Cervical spinal stenosis and sports-related cervical cord neurapraxia

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Cervical cord neurapraxia is a common sports-related injury. It is defined as a transient neurological deficit following trauma localizing to the cervical spinal cord and can be caused by hyperextension, hyperflexion, or axial load mechanisms. Symptoms usually last less than 15 minutes, but can persist up to 48 hours in adults and as long as 5 days in children. While a strong causal relationship exists between cervical spine stenosis and cervical cord neurapraxia in adult patients, this association has not been observed in children. Likewise, while repeated episodes of neurapraxia can be commonplace in adult patients, recurrences have not been reported in the pediatric population. Treatment is usually supportive, but in adults with focal cervical lesions or instability, surgery is an option. Surgery for neurapraxia in children is rarely indicated.

Key Words • neurapraxia • cervical spine • spinal cord • cervical stenosis • sports

Pathophysiology of Neurapraxia

Underlying the motor/sensory manifestations of neurapraxia is a temporary derangement of axonal permeability. Hyperextension or hyperflexion causes a mechanical injury that depolarizes the axon membrane in a reversible but sustained manner. Laboratory studies reveal that the rapid stretch experienced by the strained axon results in calcium influx, hyperpolarization, then prolonged depolarization, during which the axon is no longer excitable. In addition, anatomical strain experienced during this type of insult can result in microvascular constriction and vasoconstriction. As a result, local and regional blood flow is altered and the threat of ischemia becomes prominent. The transient nature of these physiological changes distinguished neurapraxia from irreversible neurological damage.

Cervical Spinal Stenosis

Cervical spinal stenosis is common in pediatric and adult athletes. Several methods to screen for cervical spinal stenosis in the setting of cervical cord neurapraxia have been proposed. Sagittal spinal canal diameter can be measured on lateral cervical plain radiographs and compared with standard measurements (< 14 mm in the adult...
The cervical spine is considered stenotic. The Torg ratio is calculated as the ratio of the spinal canal diameter to the vertebral body diameter at the C3–7 levels as measured on lateral plain radiographs of the cervical spine. It was developed as a measure of congenital spinal canal stenosis that theoretically minimizes the effect of variations in landmarks and radiographic technique. A Torg ratio < 0.8 is considered evidence of congenital stenosis. A criticism of this technique is that it does not take into consideration disproportionate differences in vertebral body size; football players commonly have larger vertebral bodies relative to the other spinal elements. Magnetic resonance imaging has surpassed plain radiographs and is the accepted method for evaluating spinal stenosis. Magnetic resonance imaging provides visualization of the vertebral column and intervertebral discs in relationship to the spinal cord, nerve roots, and surrounding CSF within the spinal canal. Magnetic resonance imaging demonstrates bone and discogenic encroachment on the spinal canal and spinal cord compression. The “functional reserve” of the spinal canal is indicated by the presence or absence of CSF signal surrounding the spinal cord. This can be quantified by subtracting the spinal cord diameter on a midsagittal MR image from the disc-level spinal canal diameter. Dynamic flexion and extension cervical spine MR imaging modalities have been proposed to evaluate functional stenosis, although not all centers may be capable of performing these studies.

Cervical Cord Neurapraxia in Adult Athletes

A large epidemiological study compared athletes who reported an episode of cervical cord neurapraxia to athletes and nonathletes who had never experienced neurapraxia and found that those with previous neurapraxia had significantly smaller cervical spinal canals and lower Torg ratios, suggesting an association between stenosis and neurapraxia. A smaller series of 9 rugby players with cervical cord neurapraxia demonstrated 4 athletes with Torg ratios < 0.8 and an additional 2 athletes with congenital vertebral body fusions. Another series of 2 professional football players, each with an episode of cervical cord neurapraxia, reported normal Torg ratios in both, but significant stenosis on myelography. In the largest series to date of cervical cord neurapraxia in athletes, 110 patients were evaluated after 1 episode of cervical cord neurapraxia. In this series, 80% presented with symptoms in all 4 extremities, and 40% were completely plegic; 74% were Grade I (symptoms lasting < 15 minutes). On subsequent evaluation of 104 radiographs of the athletes with cervical cord neurapraxia, 86% had Torg ratios < 0.8, and of these patients, 53 underwent MR imaging. More than 81% of these patients had evidence of cervical spinal stenosis.
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of cervical disc herniation, 25% had evidence of effacement of the thecal sac, and 34% had frank cervical cord compression. In the largest modern series, 7 10 athletes who experienced cervical cord neurapraxia underwent MR imaging that demonstrated cervical stenosis in all patients and frank cord compression in 3 (33%).

