Review Article

Central Cord Syndrome

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Abstract

Central cord syndrome is the most common type of incomplete spinal cord injury. This syndrome most often occurs in older persons with underlying cervical spondylosis caused by a hyperextension mechanism. It also occurs in younger persons who sustain trauma to the cervical spine and, less commonly, as a result of nontraumatic causes. The upper extremities are more affected than the lower extremities, with motor function more severely impaired than sensory function. Central cord syndrome presents a spectrum, from weakness limited to the hands and forearms with sensory preservation, to compete quadriparesis with sacral sparing as the only evidence of incomplete spinal cord injury. Historically, treatment has been nonsurgical, but recovery is often incomplete. Early surgical treatment of central cord syndrome remains controversial. However, recent studies have shown benefits, particularly of early surgery to decompress the spinal cord in patients with pathologic conditions revealed by radiography or MRI.

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Spinal cord injuries (SCIs) are classified as complete or incomplete. The American Spinal Injury Association (ASIA) defines complete injury as the absence of sensory and motor function below the level of injury.[1] Conversely, with incomplete injury, some neurologic function remains below the level of injury. Incomplete injuries include central cord syndrome (CCS), anterior cord syndrome, posterior cord syndrome, and Brown-Séquard syndrome.

CCS is the most common type of incomplete SCI, comprising 15% to 25% of all cases.[2-4] This syndrome was first described by Schneider et al[5] in 1954. Classic CCS presents as underlying cervical spondylosis in the older patient (aged >60 years) who sustains a hyperextension injury without any evidence of damage to the bony spine. CCS also occurs in younger persons who sustain higher-energy trauma resulting in spinal fractures or instability.

Knowledge of the organization of the spinal cord and the pathophysiology of CCS is essential in selecting the optimal treatment method. Nonsurgical management is sufficient in some instances, but recent research has shown potential benefit from surgical management.

Anatomy

Knowledge of spinal cord anatomy is critical in understanding CCS. The spinal cord fills approximately 50% of the canal in the cervical and thoracolumbar spine. Cerebrospinal fluid, epidural fat, and dura surround the cord and fill the remainder of the canal space. A myelomere is the segment of the cord from which a nerve root arises; each is located one level above the same-numbered vertebral body in the cervical and upper thoracic regions (eg, the C5 nerve root myelomere is at the level

of the C4 vertebral body).

The neural elements within the spinal cord are arranged geographically. The long tracts extending from the brain are arranged peripherally and are composed primarily of white matter. The peripheral white matter is abundant in the cervical spine because that region includes the long tracts to the cervical, thoracic, lumbar, and sacral levels. The more central gray matter contains the lower motor neurons.

The main descending motor pathway is the lateral corticospinal tract. The upper motor neuron originates in the contralateral cerebral cortex, decussates in the midbrain, and descends on the ipsilateral lateral periphery of the spinal cord. The upper motor neuron then synapses with its corresponding lower motor neurons in the anterior horn of the gray matter. The lateral corticospinal tract has traditionally been thought to be arranged with the cervical structures more centrally located and the sacral structures more peripherally located (Figure 1). Whether this lamination exists is controversial.[6] Hand and forearm musculature are primarily supplied by the large motor axons of the lateral corticospinal tract.[6,7] The ventral corticospinal tract is a minor descending motor pathway. The motor fibers in the ventral corticospinal tract do not decussate in the midbrain, and these fibers descend on the contralateral side of the spinal cord.

