Review Article

Spinal Cord Blood Supply and Its Surgical Implications

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Abstract

The blood supply to the spine is based on a predictable segmental vascular structure at each spinal level, but true radiculomedullary arteries, which feed the dominant cord supply vessel, the anterior spinal artery, are relatively few and their locations variable. Under pathologic conditions, such as aortic stent grafting, spinal deformity surgery, or spinal tumor resection, sacrifice of a dominant radiculomedullary vessel may or may not lead to spinal cord ischemia, depending on dynamic autoregulatory or collateral mechanisms to compensate for its loss. Elucidation of the exact mechanisms for this compensation requires further study but will be aided by preoperative, intraoperative, and postoperative comparative angiography. Protocols in place at our center and others minimize the risk of spinal cord ischemia during planned radiculomedullary vessel sacrifice.

General Structure of Spinal Cord Blood Supply

The spinal cord is nourished by a structure of arterial supply that follows a generally predictable path from the great vessels to the parenchyma of the white and gray matter throughout the spinal cord (Figure 1). Segmental vessels originate from the great vessels of the neck, thorax, and abdomen at each segmental level and, with few exceptions, are paired bilaterally. Although the segmental vessel typically divides into an anterior and posterior ramus, the posterior ramus is the dominant vessel and divides further into a muscular branch and a spinal branch. The spinal branch becomes an anterior radicular artery and a posterior radicular artery as it traverses the neuroforamen alongside the segmental nerve root. There are 31 paired radicular arteries, one for each segmental level, but relatively few of them contribute meaningfully to the spinal cord. Most end within the nerve root, dura, or pial plexus.

The radicular arteries that do contribute to the longitudinally oriented single anterior spinal artery (ASA), and therefore to the anterior two thirds of the spinal cord parenchyma, are named anterior radiculomedullary vessels. Gao et al[1] demonstrated the presence of 72 of these vessels across 20 human specimens, for an average of 3.6 per specimen. Conversely, Martirosyan et al[2] cited an average of 10 (range, 2 to 17) throughout the spinal cord. Anterior radiculomedullary arteries are usually not paired at any given level. Their end recipient vessel, the ASA, is mostly in continuity throughout the entire cord[3,4] and experiences both anterograde and retrograde flow, depending on the functional demand and location of dominant radiculomedullary feeder vessels. The ASA gives off many central arteries, which project and often bifurcate in the sagittal plane,

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Figure 1

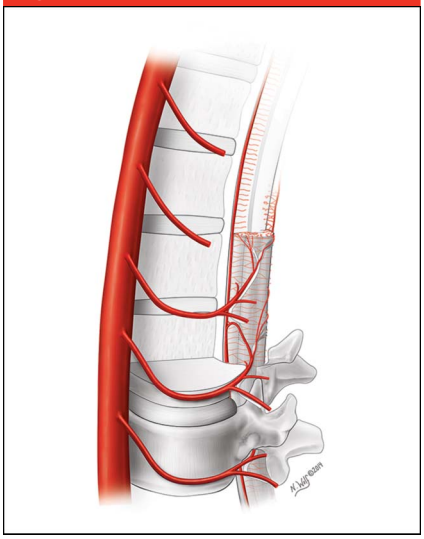

Illustration of typical spinal cord blood supply under normal conditions. One radiculomedullary vessel is shown, which supplies the anterior spinal artery through a hairpin loop.

heading to the center of the cord matter. These central arteries are most dense in the lumbosacral region, followed by the cervical and thoracic regions. These terminate in centrifugally oriented, inside-to-outside capillary beds, which are five times as dense in gray as in white matter.[5]

The posterior one third of the cord parenchyma is supplied by paired posterior spinal arteries (PSAs), which are fed by the posterior radicular arteries. This arterial supply is more similar to an arterial plexus than to the often-envisioned two longitudinal paired arteries.[6] Generally, these arteries are much smaller than the ASA and are less consequential in regard to ischemic functional deficits when disrupted. As opposed to the terminal branches of the anterior system, the PSAs supply the cord matter in a centripetal pattern. Most anatomic studies have not identified direct anastomotic connections between the anterior and posterior systems,[2] whereas others have observed a connection in certain anatomic areas, such as around the conus medullaris.[1] In any case, significant overlap and redundancy does occur between the terminal supply branches of these two systems within the parenchyma of the spinal cord itself.

Structure Unique to the Cervical Spine

The ASA in the cervical spine takes its origin from two intervertebral arteries at the very upper end of the cervical spine, where it is largest, and slowly tapers to a constant diameter for the remainder of the cervical region and into the thoracic region.[7] In the cervical spine, segmental vessels originate from the vertebral arteries in the upper portion and, in the remainder of the cervical spine, from the vertebral arteries and deep cervical, costocervical, or ascending cervical branches. The segmentals then give off anterior and posterior radiculomedullary arteries, the former of which supply the ASA, as in other regions of the spine. There are typically from one to several dominant unilateral anterior radiculomedullary vessels in the cervical spine.[1,2] Although the blood supply to the cervical spinal cord is generally thought to be redundant and well-collateralized, significant variation exists in the number of dominant supply vessels to the ASA, and their number and distribution should be well documented before considering surgical disruption.

