Chapter 53
Anesthesia for Adult Vascular Surgery and Cerebrospinal Protection

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Abstract Paraplegia is a significant complication of cardiovascular surgery, especially aortic aneurysm surgery. The patient management of cardiovascular surgery to prevent the occurrence of paraplegia caused by spinal cord injury is important during the perioperative period. Motor-evoked potentials are effective for intraoperative spinal cord monitoring, while Adamkiewicz artery identification and reconstruction, distal aortic perfusion, and cerebrospinal fluid drainage are effective for spinal cord perfusion pressure management. In addition, the pharmacological actions of anesthesia on the spinal cord are critical, and ischemic tolerance is also attracting attention. Here, we discuss the possibilities for spinal cord protection during the perioperative period.

Keywords Cardiovascular surgery • Cerebrospinal protection • Spinal ischemia

53.1 Introduction

In cardiovascular surgery, the most significant complication is paraplegia accompanying reperfusion injury or ischemic spinal cord injury. Paraplegias primarily develop after thoracoabdominal aortic aneurysm surgery. Since the spinal cord is constructed with complex blood flow control, its management is affected by a variety of factors. In addition, monitoring of spinal cord ischemia is crucial for the early detection of paraplegia. Since anesthesia affects the spinal cord in many different ways, careful selection of anesthesia is essential. Furthermore, in the perioperative period, procedures such as Adamkiewicz artery identification and reconstruction, spinal cord ischemia monitoring, spinal cord perfusion pressure management, cerebrospinal drainage, spinal epidural cooling, and pharmacotherapy are important for spinal cord protection. In this chapter, we would like to describe the anesthetic management for cardiovascular surgery and perspectives for cerebrospinal protection.

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53.2 Blood Flow in the Spinal Cord

The spinal cord is supplied by the vertebral and subclavian arteries, thoracoabdominal aorta, and segmental arteries from the internal iliac artery. The segmental arteries anastomose in the vertical direction to form the anterior spinal artery and posterior spinal artery. The anterior spinal artery continuously passes along the ventral side of the spinal cord from the basilar artery to the filum terminale, while the posterior spinal arteries are thin and discontinuous and traverse bilaterally on the dorsal side of the spinal cord. Furthermore, the pial arterial plexus forms a network of vessels that covers the surface of the spinal cord. The cervical spinal cord is supplied by the vertebral, ascending cervical, and deep cervical arteries. The anterior spinal artery of the thoracic spinal cord is supplied by the radicular arteries, which originate directly from the highest intercostal artery and dorsal aorta (1–2 arteries from the cervical spinal cord, 2–3 arteries from the thoracic spinal cord, and 1–2 arteries from the lumbar spinal cord). The largest of these is called the great radicular artery, or Adamkiewicz artery, which arises primarily on the left side, and is known to originate between T9 and T12 and between T12 and L3 in 75–80 % and 83.9 % of cases, respectively [1]. The lumbar spinal cord is supplied by the lumbar artery, whereas the sacral spinal cord is supplied by the anastomosis of the lateral sacral, median sacral, and iliolumbar arteries. Magnetic resonance angiography and multidetector-row computed tomography are utilized to identify these arteries and the blood supply (Fig. 53.1). Animal studies have found that paraplegia occurs 70 % of the time when the great radicular artery is ligated. There is a large amount of individual variation in the arteries that supply the spinal cord, and this phenomenon leads to complicated perioperative management. The “collateral network concept” of blood flow in the spinal cord proposed by Griepp et al. (Fig. 53.2) states that the spinal cord does not solely depend on the great radicular artery but is involved in a complex system of collateral pathways [2]. In addition, similarly to cerebral blood flow, spinal blood flow involves autoregulation, which is lost under conditions of hypoxemia and hypercapnia [3].

