Chapter 37

Spinal Cord Protection during Operations of the Thoracic Aorta

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Over the past three decades, much research has focused on the pathophysiology of spinal cord ischemia and methods of protecting the spinal cord during and after surgery. This research, particularly the animal studies, has been reviewed extensively by this author and others. In essence, there are three major events that contribute to spinal cord injury: (1) the degree and duration of spinal cord ischemia during the period of aortic cross-clamping; (2) failure to re-establish spinal cord blood flow during the period of aortic cross-clamping; and (3) postoperative damage to the spinal cord that, at the molecular level, involves complex biochemical cascades of events during the periods of ischemia and reperfusion of the spinal cord. In addition, postoperative events such as hypotension and respiratory problems are important contributing factors to delayed injury.[1–44]

Degree and Duration of Ischemia

Multiple studies show that degree and duration of ischemia are most critical in the development of postoperative spinal cord injury.[1,2,5,7–10,20,24–26,30–32,35,37,40,42,43] There is general agreement that the degree of ischemia is dependent on both the extent of aortic repair and available collateral blood vessels. Indeed, coarctation of the aorta, in which situation there is an extensive network of collateral blood supply, is associated with the lowest risk of postoperative spinal cord injury. In contrast, the extensive Crawford type II thoracoabdominal aneurysm repairs that involve replacement of the aorta from the proximal descending aorta to below the renal arteries are associated with the greatest risk.[9]

Multiple studies show that there is a very strong relationship between aortic cross-clamp time and the risk of neurologic injury, particularly when various protective measures are not used. Thus, performing an expeditious and efficient repair that minimizes the period of aortic cross-clamping continues to be of paramount importance in protecting the spinal cord. As part of this approach, this author and colleagues advocate a method of sequentially and segmentally repairing the aorta that maintains a maximal perfusion of the spinal cord during the period of repair.[1,5,24] Thus, only the proximal part of the descending aorta is clamped off when doing an extensive thoracoabdominal aneurysm repair with distal perfusion of the aorta maintaining intercostal and lumbar artery blood flow up to the proximal descending aorta that is clamped off. After the proximal anastomosis is completed, the clamps are moved further down (typically to above the celiac artery) and the intercostal arteries are then reattached. The proximal descending aorta clamp is then removed and the intercostals reperfused while the segment below the celiac artery clamp is then opened and repaired. This approach effectively maximizes the period of perfusion of the spinal cord during repair. Then, after the visceral segment has been repaired in the type II thoracoabdominal aneurysms, the segment is reperfused and the lower part of the aorta is subsequently repaired. After the distal anastomosis is completed, the patient is rewarmed with distal perfusion. Recently, we reported, in a prospective randomized study based on multivariable analysis, that when using the protective measures and a sequential segmental repair, the intercostal ischemia time was the best predictor of spinal cord injury.

Decreasing the Degree of Ischemia

A useful approach for reducing the degree of ischemia entails maintaining adequate blood flow to the spinal cord as much as possible while simultaneously lowering the metabolic activity of the spinal cord by using hypothermia. The distal perfusion techniques include the use of atriofemoral bypass or perfusion from the left inferior pulmonary vein to the femoral artery with centrifugal pump bypass. Femoral–femoral partial or total cardiopulmonary bypass can also be used, although these are usually reserved for those patients undergoing redo surgery or who have particularly poor pulmonary function from chronic pulmonary disease. Hypothermia can be systemically induced by using atriofemoral bypass or femoral–femoral bypass with a heat exchange in the system, or it can be done locally with epidural cooling, as advocated by Cambria and associates, or by topical cooling of the spinal cord with intrathecal cooling.[15,16,20,43]

Normothermic Distal Perfusion

In a study of 832 descending aorta repairs, in the group of patients who had atriofemoral bypass (247 patients), the incidence of paraplegia/paresis was 6%.[8] The mortality rate was 4%, versus 9% without atriofemoral bypass ($p < .05$). The incidence of acute renal failure was also reduced to 4%, as compared to 8% without atriofemoral bypass ($p < .05$). Furthermore, in a study of 1,509 thoracoabdominal aneurysm repairs, in the 258 patients who had atriofemoral bypass, the incidence of paraplegia was 14%, versus 22% in those patients who did not have atriofemoral bypass ($p = .0025$).[9] For the thoracoabdominal aneurysms, the incidence of acute renal failure was also lowered to 13% in those patients who had atriofemoral bypass, versus 19% in those patients who did not have atriofemoral bypass ($p = .032$). Borst reviewed his experience with 132 patients who underwent atriofemoral bypass with normothermia and reported a 3% mortality rate and a 2.3% paraplegia/paresis rate.[36] Thus, atriofemoral bypass alone appears to have a protective effect, even if done at normothermia.

