Hyperextension cervical spine injuries and traumatic central cord syndrome

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Traumatic central cord syndrome (TCCS), regardless of its biomechanics, is the most frequently encountered incomplete spinal cord injury. Patients with TCCS present with disproportionate weakness of the upper extremities, and variable sensory loss and bladder dysfunction. Fractures and/or subluxations, forced hyperextension, and herniated nucleus pulposus are the main pathogenetic mechanisms of TCCS. Nearly 50% of patients with TCCS suffer from congenital or degenerative spinal stenosis and sustained their injuries during hyperextension as originally described by Schneider in 1954. Immunohistochemical and imaging studies indicate mild to moderate insult to axons and their ensheathing myelin in the lateral funiculi culminating in cytoskeletal injury and impaired conduction. More than one-half of these patients enjoy spontaneous recovery of motor weakness; however, as time goes on, lack of manual dexterity, neuropathic pain, spasticity, bladder dysfunction, and imbalance of gait render their activities of daily living nearly impossible. Based on the current level of evidence, there is no clear indication of the timing of decompression for relief of sustained spinal cord compression in hyperextension injuries. Future research, taking advantage of validated digital imaging data such as maximum canal compromise, maximum spinal cord compression, and lesion length on the CT and MR images, as well as more sensitive measures of bladder and hand function, spasticity, and neuropathic pain may help tailor surgery for a specific group of these patients. (DOI: 10.3171/FOC.2008.25.11.E9)

KEY WORDS • central cord syndrome • cervical spine • spinal cord • spinal cord injury • trauma

FIRST reported by Thorburn⁷¹ in 1887, the syndrome of TCCS was defined and popularized by Schneider in 1954.^{56,58–60} Taylor,⁶⁸ Taylor and Blackwood,⁶⁹ and Schneider and colleagues^{56,58–60} related the syndrome to hyperextension of the cervical spine without concomitant fracture subluxations. Studies by Parke⁵¹ indicated a 2- to 3-mm canal compromise by hyperextension of the cervical spine due to overlapping of the laminae and the buckling of the ligamentum flavum. The share of TCCS among all the clinical syndromes following traumatic SCI is nearly 44%.⁴¹ Approximately 35–58%^{2,11,18,26,32,66,79} of patients with TCCS are those with cervical spinal stenosis who injured their spinal cord without skeletal injury during hyperextension. Although these patients clearly experience continuous discoligamentous and osseous compression, the general trend since 1954 has been reluctance to undertake aggressive treatment and hasty decompression of the spinal cord in an urgent fashion. Lack of fracture or subluxations on insensitive imaging

studies, spontaneous functional recovery, comorbidities, and risk of intraoperative worsening of neurological condition have been some of the reasons preventing surgeons from relieving spinal cord compression as soon as possible.^{2,11,18,26,32,66} This view is changing rapidly. Yamazaki et al.⁷⁹ demonstrated a direct relationship between outcome and midsagittal diameter of the spinal canal. Validated clinical and imaging studies including the Subaxial Injury Classification scoring system,⁷⁶ MCC,³¹ MSCC,³¹ and LL³¹ on MR imaging could offer powerful digital data to become more analytical, rather than descriptive, in pre- and postoperative definition of variables defining best ways to manage patients with TCCS due to hyperextension injuries.^{22,25,29,31,43,55,62,76}

Traumatic Central Cord Syndrome

Definition

Traumatic cervical central cord syndrome is a partial SCI with disproportionate motor loss in the distal upper extremities and significant involvement of bladder function with variable degrees of sensory impairment below the level of skeletal injury.^{34,56,58–60}

Abbreviations used in this paper: ASIA = American Spinal Injury Association; LL = lesion length; MCC = maximum canal compromise; MSCC = maximum spinal cord compression; SCI = spinal cord injury; TCCS = traumatic central cord syndrome.