53.3 Causes of Spinal Cord Ischemia

Ischemic spinal cord injury may occur in cardiovascular surgery, particularly in thoracoabdominal aortic aneurysm surgery. The most significant complication of spinal cord ischemia is paraplegia, with incidence rates of 0.5–1.5 % in aortic stenosis repair, 0–10 % in thoracic aortic aneurysm surgery, 10–20 % in thoracoabdominal aortic surgery, and about 40 % in extensive dissecting thoracoabdominal aortic aneurysm surgery [4]. In addition, paraplegia is also reported to occur 0.25 % of the time in lower renal artery aortic aneurysm surgery [5]. In particular, the incidence of spinal cord injury is reported to be approximately

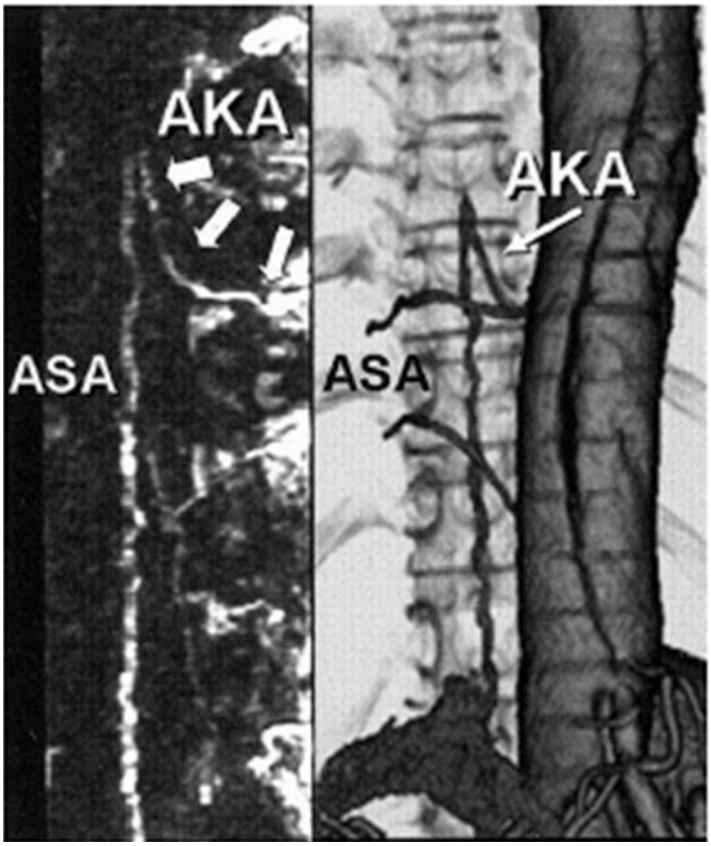

Fig. 53.1 The Adamkiewicz artery (MRA). Magnetic resonance angiography demonstrates the Adamkiewicz artery (AKA [*large white arrows*]) ascending to the anterior midsagittal surface of the spinal cord from the radicular-medullary artery originating from the dorsal branch of the intercostal or lumbar artery. It is continuous with the anterior spinal artery (ASA), with a hairpin turn in the early phase (*left*). The Adamkiewicz artery (*white arrow*) can be visualized clearly by multidetector computed tomography scans as well (*right*). Ann Thorac Surg 2006;82:592–6

20 % with vascular prosthesis implantation for Crawford II thoracoabdominal aortic aneurysms [6]. There are several causes of spinal cord ischemia, including hypertension proximal to aortic cross clamping, hypotension distal to cross clamping, elevated cerebrospinal fluid (CSF) pressure, blockage of blood flow in the intercostal and lumbar arteries, extended duration of aortic cross clamping, extensive aortic lesions, aortic dissection, and emergency surgery. Injury to the gray and white matter of the spinal cord and late-onset cell death of motor neurons are characteristically known to be caused by ischemia. Regarding mechanisms for these ischemic spinal cord injuries, glutamic acid-Ca^{2+} imbalance similar to that of the brain [7], glial cell activation after ischemia, and mitochondrial dysfunction may be involved [8].