For patients with cervical cord neurapraxia, surgery should be considered in the setting of focal lesions and associated cord compression or instability on plain radiographs and MR imaging. In two combined series, 12 (8.5%) of 142 patients underwent surgery for cord compression or spinal instability.22,24 The authors did not make general recommendations regarding surgical decision-making as they believed the number of patients was too small. Instead, they proposed that the decision to pursue surgery should be individualized based on imaging findings and patient wishes. Maroon et al.10 reported a series of 5 professional-level athletes who underwent cervical decompressive surgery and fusion for focal cord compression after an episode of cervical cord neurapraxia. All 5 returned to sports, but 2 subsequently developed career-ending adjacent-level disease. The authors suggest that it is safe for athletes to return to previous levels of activity after a single-level, radiographically confirmed fusion, but close attention should be paid as these patients may develop recurrence at the level above or below.

A previous episode of cervical cord neurapraxia may predispose athletes to recurrent episodes, but the risk of recurrence is determined by a complex interplay between the patient’s cervical spine anatomy and the type of athletic activity. One series reports 52 patients who returned to sports after cervical cord neurapraxia, 32 (62%) of whom experienced a subsequent episode. Of the athletes who returned to previous levels of activity after an episode of cervical cord neurapraxia in previous large series, none subsequently developed a permanent neurological injury.24 Conversely, of the athletes who sustained permanent neurological injury, none reported a previous episode of neurapraxia, leading the authors to suggest that cervical cord neurapraxia does not necessarily confer an increased risk of permanent injury. However, 1 case report describes a football player who became quadriplegic from a subsequent injury 1 year after an episode of cervical cord neurapraxia. Consequently, some practitioners would consider a single episode of cervical cord neurapraxia to be a contraindication to return to sports. With respect to cervical stenosis, Bailes et al.12 report included 4 athletes with cervical stenosis who returned to play after an episode of cervical cord neurapraxia. None of these athletes experienced a subsequent episode and, interestingly, all 4 had intact “functional reserve” (CSF signal surrounding the spinal cord) on MRI imaging.

In 1962, Penning described a “pincers mechanism” by which extension of the cervical spine can cause myelopathy that can also be applied to the mechanics of cervical cord neurapraxia. Penning studied lateral flexion-extension radiographs and developed a model of spinal cord “pinching” between the posterior inferior aspect of the superior vertebral body and the anterior superior aspect of the inferior lamina during extension. In addition, loss of tension on the dura and the ligamentum flavum caused these structures to protrude into the spinal canal, further decreasing the canal reserve with the neck extended. Torg et al. extrapolated these findings to explain that, during flexion, the spinal cord is compressed between the lamina of the superior level and the posterior superior aspect of the inferior vertebral body. In the stenotic canal of an adult, the pincer mechanism is likely more profound. Experimental studies in a giant squid axon model of cord deformation demonstrated that during injury there was an increase in intracellular calcium.23 Depending on the strength and duration of the injury, the chemical disturbance can be either reversible or irreversible, leading to permanent cellular damage. This can be applied to the phenomenon of sports-related cervical neurapraxia that results from a short duration injury of moderate magnitude that causes the spinal cord to be deformed by the “pincers mechanism,” which causes reversible chemical changes in the spinal cord below the level of injury. This is expressed symptomatically as a transient neurological deficit.

