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Spinal Cord Protection for Descending or Thoracoabdominal Aortic Aneurysm Repair

Nobuyoshi Kawaharada, Toshiro Ito and Tetsuya Higami Department of Thoracic and Cardiovascular Surgery Sapporo Medical University School of Medicine Japan

1. Introduction

The mortality and morbidity of extensive thoracoabdominal aorta replacement has improved markedly in recent years [1]. However, postoperative paraplegia from spinal cord infarction remains the most devastating complication that faces patients undergoing surgery on the thoracoabdominal aorta because loss of lowerlimb function imposes severe constraints on the quality of life. Additionally, paraplegia is associated with higher postoperative mortality and morbidity. Despite advances in spinal cord protection, the risk of spinal cord ischemia or infarction as a consequence of open surgical repair of thoracoabdominal aortic aneurysms (TAAAs) remains within the range of 8-28%[2,3]. The registry of the Japanese Association for Thoracic Surgery reported that the hospital mortality for surgery on the thoracoabdominal aorta was 14.2% in 561 patients during 2008 [4]. The U.S. multicenter registry in 2001 disclosed that early mortality after thoracoabdominal aortic surgery was 20% [5].

There are two major events during which injury to the spinal cord can occur. Firstly, spinal cord injury happens depending on the duration and degree of ischemia during crossclamping. The surgeon must temporarily interrupt aortic blood flow to the lower body, which renders the distal organs (including the spinal cord) ischemic, in order to resect the aneurysm. Secondly, damage may occur from the loss of blood flow to the spinal cord after the period of aortic cross-clamping because of failure to reattach the intercostal and lumbar arteries that are critical to the spinal cord blood supply.

Essentially, being a neural tissue, the spinal cord tolerates ischemia poorly and if infarction ensues, paraplegia results. A number of adjunctive measures have been used successfully to counteract the consequences brought about by spinal cord ischemia during surgical intervention and a precarious spinal cord blood supply postoperatively. The incidence of paraplegia and paraparesis at centers for aneurysm repair has been decreasing then. Occasionally, a case of spinal cord injury still occurs, and the most important factors for the prevention of either immediate or delayed paraplegia remain to be elucidated [6-10].

In this chapter, we review the contemporary anatomical and pathophysiological understanding of spinal cord blood supply and present the scientific basis for clinical interventions used during descending and thoracoabdominal aortic surgery in order to reduce the incidence of paraplegia.

2. Blood supply of the spinal cord

2.1 Arterial supply of the spinal cord

The vessels that supply the spinal cord are derived from the branches of the vertebral, deep cervical, intercostal, and lumbar arteries. The spinal cord is supplied by three longitudinal arteries, which include an anterior spinal artery and two posterior spinal arteries. These vessels are reinforced by blood from segmental vessels called radicular arteries. The anterior artery is larger than the two posterior arteries and provides 75% of spinal blood flow. The posterior spinal arteries arise as small branches of either the vertebral or posterior inferior cerebellar arteries. Between the anterior and posterior arteries, collateral blood flow is minimal.

2.2 Spinal arteries

The anterior spinal artery is formed by the union of two small branches from the vertebral arteries. It runs the length of the spinal cord in the anterior median fissure and supplies the anterior two-thirds of the spinal cord. The caliber of this artery varies according to its proximity to a major radicular artery. It is usually smallest in the T4 to T8 region of the cord.

2.3 Radicular arteries

The radicular arteries arise from the spinal branches of the vertebral, deep cervical, ascending cervical, posterior intercostal, lumbar, and lateral sacral arteries. They enter the vertebral canal through the intervertebral foramina and divide into anterior and posterior radicular arteries. The anterior radicular arteries supply the anterior spinal artery and the posterior radicular arteries contribute blood to the posterior spinal arteries. The radicular arteries supply the vertebrae, meninges, and spinal arteries. They pass along the dorsal and ventral roots of the spinal nerves to reach the spinal cord.

2.4 Anatomy of the arteria radicularis magna (artery of Adamkiewicz)

One segmental artery has assumed particular importance in the pathogenesis of spinal cord ischemia. The arteria radicularis magna (ARM), also known as the artery of Adamkiewicz, is an exceptionally large radicular artery that anastomoses into the mid-segment of the anterior spinal artery. The segmental reinforcements of blood supply from the radicular arteries are very important in supplying the anterior and posterior spinal arteries. The spinal cord may also suffer circulatory impairment if the radicular arteries, particularly the great anterior radicular artery, are narrowed by obstructive arterial disease or by ligation during surgery of the intercostal or lumbar arteries from which they arise.