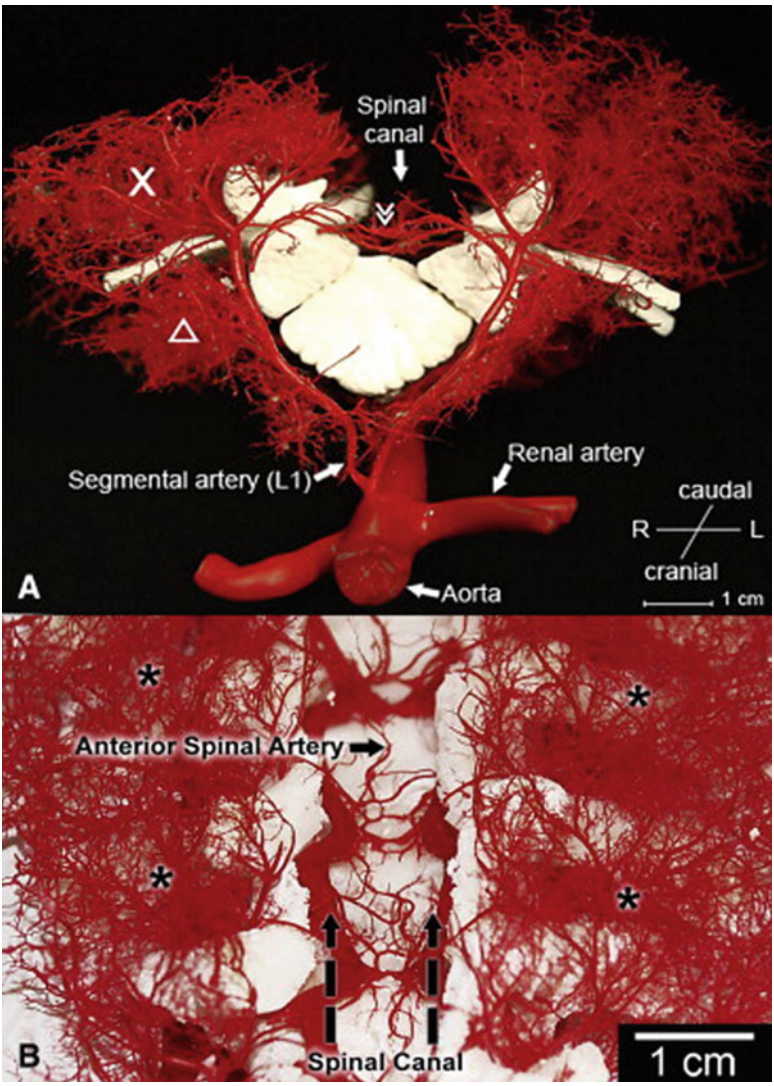

Fig. 53.2 The collateral network. Spinal cord perfusion and protection during descending thoracic and thoracoabdominal aortic surgery: the collateral network concept. Ann Thorac Surg: 83: 865–869, 2007

53.4 Intraoperative Spinal Cord Ischemia Monitoring

Several methods, such as measurements of somatosensory-evoked potential (SEP) and motor-evoked potential (MEP), are available for spinal cord ischemia monitoring. SEP monitoring detects ischemia of the sensory regions at the posterior and lateral funiculus but does not show ischemia of the motor regions at the anterior

