

Overview of Cervical Spondylosis Pathophysiology and Biomechanics

Jeffrey Mullin¹, Daniel Shedid², Edward Benzel¹

¹*Department of Neurosurgery, Center for Spine Health, Neurological Institute, Cleveland Clinic, Cleveland, Ohio*

²*University of Montreal, Centre Hospitalier de l'Université de Montréal Division of Neurosurgery, 1560 Sherbrooke Est, Montreal, Quebec H2L 4M1, Canada*

ABSTRACT

Cervical Spondylosis is one of the most common degenerative conditions of the spine; it is caused by the degeneration of cervical intervertebral discs and facet joints. These connecting structures are responsible for the transmission of forces and mobility between adjacent vertebrae. When osteophytic development compromise the canal diameter, myelopathic and/or radicular symptoms often develop. These symptoms are the result of increased pressure on the spinal cord producing neurological and vascular changes. Clinical management of cervical spondylosis requires a thorough knowledge of the biomechanics and pathophysiology of the condition.

KEY WORDS: Biomechanics, Cervical spondylosis, Pathophysiology, Spine

Cervical spondylosis is defined as “spinal canal and neural foraminal narrowing in cervical spine secondary to multifactorial degenerative changes”(40). This is one of the most common degenerative disorders of the spine, affecting 95% of patients by the age of 65 years (3,11,12,19,21,32,35). The degeneration of the intervertebral disc stems from osteophyte development of the amphiarthrodial joint, contrasting with arthritis, which is associated with diarthrodial joints and synovial space. The non-inflammatory disc degeneration is one of the defining characteristics of spondylosis (4). In this article, we will discuss the presentation, evaluation, pathophysiology, and biomechanics of cervical spondylosis.

Presentation

The majority of people with spondylosis are asymptomatic (35). Patients who are symptomatic tend to be older than 40 years old and present with 3 types of symptoms; neck pain, cervical radiculopathy, and/or cervical myelopathy (10,45). The neck pain is either acute

or chronic and often without an identifiable precipitating event (35). The pain often stems from abnormalities in structures innervated by the sinuvertebral nerve or branches of the posterior primary ramus. Among the structures that the sinuvertebral nerve innervates are the posterior longitudinal ligament, the epidural vasculature, and the spinal periosteum (10,35). Less commonly, the pain can be attributed to the facet joints, which are innervated by the primary posterior ramus (21).

Radiculopathy implies a problem with the spinal nerve root. Cervical radiculopathy symptoms can be caused by herniated nucleus pulposus or osteophytic stenosis of the spinal canal or neural foramina. Patients less than 55 years old will frequently present with cervical radiculopathy symptoms due to a herniated nucleus pulposus. Alternatively, older patients frequently have stenosis due to osteophytes (35). Symptoms can include weakness, atrophy, paresthesias, hyperesthesia, or hyperalgesia. Motor symptoms are more often associated with a soft herniated disc, whereas sensory symptoms are more often associated with a hard herniated disc (10).

Myelopathy implies a problem with the spinal cord. The clinical definition of myelopathy is “the presence of long-tract signs, which are the result of inhibition of the spinal afferent or efferent (pyramidal) nerve tracts” (24). Patient’s who are myelopathic will have symptoms including neck and shoulder pain, sensory changes (electrical shock sensation in all extremities, numbness in limbs, painful paresthesias), or motor changes (progressive spasticity, decrease dexterity, spastic gait) (3,11-13,22,23,26,39,46). Gait changes in the myelopathic patient have been shown to be decreased stride length and increased support time and these changes may be attributable to impaired proprioception and stability in the lower extremities (33). Some of the classic clinical findings in myelopathic patients are hyperreflexia, increased muscle tone, and presence of pathological reflexes, such as Babinski’s, clonus, and Hoffman’s signs. Other pathological reflexes include Oppenheim’s, Gordon’s, Schaefer’s, Bing’s, Chaddock’s, Gonda’s, and Allen’s signs (24). Late findings of atrophy, fasciculations, and sphincter dysfunction are associated with a worse prognosis for recovery (15,26,32). Atrophy will only occur distal to stenosis and if atrophy or fasciculations are present proximal to stenosis the physician must exclude other causes such as amyotrophic lateral sclerosis (24). Another clinical finding that may cloud the diagnosis is upper and lower motor neuron findings in the legs as this may indicate cervical and lumbar involvement which occurs in roughly 13% of patients (10,13). A recent study described the possible sympathetic symptoms of cervical spondylosis to include “vertigo, headache, tinnitus, nausea and vomiting, heart throb, hypomnesia, and gastroenterologic discomfort” as a result of “stimulation or compression of the sympathetic nervous system” due to degenerative changes in the cervical spine (28).

