Review Article

Update on the Diagnosis and Management of Cervical Spondylotic Myelopathy

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Abstract

Spondylotic degeneration in the cervical spine may result in static and/or dynamic spinal cord compression that can lead to the associated signs and symptoms of myelopathy. Clinical examination combined with appropriate imaging studies help to confirm the diagnosis. Classic natural history and basic science studies suggest a pernicious course of demyelination and neurologic decline in a large subset of patients. The characterization of disease severity and progression in patients with cervical spondylotic myelopathy has improved in recent years with imaging and data from prospective and multicenter studies. Additionally, advances in surgical techniques, implants, and imaging modalities have improved the identification of surgical candidates with cervical spondylotic myelopathy and associated treatment strategies. Surgical treatment, via an anterior, posterior, or a combined approach, is primarily intended to arrest neurologic progression, although it can improve function in many patients. Alignment and the characteristics and location of spinal cord compression help determine the ideal surgical approach. Distinct complications associated with each technique may be mitigated by appropriate patient selection and should be discussed preoperatively to ensure informed decision making.

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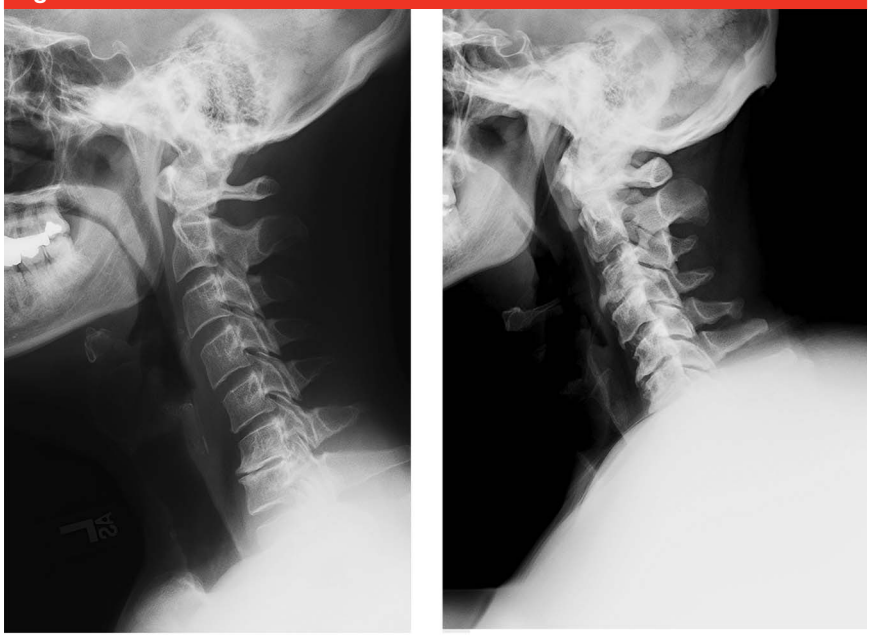
Age-related degenerative changes in the cervical spine, or cervical spondylosis, may result in chronic compression and dysfunction of the spinal cord. Evidence of cervical spinal cord compression on imaging studies is a relatively common finding with advanced age; however, a subset of patients develops the clinical syndrome of cervical spondylotic myelopathy (CSM). CSM is the most common cause of spinal cord impairment in adults and can manifest with a range of signs and symptoms, such as gait instability, diminished hand dexterity, motor weakness, sensory loss, bowel and bladder dysfunction, and ultimately significant disability and functional decline.[1]

The clinical course of CSM may involve periods of quiescent disease with step-wise deterioration; however, it also may follow a more steadily progressive course of neurologic dysfunction. Although precise identification of risk factors for neurologic decline has proven challenging, early diagnosis of CSM allows the clinician and the patient to undertake a mutual informed decision-making process regarding the risks and benefits of nonsurgical and surgical management.

Etiology and Pathophysiology

Cervical spinal stenosis is defined as a reduction in the volume of the

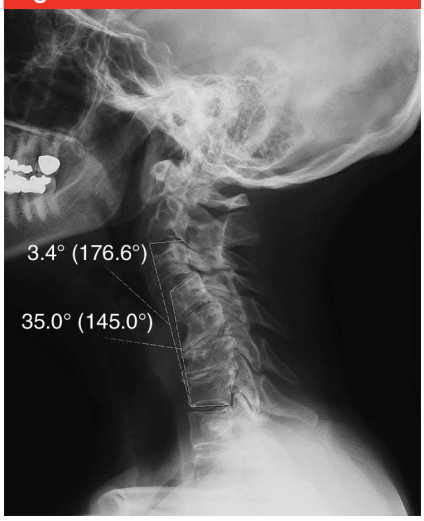
Figure 1



A B

A, Lateral radiograph demonstrating a focal pattern of degenerative disk disease at C6-C7 with disk space collapse, osteophyte formation anteriorly and posteriorly, and bony end-plate sclerosis. **B,** Lateral radiograph demonstrating more extensive spondylosis with anterior osteophytes at several levels, retrolisthesis at C3-C4 and C5-C6, and anterolisthesis at C4-C5.

