0008-3194/91/75–81/$2.00/©JCCA 1991

Cervical spondylotic myelopathy: Part I: anatomical and pathomechanical considerations*

SH Burns, DC, FCCS(C)
SA Mior, DC, FCCS(C)
SM O'Connor, DC

This two part series reviews the recent literature concerning the etiology and clinical presentation of cervical spondylotic myelopathy (CSM). CSM is the most common neurological spinal cord disorder after middle age. It is caused by the compromise of the cervical spinal cord from narrowing of the spinal canal.

In Part I, a review of the anatomy and the pathomechanics of the cervical spine pertinent to CSM is discussed. Emphasis is placed upon the intricate relationship between the osseous, neurological and vascular structures. The consequences of degenerative changes upon this relationship is evidenced by the resulting neurovascular compression. In turn, compression may lead to spinal cord ischemia with characteristic clinical results.
(JCCA 1991; 35(2):75–81)

KEY WORDS: cervical spondylotic myelopathy, cervical spine, spondylosis, myelopathy, chiropractic, manipulation.

Cet article en deux parties passe en revue la littérature récente concernant l'étiologie et la présentation de la myélopathie vertébrale cervicale (MRC). Chez l'adulte de plus de 40 ans, la MRC représente le désordre de la moelle épinière à manifestations neurologiques le plus commun. Ce désordre résulte d'un rétrécissement du canal vertébral cervical, compromettant ainsi la moelle épinière.

Dans la première partie, on discute d'anatomie et de pathomécanique de la colonne cervicale en relation avec la MRC. L'emphase portera sur la complexité des relations entre les structures osseuses, neurologiques et vasculaires. Les conséquences de changements dégénératifs sur cette relation inter-structurale se traduiront par une compression neurovasculaire. De son côté, cette compression se manifestera par des signes cliniques caractéristiques démontrant une ischémie de la moelle épinière.
(JCCA 1991; 35(2) : 75–81)

MOTS-CLÉS : myélopathie vertébrale cervicale, colonne cervicale, vertébral, myélopathie, chiropratique, manipulation.

Introduction

Cervical spondylotic myelopathy (CSM) is a neurological disease caused by the stenotic encroachment of the cervical spinal cord. The stenosis is usually secondary to degenerative changes superimposed on a congenitally narrowed spinal canal. As a result, the cord and/or its blood supply are compressed, resulting in direct mechanical damage and/or neuroischemia. Compression of the cervical nerve roots usually co-exists with cord compression, but is not an intrinsic part of CSM.

Considered to be the most common neurological spinal cord disorder after middle age,[1,2] CSM was described as a clinical entity by Brain in 1954.[1] Two years later, Clark and Robinson[3] described the degenerative process involved and its relationship to neurological symptoms. Since then, many authors have investigated CSM from various perspectives, including the neurological, pathological, biomechanical and radiological elements.

This paper reviews the recent literature underlying the pathology of CSM. A discussion of the underlying spondylotic changes and their effect upon the mechanics and vascularity of the cervical cord and vertebrae is presented.

Relevant anatomy

Certain anatomical features of the normal cervical spine warrant review, since degenerative changes ultimately affect both the static and dynamic dimensions of the spinal canal and the regional biomechanics. These are important factors in the development and progression of degenerative myelopathy.

The upper cervical spine, comprising the occiput, atlas and axis, has anatomical and biomechanical features distinct from the remainder of the cervical spine. As a consequence, degeneration in this region rarely contributes to the development of CSM. Rather CSM is encountered in the lower cervical spine, namely vertebrae C4 through C7. These are often referred to as the 'typical' vertebrae because of their similar anatomical features.

* Division of Postgraduate and Continuing Education, Canadian Memorial Chiropractic College, 1900 Bayview Avenue, Toronto, Ontario M4G 3E6.

