

Cervical Degenerative Disc Disease and Cervical Spondylotic Myelopathy

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The cervical spine is the most complicated articular system in the body, with 37 separate joints (including six intervertebral discs). The normal cervical spine moves 600 times per hour, whether the individual is awake or sleeping (2). In order to understand the pathomechanics of discopathy, one must understand normal function of the intervertebral discs. The intervertebral discs contribute approximately 22% of the length of the cervical spine. The discs function to allow motion between vertebral bodies in all planes of motion. The deformability of the intervertebral disc allows for the distribution of forces over the entire surface area of the vertebral body endplate rather than focusing loading and torsional forces at the periphery of the vertebral body (6,11). The gelatinous nucleus pulposus acts as a hydrodynamic ball bearing that converts the vertical pressures of axial loading to horizontal forces that can be absorbed by the annulus fibrosis. When axial forces are applied to the cervical spine, the annulus acts elastically (although no elastic fibers are found within the annulus) to provide for an increase in the radius of the disc. As axial forces expend themselves, the distance between the vertebral endplates decreases and the disc's radius increases. With the aging process, cervical mobility decreases and the degree of expansion on axial loading increases. Thus, the disc loses its energy absorbing capacity and sustains mostly vertical loads (11). Cervical degenerative joint disease is a dynamic process that begins with an insult to the intervertebral disc and ends at a variable point between the initial insult and the pathology associated with advanced cases of cervical spondylotic myelopathy.

Cervical Spondylosis

Cervical spondylosis is one of the most common conditions related to the cervical spine that is treated by the chiropractic physician. The symptoms related to cervical spondylosis can run the gamut from dull neck pain with no attendant neurological or orthopaedic findings to cervical spondylotic radiculopathy and cervical spondylotic myelopathy. This discussion will be directed to the more severe aspect of cervical spondylosis: cervical spondylotic myelopathy. Cervical spondylosis is said to be the most common cause of acquired spastic paraparesis in middle and late life (6). According to Rothman, cervical spondylosis is the most common cause of spinal cord dysfunction in patients over 55 years of age (3). The condition is said to be the single most underdiagnosed spinal disorder (16). Spurring in middle age and beyond is

almost a universal finding, occurring in 90% of males over the age of 50 and in 90% of females over 60 years of age (14). Similarly, radiographic evidence of spondylosis is present in 25% to 50% of the population by age 50 and in 75% to 85% of the population by 65 years of age (16).

Natural History of Cervical Disc Degeneration

At least 10% of the population recalls having pain in the neck at least three times within the past year and at least 35% of the adult population can recall at least one episode of neck pain (2). Rothman (3) stated that "it does not appear that cervical disc degeneration is a brief, self-limiting disorder but rather a chronic disease, productive of significant pain and incapacity over an extended period of time". Gore studied 205 patients for a minimum of 10 years and found that over one third of the patients studied had moderate to severe neck pain at final evaluation, thus confirming Rothman's hypothesis (1). Fifty-one percent of the adult population will experience neck and arm pain at some point in their lifetime. Predisposing factors to cervical intervertebral disc problems are heavy lifting, smoking, diving, operating heavy equipment that has a great deal of vibration, and driving (4).

In general, an acute presentation of neck pain in a young patient most likely indicates a disc extrusion. The mean age for cervical disc herniation is 37 years of age, with the incidence rate for males equal to that of females (10). The most common affected discs and nerve roots are as follows in order of decreasing frequency (11):

First	C5/C6 disc	C6 root
Second	C6/C7 disc	C7 root
Third	C4/C5 disc	C5 root
Fourth	C3/C4 disc	C4 root
Fifth	C7/T1 disc	C8 root

Greater than 80% of patients with acute neck pain as the result of disc herniation denied a history of previous cervical trauma. Approximately 85% of cases are classified as "soft" disc ruptures, 11% are "hard" disc protrusions, and 4% are soft central disc bulges (10). The term "soft disc" refers to nuclear material or a bulging annulus, while the term "hard disc" refers to the presence of an osteophyte around the bony confines of the intervertebral foramen, the posterior aspect of the vertebral body, or a prolapsed fibrotic annulus fibrosis (6). It has been noted that posterior osteophytes are always associated with evidence of disc degeneration and other degenerative changes (1). Bland indicated that any radiculopathy is a consequence of zygapophyseal joint abnormality and not a consequence of the uncinat process or the disc. He further indicates that after age 40-45, all the dural root sleeves become fibrotic and stiff and come

to the forefront in cervical radiculopathy as a causative agent (2).

In terms of treatment for cervical radiculopathy, the greater the treatment, the worse the result. There is no difference statistically between patients treated operatively and patients treated non-operatively for the same condition at 5 years. Therefore, there is the strong implication that in dealing with predominant neck pain in the absence of a neurological deficit and discrete radicular symptomatology, the results of surgery do not significantly alter the natural course of the disease (4).

Natural History of Cervical Spondylosis

Cervical spondylosis with myelopathy is a condition which has a very prolonged course marked by episodic worsening with long periods without new or worsening symptoms. Overall, the onset is insidious and the disease course is one of stepwise degeneration over time. Episodic exacerbation can occur at longer or shorter intervals for many years, with the symptomatic episodes being prolonged for weeks to months, then in the majority of cases, subsiding. A progressively deteriorating course is the exception (14,15,16). The degree of disability is established early in the course of cervical spondylotic myelopathy and, in many cases, does not change with time (15,16,20). An exception to this is seen in patients with cervical spondylotic myelopathy who were treated operatively. These patients most frequently worsened as the post-operative follow-up period lengthened (19).

The average age of onset of symptoms is 49 years, with a range of 14 years to 70 years, and 75% of the patients being between 40 and 59 years of age (15). The mean onset of symptoms prior to admission has been shown to be 45 months, with a range of one month to 313 months. In one series, 22% of the patients tolerated their symptoms for 5 to 25 years prior to admission (19). In another series, the typical patient was 53 years of age, with the range of ages in the study being between 35 and 80 years of age (16).

Clark and Robinson found that no cervical spondylosis patients ever returned to their normal state. 75% of their series of patients had episodic worsening, 20% showed steady progression, and 5% had a rapid onset of symptoms and signs followed by a lengthy disability. A spontaneous remission was highly uncommon (21). Natural history studies have failed to demonstrate factors that identify the patient at risk for rapid progression (17).

In spondylosis, it is difficult to differentiate between the effects of acute trauma and the chronic trauma of weight bearing and mobility that come under the heading of wear

and tear. Only when a single interspace can be shown to be hypermobile and when osteoarthritic changes develop at the interspace after the passage of months to years, can the changes be attributed to a single episode of trauma with any certainty (14).

The prognosis for cervical spondylosis is good, provided that the condition is recognized early, the appropriate treatment is rendered, and the patient is told how to cope with his disability. The effects of conservative care are difficult to assess in this relatively benign condition, in which prognosis may be good with or without treatment. Treatment of the cervical spondylosis patient may alleviate symptoms without influencing the course of the disease's natural history (15,16).

The following radiographs demonstrate the natural progression of cervical spondylosis from 1980 to 2006 in the same patient:

1980



2006



Pathogenesis of Cervical Spondylotic Myelopathy

Cervical spondylotic myelopathy is defined not as a single temporal or pathological entity, but as the result of a combination of degenerative events of which the initial lesion is the deterioration of the intervertebral disc (26). Cervical spondylotic myelopathy results from the interactive effects of a combination of pathological factors (11,25):

- Spondylotic transverse bars
- Hypertrophic apophyseal joints
- Pre-existing and acquired canal diameter
- Changes in the sagittal canal diameter with motion in conjunction with spurs producing irritation and compression of the spinal cord
- Interruption of the intrinsic arterial supply to the spinal cord
- Differences in spinal cord sensitivity to hypoxia
- Canal shape

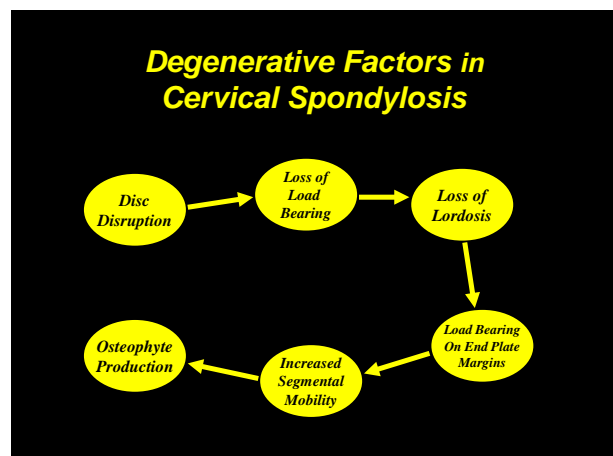
In order to understand the complex pathology of cervical spondylotic myelopathy, one must develop an understanding of each of the factors involved: the mechanical factors related to the cervical spine and spinal cord, the degenerative factors, and the vascular factors.

Degenerative Factors: Cervical Spondylotic Myelopathy

One of the precipitating factors linked to the development of cervical spondylotic myelopathy is cervical intervertebral disc degeneration. With aging, the nucleus pulposus becomes dehydrated and shrinks, and the annular function becomes less that of concentric constriction and axial load dissipation and more that of weight bearing. Thus, more segmental mobility results at the affected segment due to stretching and compression of the intervertebral disc and the degenerating disc loses its ability to distribute its load to the entire endplate and load bearing becomes the function of the margins of the vertebral body (11,14).

In a study of 20 cases of cervical spondylotic myelopathy, the anterior part of the disc was found most often to have degenerated with the resultant loss of the smooth lordotic curve (28). The loss of disc height leads to the loss of lordosis and the creation of segmental intervertebral flexion which leads to the approximation of the vertebral bodies. The mechanical compromise precipitated by the degenerating disc triggers a reactive process that produces osteophytes at the superior and inferior attachments along the periphery of the discs and the vertebral bodies. The osteophytes function to increase the surface area of the endplates, thereby reducing the force on them and to decrease motion at the degenerating joint when the osteophytes abut or spontaneously fuse. These changes are noted at the sites of greatest wear: the lower cervical spine (11,14,25,26,28,33,40).