Figure 2

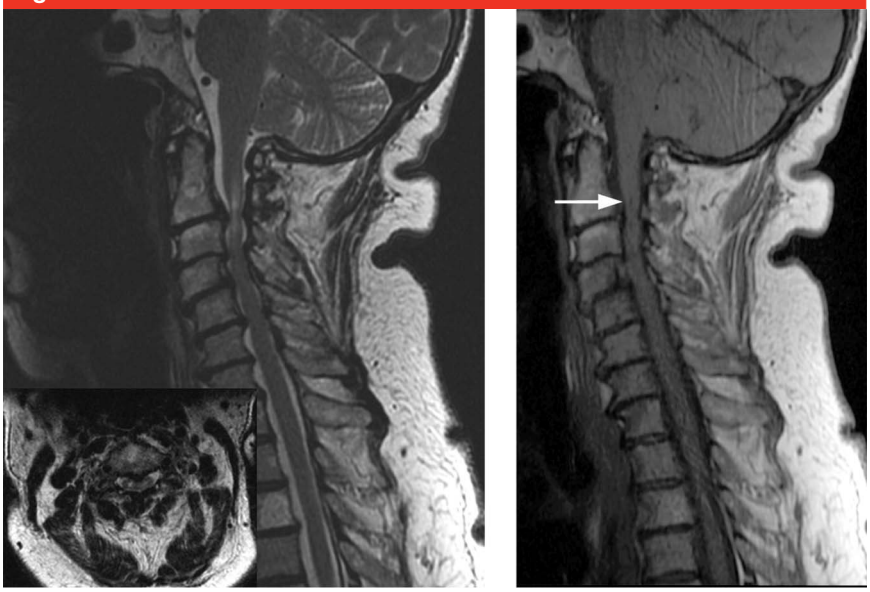


Lateral radiograph of the cervical spine demonstrating 3° of lordotic alignment and a regional kyphosis of 35° from C4-C7 with an overall sigmoid alignment in a 57-year-old woman who has mild myelopathy symptoms of diminished hand dexterity and a slight gait disturbance.

spinal canal. The clinical syndrome of CSM occurs when the stenosis impinges on the spinal cord, and the severity of CSM is generally thought to be related to the amount of mechanical compression of the various spinal cord tracts. The etiology of CSM is multifactorial and likely has both heritable and environmental contributions. A systematic review of existing family and genetic studies suggests an inherited predisposition for both CSM and ossification of the posterior longitudinal ligament (OPLL).[2] Although specific single nucleotide polymorphisms (SNPs) have been associated with OPLL (ie, collagen 6A1 and 11A2 genes), no specific SNPs or haplotypes have been conclusively associated with CSM. Also, preexisting narrowing of the spinal canal or congenital stenosis may lower the amount of spondylotic degeneration required to result in neurologic compression and therefore CSM. In one series of 63 patients with symptomatic CSM, 40 patients (63%) had developmentally narrow canals.[3]

Disk dessication and bulging occur with aging, and the degenerative disk and/or uncovertebral joint osteophytes that form may result in anterior sites of compression of the neurologic elements. Dorsally, the so-called hypertrophied or infolded ligamentum flavum and degenerative facet joints may cause compression. The degenerated spinal motion segment may result in not only direct compression of the neuronal tracts but also an ischemic insult caused by compression of the arterial blood supply to the spinal cord. However, the precise role of ischemia in the pathogenesis of CSM is currently an area of ongoing research and debate with only low-strength evidence to suggest that the area of circumferential compression is associated with deteriorating neurologic symptoms.[4] During daily range of motion, changes in the cross-sectional diameter occur that can result in dynamic compression and repetitive microtrauma. Flexion of the cervical spine may result in stretching of the cord over the anterior spondylotic bars, whereas extension may result in further buckling of the ligamentum flavum and exacerbated dorsal compression. Untoward secondary effects may follow, leading to cellular damage and subsequent clinical sequelae. This process is thought to involve a chronic inflammatory response, apoptosis of neurons and oligodendrocytes, and chronic hypoxic ischemic injury. Autopsy studies of patients with known CSM have demonstrated gray matter atrophy, neuronal loss, and white matter demyelination.[5,6]

Figure 3



A B

A, Sagittal T2-weighted magnetic resonance image demonstrating a C2-C3 disk osteophyte complex with impingement on the spinal cord and associated hyperintense signal in a 67-year-old woman who has a recent history of falls and weakness in her arms. Inset, Axial image of the spine at C2-C3 demonstrating kidney bean–shaped deformation of the cord and hyperintensity within the cord parenchyma. **B,** Sagittal T1-weighted magnetic resonance image demonstrating a hypointense region just proximal to the C2-C3 disk level (white arrow) that portends a worse prognosis.

Diagnosis

Clinical Presentation

The evaluation of the patient with suspected CSM begins with a thorough history and physical examination. The clinical manifestations related to compression of the cervical spinal cord encompass a broad spectrum of signs and symptoms. Patients with cervical spondylosis may experience symptoms of axial neck pain and limitations of range of motion.

Because of the insidious onset of CSM, patients may be unaware of subtle clinical findings, such as mild balance impairment or diminished hand dexterity. Dysfunction of the dorsal columns of the spinal cord may result in diminished proprioception and gait instability. Manifestations of balance impairment may be reported, such as the need to use the handrail while negotiating stairs or the requirement of an assistive device, such as a cane or a walker, as a result of weakness and/or imbalance. Tandem walking, the Romberg test (ie, the patient is asked to stand with the eyes closed and the arms held forward, and a positive test is indicated by loss of balance), and heel and toe walking may be assessed by the examiner to detect dysfunction.

Patients with CSM often experience difficulty with fine motor tasks, such as buttoning buttons, opening jars or doorknobs, using a computer keyboard or cellular phone, or writing. Ono et al[7] described the signs and symptoms associated with CSM that occur in the upper extremity as "myelopathy hand." This condition includes loss of power of adduction and extension of the ulnar two or three digits and the inability to grip and release the hand rapidly. The "finger escape sign," in which the ulnar two digits drift into abduction and flexion after the patient holds his or her hand with the metacarpophalangeal, proximal interphalangeal, and distal interphalangeal joints extended for more than a minute, may be present. Tests of hand dexterity may include the so-called 15-second grip-and-release test to help quantify the extent of CSM disease; normal patients are able to perform the test at least 25 to 30 times in 15 seconds.[8]

Examination of the reflexes may elicit abnormal long-tract signs, such as the Babinski sign, Hoffmann reflex (ie, flicking the fingernail to volarflex the distal interphalangeal joint of the middle finger elicits contraction of the long flexors in the thumb and index finger), and the inverted radial reflex (ie, tapping of the brachioradialis tendon elicits firing of the long finger flexors). The presence of ≥four beats of clonus also suggests upper motor neuron dysfunction. Neurologic compression of the cervical spinal nerve root may result in lower motor neuron findings (eg, hyporeflexia), whereas compression of the cervical spinal cord may result in upper motor neuron findings (eg, hyperreflexia). It should be noted that myeloradiculopathy, the coexistence of myelopathy and radiculopathy from spinal cord and nerve root compression, is fairly common in the CSM patient. In such cases, hyperreflexia may not be present secondary to nerve root compression.