Through the anterior spinal artery , the ARM supplies the major flow to the lower thoracic and lumbar cord segments. The ARM can arise from any segmental artery between T7 and L4 on either side, or directly from the aorta, but frequently originates from one of the left segmental arteries between T8 and L1. In a study of 102 cadavers, Koshino et al. found that approximately 70% of Adamkiewicz arteries originated from the intercostal and/or lumber arteries on the left side, frequently at the T8-L1 vertebral level [11]. The study reported that there was no significant correlation between the diameter of the ARM and the diameters of the intercostal and lumbar arteries, from which the ARM originated.

Furthermore, within the Th8 to L1 vertebral level, the diameters of the intercostal and lumbar arteries varied considerably and did not correlate with the diameter of the ARM.

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Morishita et al. reported that the anterior spinal artery was continuous in adult cadavers and that its diameters above and below the ARM were inconsistent. Furthermore, distal spinal blood supply becomes progressively dependent on the ARM at the narrowest point of the anterior spinal artery. Although larger compared with other radicular arteries, the ARM is of variable diameter ranging from 0.25 to 1.07 mm in cadaveric examinations [12].

3. Strategies to prevent spinal cord ischemia

3.1 Preoperative examination and monitoring of spinal cord function 3.1.1 Preoperative detection of the artery of Adamkiewicz

Kieffer et al.[13] performed preoperative spinal cord arteriography in patients with thoracic and thoracoabdominal aneurysms and identified the arteria radicularis magna (ARM) in 85%. They reported that the risk of paraplegia was 5% if the ARM was identified preoperatively and reimplanted; whereas, it was 50% if the ARM was not reattached back in the 1980s. The complexity and invasiveness of the method, however, prevented its widespread diffusion. There is growing evidence that the Adamkiewicz artery may, nowadays, be visualized through noninvasive methods, such as magnetic resonance angiography (MRA) or computed tomographic angiography (CTA)[14-16]. In a review of literature, Melissano et al.[17] revealed the identification of the artery of Adamkiewicz in 84% of patients by using MRA or CTA. Through examination of the angiographic location of the spinal cord blood supply and its relationship to postoperative paraplegia, they concluded that selective intercostal angiography was safe and the procedure provided information to help understand the mechanisms and risks of spinal cord complications after thoracoabdominal aneurysm repair.

3.1.2 Somatosensory-evoked potentials and motor-evoked potentials

Popularized by Cunningham and associates in the early 1980s [18], somatosensory-evoked potentials (SSEPs) record cortical stimulations through the scalp after peripheral electrical stimulation of the posterior tibial or peroneal nerves. SSEPs are designed to monitor spinal cord perfusion [19]. Schepens et al.[20] demonstrated the usefulness of SSEPs in lowering the incidence of spinal cord injury in patients undergoing thoracic and thoracoabdominal aneurysm repair; however, the tracing of SSEPs has several limitations. First, the tracings are altered by some anesthesic agents, hypothermia, and neuromuscular blockade. Second, SSEPs only evaluate the function of the posterior and lateral columns of the spinal cord. Third, there are reports of false-positive and false-negative responses during the intraoperative monitoring of SSEPs [21].

These limitations of SSEPs can be explained anatomically. Since SSEPs are transmitted through the posterolateral tracts, they primarily reflect ischemia in the region of the posterior spinal arteries. The SSEPs are neither sensitive nor specific monitors of the corticospinal tracts in the anterior spinal cord (supplied by the anterior spinal artery); but, the anterior spinal cord is usually the first region affected during spinal ischemia which causes paraplegia. Therefore, while SSEPs detect extensive spinal ischemia affecting global cord function, smaller degrees of ischemia limited to the anterior spinal motor territories may not be detected.

