Cervical cord neurapraxia: classification, pathomechanics, morbidity, and management guidelines

Joseph S. Torg, M.D., Thomas A. Corcoran, M.D., Lawrence E. Thibault, Sc.D., Helene Pavlov, M.D., Brian J. Sennett, M.D., R. John Naranja, Jr., M.D., and Steven Priano, M.D.

Departments of Orthopedic Surgery and Neurosurgery, Allegheny University for the Health Sciences, Philadelphia, Pennsylvania; and Department of Radiology, The Hospital for Special Surgery, New York, New York

One hundred ten cases of the transient neurological phenomenon, cervical cord neurapraxia (CCN), are presented. The authors established a classification system for CCN, developed a new computerized measurement technique for magnetic resonance (MR) imaging, investigated the relationship of the cervical cord to the canal, and analyzed clinical, x-ray, and MR data. One hundred nine males and one female were included in the study; the average age of the participants was 21 years (range 13-33 years). All episodes occurred during sports participation; 87% occurred while the patient was playing football. Follow-up review lasting an average of 3.3 years was available for 105 patients (95%).

Narrowing of the sagittal diameter of the cervical canal in the adult spine was confirmed to be a causative factor. Cervical cord neurapraxia was not associated with permanent neurological injury and no permanent morbidity occurred in patients who returned to contact activities. Of the patients returning to contact activities, 35 (56%) experienced a recurrent episode. The risk of recurrence was increased with smaller spinal canal/vertebral body ratio (p < 0.05), smaller disc-level canal diameter (p < 0.05), and less space available for the cord (p < 0.05). There was no correlation between either the classification of the CCN episode or the disease noted on MR imaging and x-ray films and the risk of recurrence.

The authors conclude that: 1) CCN is a transient neurological phenomenon and individuals with uncomplicated CCN may be permitted to return to their previous activity without an increased risk of permanent neurological injury; 2) congenital or degenerative narrowing of the sagittal diameter of the cervical canal is a causative factor; 3) the overall recurrence rate after return to play is 56%; and 4) the risk of recurrence is strongly and inversely correlated with sagittal canal diameter and it is useful in the prediction of future episodes of CCN (p < 0.001). These data will enable the physician to counsel individuals regarding a predicted risk of recurrence based on canal measurements.

Key Words * cervical spine * neurapraxia * transient quadriplegia * spinal stenosis

Previously we described cervical cord neurapraxia (CCN) as a distinct clinical entity and, using x-ray measurements, identified narrowing of the anteroposterior diameter of the cervical canal in the adult spine as a causative factor.[14,15,18,19] Characteristically, the clinical picture involves an athlete who experiences an acute transient neurological episode of cervical cord origin. The neurological symptoms
may involve both arms, both legs, all four extremities, or an ipsilateral arm and leg. The symptoms involve sensory changes with or without motor changes. The sensory changes include burning pain, numbness, or tingling; the motor changes consist of weakness or complete paralysis. The episodes typically last fewer than 15 minutes, although in some cases gradual resolution does not occur for up to 48 hours. There is complete return of motor function and full, pain-free cervical range of motion. The incidence of CCN has been estimated to be 7.3 per 10,000 football participants.[14] Those patients who return to contact activities are at risk for future episodes.

The spinal canal/vertebral body (SC/VB) ratio was developed and is studied to quantify the degree of developmental canal narrowing.[8,14,15] Previous reports have provided analyses of plain film radiographs but not of magnetic resonance (MR) images. Using modern computer technology, we have developed a system to digitize MR images so that we can determine the relationship of the spinal cord and the intervertebral disc to the bony cervical canal in this group of patients. The goals of the current report are to: 1) obtain a fuller understanding of CCN by analysis of clinical and MR imaging data; 2) determine whether a history of CCN implies a higher risk for permanent neurological injury; 3) determine which factors indicate a higher risk for recurrent episodes of CCN; and 4) delineate management guidelines and specifically deal with the issue of whether these patients can be permitted to return to contact activities.