Degenerative Factors in Cervical Spondylosis



The greatest decreases in sagittal diameter over time are at the C5/C6 and the C6/C7 levels. Decreases in sagittal diameter at these levels correlate significantly with the development or increased size of posterior osteophytes and/or disc space narrowing (1). In addition to marginal

osteophyte formation around the periphery of the disc and vertebral bodies, the uncinate joints, the apophyseal joints, the pedicles, and the laminae, also hypertrophy due to the altered biomechanics. Due to these changes, the intervertebral foramina may also become deformed and neural changes may result (11,14,33).

The most common sagittal canal diameter in the cervical region between C3 and C7 is between 17 millimeters and 18 millimeters. The degenerative process can lead to a reduction in the sagittal spinal canal diameter. Most compressive symptoms are generated in the C5 to C7 region due to the fact that the spinal canal is the smallest in this region and the spinal cord, which averages 10 millimeters in its anteroposterior diameter (with a range of 8.5 to 11.5 millimeters), occupies 3/4 of the canal in the normal cervical spine at the C6 level. In contrast, the spinal cord only occupies 1/2 of the spinal canal in the upper cervical region. It is generally felt that a sagittal canal diameter of 12 millimeters or less is critical to the development of myelopathy (25,26). Robinson investigated the sagittal canal diameters associated with the type of symptoms and signs manifested by the patient with cervical spondylosis. He found that patients presenting with spondylotic radiculopathy had sagittal cervical canal diameters averaging 11.8 millimeters and those with cervical spondylotic myelopathy had canal diameters ranging from 9 to 15 millimeters (36).

Cervical Spondylotic Myelopathy: Mechanical Factors

The spinal canal lengthens 2.8 centimeters from full extension to full flexion due to the posterior position of the neural canal in relation to the sagittal axis of flexion and extension. The spinal canal achieves its greatest sagittal diameter in flexion. In extension, the spinal canal's anteroposterior diameter may decrease by up to 2 millimeters and the ligamentum flavum, which tends to infold in extension, may further decrease the anteroposterior canal diameter (25,26,27,28,64). Panjabi and White have identified a number of elements involved statically and dynamically in the production of cervical spondylotic myelopathy (62):

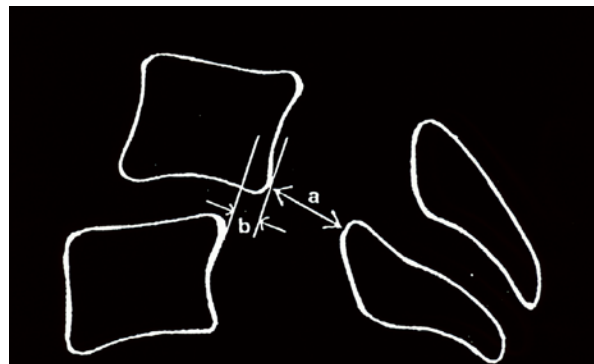
Static Factors in Cervical Spondylotic Myelopathy

- Small cervical canal
- Osteophytes
- Disc herniation
- Ossification of posterior longitudinal ligament
- Uncinate hypertrophy
- Apophyseal joint deformation/inflammation
- Hypotonic ligamentum flavum

Dynamic Factors in Cervical Spondylotic Myelopathy

- Motion: Normal and abnormal
- Loading: Normal and abnormal
- Mechanical properties of the spinal cord
- Mechanical properties of the spinal canal
- Hypotonic ligamentum flavum

Dynamic canal stenosis is one of the factors governing the pathomechanics of cervical spondylotic myelopathy caused by cervical instability. Posterior slide (retrolisthesis) of the vertebral body occurs primarily as a result of degeneration of the cervical spine due to aging changes. The sagittal canal diameter at the affected level decreases with the increase of the degree of posterior slide, followed by gradual aggravation of the clinical symptoms (23). In a series of 42 cervical spondylotic myelopathy patients, 27 of 42 demonstrated a static canal diameter measuring less than 13 millimeters while 40 of 42 demonstrated dynamic canal stenosis measuring less than 13 millimeters at least one disc level (60). With respect to dynamic cervical canal stenosis, Fukui (23) noted the distance of posterior slide, the level of posterior slide, and the number of interspaces that posterior slide was observed in spondylosis.



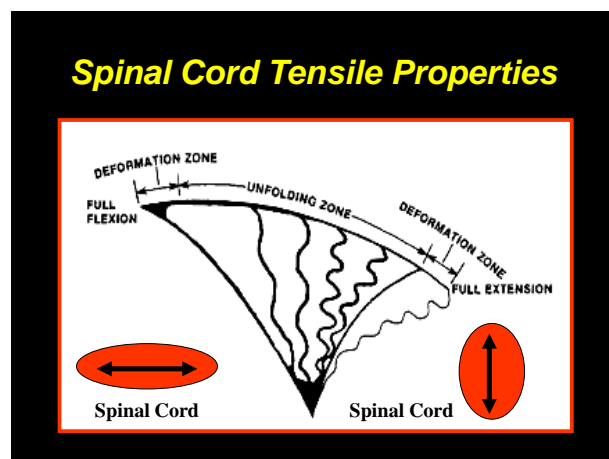
Hayashi (60) studied factors involved in dynamic cervical canal stenosis and found that posterior osteophytes were involved 30.7% of the time; primarily at C5/C6 and C6/C7. Retrolisthesis was seen 36.4% of the time; primarily at C3/C4 and C4/C5. He further noted a combination of posterior osteophytes and retrolisthesis in 19.3% of patients. The major contributing factors to spinal cord compression were disc protrusion, posterior osteophytes, retrolisthesis, and lastly, a combination of posterior osteophytes and retrolisthesis.

The number of levels of the cervical spine involved was as follows:

<u>Level of Lesion</u>	<u>Number of Patients</u>
1 level	4 patients
2 levels	9 patients
3 levels	13 patients
4 levels	14 patients
5 levels	2 patients

Spinal Cord: Mechanical Factors

According to Brieg (28), due to the cranial attachment of the dura at the foramen magnum and the caudal attachment at the coccyx via the filum terminale, *the spinal cord cannot be considered in isolation but must be treated as a continuous tract of nervous and supporting tissues*, from the mesencephalon to the conus medullaris. The static and dynamic properties of the spinal cord constitute a self-contained biomechanical compartment that is slackened in extension and stretched elastically in flexion. The cord not only moves up and down within the canal during flexion and extension but is also stretched by about 10% in full flexion (18,26). In extension, the spinal cord folds upon itself much like an accordion and widens in its anteroposterior dimension. Distinct folds are noted on the posterior surface of the spinal cord in extension. Encroachment of the ligamentum flavum may also contribute to the infolding. During flexion, the spinal cord first unfolds, with a minimum increase in tension, then it is followed by some elastic deformation near full flexion of the cervical spine. In flexion, the spinal cord also widens in its lateral dimensions (46).

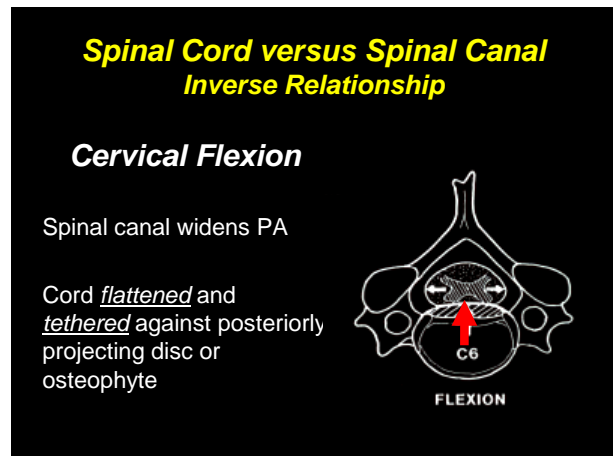


During flexion and extension movements, the axons and blood vessels of the spinal cord undergo deformation similar to that of the cord as a whole. It is evident that during postural changes in the living subject, the presence

of an unyielding pathological structure, whether located anterior to the cord (such as an osteophyte) or within it (such as a tumor), will result in incorrect loading of the nervous tissue and its abnormal deformation, particularly during the stretching phase which occurs during flexion of the cervical spine.

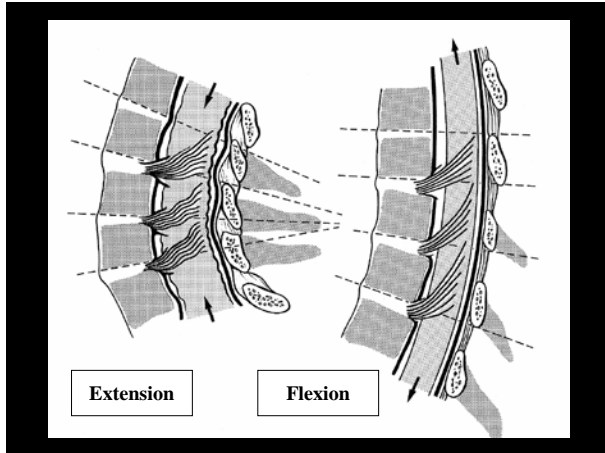
Thus, cervical spondylotic myelopathy is a neurological disorder having its origin in the tension developed in the spinal cord tissue, and the resulting distortion of both nerve fibers and blood vessels. Flexion of the cervical spine creates increased axial tension throughout the entire spinal cord, not just in the cervical region. This increased tension would force the spinal cord versus the anteriorly situated osteophytes produced due to disc degeneration. The thrust versus the osteophytes sets up local bending stresses in the cord tissue, the transverse effect of which is increased by the anchoring effects of the dentate ligaments, thus potentiating anterior cord compression.