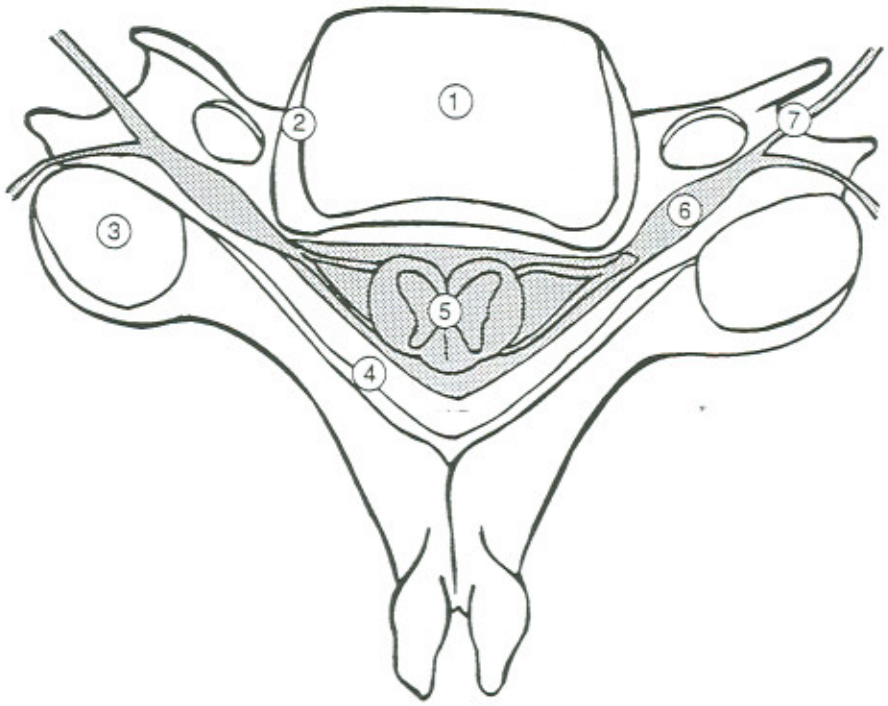

Figure 1 Schematic cross-sectional representation of the normal anatomy of a typical cervical vertebra and its surrounding soft tissue. These include: **1** vertebral body and disc; **2** uncovertebral joint; **3** zygapophyseal joint; **4** ligamentum flavum; **5** spinal cord; and **6** nerve root (after Panjabi and White[29])

The normal cervical spine has a smooth lordotic curve throughout its length. Since vertebral bodies are generally higher in their posterior aspects, the lordosis is primarily due to the intervertebral discs (IVD) being thicker anteriorly than posteriorly.[4] The IVD make up about 20% of the length of the cervical spine. Intrinsically, the centrally located nucleus pulposus constitutes about 40% of the cross-sectional area of the disc.[5] The water imbibing properties of the disc decrease after the age of 30 years, from about 80% in early life to about 60% in the geriatric spine.[6] This change in the degree of hydration is thought to play a significant role in the initial stages of spinal degeneration.

The bony elements of the cervical spine include the vertebral body anteriorly, the neural arch posteriorly, the zygapophyseal joints posterolaterally and the spinous process directly posterior. Just anterior and medial to the zygapophyseal joints lies the lateral canal, or intervertebral foramen, through which passes the emerging nerve root. On the superior lateral aspect of the body itself is a bony lip, variously known as the uncovertebral joint, the uncinate process, the neurocentral lip or the joint of Luschka. The neural arch is made up of the pedicles arising from the vertebral body and the lamina. Directly adherent to the neural arch and lying in close approximation to the spinal cord is the ligamentum flavum, or yellow ligament. Unlike the lumbar spine, where the ligamentum flavum is narrow and inconsistent, in the cervical spine it is wide and relatively thick.[7] These features are displayed in Figure 1.

The spinal cord is protected circumferentially by the bony spinal canal. Anteriorly, the cord is bounded by the vertebral bodies, their intervening discs and the overlying posterior longitudinal ligament. The cord is protected by the neural arch laterally and posterolaterally and the ligamentum flavum posteriorly. Posterolaterally the body segments, the uncovertebral joints and the zygapophyseal joints not only protect the cord, but are in close approximation to the nerve roots. (see Figure 1)

When viewed in a sagittal section, the canal has a funnel shaped configuration, being widest at the atlas and tapering in a caudal direction to about C4. Using cross sections at various cervical levels, Parke[8] has demonstrated this funnel shape on cadavers and has shown that the spinal cord occupies only about 50 percent of the canal at C1, but 75 percent of the canal at C6. It is primarily due to this dimensional feature that degenerative myelopathy is rare in the cervical spine above the C4 level.