**CLINICAL MATERIAL AND METHODS**

The study group was composed of 110 patients who experienced a witnessed episode of CCN and who presented to or were seen in consultation by the senior author (J.S.T.). A medical history was obtained and a physical examination was performed in all patients. There were 109 males and one female in the group with an average age of 21 years (range 13-33 years). All of the injuries occurred during sports-related activities: 96 cases (87%) during football; six cases (5%) during basketball; two cases (2%) during hockey; two cases (2%) during wrestling; and four cases (4%) during other sports. The level of participation in the sporting activity was professional in 28 cases (25%); collegiate in 49 (45%); high school in 29 (26%), and recreational in four cases (4%).

Twelve (11%) of the patients were treated surgically: nine patients (8%) underwent anterior cervical discectomy and fusion, two patients (2%) atlantoaxial fusion; and one patient (1%) posterior cervical decompression. Except for the two cases of atlantoaxial instability, there were no consistent indications or criteria for surgery. No authors of this report were involved in the surgical management of any of the patients.

**Clinical CCN Classification**

Information was gathered for each patient with regard to the type, duration, and anatomical distribution of the neurological symptoms of the CCN episode. This information formed the basis of a clinical classification of CCN. The CCN type is defined by the type of neurological deficit: 1) "plegia" for episodes with complete paralysis; 2) "paresis" for episodes with motor weakness; and 3) "paresthesia" for episodes that involve only sensory changes without any motor involvement. The CCN grade is defined by the duration of time that the neurological symptoms persist: Grade I, fewer than 15 minutes; Grade II, 15 minutes to 24 hours; and Grade III, longer than 24 hours. The CCN pattern is defined by the anatomical distribution of the neurological symptoms: "quad" for episodes involving all four extremities; "upper" for episodes involving both arms; "lower" for episodes involving both legs; and "hemi" for episodes involving an ipsilateral arm and leg.
Return to Contact Activities

Overall, 60% of the patients returned to sports participation at their previous level of competition. The patients returned to the sport or retired on the basis of many factors. In various cases, the patient, patient's family, multiple physician consultants, and medical and legal representatives of the sports organization had input into the decision. Because of the complexities of the decision to return, this group of patients essentially represents a random sample of the population as a whole. There was no statistical difference between the group that returned to play and the group that retired from sports participation with regard to age, sex, type of sport, CCN clinical grade, or radiological findings or measurements. Only those patients who returned to contact activities were considered in the analysis of recurrence of CCN.

Radiological Evaluation

Plain film radiographs, which routinely included anteroposterior, lateral, oblique, and lateral flexion-extension views, were available for review by the authors in 104 of the 110 cases. Routine interpretation of the plain film radiographs was performed by two of the authors (J.S.T. and T.A.C.) to identify the presence of fracture, cervical spine instability, degenerative disc disease, congenital vertebral fusion (Klippel-Feil syndrome), osteophytic ridging, limbus vertebra, atlantoaxial instability, and lordotic reversal. Atlantoaxial instability was considered in cases in which flexion-extension studies demonstrated an atlantodens interval of 5 mm or greater. Cervical spine instability was diagnosed according to the criteria of White and Panjabi.[20]

The SC/VB ratio was determined at the C3-7 levels.[8] This measurement provides a normalized size of the spinal canal, controlling for variations in x-ray magnification. The ratio for a particular cervical level is made by dividing the two distances measured on the lateral radiograph. The numerator is the distance from the midpoint of the posterior aspect of the vertebral body to the closest point of the corresponding spinolaminar line. The denominator is the anteroposterior width of the vertebral body at its superoinferior midpoint (Fig. 1). The smallest ratio, indicating the smallest canal segment, was used for analysis. Developmental spinal stenosis was diagnosed in those patients who had a SC/VB ratio of less than 0.8 at any level.[4,8,14,15] The ratio measurements were made on the basis of radiographs by using a caliper on a lighted table with loupe magnification to an accuracy of 0.25 mm.
Fig. 1. Schematic drawing of a midsagittal section through the cervical canal. A = vertebral body width; B = the canal diameter at the midvertebral body. The SC/VB ratio is obtained by dividing B by A (B/A) as measured on plain film radiography. C = disc-level canal diameter as measured on MR imaging; D = spinal cord diameter. The SAC is calculated by subtracting D from C (C - D).

