YALE JOURNAL OF BIOLOGY AND MEDICINE 66 (1993), pp. 165–177

Cervical Spondylotic Myelopathy: A Review of Surgical Indications and Decision Making

Melvin D. Law, Jr., M.D.[a], Mark Bernhardt, M.D.[b], and
Augustus A. White, III, M.D.[a]

[a]*Department of Orthopaedic Surgery and Daniel E. Hogan Spine Fellowship, Beth Israel Hospital/Harvard Medical School, Boston, Massachusetts, and*

[b]*Dickinson-Direley Midwest Orthopaedic Clinic, Kansas City, Missouri*

(Submitted March 16, 1993; sent for revision May 19; received and accepted May 26, 1993)

Cervical spondylotic myelopathy (CSM) is frequently underdiagnosed and undertreated. The key to the initial diagnosis is a careful neurologic examination. The physical findings may be subtle, thus a high index of suspicion is helpful.

Poor prognostic indicators and, therefore, absolute indications for surgery are: 1. Progression of signs and symptoms. 2. Presence of myelopathy for six months or longer. 3. Compression ratio approaching 0.4 or transverse area of the spinal cord of 40 square millimeters or less. Improvement is unusual with nonoperative treatment and almost all patients progressively worsen. Surgical intervention is the most predictable way to prevent neurologic deterioration.

The recommended decompression is anterior when there is anterior compression at one or two levels and no significant developmental narrowing of the canal. For compression at more than two levels, developmental narrowing of the canal, posterior compression, and ossification of the posterior longitudinal ligament, we recommend posterior decompression. In order for posterior decompression to be effective there must be lordosis of the cervical spine. If kyphosis is present, anterior decompression is needed. Kyphosis associated with a developmentally narrow canal or posterior compression may require combined anterior and posterior approaches. Fusion is required for instability.

CSM[c] is defined as spinal cord dysfunction secondary to extrinsic compression of the spinal cord and/or its vascular supply [1] from degenerative disease of the cervical spine. It is the most common cause of spinal cord dysfunction in patients who are older than fifty-five [2, 3]. The pathology may be associated with congenital or developmental stenosis of the cervical canal. The pathogenesis begins with degenerative changes in the disc [1] and this causes changes in the osseous and soft tissue structures. Encroachment of the available space in the spinal canal and spinal cord is caused by deformation of the facet and uncovertebral joints with associated osteophytic spurring. Soft tissue compression results from intervertebral disc herniation and invagination of the ligamentum flavum into the canal. This is thought to be due to decrease in disc space height and loss of elasticity of the ligamentum flavum [4]. There may also be thickening of the ligamentum flavum [4–6]. Ligamentous laxity from degenerative changes, inflammatory disease,

[a]*To whom correspondence should be addressed.* Augustus A. White, III, M.D., Beth Israel Hospital, 330 Brookline Avenue, Boston, MA, 02215.
[c]*Abbreviations used:* CSM, cervical spondylotic myelopathy; MRI, magnetic resonance imaging; CT, computerized axial tomography; PLL, Posterior longitudinal ligament; OPLL, ossification of posterior longitudinal ligament.

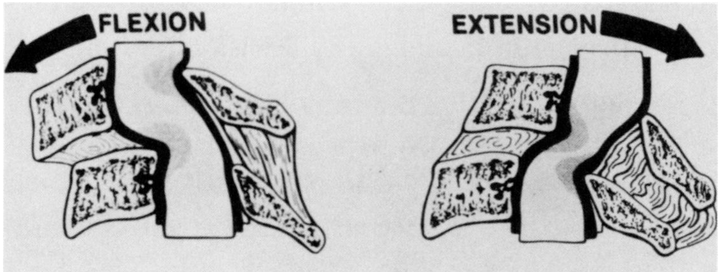


Figure 1. The pincer mechanism [4, 6] occurs when normal or abnormal motion combined with dynamic mechanical factors create various patterns of cord encroachment.

or trauma may allow anterior or posterior subluxation of the cervical vertebrae which may cause cord compression through a pincer mechanism [4, 6] (Figure 1). Physiologic motion of the neck without subluxation can produce a pincer mechanism when there are morphologic changes of the posterior vertebral body, spurring, and protrusion of the anterior lamina or ligamentum flavum into the canal [5, 7–13].

The clinical evaluation includes a history, physical exam, and radiographs. MRI and/or myelography followed by CT scanning are the most useful imaging studies for surgical planning [2, 14]. Electrophysiologic studies are usually not necessary for the diagnosis but may be useful in following the patient's progress [15].

Characteristically, patients with CSM complain of neck pain, difficulty walking, and unsteadiness of the feet. In the upper extremities, pain, numbness, paresthesias, weakness, and loss of dexterity are common complaints. There is usually coexisting compression of the nerve roots causing a radiculopathy of the involved nerve. Bladder dysfunction may occur but is not common. Coexisting lumbar spinal stenosis is common [16].

Physical examination may show upper motor neuron findings in the lower extremities and lower motor neuron findings in the upper extremities at the level of the lesion. A good phrase to remember is "uppers in the lowers and lowers in the uppers." There also may be upper motor neuron findings in the upper extremities below the level of the lesion; thus, deep tendon reflexes may be hyperactive in both upper and lower limbs. The following signs which are indicative of myelopathy may be present: the inverted radial reflex [17], Hoffmann's reflex, and clonus. Extending the neck during testing increases the sensitivity of Hoffmann's sign [18]. The inverted radial reflex is a spontaneous flexion of the digits when attempting to elicit the brachioradialis reflex. When this is present, it is almost pathognomonic of CSM. This is because there is spondylosis at C5-6 which is the most common level for this pathology. A large spur of disc has eliminated the C6 motor (brachioradialis) and compressed the cord enough to cause an upper motor neuron deficit and the spontaneous (spastic) contraction of the finger flexors which are innervated lower at C8. Abnormal plantar responses usually do not occur until myelopathy becomes severe [17]. Sensory examination may reveal changes in pain, temperature, and light touch, but more specific for myelopathy are changes in position and vibratory sensation. The earliest findings are dysdiadokinesis, difficulty with tandem gait, and subtle deficits in fine motor function.