The osteophytes encroaching upon the spinal canal may also displace the spinal cord posteriorly or posterolaterally. Additionally, focal arachnoiditis may result from the localized compression (27,28,33). When the spine is placed in extension, the thickened laminal arches and infolded ligamentum flavum may also create posterior cord compression (33).



The forces produced by the interaction of the spinal cord versus the spondylotic bar (osteophyte) reduce the spinal cord's anteroposterior diameter and force it to be wider than normal in its lateral dimension. These forces result in stretching of the lateral branches of the segmental spinal arteries which nourish the lateral columns and gray matter and may contribute to the vascular component of the disorder (27,28). The effects of compression on the spinal cord are additive, with two or more levels of minimal compression having the same effect on the spinal cord as one level of moderate compression (40).

Cord Movement in Flexion and Extension
(Alf Brieg MD)



Cervical Spondylotic Myelopathy: Vascular Factors

The vascular component of cervical spondylotic myelopathy is considered central to the disease's pathophysiological process. The vascular compromise may result at any point from the vertebral artery origin of the radicular arteries to the spinal cord itself. The spinal cord receives its blood supply from the anterior spinal artery, which is a longitudinal anastomotic chain formed by the junction of the terminal ascending and descending radicular arteries. The anterior spinal artery supplies the anterior 2/3's of the spinal cord. The two posterior spinal arteries supply each dorsal column respectively. The anterior spinal artery receives its blood supply from 2 or 3 medullary feeder arteries in the cervical region. The C1-C4 levels of the spinal cord are serviced by intracranial sources and do not contribute to the vascularization of the lower cervical spinal cord appreciably. This leaves the C5 to T1 levels of the cervical cord to be nourished almost exclusively by the variable number of medullary feeder arteries originating from the vertebral artery and traversing through the intervertebral foramen with the nerve root to reach the spinal cord. The most frequent site for medullary feeder artery occurrence is at the C6 level, with the C3 level being second, and the C4 or C5 levels being the third most probable locations for the feeder arteries to enter the spinal canal from the vertebral artery (24,26, 43).

Spinal Cord versus Spinal Canal Inverse Relationship

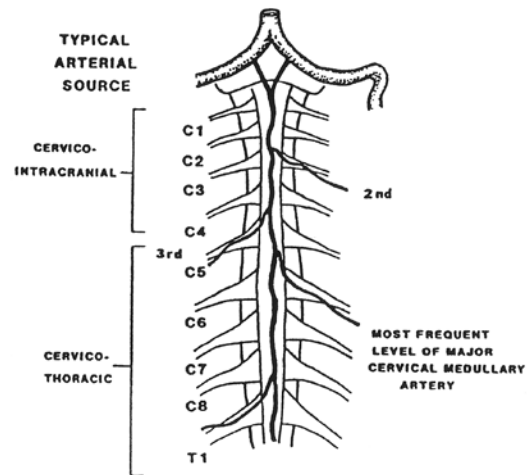
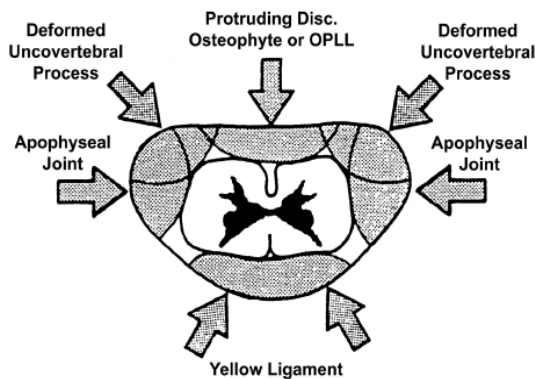
Cervical Extension

Cervical cord widens in PA dimension

Spinal canal narrows due to retrolisthesis and infolding of ligamentum flavum



Cord Compression Factors



Taylor noted that the patients with cervical spondylosis who develop myelopathy are those in whom important radicular arteries have become compressed at some point between the vertebral artery and the spinal cord, the compressing agents being either the osteophytes which encroach on the intervertebral foramen, or the thickened, hyalinized fibrous tissue which surrounds the nerve roots (36,39). Fibrosis and adhesion formation is a common

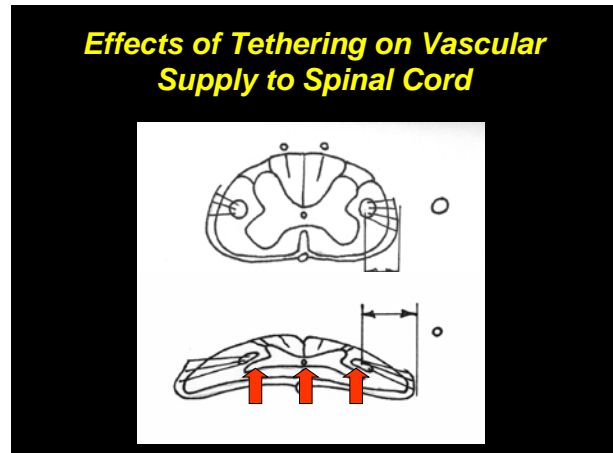
finding in the degenerating spine around the nerve root. Motion and repetitive root trauma caused by the fibrosis in and around the dural sleeve could interfere with the blood supply to the spinal cord (35,36).

Gooding noted that root sleeve fibrosis associated with degenerative changes of the zygapophyseal joints and intervertebral joints could cause spasm of the lateral spinal arteries and their branches. The vasospasm may result from direct stretching and kinking of the arteries during movements of the cervical spine or it may be the result of nociceptive stimuli mediated by sensory nerves from the joints and the autonomic pathways. These insults could result in reflex arterial spasm and paralysis of local segmental arterial autoregulation and adversely affect the spinal cord's blood supply. In an area where compression already exists, such a mechanism could cause cord ischemia, and if frequent or prolonged, result in myelopathy (35,41).

The spinal cord, as well as the entrapped nerve roots, may undergo ischemic changes in addition to compression injury. The thin walled, fragile, radicular veins may be most vulnerable to the effects of trauma and compression. Partial or complete obstruction of veins can cause raised venous pressure within the cord itself, with subsequent edema and possibly ischemia due to reduced blood flow and stagnation. Compression and ischemia are additive insults, affecting the spinal cord together at degrees of severity, which would have minimal effects on the cord if invoked separately (40). Experimentally, it has been demonstrated that at least 40% compression at one level is necessary to produce minimal neurological signs in lab animals. When the compression is applied at two or three levels, the same signs of neurological dysfunction may be produced with only a 25% reduction of the sagittal diameter at each level. Therefore, compression at multiple levels also has an additive effect and explains the clinical observation that multisegmental osteophytes of moderate size are more frequently found in patients with myelopathy (40,41,45).

As it has been demonstrated, in cervical spondylosis the arterial blood flow may be reduced in the vessels supplying the cervical cord, thus setting the stage for ischemia precipitated by the mechanical stresses applied to the vessels within the spinal cord. The long penetrating branches of the lateral arterial plexus that supplies the lateral columns are narrow. Little blood flow would occur in these vessels if they were lengthened and flattened by stresses which widen the cord laterally and flatten the cord anteroposteriorly opposite a spondylotic ridge during flexion. This positional abnormality would result in inadequate perfusion to the gray matter of the central spinal cord and the lateral columns, which contain the

lateral corticospinal tracts. Demyelination may result in the lateral columns as a result of pressure and oxygen deprivation to the oligodendroglia, the Schwann-type cells within the spinal cord that are responsible for production of the myelin sheath for the axons of the neuronal cells (25,28,36). Experimentally, hyperflexion of the cervical spine produces a transverse necrotic zone in the ventral half of the gray matter. This location is the site where the transverse vessels of the cord's arterial system are maximally elongated when the cord is compressed from the anterior as shown by Brieg (37).



It is unlikely that direct compression of the anterior spinal artery is the sole precipitating factor in the production of cervical spondylotic myelopathy. Obstruction of the anterior spinal artery in animal models only produces a pathological change in the ventral one third of the spinal cord, and not in the lateral columns which are most frequently involved in cervical spondylotic myelopathy in human subjects (25).

Systemic arterial hypertension may enhance the production of myelopathic symptoms. Systemic arterial hypertension that is 70 mm Hg above normal increases spinal cord degeneration and capillary congestion. High blood pressure increases the spinal cord congestion at the site of compression and secondarily enhances the spinal cord degeneration observed in compression myelopathy (37).

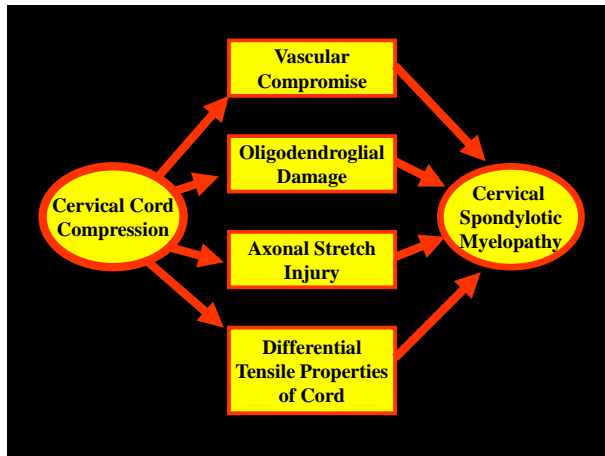
Additionally, many investigators agree that the spinal cord has an intrinsic autoregulation mechanism for its blood flow that functions within a range of arterial pressure between 70 and 150 mm Hg. Both ischemic and compressed spinal cords demonstrate lower tissue oxygenation than the normal spinal cord (44). The spinal cord also demonstrates a differential sensitivity of cord structures to anoxia. Short periods of anoxia caused by a temporary total cord lesion from which the sensory tracts

recovered completely and the motor tracts recovered incompletely. Motor neuron conduction is abolished 1.5 times faster by anoxia than interneuron conduction, and four times faster than posterior column nerve conduction (48).