FIG. 1. Neuroimaging studies obtained in a 24-year-old woman with cruciate paralysis following a motor vehicle accident. A: Midsagittal reformatted CT of the cervical spine demonstrating a Type III odontoid fracture. B: Reformatted paramedian CT scan obtained immediately after surgery demonstrating surgical fusion of the C-1 lateral mass and C-2 pars interarticularis. C: Midsagittal T2-weighted image obtained 5 weeks postinjury indicating evidence of myelomalacia in the central part of spinal cord subadjacent to the fracture. See text for more details.

Correlative Neuroanatomy, Pathogenesis, and Pathology

In primates, including man, the descending motor pathways of the corticospinal tracts pass through the internal capsule and midbrain keeping their discrete somatotopic organization. Beyond the midbrain, however, the somatotopic organization of the corticospinal tract is not discrete, leading to confusion as to the exact anatomical substrate for cruciate paralysis and TCCS.^{5,20,49,50}

For years, the presumed explanation of cruciate paralysis by Wallenberg⁷⁷ and Bell^{7,8} seemed quite logical. According to their suggestions, based on their clinical studies, corticospinal fibers serving the upper extremities were anatomically segregated in the region of the pyramidal decussation, with the upper extremity fibers being rostral and near the midline and the lower extremity fibers caudal and lateral. It was presumed, although never anatomically proven, that focal injury to the upper extremity corticospinal tract fibers near the cervicomedullary junction could produce weakness of the upper extremities, hence “cruciate paralysis.” The images in Fig. 1 were obtained in a 24-year-old woman with a Type III odontoid fracture and classic cruciate paralysis. At admission her ASIA motor score in the upper extremities was 2/50 and in the lower extremities 13/50. Eight months later, her ASIA motor score in the upper extremities was 25/50 and in the lower extremities 50/50. She was hyper-reflexic, had no difficulty with bladder function, and was able to walk independently.

Foerster³⁰ and Schneider et al.^{56–58,61} presumed a similar analogy in the pathogenesis of TCCS. The assumption was that corticospinal tract fibers subserving the upper extremities were layered more centrally and therefore were involved by a hematomyelic cavity producing weakness of the upper extremities while peripherally located fibers, innervating the muscles of the lower extremities, remained intact (Fig. 2).¹⁶ The MR image in Fig. 3 was obtained in an 85-year-old patient who had TCCS following a hyper-extension injury. Signal change is evident at the level of C3–4 and distractive extension Stage 1 at C6–7.

Several recent lines of evidence indicate that the assumptions of Wallenberg,⁷⁷ Bell,^{7,8} Foerster,³⁰ and Schneider and colleagues^{56–58,61} need to be modified. Tracing studies of Pappas et al.⁴⁹ and Marchi degeneration studies of Coxe and Landau²⁰ and Barnard and Woolsey⁵ in monkeys indicate no somatotopic organization of the corticospinal tracts at the level of pyramids or cervical spinal cord (Fig. 4). Studies of Nathan and colleagues^{44–46} in human patients tend to confirm the findings of previous investigators.

FIG. 2. Modified schematic transverse section of the cervical spinal cord indicating ascending sensory and descending motor pathways. Hatched left corticospinal tract indicates the presumed concentric layering of the corticospinal tract as proposed by Foerster and Schneider to explain TCCS. Modified from Jones: **Netter's Neurology**. Used with permission of Elsevier, Inc. All rights reserved.

Hyperextension cervical spine injuries

FIG. 3. Midsagittal reformatted T2-weighted MR image obtained in an 85-year-old man after a fall and clinical presentation of TCCS. The patient's ASIA motor score at admission was 22. The image demonstrates spinal stenosis, signal change at C3–4, and distractive extension injury at C6–7.

Correlating necropsy studies with MR imaging findings by Jimenez et al.,³⁵ Martin et al.,⁴⁰ and Quencer et al.⁵³ proved that in the majority of patients with central cord syndrome, there is no evidence of hematomyelia or significant injury to the central gray matter. Axonal disruption and swelling is widespread in the white matter of the lateral funiculi and to a lesser extent the posterior columns.