Sensory and vibratory testing in the upper and lower extremities can demonstrate deficits. Patients with advanced CSM may present with bowel or bladder dysfunction or the inability to ambulate altogether.

Radiographic Evaluation

Plain AP and lateral radiographic studies of the cervical spine provide

insight into the extent of the spondylosis. Assessment of cervical spinal alignment, disk height, the presence of end-plate sclerosis, osteophytes, spinal canal dimensions, and any translational deformity (ie, listhesis) should be systematic (Figure 1). Because of population variations in the size of the axial skeleton, there is likewise a variation in the amount of space available for the cord in terms of an absolute measurement that may be considered normal versus pathologic. Early attempts to characterize cervical stenosis on the basis of plain radiographs included the Torg-Pavlov ratio (ie, anterior-posterior diameter of the cervical spinal canal divided by the vertebral body width). A value of <0.80 was considered stenosis.[9] The introduction of a ratio avoided problems with variation in magnification; however, this value was derived in a select population of 23 athletes and did not account for soft-tissue structures. Since its introduction, the Torg-Pavlov ratio has been reported to correlate poorly with actual spinal canal diameter.[10]

Cervical spine alignment is important in the diagnosis and treatment of CSM. On plain lateral radiographs, the cervical spine can be categorized as lordotic, neutral, kyphotic, or sigmoid (S)-shaped (Figure 2). In animal models, a progressive kyphotic alignment has been shown to result in demyelination of nerve fibers in the funiculi and neuronal loss in the anterior horn.[11] CT may reveal spondylotic bars, disk osteophyte complexes, and OPLL lesions; all of these structures are important to recognize when planning a surgical decompression. Comparison of upright plain radiographs with supine radiographs or CT/MRI scans provides information about the stability of the cervical motion segments under physiologic load.

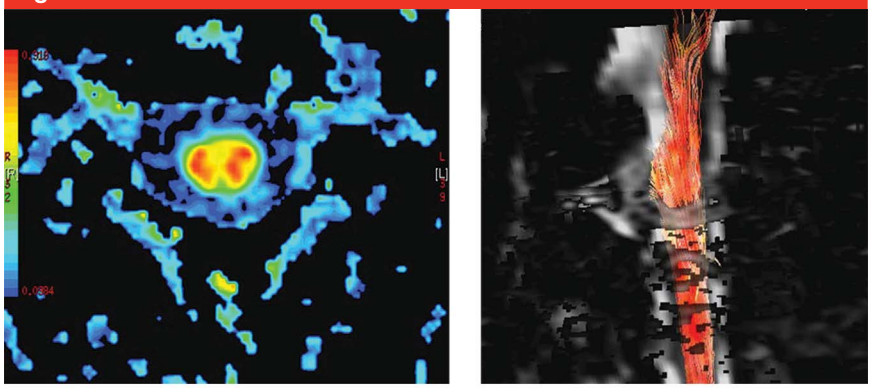
MRI is considered the benchmark imaging modality for assessing the cord status in patients with CSM. Cervical stenosis alone is a common age-related finding. Boden et al[12] performed MRI scans in asymptomatic volunteers and demonstrated that the cervical disk was degenerative or narrowed at one or more levels in 25% of persons aged <40 years and in almost 60% of persons aged >40 years. Matsumoto et al[13] performed a similar radiographic study in which 90% of asymptomatic persons had degenerative MRI findings in the cervical spine at a mean age of 48 years. When followed longitudinally for >10 years, 81% had progression of degenerative findings. Anterior compression of the dura and the spinal cord was seen in 61% of healthy volunteers.[14]

The presence of cerebrospinal fluid, which appears hyperintense both anterior and posterior to the spinal cord on T2-weighted images, provides a so-called myelography effect that can help characterize the extent of compression of the cord. In cases of severe stenosis, cross-sectional cord deformation from oval to kidney bean-shaped may be visible on axial images (Figure 3). Hyperintense signal change within the cord parenchyma on T2-weighted images was traditionally thought to be associated with increased disease severity and a poor prognosis. However, a systematic review demonstrated that high signal alone on T2-weighted images was not indicative of a worse surgical outcome[15] (Figure 3). Of note, there is evidence that the combination of high-signal intensity changes within the cervical spinal cord on T2-weighted images and low intensity on T1-weighted images is associated with irreversible injury and therefore portends a worse prognosis.[16-18]

Myelography involves intrathecal injection of a water-soluble dye contrast agent followed by plain radiography or CT. Blockage of flow of the contrast through the spinal canal may indicate regions of spinal cord compression. Myelography may be a useful study in patients who are unable to undergo MRI (eg, those with pacemakers); however, the intradural injection of contrast agent entails an invasive procedure with associated risks and thus should not be considered a first-line test in patients who are able to undergo MRI.

Dynamic studies, such as plain flexion-extension radiographs, may reveal relative instability of a motion segment (ie, anterolisthesis/retrolisthesis). Motion segments

Figure 4



A B

A and **B,** Diffusion tensor imaging of the cervical spinal cord demonstrating varying signal responses in the white matter spinal cord tracts.