This decreasing diameter of the spinal canal also has direct implication upon the cord's vascularity. The spinal cord receives most of its blood supply from the single anterior spinal artery (ASA) and the paired posterior spinal arteries (PSA). The ASA lies in the anterior sulcus of the cord; while the posterior arteries follow a torturous pathway along the posterior surface of the cord. Both the ASA and the PSA descend from the vertebral arteries above. The ASA also receives several radicular tributaries which accompany the nerve roots through the transverse foramen. The ASA supplies the majority of the cord, including the central grey matter and the anterolateral white matter. The posterior spinal arteries supply the more posterior areas of the cord including the posterior columns. (See Figure 2)

The ASA and PSA are in close approximation to the vertebral bodies, discs and to the ligamentum flavum. The neural ischemia seen in the CSM results from the susceptibility of the ASA to compression from these elements. The significance of these

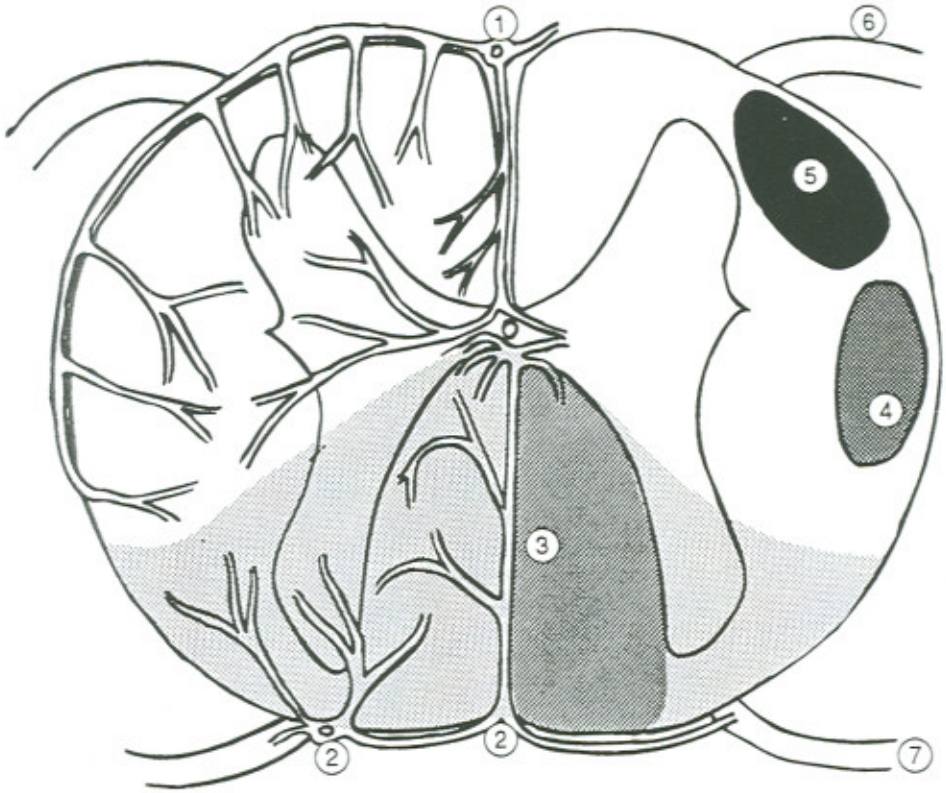

Figure 2 Schematic cross-sectional representation of the cervical spinal cord: **1** anterior spinal artery; **2** posterior spinal artery; **3** posterior column; **4** lateral corticospinal tract; **5** spinothalamic tract; **6** ventral root; and **7** dorsal root. Notice that most of the cord receives its vascular supply from the anterior spinal artery (unshaded area). The right side of the diagram illustrates the common tracts that are found in the region supplied by the anterior and posterior arteries. (after Panjabi and White[29])

bony, articular, discal, neural and vascular anatomical structures becomes apparent when the pathological features and clinical findings of CSM are discussed.