Plain radiographs, anterior posterior, oblique, and lateral views frequently demonstrate disc space narrowing. Posterior, foraminal, and uncovertebral osteophytes also may be seen. The lateral view may show spondylolisthesis, lordosis, or kyphosis. The amount of developmental stenosis should be assessed by measuring the Pavlov ratio [19, 20] (Figure 2). A ratio of 1.0 is normal, and less than 0.8 indicates a developmentally narrow

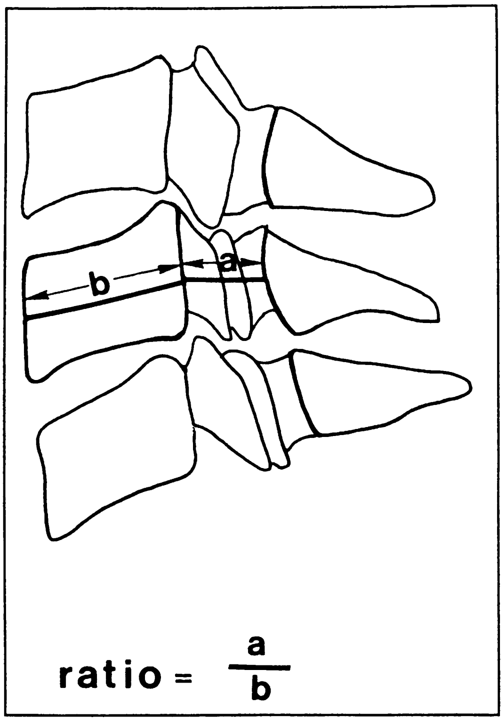


Figure 2. The normal Pavlov ratio [20] is one, a ratio of less than 0.8 indicates a narrow canal.

canal. Lateral flexion and extension radiographs are a useful test for instability [4].

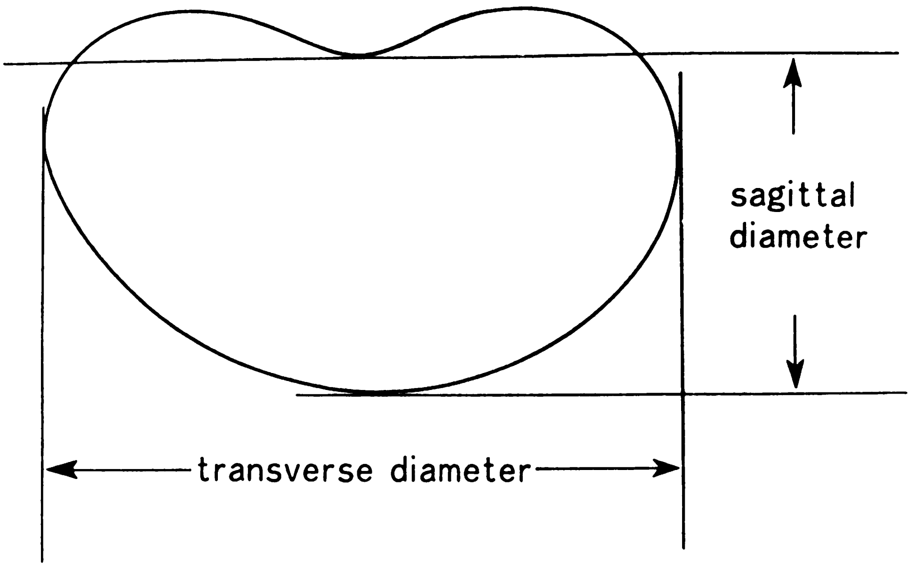
MRI is preferable for evaluation of the soft tissue structures and spinal cord [21]. Dynamic MRI scanning with flexion and extension can be very useful in documenting dynamic cord compression [5]. This may occur in the absence of instability on flexion and extension views when there is a hypotonic ligamentum flavum and dynamic annular bulging [10–12]. Myelography followed by CT scanning can be an adjunct to MRI and help distinguish disc material from osteophyte [21].

A crucial measurement that can be made on the axial MRI or myelographic CT scan is the compression ratio [6, 22] (Figure 3). The compression ratio is measured by taking the smallest anterior to posterior measurement of the spinal cord and dividing this by the broadest transverse diameter at the same level. A compression ratio of less then 0.4 or a transverse area of less than 40 square millimeters correlates with histological and clinical evidence of myelopathy and a guarded prognosis for recovery after decompression [22]. When the compression ratio is greater than 0.4 or the transverse area is greater than 40 square millimeters, improvement of these measurements on postoperative imaging correlates with clinical recovery.

Careful attention to the exam and a complete work-up is necessary to make the diagnosis and to rule out other causes of spinal cord dysfunction. These include cerebrovascular disease, arteriovenous malformation, demyelinating disease, syringomyelia, intracranial tumor, posterior fossa tumor, hydrocephalus, tabes dorsalis, myopathy, neuropathy, metabolic, and alcoholic encephalopathy.