Magnetic resonance imaging was available for review in 53 of the 110 cases. All MR images were subjected to blinded routine evaluation by an experienced musculoskeletal radiologist (H.P.) to identify the presence of degenerative disc disease, osteophytic ridging, lordotic reversals, disc protrusion, disc herniation, neuroforaminal compromise, cervical cord compression, abnormal cervical cord signal, thecal sac effacement, and stenosis.

To analyze the relationship of the spinal cord to the spinal canal, a computerized system was developed to analyze the MR images. This system consisted of a personal computer and a color scanner with a transparency adaptor. Images were digitized on the scanner and then uploaded using an imaging software package. The midsagittal T1- and T2-weighted images were digitized. Using a graphics digitizer pad with a resolution of 0.01 mm, the following measurements were made at levels C3-7: 1) to quantify spondylitic narrowing, the disc-level canal diameter was measured as the shortest distance between the intervertebral disc and the bony posterior elements; 2) the cord diameter was determined by measuring the transverse diameter of the spinal cord at the appropriate level; and 3) the space available for the cord (SAC) was calculated by subtracting the spinal cord diameter from the disc-level canal diameter (Fig. 1). The minimum disc-level canal diameter, the SAC measurements, and the average cord diameter were used for analysis in each case.

**Follow-Up Evaluation**

Follow-up evaluation was obtained by questionnaire mailing, telephone interview, or office evaluation, and was available for 105 (95%) of the 110 cases. The follow-up data were tabulated to calculate the return to contact activities or retirement from sports (Table 1). The minimum follow-up duration for any patient who returned to contact activities and did not retire was at least 12 months, including one athletic
season. The National Athletic Head and Neck Injury Registry was crossreferenced for all patients in the study to determine the occurrence of any significant injuries.

<table>
<thead>
<tr>
<th>Career Outcome</th>
<th>No. of Patients (%)</th>
<th>Follow Up (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>retired after first episode</td>
<td>42 (38)</td>
<td>49 ± 76 (1–207)</td>
</tr>
<tr>
<td>retired after recurrent episode</td>
<td>17 (15)</td>
<td>27 ± 45 (1–165)</td>
</tr>
<tr>
<td>retired after surgical complication</td>
<td>1 (1)</td>
<td>92 (92)</td>
</tr>
<tr>
<td>returned to sports activity with recurrence</td>
<td>25 (23)</td>
<td>43 ± 52 (12–228)</td>
</tr>
<tr>
<td>returned to sports activity &amp; had recurrences</td>
<td>15 (14)</td>
<td>35 ± 41 (12–148)</td>
</tr>
<tr>
<td>returned to sports activity after surgery, no recurrence†</td>
<td>5 (5)</td>
<td>15 ± 4 (12–19)</td>
</tr>
<tr>
<td>lost to follow up</td>
<td>5 (5)</td>
<td>—</td>
</tr>
<tr>
<td>totals</td>
<td>110 (100)</td>
<td>40 ± 59 (1–228)</td>
</tr>
</tbody>
</table>

* Duration of follow up is expressed as mean ± SD; range is given in parentheses. Analysis of follow up took into consideration that some patients retired from contact sports. All patients who returned to sport activities were followed for a minimum of 12 months, including one athletic season. Abbreviation: — = not applicable.
† Three of these patients had experienced recurrence before surgery was performed.

Statistical Analysis

Single and multiple regression analysis, analysis of variance, and Student's t-test were used as necessary. All statistical analyses were performed using a personal computer with commercially available software. Differences were considered significant at a probability value less than 0.05.

Sources of Supplies and Equipment

An MS-DOS (Microsoft disk operating system [Microsoft Corp., Redmond, WA])-based computer and a model IIcx color scanner (Hewlett-Packard Co., Palo Alto, CA) were used to analyze the MR images to ascertain the relationship of the spinal cord to the spinal canal. The software program used in this analysis was Adobe Photoshop, provided by Adobe Systems, Inc., Mountain View, CA. The graphics digitizer pad used was obtained from Summagraphics, Inc., Seymour, CT.

