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REVIEW ARTICLE

Cervical Spondylotic Myelopathy: Pathophysiology, Clinical Presentation, and Treatment

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Abstract Age-related changes in the spinal column result in a degenerative cascade known as spondylosis. Genetic, environmental, and occupational influences may play a role. These spondylotic changes may result in direct compressive and ischemic dysfunction of the spinal cord known as cervical spondylotic myelopathy (CSM). Both static and dynamic factors contribute to the pathogenesis. CSM may present as subclinical stenosis or may follow a more pernicious and progressive course. Most reports of the natural history of CSM involve periods of quiescent disease with intermittent episodes of neurologic decline. If conservative treatment is chosen for mild CSM, close clinical and radiographic follow-up should be undertaken in addition to precautions for trauma-related neurologic sequelae. Operative treatment remains the standard of care for moderate to severe CSM and is most effective in preventing the progression of disease. Anterior surgery is often beneficial in patients with stenotic disease limited to a few segments or in cases in which correction of a kyphotic deformity is desired. Posterior procedures allow decompression of multiple segments simultaneously provided that adequate posterior drift of the cord is attainable from areas of anterior compression. Distinct risks exist with both anterior and posterior surgery and should be considered in clinical decision-making.

Keywords cervical spine · spondylosis · myelopathy · natural history · operative treatment

Introduction

Cervical myelopathy encompasses a range of symptoms and examination findings including motor and sensory abnormalities related to dysfunction of the cervical spinal cord. Early thinking attributed the signs and symptoms of cervical myelopathy to an intrinsic dysfunction of the nervous system. It was not until the 1950s that classic anatomic studies established that spondylosis of the cervical spine is one possible etiology for compression of the spinal cord and leads to the development of myelopathic symptoms [6, 43].

The pathophysiology of CSM is now thought to be multifactorial with both static factors causing stenosis and dynamic factors resulting in repetitive injury to the spinal cord playing a role. It has been postulated that the absolute size of the spinal cord may be an important factor in the development of symptoms from CSM. The symptoms of this disorder are generally related to the degree of compression of the various spinal cord tracts. Cadaveric studies have shown that the transverse cross-sectional area of the spinal cord and the compressive ratio [compressive ratio=(sagittal diameter/transverse diameter)×100%] of the spinal cord correlate with the severity of pathologic changes [16]. In addition, autopsy examinations of patients with CSM have shown gray matter atrophy, neuronal loss, and white matter demyelination [26, 39].

The dynamic factors that contribute to the pathogenesis of CSM are complex. The spinal cord stretches with flexion of the cervical spine and shortens and thickens with

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extension. This principle was demonstrated in a classic study by Breig in which he examined the effects of spondylotic bars and positioning of the cervical spine on the spinal cord geometry and blood flow [7]. He demonstrated that extension of the cervical spine causes the cord to relax and shorten. The posterior half of the cord was found to shorten more than the anterior portion of the cord, and the axons in the posterior column assumed a spiral course and occupied a larger cross-sectional area on transverse sectioning. According to this early work, the dynamic shortening of the posterior cord and dorsal columns in extension may account for the difficulties with balance in the CSM patient.

In flexion, the stretched cord is draped over the posterior aspects of the PLL and vertebral bodies and held against any spondylotic protrusions that may be present. The axons of the cervical cord elongated and the roots are stretched. Spondylotic bars produced deep grooves on the anterior surface on the spinal cord in extension, no contact with the anterior wall in neutral position, and flattening of the cord in flexion. That is, in extension, the spondylotic bars displaced the cord, but in flexion, the cord was attenuated over the spondylotic ridge and the axons were stretched.

These studies laid the groundwork for Breig's theory that tension on the spinal cord is a key component in the development of CSM. However, he also demonstrated that the vascular supply of the cord is an important consideration. The arterioles that branch from the anterior spinal artery may be subjected to mechanical compression with flattening of the cord in the sagittal plane leading to ischemia of cord [7]. These phenomena continue to be areas of active research and form much of our modern understanding of the pathogenesis of neuron damage in CSM mediated by both ischemic and mechanical insults to the cord.