Spondylosis reviewed

Cervical myelopathy is a progressive disorder and a discussion of its pathogenesis requires an understanding of the correlation between the anatomical degenerative changes and the subsequent neurological insult. The degenerative changes associated with cervical spondylosis involve the intervertebral disc, the vertebral body, both the zygapophyseal and uncinate joints, as well as the surrounding ligaments, most significantly the ligamentum flavum. As mentioned, these changes can affect the dimensions of the canal. Although degenerative changes proceed in a somewhat sequential fashion, different vertebral motion segments will be at various stages of degeneration at any given time.

Spinal degeneration is thought to initiate with normal aging changes in the intervertebral discs, commonly beginning in the third decade of life. These start with alterations in the IVD's biochemical properties, eventually leading to internal derangement and loss of anatomical integrity.[33] In reviewing the cervical spine, Lestinini and Wiesel[4] have discussed in detail, the biochemical changes within the cervical IVD that may be responsible for the cascade of events, that ultimately lead to the gross pathological changes seen in severe spondylosis.

The first changes in the IVD are in the nucleus pulposus. As it ages, the nucleus loses water, protein and mucopolysaccharides. Consequently, the IVD begins to lose its elasticity and becomes more fibrous. In time, the water imbibing properties decrease to the point where the IVD loses the positive pressure typically seen in youth.[9,10] Furthermore, as the annular fibers slacken, the nuclear material may migrate through the layers of the annulus, increasing the extent of internal derangement.

With continued internal derangement the IVD collapses, reducing the vertical height of the vertebral column, as well as the vertical dimension of the intervertebral foramina.[11] The decrease in column height results in slackening and inward buckling of the ligamentum flavum, especially when the head and neck are put into extension. The ligament itself goes through degenerative and contractual thickening.[12,13]

As the spine loses its functional stability, secondary changes in the vertebral bodies and the surrounding joints take place. As the IVD space thins, the uncinate joints bear more weight and react with osteophyte formation.[10] Osseous lipping forms around the vertebral end-plates, which on their posterior aspects, may project into the spinal canal space. The zygapophyseal joints hypertrophy and may subsequently encroach upon both the spinal cord and roots. The disrupted intervertebral disc may harden and ossify, forming what has been termed a 'spondylotic bar'.[8]

Collectively, these changes describe circumferential encroachment around the contents of the spinal canal. The hypertrophic reactions may be thought of as an attempt to contain spondylotically-induced instability.[9,14] However, the stiffening effects of osteophytic formation at a given level may result in compensatory hypermobility at subsequent levels.[15] This sets the stage for further, progressive spondylosis in a step wise fashion throughout the remainder of the cervical spine.[8] This hypothetical process is summarized in Figure 4.

Figure 3 Photograph of a cervical vertebra showing gross degeneration around the zygapophyseal joints (arrow). These hypertrophic changes can compress the nerve roots and spinal cord of a congenitally shallow canal.

Congenital predisposition to CSM

Despite the universality of degenerative changes in the aging cervical spine, only a small percentage of the population suffers from clinically evident cervical myelopathy. It is apparent that even those with extensive, long-standing spondylotic changes do not usually suffer neurological deficit. The architecture of the normal cervical spine is such that it can accommodate quite extensive degenerative changes without cord compression, even in the lower cervical spine where the spinal cord occupies the majority of the canal.

What then is the consistent feature of patients who suffer transient or permanent neurological damage from spinal cord compression? Various investigators agree that developmental shallowness, primarily in the anterior-posterior (AP) diameter of the lower cervical canal, is the underlying criterion for susceptibility. Ehni[16,17] has extensively described the developmental variations predisposing those individuals who are at risk when degenerative changes are superimposed. He demonstrated that variations in the length and angulation of any or all of the components of the neural arch may contribute to the wide range of depths of canals and foramina. Long posteriorly directed pedicles, facets with long AP dimensions, and hemi-lamina that meet at an acute angle, all contribute to a deep canal; while short laterally-oriented pedicles, short, wide facets and lamina that meet at an obtuse angle result in a developmentally shallow AP canal. (Figure 5) Most authors concur that a sagittal diameter of 12 mm or less in the lower cervical spine, as measured on standard lateral radiographs, renders the subject vulnerable to stenotic complications. This is discussed in greater detail in Part II.