Hypothermia

Induced hypothermia, typically with deep hypothermia and circulatory arrest, was one of the early techniques used for protecting the spinal cord.[1] This author and colleagues prefer, however, to use active cooling to a moderate hypothermia range of 29° to 32°C based on the method of Swan-Ganz blood temperature monitoring.[1–5] Furthermore, we give patients a lidocaine bolus and put them on a lidocaine drip prior to inducing hypothermia to reduce the risk of any ventricular arrhythmias. In a prospective randomized study of Crawford type I and type II patients, we showed that active cooling with atriofemoral bypass to a moderate hypothermia level was associated with a reduced risk of paraplegia/paresis.[5] In a recent analysis of active cooling with moderate hypothermia and atriofemoral bypass in 72 patients, 3 patients developed a postoperative deficit (1 developed paraplegia; 2 developed paraparesis). Similarly, von Segressor found that active cooling and moderate hypothermia were associated with a reduced negative event rate postoperatively, including paraplegia, death, and surgical revisions.[31]

The use of deep hypothermia with circulatory arrest is usually reserved for complicated cases, such as those patients with distal arch involvement or rupture of the aorta associated with multiple previous descending or thoracoabdominal aorta operations or with intraoperative adverse events. Crawford and colleagues, in their study of deep hypothermia and circulatory arrest through the left chest, reported a 16% mortality rate, a 57% incidence of pulmonary complications, strokes in 9.5%, and paraplegia in 11%.[13] Similarly, Safi and colleagues, using the same technique in high-risk patients, reported a 29% mortality rate and reported that 67% developed pulmonary complications.[32] Of note, encephalopathy was noted in 33% of patients, stroke in 13%, and paraplegia/paresis in 13%.

In contrast, Kouchoukos and colleagues reported excellent results with the more routine use of deep hypothermia with circulatory arrest.[12,33] In a 152-patient series, the 30-day mortality rate was 6.6%, with a 2.7% incidence of paraplegia/paresis; in addition, only 2% developed strokes. Of note in this series, of those patients who underwent emergency operations, the mortality rate was 40%, and for type II thoracoabdominal aneurysms that included emergency surgery, it was 80%. This high mortality rate for emergency thoracoabdominal surgery has been well documented. In one study from Japan, the mortality rate within 3 months of emergency thoracoabdominal aneurysm surgery was 95%. In this author and colleagues' own experience with selective use of profound hypothermic circulatory arrest in 29 patients for aortic arch and/or descending or thoracoabdominal aneurysm repairs, the postoperative mortality rate was 10.3% and the spinal injury rate was 3.6%. Our series included patients who had the entire thoracic or entire aorta replaced in a single operation, extending from the aortic valve down to the celiac artery or to below the bifurcation. By logistic regression analysis we found no significant difference in spinal cord deficits according to the intercostal ischemia time when comparing active cooling with either moderate hypothermia or hypothermic arrest.

The problem following thoracotomy and the use of deep hypothermic circulatory arrest of a high risk of encephalopathy and neurocognitive deficits after surgery was reported in a previous series.[13,32] A possible reason for this could be that air is being trapped in the heart or aortic arch during the repair, which air then embolizes to the brain when reperfusion of the brain recommences with reestablishment of normal cardiac rhythm. Similarly, material in the descending or thoracoabdominal or dissection may embolize during the dissection or femoral perfusion, to cause neurologic injury. A method that this author and colleagues have found useful for establishing cardiopulmonary bypass in these difficult and complex operations is to anastomose an 8 mm tube graft to the right subclavian artery for arterial inflow perfusion.[44] We then place a long venous drainage catheter through the right femoral vein into the right atrium, using transesophageal echocardiography to ensure correct placement of the tip of the drainage cannula. The advantage of this technique is that the arterial inflow of the right subclavian artery can be used for antegrade brain perfusion during a period of circulatory arrest and for flushing out any potential embolic material from the aortic arch. In addition to this method, we routinely flood the field with carbon dioxide at 10 L/min so that any potential air gathering in the heart or aorta is displaced, further reducing the risk of serious air embolization to the brain. Indeed, since we began using this approach, we have been able to reduce the incidence of strokes and neurocognitive deficits.[10]

Cerebrospinal Fluid Drainage with or without Intrathecal Papaverine

The use of cerebrospinal fluid (CSF) drainage was first advocated by Blaisdell and Cooley in the 1960s.[22] Much animal research was performed with varying results as to the effectiveness of CSF drainage.[1–17] In animal studies this author and colleagues did in baboons, we were unable to show that CSF drainage alone during clamping protected the spinal cord, although with the addition of intrathecal papaverine the spinal cord blood flow was improved and paraplegia/paresis was prevented in this group of animals.[17] Also, our measurement of the anterior spinal artery size in these animals showed significant dilation of the vessel, which accounted for improved blood flow to the spinal cord.

