not identified by MRA, it was diagnosed as 'absent' ($n = 3$). In 47 patients, 51 ICA-AKAs were identified (Table 2). In four patients, there were double ICA-AKAs. Occlusion of ICA-AKA at its origin was diagnosed in six patients, in all of them on the left side. When the ICA-AKA was occluded, blood supply from adjacent intercostal or lumbar arteries was suspected to be significant. However, we were unable to distinguish the critical collateral flow to AKA.

TEVAR

To create a landing zone, a carotid–subclavian bypass was performed in two and visceral vessel bypass was performed in one. In nine patients who had extensive/multiple aneurysm(s) from the aortic arch to the descending aorta, TAR was performed using elephant trunk (ET) implantation. Regarding patients who had a history of aortic surgery, an artificial graft was used to create a proximal landing zone in 19 and a distal landing zone in three.

In all patients, TEVAR was carried out under general anaesthesia. The access route for TEVAR was a native artery in 35, an iliac conduit in 13 and a graft limb or a side branch of AAA graft in two patients.

MEP monitoring and cerebrospinal fluid drainage

In all patients trans-cranial MEPs were monitored during TEVAR and a cerebrospinal fluid drainage (CSFD) tube was placed before TEVAR in 31 patients.

Immediately after the stent graft was placed, the mean blood pressure was raised above 80 mmHg and MEP was monitored every 5 min. When the amplitude of MEPs decreased under general anaesthesia, or when symptoms and signs of SCI were noted during the postoperative period, CSFD (<15 cmH_2O) was started with the infusion of methylprednisolone (30 mg kg^{-1} bolus and 5.4 mg kg^{-1} h^{-1} for 23 h followed by 2.7 mg kg^{-1} h^{-1} for 2 days) and naloxone (1200 μg day^{-1}). Intensive spinal care with CSFD, methylprednisolone and naloxone was continued for 72 h if the symptom did not resolve or was discontinued 24 h after full recovery.

CSFD was started only after paraplegia or a decrease of less than 25% of the amplitude of MEPs was noted. CSFD was not indicated as a prophylactic measure after TEVAR.

Measurement of the aortic length

The length of 'proximal uncovered aorta' (from LSCA to stent graft), 'aortic coverage' by stent graft and 'distal uncovered aorta' (from stent graft to coeliac axis (CA)) was measured on CTA using curved planar reformation images processed in a workstation (GE Advantage workstation 4.3).

After TAR with a multibranch graft, the length of aortic coverage was measured from the distal anastomosis. This site coincided with the origin of ET and was several centimetres distal to the branch graft of LSCA. When ET was installed, the proximal edge of the stent graft was positioned inside the multibranch graft and not only inside ET. After replacement of the descending or the thoracoabdominal aorta, the position of LSCA and/or CA served as the point of reference for the measurement.

Statistical analysis

Values are the mean ± SD. Data were analysed using the chi-square test for categorical variables, and continuous variables were examined using analysis of variance (ANOVA). The level of statistical significance was set at $p < 0.05$.

Results

Mortality and morbidity

Initial success of TEVAR was achieved in all patients except for two patients with Type I endoleaks detected by CTA who were successfully treated by a repeat TEVAR. No operative (30 days) death was encountered. Injury and occlusion of access arteries occurred in one. Two patients were complicated with cerebral embolism due to the guidewire pull-through technique and atrial fibrillation.

The following three patients were complicated with paraplegia: Patient 1 was a 59-year-old man with a history of closure of ventricular septal defect, aortic valve replacement and repair of a Valsalva sinus aneurysm. He also suffered from liver cirrhosis. He developed aneurysmal dilatation of the whole thoracic aorta and underwent TAR with ET installation as the first-stage repair. MRA revealed the AKA arose from the left Th9-ICA. TEVAR with Gore TAG was performed 5 weeks later from ET (Z3) to T11. The iliac conduit was connected to the right common iliac artery but the haemostasis was time consuming because of obvious coagulopathy due to liver cirrhosis. Paraplegia was confirmed 24 h after TEVAR after the patient suffered much pain. Despite treatment for SCI, the patient could not ambulate. Retroperitoneal haematoma had to be removed twice. He eventually died from methicillin-resistant *Staphylococcus aureus* (MRSA) mediastinitis 4 months after TEVAR. The length of aortic coverage from the origin of ET to the distal flair was 325 mm.