Recent experimental studies have indicated that complete unilateral or bilateral transection of the corticospinal tracts at the level of the pyramids or cerebral peduncles renders monkeys only partially paralyzed with hand function more severely affected. As in patients with TCCS, recovery of function in these monkeys starts from the lower extremities, then proceeds to the proximal upper extremities and at last the fingers.^{14,15,37,38,63,73}

An alternative hypothesis proposed by Levi et al.³⁹ and Collignon et al.¹⁹ is that the 2 syndromes of cruciate paralysis and TCCS may result from pathological entities affecting the corticospinal tracts anywhere from the pyramids to the cervical enlargement. It is suggested that

the corticospinal tracts primarily subserve fine motor movements to the distal musculature, especially the upper limbs. Preservation of leg movement is mediated by other descending motor pathways important for locomotion.

Lesions in patients presenting with TCCS seem to comprise 3 main categories: 1) cervical spondylosis associated with segmental spinal stenosis or interspace disc/osteophyte complex; 2) fracture subluxations; and 3) sequestered disc without evidence of spinal stenosis.^{2,11,13,18,26,32,42,54,66,72,79} The proportion of each of these pathological processes in each case series is different, reflecting the nature of the studies, which are mostly uncontrolled and retrospective. In the study of Aito et al.,² 44 of 82 patients with TCCS had hyperextension injuries and the rest had fracture/subluxations or disc injury or SCI without evidence of trauma. Nineteen of 28 patients in the study of Bose et al.¹¹ had hyperextension injuries and the condition of 14 of 28 was stable according to the principals of White et al.⁷⁸ In Chen and colleagues' study of surgical treatment of TCCS, 16 of 28 patients had fracture subluxations or disc herniation and 12 had disc/osteophyte complex. In the series of TCCS cases reported by Dvorak et al.,²⁶ 43 of 70 patients with adequate follow-ups had fracture/subluxations, 25 had spinal stenosis, and 2 had ruptured discs. In 2002 Guest et al.³² evaluated early versus late surgery in 50 patients with TCCS who presented to Barrow Neurological Institute. Their retrospective study included 24 patients (48%) with spinal stenosis, 10 (20%) with fracture subluxations, and 16 (32%) with herniated discs. Between May 2007 and June 1, 2008, 42 patients with TCCS admitted to the Shock Trauma Center in Baltimore were screened for eligibility for a randomized prospective trial evaluating early (first 5 days) versus late (6 ± 1 weeks) spinal cord decompression (unpublished data). Twenty (48%) of 42 patients had either spinal stenosis or disc/osteophyte complex, 13 (33%) had fracture subluxations, 5 (12%) sequestered disc, and 3 (7%) SCI without radiological abnormality. Twelve of 20 patients with spinal stenosis also had concomitant distractive extension injury Stage 1 (Fig. 3). The C3–4 level, either by itself or in conjunction with other levels, was the most frequent level causing spinal cord compression (noted in 8 patients). It was not unusual that the site of compression was associated with a remote site of distractive extension injury.

The Syndrome

Demographic data indicate that middle-aged men are more susceptible to injuries producing TCCS. In several recent series the proportion of men ranged from 56.2 to 88%.^{2,11,18,26,32,74,79} This tendency is even more pronounced in reports of case series describing hyperextension injuries. Aito et al.² reported on a series of 44 cases managed conservatively; the patients had TCCS without radiological evidence of fracture dislocations and their mean age was 56. The mean age of patients in the case series of Dvorak et al.,²⁶ Guest et al.,³² Yamazaki et al.⁷⁹ and Uribe et al.⁷⁴ were 51, 62, 56.2, and 56 years, respectively. Very few investigators have systematically studied their patients at the time of admission using the ASIA motor function assessment system. It is not unusual for patients to experience quadriplegia immediately after an accident

