Spinal cord injury without radiographic abnormalities in children

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✓ Spinal cord injury in children often occurs without evidence of fracture or dislocation. The mechanisms of neural damage in this syndrome of spinal cord injury without radiographic abnormality (SCIWORA) include flexion, hyperextension, longitudinal distraction, and ischemia. Inherent elasticity of the vertebral column in infants and young children, among other age-related anatomical peculiarities, render the pediatric spine exceedingly vulnerable to deforming forces. The neurological lesions encountered in this syndrome include a high incidence of complete and severe partial cord lesions. Children younger than 8 years old sustain more serious neurological damage and suffer a larger number of upper cervical cord lesions than children aged over 8 years. Of the children with SCIWORA, 52% have delayed onset of paralysis up to 4 days after injury, and most of these children recall transient paresthesia, numbness, or subjective paralysis. Management includes tomography and flexion-extension films to rule out incipient instability, and immobilization with a cervical collar. Delayed dynamic films are essential to exclude late instability, which, if present, should be managed with Halo fixation or surgical fusion. The long-term prognosis in cases of SCIWORA is grim. Most children with complete and severe lesions do not recover; only those with initially mild neural injuries make satisfactory neurological recovery.

KEY WORDS • spinal cord injury • juvenile spine • increased deformability • severe neurological damage • delayed paralysis • tomography • dynamic radiography • children

Because of anatomical and biomechanical differences in the human spine at various ages, the mechanism of neural damage and degree of osteoligamentous disruptions associated with spinal cord injury may be radically different from one age group to another. Accordingly, patients with spinal cord injury can be divided into four age categories: those injured 1) during birth; 2) between infancy and 16 years; 3) between 16 years and middle age; and 4) between late-middle to old age.

The mechanism involved in intrapartum spinal cord injury is generally thought to involve longitudinal traction of the neonatal spine during breech extraction and subsequent rupture of the cord.1,29,43 Due to the extreme elasticity of the fibrocartilaginous spine and its investing soft tissues, the resulting tetraplegia is often described without radiographical evidence of fracture or dislocation.1,29,51

From 16 years to middle age (arbitrarily defined as 45 years), it is exceedingly rare to find closed spinal cord trauma without skeletal injury or dislocation.92,92

The mechanical properties of the spines of patients in this age range are such that fractures of the vertebral body, pedicles, or facets, or locking of an anteriorly dislocated facet occur before the neural structures are injured.

In patients of late-middle to old age, a syndrome of closed spinal cord injury without demonstrable skeletal injury was identified by Crooks and Birkett in 1944.33 The spines of these patients usually show significant spondylotic changes, resulting in sagittal narrowing of the central canal, and the neurological injury is most often acute central cord syndrome. The mechanism of injury has been convincingly shown by Taylor and Blackwood71,72 and Schneider, et al.,66,67 to be hyperextension.

Spinal cord injury is uncommon from infancy to 16 years. The incidence of pediatric spinal cord injuries among all spinal cord injuries has been quoted as anywhere from 0.65% to 9.47%.95,96,97,98,99,100,101 Although many children with spinal cord injury have associated vertebral injuries, a distinct group of children with...
Cord injury with normal radiography

Traumatic myelopathy have no radiographic evidence of fracture or dislocation. Twenty-four children with this syndrome of spinal cord injury without radiographic abnormality (SCIWORA) were treated at Children's Hospital of Pittsburgh (CHP) from 1960 to 1980, constituting 66.7% of all children with nonpenetrating spinal cord injuries seen at this institution during that time. Our experience shows that the pediatric syndrome of SCIWORA is characterized by clinical features and prognosis very different from those found in children with spinal cord injury and associated fracture-dislocation, or in adults with cervical spondylosis and hyperextension injury. It is probable that complex pathogenetic mechanisms unique to the spine of young children are involved in producing this syndrome. It is this specific subgroup of pediatric spinal cord trauma that this paper wishes to address.

Clinical Material and Type of Injury

Definition of the Series

The pediatric syndrome of SCIWORA is reserved for those children with objective signs of myelopathy as a result of trauma, whose plain films of the spine, tomography, and occasionally myelography carried out at the time of admission showed no evidence of skeletal injury or subluxation. We have excluded all birth injuries and children with cord injuries caused by electric shock, penetrating agents, or missiles. Since we were primarily interested in the vulnerability of the normal spine in the young to violence, we have also eliminated all congenital malformations associated with inherent instability of the spine, such as insufficiency of the transverse odontoid ligament, os odontoideum, ossiculum terminale, the Klippel-Feil syndrome, Down's syndrome, and occipitalization of the atlas.