All statistical analyses were performed using a Macintosh personal computer (Apple Computer, Inc., Cupertino, CA) with Statview software (Abacus Concepts, Inc., Berkeley, CA).

RESULTS

Patient Outcome

There were no permanent or catastrophic neurological injuries related to the occurrence of CCN. One patient suffered permanent neurological symptoms as a result of a surgical complication. That patient experienced CCN with ipsilateral arm and leg weakness and exhibited neurological improvement in the hours following the episode. He underwent a multilevel posterior cervical laminectomy for cervical canal decompression despite an improving neurological examination. Postoperatively, the patient experienced a worsening and unstable neurological condition in the recovery room and was taken immediately for
reoperation. Ultimately, he was left with permanent weakness in the ipsilateral extremities. This patient's morbidity resulted directly from a complication of surgery and was not primarily due to the episode of CCN.

Pathological Findings Found on Plain Film Radiography and MR Imaging

Evaluation of the 104 plain film radiographs revealed: seven (7%) with normal findings; 89 (86%) with cervical stenosis; 52 (50%) with osteophytic ridging; 22 (21%) with loss of cervical lordosis; and 29 (28%) with degenerative disc disease. The 53 MR images included four (8%) with normal findings; 43 (81%) with disc bulge; 19 (36%) with disc protrusion; 29 (55%) with osteophytic ridging; and 25 (47%) with neuroforaminal compromise. Other pathological findings demonstrated on plain film radiographs and MR imaging are included in Table 2. There was no evidence of posttraumatic cord swelling, deformity, or syrinx.

![Table 2: Findings on plain radiographs and MR imaging in 110 patients with CCN*](image)

<table>
<thead>
<tr>
<th>Finding</th>
<th>No. of Patients WI X-Ray Findings (104 patients)</th>
<th>No. of Patients WI MR Findings (53 patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>normal</td>
<td>7 (7%)</td>
<td>4 (8%)</td>
</tr>
<tr>
<td>degenerative disc disease</td>
<td>29 (28%)</td>
<td>43 (81%)</td>
</tr>
<tr>
<td>Klippel–Feil syndrome</td>
<td>7 (7%)</td>
<td>—</td>
</tr>
<tr>
<td>compression fracture</td>
<td>6 (6%)</td>
<td>—</td>
</tr>
<tr>
<td>teardrop fracture</td>
<td>1 (1%)</td>
<td>—</td>
</tr>
<tr>
<td>osteophytic ridge</td>
<td>52 (50%)</td>
<td>29 (55%)</td>
</tr>
<tr>
<td>limbus vertebral</td>
<td>5 (5%)</td>
<td>—</td>
</tr>
<tr>
<td>stenosis (ratio &lt;0.8)</td>
<td>89 (86%)</td>
<td>—</td>
</tr>
<tr>
<td>ADI &gt;5 mm</td>
<td>2 (2%)</td>
<td>—</td>
</tr>
<tr>
<td>lordotic reversal</td>
<td>22 (21%)</td>
<td>4 (8%)</td>
</tr>
<tr>
<td>disc protrusion</td>
<td>—</td>
<td>19 (36%)</td>
</tr>
<tr>
<td>disc herniation</td>
<td>—</td>
<td>7 (13%)</td>
</tr>
<tr>
<td>neuroforaminal compromise</td>
<td>—</td>
<td>25 (47%)</td>
</tr>
<tr>
<td>spinal cord compression</td>
<td>—</td>
<td>18 (34%)</td>
</tr>
<tr>
<td>abnormal cord signal</td>
<td>—</td>
<td>4 (8%)</td>
</tr>
<tr>
<td>thecal sac effacement</td>
<td>—</td>
<td>13 (25%)</td>
</tr>
<tr>
<td>posterior stenosis</td>
<td>—</td>
<td>2 (4%)</td>
</tr>
</tbody>
</table>

*ADI = atlantodens interval; — = not applicable.