Table 1

Considerations for Determining Surgical Approach for the Patient with Cervical Spondylotic Myelopathy

Finding	Considerations	Surgical Approach/ Procedure
High Cervical Cord Compression (occiput-C2)	Transoral decompression associated with higher infection rate. Posterior decompression may require resection of checkreins to kyphotic deformity and require fusion.	Posterior decompression and fusion
Subaxial Compression (C3-C7)		
Alignment		
Neutral or lordotic	Adequate central canal decompression may be achieved by direct anterior decompression or by cord "drift back" with a laminectomy or laminoplasty procedure.	Anterior, posterior, or combined
Kyphotic (>13°)	Associated with inadequate spinal cord "drift back" with posterior-only decompression	Anterior or combined
Modified K-line (+)	Compression is dorsal to modified K-line. Posterior-alone procedure associated with worse outcomes	Anterior or combined
Modified K-line (−)	Compression is ventral to modified K-line. Adequate "drift back" can be achieved from a posterior approach.	Anterior, posterior, or combined
Radiographic Findings		
Presence of OPLL	Anterior resection poses risk of durotomy with adherent OPLL lesions to the dura.	Anterior "floating method" versus posterior or combined procedure
OPLL and canal occupancy ratio >60%	Associated with inadequate "drift back" from posterior-only procedures	ACDF, STV, ACCF or combined
Single-level disease (anterior compression)	High fusion rate and direct decompression anteriorly	ACDF
Single-level disease (posterior compression)	Direct decompression from posterior approach	Laminoplasty, laminectomy, laminectomy and fusion
Retrovertebral disease	Anterior compression of spinal cord from posterior to vertebral body may require subtotal versus total corpectomy for decompression ± posterior stabilization	ACCF, STV, (combined if more than two levels)

(*continued*)

ACCF = anterior cervical corpectomy and fusion, ACDF = anterior cervical diskectomy and fusion, OPLL = ossification of the posterior longitudinal ligament, STV = subtotal vertebrectomy

adjacent to stiffened, spondylotic segments may exhibit hypermobility and can produce dynamic compression of the spinal cord. Dynamic MRI studies (ie, separate MRI scans performed with the neck positioned in flexion and extension) have been suggested to give a more accurate depiction of the sites of pathologic compression throughout the patient's range of motion.[19]

Diffusion tensor imaging (DTI) is a newer imaging modality with proven applications in brain pathology. It uses MRI sequences to assess the diffusion of water molecules through tissue to produce images of the white matter tracts (Figure 4). Recent data suggest that DTI may be useful in the assessment of neuronal status in CSM. The apparent diffusion coefficient, fractional anisotropy, and eigenvalues (ie, E1, E2, E3) are among the measured parameters in DTI. A prospective study reported encouraging preliminary data that show that DTI values are significantly different between grades of myelopathy.[20]

Management

Nonsurgical

Prediction of which patients will have stable disease and those that will progress remains challenging. A review of the current literature regarding the natural history of CSM suggests that between 20% to 60% of patients with mild CSM deteriorate neurologically over time in the absence of surgical intervention.[4] Once diagnosed, CSM is generally considered a disorder that is best treated surgically such that at a minimum, neurologic function is stabilized and potentially even improved. As with any surgical consideration, patient selection and mutually informed surgical decision making are paramount. Medical comorbidities, advanced age, body

Table 1 (*continued*)

Considerations for Determining Surgical Approach for the Patient with Cervical Spondylotic Myelopathy

Finding	Considerations	Surgical Approach/ Procedure
Radiographic Findings (*continued*)		
Instability (eg, spondylolisthesis, hypermobility on flexion-extensions radiographs)	Dynamic spinal cord compression may not be addressed by nonfusion procedures.	ACDF, laminectomy and fusion, or combined
Congenital stenosis	Usually multilevel compression, may not be decompressed adequately from anterior approach	Laminoplasty (laminectomy and fusion)
Myeloradiculopathy + soft-disk herniation	Soft disk may be decompressed anteriorly or posteriorly, alignment and morphology of canal compression may help dictate surgical approach	ACDF or posterior decompression (± fusion) + foraminotomy
Myeloradiculopathy + spondylotic foraminal stenosis	May achieve more thorough foraminal decompression (direct and indirect) of spondylotic disk by anterior approach	Anterior or combined
Clinical Findings		
Axial neck pain	Unlikely to be improved by a motion-preserving laminoplasty procedure	ACDF, laminectomy and fusion, or combined
Previous radiation around the neck	May raise risk of pseudarthrosis and wound complications	Laminoplasty
History of dysphagia	May be exacerbated by anterior approach	Laminoplasty, laminectomy ± fusion
Concern or history of dysphonia or vocal occupation (eg, singer, radio host)	Finite risk of dysphonia with anterior surgical procedure	Laminoplasty, laminectomy ± fusion
Chronic smoker	Nicotine inhibits fusion; if patient is unable to quit smoking consider nonfusion procedure or combined anterior and posterior approach to maximize fusion rate	Laminoplasty or combined (anterior or posterior alone—pseudoarthrosis risk)

ACCF = anterior cervical corpectomy and fusion, ACDF = anterior cervical diskectomy and fusion, OPLL = ossification of the posterior longitudinal ligament, STV = subtotal vertebrectomy

habitus, low baseline level of function, and risk factors for infection, such as diabetes and smoking, may make surgical treatment less desirable.

Nonsurgical management options for patients with mild myelopathy include physical therapy for gait training, occupational therapy for improvement of upper extremity dexterity, and neck immobilization with a hard cervical collar. Patient counseling regarding the hazards of minor cervical trauma and the potential for symptomatic worsening is appropriate in this setting.