SURGICAL INDICATIONS

The natural history of CSM has not been thoroughly defined and documented. Almost all patients worsen if left untreated and most studies report significant numbers (over 50%) of patients progressing to severe disability [23–29]. At the time of this writ-



$$\text{compression ratio} = \frac{\text{sagittal diameter}}{\text{transverse diameter}} \times 100\ (\%)$$

Figure 3. The compression ratio [22] is a good index of cord pathology. A compression ratio of 40% is considered abnormal. The cross sectional area [22] is a more reliable indicator of cord damage and clinical deficits and is abnormal at 40 square millimeters.

ing, there is no known method of identifying those patients that will develop rapid catastrophic neurologic deterioration [25]. Although the prognosis for recovery after surgery is not known [30], the best results are obtained in patients who are decompressed within six months to one year after the onset of symptoms and in those with early, mild myelopathic findings [31]. Until more is known about the natural history of CSM, we recommend that documented evidence of CSM is an indication for surgical treatment. Absolute indications for operative treatment are progression of neurologic deficits and failure to improve with nonoperative treatment, especially if CSM has been present for six months or longer. Because of the less favorable prognosis for recovery with a compression ratio of less than 40% or transverse of 40 square millimeters, we recommend that surgery be done before this degree of cord deformation occurs.

NON-OPERATIVE MANAGEMENT

The non-operative management of CSM consists of immobilization in a firm cervical orthosis, anti-inflammatory medications, physical therapy emphasizing isometric muscle strengthening, and other symptomatic measures such as heat, ice, and massage. Patients being treated nonoperatively should be monitored closely. In patients who have a high operative risk or significant co-morbid factors such as many of the elderly, cervical epidural steroid injections may be useful [32]. We advise against traction because patients can be made significantly worse by it [18]. Manipulation is contraindicated because extension of the neck narrows the spinal canal [7–12] and the foramen [33].

OPERATIVE MANAGEMENT

The primary goal of surgery for CSM is decompression of the spinal cord. There is a

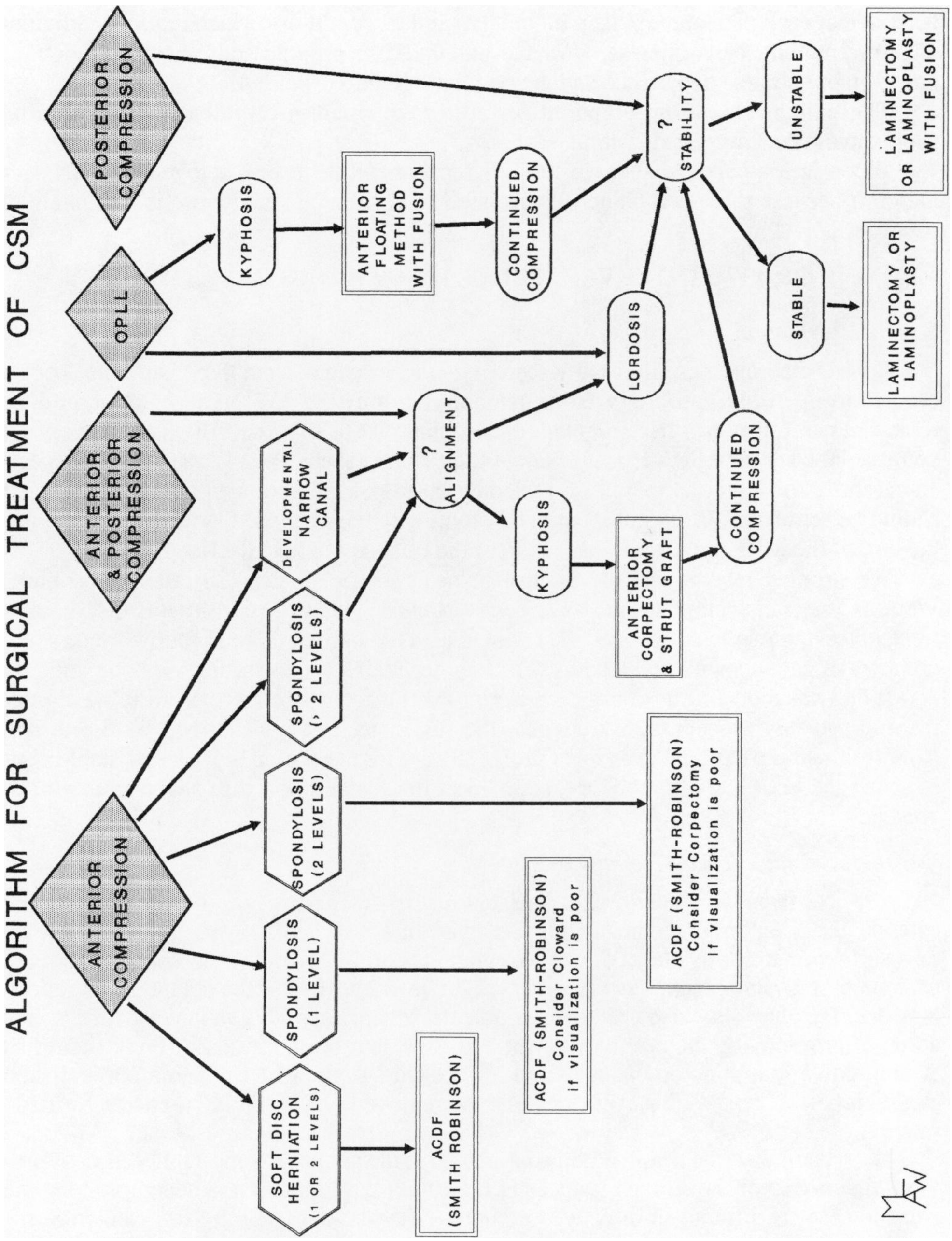


Figure 4. CSM treatment algorithm.

secondary goal which is to stabilize the region of the spinal column where there is myelopathy and instability. The goals of decompression are to remove the spinal cord and root impingement with the least surgical risk and the least disruption of the structural integrity of the spinal column [4]. Decompression may be achieved using an anterior, a posterior, or a combined approach. There is debate about which is the best approach. Patients over seventy years of age with significant neurologic deficits and medical problems also present controversies regarding operative risk-benefit issues [34, 35]. We believe that each patient presents with unique clinical conditions which requires individualized treatment. Nevertheless, we have attempted to provide some useful guidelines based on our clinical experience and the current literature (Figure 4).