Age and Sex of Patients

From 1960 to 1980, 36 children with closed spinal cord injuries were treated at CHP. Twelve children had radiographic evidence of fracture or fracture-subluxation, and 24 belonged to the SCIWORA group. Thus, the SCIWORA group constituted 66.7% of the total.

The age range varied from 6 months to 16 years, with a mean of 7.2 years. Fourteen children were younger than 8 years (58.3%), and 10 were aged from 8 to 16 years (41.7%). There were 10 males and 14 females. The follow-up period ranged from 8 months to 20 years.

Cause of Injury

The most common causes of injury were related to vehicular accidents (Table 1). Four children were in the front seat and one in the back seat during collisions. One 7-year-old child was thrown from the back seat of a motor cycle. The four children hit by automobile were all under 6 years of age. A 16-month-old child was run over on the chest by his father's truck as it was being backed out from the garage.

The next most common causes involved falls from height: one from a crib, one in the gymnasium, and three from trees. Most of these children were under 8 years old. Two toddlers fell down steps at home by escaping the confines of their walkers. The other isolated causes were mainly sports-related, and all involved older children. There was one case of child abuse.

Mechanism of Injury

In most cases, the mechanism of neural injury could be deduced from associated bone and soft-tissue injuries (Table 2). Chin laceration, mandibular fracture, facial injuries, and frontal fracture or bruising usually implied hyperextension; conversely, an occipital bruise, laceration, or fracture pointed to a flexion-type injury. These clues were particularly important in infants and young children, and in those children whose concomitant concussion or traumatic amnesia had rendered a coherent history impossible. In some cases involving older children, surface clues were corroborated by good descriptions from the patient or an eye witness. A hyperextension sprain was almost certainly involved when an 11-year-old child suffered a cervical cord injury while doing backward somersaults. A flexion mechanism was suspected in a 9-year-old boy who described the onset of symptoms occurring when his neck was forced into extreme flexion by an occipital blow during a wrestling match.

A 1-year-old victim of child abuse had subhyaloid hemorrhages, subarachnoid hemorrhage, and diffuse cerebral swelling in addition to a partial C-5 myelopathy. The known association of a whiplash-shake type of violence with this form of cerebral lesion suggested that a combination of repetitive hyperextension and flexion forces caused this child's spinal cord damage.

Two children had flexion-compression injuries to their cervical spine; one as a result of diving into

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Cause of injury in 24 children</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cause of Injury</td>
<td>Cases</td>
</tr>
<tr>
<td>hit by car</td>
<td>4</td>
</tr>
<tr>
<td>run over by car (chest)</td>
<td>1</td>
</tr>
<tr>
<td>automobile accident</td>
<td>5</td>
</tr>
<tr>
<td>motorcycle accident</td>
<td>1</td>
</tr>
<tr>
<td>fall from height</td>
<td>5</td>
</tr>
<tr>
<td>fall down steps</td>
<td>2</td>
</tr>
<tr>
<td>football tackle</td>
<td>1</td>
</tr>
<tr>
<td>diving</td>
<td>1</td>
</tr>
<tr>
<td>object fell on head</td>
<td>1</td>
</tr>
<tr>
<td>sled accident</td>
<td>1</td>
</tr>
<tr>
<td>wrestling</td>
<td>1</td>
</tr>
<tr>
<td>child abuse</td>
<td>1</td>
</tr>
</tbody>
</table>
TABLE 2