Imaging Evaluation

Various imaging studies are used in the workup of cervical spondylosis, including static or flexion-extension x-rays, CT scans, MRI and myelography. X-rays may show loss of disc space height, kyphotic changes, osteophytes, facet arthropathy, and autofusion of adjacent vertebrae (1,3,11,19,21). CT scans are useful for evaluating the spinal canal’s shape and size, transverse foramina and joint spaces (1,19). CT scans offer higher quality assessment of bony structures compared to MRI (21). MRI offers the ability to evaluate the spinal canal

dimensions, the spinal cord, intervertebral discs, and cervical soft tissues (1,3,11,17,19,21,34,39). Additionally, T2-weighted MRI often demonstrates cord signal changes at the level of compression, which represents “edema, inflammation, ischemia, myelomalacia, or gliosis” (34,35). A recent publication documented the correlation between clinically diagnosed cervical myelopathy and radiographic evaluation, specifically T2 signal changes on the spinal cord (25). Recent studies focus on using MRI to study the kinematics of the cervical spine in spondylosis patients (35). With the increase in MRI, CT myelography has largely been replaced. However some physicians contend that CT myelography is superior to MRI in distinguishing osteophytic spurs from soft tissue, disc protrusion or cartilage (3,20).

Clinicopathological distinctions

Cervical spondylosis can lead to other types of spinal cord injury, such as central cord or Brown-Sequard syndromes. (35) Spinal cord injury can result from superimposed acute injury on chronic trauma (3,11,17,26,32). Medial spinal cord lesions are often thought to be ischemic in nature due to vascular compression versus lateral spinal cord lesions that are more radicular (1,5,17,48). Medial spinal cord lesions more often present acutely with proximal lower extremity weakness as an early sign, progressing to “dorsal column sensory loss and spastic gait” (19,35,46) Due to the inherent nature of spondylosis, patients are at significantly higher risk of spinal cord injury following a relatively minor trauma (2,19,46).

In patients with cervical radiculopathy, the differential diagnosis should initially include spinal tumor, angina, RSD, infection/abscess, peripheral nerve entrapment, thoracic outlet syndrome, and brachial neuritis (35). Other disease processes that may present similarly to spondylosis are ALS, transverse myelitis, multiple sclerosis, intracranial pathology, syringomyelia, Chiari malformation, ligamentous calcification or ossification, and ankylosing spondylitis (3,5,6,16,38,45).

Pathophysiology

Primary Degenerative Phenomena

Etiology of Cervical Spondylosis

The degenerative changes of the cervical spine are responsible for the primary lesions in cervical spondylosis, while both spinal cord (neural) and

vascular compression are responsible for the myelopathic symptoms (2,12,30,32,42,47). Mechanical factors involved in the pathogenesis of cervical spondylosis have been categorized into two groups as static and dynamic factors (47).

Static factors include congenital canal stenosis, disc herniation, degenerative osteophytic growth, hypertrophy of the ligamentum flavum, and calcification of PLL. The dynamic factors are “abnormal forces on the spinal column and spinal cord during normal and abnormal movements and loads (Table 1) (35).