These and other cadaveric findings have been confirmed in vivo by dynamic MRI studies in patients with CSM. A significant increase of spinal stenosis has been observed in extension (48% of patients) more so than in flexion (24% of patients) [37]. A congenitally small spinal canal is also an important predisposing factor to the development of CSM. In one series of 63 patients with symptomatic CSM, it was found that 40 (63%) were found to have developmentally narrow canals [12]. Unique pathologic and kinematic traits may exist in the patient with a developmentally narrow canal. Dynamic MRI studies have shown that patients with a congenitally narrow cervical canal (<13 mm) have increased segmental mobility in the cervical spine which may in turn predispose to the development of radiographic dynamic cord compression and potentially clinical myelopathy [36].

Clinical Presentation and Evaluation

The clinical sequelae related to compression of the cervical spinal cord represent a broad spectrum. Affected individuals may have subtle clinical findings such as diminished hand dexterity or balance difficulties or in severe cases symptoms of incontinence and complete paralysis. Attempts to correlate the radiographic findings with clinical presentation have been made. However, it has yet to be clearly understood why clinical symptoms of myelopathy manifest in certain individuals with radiographic evidence of stenosis and not in others. The fact that radiographic spinal canal narrowing is often asymptomatic underscores the importance of a thorough history and physical examination in making the diagnosis of CSM.

Patients with mild symptoms of cervical spondylosis may report only neck pain and limited range of motion. Often with chronic CSM, patients may be unaware of subtle changes in balance or hand dexterity. The clinicians' history taking should elicit any difficulty with motor tasks such as clumsiness or slowness with activities such as buttoning buttons, using keys, or changes in hand writing. Difficulty with common modern tasks such as using a computer keyboard, pushing buttons on a cellular phone, or text messaging may be elicited as early signs of CSM. The recent use of assistive devices such as a cane, walker, or wheelchair due to weakness or balance issues may reflect progression of myelopathy. Often the earliest manifestations of balance problems are reported by the patient as the recent necessity to use a handrail while negotiating stairs. Paresthesias and weakness are often present in the upper extremities and patients may have concomitant radicular signs and symptoms. In severe cases of CSM, changes in bowel or bladder dysfunction may be present.

Physical examination should include assessment of balance such as heel-to-toe tandem walking, heel-walking, toe-walking, and Romberg's sign (patient stands with the eye's closed and arms held forward—loss of balance is considered a positive test consistent with posterior column dysfunction). Cervical range of motion should be tested in flexion, extension, lateral bending, and axial rotation. Cervical extension is often limited in the patient with CSM and should be documented should surgical intervention be planned to avoid iatrogenic hyperextension injury during intubation.

Tests of hand dexterity such as the “15 s grip-and-release” may be performed [24]. A normal patient should be able to grip and release their hand approximately 25–30 times in 15s, and patients with CSM may have difficulty with these rapid movements. “Myelopathy hand” is a finding in patients with CSM in which loss of motor strength, sensory changes, wasting of the intrinsic muscles, and spasticity may dramatically decrease upper extremity function [54]. The “finger escape sign” may be evident in which the ulnar two digits drift into abduction and flexion after the patient holds his or her hand with the MCPs, PIPs, and DIPs extended for a minute or more; however, the physiologic basis of this sign remains poorly understood.

Sensory and vibratory testing should be performed in the upper and lower extremities in addition to reflex examination. Patients with CSM will often be hyperreflexive in the upper and lower extremities. The pathologic reflexes are usually dependent on the spinal level of compression (e.g., a patient with cervical stenosis at C7-T1 may in fact have normal upper extremity reflexes that

originate rostral to the site of compression—biceps, brachioradialis, etc.). Abnormal long-tract signs such as the Babinski, Hoffman's, and inverted radial reflexes (tapping the brachioradialis tendon that elicits firing of the long finger flexors is considered a positive response). Of note, these clinical signs should not be relied upon solely for the diagnosis of myelopathy in the patient with stenosis. Recent studies have demonstrated a sensitivity as low as 58% of the Hoffman's sign for clinical myelopathy when the investigators are blinded [18]. There is some evidence however that the sensitivity of the Hoffman's sign is improved while performed with a dorsiflexion force to the DIP joint (compared to 20.5% in volarflexion) to 76.9%. However, the specificity of this clinical sign is thought to be higher with a volarflexion force—96.7% (compared to 78.0% in dorsiflexion) [45]. Cranial nerve examination should be performed for assessment of possible brain or brainstem lesions.