However, due to a great variance of sensitivity and specificity to the spinal ischemia in many clinical studies [22,23], the use of motor evoked potentials (MEPs) has been proposed. The MEP is a more logical way to detect impending paraplegia as it directly monitors nerve

conduction in the corticospinal tract. MEPs can evaluate the function of the anterior columns of the spinal cord. Use of MEPs greatly increases the sensitivity and specificity of evoked potentials in detecting spinal ischemia compared with monitoring of SSEPs alone [24]. To detect MEPs, the motor cortex or spinal cord proximal to the aortic clamp level is stimulated, and potentials are recorded in the lower spinal cord, peripheral nerves, or muscles. Unlike the SSEPs, which may have a slow response time, inadequate cord perfusion can result in loss of MEPs within as little as 1 min[25]. Laschinger et al.[26] evaluated the usefulness of MEPs to monitor spinal cord perfusion; while van Dongen et al.[27] reported that MEPs monitoring was feasible during low-dose propofol, fentanyl/50% N2O in O2 anesthesia, and partial neuromuscular blockade.

3.2 Strategy to reduce the severity of spinal cord ischemia

When prolonged spinal cord ischemia is anticipated, it is important to minimize the severity of the ischemia. Various methods have been proposed to increase the tolerance of the cord to ischemic insult [28].

3.2.1 Distal perfusion techniques

Deterioration of blood flow to the spinal cord and abdominal viscera contributes significantly to the development of ischemic complications. Distal perfusion techniques perfuse the abdominal aorta during the period of aortic cross-clamping, which permit blood supply to the spinal cord via the intercostal, lumbar, and hypogastric vessels. Although distal perfusion techniques were used by some surgeons in the 1960s , they were not widely adopted because the results were variable, such that some surgeons suggested the inefficacy of the technique due to the greater rate of incidence of paraplegia [29]. Although the clamp-and-sew technique is used successfully in most cases [30,31], several studies confirm the need for an additional protective measure if the aortic cross-clamp time is longer than 30min [31,32]. Katz et al.[33] reported a 71% incidence of spinal cord injury in patients with disease of the descending thoracic aorta and cross-clamp times longer than 30 min. To date, abundant experimental and clinical evidences reveal that these techniques do reduce the incidence of paraplegia compared with the simple clamp-and-sew approach. Notably, data from studies that concurrently employed distal perfusion and clamp-and-sew procedures have demonstrated that the exponential rise in paraplegia rates when clamp times exceeds 30 min with clamp-and-sew technique alone does not occur [34]. Several techniques have been advocated for the perfusion of the distal aorta, such as femoro-femoral bypass, passive shunts, and left heart bypass.

3.2.1.1 Passive shunts

Historically, the use of the passive shunt was the method of choice for distal perfusion. During the 1960's, the Gott shunt tube was used as a passive shunt [35]; however, according to two published reports of traumatic tear of the thoracic aorta, the Gott shunt did not decrease the incidence of paraplegia from that associated with the clamp-and-sew technique [36,37]. Moreover, the distal aortic perfusion pressure necessitated to be greater than or equal to 60mmHg in order to minimize spinal cord injury, which made the Gott shunt less desirable than techniques that allowed the flow to be actively maintained. The shunt was too small with an internal diameter of 5–6 mm; thus the blood flow was not adequate for distal aortic perfusion, and the proximal aorta could not be adequately decompressed. These problems led to the development of left heart bypass or partial cardiopulmonary bypass.

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3.2.1.2 Left heart bypass

The centrifugal pump is used for left heart bypass [38], and provides the best means of maintaining distal aortic perfusion. The left heart bypass is set up with cannulation of the left atrium or left pulmonary vein. Blood is returned to one of the femoral arteries for distal perfusion. Minimal heparinization is needed, but it is recommended especially for patients with femoral occlusive disease. With the use of the pump, the distal aortic perfusion can be maintained at 60–70 mmHg. According to Kaplan et al.,[39] active distal bypass perfusion achieved significantly greater distal aortic pressure than either the clamp and sew technique or passive shunting.

3.2.1.3 Partial cardiopulmonary bypass

To overcome the limitation of poor oxygenation of the left heart bypass, partial cardiopulmonary bypass including the artificial lung and blood reservoir in the circuit has been utilized mainly in Japan. With the aid of heart-lung bypass and full heparinization, intraoperative shift from normothermic bypass to deep hypothermia was easily achieved, and unrestricted blood aspiration was possible. Detrimental effects of prolonged cardiopulmonary bypass (CPB), including heparin-related bleeding, cannot be ignored though [40].