horn of the spinal cord. A large prospective study of thoracoabdominal aortic aneurysm cases reported that this monitoring does not improve neurological prognosis, as shown by a 13 % false-negative rate and a 67 % false-positive rate [9]. In MEP monitoring, spinal cord α-motor neurons are stimulated by transcranial electrical stimulation of the cerebral motor cortex, and evoked electromyograms from the upper and lower limb muscles are monitored. Since MEP monitoring reacts early to ischemia of the anterior horn of the spinal cord with corticospinal tract monitoring, it is useful for verifying the intercostal arteries that supply blood to the spinal cord, as well as for evaluating intercostal artery blood flow reconstruction or perfusion distal to aortic cross clamping. In addition, because a body temperature of 28–30 °C is known to have very little effect on MEP, it can also be used in surgery performed under hypothermic conditions. If there is a decrease in MEP in both lower and upper limbs, minimizing the effects of anesthetics is essential, because the decrease in MEP could be due to anesthetic agents. If the unilateral decrease in MEP in the lower limb is on the side supplied by the femoral artery, transient ischemic changes may occur in the periphery. In particular, if there is a decrease in MEP only in the lower limbs and not in the upper limbs, spinal cord ischemia may be involved (Fig. 53.3).

53.5 Anesthetic Management

Anesthetics that only have a small effect on MEP measurements are chosen when monitoring spinal cord ischemia. It is necessary to exercise care in the selection of anesthetic agents, since monitoring precision decreases with the use of muscle relaxants and the amplitude of muscular MEP decreases with the inhibition of synaptic conduction caused by nitrous oxide, desflurane, sevoflurane, and isoflurane. While propofol and midazolam are known to have less effect compared to inhalation anesthetics, they also possess inhibitory effects against MEP at high concentrations.

Remifentanil, fentanyl, and ketamine have very little effect on MEP and so are useful in surgery that requires spinal cord ischemia monitoring. Muscle relaxants should either not be used at all after anesthesia induction or used minimally under muscle relaxation monitoring. Barbiturates are reported to reduce spinal cord injury caused by aortic cross clamping [10]. Drugs such as morphine that act on μ-opioid receptors can exacerbate spinal cord motor neuropathy after spinal cord ischemia, so caution is necessary. Naloxone antagonizes the exacerbating action of morphine on spinal cord motor neuropathy and is known to be effective in patients where spinal cord ischemia is suspected [11]. Administration of fentanyl can be harmful when spinal cord ischemia is suspected. Buprenorphine and pentazocine are used in animal studies, since they are known to not worsen ischemic spinal cord injuries [12]. Ketamine is an NMDA receptor antagonist that has been reported to reduce ischemic spinal cord injuries [13]. Ketamine can also be used postoperatively due to the analgesic effects of its metabolite norketamine and its lack of damaging effects