Surgically, dilatation of spinal cord blood vessels was noted in 16 of 20 patients treated operatively for cervical spondylosis and cord compression. Swollen nerve roots were observed in 7 patients out of 20, and cord displacement was noted in 12 of the 20 patients along with increased tension imparted by the dentate ligaments (33).

According to Parke (26), a purely neuroischemic myelopathy without direct pressure on the cord could occur by a chance foraminal compression of an essential medullary feeder artery. The resultant symptoms could be consistent with the typical thrombotic cervical anterior spinal artery syndrome (Beck's Syndrome) in which the upper extremities demonstrate a flaccid paralysis and areflexia, while the lower extremities present as a spastic diplegia. There is concomitant sensory dissociation below the level of the lesion as well.

Pathogenesis of Myelopathy in CSM



Cervical Spondylotic Myelopathy Possible Pain Generators

The pain presentation of the cervical spondylotic myelopathy patient may be confusing due to the possibility of multiple pain generators contributing to the patient's perceived pain. Possible pain generators in cervical spondylosis may include any or all of the following in varying combinations:

- Local facet or discogenic pain
- Local muscular pain

- Trigger point referred pain
- Referred pain from facet joints or intervertebral discs
- Radicular pain
- Myelopathic pain

Cervical Spondylotic Myelopathy Neurologic Exam

The most common symptoms associated with cervical spondylotic myelopathy are:

- Dysesthesia in the hands
- Weakness and clumsiness in the hands with resultant deteriorating handwriting
- Weakness in the lower extremities. The weakness in the lower extremities may lead to the patient complaining of the legs feeling stiff and the patient may have to shuffle when he or she walks.
- Patients may also lose confidence in their abilities to walk and may be afraid of falling.

Of the three most common subjective complaints, gait and lower extremity related complaints are the most common. Gait may worsen later in the course of the disease if the dorsal columns become affected. The combination of a loss of dorsal column proprioception with muscle wasting and weakness leads to very unsteady ambulation and a wide-based spastic gait. These patients may require the use of external supports for walking (7,11,16,51).

The following signs and symptoms were noted in a series of 35 cervical spondylosis patients (30):

• Nerve root symptoms and signs	26
• Long tract symptoms and signs	25
• Neck pain	11
• L'hermitte's sign	7
• Impotence/sphincter disturbance	7
• Brown-Sequard Syndrome	3
• Horner's Syndrome	2

In another series of cervical spondylotic myelopathy patients cited by Clark (51), the following symptoms were identified:

• Radicular pain	39%
• Paresthetic pain	34%
• Spasticity	98%
• Weakness	65%

Thus, most of the symptoms reported are related to compression of the spinal cord, with radicular symptoms presenting at a less frequent rate than myelopathic symptoms and signs. In addition, cervical spondylotic myelopathy patients may complain of urinary frequency or

urgency, however, bladder incontinence is less commonly seen (11).

Occipital headache that is located on one or both sides and radiates through the parieto-temporal regions to the frontal region is a well known associated feature of cervical spondylosis and is seen in up to one third of all cervical spondylotic myelopathy patients. The headaches are typically worse upon awakening and subside during the day. The actual mechanisms postulated to be involved in the production of the headache are posterior cervical muscle spasms, lower cervical osteoarthritis, upper cervical joint degenerative changes, and intersegmental movement abnormalities of the upper three cervical apophyseal joints (7,11,58,75).

In addition to headaches, the cervical spondylotic myelopathy may also exhibit vertebrobasilar arterial insufficiency symptoms due to encroachment upon the vertebral arteries by osteoarthritic, hypertrophied uncinat processes. Associated symptoms may include dizziness, constriction in the neck, diplopia, tinnitus, and a "feeling of separation from the environment" (11,24).

MacNab (22) stated *if the osteophyte developing on the uncovertebral joint extends laterally the vertebral artery may become kinked. This may produce a so-called vertebral artery syndrome consisting of dizziness, which may be just a sensation of unsteadiness, or there may indeed be a true vertiginous component. The patients complain of tinnitus, which is usually associated with some loss of hearing in the upper range. In addition to these symptoms, the patient may complain of intermittent blurring of vision and occasional episodes of retro-ocular pain. Occasionally a neurocentral osteophyte may produce kinking of the artery resulting eventually in a vertebral artery thrombosis.*

The following associated symptoms and signs were seen in a study of 100 cervical spondylosis patients (43):

Brachial radiculitis	32
Headache	28
Vertigo	17
Myelopathy	13
Neck pain	13
Vertebrobasilar arterial insufficiency	5
Attacks of unconsciousness	4
Drop attacks	3
Acute lesions of the spinal cord	3

Sensory Findings

Sensory abnormalities are most commonly seen in the upper extremities and most symptoms and signs are primarily due to nerve root involvement. Patients may experience paresthesias and pain at multiple levels. Numbness and tingling may be present at the tips of the fingers, often referenced to a radicular distribution. The patients may also exhibit a decrease in light touch, pinprick, pain, and temperature sensation in the upper extremities. When the lower extremities are involved, decreases in sensation may be seen in the lateral aspects of the legs and feet. The symptoms of radiculopathy and myelopathy often co-exist or overlap. Dorsal column involvement is not common. *If the patient exhibits a loss of vibratory and proprioceptive sense there is the likelihood of a poor prognosis as dorsal column involvement is only seen in the most advanced stages of cervical spondylotic myelopathy (7,11,16,51,64).*

Motor Findings

Gait is often worse late in the course of the disease, with the patient exhibiting a wide-based, spastic gait. Ambulation is slow, shuffling, ataxic, and the patient frequently requires the use of external supports in walking. Upper extremity findings are usually noted unilaterally while it is common for the patient to display bilateral lower extremity involvement, with one leg often being affected more than the other. There are variable degrees of wasting in the upper extremity that are dependent upon the segment of the spinal cord and/or the nerve root level(s) involved. Decreased strength and fasciculations may be seen in the upper extremities (7,11,16,51). The cervical spondylotic myelopathy patient also exhibits motor findings associated with the hands. The *finger escape sign* is noted in the patient who has deficient adduction, extension, or both in the ulnar two or three digits of the affected hand (7,47).

Ebara, et al (52) described the *myelopathy hand* associated with cervical spondylotic myelopathy as one in which there is diffuse or localized muscle wasting of the extrinsic or intrinsic hand muscles. The muscle weakness, often appearing as a deficiency of extension of the fingers, was mostly seen unilaterally. If the patient presented with bilateral involvement, there was much discrepancy between the right and left hands. Sensory findings in these patients were disproportionate to motor findings, most often being minimal or absent. The myelopathic hand patients most often had sagittal cervical spinal canal diameters below 13 millimeters and multisegmental spondylosis.

Deficient hand closing is also characteristic of cervical spondylotic myelopathy patients. The normal individual can open and close the fist at least twenty times in a 10-

second period. The myelopathy patient has much difficulty performing this maneuver. The opening and closing of the fist is typically slow, difficult, and incomplete (7,16,47). Patients complaining of numb and clumsy hands should be checked for the presence of spinal cord compression between the foramen magnum and the C5 level (16).

Reflex/Pathological Reflex Findings

Cervical spondylotic myelopathy patients typically exhibit hyperreflexia in the lower extremities. Babinski's sign may be present late in the course of the disease as the degenerative changes proceed and the magnitude of spinal cord compression increases. Superficial reflexes may be absent. In the upper extremities, the deep tendon reflexes may be normal, decreased, or exaggerated, depending upon the level of involvement.

Inverted Radial Reflex

The *inverted radial reflex* may be present when cord and nerve root compression are present at the C5 and C6 levels. The reflex is seen during testing of the brachioradialis reflex. As the brachioradialis tendon is struck with the reflex hammer at the distal end of the radius, a diminished response is noted in the brachioradialis along with a reflex contraction of the spastic finger flexors, hence the term "inverted radial reflex" (7,11,51).

Dynamic Hoffman's Sign

Hoffman's sign, elicited by nipping the nail of the middle finger and observing a reflex contraction of the thumb and index finger, is evidence of an upper motor neuron lesion. In the normal patient, the Hoffman's sign is absent. Recently, a dynamic element has been suggested to add to the Hoffman's sign, which may increase the ability of the clinician to detect early spinal cord involvement in cervical spondylosis (50). It was noted that if the Hoffman's sign was checked during active flexion and extension of the cervical spine as tolerated by the patient, that the sign could be elicited in subjects that did not elicit the sign in the static, resting position. Clonus may be present at the wrist, patella, and ankle, depending upon the level and magnitude of compression (7,11,51).

L'hermitte's sign, characterized by a radiating "electric shock-like sensation" that courses through the trunk, may be elicited by flexion or extension of the cervical spine (11).

Dysphagia may be present due to anterior osteophyte encroachment upon the anteriorly situated esophagus (7,11,51).



Imaging in Cervical Spondylotic Myelopathy

Plain film radiography of the symptomatic cervical spine patient is a beneficial, relatively inexpensive, and readily available diagnostic tool. Because patients who are completely asymptomatic may have severe degenerative changes on radiographic examination, the cervical spine series must be considered to be an extension of the history and physical examination and should be evaluated in light of the patient's symptoms and signs (76).