Classification of CCN

Patients were classified according to CCN type, grade, and pattern (Table 3). The incidence of CCN type was: plegia in 44 cases (40%); paresis in 28 (25%); and paresthesia in 38 cases (35%). The incidence of CCN grade was: Grade I in 81 cases (74%); Grade II in 17 (15%); and Grade III in 12 cases (11%). The incidence of CCN pattern was: quad in 88 cases (80%); upper in 17 (15%); lower in two (2%); and hemi in three cases (3%).
Plain Film Radiography and MR Imaging Measurements

The mean values (± standard deviation [SD]) for pertinent radiological indices were determined. The plain film radiograph SC/VB ratio was 0.68 ± 0.11; the MR imaging minimum disc-level canal diameter was 9.6 ± 1.8 mm; the spinal cord diameter was 8.1 ± 0.8 mm; and the minimum SAC was 1.6 ± 1.4 mm (Table 4).

<table>
<thead>
<tr>
<th>TABLE 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>RELATIONSHIP BETWEEN CCN TYPE AND GRADE IN 110 PATIENTS</strong></td>
</tr>
<tr>
<td><strong>Type</strong></td>
</tr>
<tr>
<td>plegia</td>
</tr>
<tr>
<td>paresis</td>
</tr>
<tr>
<td>paresthesia</td>
</tr>
<tr>
<td><strong>Totals</strong></td>
</tr>
</tbody>
</table>

*Grade is based on duration of neurological symptoms: Grade I, less than 15 minutes; Grade II, 15 minutes to 24 hours; and Grade III, longer than 24 hours.*

Recurrence of CCN

Sixty-three (57%) of the patients returned to contact activities after their first episode of CCN. Of this group, 35 patients (56%) experienced a second episode of CCN. The duration of follow up, based on career outcome, is as follows: 42 patients (38%; mean follow up 49 months [range 1-207 months]) retired after their first CCN episode; 17 patients (15%; mean follow up 27 months [range 1-165 months]) returned to contact activities but retired after a recurrent episode; one patient (1%; follow up 92 months) retired after suffering an unrelated permanent neurological injury; 25 patients (23%; mean follow up 43 months [range 12-228 months]) returned to contact activities and did not experience recurrence; 15 patients (14%; mean follow up 35 months [range 12-148 months]) returned to contact activities and continued to play despite experiencing recurrence; five patients (5%; mean follow up 15 months, range 12-19 months) returned to play after surgery; and five patients (5%) were lost to follow up. The 35 patients who experienced recurrence had an average of 3.1 ± 4.0 episodes (range 2-25 episodes) (Table 1).

Predictors of Recurrence

Patients returning to football had a higher recurrence rate than those returning to other sports (p < 0.05). Thirty-two (62%) of 52 football players who returned to the sport experienced a recurrent episode of CCN compared with three (27%) of 11 players who returned to other sports. All of the radiological measurements except spinal cord diameter were predictive of recurrence. Those patients who suffered
recurrence had a smaller SC/VB ratio (mean [± SD] 0.65 ± 0.1, p < 0.05) compared with those without recurrence (mean 0.72 ± 0.1). Patients who experienced recurrence had a smaller disc-level canal diameter (mean 8.7 ± 1.7 mm, p < 0.05) compared with those who had no recurrence (mean 10.1 ± 1.2 mm). Patients who suffered recurrence had less SAC (mean 1.1 ± 1.4 mm) compared with those who had no recurrence (mean 2 ± 1.2 mm, p < 0.05). There was no difference in the size of the spinal cord diameter between the groups.

The patient's age, level of sports participation, radiographic findings, MR imaging findings, clinical CCN classification, and radiological CCN classification did not have predictive value in determining which patients were at risk of recurrence (p > 0.05); that is, the presence of disc herniation, cord compression, degenerative disc disease, or any other finding was not an indicator of whether patients would suffer future episodes of CCN.

Based on the finding that narrowing of the canal is a causative factor of CCN, the recurrence and diameter data were analyzed and correlated. Graphic plots were constructed using logistic regression analysis of the percentage risk of recurrence versus the disc-level canal diameter (Fig. 2 left) and the SC/VB ratio (Fig. 2 right). The plots demonstrate both a strong and inverse correlation between the risk of recurrence and the disc-level canal diameter and SC/VB ratio (p < 0.05).

