Spinal cord concussion

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The hallmark of concussion injuries of the nervous system is the rapid and complete resolution of neurological deficits. Cerebral concussion has been well studied, both clinically and experimentally. In comparison, spinal cord concussion (SCC) is poorly understood. The clinical and radiological features of 19 SCC injuries in the general population are presented. Spinal cord injuries were classified as concussions if they met three criteria: 1) spinal trauma immediately preceded the onset of neurological deficits; 2) neurological deficits were consistent with spinal cord involvement at the level of injury; and 3) complete neurological recovery occurred within 72 hours after injury.

Most cases involved young males, injured during athletics or due to falls. Concussion occurred at the two most unstable spinal regions, 16 involving the cervical spinal and three the thoracolumbar junction. Fifteen cases presented with combined sensorimotor deficits, while four exhibited only sensory disturbances. Many patients showed signs of recovery with the first few hours after injury and most had completely recovered within 24 hours. Only one case involved an unstable spinal injury. There was no evidence of ligamentous instability, spinal stenosis, or canal encroachment in the remaining 18 cases. Two patients, both children, suffered recurrent SCC injuries. No delayed deterioration or permanent cord injuries occurred.

Spinal abnormalities that would predispose the spinal cord to a compressive injury were present in only one of the 19 cases. This suggests that, as opposed to direct cord compression, SCC may be the result of an indirect cord injury. Possible mechanisms are discussed.

KEY WORDS - concussion - spinal cord injury - transient neurological deficits - axonal refractory period

CEREBRAL concussion is by far the most frequent type of reversible injury of the nervous system. Its clinical features have been extensively reviewed, and reproducible experimental models have allowed investigation into its pathophysiology. Spinal cord trauma often results in permanent deficits; however, in a small proportion of cases recovery is rapid and complete. Comparatively little is known about concussion injuries of the spinal cord, due to their infrequency and the lack of an experimental model. The early literature on spinal cord concussion (SCC) appears to be based on a rather heterogeneous group of cord injuries that includes cases with permanent deficits. This has led to some confusion concerning definition, clinical features, management, and outcome of SCC. A more recent study which found a high incidence of spinal stenosis in cases of cervical SCC in athletes injured through contact sports may not be applicable to SCC injuries in the general population. This is a retrospective review of 19 cases of SCC in the general population.

Clinical Material and Methods

Data were collected through a retrospective chart review of all incomplete spinal cord injuries that occurred over a 12-year period from 1975 to 1987 at two neurosurgical teaching hospitals, one adult and the other pediatric. A single adult case from a third institution was also included. A total of 19 cases of SCC were collected. This review may not include all SCC injuries treated at these institutions during this time because not all spinal cord injuries were reviewed, only those classified as incomplete injuries. Therefore, from this review it was not possible to determine the incidence of SCC injuries or what proportion of all spinal cord injuries they represent.

Spinal cord injuries were classified as concussions if they met the following three criteria: 1) spinal trauma immediately preceded the onset of neurological deficits; 2) neurological deficits were consistent with spinal cord involvement at the level of injury; and 3) complete neurological recovery occurred within 72 hours after
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injury. Follow-up data were obtained by reviewing clinic or office notes and by telephone. Spinal x-ray films (available in 13 cases) were reviewed specifically for evidence of spinal fractures, canal encroachment, ligamentous instability, spinal stenosis, or other congenital or acquired spinal abnormalities. For the remaining six patients, radiological data were obtained from x-ray reports.

Results

Clinical Presentation

Nineteen spinal cord injuries were classified as SCC's (Table 1); 10 (52%) occurred in adults aged 18 years or over (mean age 30 years), and nine (48%) in patients under the age of 18 years (mean age 12 years). The mean age of all 19 patients was 22 years. Males predominated in the under 18-year-old group (seven of nine cases). All spinal injuries were nonpenetrating; 10 occurred during athletic activities, six due to falls, and three from motor-vehicle accidents.