Structure Unique to the Thoracic Spine

In the thoracic spine, the segmental vessels come from the aorta or the subclavian artery and continue on as intercostal arteries. The number of radiculomedullary arteries in the thoracic spine is fewer than in other areas (average, one to four), and they are more spread out.[3] There is poor collateral potential in this region, and there is virtually no direct communication between the central (anterior) and peripheral (posterior) systems.[4] The ASA of the thoracolumbar spine is fed by one or two anterior radiculomedullary vessels, the most dominant of which is termed the artery of Adamkiewicz (AA).[8] This feeder vessel of the ASA can be up to 1.3 mm in diameter and occurs on the left side between T9 and T12 in 75% to 80% of cases.[9,10] The AA gives off a dominant descending branch in a "hairpin" configuration and a much smaller ascending branch as it joins the ASA. The ASA is typically continuous throughout the thoracic spine, but its caliber is significantly attenuated, especially as it approaches the AA.[9,11-13] These factors are thought to be responsible for the sensitivity of the thoracic region to ischemic insult

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of the anterior part of the spinal cord.

Structure Unique to the Lumbar and Sacral Spine

The tip of the conus medullaris may be nourished by the descending termination of the ASA directly, but there also exists a highly collateralized anastomotic network at this location termed the anastomotic loop of the conus.[7] The lower lumbar and sacral radicular arteries contribute to this highly collateralized anastomotic network rather than to the more cephalad ASA directly. Thus, although ligating a single artery or multiple sacral or lumbar radicular arteries usually does not have ischemic consequences for the conus or the cephalad spinal cord, these vessels do supply end arterioles to these regions. In fact, particulate matter clogging these end arterioles has resulted in lower extremity paraparesis, such as with inadvertent intravascular particulate steroid injection in the lower lumbar or sacral spine.[14]

Spinal Cord Ischemia During Pathologic Conditions

Spinal cord perfusion pressure is simply a function of the distal aortic perfusion pressure (or mean arterial pressure) less the extrinsic cord pressure (or cerebrospinal fluid pressure). The blood supply to the spinal cord may be interrupted iatrogenically during many different types of surgical intervention, including aortic aneurysm repair, tumor resection, and spinal deformity surgery. The mechanism of spinal cord injury may involve global hypoperfusion, as with aortic cross clamping or systemic hypotension; selective ischemia from ligation of dominant segmental vessels; or secondary insult, such as with reperfusion injury. The clinical presentation of cord ischemia ranges from complete paraparesis to asymptomatic transient abnormalities detectable only by neuromonitoring. The key clinical question driving much of the anatomic research in spinal cord ischemia during surgical interventions is which key vessels must be preserved to maintain spinal cord perfusion pressure and therefore spinal cord blood flow to avoid functional deficits.

Thoracoabdominal Aortic Aneurysm Repair

Although thoracoabdominal aortic aneurysm (TAAA) repair does not have direct relevance for the orthopaedic surgeon, its related literature is the most robust repository available for reports of ischemic spinal cord damage resulting from vascular interruption. In fact, TAAA repairs have been described as "Russian roulette for the vascular surgeon"[15] because of a reported high incidence of spinal cord ischemia, with rates of 16% to 32%, depending on the location and extent of the aneurysm.[16,17]

The mechanism of cord ischemia is multifactorial but involves the extent of disruption of segmental vessels during aortic mobilization and aneurysm repair. Several studies have highlighted the importance of preserving the dominant anterior radiculomedullary artery to the thoracolumbar spine or the AA.[18,19] In one study of open TAAA repairs, preoperative angiographic visualization of the AA outside the repair or aortic clamp area resulted in no spinal cord injury. In addition, when the AA was located within the clamped section, microrevascularization of the AA during the open procedure resulted in a 5% rate of cord ischemia compared with 50% when revascularization was unsuccessful.[18]

However, because of the confounding effect of global hypoperfusion during aortic cross clamping, studies involving open TAAA repair may not be the best model for examining the anatomic causes of spinal cord ischemia. Endovascular techniques, such as thoracic endovascular aortic repair, which selectively excludes specific segmental vessels by way of intra-aortic stents, may be a better model for examining the effect of segmental vessel sacrifice on cord ischemia. In 71 patients undergoing TAAA endovascular repair, Kawaharada et al[20] observed less spinal cord injury in a group in whom the AA was not occluded by the stent (none) compared with the group in whom the AA was occluded (10%), but the difference was not significant, and the overall event rate was low (3.6%). Schurink et al[21] analyzed 13 patients undergoing stent grafting of aneurysms at or below T8, 8 of whom had four or more segmental levels occluded by the stent, and 6 of whom had the segmental artery feeding the AA itself occluded. The authors observed two cases of intraoperative transient motor evoked potential abnormality correctable by raising the mean arterial pressure, and no cases of postoperative paraplegia.[21]

Although these and other studies indicate that the AA can be safely and permanently occluded at its more proximal segmental supply, it is important to remember that the aortic and microvascular structures of patients with TAAA are chronically degenerative in nature and thus are not normal. Chronic occlusion of segmental vessels may allow time for the development of collateral systems that supply the radiculomedullary vessels, thereby making sudden occlusion less likely to cause a catastrophic ischemic event.[22,23] A more rigorous model of critical cord blood supply may be found in other clinical scenarios.