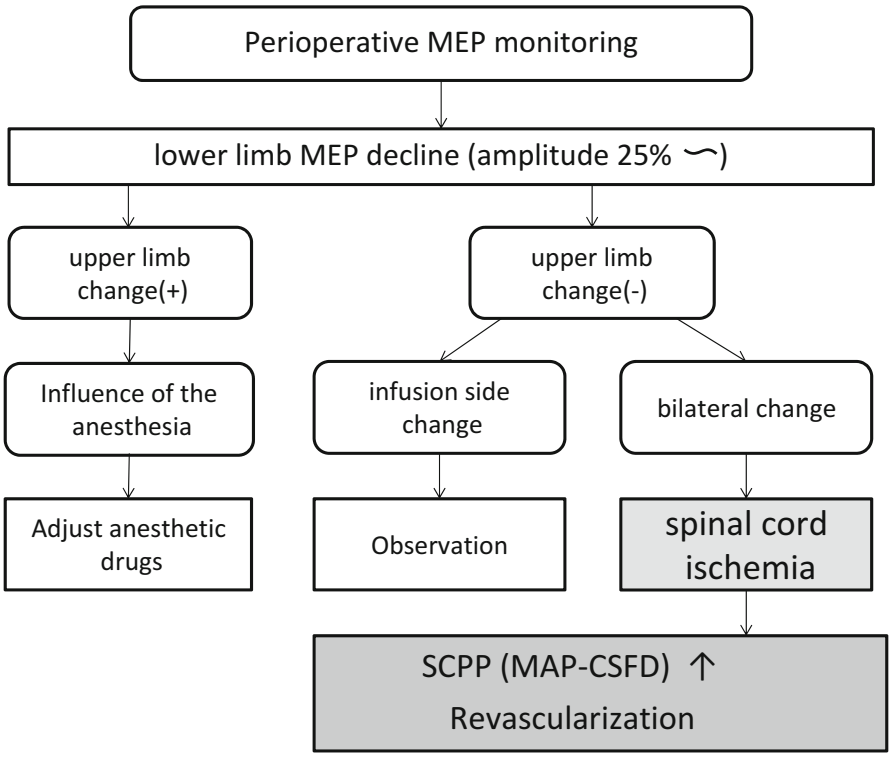

Fig. 53.3 Strategy of spinal cord ischemia protection

on the spinal cord. In addition, continuous administration of local anesthetics to the intercostal nerve block or wound area is also effective. Furthermore, dexmedetomidine has been reported to possess a spinal motor cell protective effect in animal studies, indicating its potential usefulness [14]. Ischemic preconditioning is known to be important in cardioplegia and has been reported in animal studies to possibly be effective in achieving spinal cord protection [15]. We have summarized spinal cord protection and multiple-facility clinical trials involving cerebroprotective medicines (Table 53.1).

Standard ASA monitors and an arterial line, namely, a central line to measure central venous pressure (CVP) and determine volume status, are important. A PAC may be useful to help manage hemodynamics and volume status in certain patients but is not routinely indicated. A bispectral index monitor is placed before induction. Total intravenous anesthetic technique including propofol and fentanyl infusion is often selected for general anesthesia. The infusion dose of propofol (150–250 mcg/kg/h or target-controlled infusion 3–4 mcg/mL) is titrated to effect according to BIS (bispectral index monitoring). (40–60). Remifentanil is administered at 0.2–0.5 μg/kg/min. Patients who present with cardiovascular disease often have coronary artery disease, hypertension, lung disease, diabetes, or renal disease. In order to

Table 53.1 As a result of spinal cord protection and many facilities, the clinical trial of the cerebroprotection medicine

Ca^{2+} channel blocker	*No benefit*	NMDA antagonist	Dizocilpine: protective
		Noncompetitive	Dextrorphan: protective
Na^{+} channel blocker	Riluzole: protective	Competitive NMDA antagonist	No benefit
GABA agonist	Clomethiazole: protective	Polyamine receptor antagonist	Eliprodil: not investigated
	DHEA: protective		
Free radical scavenger	Edaravone: protective	Glycine antagonist	ACEA1021: not investigated
			Gavestinel: not investigated
Neurotrophic factor	NGF: protective	AMPA/KA receptor antagonist	NBQX: protective
	BDNF: not investigated		YM872: not investigated
Ganglioside	*Modest benefit*	Metabotropic receptor antagonist	Not investigated
$MgSO_4$	Protective	Others	Piracetam: not investigated
Steroid	*Methylprednisolone: modest benefit*	Hormone	TRH: no benefit
Opioid receptor antagonist	No benefit	Mannitol	No benefit

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avoid a blood pressure drop, the induction must be performed extremely carefully in advanced cardiac dysfunction. Rocuronium bromide (0.6–0.9 mg/kg body weight), or vecuronium bromide (0.08–0.1 mg/kg body weight), or succinylcholine (1.5 mg/kg body weight) is used to facilitate intubation. These are used only at the time of anesthesia induction. Otherwise, muscle relaxants are used at the minimum concentrations required under the monitoring of the muscle relaxation level during the operation.

Weaning from cardiopulmonary bypass (CPB) may require either a change in anesthetic technique or an adjustment in dose delivery. During the rewarming process from hypothermia during CPB, there is likely to be insufficient anesthesia. Regardless of the exact nature of this change, it is vital that anesthesia is properly maintained and this should be confirmed by the operation team members.