**Fig. 2.** Graphs developed using logistic regression analysis in which the risk of recurrence can be plotted as a function of the disc-level diameter measured on MR imaging (left) and the SC/VB ratio calculated on the basis of x-ray films (right). The construction of these plots is based on the result that increased risk of recurrence is inversely correlated with canal diameter. Future CCN patients can be counseled regarding their individual risk of recurrence based on the particular size of their spinal canal.

**Age-Dependent Factors**

Increasing age was correlated with a smaller SC/VB ratio and a smaller disc-level canal diameter (p < 0.05). There was no relationship between age and recurrence, MR imaging findings, or clinical CCN classification. The average age (± SD) of the seven patients with normal plain film radiographs was 16.8 ± 2.4 years compared with 20.7 ± 4.3 years for those patients with some radiographic evidence of abnormality (p > 0.05). Three of the seven patients with normal plain film radiographs returned to contact activities and none of these three experienced recurrence of CCN.
DISCUSSION

The relationship between developmental cervical canal narrowing and CCN has been clearly established.[14,15,17-19] The mechanism of the transient neurological signs and symptoms is secondary to spinal cord compression, which occurs as a result of what has been described as the "pincers" mechanism.[10] Specifically, with hyperextension of the cervical spine the posterior inferior aspect of the superior vertebral body and the anterior superior aspect of the lamina of the subjacent vertebra--and, conversely, in flexion the lamina of the superior vertebra and the posterior superior aspect of the subjacent vertebral body--approximate with a sudden decrease in the anteroposterior diameter of the canal at that cervical level, resulting in compression of the spinal cord.[2,3,10-12,21]

An explanation for the temporary neurological dysfunction related to spinal cord deformation has been obtained from a study in which a squid axon injury model is used. Torg, et al.,[16] correlated the clinical findings of reversible cord deficit with the histochemical response of isolated neural and vascular elements subjected to controlled mechanical deformation. Neurapraxia of the cervical cord occurs as a result of hyperflexion or hyperextension of the cervical spine in an individual with a developmentally narrow (stenotic) canal. The pincer mechanism--a sudden, brief compressive deformation of the cord--is thought to produce sudden aberration of nervous function below the involved level, ranging from paresthesias to transient quadriplegia. From the engineering perspective, the spinal cord is an element with a low modulus of rigidity. Because of this particular characteristic, macroscopic loads applied to the cord result in localized tension within the tissue. Various macroscopic deformations result in local elongation (tension) of the element. The first and simplest case is pure axial elongation of the spinal cord in which all elements experience stretch. With extension or flexion (bending), the tension in the cord will vary across the diameter. Highly localized loading, such as shearing from subluxation of the vertebral elements, or focal compression, such as that experienced in a weight-drop experiment, result in elongation of the elements in the direction of the long axis of the spinal cord.

An experimental model was devised to determine the effects of high-strain uniaxial tension to various degrees of stretch in concert with changes of neurophysiology of the single axon. The giant axon of the squid Loligo pealei was selected as the isolated tissue model, and a system was designed to apply uniaxial extension at high strain rates to the preparation. The system consisted of an electromagnetic actuator, displacement transducer, isometric force transducer, and membrane potential electrodes. The actuator was programed to deform the axons to various stretch ratios at specific strain rates. Recording of the membrane potential and the cytosolic free Ca++ concentrations as a function of the strain and the tensile forces developed within the axon enabled one of us (L.E.T.) to study the response of the isolated tissue to mechanical stimulation. The purpose of the experiments was to elucidate the thresholds for the tissue response to a well-controlled mechanical insult and to relate the field observations to this isolated tissue response.