The patient should be prepared for an awake fiberoptic intubation. Positioning should avoid neck hyperextension.

When fusion is performed, we strongly recommend the use of autogenous graft over allograft because there is a higher fusion rate and postoperative collapse is less likely to occur [36].

ONE OR TWO INTERVERTEBRAL DISC LEVELS OF PATHOLOGY

Soft disc herniation

Soft disc herniations are usually seen in younger patients, and there is minimal or no contribution to the pathology from osteophytic spurring [37]. The MRI should be reviewed carefully for a free fragment of disc material behind the PLL. The disc should be resected back until the vertical fibers of the PLL are seen. The PLL should be inspected carefully for a rent and any disc material that may have herniated through the defect should be retrieved. We do not recommend routine removal of the PLL and inspection of the dura if the MRI and careful inspection of the PLL do not reveal a free fragment.

We strongly recommend the Smith-Robinson anterior fusion [38] after discectomy. We note that satisfactory results have been obtained without fusion in soft disc herniations involving one level [37, 39]. These studies have limited clinical follow-up or small numbers of patients with myelopathy. The long-term effects concerning kyphotic collapse and spondylosis occurring at the discectomy level are not known; therefore, we do not recommend this procedure. We suggest the fusion technique described by Smith and Robinson using tricortical iliac crest graft [38] because it provides the most stable construct, is the least likely to collapse, and allows for distraction if disc space narrowing is present.

Degenerative disc disease (spondylosis)

Cervical spondylosis with its associated disc space narrowing, disc herniation, and osteophyte formation is the most common cause of CSM. Osteophytes have been shown to resorb with solid fusion [40]. If the osteophytes are involved in the compression, are prominent, associated with a developmentally narrow canal, or if the compression ratio is less than 0.5 they should be removed to ensure adequate space available for the spinal cord. After removing the disc back to the PLL, the spur on the posterior body should be ground down to a thin cortical shell and left attached to the PLL. The thin cortical shell can then be cracked off. If necessary it can be removed with a small curette or Kerrison rongeur.

We recommend the Smith-Robinson fusion for the above reasons, and because it provides distraction of the collapsed disc space opening the foramen and decompressing the nerve root. It is difficult to directly decompress the nerve root in the foramen from the anterior approach. Another benefit of distraction is the tension that is placed on the ligamentum flavum which reduces an invaginated ligamentum when present. Excessive distraction should not be attempted because of the possibility of increasing tension on the spinal cord [4, 41]. Axial tension on the spinal cord may play a role in the pathophysiology of CSM [41].

A limitation of the Smith-Robinson technique is the potential for less visualization posteriorly for osteophyte removal. The Cloward procedure [42] provides improved visualization, but it is probably less stable [43], provides no distraction, and there is a potential for more postoperative collapse because the anterior end plates are removed. For two level involvement, vertebral corpectomy provides the best visualization but is potentially the most destabilizing procedure and it has a higher complication rate [44].

One or two level spondylosis with mild developmental narrowing of the canal

We define mild developmental narrowing of the canal as a Pavlov ratio of less than 1.0 and greater than 0.8. The Pavlov ratio is not affected by variations in magnification that occurs with radiographs; therefore, it is a better guide than measuring the size of the canal directly.

If the compression is purely anterior, a successful result can be expected from the anterior approach. If the adjacent levels have degenerative changes, the patient is at risk for developing stenosis adjacent to the level of anterior fusion and consideration should be given to posterior decompression. We prefer the anterior approach when it is apparent that there will be enough space available for the cord after decompression. This is recommended because it limits the surgery to the involved levels and addresses the pathology directly. In our opinion, if progressive improvement of myelopathy does not occur within three to six months and postoperative imaging studies do not show a significant improvement in the compression ratio, we suggest posterior decompression.

One or two level spondylosis with narrowing of the canal, posterior, or combined anterior and posterior compression

When there is posterior compression, combined anterior and posterior compression, or a developmentally narrow canal, a posterior approach is preferable [16, 45]. The anterior approach in this situation has a higher risk because the dura and spinal cord are pressed against the PLL in the stenotic canal. Epstein has suggested that the posterior decompression should extend one or two levels above and below the level of anterior pathology to allow the dura and cord to migrate dorsally [16]. The decision of whether to decompress one level or two levels adjacent to the compression is based on the amount of stenosis and anterior compression, the more severe the pathology, the more decompression is needed. We suggest that any level with a compression ratio of 0.5 or less should be decompressed. Assessment of decompression intraoperatively can be difficult. Instillation of water soluble contrast may be helpful in preventing inadequate decompression [46]. When in doubt, it is preferable to err on the side of too much decompression [47].

MORE THAN TWO INTERVERTEBRAL DISC LEVELS OF SPONDYLOSIS

When more than two disc levels are involved in the pathology, we recommended the posterior approach. This is somewhat controversial because successful results can be obtained with the anterior approach [48–50]. Disadvantages of the anterior approach in this situation are destabilization of the spine, fusion of multiple levels, and graft dislodgement [51, 52]. The advantages of the posterior approach are direct visualization of the nerve roots [53] and the avoidance of multiple levels of fusion.

Another controversial question is whether laminoplasty or laminectomy should be the posterior procedure of choice [16, 54–59]. The individual situation of each particular patient should be the deciding factor. Post laminectomy kyphosis is uncommon [16, 58–61] except in the growing cervical spine. Laminectomy has the mythological disadvantage of destabilizing the spine and allowing kyphosis to occur. A well designed, controlled, prospective study with a five-year follow-up comparing laminectomy and laminoplasty revealed no difference [58]; another more recent study also supports no difference between laminectomy and laminoplasty [60].