Figure 1

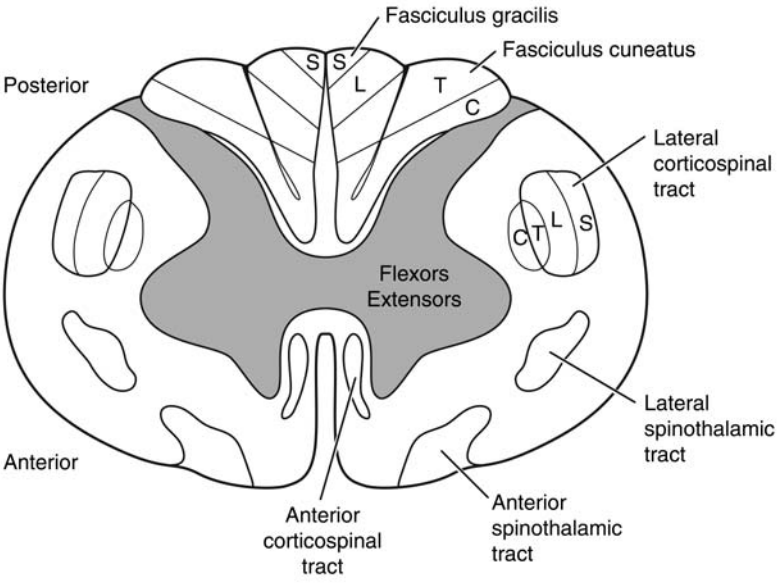

Illustration of the cervical spinal cord (axial cut). Note the orientation of the lateral corticospinal tract and dorsal column tracts (fasciculus gracilis, fasciculus cuneatus), with the sacral structures being more peripherally located and the cervical structures more centrally located. This view illustrates why central cord syndrome preferentially affects the upper extremities. C = cervical, L = lumbar, S = sacral, T = thoracic. (Adapted with permission from Gupta MC, Benson DR, Keenan TL: Initial evaluation and emergency treatment of the spine-injured patient, in Browner BD, Jupiter JB, Levine AM, Trafton PG, eds: *Skeletal Trauma: Basic Science, Management, and Reconstruction*, ed 3. Philadelphia, PA, Saunders, 2003, pp 685-707.)

The major ascending sensory pathways include the posterior column tracts (fasciculus gracilis, fasciculus cuneatus) and the smaller lateral spinothalamic tracts (Figure 1). Sensory neuron cell bodies are located in the dorsal root ganglion, and sensory input enters the posterior horn of the gray matter. Pain and temperature input immediately cross to the opposite side of the spinal cord and ascend in the contralateral lateral spinothalamic tract. Conversely, proprioception and vibratory sensation ascend ipsilaterally in the posterior column of the spinal cord and cross only after reaching the brain stem. Similar to the lateral corticospinal tract, the dorsal columns are arranged such that the sacral structures are more peripherally located and the cervical

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Figure 2

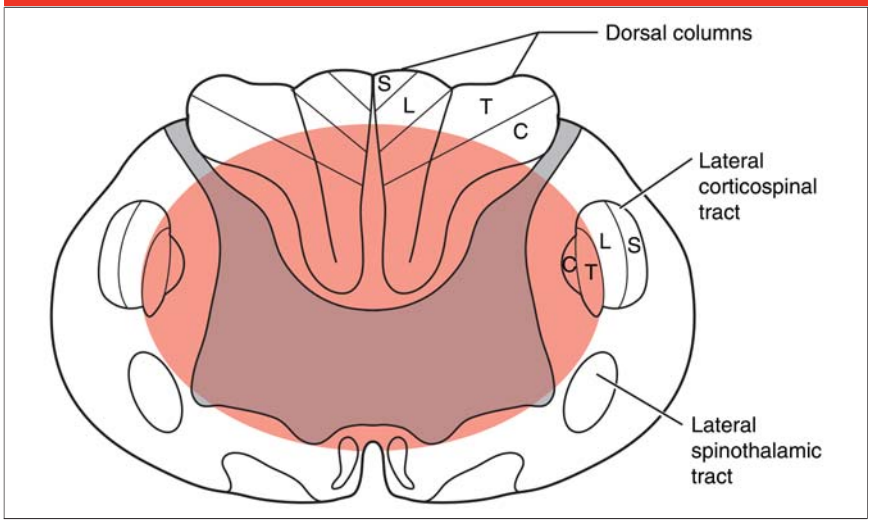


Illustration of central cord syndrome (CCS) in the cervical spinal cord (axial cut). The colored area is affected in cases of CCS. Note that the sacral structures are more peripheral in the dorsal columns and the lateral corticospinal tract; thus, those structures are preferentially spared in persons with CCS. C = cervical, L = lumbar, S = sacral, T = thoracic. (Adapted with permission from Gupta MC, Benson DR, Keenan TL: Initial evaluation and emergency treatment of the spine-injured patient, in Browner BD, Jupiter JB, Levine AM, Trafton PG, eds: *Skeletal Trauma: Basic Science, Management, and Reconstruction*, ed 3. Philadelphia, PA, Saunders, 2003, pp 685-707.)

structures are more centrally located (Figure 1).

Mechanism and Pathophysiology

SCI can be divided into two pathophysiologic phases: primary and secondary. Primary injury occurs at the time of the inciting trauma. Such injury can be caused either directly by excessive flexion, extension, and/or rotation of the spinal cord or indirectly by displaced bone or disk material having an impact on the spinal cord. Secondary injury occurs after the inciting traumatic event. Such injury is caused by an incompletely understood complex reaction involving a combination of an inflammatory response and neuronal cell apoptosis (ie, programmed cell death).