Surgical

Preoperative Planning

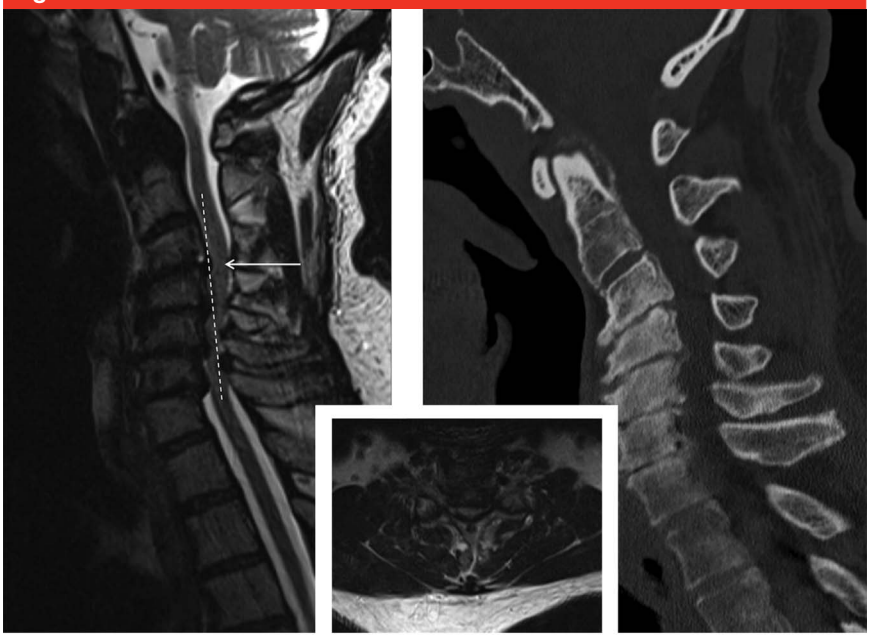
Prior to any surgical procedure, careful attention must be given to the positioning of the patient with CSM. Assessment of a patient's preoperative cervical spine range of motion is critical. It is important to keep the patient's neck within the range of comfort as assessed before surgery to avoid injury during intubation, transferring, or positioning.

Cervical spine extension is often limited in the spondylotic spine, which may make standard intubation difficult. Fiber-optic intubation or other video-assisted techniques can be helpful to maintain the neck in a neutral position and to avoid iatrogenic neurologic injury. In some cases, awake fiber-optic intubation is preferred because this enables neurologic assessment postintubation.

Intraoperative Considerations

Intraoperative neuromonitoring (IONM) modalities include somatosensory-evoked potentials (SSEPs), transcranial motor-evoked potentials (TcMEPs), and electromyography (EMG). IONM allows

Figure 5



A B

T2-weighted magnetic resonance image **(A)** and sagittal CT reformatted image **(B)** demonstrating mild cervical spondylotic myelopathy with kyphotic alignment and severe stenosis at several motion segments in the subaxial region in a 71-year-old man. Modified K-line (dotted line) and white arrow demonstrate that the degree of spinal cord drift back that may be expected from a posterior-only procedure may be inadequate. Compression is present both at the disk level and posterior to the vertebral body. Inset, The axial image demonstrates compression from the intervertebral disk anteriorly and from the buckled ligamentum flavum posteriorly.

assessment of neurologic function before and during surgery for CSM. A precise correlation between IONM recordings, MRI studies, and examination findings has been difficult to attain. Haghighi et al[21] reported that baseline SSEPs may show increased latency in CSM compared with cervical radiculopathy patients, and that TcMEPs were more likely to be absent in distal muscles. In predicting postoperative neurologic deficits, a prospective study of 1,055 patients showed SSEPs had a sensitivity, specificity, and positive predictive value (PPV) of 52%, 100%, and 100%, respectively, and a negative predictive value (NPV) of 97%.[22] MEPs had a sensitivity, specificity, and PPV of 100%, 96%, and 96%, respectively, and an NPV of 100%. EMG had a sensitivity of 46%, a specificity of 74%, a PPV of 3%, and an NPV of 97%. Moreover, persistent intraoperative changes on TcMEPs have been associated with new postoperative neurologic deficits.[23] As such, multimodality IONM with EMG and SSEP and selective use of MEPs may assist the surgeon in predicting and potentially preventing iatrogenic neurologic deficits in the CSM patient.

The anesthesiologist may be advised to avoid undue hypotension during surgery because it may compromise perfusion of the spinal cord. In general, a mean arterial pressure of >80 mm Hg is recommended. Both noninvasive blood pressure cuff and arterial line measurements (with the water column at the level of the heart) are critical. If TcMEPs are to be used, long-acting paralytic agents should be avoided because they can interfere with the signals. In the event of an intraoperative neurophysiologic alert, an appropriate response begins with an intraoperative pause, communication between the attending anesthesiologist, surgeon, and neuromonitoring team to ensure that the alert was not triggered by a technical issue (eg, lead placement), and ensuring blood pressure (mean arterial pressure >80) and oxygen saturation are adequate. Any surgical interventions performed before the alert may be sequentially reversed (eg, removing strut graft, cage, restoring preintervention alignment) until the signals return to baseline. If the alert persists, a wake-up test may be performed. This involves lightening of the anesthesia while asking the patient to perform motor functions of the arms and legs.

Surgical Approaches

Decompressive and reconstructive surgical techniques for the treatment of CSM may be broadly divided into anterior, posterior, and combined (ie, anterior and posterior) surgical approaches. A systematic review comparing these approaches found similar comparative effectiveness and safety that suggests that the location of the pathoanatomy may guide surgical decision making.[24] Consideration must be given to the sagittal alignment of the cervical spine when performing a posterior technique that relies on a "drift back" of the thecal sac away from ventral sites of compression. A regional kyphosis (>13°) has been associated with unfavorable outcomes following posterior-only surgery for CSM.[25] Traditionally, anterior approaches for CSM were preferred for patients with one- or two-segment pathology, whereas posterior techniques were preferred for patients with multisegmental (more than two levels)

compressive disease (Table 1). However, in recent years, anterior and posterior techniques have been increasingly used for multisegmental disease with similar outcomes. The modified K-line (ie, a line connecting the midpoints of the spinal cord at C2 and C7) is a preoperative index that may help predict if adequate posterior drift back will be achieved from anterior sites of compression[26] (Figure 5).