Spinal Deformity

Study of the neurologic effects of segmental vessel sacrifice demonstrates

that segmental vessels must be ligated for exposure during anterior thoracolumbar spine exposure and deformity correction; early reports raised concerns that sacrifice of segmental vessels supplying dominant radiculomedullary vessels can lead to paraparesis. Apel et al[24] reported on three cases of congenital kyphoscoliosis in which complete loss of somatosensory-evoked potential (SSEP) tracings was observed within 5 minutes after ligation of segmental vessels around the curve apex from T3 to T9. Each case resulted in postoperative paraparesis. This prompted the authors to institute a protocol in 44 patients whereby segmental vessels being considered for ligation were temporarily occluded before permanent ligation. In seven cases, SSEP amplitude changes of >50% were observed within 5 minutes of temporary occlusion of a segmental vessel at the curve apex; all of these returned to baseline within 5 to 19 minutes after removal of the occlusive clamp and preservation of the vessel. No cases of postoperative paraparesis were observed.[24]

A temporary occlusion protocol before permanent segmental vessel division has been advocated by other authors.[25] However, the importance of this has also been brought into question.[4,26,27] Bassett et al[4] studied 15 patients undergoing anterior spinal surgery for kyphoscoliosis. Using angiography, the authors identified 32 total dominant radiculomedullary vessels supplying the thoracolumbar ASA in their population. Nine of these vessels in eight patients were within the surgical field and were temporarily occluded with no SSEP changes. However, in seven of these eight patients, other radiculomedullary vessels outside the surgical field were not occluded. The eighth patient had no changes after occlusion of the one dominant segmental vessel supplying the lone radiculomedullary vessel, but it was located at L1, an area known for its rich perimedullary collateral supply.[4]

Another study retrospectively analyzed 1,197 patients who underwent anterior spinal surgery for kyphoscoliosis without using a regular temporary occlusion protocol before dividing segmentals. There were no cases of postoperative paraplegia. However, in one patient, revision surgery on the convex side of the deformity was undertaken after prior surgery on the concave side. In this patient, the authors did temporarily occlude the segmental vessels in the surgical field, and for one vessel, there were immediate SSEP changes; thus, the vessel was preserved.[26] In addition to standard neuroprotective measures, such as avoidance of intraoperative hypotension, these and other authors advise dividing segmentals as far from the neuroforamen as possible, dividing segmentals unilaterally on the convex side of the deformity only (which may play a lesser role in vascularization of the cord as a result of chronic vessel tension), and considering temporary occlusion or "soft clamping" when a thoracic-only deformity with intraspinal abnormality at the same levels is encountered.

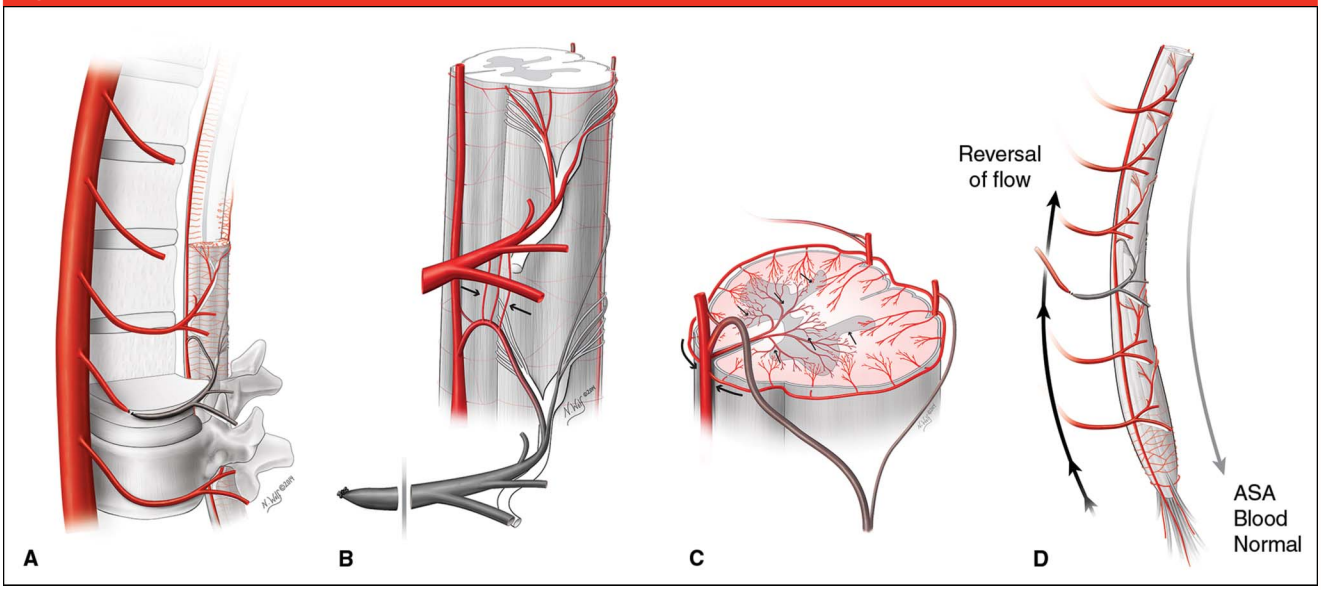
Oncologic Resection of Intraspinal Neoplasms

Resection of malignant neoplasms of the spinal axis using the technique of total en bloc spondylectomy presents a distinct challenge with regard to maintenance of spinal cord blood supply. Typically, a more extensive, often 360° exposure is required over at least two spinal motion segments to isolate the resection level for univertebral disease. Also, the tumor extent may make it impossible to safely preserve certain vessels, even if there is preoperative concern for their critical role in cord blood supply.