Pathogenesis of Cervical Spondylosis

Degenerative changes begin with intervertebral disc desiccation, which is associated with increase in the ratio of keratin sulfate to chondroitin sulfate (4,14,19). Along with desiccation, the nucleus pulposus shrinks, loses elasticity, and becomes more fibrous due the loss of water, protein and mucopolysaccharides during the aging process (3,4,14,32,42,46). Disc height is initially lost in the ventral portion of the disc, which results in a decrease in cervical lordosis. Unfortunately, this process results in positive feedback cycle due to the increase in forces applied ventrally and eventually may lead to kyphotic deformity (Figures 1, 2) (4). These early changes ultimately lead to the main pathophysiological process of cervical spondylosis, a reduction in sagittal spinal canal diameter (35). Additionally, these changes cause a transfer of axial load onto the facet joints, resulting in hypertrophy of those joints that further decreases the spinal canal's diameter (32).

The annulus fibrosis is thinner dorsally, thus making it easier for the nucleus pulposus to dissect through and cause disc herniations into the spinal canal (19,32,46). Preceding disc herniation, the peripheral fibers of the annulus fibrosis and Sharpey's fibers dissect from the vertebral body edges (35). Another early degenerative change is the posterior longitudinal ligament beginning to pull away from the vertebral bodies near the end plates (42,46). Eventually, abnormal cervical movement

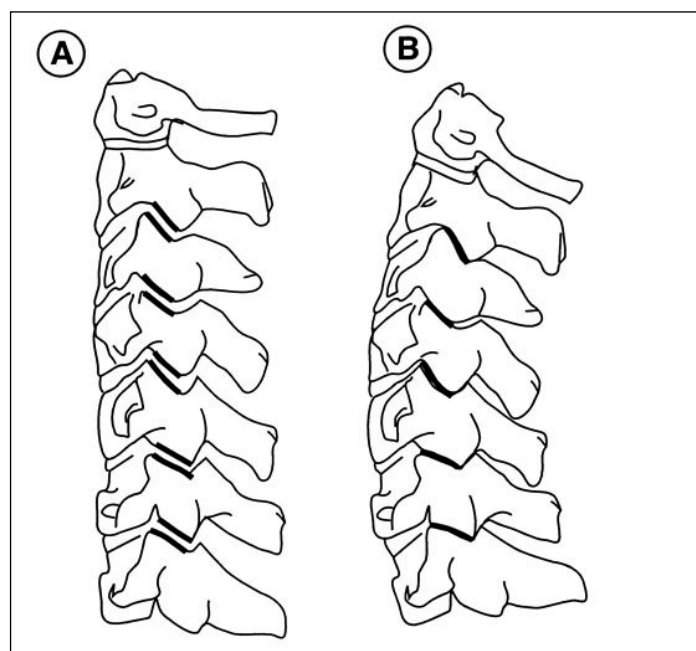


Figure 1: A) In a neutral spinal orientation, the facet joints of the cervical spine are unloaded during moderate axial loading. B) In a lordotic orientation (relative extension), however, they are loaded and thus subjected to injury during axial loading.

result either from the pain of disc herniation or annular protrusion, worsening degenerative changes, or increased ligamentous laxity (46). Where the PLL peels off the dorsal vertebral body, reactive bone formation begins forming spondylotic bone spurs, which may be as large as the width of the vertebral body (31,42). These osteophytic growths may project into the intervertebral foramina (35). Some have referred to these degenerative changes as cervical hyperostotic myelopathy (29). Of note, the increase in joint motion causes an acceleration of osteophyte growth, and this is most pronounced at C5-C6 and C6-C7(2,32). As expected, these two levels are also the most often affected by spondylosis, with C6-C7 affected more commonly than C5-C6 (38). The growth of osteophytes, along with degenerative changes, leads to decrease in sagittal spinal canal diameter, the main pathophysiological process in cervical spinal spondylosis.

In addition to the above-described osteophytic and degenerative changes, a third modality that causes spinal

Table 1: Mechanical factors in pathogenesis of spondylosis (35)

Static	Congenital canal stenosis, disc herniation, degenerative osteophytic growth, hypertrophy of the ligamentum flavum, calcification of PLL.
Dynamic	Abnormal forces on the spinal column and cord during movement.