As Schreiber reported, a meta-analysis showed that the positive effects of the designated techniques (positive airway pressure, low-volume ventilation, or vital capacity maneuvers during CPB) are probably short-lived with a questionable impact on the long-term clinical outcome of the treated patients [16]. Based on

the available data, it might be impossible to advise an optimal or best-evidence strategy of lung preservation during CPB.

53.6 Prevention of Spinal Cord Ischemia

Katz et al. reported that spinal cord ischemia can occur 71 % of the time when aortic cross clamping continues for ≥30 min [17]. In this case, performing distal aortic perfusion (left heart bypass or femoral artery bypass) to increase spinal cord perfusion is known to be effective [18, 19]. Jex et al. reported that the risk of spinal cord ischemia decreases from 44 to 8 % when distal aortic perfusion is performed [6]. In addition, CSF drainage (CSFD) is known to be effective in spinal cord ischemia. Spinal cord perfusion pressure decreases when CSF pressure elevates after aortic cross clamping, leading to an increased risk of spinal cord ischemia. CSFD thus holds a valuable potential for treating spinal cord ischemia and is used frequently [20]. In general, CSF pressure is managed with a target of 10 cm H_2O. However, since there can be severe complications such as epidural hematoma and subdural hematoma, proper attention must be given to the properties and volume of drainage. Under hypothermic conditions, the oxygen requirement of the central nervous system is known to decrease by 6–7 % for every 1 °C decrease in body temperature. Such decreases are also known to lower tissue metabolism and suppress membrane stabilization and excitatory neurotransmitter release, validating the protective effect of the reperfusion phase. Spinal epidural cooling is a method that lowers the temperature of the local spinal cord with a cool perfusion solution and has been reported to reduce nerve damage [21]. One study reported that a combination of CSFD and spinal epidural cooling decreased the incidence of paraplegia from 23.9 to 2.9 % [22].

53.7 Treatment and Management During Spinal Cord Ischemia

When spinal cord ischemia is suspected following reduced or lost MEP, procedures such as sustained perfusion pressure, confirmation of blood supply status, management of CSFD, reconstruction of an intercostal artery, or selective perfusion are performed to increase spinal cord blood flow. If this occurs during CPB, perfusion pressure is increased by raising the flow rate and blood pressure distal to cross clamping, and selective intercostal artery perfusion is performed, if possible, with the goal of achieving MEP recovery.

53.8 Postoperative Management

About 20–40 % of postoperative paraplegia is thought to be of late-onset development; therefore, sustaining spinal cord perfusion pressure in postoperative management is necessary and extremely important. When CSFD and blood pressure are raised to increase perfusion pressure, paraplegia may improve. Risk factors for spinal cord ischemia include hypotension (blood pressure $\leq$60 mmHg), anemia (hemoglobin $\leq$10 g/dL), and low cardiac output ($\leq$2.0 L/min) [23]. In addition, Etz et al. reported that CVP was higher and mean blood pressure was significantly lower in patients who experienced late-onset paraplegia [24]. Management of the patient's general condition that focuses on spinal cord perfusion pressure throughout the perioperative period is therefore necessary. Since the duration of postoperative sedation can be long and discovery can be delayed, monitoring spinal cord function in the intensive care unit is also considered critical.

53.9 Conclusion

In cardiovascular surgery, ischemic spinal cord injury is a significant complication. Since the spinal cord is under complex blood flow control, circulation management that focuses on spinal cord perfusion pressure is crucial. In addition, anesthesia should be selected with consideration of its pharmacological actions on the spinal cord, as well as monitoring the spinal cord. MEP is considered to be useful when monitoring for spinal cord ischemia, and the management of spinal cord perfusion pressure and revascularization together with early diagnosis are essential. For spinal cord protection, the combination of methods such as CSFD and spinal epidural cooling is also effective. Furthermore, postoperative management is a key factor, since patients can develop late-onset spinal cord injuries.

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