Concussion injuries occurred in two regions of the spine: 16 cases (84%) in the cervical area and three cases (16%) at the thoracolumbar junction. In two cervical SCC's the clinical findings suggested that the upper cervical cord was involved. The decreased left facial pinprick sensitivity associated with the left hemiparesis in Case 10 suggested involvement of the pars caudalis of the nucleus of the trigeminal spinal tract, which extends from the lower medulla down to C-3.1 The pattern of weakness in Case 9 (arm and opposite leg) has been described in lesions involving the decussation of the pyramids at the cervicomedullary junction.2

Fifteen patients (79%) presented with a combined sensorimotor deficit. Quadriaparesis was the commonest single type of motor deficit in cervical SCC. Two of the three patients with thoracolumbar injuries presented with hemiparesis. Four cases (21%), three cervical and one thoracolumbar, presented with only sensory deficits. No patient was known to have suffered any previous spinal cord injury. Seven patients had an associated cerebral concussion. No hemodynamic instability was documented in any of the 19 cases.

Radiographic Findings

Spinal x-ray films, obtained in all 19 cases, were normal in 13 of the 16 cases of cervical injuries and in all three thoracolumbar injuries. Congenital anomalies of the cervical spine were found in two of the three cases of cervical SCC with abnormal x-ray films (Cases 15 and 16). Both cases involved partial fusion of the facet joints at C-4 and C-5, bilaterally, as well as spina bifida occulta (at C-4 and C-5 in Case 15 and involving the posterior arch of C-1 in Case 16). No ligamentous instability was detected on flexion or extension views in these two patients. The third patient with abnormal x-ray films (Case 6) suffered an unstable spinal injury: a fractured facet and lamina of C-6 with a 3-mm anterior subluxation of C-5 and C-6.

The x-ray films of 12 patients with cervical SCC were available for review, including the three cases reported as abnormal. These films were specifically reviewed for any underlying spinal abnormalities that might predispose the cord to direct compression. Two radiological methods were used to screen for spinal stenosis in the cervical spine. In the first method, the smallest anteroposterior (AP) diameter of the spinal canal was measured at each vertebral body level from C-3 to C-6. In adults, the mean AP diameter of the spinal canal at these levels is normally 16 to 18 mm and the canal is stenotic if it is 14 mm or less.3,27 In all 12 cases of cervical SCC in which the x-ray films were reviewed, the AP diameter of the canal was a minimum of 17 mm (Table 1). In the second method, devised by Torg, et al.,27 the AP diameter of the spinal cord canal (C) is compared to the AP diameter of the vertebral body (B) at the same level of the cervical spine. Between C-3 and C-6 in the normal cervical spine, C is usually equal to or greater than B such that the ratio C/B is equal to or greater than 1:1. Congenital stenosis exists if this ratio is less than 0.8:1.27 The minimum C/B ratio was 0.9:1 among all 12 cases of cervical SCC in which x-ray films were available for review (Table 1).

Nine of these 12 cervical SCC's had flexion and extension views which revealed no evidence of ligamentous instability. Specifically, the two patients with bilateral facet joint fusion did not exhibit any increased mobility at levels adjacent to the fused segments. The x-ray films of two of the three patients with thoracolumbar injuries were reviewed and both were normal including flexion and extension views done in one case.

In summary, radiological studies in these 19 cases of SCC revealed only one unstable spinal injury (Case 6). In the remaining 18 cases, no spinal abnormalities which might predispose the cord to direct compression during trauma, such as spinal stenosis or ligamentous instability, were detected. Myelography was not performed because neurological recovery was rapid and complete in all cases.