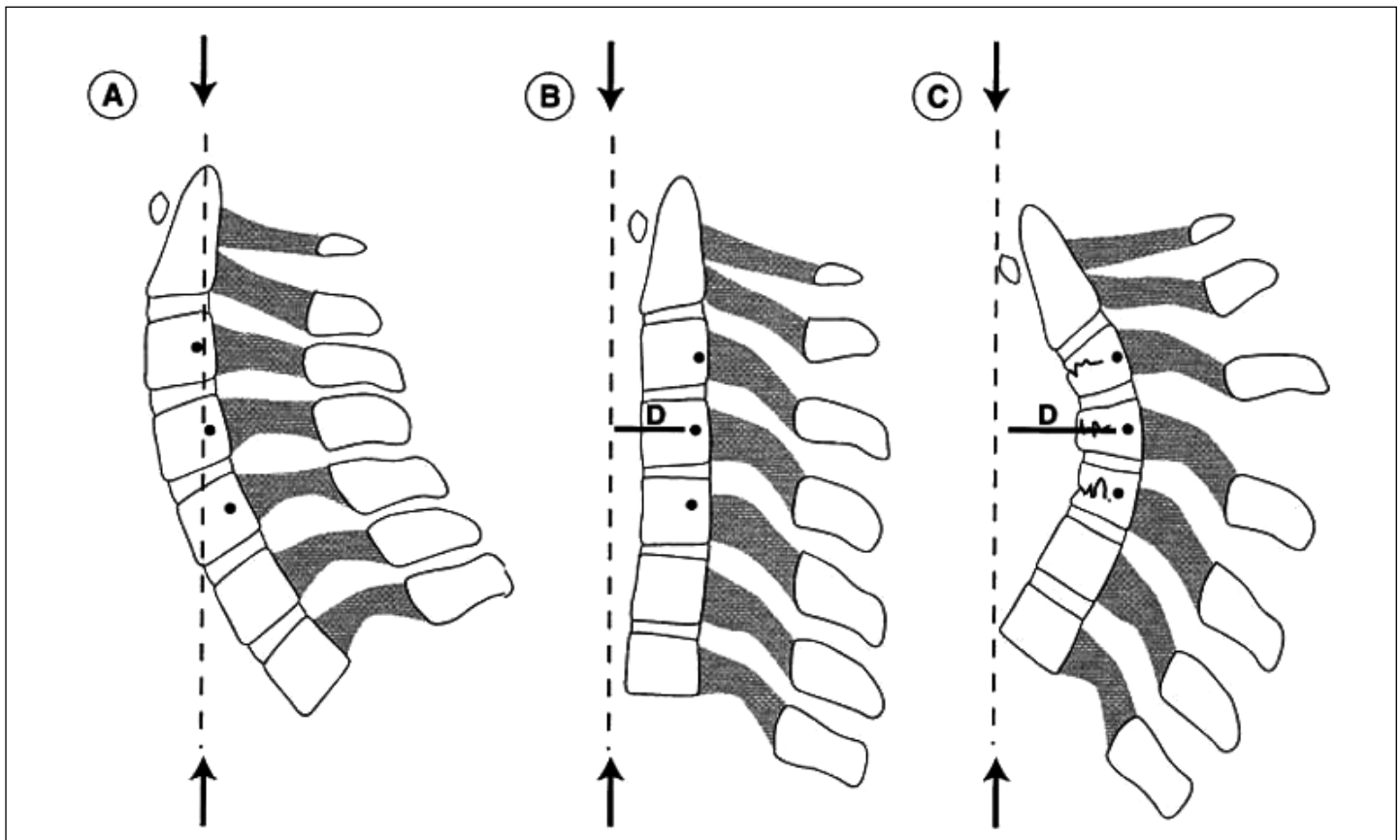


Figure 2: Sagittal magnetic resonance imaging (MRI) of the cervical spine showing ventral spinal cord compression from disc herniation at C3-4 and vertebral body osteophytes. Note the compression of the spinal cord in extension. Reprinted with permission of AANS publications.

canal narrowing is congenital cervical stenosis (3,6,22). The average cervical spondylolytic patient's spinal canal is 3 mm smaller than the average population. More pronounced narrowing is observed in congenital stenosis patients (3,38). The average adult spinal canal diameter has been reported to be 17 to 18 mm between C3 and C7 (3,18). Within the literature, there exists some minor disagreements over at what diameter patients will suffer from spondylosis and the range reported is 13-14.8 mm (3,18,19,39,47). In patients with congenital stenosis, these degenerative changes are magnified, leading to earlier onset of myelopathy (6,11,17,19,46-48).