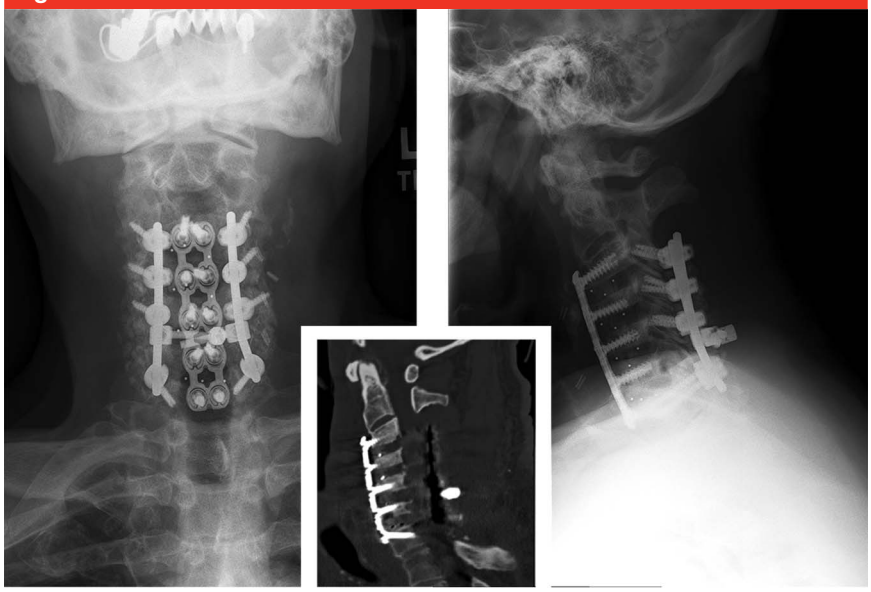
The standard anterior approach to the cervical spine, using the interval between the sternocleidomastoid muscle and the strap muscles, is the workhorse of anterior surgery for CSM. If necessary, it is easily converted into an extensile exposure that allows access from the atlantoaxial articulation to the cervicothoracic junction. Anterior cervical decompression and fusion, anterior subtotal vertebrectomy, and anterior cervical corpectomy are all techniques in the surgeon's armamentarium; the choice of which technique to use depends on the desired region of decompression and stabilization. For example, in patients with retrovertebral disease, resection of a portion of the end plate (ie, subtotal vertebrectomy) or anterior cervical corpectomy may be required for adequate anterior decompression (Figure 5). Depending on the extent of decompression and the stability of the surgical construct, a simultaneous posterior decompression and fusion procedure may be required (Figure 6).

Posterior techniques include laminectomy alone, laminectomy and fusion, or laminoplasty. Multilevel laminectomy without fusion was once a commonly performed procedure for CSM; however, increasing recognition of the potential for destabilization and subsequent postlaminectomy kyphosis with the potential for neurologic deterioration have made its use less widespread.[27] The stiffening of the spine that occurs with age may make laminectomy alone a viable surgical option with careful surgical technique that preserves the facet capsules. Laminectomy and fusion with lateral mass screw fixation has mitigated issues with postoperative progressive kyphosis and has largely replaced stand-alone laminectomy.

Several modifications of laminoplasty techniques have been developed, and cervical laminoplasty procedures are broadly categorized into two common subtypes. In 1977, Hirabayashi et al[28] described a technique known as expansive open-door laminoplasty (also known as ELAP or open-door). In 1982, Kurokowa et al[29] described a spinous process-splitting technique (ie, double-door or French-door) laminoplasty. Regardless of the technique, the principles of laminoplasty usually involve a nonfusion method of widening the spinal canal via the lateral mass-laminar junction and using suture, bone, a hydroxyapatite spacer, or a metallic or other implant to keep the lamina in an expanded position (Figure 7). Laminoplasty and fusion may also be considered to improve the bone graft surface area in patients with a challenging fusion environment.

Alternative procedures for surgical treatment of CSM have been proposed. Skip laminectomy involves standard laminectomies performed at selective levels and partial laminectomies performed at the cephalad halves of the lamina at other levels to preserve the extensor muscle (ie, multifidus and semispinalis cervicis) attachments to the spinous processes.[30] Compared with traditional laminoplasty procedures, similar results have been reported. Cervical total disk replacement (CTDR) has been used for the treatment of both one-level[31] and two-level CSM.[32]