Management

All 19 cases were initially managed as potentially unstable spinal injuries. Bed rest and log-rolling were used in all cases, and in the 16 patients with cervical injuries the neck was immobilized as well: a cervical collar was used in 14 cases, Crutchfield tongs and cervical traction were applied in one (Case 10), and halo traction was used in the patient with C5–6 fracture subluxation (Case 6). Once neurologically recovered, patients with normal or stable spinal x-ray films were mobilized. The C5–6 fracture subluxation was reduced after 2 days of halo traction and the patient was mobilized in a halo vest for 3 months. Steroids were used in only three cases and were discontinued soon after complete neurological recovery.
TABLE 1
Clinical summary of 19 cases of spinal cord concussion*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Mechanism of Injury</th>
<th>Medical History</th>
<th>Motor Deficits</th>
<th>Sensory Deficits</th>
<th>Duration of Deficits</th>
<th>X-Ray Findings</th>
<th>Minimum AP Canal C3–6 Diameter</th>
<th>Flexion &amp; Extension</th>
<th>Minimum Ratio C/B †</th>
<th>Management</th>
<th>Follow-Up Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>60, F</td>
<td>fall</td>
<td>weakness × 4, lt &gt; rt</td>
<td>quadriaparesis, LE &gt; UE, lt &gt; rt</td>
<td>↓ PP, lt</td>
<td>&lt; 48 hrs</td>
<td>normal</td>
<td>normal</td>
<td>not done</td>
<td>19 mm</td>
<td>1.1</td>
<td>cervical collar</td>
</tr>
<tr>
<td>2</td>
<td>20, M</td>
<td>MVA</td>
<td>numbness × 4</td>
<td>normal</td>
<td>↓ PP, lt hand</td>
<td>&lt; 24 hrs</td>
<td>normal (including CT)</td>
<td>normal</td>
<td>NA</td>
<td>NA</td>
<td>cervical collar</td>
<td>none</td>
</tr>
<tr>
<td>3</td>
<td>20, F</td>
<td>gymnastics</td>
<td>numbness &amp; weakness × 4</td>
<td>weakness UE, lt DTR ↓, toes ↓ × 2</td>
<td>↓ PP &amp; DC, lt UE &amp; LE</td>
<td>&lt; 48 hrs</td>
<td>normal</td>
<td>normal</td>
<td>17 mm</td>
<td>0.9</td>
<td>cervical collar</td>
<td>none</td>
</tr>
<tr>
<td>4</td>
<td>20, F</td>
<td>fall</td>
<td>paralysis × 4</td>
<td>weakness UE × 2 &amp; lt LE, lt toe ↑</td>
<td>normal</td>
<td>&lt; 24 hrs</td>
<td>normal</td>
<td>normal</td>
<td>18 mm</td>
<td>1.3</td>
<td>cervical collar</td>
<td>none</td>
</tr>
<tr>
<td>5</td>
<td>38, M</td>
<td>hockey</td>
<td>numbness &amp; weakness × 4</td>
<td>normal</td>
<td>normal</td>
<td>&lt; 1 hr</td>
<td>normal</td>
<td>normal</td>
<td>22 mm</td>
<td>1.2</td>
<td>cervical collar</td>
<td>none</td>
</tr>
<tr>
<td>6</td>
<td>47, F</td>
<td>MVA</td>
<td>weakness lt LE</td>
<td>normal</td>