The facet joints are very important for spinal stability. In cadaver studies, it has been demonstrated that preservation of 50%t of the facet joint preserves stability [62]. This study was done using normal spines; theoretically, in cervical spondylosis, there may be compromise of the facet joints leading to more loss of support with partial facet resections [53]. In the case where a partial facetectomy presents questions about stability, we recommend laminoplasty. If a destabilizing facet resection is required, a posterior fusion should be performed. However, in this case laminoplasty may be considered at the stable levels combined with fusion at the unstable level (or levels).

There is loss of motion from laminoplasty especially in neck extension; however, this may help prevent the progression of spondylosis and development of deformity. Laminoplasty has the disadvantage of the lamina closing back down causing recurrent stenosis [55].

Cervical lordosis must be present for the success of posterior decompression. A bowstring effect occurs allowing dorsal migration of the spinal cord away from the anterior compression thereby providing decompression [16]. This also allows the spinal cord to shorten which decreases the axial tension and improves perfusion of the cord [16]. Kyphotic deformity of the cervical spine is a contraindication to posterior decompression [16, 63] as the cord cannot be expected to migrate away from the anterior compressing structures.

For laminectomy, we suggest a technique similar to the open door laminoplasty technique [57]. A high speed burr and small kerrison are used to thin out and cut the lateral most aspect of the lamina. Instead of hinging open the opposite side, as one would do for the laminoplasty, the same technique of cutting the lamina is used. The lamina can then be lifted and freed from the underlying dura. This technique avoids having to place instruments beneath the lamina into the canal. There have been several techniques for laminoplasty described [54, 56–60]. The relative advantages of these surgical variations on a theme have not yet been determined.

It is useful to point out that in the operative procedure, the erector spinae muscles should not be dissected laterally beyond the facets. This is in order to avoid denervation of the posterior cervical muscles which may cause postoperative kyphosis [16]. A five millimeter thick free fat graft should be placed over the exposed dura to minimize adhesions or formation of a post laminectomy membrane which may cause post laminectomy neurologic deterioration [64].

More than two level spondylosis with or without posterior compression, with or without a developmentally narrow canal

In these situations, we recommend a laminectomy in most patients. This is a safe, reliable, and effective procedure. If there are concerns about post laminectomy instability because of hypolordosis, facet resection or deformity, hypermobility, or erector spinae muscle dysfunction, a laminoplasty can be considered.

Kyphosis and CSM

The preferred approach is anterior corpectomy and strut grafting [52, 54]. The lateral borders of the vertebral body should be preserved. For two disc levels, iliac crest strut graft is adequate; for greater than two levels, a fibular strut graft is superior [48, 52]. Post operative use of halo immobilization minimizes the probability of graft dislodgement, spinal malalignment, and pseudoarthrosis. Anterior internal fixation with plates and screws is becoming a popular technique to augment stability of these constructs and possibly eliminate the need for a halo [65–68]. We recommend a conservative and gradual approach to the use of anterior internal fixation until the risks and benefits are more clearly delineated.

In the unusual situation of spondylosis with kyphosis and posterior compression or developmental narrowing of the canal, an anterior approach can be done if it appears there will be adequate space available after anterior decompression. Otherwise a combined anterior and posterior decompression is considered.

Instability and CSM

When cervical instability is present, a fusion is necessary. The guidelines for anterior and posterior decompression are similar. Each case should be individualized because of different levels and degrees of instability. In severe instability and instability with kyphosis, anterior and posterior approaches for decompression, fusion and stabilization should be considered [69].

Anterior decompression and fusion

Bone grafts should be appropriately sized and fashioned to make the most stable constructs [48, 68, 70], and consideration should be given to halo immobilization to maintain the position. Catastrophic neurologic injury can result from graft dislodgement with cord impingement [52, 70, 71]; this has been reported even with use of the halo [70].

Internal fixation has the theoretical advantages of improving the stability of the graft construct and possibly eliminating the need for a halo in selected patients [67, 72]. The anterior plate fixation system developed by Morscher [73] is appealing because there is no requirement that the screws penetrate the posterior cortex. The screws are locked into the plate minimizing the complication of the screw loosening and backing out causing esophageal erosion and other complications.

Posterior decompression and fusion

Posterior cancellous iliac crest bone graft is preferred; however, after laminectomy there may be sufficient bone to serve as graft for posterior fusion.

If the facets are intact and will hold wire fixation after posterior decompression, the posterior cervical wiring technique described by Robinson and Southwick [74] is a useful procedure that adds little to the operative risks. When the facets are deformed by the disease or removed by the decompression, posterior cervical plates or rods with screws that achieve fixation to the lateral masses is a reasonable option and is being employed more frequently for posterior cervical fusions [67, 75]. This technique carries the risk of injury to the nerve root or vertebral artery [67]; therefore, a thorough knowledge of the anatomy and a careful precise application of well practiced technique is required to avoid these complications.

Ossification of the Posterior Longitudinal Ligament (OPLL)

This condition was previously thought to be confined to Asians [51, 56]; however, it is being reported with increasing frequency in Occidental patients [51, 71]. When OPLL occurs in non-orientals, the clinical, radiographic, and histologic features are identical to those of oriental patients [51]. About one-half of patients with OPLL have diffuse idiopathic skeletal hyperostosis (Forestier's disease) on plain radiographs [51, 76]. Enhanced CT scanning is the best method for confirming the diagnosis of OPLL [51]. Awareness of the possibility of OPLL occurring in non-orientals and accurately making the diagnosis is important when managing patients with CSM to avoid catastrophic surgical complications [6, 51 ,71].