Trauma is the most common cause of CCS.[2,4,5,8-10] CCS occurs most often after motor vehicle accidents, falls, and diving injuries. CCS can also result from nontraumatic causes, such as spinal epidural abscess.[11]

The classic presentation of CCS involves an older patient with underlying cervical spondylosis who sustains an injury as the result of hyperextension of the head and neck relative to the torso.[5,8,9,12] The hyperextension mechanism often seems to be very mild but in the setting of cervical spondylosis can result in marked neurologic injury. Persons with CCS have a smaller sagittal diameter of the cervical spinal canal compared with the average, and >90% of patients with CCS aged >40 years have been shown to have underlying cervical spine conditions, such as spondylosis with osteophyte formation, canal stenosis, and ossification of the posterior longitudinal ligament.[8] Although some patients experience minor symptoms, the underlying cervical spondylosis often is asymptomatic before injury.[5,12,13] Vertebral fractures and dislocations typically are absent in persons in this age group with CCS. The cervical cord can be injured by direct compression from buckling of the ligamenta flava into an already narrowed spinal canal.

Younger persons with congenital cervical stenosis also are at increased risk of sustaining CCS as a result of hyperextension injury. CCS and its variants, including transient quadriplegia, cervical cord neurapraxia, and burning hand syndrome, have been reported in several football players with congenital stenosis.[14-16] Football players are at increased risk because of the demands of the game, particularly tackling. The terms "transient quadriplegia" and "cervical cord neurapraxia" are used interchangeably in the literature to describe significant, sometimes complete, upper and lower extremity weakness and sensory disturbances that typically last 10 or 15 minutes and then resolve on their own.

In younger persons without preexisting stenosis or spondylosis, a higher-energy traumatic mechanism is required to cause CCS. Often, the mechanism consists of a severe spinal column injury with an associated fracture-dislocation, resulting in an unstable spine.[8,9,17] A third subset of patients includes younger persons with traumatic disk herniation that results in CCS in the absence of spinal fracture or instability.[17]

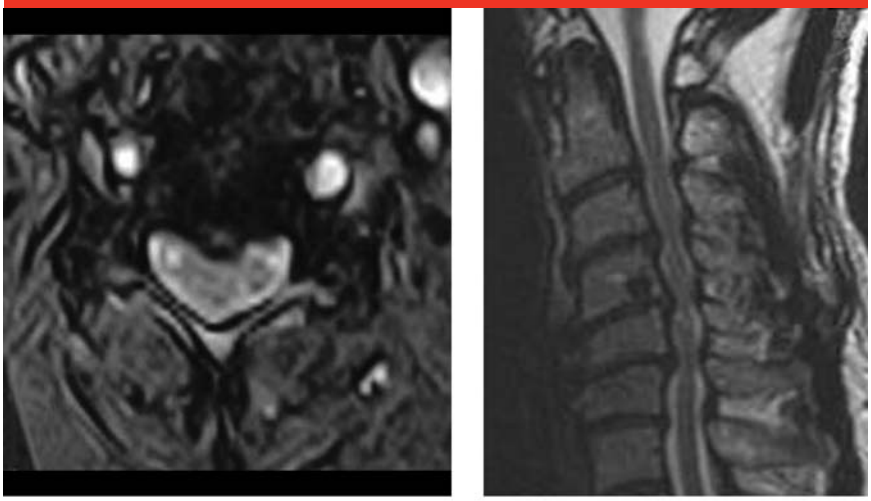
CCS originally was theorized to consist of injury to the central gray matter and the central portion of the long tracts, with preservation of the peripheral structures (Figure 2). Injury to the central gray matter and cord hemorrhage were thought to be the main causes of CCS.[5] However, recent studies have shown that the lateral cortical spinal tract in the mid to upper cervical spine contains the main area of pathologic abnormality.[6,18,19]

MRI studies have failed to show any evidence of cord hemorrhage.[8,19,20] Two autopsy studies found no evidence of lower motor neuron injury or cord parenchyma hemorrhage; however, diffuse injury to the large-diameter motor axons in the lateral corticospinal tract was noted.[18,19]

Patients with CCS exhibit wallerian degeneration of the axonal tracts distal to the zone of injury in the lateral corticospinal tracts.[18] In a feline model of SCI, large-diameter axons were shown to be more susceptible to injury than smaller axons.[21] Trombly and Guest[11] presented the only report of neuromonitoring of a case of CCS. The motor-evoked potentials were more severely affected than the somatosensory-evoked potentials, and the hand musculature, particularly the abductor pollicis brevis, was most severely affected. Thus, injury to the large myelinated axons of the lateral corticospinal tract seems to be the main cause of the deficits associated with CCS. This explains the primary involvement of the fine motor movements of the distal upper extremity.[6,7]