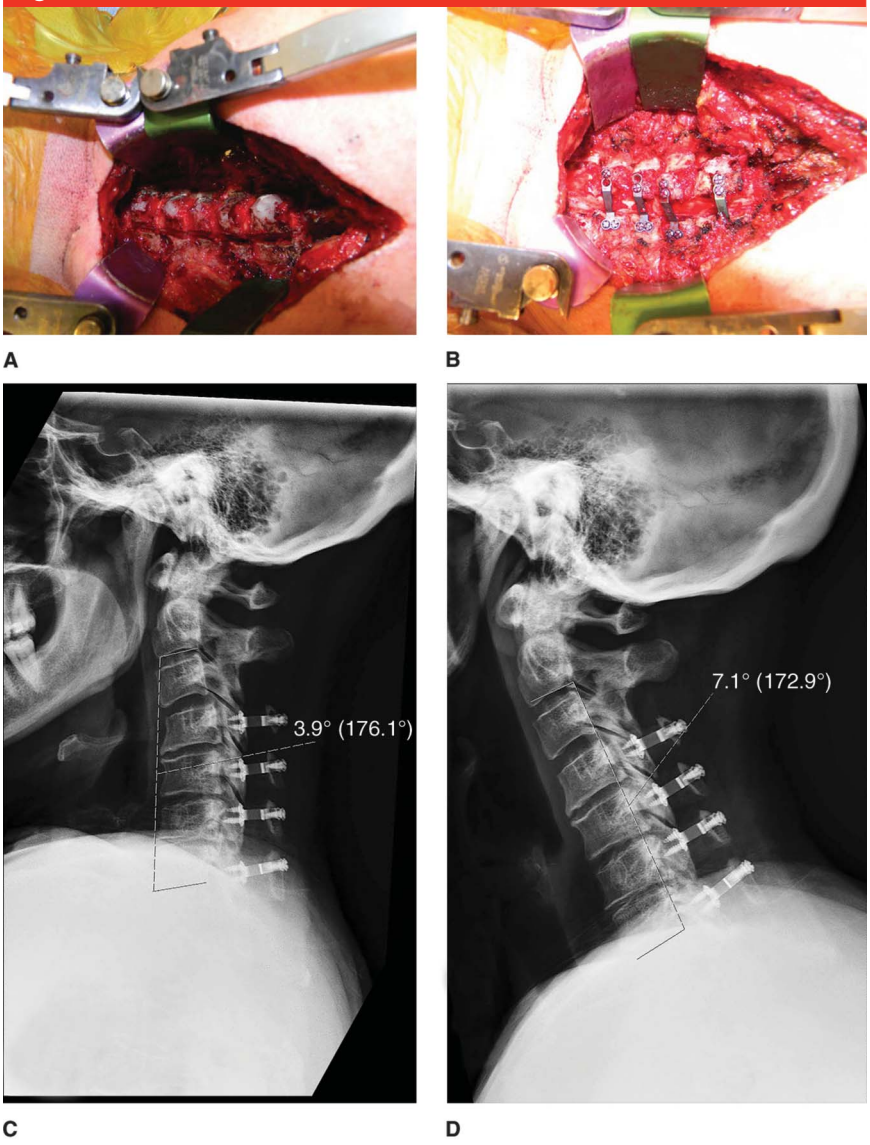
Figure 6



A B

AP **(A)** and lateral **(B)** postoperative radiographs of the spine in the same patient shown in Figure 5 following combined anterior (C3-C7 anterior diskectomy and fusion with subtotal vertebrectomies and plate fixation) and posterior (C3-C7 laminectomy and fusion) surgery. Inset, Sagittal CT of the cervical spine demonstrating regions of decompression anteriorly and posteriorly.

Figure 7



A and **B,** Intraoperative images of the spine demonstrating modified expansive open-door laminoplasty. Cranial is to the left and caudal is to the right in both images. A trough was created on both sides at the spinolaminar junction (left) and the lamina door was hinged on the right and held open on the left with plate fixation at C4, C5, C6, and C7. Flexion **(C)** and extension **(D)** radiographs obtained 6 months after surgery demonstrating segmental plate fixation with preservation of 10° of motion.

Table 2

Nurick's Classification System for Myelopathy

Grade	Root Signs	Cord Involvement	Gait	Employment
0	Yes	No	Normal	Possible
I	Yes	Yes	Normal	Possible
II	Yes	Yes	Mild abnormality	Possible
III	Yes	Yes	Severe abnormality	Impossible
IV	Yes	Yes	Only with assistance	Impossible

Some data suggest equivalence of CTDR with fusion at early follow-up.[32] Despite early encouraging results, using CTDR for primary treatment of CSM remains controversial and lacks long-term follow-up.

Outcomes and Complications of Surgery

Several measures of disease severity have been developed to characterize the CSM disease burden. The Nurick classification for myelopathy,[33] the Japanese Orthopaedic Association (JOA) scale,[34] and Benzel's modified Japanese Orthopaedic Association (mJOA) scale[35] are among the more commonly used instruments (Tables 2 through 4). According to the Benzel mJOA scale, the severity of CSM can be categorized as mild (mJOA ≥15), moderate (mJOA = 12 to 14), or severe (mJOA <12). The Neck Disability Index (NDI)[36] is a modification of the Oswestry Disability Index as a self-reported measure of neck pain, lifting, driving, sleeping, and work activities. The 30-meter walk test, the Myelopathy Disability Index (MDI), and the Medical Outcomes Study 36-Item Short Form, version 2 (SF-36v2) are among the more commonly used outcome instruments for CSM patients. Complementary outcome measures include the quick-DASH; Berg balance scale; graded redefined assessment of strength, sensibility, and prehension test; and grip dynamometer.[37]

Although many retrospective studies have reported on the outcomes of surgical treatment of CSM over the past several decades, a more recent large prospective multicenter study was performed. The AOSpine North American CSM study followed a total of 278 patients from 12 sites.[38] Patients underwent anterior, posterior, or a combined operation for CSM at the discretion of the treating surgeon

and were assessed using the mJOA scale, the Nurick scale, NDI, and SF-36v2 (Physical and Mental Component scores). Patients who were selected for anterior surgery tended to be younger and had less severe myelopathy than did those who underwent posterior procedures, and improvement in the mJOA score was significantly lower in the anterior group (however, baseline characteristics were different). The extent of improvement in Nurick grade, NDI, and SF-36v2 physical and mental scores did not differ between groups. This large observational prospective study suggests that both anterior and posterior approaches have equivalent efficacy.