3.2.2 Cerebrospinal fluid drainage

Blaisdell and Cooley[41] and Miyamoto et al.[42] reported that CSF drainage was beneficial for reducing the incidence of spinal cord injury in a dog model. It was shown that the relative spinal cord perfusion pressure increased with the CSF drainage, which led to reduction of spinal cord injury. McCullough et al.[43] also reported the protective effect of CSF drainage in reducing the incidence of paraplegia in a canine model. The combined effects of decreased arterial pressure and increased CSF pressure during aortic crossclamping resulted in decreased spinal cord perfusion pressure. The perfusion pressure can be maintained by decreasing CSF pressure through CSF drainage. Based on the results of these experiments, the concept of CSF drainage has been applied clinically. Crawford et al.[44] performed a prospective randomized study on the effectiveness of CSF drainage for preventing paraplegia and reported that it was not beneficial in this regard. Conversely, a more recent prospective randomized study of CSF drainage by Coselli et al.,[45] and a report by Safi et al., [46] showed that CSF drainage did help to prevent spinal cord injury. Many reports regarding reversal of paraplegia by commencing CSF drainage after surgery or endovascular stent-grafting also substantiated the importance of lowering the CSF pressure.

The use of CSF drainage as a therapeutic measure for delayed-onset paraparesis or paraplegia after open or endovascular repair is more accepted because there are several published case reports and anecdotal accounts of successful reversal of paraplegia by employing CSF drainage [47,48]. Wada et al.[49] manipulated the mean arterial and CSF pressures intraoperatively and found that ischemic SSEPs normalized when a combination of CSF drainage and arterial pressure manipulation was used to obtain a spinal perfusion pressure above 40mmHg. On the basis of their data, spinal perfusion pressure should always be maintained above 40mmHg, which confirmed previous similar observations from animal studies. Manipulation of spinal perfusion pressure assumes greater importance in patients with respiratory compromise, as autoregulation of spinal blood flow is lost with

hypoxia and hypercarbia, thereby, making spinal blood flow more sensitive to changes in perfusion pressure [50].

In view of these encouraging clinical results, CSF drainage has been incorporated as one of the most important components in the modern multimodality approach to spinal cord protection. Since not all cases of spinal cord ischemia are accompanied by increased CSF pressure; however, CSF drainage alone cannot be relied upon to prevent or reverse paraplegia and should be regarded as part of a multimodality approach to the prevention of spinal cord injury. Most importantly, this procedure is not immune to serious complications, such as intracranial bleeding, perispinal hematoma, and meningitis.

3.2.3 Systemic hypothermia

Systemic hypothermia is the most reliable protective adjunct for the prevention of spinal cord injury and is used by many surgeons [51,52]. Hypothermia is one of the most promising methods for protecting neural tissue during ischemia with the advantage that even longer periods of ischemia are tolerated compared with normothermic techniques. Because ventricular fibrillation or severe bradycardia is invariable with profound hypothermia, total body circulatory arrest is necessarily a component of this technique. Experimental work has shown that during the periods of aortic cross-clamping, hypothermia confers a protective effect on spinal cord function [53]. Hypothermia increases the tolerance of neural tissue to ischemia [54,55] by decreasing oxygen demand and metabolic rate, and mild hypothermia confers a marked protective effect on the spinal cord [56]. Kouchoukos and Rokkas reported an 8% 30-day mortality with a paraplegia or paraparesis rate of 2.8%. They concluded that the use of hypothermic CPB and circulatory arrest provided substantial protection against paraplegia and allowed complex operations on the descending thoracic and thoracoabdominal aorta to be performed safely [57]. Depending upon the extent of aortic replacement and vessel reimplantation, the whole procedure may be undertaken during circulatory arrest, or, for more extensive thoracoabdominal procedures, circulation is resumed after completion of the proximal and intercostal anastomoses. The advantages of this approach, in terms of spinal protection, are more uniform cooling of the cord, avoidance of the need for selective intercostal or visceral perfusion, and ability to perform open aortic and intercostal anastomoses; thus, potential steal phenomenon can be avoided. Okita et al. reported the clinical advantages of using deep hypothermia in patients for thoracoabdominal repair [58]. The main candidate for deep systemic hypothermia is a good-risk patient with less blood reserve in the spinal cord, such as one with a prior aortic replacement, severe atherosclerosis, or chronic aortic dissection.