CCS presents on a spectrum, from weakness limited solely to the hands and forearms with sensory preservation, to complete quadriparesis with sacral sparing as the only evidence of incomplete SCI. The upper extremities are more severely affected than the lower extremities. In particular, the hands and forearms are most affected. Bladder dysfunction, typically urinary retention and bowel and sexual dysfunction, may be present in more severe cases. Motor function return, if any occurs, proceeds in a caudad to cephalad manner. Toe flexors are the first to return, followed by the toe extensors, and then the structures innervated by the lumbar cord (eg, those that enable ankle dorsiflexion). Recovery is usually less complete in the upper extremities than in the lower extremities.

Figure 3



Axial **(A)** and sagittal **(B)** T2-weighted magnetic resonance images of a 72-year-old man with underlying cervical spondylosis who developed central cord syndrome after a hyperextension injury.

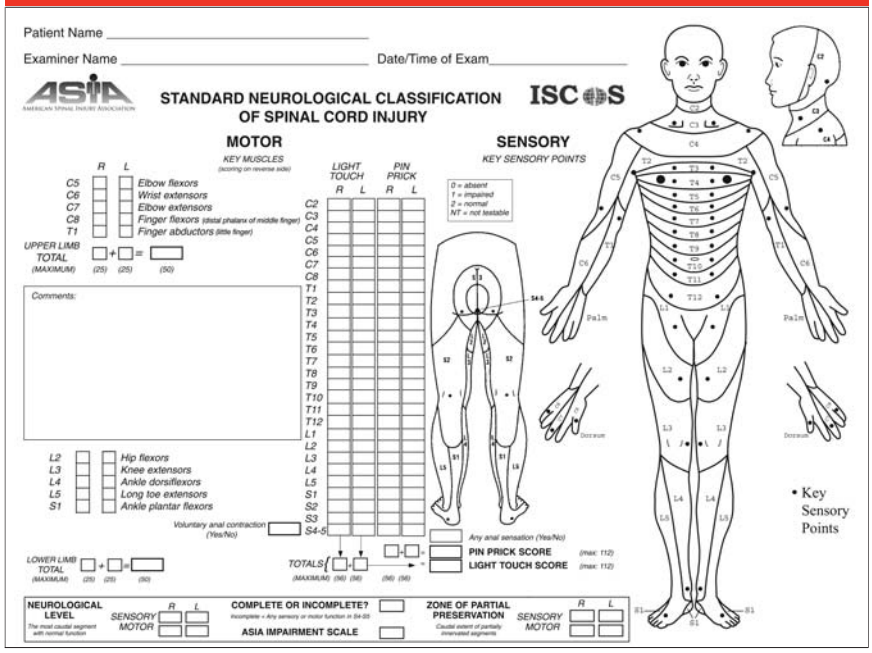
Diagnosis and Evaluation

Rapid and accurate diagnosis is essential in the patient with suspected SCI. A full radiographic evaluation should be done, typically consisting of cross-table lateral, AP, and open-mouth odontoid views. Coronal and sagittal reconstruction CT scans can be obtained to gain a better understanding of bony injury and to detect injuries that are not obvious on plain radiographs. CT is also helpful in assessing possible injuries to the occipitocervical and cervicothoracic junctions. In trauma centers, CT frequently is performed either in addition to or instead of traditional radiography. Because of the approximately 10% to 20% incidence of noncontiguous spine fractures, when a cervical spine fracture is identified, the entire spine often is imaged with either plain radiography or CT.[22,23] MRI can be useful in further assessing the presence of soft-tissue injury or cord compression. MRI evaluation typically includes axial, coronal, and sagittal sections of T1- and T2-weighted images (Figure 3).

Short tau inversion recovery (STIR) sequences can complement the sagittal sections. Hyperintense signal within the parenchyma of the cervical spinal cord is typically demonstrated on T2-weighted MRI and STIR sequences in patients with CCS. This finding is consistent with edema without any evidence of parenchymal hemorrhage.[19,20] T2-weighted imaging and/or STIR sequences are critical in evaluating injury to the anterior and posterior soft tissues, such as the intervertebral disk and posterior ligamentous tension band. T2-weighted MRI may also reveal prevertebral hyperintensity, which has been shown to be a predictor of spinal instability.[20] Underlying cervical spondylosis and stenosis can be assessed with plain and advanced imaging studies.