Mechanism of injury and diagnostic clues in 24 children

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>Mechanism</th>
<th>Diagnostic Clues</th>
</tr>
</thead>
<tbody>
<tr>
<td>2½</td>
<td>hyperextension</td>
<td>depressed frontal fracture</td>
</tr>
<tr>
<td>3*</td>
<td>hyperextension</td>
<td>chin laceration; mandibular fracture</td>
</tr>
<tr>
<td>3</td>
<td>hyperextension</td>
<td>frontal laceration</td>
</tr>
<tr>
<td>8½</td>
<td>hyperextension</td>
<td>forehead bruise</td>
</tr>
<tr>
<td>10</td>
<td>hyperextension</td>
<td>patient’s description</td>
</tr>
<tr>
<td>11</td>
<td>hyperextension</td>
<td>backward somersaulting</td>
</tr>
<tr>
<td>14</td>
<td>hyperextension</td>
<td>patient’s description (football tackle)</td>
</tr>
<tr>
<td>15</td>
<td>hyperextension</td>
<td>forehead &amp; facial lacerations</td>
</tr>
<tr>
<td>15*</td>
<td>hyperextension</td>
<td>mandibular fracture; anterior neck lacerations; chin abrasion</td>
</tr>
<tr>
<td>16</td>
<td>hyperextension</td>
<td>rt frontal, facial lacerations</td>
</tr>
<tr>
<td>11*</td>
<td>flexion</td>
<td>mother’s description (fell down steps)</td>
</tr>
<tr>
<td>2</td>
<td>flexion</td>
<td>occipital bruise</td>
</tr>
<tr>
<td>2½</td>
<td>flexion</td>
<td>occipital bruise</td>
</tr>
<tr>
<td>4</td>
<td>flexion</td>
<td>occipital bruise</td>
</tr>
<tr>
<td>4</td>
<td>flexion</td>
<td>occipital bruise</td>
</tr>
<tr>
<td>6*</td>
<td>flexion</td>
<td>occipital bruise</td>
</tr>
<tr>
<td>7*</td>
<td>flexion</td>
<td>occipital laceration &amp; fracture</td>
</tr>
<tr>
<td>9</td>
<td>flexion</td>
<td>struck on occiput while wrestling</td>
</tr>
<tr>
<td>1½</td>
<td>repetitive flexion &amp; extension</td>
<td>child abuse: shake &amp; whiplash injury</td>
</tr>
<tr>
<td>8</td>
<td>flexion-compression</td>
<td>hit vertex while diving</td>
</tr>
<tr>
<td>10</td>
<td>flexion-compression</td>
<td>heavy object fell on vertex</td>
</tr>
<tr>
<td>4</td>
<td>longitudinal distraction</td>
<td>head caught by tow chain, dragged</td>
</tr>
<tr>
<td>5*</td>
<td>longitudinal distraction</td>
<td>lap seat belt injury; associated L2-3 transverse fracture</td>
</tr>
<tr>
<td>1½</td>
<td>direct crush injury ? hyper-extension</td>
<td>run over by truck, lying on abdomen; tire marks</td>
</tr>
</tbody>
</table>

* Severe hypotension (systolic pressure < 60 torr) present at admission.

shallow water, and one from a direct vertex hit from a falling object. Both had signs of soft-tissue bruising in the vertex.

A 5-year-old child was strapped to the front passenger seat by a lap seat belt during a head-on collision. He suffered a transverse body fracture of the L-2 vertebral body (Chance fracture) and a complete midthoracic transverse myelopathy. Since a flexion-distraction force of great magnitude is necessary to produce the type of lumbar fracture in question, the same force vector may account for his thoracic cord injury. Similar distraction forces were likely responsible for the cervical cord injury of a 4-year-old child whose head was caught by a tow chain which dragged his body a considerable distance.

There was one case of direct crush injury to the thoracic spine in a 16-month-old child who was run over by a truck while lying on his abdomen, evidenced by fresh tire markings on his back. The forces involved would tend to cause a backward bending on the spine.

Flexion is as frequently implicated as hyperextension as the mechanism of injury (Table 2). However, if the mechanism of injury is correlated with age, flexion and hyperextension appear to involve two different age groups. Of the eight children who sustained flexion injury, seven were younger than 8 years. Conversely, seven of the 10 with hyperextension injury were older children. Both cases of longitudinal distraction and the one case of direct crush injury to the thoracic spine occurred in very young children.

Table 2 also indicates six instances where severe hypotension (systolic pressure less than 60 torr) was present at the time of admission. In all six cases, this was due to blood loss from thoracoabdominal injuries, multiple long-bone fractures, or soft-tissue lacerations.

Associated Extraneural Injuries

Three children had intra-abdominal hemorrhage caused, respectively, by a mesenteric tear, a ruptured spleen, and a liver laceration; all three had significant hypotension at the time of admission. One child was in hypovolemic shock because of a severe oropharyngeal tear and another because of a massive avulsion of the occipital scalp over a depressed fracture.