The PLL ossifies and thickens causing anterior compression of the spinal cord. In some cases, stability is enhanced by the ossified PLL allowing for a more extensive decompression [6]. At the time of presentation, there may be few levels of involvement; however, the disease may progress to involve the PLL at multiple levels [49].

We feel that the best surgical procedure for this condition is posterior decompression. This is largely because of serious and potentially fatal complications that can occur with anterior decompression [6, 51, 71]. Because the dura may be absent [71] or the ossified PLL may be adherent to the dura, attempts to remove the ossified PLL can result in major dural injuries that are often difficult or impossible to repair [51]. In a series of twenty-two patients who underwent anterior decompression, there were numerous complications, and three patients developed significant postoperative neurological deficits that were thought to be directly related to manipulation of the ossified PLL during surgical removal [51]. Anterior decompression is required when OPLL is associated with kyphosis. The anterior floating method where the disc and a portion of the vertebral body is removed allowing the ossified PLL to be gently pulled forward without actual removal [77, 78] has had an acceptable success rate, but the complication rate is still higher than posterior decompression [77].

CONCLUSION

Although the treatment of CSM is controversial, this review presents our recommended approach based on our experience and the most current literature. More prospective controlled studies are needed to provide the information that is necessary to make definitive decisions about which patients should be operated and what operation should be done [79, 80]. When more studies are completed, we will be able to keep the risk-benefits of our surgical decision making in the patients favor. In the meantime, if we decompress the spinal cord or nerves with the least possible surgical risk and the least disruption of the structural integrity of the spinal column, we will be making informed rational decisions for our patients with all the various manifestations of CSM.

REFERENCES

1. Parke, W. W. Correlative anatomy of cervical spondylotic myelopathy. Spine 13:831–837, 1988.
2. Bernhardt, M., Hynes, R. A., Blume, H. A., and White, A. A., III. Current Concepts Review: Cervical spondylotic myelopathy. J. Bone and Joint Surg. 75A:119–128, 1993.
3. Whitecloud, T. S. Anterior surgery for cervical spondylotic myelopathy. Smith-Robinson, Cloward, and vertebrectomy. Spine 13:861–863, 1988.
4. White, A. A., III and Panjabi, M. M. Clinical Biomechanics of the Spine. Second edition. Philadelphia, J. B. Lippincott, 1990, pp. 314, 511–528.
5. Epstein, N. E., Hyman, R. A., Epstein, J. A., and Rosenthal, A. D. Technical Note: "Dynamic" MRI scanning of the cervical spine. Spine 13:937–938, 1988.
6. White, A. A., III and Panjabi, M. M. Biomechanical considerations in the surgical management of cervical spondylotic myelopathy. Spine 13:856–860, 1988.
7. Adams, C. B. T. and Logue, V. Studies in cervical spondylotic myelopathy. I. Movement in the cervical roots, dura, and cord and their relations to the course of the extrathecal roots. Brain 94:557–568, 1971.
8. Adams, C. B. T. and Logue, V. Studies in cervical spondylotic myelopathy. II. The movement and the contour of the spine in relation to the neural complications of cervical spondylosis. Brain 94:569–586, 1971.
9. Adams, C. B. T. and Logue, V. Studies in cervical spondylotic myelopathy. III. Some functional effects of operations for cervical spondylotic myelopathy. Brain 94:587–594, 1971.
10. Gruninger, W. and Gruss, P. Stenosis and movement of the cervical spine in cervical myelopa-