In evaluating a patient with SCI, it is essential to determine the extent of neurologic injury (ie, incomplete versus complete). Incomplete injuries have a greater chance for neurologic recovery, whereas motor recovery is achieved in only 3% of patients with

Figure 4



American Spinal Injury Association Standard Neurologic Classification of Spinal Cord Injury Worksheet.[1] (Reproduced with permission from the American Spinal Injury Association.)

Table 1

Motor Grading Scale for Suspected Spinal Cord Injury[1]

Grade	Description
0	Total paralysis
1	Palpable or visual contraction
2	Active movement, full range of motion, gravity eliminated
3	Active movement, full range of motion, against gravity
4	Active movement, full range of motion, against some resistance
5	Active movement, full range of motion, provides normal resistance

complete injury during the first 24 hours and never after 24 to 48 hours.[10,24] Complete injuries originally were defined as the absence of motor and sensory function more than three levels below the zone of injury. Incomplete injuries, in contradistinction, involve some preservation of motor or sensory function below the level of injury. The ASIA has more recently redefined complete SCI as the absence of sensory and motor functions in the lowest sacral segments (ie, S4-S5).[1] Thus, to diagnosis SCI as complete, the physician must test touch and pinprick sensation in the perianal area as well as voluntary contracture of the external anal sphincter.

Sacral sparing is an important indicator of incomplete SCI because it signifies at least partial continuity of the long white-matter tracts (ie, corticospinal and spinothalamic) from the conus medullaris to the cerebral cortex. At the time of initial evaluation of a patient who has sustained SCI, sacral sparing may be the only neurologic function present to differentiate incomplete from complete SCI. Evaluation of sacral sparing consists of perianal sensation, rectal tone, and activity of the great toe flexor.

Spinal shock can occur after severe SCI. This is defined as a state of complete areflexia and usually resolves within 24 hours of the time of injury.[10] The completeness of the neurologic injury cannot be determined until the spinal shock has resolved. The return of the bulbocavernosus reflex heralds the end of spinal shock. The clinical test assesses the integrity of the intact S3-S4 arc and is performed by squeezing the glans penis, placing pressure on the clitoris, or tugging on a Foley catheter. An intact reflex will result in contraction of the anal sphincter.

An accurate system of clinical neurologic assessment and recording influences treatment decisions, allows for reliable serial monitoring, and provides prognostic information. The ASIA worksheet details the physical examination (Figure 4). Each of the 28 dermatomes can be assessed bilaterally for sensory function to pinprick and light touch. Normal sensation is grade 2, altered is grade 1, and absent is 0. Ten key muscle groups, five in the upper extremities and five in the lower extremities, are tested bilaterally and graded on a standard scale of 0 to 5 points (Table 1). The motor scores are summed to obtain the ASIA motor score (maximum, 100). The ASIA has classified the level of impairment from complete SCI to varying levels of incomplete SCI (Table 2). The ASIA defines the level of injury as the most caudal level that has intact motor and sensory function on both sides of the body.

Management

Nonsurgical

In most cases of CCS, the patient experiences considerable neurologic

and motor recovery without surgical intervention.[5,8,12,25-28] The work conducted by Schneider et al[5] in 1954 suggested that the natural history of CCS led to spontaneous recovery and that surgical management was unnecessary and possibly harmful. Optimal medical management, early immobilization, and possibly intravenous steroids have improved the overall prognosis of CCS.[5,25-28]

Proper medical management of the person with CCS requires that the patient be placed in intensive care during the initial period after injury. Central venous and indwelling arterial catheters can be used for close monitoring of hemodynamics and responses to therapy. A Swan-Ganz catheter may be necessary. Maintaining adequate blood pressure (mean arterial pressure >85 mm Hg) by volume resuscitation supplemented by vasopressors, if needed, has been shown to improve neurologic outcome.[25] Blood pressure augmentation presumably maximizes spinal cord perfusion and limits secondary injury.

Although controversial, intravenously administered methylprednisolone is the most commonly used pharmacologic treatment for complete and incomplete SCI. The National Acute Spinal Cord Injury Study II and III trials established the standard dosing of a 30 mg/kg bolus followed by 5.4 mg/kg/hr.[26,27] The infusion is continued for 24 hours when started within 3 hours of the time of injury or for 48 hours when started between 3 and 8 hours from the time of injury. No proven benefit of steroid administration has been shown when the patient presents >8 hours from the time of injury.[26,27] However, a recent and extensive literature review, which was formulated into clinical practice guidelines on the early acute management of adults with SCI, showed that there currently is no evidence for the use of any neuroprotective agent, including steroids.[29] Administration of steroids may adversely affect patient outcome, especially in patients with penetrating injuries.