Teresi, et al (77) demonstrated the widespread occurrence of degenerative changes in asymptomatic patients through the use of magnetic resonance imaging in a group of asymptomatic patients with evidence of cervical degenerative disc disease. They found that a wide variety of abnormalities may be seen in asymptomatic patients, particularly the elderly. They noted the following:

- Disc protrusion may be seen in 57% of patients between under 45 and 55 years of age

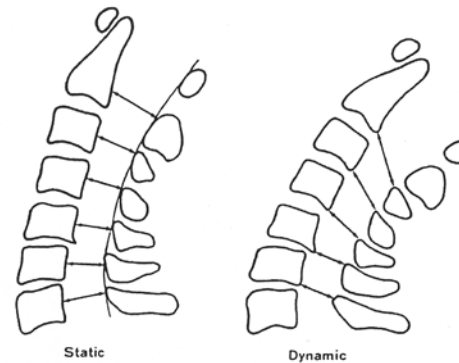
- Disc protrusion was seen in 57% of patients under 64 years of age
- Posterolateral disc protrusion were noted in 9%
- Spinal cord impingement was seen in 16% under 64 years of age and in 26% of those over the age of 64
- Spinal cord compression was identified in 7%

Teresi also saw that the percentage of cross-sectional area reduction never exceeded 16% in asymptomatic patients and averaged approximately 7%. Long tract signs generally were not manifested until there was a 30% reduction in the cord's cross-sectional area.

The lateral cervical view is the single most important film for the evaluation of the degenerative spine, providing a wealth of information. The lateral view is useful in assessing the intervertebral disc spaces, osteophytosis, cervical spine curvature and alignment, apophyseal joints, and bony sagittal cervical canal diameter measurements. The oblique views may be utilized to assess the degree of encroachment upon the intervertebral foramen. Osteophytes are not thought to physically encroach upon the exiting nerve roots until they obliterate more than 50% of the foraminal volume (76).

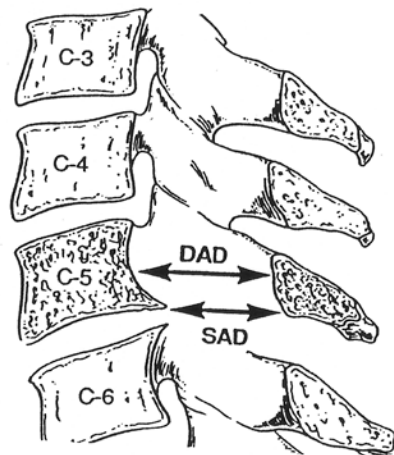
Sagittal spinal canal diameter in the lower cervical spine averages 17-18 millimeters in the normal subject. Most compressive symptoms are generated in the C5 through C7 levels due to the fact that the cervical spinal cord occupies 3/4 of the canal at this level in the normal patient (25,26). Sagittal canal diameter is best assessed via the lateral cervical radiograph taken at 72 inches, non-bucky. Many experts feel that since there is the propensity for dynamic canal stenosis, the sagittal canal diameter should also be assessed on flexion and extension lateral cervical radiographs as well (26,36,55). The minimal sagittal spinal canal diameter considered to be normal in the lower cervical region is 13 millimeters. Sagittal canal diameters less than or equal to 12 millimeters are considered to be critical to the development of myelopathy. It should be noted that the spinal cord's average anteroposterior diameter is 10 millimeters, with a range from 8.5 millimeters to 11.5 millimeters (26). The measurement of most use in the assessment of sagittal canal diameter is the *functional sagittal canal diameter* (36). The functional sagittal canal diameter is assessed by measuring the distance between the tip of the most prominent osteophyte on the posterior border of adjacent spondylotic vertebrae to the superior aspect of the spinolaminar junction line of the inferior vertebra. This measurement provides a better evaluation of the space available for the spinal canal's contents. This measurement should be assessed on the flexion and extension views as well in order to compare static and dynamic findings (36).

Static and Dynamic Spinal Canal Assessment



The *segmental stenotic index* has been used to identify patients that may be at risk for the development of cervical spondylotic myelopathy (62). The segmental stenotic index is calculated by assessing the difference between the developmental anteroposterior diameter (measured from mid vertebral body to mid lamina) and the spondylotic anteroposterior diameter (a.k.a functional sagittal canal diameter). A segmental stenotic index of 1.5 millimeters or more may indicate a patient at risk for the development of cervical spondylotic myelopathy.

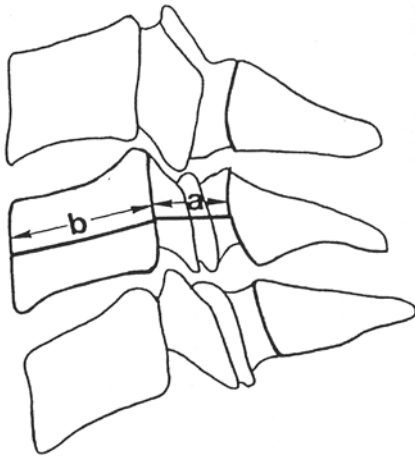
Segmental Stenotic Index Assessment



Viedlinger (30) described a magnification factor that is useful when considering sagittal canal diameter measurements. Due to the width of the shoulders between male and female patients, 10 millimeters is approximately 11.18 millimeters in the male and 11.11 millimeters in the female. Therefore, one should consider that mensurations made on 72-inch lateral cervical radiographs are, in reality, anatomically smaller than actually measured.

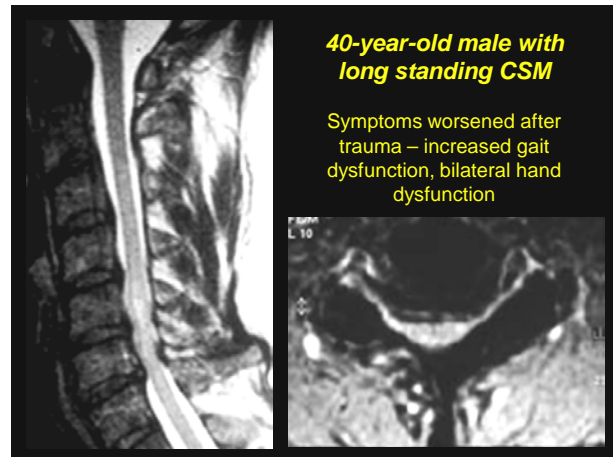
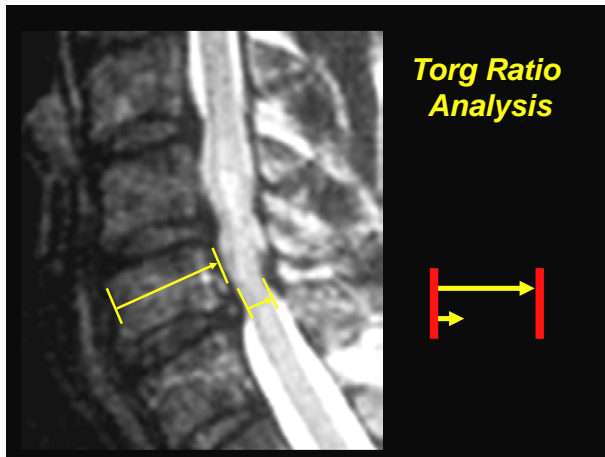
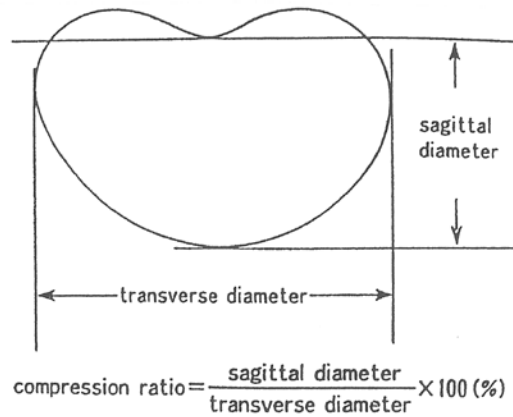
As a general rule of thumb the spinal canal can be related to the size of the articular pillars (71). Approximately 1/3

of the canal should be visible posterior to the articular pillars. Also, the anteroposterior diameter of the spinal canal in the cervical region should be approximately equal to the anteroposterior diameter of the vertebral body at that level. Thus, a normal canal should have a ratio of approximately 1:1. Torg (42) utilized the ratio of canal size to vertebral body size in assessing patients at risk for spinal cord injury due to a stenotic spinal canal. The ratio of the spinal canal to the vertebral body is the distance from the mid-point of the posterior aspect of the vertebral body to the nearest point on the corresponding spinolaminar junction line divided by the anteroposterior width of the vertebral body. Utilizing this method, a measurement of less than 0.80 indicates significant stenosis, as normal spines demonstrate a measurement closer to 1.00.

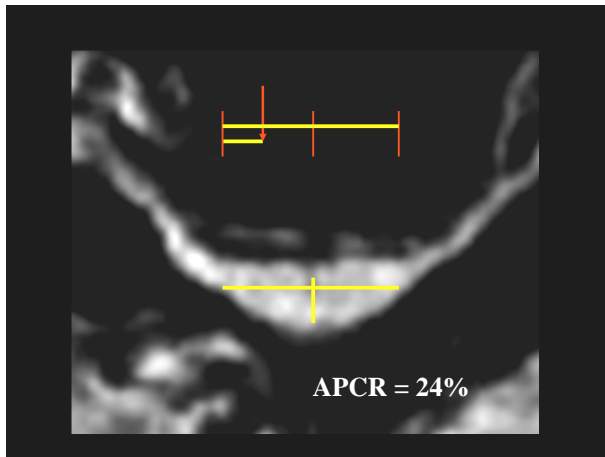


considered abnormal. The AP compression ratio has been studied by Ogino (31). He assessed the APCR at the most damaged spinal cord segment and found that when the APCR was 40%-45% (which is nearly in the normal range) that there was flattening of the gray matter of the spinal cord and mild, localized demyelination of the lateral corticospinal tracts. An APCR of 22%-39%, which is significantly reduced, was associated with rarefaction of the gray matter, formation of small cavities and neuronal loss in the gray matter, and diffuse demyelination of the lateral corticospinal tracts. When the APCR measured between 12% and 19% there was extensive gray matter necrosis and cavitation. There was also significant necrosis and gliosis within the lateral columns and dorsal columns.