Role of vascular ischemia

The signs and symptoms in CSM are not only due to mechanical compression of the spinal cord, but also to compression of the cords' vasculature. This can involve compromise of the arterial supply, the venous drainage, or both. When venous flow is obstructed, the cord becomes edematous and neurological symptoms are widespread and diffuse. Compression of the arterial supply leads to transient neural deficits which may become permanent if the ischemia leads to regional cord necrosis.

The role of vascular compression in CSM has been recognized since the 1950's, when Allen[18] observed blanching of the cord as a spondylotic neck was flexed during laminectomy. Mair and Druckman[19] demonstrated pathological changes in the

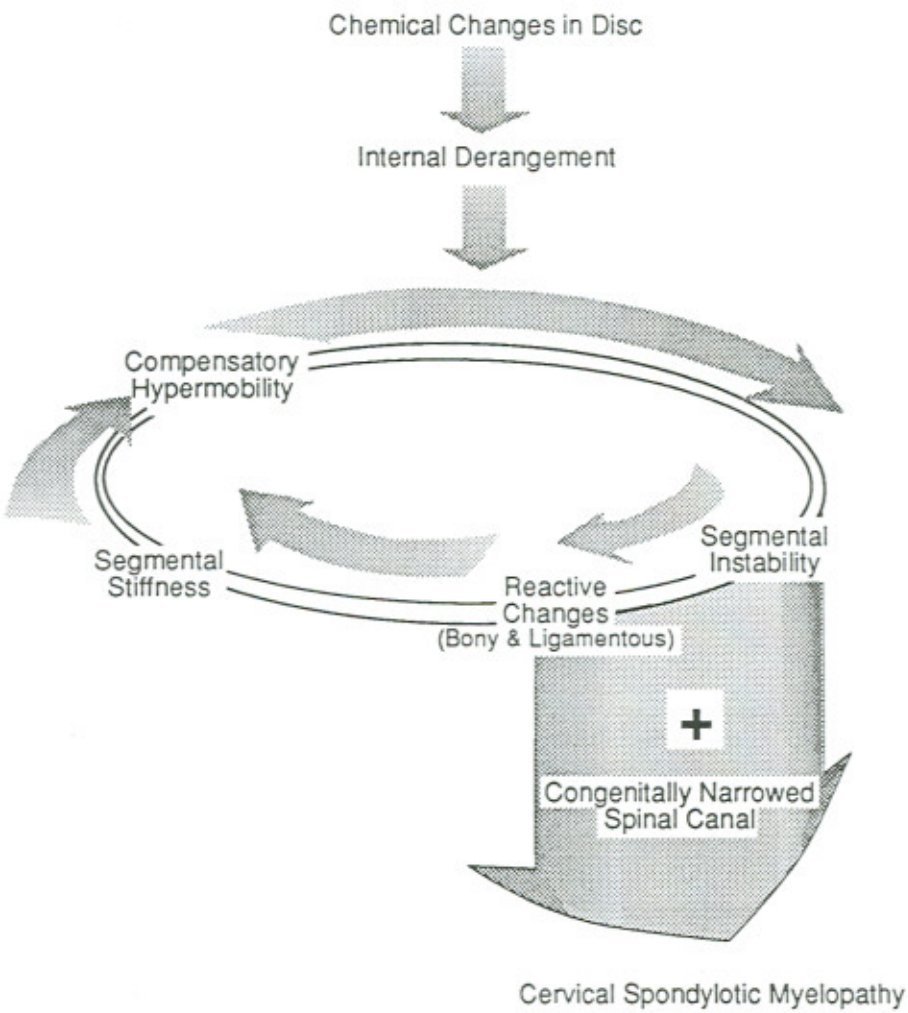

Figure 4 Graphic summary of a pathological model illustrating the changes hypothesized to lead to cervical spondylosis.