Four patients had skull fractures: two frontal, one occipital, and one basilar. Both children with frontal fractures also had associated mandibular fractures. Two children had femoral-tibial fractures and one had pelvic fractures.

Neurological Status

Level and Type of Neurological Lesions

Five lesions involved the upper cervical cord (C1-4), 15 involved the lower cervical cord (C-5 and C-8), and four involved the thoracic cord.

Neurological examination at the time of admission revealed four individual syndromes: complete physiological cord transection, central cord syndrome, Brown-Séquard syndrome, and partial cord syndrome (Table 3). The latter category included those patients with partial preservation of function below the level of the lesion, but whose pattern of neurological deficits could not be classified as either central cord or anterior cord syndromes.

There were seven cases of complete transection. Four of the ten patients with central cord syndrome were classified as severe because of profound weakness of hand grip and forearm musculature and sufficient weakness of the lower extremities to prevent ambulation. The other six children had a mild central cord syndrome, and either remained ambulatory or showed rapid improvement in lower extremity functions within the first 24 hours. The patient with Brown-Séquard syndrome had a hemihypalgesia and mild weakness in the contralateral arm, and a corresponding Babinski response. There were three patients each with severe and mild partial cord syndrome. In all, 14 children (58.3%) either sustained complete physiological transection or serious damage to their spinal cord.
Table 3
Types of neurological syndromes in 24 children

<table>
<thead>
<tr>
<th>Neurological Syndromes</th>
<th>No. of Cases</th>
<th>Percent of Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>complete cord transection</td>
<td>7</td>
<td>29.1</td>
</tr>
<tr>
<td>central cord, severe</td>
<td>4</td>
<td>16.7</td>
</tr>
<tr>
<td>central cord, mild</td>
<td>6</td>
<td>25.0</td>
</tr>
<tr>
<td>partial cord, severe</td>
<td>3</td>
<td>12.5</td>
</tr>
<tr>
<td>partial cord, mild</td>
<td>3</td>
<td>12.5</td>
</tr>
<tr>
<td>Brown-Séquard</td>
<td>1</td>
<td>4.2</td>
</tr>
</tbody>
</table>

Table 4
Correlation between level and severity of neurological injury

<table>
<thead>
<tr>
<th>Level of Injury</th>
<th>No. of Cases</th>
<th>Severity of Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1-4</td>
<td>5</td>
<td>Complete: 3, Severe: 2, Mild: 0</td>
</tr>
<tr>
<td>C5-8</td>
<td>15</td>
<td>Complete: 1, Severe: 4, Mild: 10</td>
</tr>
<tr>
<td>T1-6</td>
<td>4</td>
<td>Complete: 3, Severe: 1, Mild: 0</td>
</tr>
</tbody>
</table>

Level Related to Severity of Neurological Injury

The upper cervical cord and thoracic cord were equally prone to serious injuries (Table 4). Six of the seven complete transections in the series occurred in these two regions. On the other hand, the lower cervical spine appeared to be relatively resistant to injury. Only one case of complete transection and four of the severe cord syndromes involved the C5–8 segments, and 10 of the 15 injuries to this region were mild or moderate.

Age Related to Neurological Status

Figure 1 suggests that the spines in infants and young children were more vulnerable to deforming forces than the spines of older children. Children aged from 6 months to 8 years suffered much more devastating neurological injuries: seven of the 14 younger children had complete transections, and six had either a severe central cord syndrome or a severe partial cord syndrome. Only one child in this age group who suffered a flexion injury to the cervical spine escaped serious cord damage. Conversely, all but one of the children aged 8 to 16 years had mild to moderate neurological damage. The one exception was an 11-year-old boy with a nearly complete paraplegia at T-5 following a violent extension injury.

The same age-related difference exists within the subgroup of the 10 cases of central cord syndrome. The four severe cases were 4 years old or younger, and the six mild cases were older than 8 years.

Figure 2 shows the age distribution in relation to the level of neurological injury. All five cases of upper cervical cord damage occurred in the younger group, whereas injuries to the lower cervical segments were evenly distributed in the entire age span of the series.