Patient 2 was an 81-year-old man with ascending, arch and descending aorta aneurysms. MRA revealed the AKA branching from the left Th9-ICA. Four weeks after TAR with ET installation, TEVAR with TAG was performed from ET (Z3) to T12 (Fig. 1). The iliac conduit was required and

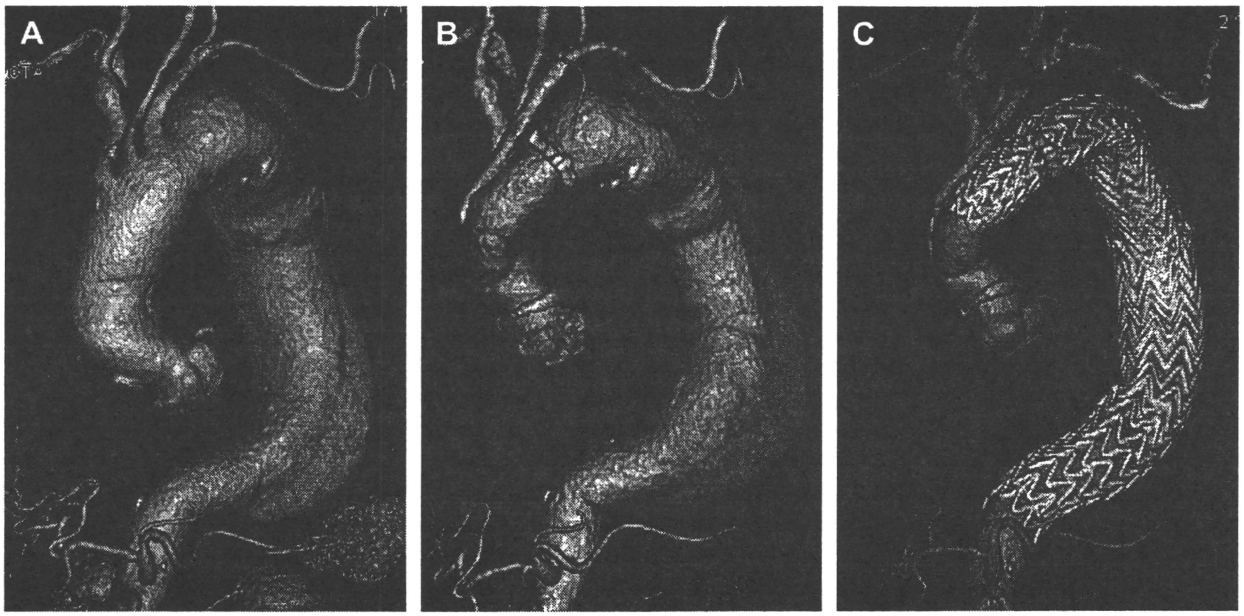

Figure 1 Sequence of CTA in Patient 2. Panel A: Preoperative, Panel B: After total arch replacement, Panel C: After TEVAR.