Any patient with suspected CCS should be promptly immobilized with a hard cervical orthosis (eg, Philadelphia collar) to prevent further motion injury. The hard collar is used for at least 6 weeks or until neck pain has resolved and associated neurologic improvement is noted. Patients with no evidence of axial skeletal instability after complete radiographic evaluation are kept in the hard cervical orthosis and are mobilized early after medical stabilization. Patients with unstable fractures or dislocations can be treated surgically or with application of cervical tongs and/or halo rings in the emergency setting to facilitate skeletal traction and early closed reduction.[12,30] Traction provides the most urgent form of spinal cord decompression; early closed reduction is associated with neurologic improvement.[30,31]

Early mobilization and rehabilitation with physical therapy and occupational therapy are essential once the patient is medically stable. Retraining hand function and gait are the main goals. Many patients benefit from initial intensive inpatient rehabilitation and, after certain milestones are met, from outpatient therapy.

Table 2

American Spinal Injury Association Impairment Scale for Spinal Cord Injury[1]

Grade	Description
A	Complete: no motor or sensory function is preserved in the sacral segments (S4-S5)
B	Incomplete: sensory function is preserved below the level of injury, but no motor function is preserved
C	Incomplete: motor function is preserved below the level of injury, but more than half the affected muscles have a grade ≤3
D	Incomplete: motor function is preserved below the level of injury and at least half the affected muscles have a grade ≥3
E	Normal: motor and sensory function are normal

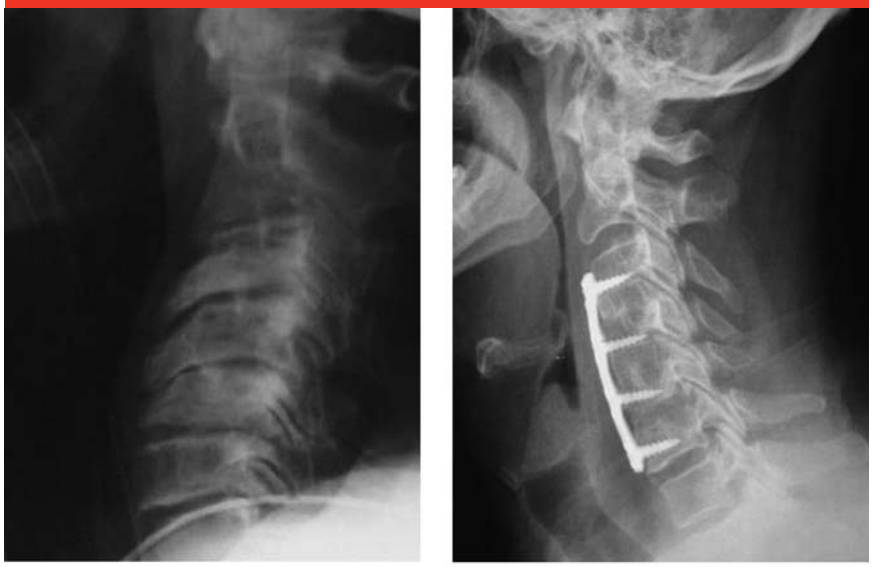
Surgical

Surgery is an adjunct to medical management in the patient with CCS; all of the aforementioned principles of medical management should be included in the treatment regimen regardless whether surgical intervention is indicated. Older studies suggested that surgical management of CCS might be detrimental and ineffective. Schneider et al[5] and Morgan et al[32] showed that decompression laminectomy did not improve patient neurologic status. One patient woke up quadriplegic after surgical decompression.[5] However, nonsurgical management of CCS has not been successful in certain patients, and more recent research has suggested that surgical intervention may be beneficial in certain subsets of persons with CCS.[13,17,20,30,32-35]

Indications

Spinal instability is the only absolute indication for surgical intervention. Spinal instability has been defined as angular displacement >11° compared with an adjacent vertebra or vertebral body translation >3.5 mm.[36] Instability can worsen secondary injury to the spinal cord by acting as a dynamic factor, further damaging the cord.

Figure 5



A B

Preoperative **(A)** and postoperative **(B)** lateral plain radiographs of the same patient as in Figure 3. The patient underwent C3-C6 anterior decompression and fusion with instrumentation because of early neurologic deterioration.