On the other hand, the employment of CPB necessitates full heparinization; wherein, hypothermia itself may cause coagulopathy. The resultant intrabronchial bleeding in the left lung is then problematic. Furthermore, the requirement for full CPB and profound hypothermia introduces additional new problems and potential complications, including cardiac dysfunction (due to ventricular distension during cooling), brain injury, and possibly higher infection risk. Various results in the literature are demonstrated, and there are several small series reporting the high morbidity and mortality associated with this technique [59]. For this reason, most surgeons reserve deep hypothermic circulatory arrest techniques for only the most complex cases.

3.2.4 Regional cooling

Apart from systemic hypothermia, regional hypothermia has been used for spinal cord protection. Experiments have shown that regional hypothermic perfusion applied to the epidural or intrathecal space may protect the spinal cord during cross-clamping of the aorta [60]. Direct cooling of the spinal cord has been applied in both the laboratory and clinical settings, and has the theoretical advantage of deep cooling of the spinal cord whilst avoiding the drawbacks of profound systemic hypothermia.

In 1961, Albin et al. demonstrated the effect and safety of regional spinal cord cooling [61]. In 1993, Tabayashi et al.[62] and Marsala et al.[63] evaluated the effect of spinal cord cooling during spinal cord ischemia and reported its usefulness in preventing ischemic spinal cord injury. Davison et al.[64] devised and applied this method in eight patients undergoing thoracic or thoracoabdominal aneurysm repair in 1994 and reported that epidural cooling was a safe and effective technique of increasing the ischemic tolerance of the spinal cord.

The most systematically applied in the clinical setting has been the technique of Cambria et al.[65]; in which, normal saline at 4°C is continuously infused into the epidural space through a catheter. Using epidural cooling with CSF drainage, segmental artery reimplantation, and almost exclusive use of a clamp-and-sew technique without distal bypass (98% of patients), Cambria et al.[66] reported a paraplegia rate of 2% in 170 cases. The major risk associated with this approach is a potential for an increase in CSF pressure; hence, the necessity for CSF pressure monitoring and drainage is described. Their data show that epidural cooling is an effective method of spinal protection and may offer an alternative to distal bypass. Whilst regional cooling has been shown to be a safe alternative to distal perfusion in the majority of cases, it is not known whether it adds further protection if used in addition to distal perfusion.

3.2.5 Segmental artery perfusion

Some studies further attempt to reduce spinal cord ischemia by continuously perfusing the lower intercostal arteries. Experimentally, it has been demonstrated in pigs that segmental artery perfusion can protect the spinal cord for up to 60 min of ischemia [67]. In this study, the control group has simple aortic cross-clamping without distal perfusion, which is not reflective of the clinical scenario where adjuncts are frequently used. Selective spinal cord perfusion has been applied clinically utilizing a special cannulae [68] or through a Dacron graft [69].

Kawaharada et al. reported that trials to perfuse the critical intercostal arteries have been tried based on preoperative identification of the ARM [70]. Perfusion of the intercostal arteries from the study on the measurement of blood flow of the intercostal artery or lumbar artery with the use of transthoracic Doppler sonography was conducted [71]. They attempted to perfuse the intercostal arteries mainly through the Adamkiewicz artery for the purpose of maintaining spinal cord perfusion pressure during the cross-clamping of the aorta. By this technique, total blood flow quantity in the spinal cord becomes equal to or more than a certain constant level. Selective spinal perfusion maintains the quantity of total blood flow in the spinal cord and is very useful for reducing the incidence of ischemic injury of the spinal cord during operation.

3.2.6 Spinal cord steal syndromes

Steal is one concept that unifies most of the various successful strategies for reduction of paraplegia rates. When the aneurysm is opened, free back-flow of blood from the patent

intercostal arteries is usually observed with retrograde flow into the operating field through the opened intercostal and lumbar vessels, instead of going through the ASA due to a steal mechanism [72].