thy. Paraplegia 20:121–130, 1982.
11. Penning, L. Some aspects of plain radiography of the cervical spine in chronic myelopathy. Neurology 12:513–519, 1962.
12. Reid, J. D. Effects of flexion-extension movements of the head and spine upon the spinal cord and nerve roots. J. Neurol. Neurosurg. Psychiatry 23:214–221, 1960.
13. White, A. A., III and Panjabi, M. M. Biomechanics of nonacute cervical spinal cord trauma. Spine 13:838–842, 1988.
14. Alker, G. Neuroradiology of cervical spondylotic myelopathy. Spine 13:850–853, 1988.
15. Cusick, J. F. Monitoring of cervical spondylotic myelopathy. Spine 13:877–880, 1988.
16. Epstein, J. A. The surgical management of cervical spinal stenosis, spondylosis, and myeloradiculopathy by means of the posterior approach. Spine 13:864–869, 1988.
17. Simeone, F. A. and Rothman, R. H. Cervical disc disease. In The Spine. Edition 2, Philadelphia, W. B. Saunders, 1982, pp. 440–476.
18. Denno, J. J. and Meadows, G. R. Early diagnosis of cervical spondylotic myelopathy: a useful clinical sign. Spine 16:1353–1358, 1991.
19. Pavlov, H., Torg, J. S., Robie, B., and Jahre, C.: Cervical spinal stenosis: determination with vertebral body ratio method. Radiology 164:771–775, 1987.
20. Torg, J. S. Pavlov's ratio: determining cervical spinal stenosis on routine lateral roentgenograms. Contemp. Orthop. 18:153–160, 1989.
21. Brown, B. M., Schwartz, R. H., Frank., E, and Blank, N. K. Preoperative evaluation of cervical radiculopathy and myelopathy by surface-coil MR imaging. A.J.R. 151:1205–1212, 1988.
22. Fujiwara, K., Yonenobu, K., Ebara, S., Yamashita, K., and Ono, K. The prognosis of surgery for cervical compression myelopathy. An analysis of the factors involved. J. Bone and Joint Surg. 71B:393–398, 1989.
23. Clarke, E. and Robinson, P. K. Cervical myelopathy: a complication of cervical spondylosis. Brain 79:483–510, 1956.
24. Epstein, J. A., Janin, Y., Carras. R., and Lavine, R. S. A comparative study of the treatment of cervical spondylotic myeloradiculopathy. Experience with 50 cases treated by means of extensive laminectomy, foraminotomy, and excision of osteophytes during the past 10 years. Acta Neurochir. 61:89–104, 1982.
25. LaRocca, H. Cervical spondylotic myelopathy: natural history. Spine 13:854–855, 1988.
26. Lees, F. and Turner, J. W. A. Natural history and prognosis of cervical spondylosis. British Med. J. 2:1607–1610, 1963.
27. Montgomery, D. M. and Brower, R. S. Cervical spondylotic myelopathy. Clinical syndrome and natural history. Orthop. Clin. North Am. 23:487–493, 1992.
28. Nurick, S. The natural history and the results of surgical treatment of the spinal cord disorder associated with cervical spondylosis. Brain 95:101–108, 1972.
29. Symon, L. and Lavender, P. The surgical treatment of cervical spondylotic myelopathy. Neurology 17:117–127, 1967.
30. Gregorius, F. K., Estrin, T., and Crandall, P. H. Cervical spondylotic radiculopathy and myelopathy: a long-term follow-up study. Arch Neurol. 33:618–625, 1976.
31. Lesoin, F., Bouasakao, N., Clarisse, J., Rousseaux, M., and Jomin, M. Results of surgical treatment of radiculomyelopathy caused by cervical arthrosis based on 1000 operations. Surg. Neurol. 23:350–355, 1985.
32. Murphy, M. J. and Lieponis, J. V. Nonoperative treatment of cervical spine pain. In: The Cervical Spine. Edited by the Cervical Spine Research Society Editorial Committee. Edition 2, Philadelphia, J. B. Lippincott, 1989, pp. 670–677.
33. Yoo, J. U., Dewei, Z., Edwards, W. T., Bayley, J. C., and Yuan, H. A. Effect of cervical spine motion on the neuroforaminal dimensions of human cervical spine. Spine 17:1131–1136, 1992.
34. Ono, K., Ota, H., Tada, K., and Yamamoto, T. Cervical myelopathy secondary to multiple spondylotic protrusions. A clinicopathologic study. Spine 2:109–125, 1977.
35. Taylor, J., Johnston, R. A., and Caird, F. I. Surgical treatment of cervical spondylotic myelopathy in elderly patients. Age Ageing 20:407–412, 1991.
36. Brown, M. D., Malinin, T. I., and David, P. B. A roentgenographic evaluation of frozen allografts versus autografts in anterior cervical spine fusions. Clin. Orthop. 119:231–236, 1976.
37. Selladurai, B. M. Cervical myelopathy due to nuclear herniations in young adults: clinical and radiologic profile. Results of microdiscectomy without interbody fusion. J. Neurol. Neurosurg. Psychiatry 55:604–608, 1992.
38. Smith, G. W. and Robinson, R. A. The treatment of certain cervical spine disorders by anterior removal of the intervertebral disc and interbody fusion. J. Bone and Joint Surg. 40A:607–624,

1958.
39. Rosenorn, J., Hansen, E. B., Rosenorn, M. A. Anterior cervical discectomy with and without fusion: a prospective study. J. Neurosurg. 59:252–255, 1983.
40. White, A. A., III Southwick, W. O., DePonte, R. J., Gainor, J. W., and Hardy, R. Relief of pain by anterior cervical spine fusion for spondylosis. A report of sixty-five patients. J. Bone and Joint Surg. 55A:525–534, 1973.
41. Breig, A. and el-Nadi, A. F. Biomechanics of cervical spinal cord relief of contact pressure on and overstretching of the spinal cord. Acta Radiol. 4:604–624, 1966.
42. Cloward, R. B. The anterior approach for removal of ruptured cervical disks. J. Neurosurg. 15:602–617, 1958.
43. White, A. A., III Jupiter, J., Southwick, W. O., and Panjabi, M. M. An experimental study of the immediate load bearing capacity of three surgical constructions for anterior spine fusions. Clin. Orthop. 91:21–28, 1973.
44. Saunders, R. L., Bernini, P. M., Shirreffs, T. G., and Reeves, A. G. Central corpectomy for cervical spondylotic myelopathy: a consecutive series with long-term follow-up evaluation. J. Neurosurg. 74:163–170, 1991.
45. Epstein, J. A., Epstein, B. S., and Lavine, L. S. Cervical spondylotic myelopathy: the syndrome of the narrow canal treated by laminectomy, foraminotomy, and the removal of osteophytes. Arch Neurol. 8:307–317, 1963.
46. Walker, J., Gillespie, R., Davis, J., and Dawson, W. Water-soluble contrast medium for intraoperative evaluation of anterior cervical discectomy: Technical note. J. Neurosurg. 68:491–492, 1988.
47. Epstein, J. A. Personal communication. February 4, 1993.
48. Bernard, T. N. and Whitecloud, T. S. Cervical spondylotic myelopathy and myeloradiculopathy: anterior decompression and stabilization with autogenous fibula strut graft. Clin. Orthop. 221:149–160, 1987.
49. Jamjoom, A., Williams, C., and Cummins, B. The treatment of spondylotic cervical myelopathy by multiple subtotal vertebrectomy and fusion. Br. J. Neurosurg. 5:249–255, 1991.
50. Saunders, R. L., Bernini, P. M., Shirreffs, T. G., and Reeves, A. G. Central corpectomy for cervical spondylotic myelopathy: a consecutive series with long-term follow-up evaluation. J. Neurosurg. 74:163–170, 1991.
51. McAfee, P. C., Regan, J. J., and Bohlman, H. H. Cervical cord compression from ossification of the posterior longitudinal ligament in non-orientals. J. Bone and Joint Surg. 69B:569–575, 1987.
52. Zdeblick, T. A., and Bohlman, H. H. Cervical kyphosis and myelopathy. Treatment by anterior corpectomy and strut-grafting. J. Bone and Joint Surg. 71A:170–182, 1989.
53. Raynor, R. B. Anterior or posterior approach to the cervical spine: an anatomical and radiographic evaluation and comparison. Neurosurgery 12:7–13, 1983.
54. Herkowitz, H. N. A comparison of anterior cervical fusion, cervical laminectomy, and cervical laminoplasty for the surgical management of multiple level spondylotic radiculopathy. Spine 13:774–780, 1988.
55. Herkowitz, H. N. The surgical management of cervical spondylotic radiculopathy and myelopathy. Clin. Orthop. 239:94–108, 1989.
56. Hirabayashi, K., Miyakawa, J., Satomi, K., Maruyama, T., and Wakono, K. Operative results and postoperative progression of ossification among patients with ossification of cervical posterior longitudinal ligament. Spine 6:354–364, 1981.
57. Hirabayashi, K. and Satomi, K. Operative procedure and results of expansive open-door laminoplasty. Spine 13:774–780, 1988.
58. Hukuda, S., Ogata, M., Mochizuki, T., and Shichikawa, K. Laminectomy versus laminoplasty for cervical myelopathy. A brief report. J. Bone and Joint Surg. 70B:325–326, 1988.
59. Yoshida, M., Otani, K., Shibasaki, K., and Ueda, S. Expansive laminoplasty with reattachment of spinous processes and extensor musculature for cervical myelopathy. Spine 17:491–497, 1992.
60. Nakano, N., Nakano, T., and Nakano, K. Comparison of the results of laminectomy and open door laminoplasty for cervical spondylotic myeloradiculopathy and ossification of the posterior longitudinal ligament. Spine 13:792–794, 1988.
61. Jenkins, D. H. R. Extensive cervical laminectomy. Br. J. Surg. 60:852–854, 1973.
62. Raynor, R. B., Pugh, J., and Shapiro, I. Cervical facetectomy and its effect on spine strength. J. Neurosurg. 63:278–282, 1985.
63. Tencer, A. F., Allen, B. F., and Ferguson, R. L. A biomechanical study of thoracolumbar spinal fractures with bone in the canal: Part I, The effect of laminectomy. Spine 10:580–585, 1985.