Several clinical measures of disease severity have been developed such as the Japanese Orthopaedic Association (JOA) [25], Nurick [38], and Chile's modified Japanese Orthopaedic Association (mJOA) [10] scoring systems. These popular scales provide the clinician with a metric to quantify the extent and progression of disease (Tables 1, 2, and 3). However, they are not without their limitations. The mJOA translates the Japanese functional grading of the ability to use chopsticks to a more pertinent evaluation in western cultures (ability to use fork and knife). For instance, there remains inter-observer potential for variation in these systems when assessing items such as "mild" versus "severe" sensory loss. These tools should be utilized to augment a carefully documented history and physical exam and by no means serve to replace astute clinical acumen.

Plain X-ray radiographic evaluation of the patient with CSM should include AP and lateral views. Narrowing of the disc space, facet joint arthrosis, bone spurs, ossification of the posterior longitudinal ligament (OPLL), and kyphotic alignment may be visualized on a standard lateral plane X-ray. Spondylotic changes often lead to a stiffening of the involved segments. Adjacent segments of the spine may be hypermobile to compensate for the decreased motion at the spondylotic levels. This hypermobility can result in a dynamic compression of the spinal column and may not be seen on routine MRI imaging. Therefore, flexion-extension radiographs should be included in the radiographic evaluation of the patient with CSM. Additional oblique views are useful in for visualizing foraminal narrowing. Comparison of standing radiographs to supine radiographs provides important information about the stability and motion of the cervical spine under a physiologic load.

Magnetic resonance imaging is an important part of the workup. Disc herniations, facet joint hypertrophy, folding of the ligamentum flavum, cord edema, and the sagittal diameter of the cord are indicative of the extent of the pathology. The presence of bone spurs or any ossification of the posterior longitudinal ligament (OPLL) as a source of compression are best visualized on cervical CT scan and are important for operative planning. CT myelography is a useful modality to characterize compression of the spinal cord more accurately than MRI scanning and in patients unable to get an MRI scan. Moreover, a CT myelogram may be utilized as a dynamic study—allowing the visualization of contrast flow through the CSF in flexion, extension, and lateral bending.

Radiographic attempts to characterize patients with cervical stenosis have included the Torg–Pavlov ratio (diameter of the cervical canal:width of vertebral body <0.80 is indicative of stenosis) [42]. Although the use of a ratio avoids problems with variation in magnification when measurements are made from plain radiographs, these measures were developed in a select population of 23 athletes and have since been reported to be unreliable and to correlate poorly with the true diameter of the canal [4].

An "absolute stenosis" has been defined as a sagittal canal diameter <10 mm and a "relative stenosis" as a canal diameter <13 mm, and a normal sagittal diameter in the mid-cervical spine of 17–18 mm [1, 55]. However, these absolute measurements of sagittal plane diameter are subject to genetic variation between individuals of different sizes. In his classic study, Boden reported a high false positive rate of stenosis using such absolute measurements in asymptomatic individuals. In his study, cervical canal stenosis was observed on MRI imaging in 14% of individuals less than age 40 and 28% of those over the age of age 40. Disc degeneration or narrowing was also observed at one or more levels in 25% of individuals less than age 40 and in almost 60% of those over the age of 40 [5]. As such, the diagnosis of CSM requires consideration of history, physical examination, and imaging studies for each individual patient.