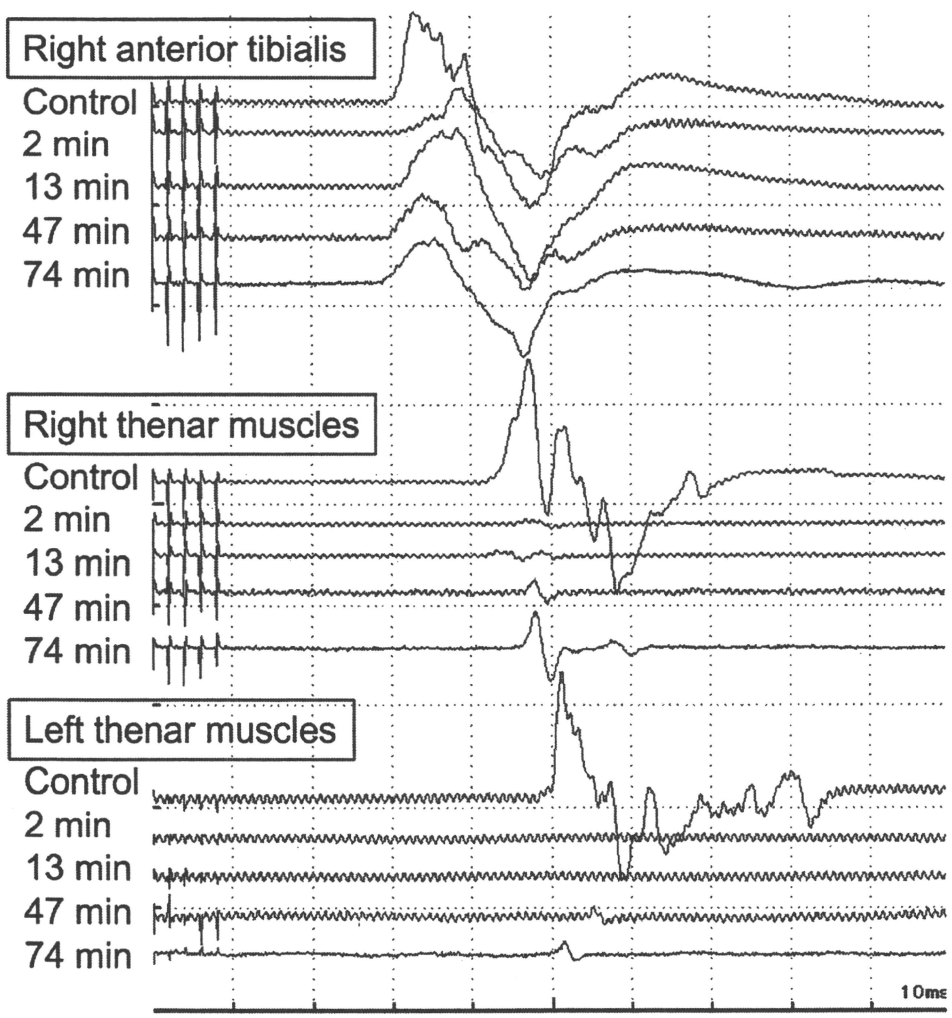

Figure 2 Sequence of MEPs in Patient 3. MEPs of the right anterior tibialis and both thenar muscles before TEVAR over Th9-ICA (control) and 2, 13, 47, and 74 minutes after TEVAR.

haemostasis took a long time due to co-existing consumption coagulopathy caused by aortic lesions. Six hours after TEVAR, the patient suddenly complained of back pain and paraplegia was confirmed. One hour after intensive spinal treatment, he could move his legs and on the next morning he could walk. The retroperitoneal, femoral and brachial haematomas were removed twice. The length of aortic coverage was 302 mm.

Patient 3 was a 78-year-old woman who had undergone TEVAR (Z3 to T7) for a proximal descending aortic aneurysm 6 months earlier. MRA revealed the ICA-AKA branching from the left Th9-ICA. Due to the rapid growth of the distal descending aortic aneurysm, TEVAR was performed again from the previous stent graft to L1. The CA was closed to create a distal landing zone. Immediately after the deployment over the Th9-ICA, the MEPs of both thenar muscles diminished and the amplitude of the MEPs of the right anterior tibialis decreased about 50% from the control amplitude (Fig. 2). Despite treatment for SCI, ankle dorsiflexion was slight when she awoke from anaesthesia. Intensive spinal care was continued and she gradually gained muscle strength within 3 h after TEVAR. On the following morning she could ambulate. The length of aortic coverage after the first TEVAR was 157 mm and was extended to 308 mm by the second TEVAR.

Incidence of paraplegia

The 50 patients were classified into group A: 17 patients whose patent ICA-AKA was not covered by TEVAR, group B: 24 patients whose ICA-AKA was covered by TEVAR and group C: nine patients in whom no patent ICA-AKA was identified. Group C included six patients whose ICA-AKA occluded at its origin and three patients whose ICA-AKA was absent.