The various collaterals of the spinal cord arterial supply mean that blood can be diverted toward or away from the spinal cord and toward or away from other competing vascular beds in the cervical, thoracic, and lumbar/hypogastric regions. The importance of steal syndromes in the context of aortic surgery has been less appreciated. Although the possibility of steal as a cause of paraplegia after aortic resection was suggested by Cole and Gutelius [73] in 1969, it was largely unacknowledged until only recently. In the early 1990s, Wadouh et al.[74] revisited the concept of steal and suggested that spinal cord injury during aortic clamping resulted from a steal phenomenon. Using a pig model, they concluded that after aortic cross-clamping, blood had the tendency to drain away from the spinal cord than to supply it longitudinally. These experiments suggest that simple clamp techniques, especially where a proximal clamp only is applied, may result in more steal away from the cord compared with other approaches. Kawanishi et al. had experimentally demonstrated that control of the intercostal back-flow in the rabbit's opened aorta could reduce the incidence of spinal cord ischemia [75]. Using aortic injection and spinal artery perfusion in cadavers, Biglioli et al. [76] demonstrated the anatomical existence of a functional steal pathway; wherein, the blood was diverted from the spinal cord to the ARM during aortic clamping.

Another potential steal pathway is into the open thoracic cavity. If the aneurysm is opened prior to ligation of segmental arteries, back-bleeding is evident from the intercostal and lumbar orifices into the aorta. As there is no resistance to blood flow from these segmental vessels, blood from the anterior spinal artery will preferentially bleed out into the thorax through the ARM; thus, the spinal ischemia that has already resulted from the loss of intercostal supply from the excluded aortic segment is compounded [77]. The clinical existence of spinal steal as a possible cause of neurological injury is supported by the relatively low incidence of paraplegia in endovascular stent graft procedures, which entirely avoid steal phenomenon. Thus, external clamping of the intercostal arteries before opening the aneurysm or intraluminal insertion of a balloon-tipped catheter into the intercostal arteries after opening the aneurysm is a routine procedure to minimize blood reflux from the intercostal arteries. It seems prudent therefore to include measures for prevention of steal in spinal protection strategies.

3.2.7 Management of segmental arteries

3.2.7.1 Reattachment of the intercostal and lumbar arteries

Reattachment of segmental arteries after reconstructive surgery of the aorta is a controversial subject. A markedly lower incidence of spinal cord ischemia after endovascular stent-grafting stimulated the controversy, as well as a search for insights regarding the etiologies of spinal cord ischemia [78]. Some surgeons avoid intercostal reattachment. In fact, Acher et al.[79] demonstrated excellent operative results without reattachment of intercostal arteries. They reported that quick oversewing of the intercostal arteries with CSF drainage and naloxone administration could help reduce the incidence of spinal cord injury. Griepp et al.[80] also showed that reattachment could be avoided by ligating the intercostal arteries before aortic cross-clamping while monitoring somatosensory evoked potential. These reports suggest that existing collateral vessels might

improve the perfusion pressure. However, in 2008, Acher et al. [81] reported their clinical experience with thoracoabdominal aorta repair and the impact of intercostal artery implantation, in which the incidence of paraplegia decreased from 4.83% to 0.88% for neuroprotective strategies. The concept of oversewing segmental vessels with appropriate monitoring of the spinal cord is based on the fact that many clinical studies demonstrate the increasing rate of postoperative paraplegia with the increase in cross-clamping time. Time might be saved by not reattaching the noncritical segmental arteries. However, when a large number of intercostal and lumbar arteries are oversewn, the risk of neurological complications may be increased. Some surgeons attempt to identify the critical segmental vessels and selectively reimplant them. Traditionally, which vessels are important is decided intraoperatively by observing the intercostal arteries and reimplanting the larger vessels and those with greatest back-bleeding. Other surgeons base their decisions on the extent of resection; in which, vessels are reimplanted only during extensive thoracoabdominal resections. Anatomical studies have, however, shown no correlation between the size of the intercostal arteries and their likelihood of feeding the ARM [11]. The assumption that the arteries with greatest back-bleeding should be implanted is also flawed, since the presence of bleeding after aortic transection implies that a vessel is well collateralized and is effectively stealing blood retrograde from the spinal cord; hence, such vessels can be ligated without consequence. In contrast, vessels that do not back-bleed suggest a lack of collateralization and their reimplantation may improve spinal circulation.

Most surgeons who advocate selective reimplantation do not rely on intraoperative assessment but undertake preoperative angiography to localize the ARM. Preoperative localization helps target intercostal reimplantation, such that only a few intercostal arteries are reimplanted. Preoperative detection of an intercostal artery that may be related to the ARM is useful for establishing the best operational strategy for thoracoabdominal aortic aneurysm repair because surgical repair can be performed while taking care to revascularize the intercostal and lumbar arteries at or near the level of the ARM. Consequently, the occurrence of spinal cord injury can be reduced.