<td>↓ DC lt LE, normal PP</td>
<td>36 hrs</td>
<td>C5-6 fracture-subluxation</td>
<td>not done</td>
<td>20 mm</td>
<td>1.2</td>
<td>halo traction 2 days; halo vest 3 mos</td>
<td>normal, 7 mos; solid fusion, 3 mos</td>
</tr>
<tr>
<td>7</td>
<td>18, M</td>
<td>football</td>
<td>paralysis × 4 (30 min)</td>
<td>weak finger extensors, toes ↓ × 2</td>
<td>normal</td>
<td>12 hrs</td>
<td>normal</td>
<td>NA</td>
<td>NA</td>
<td>cervical collar</td>
<td>none</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>15, F</td>
<td>horse-back riding</td>
<td>paralysis × 4</td>
<td>normal</td>
<td>↓ PP T12–Si × 2</td>
<td>&lt; 24 hrs</td>
<td>normal</td>
<td>not done</td>
<td>NA</td>
<td>NA</td>
<td>cervical collar</td>
<td>normal, 1 mo</td>
</tr>
<tr>
<td>9</td>
<td>8, M</td>
<td>wrestling, hyperflexion</td>
<td>paralysis lt UE, LE × 2</td>
<td>weakness lt UE, rt LE, DTR normal</td>
<td>↓ PP &amp; DC, lt UE &amp; rt LE</td>
<td>&lt; 48 hrs</td>
<td>normal</td>
<td>not done</td>
<td>20 mm</td>
<td>1.1</td>
<td>cervical collar, Decadron 3 days later</td>
<td>normal, 2 yrs</td>
</tr>
<tr>
<td>10</td>
<td>13, M</td>
<td>fall</td>
<td>weakness lt UE, numbness lt LE</td>
<td>lt hemiparesis, lt toe ↑</td>
<td>↓ PP &amp; DC, lt C2–T7, ↓ PP lt face, ↓ lt corneal reflex</td>
<td>&lt; 24 hrs</td>
<td>normal</td>
<td>not done</td>
<td>NA</td>
<td>NA</td>
<td>cervical collar, traction 2 wks</td>
<td>none</td>
</tr>
<tr>
<td>11</td>
<td>12, M</td>
<td>wrestling</td>
<td>numbness lt side</td>
<td>normal</td>
<td>↓ PP lt thigh &amp; lt side trunk</td>
<td>2–3 hrs</td>
<td>normal</td>
<td>normal</td>
<td>18 mm</td>
<td>1.0</td>
<td>cervical collar</td>
<td>normal, 3 mos</td>
</tr>
<tr>
<td>12</td>
<td>14, M</td>
<td>fall</td>
<td>paralysis LE × 2, numbness × 4, catheterized</td>
<td>weakness × 4, toes ↑ × 2, DTR ↑</td>
<td>normal</td>
<td>12 hrs</td>
<td>normal</td>
<td>normal</td>
<td>17 mm</td>
<td>0.9</td>
<td>cervical collar, Decadron 1 day</td>
<td>none</td>
</tr>
<tr>
<td>13</td>
<td>11, M</td>
<td>hockey</td>
<td>lt-sided weakness &amp; numbness</td>
<td>lt hemiparesis normal</td>
<td>normal</td>
<td>2–3 hrs</td>
<td>normal</td>
<td>normal</td>
<td>18 mm</td>
<td>1.1</td>
<td>cervical collar</td>
<td>recurrent cervical concussion at 3 mos, no recurrence, 1 1/2 yrs later</td>
</tr>
<tr>
<td>14</td>
<td>13, M</td>
<td>soccer</td>
<td>paralysis × 4 (2 min)</td>
<td>normal</td>
<td>normal</td>
<td>&lt; 6 hrs</td>
<td>normal</td>
<td>normal</td>
<td>19 mm</td>
<td>1.0</td>
<td>cervical collar</td>
<td>normal, 3 yrs</td>
</tr>
</tbody>
</table>