Assessment of stability can be difficult, and integrity of the discoligamentous complex is the key factor in determining the stability of the spinal motion segment.[37] Surgical intervention for CCS without spinal instability is controversial. No evidence currently supports decompression in a patient who demonstrates neurologic improvement. Patients with persistent cord compression, failure of motor recovery, or prolonged neurologic plateau or deterioration, however, may benefit from surgical intervention.[17,19,22,32,34,35] Cord compression can occur as a result of a herniated disk, an epidural hematoma, or bone fragments invading the canal. Early removal of the offending spinal cord compression may prevent the progression of chronic myelopathic changes and may lead to improved recovery and overall function.[17,19,32] Underlying spinal stenosis and spondylosis are further relative indications for surgical management.

Timing

The timing of surgical intervention in persons with CCS is controversial, with two exceptions: (1) overt spinal instability with acute dislocation in which early reduction and fixation should be performed and (2) in rare cases of progressive neurologic deficit. Recent studies have attempted to elucidate the optimal surgical protocol for CCS. With traumatic CCS, early surgical intervention (≤24 hours of injury) performed on patients with pathologic abnormality (ie, fracture-dislocation, acute herniated disk) confirmed with radiography or MRI was associated with greater motor and neurologic recovery than was late surgical intervention (>24 hours after injury).[30,34] La Rosa et al[35] conducted a systematic analysis of the literature and concluded that early surgical intervention (≤24 hours of injury) was associated with better neurologic outcomes than either delayed surgical intervention or nonsurgical management.

Although no guidelines currently are available for the timing of decompression in cases of acute CCS, studies suggest that early decompression is feasible and may result in improved outcomes, especially in the patient with progressive neurologic deterioration.[30,33-35] However, there is no evidence to support when, or even if, surgery should be performed on a patient who shows neurologic improvement.

Procedures

Surgical options are dictated by the pathologic abnormality. Imaging techniques such as CT and MRI allow the surgeon to identify the site of compression. The extent of surgery is individualized for each patient, and multilevel stenosis is a typical presentation (Figure 5). An anterior, a posterior, or a combined approach is used to relieve pressure on the cord.

Clinical Outcomes

Nonsurgical

Currently, no prospective, randomized studies have been published comparing nonsurgical with surgical treatment of CCS. However, several studies have reported good results with nonsurgical management.[4,5,9,10,28] Recovery is gradual and often incomplete and is related to the severity of the injury. Pain typically is not a major sequela of CCS. The progression of neurologic and motor recovery usually begins in the lower extremities, continues with improved bladder and bowel control, and ends with upper extremity control. Restoration of hand function is variable; in some patients, hand function does not recover. Hand function impairment is the most common long-term disability associated with CCS.[4,9] Although most patients achieve major neurologic improvements, some face considerable long-term functional impairment.

Good prognostic factors in cases of CCS include young age, preinjury employment, level of education, absence of spinal cord signal abnormality shown by MRI, higher initial ASIA motor score, absence of spasticity, early motor recovery, and good hand function.[2,4,5,8,9,12,17,28,38] Spinal column instability, degree of canal stenosis, persistent spasticity, and medical comorbidities all correlate with poorer neurologic recovery.[2,20,28,39]

In their original reports in 1954 and 1958, Schneider and colleagues[5,12] noted that of 17 patients treated medically, 2 died without improvement, 14 achieved neurologic improvement but still had residual deficits, and 1 regained complete function. In 1971, Bosch et al[2] presented one of the first studies that included a long-term follow-up period (4 months to 26 years) of patients with CCS who were treated nonsurgically. They noted that at least some return of neurologic function occurred in 75% of the 42 patients. Independent walking improved from 19% immediately after injury to 57% after rehabilitation. Likewise, bladder control improved from 17% to 53%. Importantly, the authors noted that only 43% of patients regained functional use of their hands and that 24% of patients who initially experienced neurologic improvement reported a plateau, followed by deterioration in neurologic and functional recovery. This clinical course suggested a chronic form of CCS marked by spasticity and pyramidal tract involvement.

In 2000, Newey et al[38] presented long-term (mean, 8.6 years), retrospective outcomes of 32 patients with CCS who were treated nonsurgically and reported on the differences in recovery related to patient age. All six patients aged <50 years could walk independently and had bladder continence. In patients aged 50 to 70 years, 77% could ambulate independently, and 69% had bladder control. Of the three surviving patients aged >70 years, only one could ambulate independently, and none had bladder control.