In 1991, we performed a prospective randomized study of CSF drainage, although this was limited to a maxium of 50 cc of drainage during a period of aortic cross-clamping and the pressure was kept at 10 mm Hg.[24] Of note, postoperative CSF drainage was not used in this study. This study failed to show any immediate benefit in humans when using CSF drainage with the described protocol, although the incidence of delayed spinal cord injury appeared to be reduced in this group of patients when episodes of hypotension or respiratory failure occurred ($p = .08$). Subsequently, two prospective randomized studies were performed, one by us and one by Coselli and colleagues, that show that for Crawford type I and type II thoracoabdominal aneurysms, CSF drainage significantly reduces the incidence of deficits.[5,35,42] In our study, we also used intrathecal papaverine prior to aortic cross-clamping. During the period of aortic cross-clamping, CSF was allowed to drain freely as long as it was not bloodstained; after aortic clamping, the CSF drainage was stopped until the patient arrived in the intensive care unit. In the intensive care unit, CSF drainage was continued if the CSF pressure exceeded approximately 7 cm of water so that a rate of drainage of approximately 15 to 25 mL an hour was enabled. In that prospective randomized study, the incidence of permanent deficits was 3%. The study by Cosselli documented similar results for type I and type II thoracoabdominal aneurysms. Furthermore, Safi and colleagues advocate the combination of CSF drainage with distal aortic perfusion at normothermia, and in a recent study of descending aortic repairs, the occurence of deficits was 0.9% (1 of 105 patients), and for their thoracoabdominal type I and type II, the incidence was 6% (14 of 239 patients).[7,30,32,39]

Thus, based on our own studies and on the reports by Safi and Coselli, we recommend CSF drainage both during the period of aortic cross-clamping and postoperatively. Indeed, in our study we continued to drain CSF postoperatively for 40 to 48 h after surgery. Of particular note, we and others have found that when delayed paraplegia/paresis occurs, the reinsertion of a catheter for CSF drainage may reverse the incidence of delayed deficits.

For the type I and type II thoracoabdominal aneurysms, the incidence of neurologic deficits was significantly reduced to 3% with CSF drainage and intrathecal preservative papaverine. Although this author and colleagues do not have a prospective randomized study specifically assessing intrathecal papaverine, based on our animal studies, we believe that intrathecal papaverine, has an additive protective effect on the spinal cord, including improving collateral blood flow down the longitudinal vessels on the spinal cord.[1,17] In our first study of intrathecal papaverine in a pilot study of 34 patients, the incidence of deficits was reduced to 2.9%. The one event was related to a delayed deficit. In a more recent evaluation of 61 patients treated with CSF drainage and intrathecal papaverine, 3 patients developed permanent paraplegia/paresis (4.9%). We have also done animal studies using intrathecal perfusion of the spinal cord with cold Ringer lactate and added papaverine to prevent any spinal cord spasm. In the porcine model that we used, the combination significantly reduced postoperative deficits. Cambria and colleagues took a different approach, and in a recent report of 170 patients, they found that using their method of epidural cooling reduced the incidence of spinal cord injury when compared to the historical controls.[20,43]

Finally, in our prospective randomized study showing the relationship of spinal cord injury to the time of aortic cross-clamping, the sigmoid curve shifted to the right with the use of either CSF drainage or intrathecal papaverine. It was shifted even further to the right with active cooling with atriofemoral bypass, and when the two methods were combined, this resulted in the greatest protective effect (Table 37-1).