Cervical Spine Posture

The upper cervical spine is anatomically complex, allowing for varying degrees of motion in multiple directions as summarized in Table 2 (35). In addition to providing flexibility, the cervical spine also functions to support the cranium and protect the spinal cord (4). The lower parts of the cervical spine do not provide the same degree of motion. A significant feature of this part of the

Table 2: Movements allowed in the craniocervical region

Joint	Motion	Range of Motion (degrees)
Occiput-C1	Combined flexion/extension	25
	Lateral bending (unilateral)	5
	Axial rotation (unilateral)	5
C1-C2	Combined flexion/extension	20
	Lateral bending (unilateral)	5
	Axial rotation (unilateral)	40

spine is the lordotic posture, and it is believed that this may play a role in preventing spinal cord injury. By being in lordosis, the cervical axial loads are symmetrically displaced. Also providing protection is the coronal orientation of the facet joints, which limits movement only in the direction of extension, where the spine is most able to resist axial loading in this direction. The facet joints are most effective in participating in axial load support when in extension (35).

Late Pathological Features

As discussed earlier, osteophytes originate from the vertebral bodies and extend into the spinal canal while some eventually progress to cross the intervertebral disc space. When this occurs, the vertebral bodies may combine and fuse (35). If this autofusion occurs, patients may experience a paradoxical increase in stability of their cervical spine (5,19). This autofusion also results in decreased cervical motion and may be responsible for the improvement of symptoms in patients with mild or moderate myelopathy (2). However, this process occurs in the setting of relative kyphosis and may place additional stress on adjacent cervical levels and lead to progression of spondylotic abnormalities at the rostral and caudal levels (35).

Secondary Compressive Processes

Spinal Cord Compression

Cervical compression in the sagittal plane is the most frequently cited cause of spondylotic myelopathy (5,11,18,42,47,48). This compression may be ventral via osteophytes or dorsally via bulging ligamentum flavum (42,43,47). With ventral compression, symptoms can worsen with flexion and especially with kyphosis (9,27,46). In flexion, the spinal cord's lateral and ventral columns are deformed and the sagittal dimensions are reduced, and this is attributed to axial tension (19). On the other hand, extension may result in dorsal compression of the spinal cord due to impingement from the ligamentum flavum, often with resultant myelopathy or Lhermitte's sign (19,22,27,38,39,42,46). Several studies have demonstrated that spondylotic processes cause significant indentation of the cervical spinal cord during hyperextension, and to a lesser degree during lateral bending and axial rotation (9,38,44). Flexion and extension MRI images have shown that increased stenosis is two times more likely during extension than during flexion (Figure 3) (37). Normal spinal cord movement is altered by ventral osteophytes, which causes ventral deformities to occur (15,39). A study by Brieg et al has shown that the dorsal half of the spinal cord shortens more than the ventral half and osteophytes cause deep furrows in the ventral surface during extension (9). It has been shown that the spinal cord regions containing nerve tracts for the upper extremities undergo five times more compressive stress than the tracts for the lower extremities (42).



Figure 3: The nonpathological state where the dorsal vertebral body height is less than the ventral vertebral body height results in normal cervical lordosis (A). Loss of the ventral disc interspace height which occurs with the natural degenerative process results in loss of lordosis (B). This causes elongation of the moment arm applied to the spine (D), leading to ventral vertebral body compression. A further exaggeration of pathological kyphotic posture may then ensue (C). Reprinted with permission of AANS publications

Another change in normal biomechanics that has been described is a pincer phenomenon, the result of unstable cervical segments undergoing subluxation during flexion, extension or both. This subluxation results in pinching of the spinal cord, while further exacerbation is caused by large osteophytes and ligamentous laxity with instability (22,23,35,46,47).