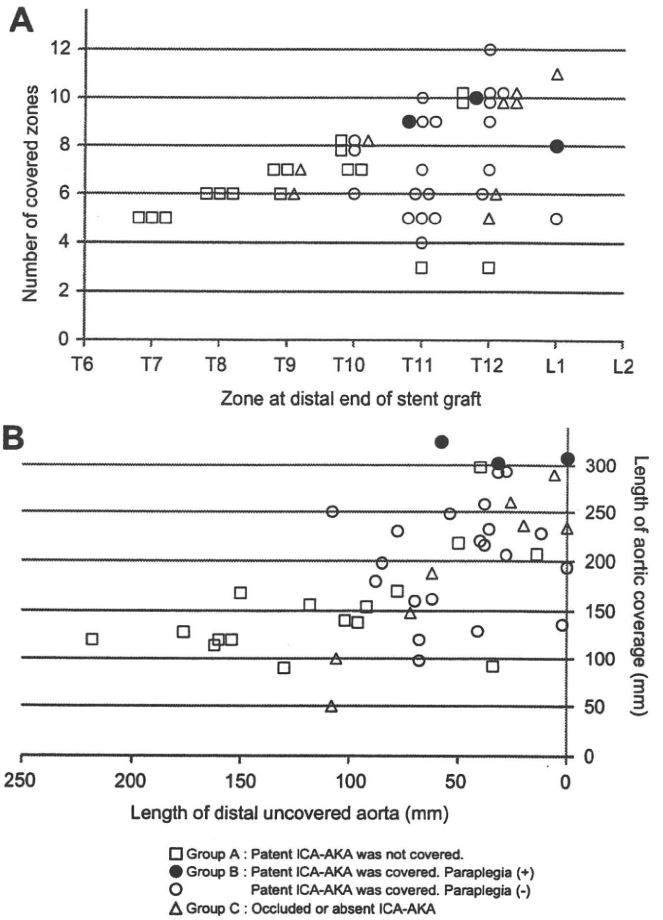

Figure 3 Distribution of patients with paraplegia in accordance with covered aorta and distal uncovered aorta expressed as number of aortic zones (Panel A) and measured length (Panel B).

Table 3 Comparison of patients with and without paraplegia.

	Paraplegia $N = 3$	No paraplegia $N = 47$	p
Age (year-old)	72.7 ± 11.9	76.1 ± 6.1	.3736
Male gender	2 (67%)	39 (83%)	.5094
ASA classification	3.7 ± 0.6	3.1 ± 0.8	.2026
Renal dysfunction	1 (33%)	18 (38%)	.3362
History of aortic repair (descending, thoracoabdominal, abdominal)	1 (33%)	19 (40%)	.8060
LSCA patency	3 (100%)	47 (100%)	—
RIIA patency	3 (100%)	47 (100%)	—
LIIA patency	3	44	.5359
Op time (minutes)	252 ± 117	141 ± 76	.0200
Blood loss (ml)	557 ± 274	363 ± 423	.4482
Use of an iliac conduit	2 (67%)	15 (32%)	.2335
Zones of aortic coverage	9 ± 1	7.2 ± 2.2	.1691
Proximal uncovered aorta (mm)	0	36 ± 49	.2191
Aortic coverage (mm)	312 ± 12	179 ± 64	.0009
Distal uncovered aorta (mm)	30 ± 29	72 ± 52	.1839
Hypotension	2 (67%)	3 (6%)	.0116

LSCA: Left subclavian artery, R(L)IIA: Right internal iliac artery.

Table 4 Reported incidence of spinal cord injury (SCI).

	Patients	Location	N	SCI	
Criado, 2002	TEVAR	Arch-descending	47	0	(0.0%)
Bergeron, 2003	TEVAR	Descending	38	0	(0.0%)
Czerny, 2004	TEVAR	Descending	54	0	(0.0%)
Orend, 2003	TEVAR	Various	74	2	(2.7%)
Mitchell, 1999	TEVAR	n/d	103	3	(2.9%)
Makaroun, 2005	TAG phase II	Various	142	4	(3.0%)
Ellozy, 2003	TEVAR	Descending	84	3	(3.6%)
Morales, 2007	TEVAR	n/d	186	7	(3.8%)
Bell, 2003	TEVAR	Various	67	3	(4.0%)
Greenberg, 2008	TEVAR	TAAA	352	15	(4.3%)
Gravereaux, 2001	TEVAR	Descending	53	3	(5.7%)
Greenberg, 2005	TXI & TXII	Various	100	6	(6.0%)
Sandroussi, 2007	TEVAR	n/d	65	4	(6.2%)
Cheung, 2005	TEVAR	Various	75	5	(6.5%)
Amabile, 2008	TEVAR	Descending	67	5	(7.5%)
Feezor, 2008	TEVAR	n/d	326	33	(10.0%)