Based on an early report that up to four unilateral nerve roots and their accompanying radicular vessels can be sacrificed in dogs without cord ischemia,[28] Nambu et al[29] and Ueda et al[30] undertook extensive experimental work investigating the importance of preserving cord-supplying vasculature during total en bloc spondylectomy. In several initial reports, these authors showed only modest decreases (15% to 25%) in spinal cord blood flow without change in motor evoked potentials (MEPs) or spinal cord evoked potentials (SCEPs) after bilateral three-level segmental vessel ligation at T11-T13.[29,30] This area in dogs corresponds to the thoracic watershed area in humans where the AA is most often located. In follow-up studies that sought to define the limits of segmental vessel sacrifice in this area without effect on cord health, these authors observed blood flow to the cord dropping to less than half of normal only in dogs that had bilateral five-level (44%) and seven-level (25%) segmental vessel sacrifice; in addition, only in dogs with five or more bilateral levels ligated were any MEP, SCEP, or neurologic examination abnormalities detected.[31] In a refinement, the group studied segmental sacrifice over multiple levels that specifically included the canine equivalent of the AA based on preoperative angiogram; only dogs with greater than three-level bilateral segmental vessel ligations including the AA were abnormal with regard to MEP, SCEP, and neurologic examination.[32] It is on the basis of these studies that this group currently advocates for safe, bilateral three-level segmental sacrifice, including the AA, in humans.

These authors have retrospectively reported on their experience with 180 cases of total en bloc spondylectomy in humans; 15 had the AA involved at the resection level and underwent target vertebra preoperative embolization and three-level segmental vessel sacrifice, including the AA. There were no

Figure 2



A through **D**, Illustration of disrupted spinal cord supply following ligation of a key segmental artery (shown in gray) (**A**) with three possible compensatory mechanisms for reconstitution of the anterior spinal artery (ASA). Without direct supply to the ASA via the typical flow from the segmental artery to the radiculomedullary artery (RMA), the ASA may be reconstituted by collaterals emanating from an adjacent segment radicular artery (**B**), communication between the posterior spinal arterial system and the ASA system via the pial plexus and areas of spinal cord parenchymal overlap (**C**), or compensatory dynamic reversal of flow in the ASA itself using supply from distant RMAs or the anastomotic loop of the conus (**D**).

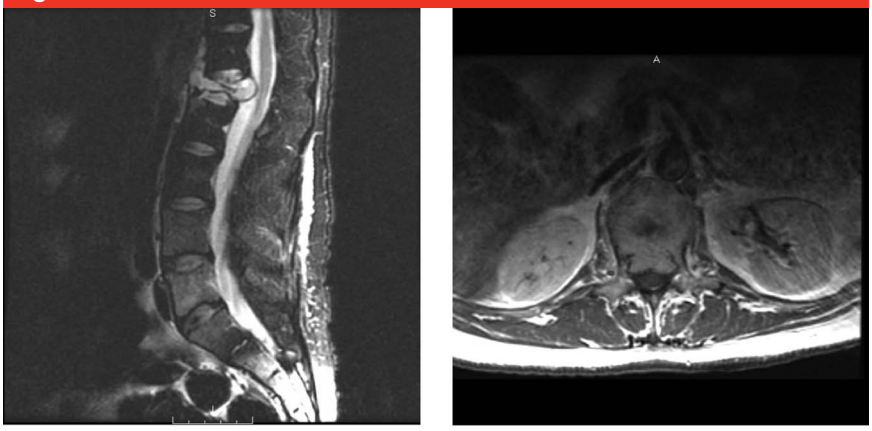
cases of neurologic deterioration or paraplegia after surgery.[33] Other authors have also suggested that ligating the AA may be safer than previously thought and have demonstrated postoperative development of new collaterals to the ASA after AA ligation.[34,35]

The explanations given for the somewhat counterintuitive finding of normal spinal cord function after ligation of the dominant anterior radiculomedullary vessel to the ASA are theoretical. Tomita et al[36] claim that there must be anastomotic communication between the posterior spinal artery and anterior spinal artery systems through the intercanal, dural, or pial plexus (Figure 2, C). Some authors have corroborated this in a limited fashion in anatomic dissections by demonstrating communication at the conus medullaris level,[1] whereas others have claimed that no functional communication exists.[2] Kawahara et al[37] have demonstrated that spinal shortening during total en bloc spondylectomy may increase blood flow to the cord. Other anatomic explanations are that proximal ligation of a segmental artery or even of an anterior radiculomedullary artery itself does not disrupt the afferent supply to the ASA because of preexisting, more distal contributions to the critical anterior radiculomedullary artery (Figure 2, B). These more distal contributions may not be seen on a preoperative angiogram because they are dynamically recruited only after the pathologic condition is initiated. Finally, it is possible that anterograde or retrograde flow dynamics through the ASA itself from distant cephalad or caudad anterior radiculomedullary vessels compensate for a given anterior radiculomedullary division (Figure 2, D).