Anteroposterior Compression Ratio (APCR)

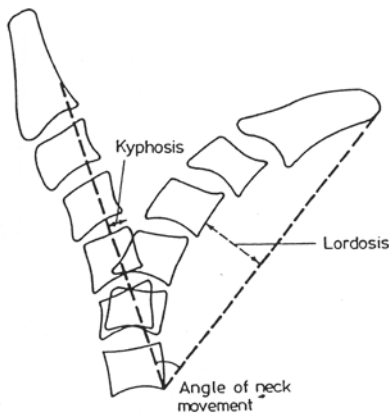


The AP compression ratio (APCR) is a measurement of the cross-sectional area of the spinal cord that is obtained from a CT scan with contrast. The normal APCR is at least 45%. Percentages for the APCR less than 40% are



In a series of 45 patients studied by Saunders (18), cervical range of motion in the cervical spondylosis patient was found to be an indicator of a patient at risk for deterioration and further disability. Saunders found that when the total range of motion (as assessed through the method illustrated below) was greater than 62 degrees the patient's condition was more likely to deteriorate. When the total range of motion was less than 62 degrees, the disability tended to remain nonprogressive. This measurement may prove of use in identifying those patients whose condition may not be amenable to an extended course of conservative care.

Total Range of Motion Measurement Technique



Shirasaki, et al (57) studied factors which predisposed the patient to instability after anterior cervical fusion surgery and identified two structural characteristics that predisposed the patient to instability post-surgically: the pre-operative segmental range of motion, and the facet angle. When the segmental range of motion at the level adjacent to the site for the proposed fusion is already great, it will be further increased by compensatory motion after

surgery, leading to disc degeneration and spondylotic changes. The facet joint acts to restrain anterolisthesis by its angle of inclination, or facet angle. A facet joint cannot act to restrain anterior slippage if the facet angle is large. Thus, facet angle and segmental range of motion should be considered in all surgical fusion candidates.

Magnetic Resonance Imaging of Cervical Spondylotic Myelopathy

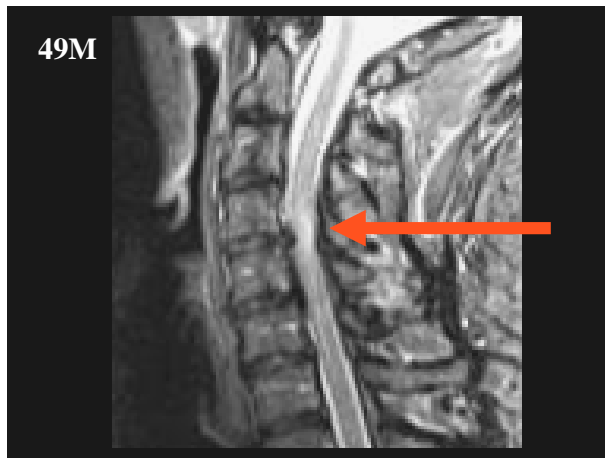
Magnetic resonance imaging is of value in assessing the soft tissue structures associated with the cervical region. However, the use of MRI may not fully demonstrate the nature and extent of the lesion or lesions associated with cervical spondylotic myelopathy due to the static nature of the test and its inability to assess the cervical spondylotic myelopathy patient in the flexed and/or extended postures.

Considerable attention has been given to the use of MRI in identifying intrinsic cord pathology secondary to cord compression resulting from extradural space-occupying lesions. Takahashi et al (132) studied MRI scans from 128 patients with compression lesions in the cervical spinal canal to determine whether a high signal intensity lesion within the spinal cord was present on T2-weighted images. The study revealed the presence of high signal intensity in 18.8% of the patients and was generally observed in patients with constriction or narrowing of the spinal canal. The pathophysiological basis for the presence of the high signal intensity on the T2-weighted images was hypothesized to be myelomalacia or cord gliosis secondary to long-standing cord compression.

Giammona et al (137) studied MRI scans from 56 patients with cervical spinal cord compression and noted increased signal intensity on T2-weighted images in 21.4% of the study group. This subgroup also demonstrated evidence of a longer mean duration of symptoms, and, in most patients, more serious clinical conditions. Mifsud and Pullicino (140) studied the frequency and cause of T2-weighted spinal cord hyperintensity through an analysis of 273 cases of cervical spondylosis and 36 cases of cord compression secondary to cervical spondylosis. The found increased signal intensity on T2-weighted images present in 19 (7%) of 273 scans from cervical spondylosis patients and in 19 (53%) of 36 scans of patients with cord compression. The authors noted that the presence of T2 spinal cord hyperintensity related to the severity of the cervical spondylosis.

Wada et al (93) studied fifty patients with cervical spondylotic myelopathy through the use of computed tomographic myelography and magnetic resonance imaging before and after surgical intervention to identify the recovery rates and clinical parameters suggestive of

poor outcome. They found that patients with multisegmental areas of high signal intensity on T2-weighted magnetic resonance images tended to have poorer results. Post-operative computed tomographic myelography indicated that the multisegmental areas of high signal intensity on T2-weighted MRI probably represented cavitation in the central spinal cord. Bucciero et al (136) reviewed magnetic resonance images from 35 cervical spondylotic myelopathy patients whose films demonstrated increased signal intensity at the most compressed segment of the cervical spinal cord on T2 and proton density weighted images. All patients underwent surgery and were subsequently re-evaluated via MRI. Postoperatively, the high MR signal intensity disappeared in 3 cases, decreased in 20 cases, and did not change in the remaining 12 cases. The reversible increased signal intensity on T2-weighted images was attributed to edema or transient ischemia. Irreversible changes were attributed to myelomalacia or gliosis of the spinal cord.



High intensity zone in patient with myelopathic symptoms

Wada et al (107) also performed a retrospective study of MRI and computed tomography after myelography in cervical spondylotic myelopathy to determine if the presence or absence of high signal intensity areas in the spinal cord were correlated with clinical symptoms and surgical outcomes. Seventy-four percent of patients in the study demonstrated high intensity areas on T2-weighted MRI. Patients with multisegmental (linear) high intensity areas frequently manifested muscle atrophy in the upper extremities. Multisegmental (linear) high intensity areas on T2-weighted MRI scans were associated with clinical evidence of extensive anterior horn cell damage and radiographic evidence of gray matter cavitation.

Ratliff and Voorhies (94) studied increased T2-weighted signal intensity in patients with cervical myelopathy and found a rapid resolution of radiographic abnormalities after surgical decompression and fusion. However,

clinical improvement did not parallel radiographic resolution.

Kameyama et al (95) studied the outcome from laminoplasty for cervical spondylosis to determine the efficacy of MRI and motor evoked potentials for surgical indications and prognosis. The authors studied 31 cases of cervical myelopathy for which surgery was considered. Twenty-one patients had cervical spondylosis and ten patients suffered from ossification of the posterior longitudinal ligament. Magnetic resonance imaging revealed the deformed spinal cord in the involved area, and the signal intensity of the involved spinal cord in the T2-weighted image was remarkably high. The signal intensity ratio was significantly higher in the poor-outcome patients than in the good outcome patients. The authors concluded that a high signal intensity in the T2-weighted image and a prolonged conduction time or absence of motor-evoked potentials largely corresponded to the clinical and other investigative features of myelopathy responsible for a poor outcome.

Kameyama et al (98) studied intramedullary high-intensity lesions observed on T2-weighted MRI in patients with cervical myelopathy due to cervical spondylosis and ossification of the posterior longitudinal ligament. The pathophysiological basis for the increased signal intensity was presumed to vary from acute edema to chronic myelomalacia. They considered the intramedullary lesion to be the main site of the lesion responsible for the patient's abnormal signs and symptoms because of the correlation between the neurological level of involvement and the level of the spinal cord affected by high signal intensity.

In a third study, Kameyama et al (133) studied the MRI scans from three patients with cervical spondylotic amyotrophy, a clinical syndrome in cervical spondylosis characterized by severe muscle atrophy in the upper extremities with an absent or insignificant sensory deficit. The patients in this study demonstrated segmental muscular atrophy in the upper extremities with insignificant sensory findings. Sagittal T2-weighted magnetic resonance images demonstrated multi-segmental linear high signal intensity within the cord at the compression sites. The areas of high signal intensity appeared to be located in the region of the anterior horns on the axial images. Cord compression was noted to be less severe when the neck was in the neutral position and spinal canal stenosis was increased when the neck was extended. The pathophysiological mechanism was attributed to multisegmental damage of the anterior horns caused by dynamic compression, which possibly created a circulatory insufficiency.

Al-Mefty et al (138) reported on 18 cases in which the magnetic resonance imaging demonstrated two distinct types of lesions in patients with cervical spondylotic myelopathy. In the first type, localized spinal cord changes at the level of compression (consistent with myelomalacia) were revealed as areas of high signal intensity on T2-weighted axial images. The second type of lesion (consistent with either cystic necrosis or secondary syrinx formation) was best characterized as a local and/or multisegmental lesion that extended longitudinally up and/or down within the spinal cord on T2-weighted images.

Histological studies of patients with spinal cord compression and the presence of increased signal intensity on T2-weighted images were performed (135). The study revealed distinct differences in the severity and extent of pathological changes based on the shape of the compressed spinal cord as noted on axial images. The cords were classified into two groups: boomerang-shaped or triangle-shaped cross-sections. Boomerang-shaped cords demonstrated major pathological changes restricted to the gray matter, with relative preservation of the white matter. Secondary wallerian degeneration was restricted to the fasciculus cuneatus, the fibers of which were derived from the affected segments. Triangle-shaped cords exhibited more severe pathological changes that extended over more than one segment, and both descending degeneration of the pyramidal tracts and ascending degeneration of the posterior columns, including the fasciculus gracilis.