The sagittal bowstring effect is when an “effective kyphosis” results in spinal cord tethering over the kyphosis in the sagittal plane during flexion (Figure 4) (4-6,18,19,35). An “effective kyphosis” is when no part of the dorsal aspects of C3 through C7 vertebral bodies crosses an imaginary line; this line is “associated with a zone of uncertainty... , within which the surgeon’s bias” determines the principal configuration in the midsagittal section, lordosis or kyphosis (Figure 5) (4,35). A coronal bowstring effect is also possible, often caused by nerve roots or dentate ligaments tethering the cord. In coronal bowstring, laminectomy is not effective in releasing the tethered spinal cord, and a ventral decompression is therefore required (Figure 6) (4,35).

Pathological and Histological Changes

Histopathological changes are most frequently demyelination of the lateral columns at the level of the osteophytic growth and the dorsal columns are also frequently affected. When the corticospinal tract is compressed, the changes occur caudal to the compressed level (35). With severe and chronic compression, necrosis and cavitation of the gray matter ensues (7,8,19,27,32). The term “butterfly” partial necrosis has been used to describe the areas affected by necrosis; the lateral columns, the lateral portions of the gray matter, and the ventral parts of the dorsal columns (Figure 7). This pattern of necrosis is thought to be due to occlusion of the arterial or venous systems or both (4,23,32).

Effect of Trauma

The central cord syndrome is often associated with acute spinal cord injury but can also occur in chronically injured spinal cords. A collection of chronic minor injuries may be minor but the collection of repetitive, concussive injuries may be significant and result in the central cord syndrome (19,35,47). Additionally, spondylosis patients may suffer from spinal concussions, contusions and the Brown-Sequard syndrome (15,16).

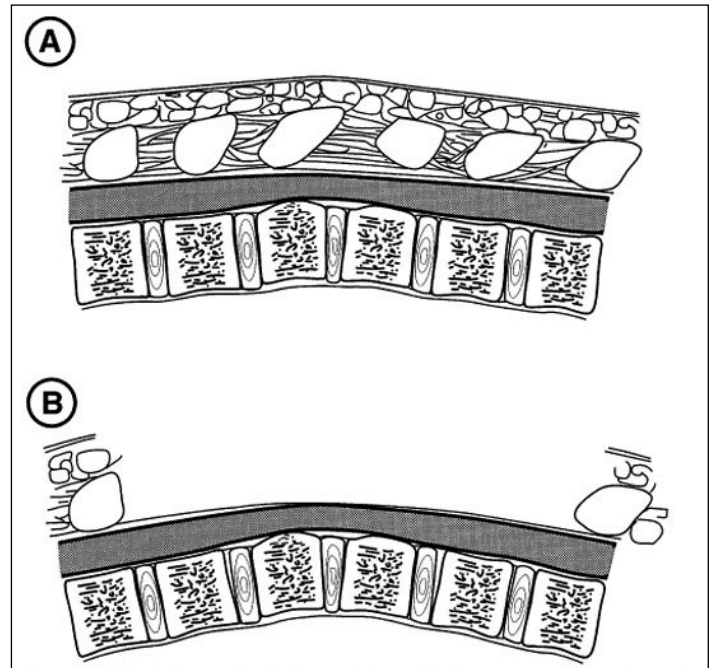


Figure 4: Kyphosis associated with cervical spondylosis causes neural injury, in part, by tethering the spinal cord over a ventral mass via the “sagittal bowstring” effect (A). Dorsal decompression (i.e. via laminectomy) may worsen deformation (B). Reprinted with permission of AANS publications.