Natural History

To date, the natural history of CSM has not been clearly defined, and many of the existing studies are limited. Most

Table 1 Nurick's classification system for myelopathy [38]

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

Table 2 The Japanese Orthopaedic Association (JOA) scale for spondylotic myelopathy [25]

			Points
Motor	Upper extremity	Unable to eat with spoon or chopsticks	0
		Possible to eat with spoon 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

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authors agree that there is a tendency of patients with CSM to progress to more severe disease, however, the proportion of patients that deteriorate and the rate of decline remains a subject of debate. Predominantly Class III evidence sug-

Table 3 Chile's modified Japanese Orthopaedic Association (mJOA) scale for spondylotic myelopathy [10]

			Points
Motor	Upper extremity	Unable to feed oneself	0
		Unable to use knife and fork; able to use spoon	1
		Able to use knife and fork with much difficulty	2
		Able to use knife and fork with slight difficulty	3
		Normal	4
	Lower extremity	Unable to walk	0
		Can walk on flat floor with walking aid	1
		Can walk up and/or down stairs with handrail	2
		Lack of stability and smooth gait	3
		Normal	4
Sensory	Upper extremity	Severe sensory loss or pain	0
		Mild sensory loss	1
		Normal	2
	Lower extremity	Severe sensory loss or pain	0
		Mild sensory loss	1
		Normal	2
Trunk		Severe sensory loss or pain	0
		Mild sensory loss	1
		Normal	2
Bladder function		Unable to void	0
		Marked difficulty with micturition (retention)	1
		Difficulty with micturition (frequency, hesitation)	2
		Normal	3
Total			17

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gests that patients with CSM experience stable quiescent periods with an intervening slow and stepwise decline in function [34]. However, some authors have described stable neurological dysfunction for extended periods of time in most individuals and rapid decline in others. Clarke and Robinson reported a retrospective series of 120 patients with CSM. Of those patients, 75% experienced long periods of consistent disease with a series of intervening episodes during which they experienced new symptoms and signs. Of those 75%, two out of three patients also deteriorated between episodes. An additional 20% of patients with CSM followed a slow, steady progression of disease (with one in five patients showing a late improvement). The remaining 5% showed rapid deterioration of symptoms and signs followed by long stable periods. This study was, however, limited by a heterogenous patient population and the lack of objective outcome measures [11].

Lees and Turner studied 44 patients (age 31–80 years) with radiographic cervical spondylosis and a neurologic exam consistent with myelopathy (extensor plantar responses) and characterized a pattern of progression in patients with symptoms for more than 10 years [31]. They demonstrated that very few patients have a steady progression of symptoms. The more common scenario in their study was that myelopathic disease was quiescent for periods of time with intermittent periods of rapid decline in neurological function.

Barnes and Saunders reported that the majority of patients remain stable for years with deterioration more prevalent with female gender and increased ROM [2]. A longer duration of symptoms has also been reported to portend a poor prognosis [47, 49]. More recent investigations have suggested that 62% of patients with mild CSM will not deteriorate or undergo surgery at 10 years, and that mal-alignment and instability are adverse prognostic factors [41]. One systematic review of the literature regarding the natural history of CSM suggest a mixed course with many patients having quiescent disease for long periods of time and others experiencing a slow, stepwise decline [34].

Conservative Management

Nonsurgical care for patients with radiographic evidence of cervical stenosis without clinical signs or symptoms of myelopathy is a treatment option. Such patients should be advised regarding the potential of a hyperextension force to result in spinal cord injury (e.g., central cord syndrome) with even minor trauma (such as a low-energy fall or rear-impact motor vehicle collision). Patients with mild clinical disease such as hyperreflexia or slight balance disturbance may be observed with close clinical and radiographic follow-up. The biologic and physiologic age, activity level, and functional status should all be taken into consideration when considering non-operative treatment.

Prospective studies in which patients were randomized into conservative and operative treatment groups have suggested that patients with mild CSM do not frequently progress at early time points (2–3 years) [27, 28, 32]. However, other studies have suggested that conservative therapy will be successful in preventing progression in 70% of patients at a minimum of 1-year follow-up with the remainder requiring surgery due to progressive symptoms.