Potential complications related to cervical spine surgery for CSM include dysphagia, dysphonia, Horner syndrome (ie, miosis, pupillary constriction, enophthalmos, anhidrosis, and ptosis as a result of sympathetic chain dysfunction), cerebrospinal fluid leak, esophageal perforation, iatrogenic neurologic injury, persistent axial neck pain, pseudarthrosis, and infection. Of the 302 patients that were followed prospectively for 2 years in the AOSpine North America CSM study, perioperative complications (<30 days) were reported in 15.6% and delayed complications (>30 days) in 4.4%.[39] Interestingly, the incidence of C5 palsy (traditionally associated with posterior laminectomy/laminoplasty procedures) was not associated with either the anterior or posterior surgical approach. Posterior procedures had a higher rate of wound complications (4.7%) compared with anterior procedures (0.6%). Multivariate analysis demonstrated that older patients, longer surgical times, and combined anterior-posterior procedures were associated with an elevated risk of complications. Despite the finite risk of complications, surgical intervention for CSM has been associated with improvement in functional, disability-related, and quality-of-life outcomes for mild, moderate, and severe CSM.[40]

Table 3

The Japanese Orthopaedic Association Scale for Spondylotic Myelopathy[22]

Motor	Activity/Finding	Points
Upper extremity	Unable to eat with spoon or chopsticks	0
	Possible to eat with spoon but not chopsticks	1
	Possible to eat with chopsticks but not adequate	2
	Possible to eat with chopsticks but awkward	3
	Normal	4
Lower extremity	Impossible to walk	0
	Need a cane or aid on flat ground	1
	Need a cane or aid on stairs	2
	Possible to walk without a cane or aid but slow	3
	Normal	4
Sensory		
Upper extremity	Apparent sensory loss	0
	Minimal sensory loss	1
	Normal	2
Lower extremity	Apparent sensory loss	0
	Minimal sensory loss	1
	Normal	2
Trunk	Apparent sensory loss	0
	Minimal sensory loss	1
	Normal	2
Bladder Function	Complete retention	0
	Severe disturbance	1
	Mild disturbance	2
	Normal	3
		Total = 17

Future Directions

Several obstacles remain in trying to fill the remaining gaps in the current knowledge base on the natural history and efficacy of surgical intervention for CSM. Large randomized prospective studies of nonsurgical versus surgical treatment of CSM in modern spine care practice have remained elusive in providing information on optimal timing of surgical intervention. This may reflect the difficulty in maintaining surgeon equipoise in this patient population; randomization of a patient with CSM into a nonsurgical group may place the patient at risk for neurologic deterioration without intervention and may present an ethical dilemma for the investigator.

Surgery is effective in preventing the progression of neurologic decline in patients and may improve quality of life; however, some patients with CSM may have residual neurologic deficits following surgery given the chronicity of the disease. Pharmacologic interventions, such as riluzole, a sodium and glutamate-blocking medication that is FDA-approved for the treatment of amyotrophic lateral sclerosis, are currently under

Table 4

The Modified Japanese Orthopaedic Association Scale for Spondylotic Myelopathy[35]

Motor	Activity/Finding	Points
Upper extremity	Inability to move hands	0
	Unable to use a spoon but able to move hands	1
	Unable to button shirt but able to eat with a spoon	2
	Able to button shirt with great difficulty	3
	Able to button shirt with slight difficulty	4
	No dysfunction	5
Lower extremity	Complete loss of motor and sensory function	0
	Sensory preservation without ability to move legs	1
	Ability to move legs but unable to walk	2
	Able to walk on flat floor with a walking aid	3
	Able to walk up and/or down stairs with a handrail	4
	Moderate to significant lack of stability but able to walk up and/or down stairs without a handrail	5
	Mild lack of stability but able to walk unaided with smooth reciprocation	6
	No dysfunction	7
Sensory		
	Complete loss of hand sensation	0
	Severe sensory loss or pain	1
	Mild sensory loss	2
	No sensory loss	3
Sphincter Dysfunction		
	Inability to micturate voluntarily	0
	Marked difficulty with micturition	1
	Mild to moderate difficulty with micturition	2
	Normal micturition	3
	Difficulty with micturition (frequency, hesitation)	2
	Normal	3
		Total = 18

investigation in the postoperative CSM population. Preclinical studies indicate that the administration of riluzole may further improve the results of surgical decompression for CSM. The CSM-Protect Trial is an ongoing phase III randomized controlled trial that is being used to investigate the functional recovery of CSM patients undergoing decompressive surgery in addition to receiving riluzole versus those undergoing decompressive surgery and receiving a placebo medication.[41] The potential for medical augmentation of surgical practice may help mitigate the chronic inflammatory response, apoptosis, and hypoxic ischemic injury stages in the pathogenesis of CSM.

Summary

CSM is the most common disorder of the spinal cord and early identification of affected patients is important to prevent functional decline and to improve quality of life. It is likely that there is a heritable predisposition to CSM that is exacerbated by age-related spondylotic degeneration. History and physical examination can help confirm the diagnosis by eliciting neurologic deficits, the presence of abnormal long-tract signs, or findings of myelopathic hands. Radiography, CT, MRI, and dynamic studies are used to help confirm the diagnosis of CSM. Following diagnosis, the patient may be counseled that the natural history is often characterized by stable, quiescent disease; however, others may follow a more progressive and pernicious decline. Hyperintense T2-weighted magnetic resonance images and hypointense T1-weighted magnetic resonance images portend a worse prognosis. Surgical intervention by anterior, posterior, or combined approaches can stabilize and potentially improve the patient's neurologic status. Cervical spine alignment, number of motion segments involved, morphology, and location of the spondylotic compression will help guide surgical decision making. A large prospective multicenter study demonstrated that patients treated with anterior techniques tend to be younger with less severe CSM and more focal pathology; however, both anterior and posterior procedures are effective. Surgical intervention is most effective to stabilize neurologic decline; however, improvement is less predictable and increased risks exist in older patients undergoing longer procedures and combined anterior-posterior approaches.

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Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 14, 31, 39, and 40 are level II studies. References 2, 3, 8, 12, 13, 15, 18, 20, 22, 25, 34-36, and 38 are level III studies. References 7, 9, 10, 16, 17,

19, 21, 23, 24, 26-30, and 32 are level IV studies. References 1, 4, 33, 37, and 41 are level V expert opinion.

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