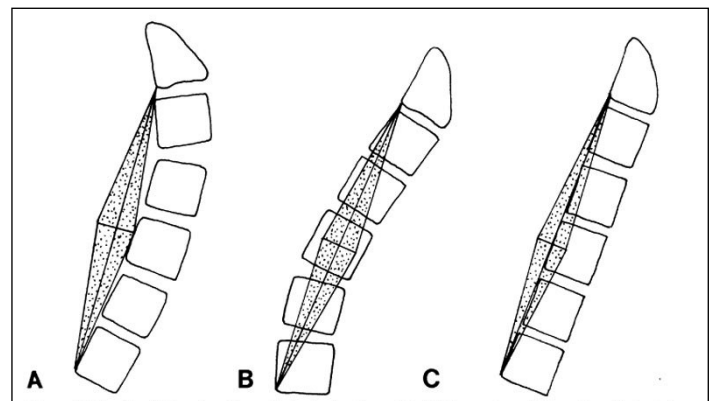


Figure 5: A midsagittal section of the cervical spine configured in lordotic posture (“effective” cervical lordosis). A line has been drawn from the dorsocaudal aspect of the vertebral body of C2 to the dorsocaudal aspect of the vertebral body of C7 (solid line). The “gray zone” is outlined by the other lines (A). A midsagittal section of a cervical spine in kyphosis (“effective” cervical kyphosis). Note that portions of the vertebral bodies are located dorsally to the gray zone (B). A midsagittal section of a “straightened” cervical spine (C). Note that the most dorsal aspect of vertebral bodies are located within, but not dorsally to, the gray zone. Reprinted with permission of AANS publications.

Vascular Compression

Evidence suggestive of vascular mechanisms in cervical spondylosis is necrosis is found in the spinal gray matter that is more sensitive to ischemia. Spinal