Reestablishment of Spinal Cord Blood Flow

In 90% of patients the artery of Adamkiewicz arises between T6 and L1, and in 80% of patients the vessel arises from the left segmental intercostal or lumbar arteries.[1,17] While this is the largest of the radicula arteries that supply the spinal cord, the other thoracic radicula arteries are also important to spinal cord blood supply. The reason for this is that the other higher thoracic intercostal arteries that give off the thoracic radicula arteries supply the anterior spinal artery with blood flow up and down the length of anterior spinal artery. This differs from the artery of Adamkiewicz, which is also known as the arteria radicularis magna (ARM), because this artery takes a hairpin bend where it

TABLE 37-1. Techniques that Protect the Spinal Cord

1. Expeditious and efficient repair
   Sequential segmental repair
   Transection of the aorta
   Extensive use of second-stage elephant trunk technique
   Open anastomosis in selected patients
2. Distal perfusion
   Atriofemoral or left inferior pulmonary vein to femoral or partial or total cardiopulmonary bypass
3. Hypothermia
   Systemic
   Local
   Deep hypothermia with circulatory arrest
4. CSF drainage with or without intrathecal papaverine
5. Reattachment of all segmental arteries from intercostal T6 to and including L1
6. Prevention of postoperative hemodynamic instability or deoxygenation or respiratory problems
7. Consideration of pharmacologic agents
8. Consideration of monitoring techniques
   Motor-evoked potentials or spinal cord oxygen saturation

CSF = cerebrospinal fluid.

joins the anterior spinal artery and perfuses the segment of the spinal cord below it, typically the lumbar expansion of the spinal cord. In animal experiments, tying off the ARM has resulted in a 50 to 100% incidence of paraplegia or paresis, particularly in monkey experiments. In the experience of this author and colleagues, however, the ARM may not be quite so critical in spinal cord protection in humans. Rather, in humans, collaterals have developed to the spinal cord because the segmented intercostal or lumbar arteries have been occluded by dissection or clot. We believe that collateral blood flow is an even more important factor in protecting the spinal cord, particularly when dissection or clots within an aneurysm occlude intercostal arteries, especially the artery supplying the ARM. Hence, maintaining blood flow through the other thoracic and lumbar arteries is important, particularly because this can be a vital source of collateral blood flows, as we have documented in some of our studies by using hydrogen mapping of blood-flow patterns to the spinal cord.[4,14,20]

Although preoperative angiography to document the blood supply to the spinal cord has been attempted, there are several problems with this technique.[1] First, patients often require an anesthetic, which procedure has associated risks. Second, the time to do the mapping of the entire potential spinal cord blood supply is lengthy. Third, it is documented that paraplegia or paresis can be induced by the mapping of the spinal cord.[1] Finally, patients have died during the procedure from rupture of the aorta or from complications such as renal failure caused by the large dye load required to perform the mapping.[1] Thus, we do not advocate the use of preoperative angiography for assessment of blood supply for thoracoabdominal aneurysm repairs. Based on our cadaver dissections examining the spinal cord blood supply in humans, as well as on our studies in primates and the use of hydrogen mapping, we advocate that the critical vessels be reattached during surgery from T6 down to L2. In 98 patients undergoing type I or type II thoracoabdominal aneurysm repairs in whom we carefully examined the patency or occlusion of the segmental arteries, we found that the management of these vessels strongly influenced outcome postoperatively.[10] If patients had patent intercostal or lumbar arteries between the T10 and L1 segments that were not reattached during the time of the aortic repair, the incidence of paraplegia/paresis postoperatively was 63%, in contrast to a 25% incidence rate in patients in whom these vessels were present and reattached (p = .05). Similarly, studies by Ross and colleagues and by Safi and colleagues show a higher incidence of paraplegia/paresis when patent intercostal or lumbar arteries were not reattached at the time of surgery in these segments.[38,39]

For patients with descending aortic aneurysm, the importance of these vessels is not quite as clear. The reason for this is as discussed above; the radicula arteries that join the anterior spinal artery can perfuse the spinal cord both upward and downward. Thus, any vessels that are sacrificed during the repair may be compensated for from other radicula arteries and from segments above or below the segment that was repaired. Thus, collateral blood flow may continue to perfuse the anterior spinal artery adequately. This author and colleagues' research in animals, however, shows that if only the ARM is perfused, then the blood flow through this vessel does not protect the lower thoracic spinal cord. In a series of 132 descending aortic repairs, Borst found that all the paraplegia/paresis events that occurred were in those patients in whom the repair was continued to below T8.[36] Similarly, in our study of 832 patients undergoing descending aortic repairs, the patients who had the distal half of the descending aorta replaced had the highest risk of developing paraplegia or paresis.[8] In Estera and colleagues' study of 148 patients undergoing descending aortic repairs in combination with CSF drainage and distal aortic perfusion, the repair of the distal descending aorta was associated with the greatest risk.[30] Similarly, Griepp and colleagues found that clipping most of the descending intercostal arteries was safe, but they did note that the risk of paraplegia was increased with the increasing number of segmental intercostal or lumbar arteries being divided.[40] Based on this research in both animals and humans, we recommend that if the descending aorta is replaced below T8, then these vessels should be preserved.