Given the paucity of definitive data regarding the natural history of CSM, a discussion with the patient regarding the severity of symptoms, the degree of disability, and the potential for progression or spinal cord injury should be undertaken prior to conservative treatment. Collar immobilization could be considered in patients with radiographic evidence of severe stenosis despite mild clinical symptoms given the potential for catastrophic neurologic injury. It should be noted that even a hard cervical collar restricts cervical range of motion only a limited amount and cervical spine injury is possible in the patient with a stenotic canal despite collar usage. Potential adverse effects of prolonged collar immobilization such as muscle deconditioning and restrictions in motion due to their cumbersome nature in young active patients with mild myelopathy should be considered.

Indications for Surgery

The decision for operative treatment of CSM must take into consideration the patient's age, baseline function, rate of deterioration, severity of symptoms, and overall health. It is generally agreed upon that patients with ongoing symptoms refractory to conservative measures, those with progressive symptoms, bowel or bladder dysfunction, or overt weakness should be considered for operative intervention. The natural history studies discussed previously suggest periods of stable function with intervening episodes of rapid deterioration that exist which makes operative treatment for patients with symptomatic CSM a common practice.

Patients with CSM are generally counseled that operative treatment is most effective to prevent further decline in function, but may not result in substantial spontaneous improvement from their current level of function. Surgery may be recommended earlier for patients with myelomalacia (seen as edema within the cord on MRI) or severe radiographic stenosis. There is some data to suggest that intramedullary high signal intensity is a poor prognostic indicator for neurological recovery [52]; however, the relative importance remains controversial. In general, signal change within the spinal cord itself may not necessarily correlate with neurological function or postoperative recovery; however, it should be noted and documented as evidence of the extent of CSM pathology.

A Cochrane Review of randomized controlled trials for the role of surgery in mild CSM concluded that the early results of surgery were superior to non-operative treatment in terms of pain, weakness, and sensory loss. However, no significant differences were found at 1 year. Another trial reviewed found no significant differences between operative and non-operative care up to 2 years after treatment. Overall, the data from the reviewed trials was inadequate

to provide definitive conclusions on the role for operative treatment of CSM [15].

Surgical Techniques

There have been many different surgical techniques described for the treatment of CSM. Regardless of the surgical approaches chosen, the goal of operative treatment is the decompression of the spinal cord (while preserving alignment and stability) with consideration of both static and dynamic factors. Surgical intervention in patients with moderate to severe myelopathy can be expected to halt the progression of symptoms. Fujiwara et al. [17] analyzed the factors involved in the prognosis of CSM in 50 patients by CT myelography and found that in patients with a transverse area of the cord <30 mm^2, the results were poor. Age and preoperative neurological function were also found to play a role on outcome. Patients with larger transverse area of the cord, younger patients, patients with shorter duration of symptoms, and single rather than multiple levels of involvement can be expected to have a better prognosis with surgical intervention [17, 30].

Special consideration is given to the positioning of the patient with CSM. Documentation of the preoperative cervical range of motion is essential. The spondylotic patient with a tight cervical canal may sustain severe neurological injury if excessive extension is achieved during intubation on the operating table. Fiber-optic intubation is a useful adjunct in the CSM patient with limited extension. Discussion between the surgeon and anesthesia team will permit safe intubation, induction of anesthesia, and positioning.

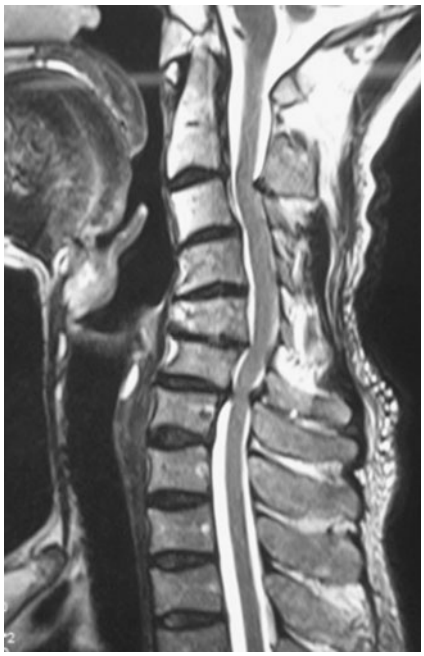

Fig. 1. A patient presenting with CSM with predominantly C5–C6 and C6–C7 stenosis on sagittal T2-weighted MRI scan and a regional fixed cervical kyphosis from C4–C6