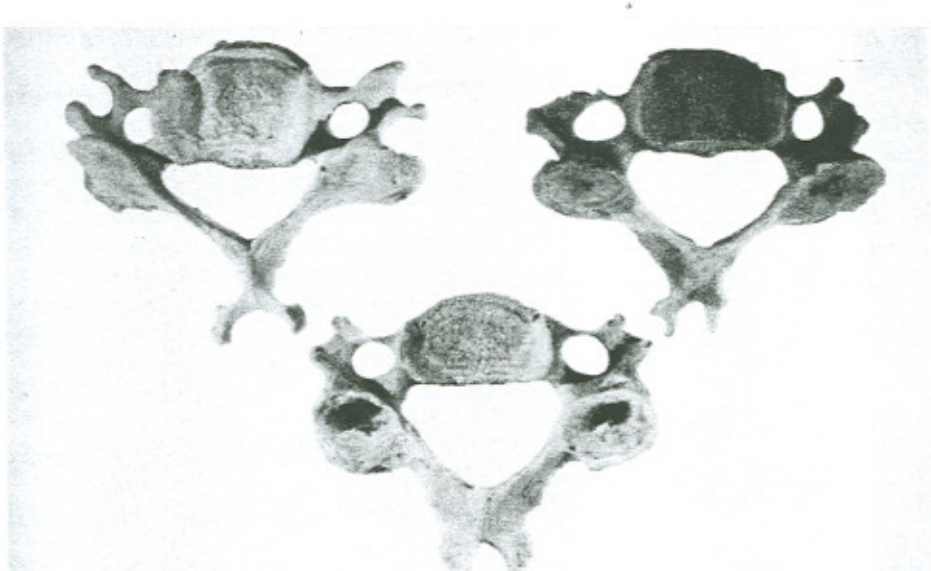

Figure 5 Photograph of a C5 vertebra obtained from three different cadaver specimens. Note the inherent difference in the shape of the spinal canal.

cord corresponding to the watershed area of the ASA in cadavers. These findings have been reproduced experimentally on canine models.[20,21]

The neurological deficits resulting from vascular compromise usually correspond to the area of the cord supplied by the ASA.[7,11,22] Parke[8] emphasizes the vulnerability of the ASA in its mid-sagittal position on the cord. The significance of compression of this artery is evident as it supplies approximately 75% of the cord, including all the grey matter and the anterolateral white matter.[23] Furthermore, as Parke[8] points out, the major lateral feeder arteries to the ASA are most often found at the C4–5–6 levels. It is at these levels that the greatest degree of lateral canal stenosis, which usually accompanies central stenosis, occurs. The degenerative changes that lead to nerve root radicular signs can, therefore, also affect the 'cord proper' by compressing the tributaries to the ASA.

By contrast, the posterior spinal arteries do not usually become compromised. This is likely due to their zig-zag configuration on the posterior cord surface, which allows them to elongate during neck movements without undergoing tension, even in a spondylotically-tethered cord.[7,24]

Pathomechanics of CSM

Until now, we have been discussing CSM in static terms. However, the signs and symptoms of CSM are most often dependent upon the position and movement of the patients' neck. The most important movements to consider are flexion and extension, since these have the greatest influence on the AP diameter of the canal and on tensile stresses within the cord. This is true for both mechanical cord compression and arterial compression. The dynamics of cervical spine motion, combined with co-existing anterior and posterior canal encroachment, are such that symptoms may arise from flexion, extension, or both. The movements of lateral flexion and extension play a greater role in nerve root compression that often co-exists with myelopathy.[8]

Pre-morbid mechanics

In the normal cervical spine, forward flexion results in a smooth reversal of the lordosis throughout its length. The column and cord lengthen by about 3 mm and the elasticity of the cord accommodates this lengthening without significant tensile stresses being generated.[8] There is an increase of about 2 mm in the available space within the canal. This is due to the resulting segmental configuration and the decrease in the circumference of the cord as it slightly stretches.[24,25] In extension, the lordosis increases and the space available in the nerve root canal decreases slightly.[8] In flexion, the reverse occurs as the space decreases and the cord's diameter increases when it slackens.[26,27]

In the past, controversy existed as to whether the cord lengthened in an accordian-like fashion, or through translational sliding within the canal. This has importance when considering the implications of nerve root tethering. However, it seems that during flexion both types of motion occur.[13,25,28]

In the healthy spine, there is sufficient room to accommodate the changes in canal and cord dimensions that occur with neck movements. When the vertebral column is stable, there is little or no segmental translation during flexion or extension. Although the cord and dura may flatten slightly against the bodies in flexion, the bony configuration is curvilinear, so that angular bending or shearing forces are avoided. However, this does not occur in the degenerated spine.