Monitoring and Timing

Following the sacrifice of key spinal cord supply vasculature, the options for intraoperative monitoring of deficit include SSEPs, continuous electromyographic monitoring (EMG), MEPs, and the intraoperative Stagnara wake-up test. SSEP monitoring remains the preferred method and is unlikely to be affected by anesthetics, but because it predominantly monitors the dorsal columns, it is at best an indirect measure of the lateral corticospinal tracts and the motor neurons, which are supplied by the ASA. False-negative readings can occur, with rates as high as 9%.[38,39] Free-running or continuous EMG monitoring, like SSEP monitoring, is a good measure of individual nerve root irritation during surgery and has the benefit of detecting spontaneous nerve root activity in response to stretch or compression. In contrast to both, transcranial MEPs directly measure the anterior cord motor pathways. However, they are susceptible to the anesthetic effects of halogenated compounds, nitrous

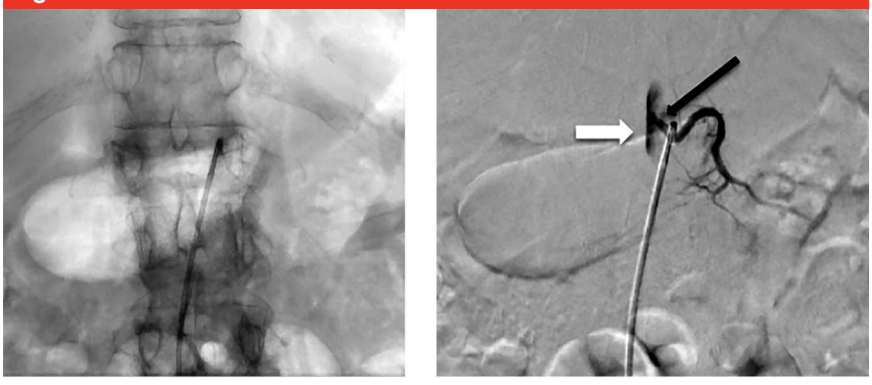
Figure 3



A B

Midsagittal T2-weighted (**A**) and axial T1-weighted (**B**) postcontrast magnetic resonance images of the spine in a 58-year-old woman with a history of small-cell osteosarcoma of the right hemipelvis depicting the tumor extent, with pathologic collapse of the vertebra and extraosseous epidural tumor extension. She presented with a solitary L1 metastasis 3 years after local and systemic treatment of her pelvic disease.

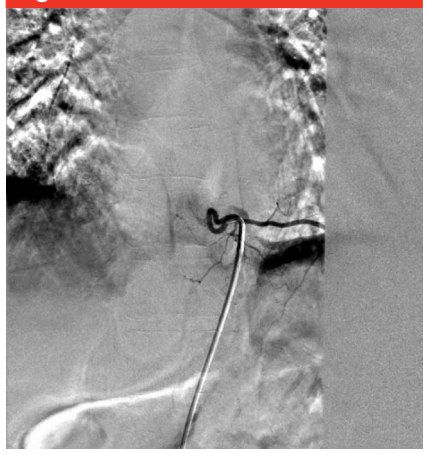
Figure 4



A B

A and **B**, Spinal arterial angiography images with overlying radiograph of the same patient shown in Figure 3, depicting the left-sided dominant thoracolumbar radiculomedullary artery, or artery of Adamkiewicz (black arrow). Filling of the anterior spinal artery is also seen (white arrow).

Figure 5



Spinal arterial angiography image with overlying radiograph depicting the comparatively minor contribution of a second radiculomedullary artery located on the left side at T10 in the same patient shown in Figures 3 and 4.

oxide, or neuromuscular blockade and are associated with complications from muscle contraction, intraoperative movement, and aberrant electrical signals affecting the brain or cardiac systems.[40] Most authors feel that a combined approach using SSEP and MEP is the safest configuration, with a sensitivity and specificity for detecting cord ischemia of 100% and 91%, respectively.[41] The Stagnara wake-up test is effective but used less commonly today because a relatively long lag time is required to reverse anesthesia and make an accurate determination of cord function clinically.[42]