Broadly speaking, surgical options may be divided into anterior, posterior, or combined anterior and posterior approaches. The choice of approach remains a source of active debate among practitioners. Factors involved in the decision-making progress include sagittal alignment, number of diseased segments, stenosis morphology, history of prior surgery, and bone quality. The overall cervical spine alignment in the sagittal plane is thought to be an important consideration when choosing between an anterior and posterior approach. Traditionally, in the setting of a fixed cervical kyphosis, an anterior procedure or combined anterior and posterior procedures have been performed (Fig. 1). In the presence of a fixed cervical kyphosis, an anterior procedure will allow direct decompression of the spinal canal in addition to indirect decompression by correction of the deformity. A posterior procedure in this setting has the theoretical risk of leaving the spinal cord compressed anteriorly against any spondylotic bars or disc bulges in the kyphotic segment of the spine. Postoperative MRI scanning has demonstrated that posterior migration of the thecal sac following laminectomy may be inadequate to clear compression from anterior osteophytes in patients with neutral to kyphotic alignment and may account for some instances of poor outcome after laminectomy for CSM [3].

A recent large prospective observational multicenter study of 280 patients sought to address the issue of anterior versus posterior surgery [13]. At 12-month follow-up, patients with CSM showed significant improvements in both disease-specific and general health-related outcome measures with either anterior or posterior surgery (mJOA, Nurick scale, NDI, and SF-36). Patients treated with anterior surgery generally had more focal pathology, limited to a few levels, compared to patients treated with posterior surgery.

There have been reports of patients with myelopathy being treated by one-level [51] and multiple-level [50] cervical disc arthroplasty. Although several studies to date have shown the equivalence of cervical disc arthroplasty and arthrodesis for the treatment of cervical radiculopathy

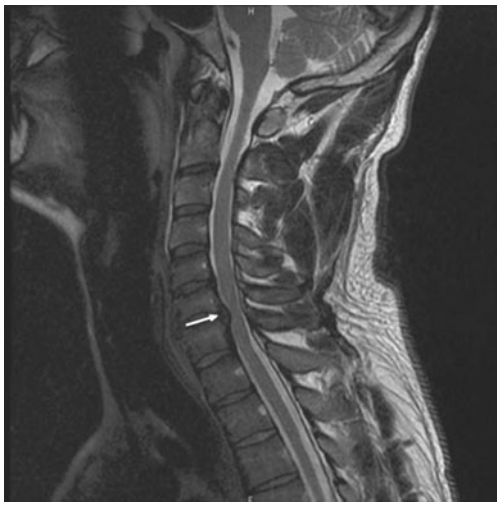

Fig. 2. Sagittal T2-weighted MRI scan of a patient with predominantly C6–C7 stenosis, retrovertebral compression at C6 and C7, and preservation of cervical lordosis

[21, 44, 48], there remains a concern that motion preservation at the myelopathic spinal segment may allow continued microtrauma to the spinal cord. Recent multicenter cross-sectional studies have shown similar improvements in neurological status in patiens undergoing arthrodesis and arthroplasty at the 2-year time point [46]. Despite early encouraging results, the future role that motion preservation will play in the myelopathic patient remains to be determined and is still considered investigative at the present time.

Anterior procedures include single or multilevel anterior cervical discectomy and fusion (ACDF) or hemi-corpectomy or corpectomy and fusion depending on the desired regions of decompression. Anterior surgery is generally recommended for patients with disease limited to a few segments (Fig. 2) and patients with a fixed cervical kyphosis. Choice of interbody graft and cervical plate for graft containment for ACDF procedures is chosen based on the individual patient and surgeon's preferences. Commerically available allograft interbody spacers avoid the donor site morbidity associated with conventional iliac crest harvesting. Synthetic interbody spacers are also an option, most commonly composed of a PEEK (polyether–etherketone) polymer, carbon fiber, or a PEEK/carbon fiber hybrid material. Autograft through open or percutaneous iliac crest aspiration offers the theoretical benefit of improved biology for enhancing a fusion. With regard to plate choice, locking plates (in which the screw head is threaded and forms a "fixed-angle device" with the plate) may prevent graft subsidence in older patients with osteoporotic bone whereas variable angle plates will allow some settling of the graft for compression at the graft site once a patient is upright and the spine experiences a physiologic load. Newer dynamic anterior cervical plates offer a design that allows load sharing between the vertebral body and the plate despite subsidence of the interbody bone graft [8, 9]. Although a clinical advantage of dynamic plate stabilization over locked plate fixation has not been clearly established, both techniques have inherent risks of late instrumentation-related complications.