Ishida and Tominaga[8] reported the first prospective study of patients with CCS in 2002. Their study was limited to patients with weakness in the upper extremities only. At 2-year follow-up of the 22 patients, all of whom were treated nonsurgically, the authors observed that none had any fractures or dislocations, 77% had achieved full motor recovery, 23% had mild dysfunction or weakness of the hands, and none had severe dysfunction. Motor and sensory recovery occurred rapidly during the initial 3 weeks and, in most patients, reached a plateau within approximately 6 weeks. The absence of abnormal MRI signal intensity was the best predictor of recovery at final follow-up, suggesting less severe injury in cases with normal MRI findings. Early neurologic improvement and younger age were also found to be significant contributors to improved outcome. Of note, neurologic improvement during the first 6 weeks was a stronger predictor of final neurologic function than was admitting neurologic status. These results are promising and show excellent prognosis for nonsurgical treatment of the less severe form of CCS and for persons with symptoms limited to the upper extremities. However, caution is required when attempting to extrapolate these results to more severe forms of CCS.

In 2005, Dvorak et al[28] reported a rigorous study of patients with CCS. In contrast to the study conducted by Ishida and Tominaga,[8] Dvorak et al[28] did not exclude the more severe forms of CCS. Their prospective analysis had a minimum 2-year follow-up (average, almost 6 years), during which all patients underwent formal evaluation and calculation of ASIA motor scores within 72 hours of injury and at follow-up visits. The authors found a marked increase in ASIA motor scores from a mean score of 58.7 at the time of injury to a mean score of 92.3 at last follow-up. The best predictor of final ASIA motor score was the initial score at the time of injury. Level of formal education was another noteworthy prognostic factor, with patients who had higher levels of education achieving greater recovery. At last follow-up, 81% of patients reported bowel and bladder continence, and 86% were capable of independent ambulation. However, 59% had a large degree of spasticity, and 34% expressed dissatisfaction with their symptoms.

Surgical

Patients with persistent cord compression, failure of motor recovery, or prolonged neurologic plateau or deterioration may benefit from surgical intervention.[17,19,22,32,34,35,38] However, few data directly compare surgical and nonsurgical management.

Bose et al[40] retrospectively compared motor function recovery in 28 patients with CCS who were treated either surgically or nonsurgically. A greater degree of recovery was noted in the surgical group. Surgery was performed on patients with spinal instability and on those who had failed to improve progressively after an initial period of improvement and who had evidence of persistent compression.

Chen et al[17] conducted an initial retrospective study and found two groups that benefited from surgical intervention: younger patients regardless of radiographic abnormalities and older patients with clinically correlated encroaching cord lesions. Chen et al[13] subsequently conducted a prospective study of 37 patients with preexisting cervical spondylosis and in-

complete cord injury who were operated on 2 to 14 days after injury. The surgical group experienced more rapid neurologic recovery than did the nonsurgical group. Recovery was significantly slower for the nonsurgical group ($P = 0.005$), and the hospital stay was longer. However, by 2 years, no significant difference was shown between the two cohorts ($P = 0.06$). Patients with cervical stenosis affecting more than three vertebral levels experienced poorer outcomes whether treated surgically or nonsurgically.

Most studies that describe surgical treatment are retrospective analyses that are subject to selection biases and confounding variables. No prospective randomized studies have been performed comparing surgical with nonsurgical management. Thus, the true benefit of surgical treatment is unclear. Well-designed prospective studies to examine the value and timing of surgical intervention are needed.

Summary

CCS is the most common incomplete SCI. With the increasing age of the population, physicians will be encountering more patients with CCS. Thus, it is essential to understand the anatomy, pathophysiology, and treatment of CCS. Although most patients with CCS can expect gradual, albeit often incomplete, recovery with nonsurgical treatment, recent studies have shown potential benefits of early surgery. All CCS patients should receive optimal medical management and early immobilization. In our practice, the primary surgical indication is fracture with associated central cord injury. The fractures typically present as extension-distraction injury. Early surgical treatment is recommended in the absence of spinal instability in the patient with neurologic deterioration. Further research is needed to determine the best treatment algorithm and the timing of surgical intervention, if chosen, for any nonprogressive neurologic deficit that remains static.

References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 26 and 27 are level I studies. References 8, 13, and 33 are level II studies. Level III studies include references 9, 10, 17, 25, 28, 30, 34, 35, and 40. Level IV studies include references 2, 4, 5, 11, 12, 14-16, 22-24, 31, 32, and 37-39. Reference 29 is a level V study.

Citation numbers printed in **bold type** indicate references published within the past 5 years.

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