64. Oiwa, T., Hirabayashi, K., Uzawa, M., and Ohira, T. Experimental study on post laminectomy deterioration of cervical spondylotic myelopathy. Influences of intradural surgery and persistent spinal block. Spine 10:717–721, 1985.
65. Aebi, M., Zuber, K., and Marchesi, D. Treatment of cervical spine injuries with anterior plating. Indications, techniques, and results. Spine 16(3 suppl):S38–S45, 1991.
66. Hall, D. J. and Webb, J. K. Anterior plate fixation in spine tumor surgery. Spine 16(3 suppl):S80–S83, 1991.
67. Montesano, P. X., Juach, E. C., Anderson, P. A., Benson, D. R., and Hanson, P. B. Biomechanics of cervical spine internal fixation. Spine 16(3 suppl):S10-S16, 1991.
68. Ripa, D. R., Kowall, M. G., Meyer, P. R., and Rusi, J. J. Series of ninety-two traumatic cervical spine injuries stabilized with anterior ASIF plate Spine 16(3 suppl):S46–S55, 1991.
69. Cusick, J. F. Pathophysiology and treatment of cervical spondylotic myelopathy. Clin. Neurosurg. 37:661-681, 1991.
70. Stauffer, E. S. and Kelley, E. G. Fracture-dislocations of the cervical spine: Instability and recurrent deformity following treatment by interbody fusion. J. Bone and Joint Surg. 59A:45–48, 1977.
71. Smith, M. D., Bolesta, M. J., Leventhal, M., and Bohlman, H. H. Postoperative cerebrospinal-fluid fistula associated with erosion of the dura: findings after anterior resection of ossification of the posterior longitudinal ligament in the cervical spine. J. Bone and Joint Surg. 74A:270–277, 1992.
72. Ulrich, C., Woersdoerfer, O., Kalff, R., Claes, L., and Wilke, H-J. Biomechanics of fixation systems to the cervical spine. Spine 16(3 suppl):S4-S9, 1991.
73. Morscher, E., Sutter, F., Jenny, H., and Olerud, S. Die vordere Verplattung der Halswirbelsaule mit dem Hohlschrauben-Plattensystem aus Titanium. Chirurg. 57:702–707, 1986.
74. Robinson, R. A. and Southwick, W. O. Surgical approaches to the cervical spine. In: American Academy of Orthopaedic Surgeons: Instructional Course Lectures 17. St. Louis, C. V. Mosby, 1960 pp. 299–330.
75. Anderson, P. A., Henley, M. B., Grady, M.S., Montesano, P. X., and Winn, H. R. Posterior cervical arthrodesis with AO reconstruction plates and bone graft. Spine 16(3 suppl):S72–S79, 1991.
76. Pouchot, J., Watts, C. S., Esdaile, J. M., and Hill, R. O. Sudden quadriplegia complicating ossification of the posterior longitudinal ligament and diffuse idiopathic skeletal hyperostosis. Arthritis Rheum. 30:1069-1072, 1987.
77. Kamikozuru, M. Significance of the anterior floating method for cervical myelopathy due to ossification of the posterior longitudinal ligament. Nippon Seikeigeka Gakkai Zasshi 65:431–440, 1991.
78. Tsuyama, N. Ossification of the posterior longitudinal ligament of the spine. Clin. Orthop. 184:71–84, 1984.
79. Rowland, L. P. Surgical treatment of cervical spondylotic myelopathy: time for a controlled trial. Neurology 42:5–13, 1992.
80. White, A. A., III. Introduction. Symposium on cervical spondylotic myelopathy. Spine 13:829–830, 1988.