The results of intraluminal grafting and the incidence of paraplegia/paresis are of interest.[6,41] Because these patients treated by stent grafts do not have significant ischemia during the insertion of the grafts, the incidence of paraplegia/paresis in these patients is largely dependent on the occlusion of intercostal vessels or lumbar arteries or the embolization of these vessels. Thus, just as we showed in 832 patients who underwent aortic repairs, the incidence of paraplegia/paresis increased when the abdominal aorta had been previously repaired.[8] The Stanford studies with intraluminal grafts found similar results.[6,41] In those patients in whom the lower descending aorta was occluded and who had previously undergone abdominal aneurysm repairs, the incidence of paraplegia/paresis was increased.

This author and colleagues have used intraoperative methods in an attempt to determine which vessels need to be reattached at the time of surgery so as to shorten the period of aortic cross-clamping. Our work with hydrogen mapping of the spinal cord showed that this method was accurate and did tend to reduce the period of aortic cross-clamping, although the equipment required is quite cumbersome and it is largely a research method.[4,14,26] Similarly, our research demonstrates that monitoring the spinal cord oxygen saturation with a platinum electrode alongside the spinal cord by using the polargraphic technique was also valuable. The oxygen saturation alongside the spinal cord drops within 30 to 90 s with aortic cross-clamping both in humans and in porcine experiments. With the reestablishment of blood flow to the spinal cord, this is rapidly corrected if the critical intercostal or lumbar arteries have been reattached.[1,4,14,20] This is a method that may have future prospects for monitoring adequate spinal cord blood-flow reestablishment. Our research also shows that, based on using postoperative highly selective angiography in some patients, the reattached intercostal or lumbar arteries clot off in the postoperative period. This is clearly a surgical technique issue that may account for some of the postoperative spinal cord injuries, particularly the delayed deficits associated with hypotension, as we have shown in the porcine model. One operative modification that we have undertaken to reduce this risk is that we no longer use catheters in the individual intercostal or lumbar arteries to prevent backbleeding. This should reduce the risk of injuries and the risk that rupture of these arteries will result in clot formation.

Prevention of Postoperative Hemodynamic Instability and Induced Hypertension

With the increasing success of spinal cord protection during the period of aortic cross-clamping, we have seen a greater proportion of delayed deficits post surgery even though the total number of events has been reduced. To reduce the risk of delayed deficits, we now routinely maintain our patients' postoperative blood pressure at a higher level, typically in a range above 80 mm Hg mean pressure. While this does increase the potential risk of postoperative bleeding, we believe this has contributed to a lower incidence of delayed deficits. In a recent analysis of 132 descending or thoracoabdominal aneurysm repairs, 6 patients developed a delayed deficit (4.5%). In these patients, the deficits were a result of hypotension or pulmonary complications that required the patients to be re-intubated. This further supports the use of CSF drainage for at least the first 2 days post-operation, which is when the patients are most likely to have hypotension or respiratory problems. In addition, extubation is delayed to the second postoperative day.

The Use of Medications

Many animal studies show that specific medications or agents may reduce the incidence of neurologic injury.[1,17] However, there are no successful prospective randomized studies in humans demonstrating that these agents are effective in reducing the incidence of spinal cord injury. Nonetheless, this author and colleagues did notice in one of our prospective randomized studies that patients who received lidocaine for myocardial arrhythmias during surgery tended to have a lowered incidence of postoperative paraplegia or paresis ($p = .1$), albeit not statistically significant.[24] Therefore, we continue to use lidocaine to protect the spinal cord. Animal studies also show that analogs of lidocaine are effective in protecting the spinal cord. In our animal studies, we have examined many agents for spinal cord protection, including superoxide dismutase, allopurinol, steroids, mannitol, flunarizine, naloxone, Selfotel, thiopental, anti-inflammatory agents, white-cell-poor blood transfusions, and angiotensin-converting enzyme blockers. None of these, however, have convincingly reduced the incidence of paraplegia or paresis. Undoubtedly, in the future, agents will be found that are effective in combination with the other documented methods to further reduce the incidence of postoperative paraplegia/paresis.

Conclusion

The incidence of paraplegia/paresis in patients with descending or thoracoabdominal aneurysms has declined significantly in recent years. This is largely a result of extensive research in this area using animal studies and prospective randomized trials in humans. For example, in a recent review of 132 of our patients who underwent descending or thoracoabdominal operations, the incidence of permanent deficits was reduced to 3.8%. This has also contributed to better early and long-term survival rates after surgery for this series of patients, with a mortality rate of 8.3%.

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