*In Cases 1 to 16, injury was sustained at the cervical level, and in Cases 17 to 19, at the thoracolumbar level. AP = anteroposterior; UE = upper extremity; LE = lower extremity; DC = dorsal column function; PP = pinprick sensation; CT = computerized tomography; MVA = motor-vehicle accident; DTR = deep tendon reflex; NA = not available; × 4 = all limbs; × 2 = bilateral; ↑ = increased; ↓ = decreased. Decadron = dexamethasone.

† Ratio of the AP diameter of the spinal cord canal (C) to the AP diameter of the vertebral body (B).

TABLE 1 (continued —)
Spinal cord concussion

**TABLE 1 (continued)***

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Mechanism of Injury</th>
<th>Medical History</th>
<th>Motor Deficits</th>
<th>Sensory Deficits</th>
<th>Duration of Deficits</th>
<th>X-Ray Findings Neutral</th>
<th>Flexion &amp; Extension</th>
<th>Minimum AP Canal Diameter C3-6</th>
<th>Minimum Ratio C/B†</th>
<th>Management</th>
<th>Follow-Up Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>13, M</td>
<td>hockey</td>
<td>weakness UE</td>
<td>weakness UE</td>
<td>PP C5-T7 x 2</td>
<td>72 hrs, normal power at 48 hrs</td>
<td>normal</td>
<td>18 mm 1.1</td>
<td>normal cervical collar, Decadron 4 days</td>
<td>normal, 3 yrs later, no contact sports</td>
<td>normal</td>
<td>10 yrs</td>
</tr>
<tr>
<td>6</td>
<td>13, F</td>
<td>skating, hyperflexion</td>
<td>neck pain, numbness LE</td>
<td>normal</td>
<td>PP below thighs x 2</td>
<td>72 hrs</td>
<td>normal</td>
<td>partial fusion C4-6, bifid posterior arch C-1</td>
<td>normal cervical collar at 3 yrs</td>
<td>normal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>39, F</td>
<td>fall</td>
<td>weakness LE</td>
<td>paraparesis</td>
<td>L-1 sensory level (PP) ↓ DC LE x 2</td>
<td>72 hrs</td>
<td>normal</td>
<td>not done</td>
<td>—</td>
<td>bed rest</td>
<td>none</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>23, M</td>
<td>fall</td>
<td>numbness LE</td>
<td>tender L5-S1, no weakness</td>
<td>PP below knees x 2</td>
<td>2-3 hrs</td>
<td>normal</td>
<td>not done</td>
<td>—</td>
<td>bed rest</td>
<td>none</td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>19, M</td>
<td>MVA</td>
<td>paraparesis LE</td>
<td>normal</td>
<td>L-1 sensory level (PP) ↓ DC LE x 2</td>
<td>24 hrs</td>
<td>normal</td>
<td>normal thoracolumbar level</td>
<td>—</td>
<td>bed rest</td>
<td>normal (6 mos)</td>
<td></td>
</tr>
</tbody>
</table>

* In Cases 1 to 16, injury was sustained at the cervical level, and in Cases 17 to 19, at the thoracolumbar level. AP = anteroposterior; UE = upper extremity; LE = lower extremity; DC = dorsal column function; PP = pinprick sensation; CT = computerized tomography; MVA = motor-vehicle accident; DTR = deep tendon reflex; NA = not available; x 4 = all limbs; x 2 = bilateral; † = increased; ↓ = decreased. Decadron = dexamethasone.

† Ratio of the AP diameter of the spinal cord canal (C) to the AP diameter of the vertebral body (B).

**Clinical Outcome**

All patients made a full neurological recovery, many showing signs of improvement within the 1st hour after injury. All deficits had cleared by 24 hours in 14 cases (73%), and all but three patients had completely recovered by 48 hours. These remaining three patients exhibited minor but consistent sensory disturbances which persisted for up to 72 hours even though their motor deficits had completely resolved within 24 hours.

**Follow-Up Results**

Follow-up data were available in 11 patients, ranging from 1 month to 3 years after injury (mean 16 months). Two patients, both children (Cases 9 and 13), suffered a recurrent SCC: one 3 weeks and the other 3 months after the initial injury. In these two cases, the initial and recurrent SCC’s both involved the cervical cord. Spinal x-ray films, including flexion and extension views, were normal after both the initial and recurrent injuries. A metrizamide-enhanced computerized tomography scan, performed on one of these two patients, was normal. A review of the details of the initial concussion injuries in these two cases revealed no unusual features, although in one case the sensory deficits persisted beyond 48 hours. In the two patients with recurrent SCC’s, all deficits cleared within 48 hours. Follow-up monitoring of one of these two patients at 1½ years revealed no further recurrences. The other patient was lost to follow-up review. No known permanent spinal cord injuries or nontraumatic spinal disorders occurred during the follow-up period.