These experiments demonstrated a mechanically induced and spontaneously reversible depolarization that is dependent on the rate and magnitude of the applied stimulus. A low rate of deformation produced only a small reversible depolarization. As the rate of loading was increased, the magnitude of the depolarization and the recovery time to the original resting potential increased in a nonlinear fashion. An immediate and direct effect of mechanical deformation on the calcium ion concentration within the axon was observed. Increases in cytosolic Ca++ correlated with the degree and reversibility of neuronal dysfunction.
Neurological recovery from mechanical deformation was inversely proportional to a rise in the \([\text{Ca}^{++}]_i\) concentration, which in turn is directly proportional to the amount and rate of tension applied to the axon.[16] On the basis of laboratory and clinical data, parameters characteristically associated with cord neurapraxia and transient quadriplegia (a completely reversible lesion) are developmental narrowing of the cervical spine at one or more levels. Cord deformation must occur rapidly and is attributable to a hyperflexion or hyperextension mechanism. Disruption of cell membrane permeability results in an increase in \([\text{Ca}^{++}]_i\) concentration. With stability of the cervical spine intact and cell anatomy preserved, the deleterious affects of local anoxia secondary to venous spasm does not impede recovery of axonal function and complete neurological recovery occurs.[16]

This series confirms narrowing of the sagittal diameter of the cervical canal in the adult spine and the concomitant decreased space available for the cord as the cause of CCN. We measured the SC/VB ratio from plain film radiographs to quantify developmental narrowing and the disc-level canal diameter and SAC from midsagittal MR images to quantify spondylitic narrowing (Fig. 1). As a group, the spondylitic diameter averaged 10.1 mm compared with the 12.5 mm diameter that is generally considered to be the low end of the normal range.[7] Torg, et al.,[15] documented an average ratio of greater than 0.97 in a control group compared with the present group, which had a mean SC/VB ratio of 0.68.

Cervical cord neurapraxia was associated with complete neurological recovery in 109 of the 110 cases and there was no permanent morbidity in the 63 patients who returned to contact sport activities. The one case of irreversible neurological injury was a direct complication of surgery.

Clinically, CCN is a transient neurological entity that appears in the absence of instability or structural deficiency of the cervical spine. Patients with CCN may be advised that they are not at increased risk of permanent injury with return to contact sport activities. The presence of developmental or spondylitic stenosis, regardless of the degree of canal narrowing, does not result in irreversible cord injury. The MR images obtained in three patients with significant cervical canal stenosis and cervical spine spondylosis who returned to professional football are shown in Figs. 3 and 4. It is noteworthy that 10 patients with actual cord compression caused by degenerative discs were safely returned to contact activities.

![Fig. 3. Midsagittal (left) and C4-5 axial (right) MR images obtained in a 23-year-old professional football defensive back who experienced CCN of the paresis type, Grade I, in a quad pattern. The patient did not experience recurrence despite spinal cord compression and deformity at the C4-5 disc level. His disc-level canal diameter measures 8 mm. A patient](image-url)
with this diameter would be counseled that the chance of recurrence is 65 to 70% (see Fig. 2 left).

Overall, 35 (56%) of the patients who returned to contact sport activities after a CCN episode experienced a recurrent episode. Football participation and the degree of stenosis were the only factors that were indicative of risk of recurrence. The disc-level canal diameter measurement obtained from MR imaging was the best predictor of risk of recurrence, followed by the SC/VB ratio and then the SAC measurement (p < 0.05). Although a measure of the disc-level diameter on MR imaging has the smallest confidence intervals in quantifying risk, it is only marginally better than the SC/VB ratio. Considering the technical requirements for making an accurate MR measurement, as performed with our technique, the SC/VB ratio is the method of choice in general practice. Although Herzog, et al.,[4] have analyzed the SC/VB ratio and questioned whether it is overly sensitive for the diagnosis of cervical stenosis, it is a reliable, easy, accurate, and available method to quantify the risk of CCN recurrence. Using the risk-versus-diameter plots (Fig. 2), a physician can counsel patients with CCN about their individual risk of recurrence. The appropriate diameter is measured, found on the X-axis, and the corresponding risk of recurrence is determined. For example, as seen in Fig. 2 right, a CCN patient with an SC/VB ratio of 0.5 has approximately a 75% risk for a recurrent episode of CCN.