FIG. 4. Photographs of sections from the brain of a squirrel monkey following injection of 0.05 μ l 2% wheat germ agglutinin conjugated with horseradish peroxidase in the foot region of the left and hand region of the right hemisphere precentral cortex. The sections were reacted with tetramethylbenzidine and then stained with neutral red, mounted, and examined with polarized-light microscopy. A: Transverse section through the medulla at the level of the inferior olive. The evenness in the fiber labeling of the pyramids is easily seen. Fine fibers leaving the pyramids (p) on the left side correspond to the forelimb injection and cross the brainstem to terminate the lateral reticular formation (lrn). Bar = 1 mm. B: Transverse section through the rostral part of the pyramidal decussation. Heavy terminal labeling is visible in the gracile nucleus (gr) on the left while more scattered labeling is seen in and around the cuneate nucleus (cu) on the right. The pyramidal tracts (px) are decussating symmetrically. Bar = 1 mm. C: A slightly more caudal section than shown in B, but still in the pyramidal decussation. The gracile nucleus is still heavily labeled; the labeling in the cuneate nucleus has become more focused and has shifted medially. Decussating pyramidal fibers have moved dorsally and laterally but still occupy symmetrical positions in their respective halves of the brainstem. Bar = 1 mm. D: Transverse section immediately caudal to the pyramidal decussation. The labeled corticospinal (cs) tracts form roughly circular patterns that occupy symmetrical areas on both sides. The gracile labeling is largely absent, while the cuneate labeling is dense and sharply defined. A second area of corticospinal termination (curved arrow) is visible on the medial border of the right corticospinal tract. This area lies in Kuypers' internuncial region. Some of the corticospinal terminals are present in the cuneate nucleus (long arrow) and the rest are in the central gray area (short arrow). Bar = 1 mm. Modified from Pappas et al.: *J Neurosurg* 75:935–940, 1991, with permission.

and to recover gradually so that by the time they arrive at the emergency department they are complaining of weakness and an extreme burning sensation of the arms and hands, which is very uncomfortable upon touch. The admission ASIA motor score for all the 14 conservatively treated patients in the case series reported by Bose et al.¹¹ was 50.6 and for those managed surgically it was 58.5. In the case series reported by Guest et al.,³² the ASIA motor scores for patients who did not have fractures and who were treated surgically less than 24 hours and more than 24 hours after injury were 56.8 and 61.7, respectively. Among the 42 previously mentioned patients with TCCS admitted to our center and screened for enrollment in the randomized trial of the timing of surgery, the mean ASIA motor score was 77.

Imaging Studies

Computed tomography and MR imaging studies of the cervical spine and, when indicated, dynamic studies will essentially rule out the possibility of skeletal damage, discoligamentous injuries, and hidden fractures.^{1,3,4,6,9,10,21,23,24,33,36,64,67,70,75} New technology even en-

ables us to measure the degree of canal compromise and the extent of spinal cord compression.^{29,31,43,55} (Figs. 5, 6, and 7).

Management

Class II and III evidence support early surgical intervention in TCCS due to herniated nucleus pulposus and unstable skeletal injuries. The objectives behind this approach are spinal cord decompression, alignment and internal fixation, and thus interruption and/or prevention of further secondary insults. This concept is not universally accepted in stable TCCS due to forced hyperextension superimposed on spinal stenosis. Spontaneous recovery of function, comorbidities, lack of proved instability, and a less aggressive approach recommended by surgeons on the basis of the experience of Schneider and colleagues are only a few reasons.^{2,11–13,17,25,26,32–34,42,47,54,56–58,60,61,65,66,74,79} In 2007, Aito et al.² compared long-term motor and functional recovery, including ASIA impairment scale, Walking Index for Spinal Cord Injury, and Functional Independent Scale (FIM) scores of 38 patients treated surgically and 44 patients who were treated conservatively. The