With this reconstruction method, the operation time, distal perfusion time, and clamp time needed for thoracoabdominal aortic aneurysm repair may be reduced. However, there is still no prospective randomized study that shows a significant reduction in the risk of postoperative paraplegia through reattachment of the segmental arteries. With the refinement of techniques in identifying the critical intercostal arteries and monitoring of evoked potentials, selective reattachment of the critical segmental arteries may be achieved.

3.2.7.2 Sacrificing of the intercostal and lumbar arteries

Systematic sacrifice of the intercostal vessels has been employed by Griepp et al.[80] and Gala et al.[82]. Intercostal reimplantation is not an integral part of their technique and is only undertaken if evoked potentials suggest spinal ischemia when intercostal arteries are occluded. Vessels are occluded thrice every 10 min, after which motor and sensory potentials are recorded. If the evoked potentials remain normal after 5 min of occlusion, then these vessels are sacrificed. The advantages of the Griepp approach include reduction of aortic cross-clamp time (and hence decrease in the overall duration of spinal ischemia), diminution of steal (as all intercostals are ligated prior to aortic transection), and prevention of unnecessary reimplantation of intercostals (with its attendant risks). The postoperative spinal circulation is more predictable and is not subject to abrupt changes resulting from problems with intercostal reimplantation (such as acute occlusion from thrombosis, which

has been postulated as one mechanism for delayed-onset paraplegia). Other potential advantages are the avoidance of technical problems associated with performing anastomoses or oversewing intercostals in a severely atherosclerotic aneurysm and the absence of residual aortic tissue in the replaced segment of the aorta.

Sacrificing of the intercostal inflow to the spinal circulation, however, means that the part of the thoracic cord is totally dependent upon extrasegmental supply and inflow from the cervical and hypogastric/lumbar arteries postoperatively. The spinal perfusion pressure, therefore assumes greater importance as the principal determinant of spinal blood flow to critical regions. For this reason, evoked potentials are monitored until the patient is awake and can be evaluated neurologically. Mean arterial pressures are kept in a supranormal range (80-90 mmHg mean) and, with concomitant CSF drainage, cerebrospinal pressures are kept below 10 mmHg. Sustained hypotension in the absence of the intercostal inflow will almost certainly result in paraparesis, which, if untreated, could develop into paraplegia.

4. Conclusions

Reconstructive surgery of the thoracoabdominal aorta remains a challenging surgical procedure with a recognized incidence of postoperative neurological complications. The complications pose not only the physical disability described but also a higher mortality rate among patients. The etiology of the aforementioned postoperative neurological problems has now been well described, and attempts have been made to reduce the incidence based on our knowledge of the pathophysiology of spinal cord ischemia. In open repair, recognized important strategies that reduce the risk of paraplegia include maintenance of the total amount of blood flow of the spinal cord during the cross-clamping of the aorta, provision of maximum collateral blood flow, reduction of nervous tissue oxygen demand, prolongation of ischemic tolerance of the spinal cord, and reduction of reperfusion injury. However, understanding the development and prevention of spinal cord complications is a prerequisite for a successful surgery on the thoracoabdominal aorta.

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Diagnosis, Screening and Treatment of Abdominal, Thoracoabdominal and Thoracic Aortic Aneurysms Edited by Prof. Reinhart Grundmann

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This book considers mainly diagnosis, screening, surveillance and treatment of abdominal, thoracoabdominal and thoracic aortic aneurysms. It addresses vascular and cardiothoracic surgeons and interventional radiologists, but also anyone engaged in vascular medicine. The high mortality of ruptured aneurysms certainly favors the recommendation of prophylactic repair of asymptomatic aortic aneurysms (AA) and therewith a generous screening. However, the comorbidities of these patients and their age have to be kept in mind if the efficacy and cost effectiveness of screening and prophylactic surgery should not be overestimated. The treatment recommendations which will be outlined here, have to regard on the one hand the natural course of the disease, the risk of rupture, and the life expectancy of the patient, and on the other hand the morbidity and mortality of the prophylactic surgical intervention. The book describes perioperative mortality after endovascular and open repair of AA, long-term outcome after repair, and the cost-effectiveness of treatment.

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