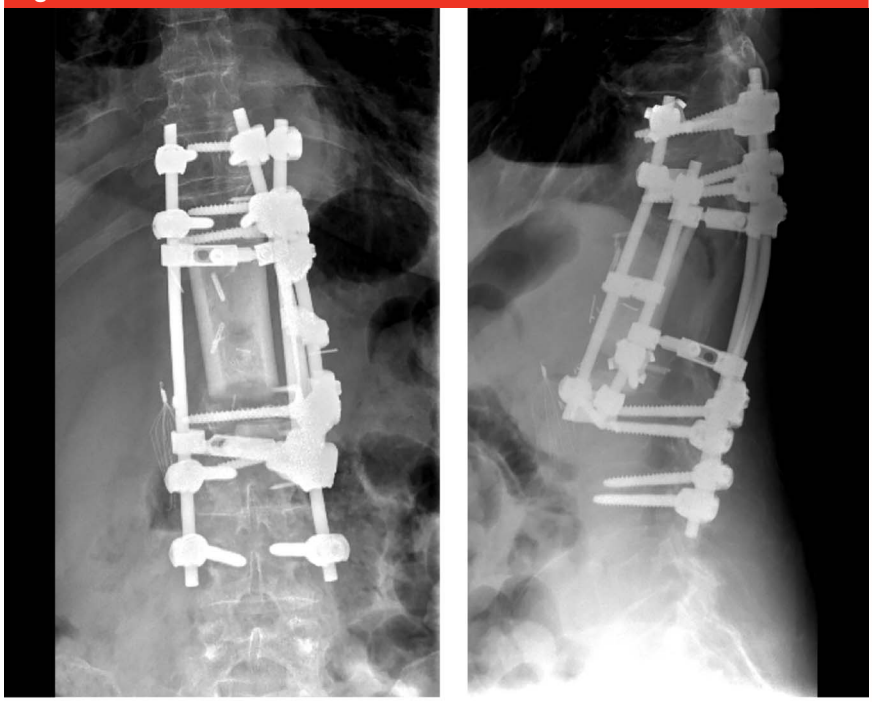
Many authors have commented on the timing of neurophysiologic monitoring changes after maneuvers that affect cord blood flow, but the duration is variable. MEP and SEP changes have been reported to occur within 5 to 10 minutes after temporary or permanent occlusion of key segmental vessels and to return to baseline in reversible scenarios, such as "soft clamping," within 5 to 19 minutes of clamp removal.[24,43] However, although combined neurophysiologic monitoring is thought to be an adequate proxy for clinical motor function, the correlation is not perfect. Irreversible SSEP/MEP changes may indeed be accompanied by postoperative paraplegia, but not always, and the reverse is true, as well, whereby false-negative readings occur.[39] The report by Svensson et al[16] of 1,509 TAAA procedures had a 16% clinical postoperative neurologic deficit rate, but only 21% were observed in the immediate postoperative period; 32% were not observed for several days after surgery, and the range of onset was 1 to 21 days.[16] Neurologic damage from altered blood supply is complex and may be related to local ischemia, global perfusion dynamics,

reperfusion injury, or a combination of the three.

Protective Maneuvers

Standard neuroprotective maneuvers for any surgical procedure involving the spinal cord include maintenance of hemostasis, maintenance of mean arterial pressure, maintenance of end-tissue oxygenation, and avoidance of mechanical stressors such as distraction or rapid deformation. However, because spinal cord perfusion pressure is simply a function of mean arterial pressure minus the cerebrospinal fluid pressure, other methods have been developed to balance this equation in a favorable way. One example is controlled cerebrospinal fluid drainage via lumbar cannulation of the intrathecal space. A recent Cochrane review of three large trials investigating cerebrospinal fluid drainage in the setting of TAAA repairs found evidence, albeit limited, that cerebrospinal fluid drainage improves neurologic outcome in open TAAA.[44] Although less germane to tumor resection or deformity surgery, another neuroprotective maneuver used during TAAA repair involves distal aortic perfusion during cross-clamping of the aorta to maintain distal aortic perfusion pressure. A Cochrane review found no high-quality randomized studies to support this technique but did highlight several observational studies that suggest a neuroprotective effect with improved outcomes.[45] In addition, other authors have reported low rates of neurologic complications during TAAA using a protocol that involves reimplantation of segmental vessels previously divided for exposure;[46-49] however, this protocol has been called into question by others,[19] who have reported low rates of neurologic compromise without reimplantation, and it is not commonly

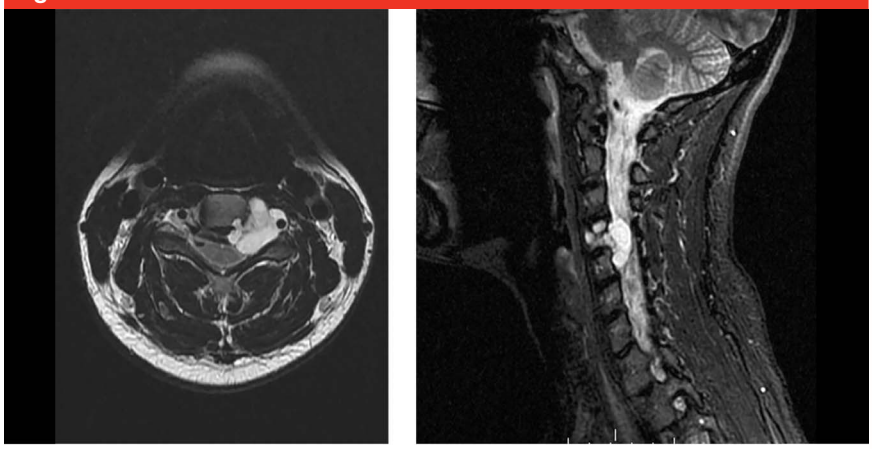
Figure 6



A B

Postoperative PA (**A**) and lateral (**B**) radiographs depicting the spinal reconstruction in the same patient shown in Figures 3 through 5, following total spondylectomy of L1, with partial spondylectomies of T12 and L2, including posterior segmental fixation, femoral allograft interbody reconstruction, Kaneda-type rod fixation of the anterior vertebrae, and linkage of the posterior and anterior systems via a crosslink device.