Differential Diagnosis in Cervical Spondylotic Myelopathy

A differential diagnosis of cervical spondylotic myelopathy should include the following conditions:

- Multiple sclerosis
- Amyotrophic lateral sclerosis
- Spinal cord tumor
- Syringomyelia
- Subacute combined degeneration
- Cervical disc extrusion
- Cerebral hemisphere lesion
- Low-pressure hydrocephalus

Meyer and Sandovss (146) noted that the differential diagnosis between cervical spondylotic myelopathy and multiple sclerosis or other neurological deficits may be difficult. They reported on 4 patients with presumed chronic cervical spondylotic myelopathy who also had unsuspected multiple sclerosis.

Burgerman et al (147) reported on six patients who were affected by both multiple sclerosis and cervical spondylosis. The authors noted that diagnostic evaluations of patients with symptoms of cervical spondylosis (particularly younger patients) should include consideration of the possible coexistence of multiple sclerosis and the evaluation of the patient with known multiple sclerosis who develops new signs of cervical spinal cord dysfunction should always include spinal neuroimaging studies. Bashir et al (182) reported on 14 patients with multiple sclerosis and coexisting spinal cord compression secondary to cervical spondylosis or cervical disc disease.

Differentiation of MS from CSM

	Cervical Spondylotic Vascular Myelopathy	Multiple Sclerosis
Onset	A few hours	Slower
Duration of Relapses	A few hours to 1-2 days	Days to weeks
Clinical Presentation	Same each time but may be more severe with repeat episodes	Variable clinical picture
Symptoms and Signs	Mainly motor	Mixed motor and sensory

Syringomyelia has been associated with cervical spondylotic myelopathy and should be included in a differential diagnosis. Yu and Moseley (149) retrospectively studied 88 cases of proven syringomyelia to explore the relationship between syringomyelia and cervical spondylotic myelopathy. They found that there was no significant increase in incidence of syringomyelia with cervical spondylotic myelopathy but the coexistence of the two conditions may contribute to the progression of cervical spondylotic myelopathy in some patients. The coexistence of syringomyelia and cervical spondylotic myelopathy has been reported, and syringomyelia may be a complicating factor to cervical spondylotic myelopathy (150,151,152).

Treatment of Cervical Spondylotic Myelopathy

Lees and Turner (15) indicated that cervical spondylotic myelopathy is a disease that is best treated through very

conservative therapy. McCormack and Weinstein (100) noted that the results of conservative treatment in most cases of cervical spondylosis are so favorable that surgical intervention is not considered unless pain persists or unless there is progressive neurological deficit. Hunt outlined conservative and surgical treatments for cervical spondylotic myelopathy and indicated that, for the most part, a balance between judicious exercise to maintain muscular and ligamentous tone that is supplemented by rest, brief periods of immobilization, salicylates, and symptomatic therapy (massage, heat, counterirritants) is the treatment of choice (14). Hunt noted that a "failure of conservative care" must be defined in terms of time, since many weeks or months may be required before symptoms subside; yet it is probable that the patient who has recovered with an unoperated spine is better off than the patient who has had a spinal fusion.

Truumees and Herkowitz (169) indicated that a trial of nonsurgical management is recommended in most patients with radiculopathy or mild myelopathy. Surgical intervention should be reserved for patients with neurological complaints and in whom non-surgical measures have failed, as well as those patients with more pronounced myelopathy.

Wang et al (178) studied 56 patients with cervical spondylotic myelopathy and found that conservative management resulted in 35% improvement and operative management resulted in 43% improvement. They indicated that if a patient has received conservative treatment, routine re-evaluation is essential for the early detection of progressive compression myelopathy signs. Lehman (180) commented that conservative therapy helps in one-half of cervical spondylotic myelopathy patients and surgery may be beneficial in up to 75% of patients.

Physical therapy, according to MacNab (22) does not reverse degenerative changes. Loy (131) assessed the relative efficacies of physiotherapy and electroacupuncture in the treatment of cervical spondylosis and found that, while both methods were effective, electroacupuncture produced an earlier symptomatic improvement with increased neck movement. Electroacupuncture was most effective in patients with mild degenerative changes in the cervical spine. Cervical traction has the greatest value during the acute phase of the lesion.

Swezey et al (99) studied the effects of home cervical traction therapy on patients with mild to moderately severe cervical spondylosis. The patients in the study utilized over-the-door cervical traction 5 minutes per

session, twice per day. Over-the-door home cervical traction therapy provided symptomatic relief in 81% of the patients. Prolonged physical therapy, especially if it involves taking more than two or more hours away from activities of daily living, may be more disabling than the disease itself (22).

Cervical collars may be beneficial to the patient because they discourage cervical extension, they provide the patient with a "rest" position during activities of daily living, and they may possibly provide an analgesic effect due to the firm pressure that the collar exerts versus the skin of the neck.

McCormack and Weinstein (100) indicated that because many patients have nonprogressive minor impairment, neck immobilization is a reasonable treatment in cervical spondylotic myelopathy patients presenting with minor neurological findings or in whom operative treatment is contraindicated. They indicate that the use of collars will result in improvement in 30% to 50% of patients. Misra and Kalita (154) studied the effect of collar therapy in 20 patients with cervical spondylotic myelopathy through the use of motor evoked potentials. Patients were evaluated following 6 and 12 weeks of collar therapy. Walking difficulties were present in 18 patients and leg weakness ranged between 2/5 strength and 4/5 motor strength on a zero to 5 motor grading scale. Joint position sense was impaired in 14 patients. They found that one Nurick's grade of improvement occurred in 12 patients and two Nurick's grades of improvement occurred in 3 patients at six weeks. The authors also noted that improvement was more pronounced at 6 weeks as compared to 12 weeks after collar therapy.

Chiropractic management of the cervical spondylotic myelopathy patient should be directed in a "full spine" approach. Degenerative changes, instability in the mid-cervical region, and neurbiomechanical hazards may preclude the application of high-velocity, low-amplitude techniques. Attention should be given to postural abnormalities exhibited by the patient. Treatments may include stretching and strengthening exercises as well as spinal manipulation above and below the involved segments of the cervical spine.

Brieg (27) indicated the importance of a postural approach to patient care. Brieg noted that the magnitude of the tension developed in the spinal cord depends firstly on the anatomical factor of body posture, which determines the relative lengths of the spinal canal and spinal cord. Conductivity and circulation can often recover by eliminating tension due to the structures impinging on the spinal cord and the

exiting nerve roots resulting from postural alterations. Total tension induced by the pathology may lead to neural dysfunction with the most significant consequence of overstretching of the spinal cord's nerve fibers being impairment of conductivity. It would seem logical that if postural alterations can impair cord function, then postural training may improve cord function.

Extension traction has been advocated for the use in the functional restoration of the cervical lordosis in patients who have had a loss of the normal cervical curve. This procedure involves the sustained use of traction that holds the cervical spine in an extended posture. From a neurological perspective, patients with a congenitally small cervical spinal canal and patients with acquired cervical spinal canal stenosis secondary to spondylosis are not candidates for this procedure. This therapy is contraindicated because of the biomechanical interaction of the vertebrae comprising the cervical spinal canal, the ligamentous support structures of the region, and the spinal cord residing within the cervical spinal canal. Extension causes the cervical spinal canal's sagittal diameter to decrease in the lower cervical region while simultaneously causing the spinal cord to fold down upon itself and widen in its anteroposterior dimension. In the patient with a normal cervical sagittal canal diameter (17-18 millimeters) the sustained extension traction position should not be hazardous. However, in the case of the patient with spinal canal compromise due to congenital stenosis or acquired central canal stenosis, the extension traction procedure may be neurologically detrimental to the patient, as this position may produce spinal cord compression (23,24,74). A patient with a sagittal canal diameter of 13 millimeters or less should not be subjected to the procedure.

Brieg noted that the pincer action set up in extension can set up short range axial tension in the tissue of the cervical spinal cord. The pincer action is produced by osteophytes approaching the posterior wall that consists of the dura, the ligamentum flavum, and the vertebral arches. It occurs when a large osteophytes projects into the cervical canal of normal size or when a relative small osteophytes projects into a shallow canal. The localized tension developed by the pincer action can be so great as to cause tearing of the nerve fibers and even blood vessels.

The hazards of prolonged hyperextension to the cervical spondylosis patient have been described in literature concerning myelographic procedures in the cervical region (65). Prolonged hyperextension of the head and neck, necessary to prevent the escape of oil into the

intracranial cavity, is often painful and dangerous because of the maximal impingement on the cord produced by the hyperextension maneuver. Greene et al (101) reported on a case in which gentle cervical hyperextension caused quadriplegia in an older man with symptomatic cervical spondylosis. Whiteson et al (109) reported on a patient who suffered compressive myelopathy secondary to passive hyperextension of the cervical spine during a dental procedure. The patient had been asymptomatic prior to the procedure and subsequent imaging revealed the presence of advanced spondylosis and spinal stenosis. The authors noted that hyperextension of the cervical spine with spondylosis changes can lead to compression myelopathy. They recommended preventive measures for individuals with asymptomatic spondylosis changes and education of all health-care professionals to avoid abrupt or prolonged hyperextension of the cervical spine.

Anesthesiologists have identified the hyperextension position as a position hazardous to the cervical spondylosis patient. Hyperextension, or excessive manipulation of the neck during intubation for anesthesia should be avoided. It is conceivable that, during the induction of anesthesia, hyperextension may further damage a spinal cord that has already been displaced or compromised by posterior osteophytes. This additional damage may result in damage that is permanent (33). Additional reports indicate that blanching of the spinal cord can be produced by hyperextending the cervical spine during surgical procedures for cervical spondylosis myelopathy, thus demonstrating the sensitivity of the spinal cord to circulatory alterations in the presence of a compromised cervical spinal canal (34). Treatment in extension may also be detrimental to exiting nerve roots (59). Thus, the physician seeking to utilize extension traction in the treatment of cervical curve reversals should carefully screen each patient and rule out contraindications for the procedure.

