Spinal cord protection during thoracoabdominal aortic aneurysm surgery

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Introduction
Postoperative paraplegia including paraparesis is a devastating complication after descending thoracic aortic aneurysm (DTAA) and thoracoabdominal aortic aneurysm (TAAA) repair surgery. This lecture will be focused on the pathophysiological mechanisms for the development of postoperative paraplegia, the strategies to prevent the development of postoperative paraplegia, and anesthetic managements during the monitoring of motor evoked potentials (MEPs).

Incidence of postoperative paraplegia
The incidence of paraplegia or paraparesis is reported to be 0% to 10% for thoracic aneurysm repair and 10% to 40% for TAAA (1). Patients with Crawford type II of TAAA have the highest risk of paraplegia. Risk factors for paraplegia after TAAA include emergency presentation, prolonged aortic cross-cramping time, extensive aneurysm (Crawford type I and II), postoperative hypotension, advanced age, previous abdominal aortic aneurysm repair, severe atherosclerotic disease and diabetes. High mortality rate in patients undergoing DTAA and TAAA, ranging from 5% to 20% may underestimate the rate of paraplegia associated with these procedures.

Anatomy for spinal cord blood flow
The spinal cord receives the blood supply from one anterior spinal artery and two posterior spinal arteries. The anterior spinal artery supplies the motor tracts in the spinal cord. The upper cervical segment of the spinal cord receives from the branches of vertebral arteries. The thoracic portion of the anterior spinal artery is supplied by the anterior radicular arteries and the largest of the radicular arteries is called as the great radicular artery (GRA) or the artery of Adamkiewicz. The artery of Adamkiewicz has a variable origin, but generally originates from T8 and L1 in the majority of patient. The lumbar portion of the anterior spinal cord receives from the lumbar and pelvic circulation.

Preoperative assessments.
Preoperative spinal cord angiography in patients with DTAA and TAAA has been reported (2). Based on these results, precise identification of intercostals arteries giving rise to the GRA can be performed and reimplantation of these vessels may be planned preoperatively. However, recent evidence indicated that even in patients with an identified and reimplanted GRA, spinal cord injury can develop postoperatively. In contrast, a number of patients who did not receive reimplantation of the GRA had no postoperative paraplegia. The usefulness of preoperative identification of the GRA remained controversial.

Mechanisms of development of paraplegia
Intraoperative ischemia of the spinal cord is partly related to interruption of blood flow through the intercostals arteries associated with GRA, which is resulted from cross-clamping of the aorta and/or surgical ligation during aneurysm resection. However, spinal blood flow is unlikely depend on a single GRA. Conversely, spinal cord integrity is maintained by an extensive network of collateral vessels from lumbar arteries and pelvic circulation. Recent evidence indicated that a reduction in collateral perfusion during the cross-clamping of the aorta is more related to spinal cord ischemia, compared with an interruption of blood flow through GRA (3).

Prevention of postoperative paraplegia
Blood pressure management
Spinal cord perfusion pressure (SCPP) during clamping the aorta can be determined by the following formula:

\[ \text{SCPP} = \text{MAP} - (\text{CSFP or CVP} \ [\text{whichever is greater}]) \]

Where MAP = mean arterial pressure; CSFP = cerebrospinal fluid pressure; and CVP = central venous pressure. The MAP levels to maintain spinal cord blood flow can vary, but SCPP is required to maintain at least 60 mmHg. The maintenance of CSFP and CVP at low levels is also required. During clamping the aorta, MAP is maintained at 75-85 mmHg in normotensive patients, probably higher in hypertensive patients. In cases in which spinal cord ischemia developed, MAP is recommended to increase to the levels of more than 95 mmHg.

Cerebrospinal fluid drainage
In order to maintain SCPP, lumbar CSF drainage can be inserted. Although there have been no definitive evidence that the use of CSF drainage reduce spinal cord injury, the available data supports the use of CSF drainage in patients with a risk of paraplegia after TAAA. CSFP is maintained at less than 10 mmHg (13 cmH2O). Drainage of more than 10-15 ml/h should be avoided
to prevent the development of complications such as intracranial hemorrhage. The incidence of intracranial hemorrhage is reported to be 0.5-3.5% and, once intracranial hemorrhage developed, mortality can be as high as 40%. (1) Bloody CSF can be an indicator of intracranial hemorrhage during the monitoring of CSFP.

**Monitoring of motor function**

Functional integrity of motor function can be monitored using motor evoked potentials (MEPs). After the recent progress in the stimulating device using the multi-pulse, MEP monitoring has become feasible during the TAAA. To stimulate motor cortex, transcranial electrical stimulation with a train of 4-6 pulses with an interstimulus interval of 2 msec (500Hz) is used. Myogenic MEP is more sensitive to spinal cord ischemia compared to spinal MEP. MEP monitoring can be used to assess whether spinal cord perfusion during cross-clamping of the aorta is adequate, to assess whether surgical reconstruction is necessary, and to assess the prognosis of motor function, although cut-off points of MEP amplitude to predict postoperative motor deficits remained controversial (4,5). Anesthetic managements are important for successful MEP monitoring. Total intravenous anesthesia using propofol and opioids is recommended. However, occasionally, the use of propofol may be limited, because cardiopulmonary bypass, hypothermia, and occlusion of the aorta can affect the metabolism and concentration of propofol, resulting in the fluctuation of MEP responses. In such cases, ketamine-based anesthesia may be required because ketamine has no suppressive effects on myogenic MEPs.

**Hypothermia**

Hypothermia can be the most reliable technique to protect the ischemic spinal cord injury. Since a reduction of one degree centigrade reduces oxygen requirement by approximately 5%, a twofold prolongation of tolerated cross clamp time can be achieved by even inducing mild hypothermia (34 degrees C). In addition to a reduction of oxygen demand, a variety of mechanisms have been reported regarding the neuroprotective effects of hypothermia. Clinically, both systemic and regional hypothermia can be applied for TAAA surgery. MEP monitoring is feasible as long as hypothermia >28 degrees C is used. In contrast, when circulatory arrest with deep hypothermia is used, usefulness of MEP monitoring is questionable.

**Treatment/prevention of postoperative paraplegia**

Delayed-onset paraplegia is common after TAAA surgery. Maintenance of SCPP is also important postoperatively, so that MAP and CSFP/CVP should be maintained at the proper levels. CSF drainage can be applied for 48 to 72 hours. The use of morphine would be better avoided postoperatively, because it may worsen the mildly ischemic spinal cord injury (6). Although
evidence is not clear, low dose of naloxone may be administered postoperatively.

References