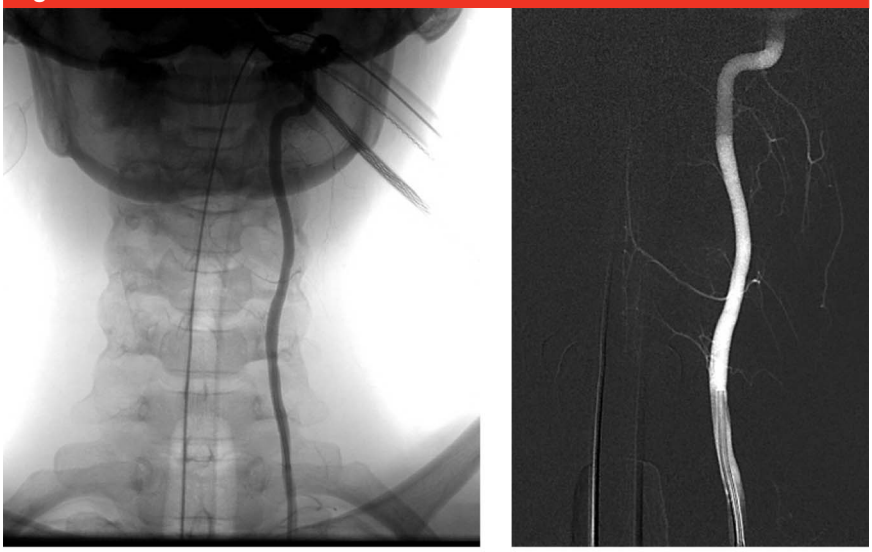
Figure 7



A B

A, Axial short tau inversion recovery (STIR) magnetic resonance image of the cervical spine in a 30-year-old woman who presented with a 2-year history of left periscapular shoulder pain demonstrating tumor encasement of the left vertebral artery and extensive left neuroforaminal involvement. **B**, Left-sagittal STIR magnetic resonance image demonstrating the tumor extent across multiple segments in the midcervical spine.

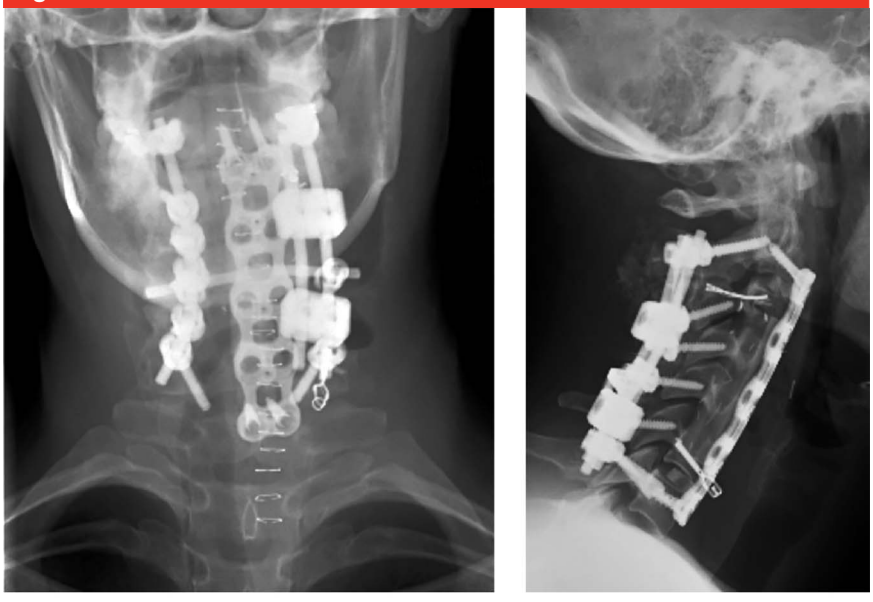
Figure 8



A B

Standard (**A**) and reversed (**B**) spinal angiogram images in the same patient shown in Figure 7, demonstrating the lone dominant C5 radiculomedullary artery and its contribution to the anterior spinal artery.

Figure 9



A B

Postoperative AP (**A**) and lateral (**B**) radiographs of the cervical spine reconstruction, in the same patient shown in Figures 7 and 8.

done at our center. Other potentially clinically useful maneuvers include epidural or systemic cooling;[50] also, recent experimental evidence in animals has demonstrated a neuroprotective effect of intravascular injection of a direct free-radical scavenger agent.[51] Aside from the limited support available for the use of cerebrospinal fluid drainage, evidence for most modalities is lacking.

Common Protocols at the Authors' Center

Currently, to minimize the risk of acute or postoperative ischemic injury to the spinal cord during tumor resection, our group uses several methods in the preoperative, intraoperative, and postoperative stages. Preoperatively, we use angiography to identify critical anterior radiculomedullary vessels, and we favor a multidisciplinary approach to the preoperative planning involving thoracic surgery, vascular surgery, and neurointerventional radiology. We frequently use preoperative embolization of the tumor levels, depending on the presence of visualized collateral supply to the cord.

Intraoperatively and postoperatively, we maintain the mean arterial pressure at >90 mm Hg; however, relatively higher pressures must be balanced with intraoperative bleeding risk. We use combined SSEP/MEP monitoring for every case. In high-risk patients, such as those with comorbid risk factors for ischemia (eg, old age, peripheral vascular disease, kidney disease) or with disease-related risk factors (eg, planned sacrifice of major radiculomedullary vessel or vertebral artery), we occasionally insert prophylactic cerebrospinal fluid drains, which are initially clamped and used simply to monitor cerebrospinal fluid pressure. Depending on the patient risk profile and any neurologic changes that occur intraoperatively or postoperatively, we initiate strict maintenance of cerebrospinal fluid pressures <10 mm Hg[52,53] with continued maintenance of the mean arterial pressure at >90 mm Hg. In the case of a postoperative neurologic change, we also advise the use of axial imaging to rule out other

causes of neurologic change, such as postoperative hematoma.