Surgical Treatment

Surgical treatment of cervical spondylosis myelopathy is unstandardized and associated with immediate, as well as long-term complications. Congenitally narrow canals, as well as compensatory subluxation of vertebrae above spontaneously fused segments, may predispose the patient to myelopathy (62). The most difficult and dangerous decompressions occur in the patient with the congenitally narrowed spinal canal, especially when the spine is straight. Less posterior decompression can be achieved in the straight or kyphotic spine than in the spine with a normal curvature (14). Batzdorf found that the patient with the relatively

normal cervical curvature demonstrated the greatest improvement in symptoms and signs postoperatively (53).

The patients with moderate to severe disability at the time of the initial evaluation are probably the best candidates for aggressive treatment, as the chance for improvement through a protracted regimen of conservative care is negligible. Good prognosis for surgery is associated with the young patient, symptom duration of less than one year, unilateral motor deficits, Lhermitte's sign, and a limited number of levels of involvement (16,17,19,43). According to Law et al (174), absolute indications for surgery are: progression of signs and symptoms, presence of myelopathy for six months or longer, and a compression ratio approaching 0.4. Naderi et al (176) indicated that age, abnormal cervical curvature, and the presence of pre-operative high signal intensity within the spinal cord on T2-weighted magnetic resonance imaging studies tend to predict less post-operative neurological improvement.

Anterior Discectomy and Fusion

There is a general tendency toward improvement in disability after anterior interbody fusion. Many anterior discectomies with fusion have been carried out with immediate success, but long-term complications related primarily to accelerated degeneration of adjacent discs occur (14,19,65,66). Following cervical laminectomy there is a tendency for late worsening of the patient's disability, which may occur up to 8 to 12 years after the initial period of post-operative improvement and plateau (19, 65). Indications for anterior decompression are cases in which there is anterior compression at one or two levels and no significant developmental narrowing of the spinal canal (174).

Disadvantages to anterior discectomy and fusion include the following (66):

- Neurological injury, including radiculopathy, recurrent laryngeal nerve palsy, and permanent myelopathy
- Hoarseness
- Graft extrusion
- Graft donor site pain
- Lateral femoral cutaneous nerve injury
- Pseudoarthrosis

Early surgical follow-up studies indicated that there was a tendency toward resorption of posterior osteophytes after successful spinal fusion in the cervical spine. DePalma and Cooke reported that in 58% of their cases with posterior osteophytes, there was radiographic evidence of complete resorption (66). Yang reported

the presence of posterior osteophytes on radiographs of all 69 patients in his series, with resorption or blunting of posterior osteophytes noted in one-third of patients at follow-up. He found that an average of 21 months was required for resorption to occur (23). However, evaluation of bone spurs via radiographs is difficult to perform properly due to positioning problems (68).

Chiles et al (164) reported on 76 patients treated with anterior cervical decompression and fusion by one surgeon and noted that strength improved at rates of 80-90% in individual muscle groups after anterior cervical decompression. However, fewer than half of all patients experienced functional improvement in the lower extremities. Sinha et al (171) reported on the outcome of anterior discectomy in 105 cases of cervical spondylosis. He found that 89% showed improvement, 4% were unchanged, and 7% worsened. He felt that anterior cervical discectomy had less risk of overall complications than decompressive laminectomy.

Another complication of anterior cervical decompression and fusion centers around the production of symptoms and signs at areas adjacent to the fusion site due to instability induced in those levels as a consequence of the fusion. Chiles (164) noted that recurrent symptomatic spondylosis at unoperated levels was calculated to occur at an incidence of 2% per year. Hilibrand et al (168) studied the incidence, prevalence, and radiographic progression of symptomatic adjacent-segment disease, which was defined as the development of new radiculopathy or myelopathy referable to a motion segment adjacent to the site of previous anterior cervical arthrodesis. They noted that symptomatic adjacent-segment disease occurred at a relatively constant incidence of 2.9% per year during the ten years post-surgery. The risk of new disease at an adjacent level was significantly lower following multilevel arthrodesis than it was following single-level arthrodesis. Thus, symptomatic adjacent-segment disease may affect more than one-fourth of all patients within ten years after an anterior cervical arthrodesis. The greatest risk factors for new disease appeared to be a single-level arthrodesis involving the fifth or sixth cervical vertebra and pre-existing radiographic evidence of degeneration at adjacent levels. Matsunaga, et al (194) analyzed the change in strain distribution of intervertebral discs present after anterior cervical decompression and fusion. The authors noted that, in cases of double and triple-level fusions, the shear strain on adjacent segments increased approximately 20% one year post-surgery. During the follow-up period, 85% of discs that demonstrated abnormal increases in longitudinal strain after surgery herniated and caused spinal cord compression. They concluded that close

attention should be paid to the long-term biomechanical changes in the unfused segments of the cervical spine.

Dowd and Wirth (173) performed a prospective, randomized trial to compare the effects of anterior cervical discectomy with anterior cervical discectomy and fusion. They found that patients who underwent anterior cervical discectomy had shorter operative times, shorter hospital stays, and less need for analgesia. Patient satisfaction and return to preoperative activity level was similar for the non-fusion and fusion groups. The authors concluded that the addition of a fusion procedure to the anterior cervical discectomy may be unnecessary.

Posterior Decompression - Laminectomy

Indications for posterior decompression are compression at more than two levels, developmental narrowing of the spinal canal, posterior compression, the presence of a straight or lordotic spine, and ossification of the posterior longitudinal ligament (174,177). Cervical laminectomy has a number of disadvantages associated with the procedure (68,181):

- Manipulation of the spinal cord at surgery
- Vertebral dislocation or Subluxation
- Formation of scar tissue after surgery
- Risk of increased neurological damage by attempting to remove anteriorly situated osteophytes or disc material by way of a posterior approach (There is a 19% to 53% neurological loss rate associated with laminectomy)
- Production of kyphotic deformity
- Postoperative radiculopathy

With respect to the post-laminectomy kyphosis, at least one of the elements that make up the posterior column must be left intact in addition to the entire anterior column to maintain stability in the cervical spine. In laminectomy, the posterior column supporting structures are profoundly interrupted. From the posterior approach, facetectomy is done in addition to laminectomy to achieve nerve root decompression. Not more than twenty-five percent of the facet joint should be removed. When more than fifty percent of the facet joint is removed there is a statistically significant loss of stability in flexion and torsion. Some authors recommend posterior laminectomy only when the cervical lordosis is intact (174).

Kaptain et al (165) studied 46 patients who underwent laminectomy for cervical spondylotic myelopathy and found that kyphosis developed in 21% of patients who underwent the procedure. Progress of the kyphotic

deformity appeared twice as likely if preoperative radiographic studies demonstrated a straight spine. Sasai et al (170) performed a retrospective radiographic analysis of 31 patients undergoing laminoplasty for cervical spondylotic myelopathy to assess the consequences of the procedure on post-operative lordosis. They found that the lordosis demonstrated a significant decrease in 87% of patients over an average of 3.1 years. Loss of lordotic alignment was attributed to detachment of the semispinalis muscle on C2 and the nuchal ligament on C6-C7 because of the posterior approach and/or section of the ligamentum flavum at C2-C3.

Shinomiya et al (205) described the relationship between the loss of the posterior epidural ligaments and cervical spinal cord injury. The role of the posterior cervical epidural ligaments is to anchor the posterior dura mater to the ligamentum flavum and theoretically prevent injury to the spinal cord by resisting collapse of the dura during cervical flexion. Posterior laminectomy destroys the attachments for these ligaments. The authors found that reduced blood flow and changes in motor and sensory evoked potentials were noted in the spinal cord at 50 degrees, 60 degrees, and 70 degrees of flexion in test animals that underwent laminectomy. No changes in blood flow or evoked potentials were noted in the control group during flexion of the cervical spine. The study concluded that the loss of the posterior cervical epidural ligaments allows anterior displacement of the posterior dura mater in flexion, which may lead to flexion myelopathy.

The facet joints account for greater than 40% of the cervical spine's stability. Laminectomy and facetectomy result in 60% of the cervical spine's stability in flexion being lost (69,70,72). Risk factors for the development of post-laminectomy kyphosis are as follows (72):

- Children or adults less than 25 years of age
- Patients who have undergone laminectomy combined with foraminotomy or extensive dissection about the facets
- Patients undergoing laminectomy following trauma, in whom spinal stability is already compromised

Sinha et al (171) reported on 119 cases of patients treated via decompressive laminectomy for cervical spondylosis. His results indicated that 68% improved, 15% were unchanged, 15% worsened, and 1.6% died. Arnasson et al (172) reported that 69% of cervical spondylotic myelopathy patients treated with posterior surgery improved. Guigui et al (175) studied 60 patients over the age of 30 who underwent a

laminectomy of more than 3 levels without fusion for myelopathy secondary to cervical spondylosis. The patients in the study were followed for an average of 5 years through the use of lateral cervical radiographs that included neutral lateral, flexion, and extension films. They found that 31% of the patients developed post-operative changes in cervical spine curvature. Twenty-five percent of patients demonstrated one or more destabilized levels, and destabilization required repeat surgery in 3 patients. It was noted that all levels found destabilized on the post-operative radiographs were also hypermobile on pre-operative radiographs.

Combined Approach: Anterior Discectomy and Fusion with Posterior Laminectomy

A small subclass of patients treated operatively has undergone surgery according to a protocol involving both an anterior approach and a posterior laminectomy. The combined approach may be necessary in cases where the patient has kyphosis associated with a developmentally small spinal canal and multilevel pathology (174). Jeffreys (167) reported on 137 patients with severe disability that were treated with the anterior approach and posterior laminectomy. Fifty-two percent of patients returned to full employment following surgery, 39% returned to light employment, and 9% remained disabled though improved from the pre-operative status. Sinha et al (171) reported on 6 cases in which patients were treated via the combined approach. In the combined approach, 66% showed improvement post-operatively, 17% remained unchanged, and 17% died.

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