**Discussion**

**Terminology**

Cerebral concussion is a clinical syndrome, defined as a transient impairment of neural function due to mechanical forces. Through clinical review and long-term follow-up monitoring, important neurological sequelae of single and repeated cerebral concussions have been identified. Concussive injuries of the brain have been reproduced experimentally, resulting in a better understanding of the biomechanical, metabolic, vascular, pathological, and electrophysiological aspects of this syndrome.

Irreversible spinal cord injuries have also been studied extensively, but SCC remains poorly understood because it occurs infrequently and no good experimental model exists. This present review of SCC in...
the general population was undertaken in an attempt to identify any predisposing factors, and to assess the risk of future spinal cord injuries in this group.

Literature Review
Obersteiner first introduced the term "spinal cord concussion" in 1879 to describe spinal cord injuries in which complete neurological recovery occurred within 24 to 48 hours after injury. He believed that it consisted of a "molecular disturbance of neurons due to trauma." However, much of the subsequent literature on SCC does not adhere closely to this definition; spinal cord injuries in which the cord was thought not to have been compressed were classified as SCC’s, even though the neurological deficits were permanent. Groat, et al., re-emphasized the reversibility of a true SCC, and stressed that it should be considered a functional block of neural activity, similar to the mechanism of cerebral concussion postulated 4 years before by Denny-Brown and Russel.

There have been few other recent reviews of transient spinal cord injuries. Torg, et al., estimated that 1.3 of every 10,000 United States college and professional football players sustained an SCC in 1984. Undoubtedly, it occurs less frequently in the general population but the true incidence may be difficult to obtain as many cases may go unrecognized due to the transient and often subtle symptomatology of SCC.

Torg, et al., presented 32 college or professional athletes, most of whom were football players, who had suffered transient cervical cord injuries. Thirty of these fulfilled the three criteria for an SCC used in the present review. Six of their patients had a previous history of at least one traumatic cervical cord injury, all transient in nature. None of the 19 patients in the present review gave a history of a previous spinal cord injury, but there were no other differences in the clinical presentations of our cases of cervical SCC’s compared to those presented by Torg, et al.

Comparison of Radiological Findings
All 30 patients with cervical SCC presented by Torg, et al., had cervical spine abnormalities; 26 had an associated spinal stenosis, 20 congenital and six acquired. Four of their patients had instability of the posterior spinal ligaments based on widening of the interspinous space on neck flexion. Based on the high incidence of spinal stenosis and ligamentous instability found in their review, Torg, et al., concluded that transient cervical cord injuries are most likely due to a transient compression of the spinal cord at the time of injury. Holmes, in 1915, was perhaps the first to suggest that spinal stenosis predisposed patients to SCC, postulating that such injuries were due to a slapping of the cord against the spinal canal.

In our review, radiological evidence to suggest that spinal cord compression occurred at the time of injury was found in only one case (C5–6 fracture subluxation in Case 6). Although the criteria for spinal stenosis used in this review were identical to those used by Torg, et al., none of the patients in our series exhibited radiological evidence of either congenital or acquired spinal stenosis. It appears that spinal motion does play a role in SCC, because all 19 concussion injuries occurred at either the cervical or thoracolumbar regions, the two most mobile areas of the vertebral column. Nevertheless, no spinal hypermobility was detected in any of the 12 patients in whom flexion-extension x-ray views were obtained (Table 1). As no patient underwent myelography, it is impossible to absolutely rule out the presence of a soft-tissue lesion, such as an intervertebral disc herniation or ligamentous hypertrophy; however, these abnormalities are uncommon in this younger age group.