The three patients who developed paraplegia were in group B, that is, ICA-AKA was covered by TEVAR. The estimated incidence of permanent and transient paraplegia was 3.7% in all patients subjected to TEVAR (81 patients), 4.5% in patients in whom part or the entire distal aorta was covered, regardless of ICA-AKA identification by MRA (66 patients) and 6.0% in those whose ICA-AKA was identified (50 patients, groups A, B and C). The incidence increased to 12.5% only when the patent ICA-AKA was covered by TEVAR (24 patients, group B).

Comparison of patients with and without paraplegia after TEVAR showed that the operation time and the length of aortic coverage were significantly longer in those with paraplegia (Table 3). Episodes of hypotension below 80 mmHg for more than 10 min during and after surgery were more frequent in patients with paraplegia. No difference was found in the patency rate of LSCA[27] and IIA. Other risk factors previously reported[28] such as the abdominal aortic surgery and the renal dysfunction showed no difference.

Fig. 3 shows the occurrence of paraplegia in relation to aortic coverage and distal aortic uncoverage length. When these were divided into zones, the stent grafts in the three patients with paraplegia were placed at T11 or distal to it and covered more than eight zones. Fourteen patients without paraplegia had the same range of intervened zones (Fig. 3A). In the three patients with paraplegia, the length of aortic coverage was more than 300 mm. The length of distal uncovered aorta was within 60 mm. Four other patients whose length of aortic coverage was between 270 mm and 300 mm and the length of distal uncovered aorta was less than 60 mm did not experience paraplegia (Fig. 3B).

Discussion

We have reported the low risk of paraplegia for patients subjected to descending and thoraco-abdominal aorta open repair by combined use of AKA identification by MRA and MEP measurement.[22] The risk of paraplegia has been considered to be lower after TEVAR than after open repair, but the incidence of SCI in the previous reports varied (Table 4). This variation might be due to differences in case mix as the area subjected to TEVAR was not the same and was not specified in some of the reports.[23]

The theoretical advantages of TEVAR concerning protection of the spinal cord are the maintenance of distal perfusion, stable haemodynamics and no reperfusion of the spinal cord.[12] However, additional ICAs are sacrificed for the landing zones and revascularisation of the ICAs is impossible. Paraplegia occurs after TEVAR when the arteries that supply the spinal cord are sacrificed, as well as after a period of hypotension or as a result of emboli from aortic atheromatous lesions.[29]

We encountered paraplegia in three patients whose patent ICA-AKA was covered and the length of aortic coverage was more than 300 mm. The rate of permanent and transient paraplegia was 12.5% (1/8), when the patent ICA-AKA was covered by TEVAR. This result was in agreement with that of a previous study showing that SCI occurred in 9.1% of the patients with occlusion of the ICA-AKA.[13] The authors did not encounter paraplegia in patients whose ICA-AKA was patent. This fact is also relevant to our result as none of our patients, whose ICA-AKA was already occluded at its origin before TEVAR or was absent, experienced paraplegia.

The spinal cord blood supply depends on many interchangeable collateral arteries that supply the anterior spinal cord artery, rather than a single dominant AKA.[21] However, the importance of the patency of the ICA-AKA during TEVAR[13] and the restoration of blood flow in the spinal cord after revascularisation of ICA-AKA at the aortic replacement have been reported.[22] Patency of ICA-AKA is sufficient to prevent paraplegia and occlusion of the patent ICA-AKA is critical. To preserve the patency of ICA-AKA, this should be identified preoperatively to allow the creation of an adequate landing zone.[12]

The rate of paraplegia, 12.5%, means that 87.5% (7/8) patients did not develop paraplegia after the coverage of patent ICA-AKA. Between three patients who suffered paraplegia and 21 patients who did not in group B, the threshold of the length of aortic coverage was 300 mm. The length of aortic coverage has been described as a risk for SCI in previous reports.[13,30] Amabile et al. reported that