Spondylotic mechanics

In the degenerated spine, the resulting aberrant mechanics, when superimposed on a congenitally narrow canal, render the cord and its vasculature vulnerable to compression during movement. White and Panjabi,[29] in applying engineering principles to study the effects of cervical stenosis, agree that neurological or vascular insult can result at the extreme of either flexion or extension. They have isolated the four main destructive forces in CSM as: 1) direct compression, 2) bending, 3) stretching (tension) and 4) shear. The first two result from osteophytes pressing on the cord, while tension is secondary to fibrotic tethering within the dura of the cord and nerve roots. Shear forces arise within the cord when the unstable vertebra translates anteriorly or posteriorly across the others. As the cord stretches in flexion, it may have to bend over an anteriorly located osteophytic spur or discal spondylotic bar. This results in mechanical deformation and/or vascular compression. In addition, fibrosis within the nerve root sleeve may tether the cord, further increasing the tension. In extension, the caudal aspects of the stenosed canal cannot accommodate the increased cross-sectional area of the cord, as tension is lessened. Further extension movements may lead to cord compression from posterior protrusion of the discs' annulus and/or inward buckling of the thickened ligamentum flavum.

The effect of these forces upon the cord, can be predicted if the spinal cord is viewed as an elliptical structure. Each of the forces described have their greatest effect at different zones in elliptical structures, such as the spinal cord. As White and Panjabi[29] point out, these selective areas correlate well to known patterns of ischemia or column destruction. Ogino et al.[30] noted that various areas within the cord have different vulnerabilities to compression. The posterolateral columns seem most susceptible to mechanical compression, while the anterior cord suffers most from ischemia or infarct.

Studies show that the majority of patients with severe myelopathy have spondylosis-induced instability.[31,32] Adams and Logue[33,34] identified a significant difference in the quality and quantity of cervical vertebral motion between spondylotic patients with myelopathy compared to those without. They have noted that immobilizing myelopathic patients with a collar is often as effective as surgical decompression. Barnes and Saunders[32] followed conservatively-managed CSM patients and concluded that increased cervical mobility should be used as a criteria to help identify those likely to deteriorate.

It can be seen, therefore, that the above principles have important implications in the management and the development of a predictive algorithm relating internal stresses to degenerative pathology.

Summary

In this the first of two parts, we have reviewed the relevant anatomy and pathomechanics related to cervical spondylotic myelopathy. The normal relationship between the osseous, vascular and neurological structures helps explain the predilection of certain patients to CSM.

In summary, the spinal cord makes up about 75% of the available canal space. The cord is mostly supplied by the anterior spinal artery. The cord and artery may become compromised as a result of progressive degenerative changes that also affect the functional stability of the cervical motion segments. When altered stability is combined with a narrowed sagittal canal diameter further cord compression may ensue at the extremes of either flexion and/or extension. Such compression of the cord and its adjacent anterior spinal artery, renders the patient vulnerable to cord ischemia and the resulted neurological complications.

In Part II, we will review the clinical manifestations of CSM, the imaging modalities available for the assessment and the management of these patients.

Acknowledgements

The authors would like to acknowledge the financial assistance of the Chiropractic Foundation for Spinal Research. We also appreciate the help of Ms. Carol Hagino with the graphics and Mr. Dan McGinty, CMCC Audiovisual Department, with the photographs.