Patients with predominantly dorsal compression due to ligamentum flavum infolding and patients with multisegmental stenotic disease may be better suited for a posterior procedure in the setting of a neutral to lordotic alignment. Posterior procedures for CSM include central decompression alone by removal of the posterior elements (laminectomy), decompression and fusion (laminectomy and fusion), and decompression by hinging open of the lamina (laminaplasty). Laminaplasty is a relatively motion-preserving procedure in which the dorsal arch of the lamina is fixed by plate, wire, cable, spacer between the lamina and the lateral mass, or suture technique to maintain its protective role over the dorsal thecal sac.

Complications of Surgery

In addition to complications of bleeding, infection, nerve damage, dural leak or fistula seen with any spinal surgery, unique risks exist with anterior and posterior procedures for CSM. ACDF or anterior corpectomy by conventional Smith-Robinson approach carries the risk of recurrent laryngeal nerve injury, superior laryngeal nerve injury, esophageal injury, vertebral artery injury [53], hardware breakage or migration, pseudoarthrosis, graft dislodgement, Horner's syndrome, and airway compromise [14]. Most patients undergoing anterior cervical surgery will experience moderate dysphagia that is usually self-limited and should be made aware of this preoperatively.

Laminectomy for the treatment of CSM has been associated with reports of neurologic re-stenosis, or the development of a "post-laminectomy" membrane [56] and soft tissue impingement on the unprotected thecal sac remains a possibility. Post-laminectomy kyphosis is a potential complication following multilevel posterior decompression procedures [35]. Fusion should be considered in patients undergoing laminectomy with any pre-existing instability or in patients in whom a wide decompression is desired. Fusion procedures in the cervical spine (either anterior or posterior) carry the theoretical risk (albeit controversial) of adjacent segment degeneneration (ASD). Symptomatic ASD has been shown to occur at an incidence of 2.9% per year and survivorship analysis predicts that 25% of patients will develop ASD within 10 years after an ACDF for instance [23]. A portion of these patients will require revision surgery for symptomatic ASD [19, 20]. Risks unique to laminaplasty include premature lamina closure after open-door technique with resultant re-stenosis [33] and axial neck pain [40]. Cervical nerve root palsy (most commonly C5 palsy) remains a risk predominantly of posterior cervical decompression [29] but has also been reported to occur following anterior decompression [22].

Summary

Age-related changes in the spinal column result in a degenerative cascade with resultant disc dessication, facet joint hypertrophy, ligamentum flavum infolding, and kyphotic collapse. Genetic, environmental, and occupational influences may play a role in this degenerative process. These spondylotic changes may result in direct compressive and ischemic dysfunction of the spinal cord known as CSM. Both static and dynamic factors play a role in the pathogenesis and should be considered when considering treatment options. CSM may present as subclinical stenosis seen on imaging studies or may follow a more pernicious and progressive course. Most reports of the natural history of CSM involve periods of quiescent disease with intermittent episodes of neurologic decline. If conservative treatment is chosen for mild CSM, close clinical and radiographic follow-up should be undertaken in addition to precautions for trauma-related neurologic sequelae with even low-energy impact. Operative treatment remains the standard of care for moderate to severe CSM and is most effective in preventing the progression of disease. Anterior surgery is often beneficial in patients with stenotic disease limited to a few segments or in cases in

which correction of a kyphotic deformity is desired. Posterior procedures allow decompression of multiple segments simultaneously given adequate posterior drift of the cord is attainable with both anterior and posterior surgery and should be included in the clinical decision-making.

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