FIG. 5. Measurement of MCC following distractive flexion injury. A = midsagittal diameter 2 segments above the site of injury; I = midsagittal diameter at the level of injury; B = midsagittal diameter 2 segments below the site of injury. $MCC (\%) = [1 - I/(A+B)/2] \times 100$. (See Furlan et al.)

conservative-treatment group suffered hyperextension injuries and the surgical-treatment group skeletal and discoligamentous injuries. The authors noted no statistically significant difference in functional outcome between the 2 groups. In 1984, Bose et al.¹¹ from Thomas Jefferson University compared 2 groups of patients with TCCS—14 patients whose condition was unstable and who were treated surgically, and 14 patients whose condition was stable and who were treated conservatively; they noted better motor scores in the surgically treated group. The authors concluded that operative intervention was safe and when chosen properly could result in better motor recovery. In 2002, Guest et al.³² reviewed their experience with TCCS at Barrow Neurological Institute. In 2 groups of surgically treated patients, early surgery (within 24 hours of injury) was compared with late surgery (> 24 hours after injury). The conclusion was that early surgery

FIG. 6. Measurement of MSCC following TCCS. a = spinal cord diameter on T2-weighted images at 2 cord segments above the injury; i = spinal cord diameter at the level of injury; b = spinal cord diameter 2 segment below the level of injury. $MSCC (\%) = [1 - i/(a+b)/2] \times 100$. (For more information, see Furlan et al.)

in patients with skeletal injuries or disc herniation resulted in better motor recovery. The timing of surgery did not affect motor recovery in patients suffering from spinal stenosis. Patients older than 60 years and those with bladder dysfunction fared worse than younger patients without bladder dysfunction at the time of admission. Younger patients with TCCS seem to do better than older patients.^{32,52} In 2005, Yamazaki et al.⁷⁹ analyzed their experience with 47 patients with spinal stenosis and found a significant relationship between the sagittal diameter of the spine and functional outcome. Evidence is accumulating that earlier decompression of a compressed spinal cord in traumatic spinal cord injuries is safe and may in fact promote recovery of function.^{28,48} Preliminary results of a prospective multicenter trial to evaluate the role and timing of decompression in patients with cervical SCI reported by Fehlings et al.²⁷ indicated better functional outcome when the spinal cord was decompressed within 24 hours of injury as compared with decompression after 24 hours of trauma. Although patients with TCCS were included in the study, the exact influence of the timing of decompression on hyperextension injuries remains to be elucidated.

What approach should be taken for spinal cord decompression in patients with hyperextension injuries is not well established.^{11,13,25,28,32,33,48,74,79} In a recent review of the current literature, Dvorak et al.²⁵ were not able to establish a standard algorithm for management of subaxial cervical spine injuries. Based on the Subaxial Injury Classification⁷⁶ classification system, the options were as follows. In patients with normal-looking sagittal balance of the cervical spine, laminectomy with or without internal

FIG. 7. Measurement of LL on T2-weighted MR images in mm. Type 1 injury is defined as any injury with bleeding inside the spinal cord, Type 2 is defined high signal without bleeding and more than one skeletal segment in length, and Type 3 is defined as high signal without evidence of bleeding confined to 1 skeletal segment. (For details, see Furlan et al.³¹ and Schaefer et al., 1992.)

fixation is an option, but in patients with a straight cervical spine or kyphotic deformity, surgical decompression should be from multilevel discectomy or corpectomy followed by internal fixation.

Conclusions

Traumatic central cord syndrome is the most frequent syndrome encountered after an incomplete cervical SCI. Almost 50% of all cases of TCCS are due to hyperextension injuries involving old male patients and usually due to a fall. Advanced age, comorbidities, spontaneous recovery of function, and a negative outlook toward surgery make any sort of recommendation for the timing of surgery at the level of an option. Future research should be multicenter, prospective, and analytical rather than descriptive, taking advantages of digital data, such as MCC,³¹ MSCC,³¹ LL³¹ and midsagittal diameter^{31,79} to define variables best fit for surgery and prediction of treatment effect.

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Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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