References

1 Brain R. Cervical spondylosis. Ann Intern Med 1954; 41:439.
2 Simeone FA, Rothman RH. Cervical disc disease. The Spine. Philadelphia: WB Saunders Company, 1982: 440–476.
3 Clarke E, Robinson PK. Cervical myelopathy: a complication of cervical spondylosis. Brain 1956: 79:83.
4 Lestinini W, Wiesel S. The pathogenesis of cervical spondylosis. Clin Orthop Rel Research 1989; 239:69–93.
5 Coventry MB. Anatomy of the intervertebral disc. Clin Orthop Rel Research 1969; 67:9.
6 Coventry MB, Ghormley RK, Kernohan JW. The intervertebral disc: its microscopic anatomy and pathology, part II. Changes in the intervertebral disc concommitant with age. J Bone Joint Surg 1945; 27:223–237.
7 Cailliet R. Neck and arm pain. 2nd ed. Philadelphia: FA Davis, 1981.
8 Parke WW. Correlative anatomy of cervical spondylotic myelopathy. Spine 1988; 13:831–837.
9 Wilkinson M. Cervical spondylosis. Its early diagnosis and treatment. 2nd ed. London: Heinemann Medical Books, 1971:37.
10 Ferguson RJL, Caplan LR. Cervical spondylotic myelopathy. Neurologic Clinics 1985; 3:373–382.
11 Hoff J, Nishimma M, Pitts L, Vilnis V, Tuerk K, Lagger R. The role of ischemia in the pathogenesis of cervical spondylotic myelopathy. A review and new microangiographic evidence. Spine 1977; 2:105–108.
12 Breig A, Turnbull I, Hassler O. Effects of mechanical stresses on the spinal cord in cervical spondylosis; a study of fresh cadaver material. J Neurosurg 1966; 25:45.
13 Adams CBT, Logue V. Studies in cervical spondylotic myelopathy. I. Movement of the cervical roots, dura and cord, and their relation to the course of the extrathecal roots. Brain 1971; 94:557–568.
14 Jeffreys E. Disorders of the cervical spine. London: Butterworth, 1980: 90–105.
15 McGregor M, Mior S. Anatomical and functional perspectives of the cervical spine: part III: the unstable cervical spine. J Can Chiropr Assoc 1990; 34(3):145–152.
16 Ehni G. Cervical arthrosis. Diseases of the cervical motion segments. Chicago: Year Book Medical Publishers, 1984: 26–43.
17 Ehni G. Developmental variations, including shallowness of the cervical canal. In: Post MJ (ed). Radiographic Evaluation of the Spine. New York: Masson, 1980: 469–474.
18 Allen KL. Neuropathies caused by bony spurs in the cervical spine with special reference to surgical treatment. J Neurol Neurosurg Psychiatry 1952; 15:20.
19 Mair WGP, Druckman R. The pathology of spinal cord lesions and their relation to the clinical features in protrusions of cervical intervertebral discs. Brain 1953; 76:70.
20 Gooding MR, Wilson CB, Hoff JT. Experimental cervical myelopathy: effect of ischemia and compression of the canine cervical spinal cord. J Neurosurg 1975; 43:9.
21 Hukuda S, Ogata M, Katsuura A. Experimental study on acute aggravating factors of cervical spondylotic myelopathy. Spine 1988; 13:15–20.
22 Taylor AR. Vascular factors in the myelopathy associated with cervical spondylosis. Neurology 1964; 14:61–63.
23 Suh TH, Alexander L. Vascular system of the human spinal cord. Arch Neurol Psychiatry 1939; 41:659–677.
24 Robinson RA, Afeiche N, Dunn EJ, Northrup BE. Cervical spondylotic myelopathy: etiology and treatment concepts. Spine 1977; 2:89–99.
25 Breig A. Adverse mechanical tension in the central nervous system. New York: J Wiley and Sons, 1978.
26 Penning L. Functional pathology of the cervical spine. Baltimore: Williams and Wilkins, 1968.
27 Penning L, Wilmik JT, vanWoerden HH, Knol L. CT myelographic findings in degenerative disorders of the cervical spine: clinical significance. Am J Neuro Rad 1986; 7:119.
28 Rayner RB, Koplik B. Cervical cord trauma. The relationship between clinical syndromes and force of injury. Spine 1985; 10:193–197.
29 Panjabi M, White A. Biomechanics of nonacute cervical spinal cord trauma. Spine 1988; 13:838–842.
30 Ogino H, Tada K, Yonenobu K, Yamamoto T, Ono K, Namiki H. Canal diameter, anteroposterior compression ratio, and spondylotic myelopathy of the cervical spine. Spine 1983; 8:1–15.
31 Bohlman HH. Cervical spondylosis with moderate to severe myelopathy: a report of seventeen cases treated by Robinson anterior cervical discetomy and fusion. Spine 1977; 2:151–561.
32 Barnes MP, Saunders M. The effect of cervical mobility on the natural history of cervical spondylotic myelopathy. J Neuro Neurosurg Psychiatry 1984; 47:17–20.
33 Bishop PB. Proteoglycans and degenerative spondylosis. J Manip Physio Ther 1988; 11(1):36–40.