Intraoperatively, when dividing segmental vessels or nerve roots containing radicular vessels, we attempt to divide only the vessels that are necessary for exposure and safe resection of the tumor, and we divide them as far laterally as possible. We regularly use a "soft clamping" protocol, in which key segmental or radicular vessels to be divided are temporarily clamped and the neurophysiologic monitoring observed for 10 minutes before permanent division. If neuromonitoring changes are observed during this "soft clamping," the clamp is removed and attempts to spare the segmental vessel in question are undertaken. Often, the vessel in question cannot be safely spared, given the tumor extent, and although attempts at revascularization can be considered, a difficult decision must at times be made regarding paraplegia risk versus oncologic resection margin. This is discussed preoperatively with every patient. Advances in the techniques of radiotherapy delivery both in and out of the operating room have helped address close or intentionally positive margins, but the clinical scenario remains challenging.

Case Examples

The following case examples illustrate sacrifice of dominant radiculomedullary vessels without postoperative paraplegia.

Case 1

A neurologically normal 58-year-old woman with a history of small-cell osteosarcoma of the right hemipelvis presented with a solitary L1 metastasis 3 years after local and systemic treatment of her pelvic disease (Figure 3). Based on a necrosis rate of 50%, her tumor responded poorly to systemic chemotherapy. Aside from the spinal disease, there was no evidence of other metastasis or local recurrence in the pelvis. She underwent preoperative radiotherapy with 19.8 Gy to the L1 tumor site. She underwent standard preoperative staging and imaging procedures, including preoperative thoracolumbar angiography. Angiography demonstrated a dominant anterior radiculomedullary vessel originating from the left L1 segmental vessel and supplying the ASA (Figure 4). There was a minor contribution to the ASA via a second radiculomedullary artery on the left at T10 (Figure 5).

With a thorough understanding of the neurologic risks, the patient elected to undergo en bloc resection of the isolated metastasis at L1. This was performed as a two-stage procedure, with the first stage involving posterior decompression and partial transpedicular osteotomies at T12 and L2 and posterior segmental instrumentation and fusion with iliac crest autograft from T10 to L4. T12 and L1 nerve roots along with their radicular vessels were ligated bilaterally, given the extent of the tumor. The second stage was performed 2 days later and consisted of left-sided thoracotomy and en bloc excision of L1, along with partial segments of T12 and L2, with femoral strut allograft reconstruction and Kaneda-type anterior instrumentation (Figure 6). The patient did receive 10 Gy of intraoperative P32 dural plaque radiation, as well as a postoperative boost with protons, for a total of 68.4 Gy. Intraoperative margins were microscopically positive at the dural margin, as planned. Given the L1 nerve root sacrifice, the patient had postoperative hip flexor weakness, but there was no evidence of global paraparesis, as might be predicted following sacrifice of the AA at L1. One explanation for this is that the minor T10 radiculomedullary artery compensated for the sacrificed dominant AA.

Case 2

A neurologically normal 30-year-old woman presented with a 2-year history of left periscapular shoulder pain. She had imaging and biopsy studies consistent with a conventional-type chordoma involving the C4 and C5 cervical segments, as well as the left C4-5 neuroforamen (Figure 7). The tumor extent was predominantly left-sided with encasement of the left vertebral artery and was not metastatic. She underwent preoperative cerebral angiography, which demonstrated a co-dominant vertebral artery system with adequate contralateral reflux through the circle of Willis and the presence of a single dominant radiculomedullary vessel emanating from the left vertebral artery at C5 (Figure 8). There were no identifiable tumor supply vessels for embolization. She underwent preoperative combinatorial proton- and photon-based radiation to 50.4 Gy.

With a thorough understanding of the neurologic risks, the patient elected to undergo en bloc resection of her tumor. This was performed as a two-stage procedure. The first stage involved posterior decompression, partial posterior osteotomies at C3 and C6, posterior instrumentation from C2 to C7, and ligation of the left vertebral artery at C3 and C6. In addition, the C4 and C5 nerve roots were ligated, along with the dominant radicular vessel. Prior to ligation, however, temporary vascular clips were used for 10 minutes to test for neurologic potential changes, with no events observed. Throughout the remainder of the first-stage operation, there were no significant changes from baseline in the SSEPs or MEPs. A wake-up test was not performed.

The second stage of the resection involved completion osteotomies and a hemivertebrectomy of the most caudad portion of C3, the entirety of C4 and C5, and the most cephalad

portion of C6. The right vertebral artery, including segmental vasculature, was not dissected free because it was not involved by tumor. The tumor specimen was removed en bloc, and the defect was reconstructed with free autogenous fibula graft and an anterior plate (Figure 9). The postoperative course was not significant for any sensorimotor neurologic deficit except for weakness (3/5) in the left deltoid and subtle weakness (4/5) in the left biceps, given the oncologically scheduled root sacrifice at C5. By 8 months postoperatively, the motor function in the left biceps and deltoid muscle groups had normalized.

Summary

The blood supply to the spine is complex and may have dynamic autoregulatory or anastamotic mechanisms to compensate for loss of critical vessels that feed the ASA during surgical procedures. Elucidation of the exact mechanisms for this compensation requires further study but will be aided by preoperative, intraoperative, and postoperative comparative angiography. Relatively little study has been dedicated to the cervical spine, and this region in particular could be an area of future investigation.

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