Cervical Cord Neurapraxia in Pediatric Athletes

In the large Torg et al.22 series, 7 patients had normal Torg ratios (that is, no evidence of cervical spinal stenosis), and the mean age of these patients was 17 years old. Only 1 study has specifically evaluated the association between cervical spinal stenosis and cervical cord neurapraxia in pediatric patients. Boockvar et al.6 retrospectively reviewed 13 children younger than 16 years of age who presented to the Children’s Hospital of Philadelphia with cervical cord neurapraxia. The most common mechanism of injury was hyperflexion (38%). There were significant differences in symptomatology relative to adult athletes. In contrast to adult patients, the majority of children (77%) reported neck pain and decreased cervical range of motion. The distribution of deficits was most commonly upper-extremity paresis (38%), followed by quadripareisis (31%), hemiparesis (23%), and lower-extremity paresis (8%). The duration of symptoms was longer than in adults, with a mean duration of 26 hours, with 1 patient experiencing quadripareisis and paresthesias for 5 days. The majority of patients had combined motor and sensory disturbances (85%). No patients were completely plegic.

Torg ratios were calculated for all 13 patients. Interestingly, all patients had Torg ratios > 0.8 indicating that none had cervical spinal stenosis by traditional radiographic criteria. Magnetic resonance imaging was performed within 24 hours of injury and none of the patients demonstrated evidence of spinal cord or extraneural pathology, which often appear in adults. No patients were treated with cervical spine surgery. Neurological symptoms resolved in all patients. Follow-up flexion-extension radiographs confirmed cervical stability. Ten of 13 patients had long-term follow-up, and all of these patients had returned to previous levels of activity including sports. None reported recurrence of neurapraxia symptoms. None had experienced a subsequent permanent neurological injury. Although the number of patients is small, this evidence suggests that children can safely return to athletic activities after an episode of cervical cord neurapraxia. Similarly, in the series by Torg et al.,22
3 of the 7 patients with cervical cord neurapraxia and normal Torg ratios returned to contact sport activity with no recurrence. Future large-scale studies are needed to confirm that cervical cord neurapraxia does not incur an increased risk of future neurological injury.

The observation that cervical cord neurapraxia in children is not associated with cervical spinal stenosis is indicative of a different mechanism of neurological deficit in this unique population. In contrast to adults, the pediatric cervical spine is more mobile, likely due to more compliant ligaments,13 underdeveloped paraspinous musculature,14 increased water content of intervertebral discs,9 and immature facet joints.8 It was proposed that in this setting, the mobility of the spine allows the spinal cord to stretch past its tolerance or allows the spinal cord to forcibly contact the bony elements of the spine resulting in transient neurological symptoms. Therefore, even in the absence of cervical spinal stenosis, injury can occur. The phenomenon of spinal cord injury without radiographic abnormality describes the potential consequence of this increased mobility.17 Spinal cord injury without radiographic abnormality is generally associated with extreme forces such as a motor vehicle accident. Cervical cord neurapraxia in children can be considered a mild form of spinal cord injury without radiographic abnormality in which the forces that deform the spine are sufficient to cause reversible perturbation of spinal cord physiology without permanently damaging the cord.

Guidelines for Return to Play After Cervical Neurapraxia

Clearance of athletes for resumption of physical and athletic activity is a highly controversial topic and one that is often without consensus opinion.15 Fundamental requirements for returning to athletic activity after a cervical injury with neurapraxia should include normal strength, painless range of motion, and a stable vertebral column.14 Bailes2 suggests that patients with MR imaging evidence of CSF signal surrounding the cervical cord may be safe to return to play. Further considerations should be the mechanism of the original injury, objective physical examination and radiographic findings, and the athlete’s recovery response.26 Page and Guy16 recommend that absolute contraindications for return to play after cervical neurapraxia are ligamentous instability, a single neurapraxic event with evidence of cord damage, multiple events, and/or events with symptoms lasting longer than 36 hours.

Conclusions

Cervical cord neurapraxia is common in adult and pediatric athletes. Cervical cord neurapraxia is associated with cervical spinal stenosis in adult athletes but not in the pediatric population. This observation likely highlights a mechanistic difference in the injury in the two different age groups. In adults, a stenotic canal will predispose patients to cervical cord injury at the level of stenosis following an extension, flexion, or axial load injury. Therefore, surgery should be considered for a focal lesion causing cord compression. In comparison, the pediatric spine demonstrates increased mobility, predisposing the spinal cord to contact with bony elements with stretching even in the absence of a focal stenosis. Although symptoms invariably resolve, recurrences are not uncommon, most notably in adults. Patients should be advised of this risk when considering return to sports-related activities.

Disclosure

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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