Experimental Studies
The majority of experimental models of spinal cord injury produce an irreversible cord injury, but Parkinson and coworkers have attempted to develop a model that results in a reversible cord injury. In decerebrate preparations of the frog, they dropped a weight directly onto the posterior neural arch such that no direct cord compression occurred. Evoked potentials (EP’s) were used to monitor the transmission of impulses through the spinal cord at the level of the injury. Blows to the mid-thoracic spine resulted in immediate abolition of the EP’s which, on average, completely recovered within 30 minutes. Although recovery of EP’s does not exclude an underlying permanent cord injury, these studies do suggest that direct trauma to the posterior neural arch without any obvious direct cord injury can result in an apparently reversible block of impulse transmission through the long tracts of the spinal cord. Parkinson, et al., postulated that this transient cord dysfunction may be due to the absorption of brief bursts of kinetic energy by the spinal cord. The absence of pathological changes in these “concussed” spinal cords would have further supported such a hypothesis, but no pathological studies were presented.

Pathophysiology
The experimental work by Parkinson, et al., together with the lack of any radiological evidence of spinal stenosis or ligamentous instability in 18 of our 19 cases of SCC suggest that concussive injuries of the spinal cord may be the result of an indirect injury in which the injuring force is transmitted to the cord without direct cord compression. The pathophysiology of SCC is unknown, but several theoretical mechanisms appear to be unlikely. Neither focal demyelination of axons in the long tracts of the spinal cord or spinal cord edema would account for the immediate onset or rapid recovery of deficits following injury. Although the time course of the neurological deficits in SCC is suggestive of an underlying transient ischemia, Kobrine, et al., have shown experimentally that conduction through
the long tracts of the spinal cord is relatively insensitive to ischemia. Simultaneous monitoring of spinal evoked responses (SER) and spinal cord blood flow (SCBF) revealed that the SER did not change until the SCBF fell below 25% of normal and that SER disappeared only after 10 minutes or more of absolute ischemia (SCBF = 0).

Based on the immediate onset and the rapid and complete recovery of neurological deficits in SCC, it has been postulated that this entity involves functional disturbance of the axonal membrane without disruption of its structural integrity.\(^1\)\(^3\) Studies carried out by Hodgkin and Huxley\(^7\) on the giant squid axon revealed that propagation of an action potential requires movement of ions, primarily Na\(^+\) and K\(^+\), into and out of the axon through ion channels located within the axonal membrane. During each action potential there normally exists an absolute refractory period during which the segment of the axon just depolarized is completely unresponsive to any subsequent impulse. During this period, which is normally very short, there exists a physiological block of impulse conduction that persists until repolarization of that axon segment is complete. Therefore, SCC could be due to a prolongation of the absolute refractory period of axons in the tracts of the spinal cord. This could come about through a delay in the closure of Na\(^+\) channels and/or a delay in the opening of K\(^+\) channels following depolarization. The mechanisms by which trauma might result in such functional disturbances in the axonal membrane remain speculative. Experimental work in cerebral concussion reveals that a prolonged period of neuronal inactivity is immediately preceded by a short phase of intense neuronal hyperactivity that occurs at the time of head injury.\(^\text{13-17}\) At present there is no clinical or experimental evidence to suggest that such a phase of excitation of long tract axons of the spinal cord occurs in SCC.

**Management Considerations**

By definition, the short-term prognosis for SCC injuries is excellent. Long-term outcome is more difficult to assess given the small patient population and relatively short follow-up in this review. Combining the patients in this study with those of Torg, et al.,\(^27\) follow-up data on a total of 49 SCC injuries reveal that seven patients suffered recurrent cord injuries, all of which were concussion injuries. No known permanent cord injuries occurred. Torg, et al., interviewed 117 patients rendered quadriplegic due to football injuries and found only two with a previous history of SCC. In their review, recurrent SCC only occurred in athletes who resumed contact sports. It will be difficult to reduce the risk of recurrent SCC involving the general population unless predisposing factors that can be modified are identified in this group. Contrary to the review by Torg, et al., no predisposing factors have been identified in the 19 patients in our series. Longer follow-up monitoring of a larger number of SCC injuries will be required to determine if these patients are at an increased risk for permanent spinal cord injuries or nontraumatic spinal cord disorders. Few generalizations can be made about the long-term management of SCC based on this present review. Each case should be dealt with on an individual basis according to the clinical and radiological data, and the age and lifestyle of the patient.

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