Fig. 4. Left: Midsagittal MR image obtained in a 31-year-old professional football defensive back who experienced CCN of the paresthesia type, Grade I, in a quad pattern. The patient returned to contact sport activities, experienced one recurrent episode, and continued to play football. The MR image demonstrates osteophyte formation, no space available for the cord, and a minimum disc-level diameter of 7 mm. Our data indicate that a patient with this diameter would have an 80% chance of recurrence (see Fig. 2 left), consistent with the patient's actual history. Right: Midsagittal MR image obtained in a 32-year-old professional
football running back who experienced CCN of the paresis type, Grade III, in a quad pattern 12 years prior to this evaluation. The patient returned to football without recurrence of CCN but was evaluated for C-7 radiculopathy that resolved with nonoperative treatment. The MR image is significant for disc herniation, spinal cord compression, and a minimum disc-level diameter of 7.5 mm.

The diameters used to measure developmental and spondylitic narrowing were both inversely correlated with age (p < 0.05); that is, as a cross section, the older patients tended to have more developmental and spondylitic narrowing. There was a subset of seven patients who had no radiographic evidence of developmental or spondylitic narrowing. The occurrence of CCN in the younger group with normal radiographs is attributed to increased mobility of the cervical vertebrae due to the physiological laxity of immaturity. Although not substantiated statistically because of the small population, we hypothesize that with maturity there will be decreased laxity, protective muscle hypertrophy, and a decreased chance for recurrence. This contrasts with an older patient in whom there may be existing stenosis, advancing degeneration, and an increasing risk of recurrence.

The type of CCN neurological deficit is almost equally divided among paralysis, motor weakness, and sensory changes alone. However, the pattern is four-extremity involvement in the majority of cases (80%), and 74% of the episodes are Grade I, lasting less than 15 minutes. The involvement of the lower extremities alone or an ipsilateral arm and leg are relatively rare and occur in less than 5% of cases. Several investigators have observed an inverse correlation between increased posttraumatic myelopathy and the sagittal diameter of the cervical canal in patients suffering injuries involving fracture, dislocation, or cervical spine instability.[1,3,5-7,9,15] In our series, there was no correlation between the severity of the clinical manifestations and the degree of narrowing. The important difference between our study and previous studies that show a correlation between stenosis and permanent injury is that our patients did not experience loss of cervical spine stability at the level of spinal canal narrowing. The occurrence of spinal cord deformation is related to the presence of a narrow canal, but the amount and duration of cord deformation and, therefore, the severity of the symptoms in accordance with the squid axon model, does not correlate with the measurements from static radiological studies. Analyzing only the radiological studies disregards the biokinetics of a particular episode.

Abnormal plain film radiography and MR imaging findings are common in individuals with CCN. Only 6% of the radiographs and 8% of the MR images were interpreted as normal. Except for the atlantoaxial fusion in the two cases of atlantoaxial instability, there were no consistent clinical or radiographic criteria for surgical treatment. The patients who underwent surgery were not necessarily the patients with the most severe disease. Five patients returned to contact sport activities after anterior cervical discectomy and fusion. Although none of these five patients had recurrence of CCN, the impact of surgical intervention could not be statistically substantiated because of the small size of the group. We believe that surgical indications should be determined on an individual basis. A more recent epidemiological study has demonstrated that developmental narrowing of the cervical canal in a stable spine does not appear to predispose an individual to permanent catastrophic neurological injury and, therefore, should not preclude an athlete from participation in contact sports.[13]

CONCLUSIONS

We infer from the data that CCN is a transient clinical entity. The cause of this lesion is cord compression related to developmental or spondylitic narrowing of the sagittal diameter of the cervical
canal. Routine plain film radiography and MR imaging examinations should be performed in all cases. Patients without spinal instability can return to contact sport activities without increased risk of permanent neurological injury. The overall recurrence rate is 56% in those who return to sports and it is strongly correlated to the degree of narrowing of the cervical canal. Data provided by this series can be used to counsel and quantify the risk of recurrence for individual patients.

References


Manuscript received February 28, 1997.

Accepted in final form July 8, 1997.

Address reprint requests to: Joseph S. Torg, M.D., Department of Orthopaedic Surgery, Allegheny University for the Health Sciences, Philadelphia, Pennsylvania 19107.