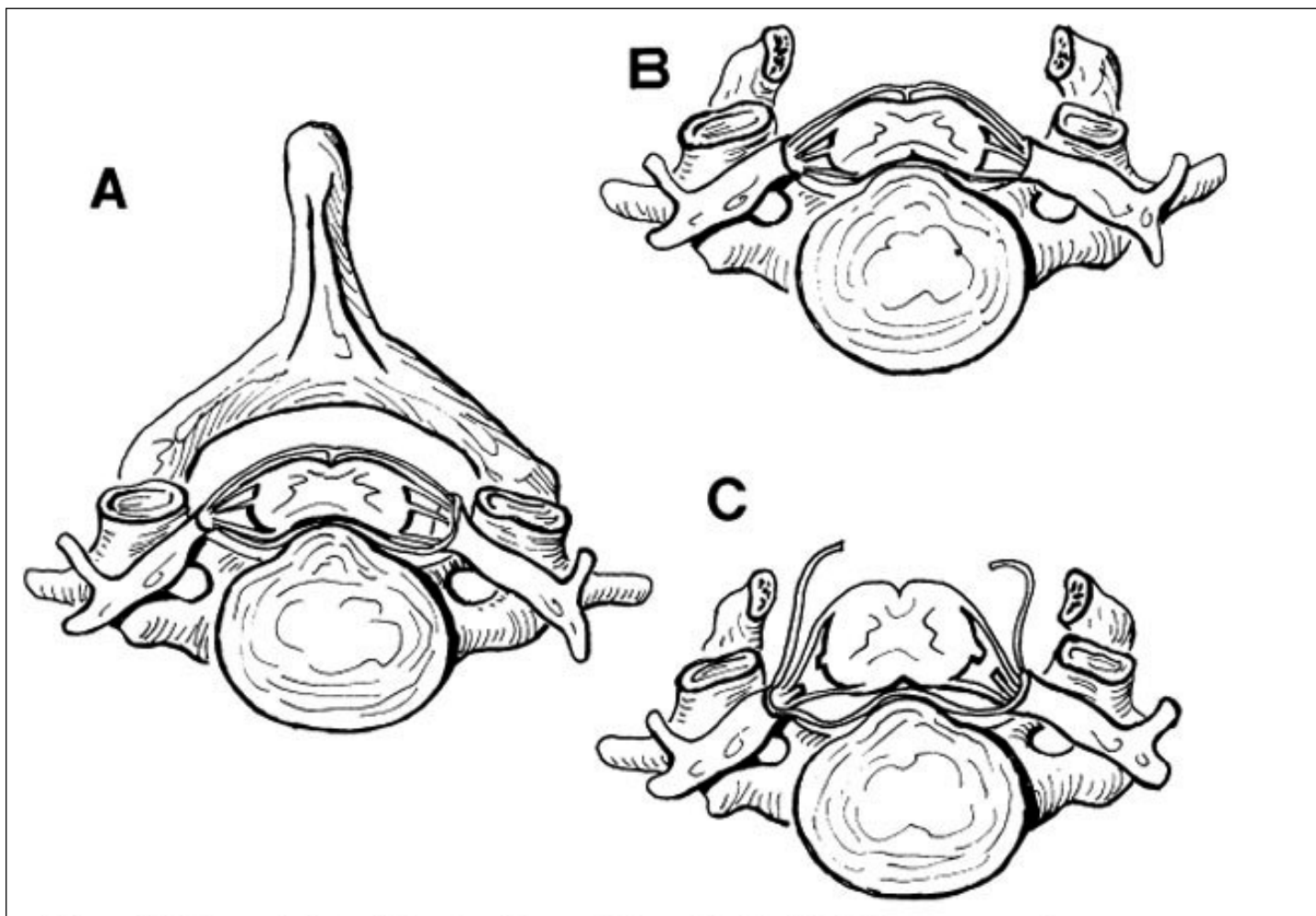


Figure 6: Coronal plane tethering (“coronal bowstring” effect). The nerve roots, or more commonly, the dentate ligaments may tether the spinal cord in the coronal plane (A). Laminectomy may not relieve the distortion (B). Ventral decompression is a more commonly considered approach (C). Reprinted with permission of AANS publications.

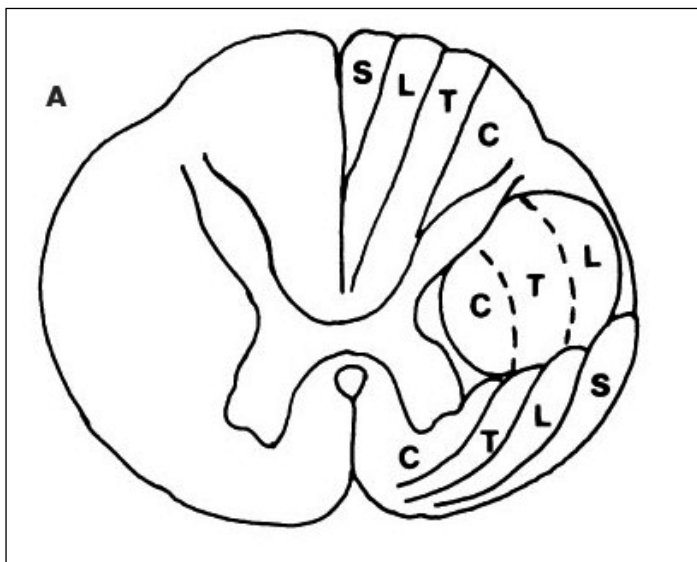


Figure 7: Diagrammatic axial sections of the spinal cord demonstrating the somatotropic orientation of the spinal tracts. The zone of “butterfly” partial necrosis is observed in the cervical spinal cord of patients with cervical spondylotic myelopathy (grayed region). The necrosis affects the lateral columns, lateral portion of the central gray matter and ventral portions of the dorsal column. Reprinted with permission of AANS publications.

cord vascularization is more tenuous between C5 and C7, and occasionally acute presentations of cervical spondylotic myelopathy are attributed to spontaneous thrombosis of spinal arteries leading to ischemia (7,10,34,46). Among the local arterial supply vessels that are compressed in spondylosis are the anterior spinal artery, the posterior spinal artery, radicular arteries, pial plexuses, and penetrating spinal cord vessels (3,4,5,17-19,22,31,32,42,43,46,47).

In addition to arterial blood supply being compromised, venous blood flow may also be affected. The anterior median spinal vein, which receives flow from central sulcal tributaries, may be compressed by ventral osteophytes. Delayed syringe cavitation development may be caused by venous infarction (32).

CONCLUSION

Cervical spondylosis is a complex pathology. The management requires an extensive knowledge of the anatomy, biomechanics and surgical options.

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Address correspondence to: Edward C. Benzel, Chairman, Department of Neurosurgery, Cleveland Clinic, 9500 Euclid Avenue S-80 Cleveland OH 44195

GSM: 216 444 7381

Fax : 216 445 4527

email: benzele@ccf.org