Mechanism of Injury Related to Severity and Level of Neurological Lesion

Table 5 attempts to define the relationship between the types of deforming forces to the spine and the severity of the resultant neurological damage. Flexion forces to the spine appear to produce more serious neural injuries than extension forces. One explanation is that the pediatric spine is more resistant to extension forces than to flexion forces. However, if the ages of these patients were taken into consideration, it is clear that the three cases of complete cord transection due to hyperextension and the six cases of severe to complete cord injury due to flexion all occurred in children under 6 years. Thus, an alternative explanation may again be related to the aforementioned suggestion that the spines in infants and young children are inherently more susceptible to injury than those of older children.
more deformable and, therefore, provide less effective protection for the subjacent spinal cord regardless of whether flexion or extension forces were involved. Certainly, within the subgroup of patients subjected to hyperextension, the younger patients all had worse neural damage than the older ones (Fig. 1).

Repetitive flexion-extension as seen in the whiplash-shake type of child abuse, longitudinal distraction of the spine, and direct crush injury were all associated with severe neural damage (Table 5). However, it must be remembered that these injuries all involved extremely violent forces and were also inflicted on infants and young children. Both patients who suffered vertical loading to their vertebrae had relatively mild neural lesions.

Table 6 relates the mechanism of injury to the level of the neurological lesion. It appears that the upper and lower halves of the cervical cord are susceptible to different types of deforming forces. All five cases of upper cervical cord injuries in the series were caused by flexion forces, while the predominant mechanism injuring the lower four cervical segments was hyperextension. Again, if one includes the age factor in the analysis, it is apparent that if flexion forces are applied to the very young spine, the upper four segments of the cord are most likely to be injured; but in children older than 8 years, both flexion and extension forces are more likely to injure the lower cord segments.

Table 6 also shows that thoracic cord injury can be caused by several different mechanisms. It is probable that what determines the severity of thoracic cord injury is the magnitude and not the direction of the deforming vector.

**Delayed Onset of Neurological Signs**

Thirteen patients in this series (54%) had delayed onset of their neurological deficits following spinal trauma (Table 7). The time interval between injury and the appearance of objective sensorimotor dysfunction, the "latent period," ranged from 30 minutes to 4 days, with a mean of 1.2 days. There was no uniformity in either the age or mechanism of injury in this subgroup. The age range varied from 6 months to 15 years, and the mechanisms included six cases of hyperextension, three of flexion, two of flexion-compression, one of longitudinal distraction, and one of direct crush injury to the thoracic spine.

Immediately following injury, the child in Case 1 was noted to have transient clumsiness in his extremities which disappeared within minutes; he became profoundly quadriparetic 2 days later. Seven other children recalled transient neurological symptoms that were initially ignored by the patients and often by their physicians. Four children (Cases 2, 6, 7, and 8) had paresthesia in all extremities lasting from 5 minutes to 1 hour. A 10-year-old girl who suffered a flexion-compression injury from diving into shallow water (Case 5) had transient tingling in both upper extremities and had a subjective feeling of "total body paralysis," although no objective deficits were found. She developed a severe central cord syndrome 4 hours later. In Case 3, the child described a similar feeling of subjective paralysis, was examined by a neurosurgeon, found to be normal, and sent home. He was well for 2 days but, while playing basketball 48 hours following his injury, he rapidly developed arm and hand weakness and paresthesia in both legs. The child in Case 4 recalled a tingling numbness in his hands and a lightning sensation shooting down both legs, together with a subjective "heaviness" in the lower part of his body. These symptoms cleared within 15 minutes, but 24 hours later he presented with a typical C-5 central cord syndrome. During his 2-day hospital stay, his weakness improved dramatically, and he was sent home with a stiff cervical collar. Two days later, he removed his collar while playing and promptly noticed symptoms caused by a recurrence of his myelopathy.

Although these patients were well during the "latent period," sensorimotor paralysis progressed inexorably once it began, evolving into complete transverse myelopathy in two cases, severe partial cord syndrome in four, and mild to moderate central cord syndrome in seven children.

**Radiographic Diagnosis**

The radiographic tests performed are summarized in Table 8. All patients had plain films of the spine taken on admission. The entire spine was studied in...
TABLE 7

Delayed neurological signs in 13 children

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Mechanism of Injury</th>
<th>Initial Transient Symptoms</th>
<th>“Latent Period”</th>
<th>Neurological Manifestations</th>
<th>Myelography</th>
<th>Final Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>flexion</td>
<td>mother noted child not moving limbs immediately after injury; cleared quickly</td>
<td>2 days</td>
<td>severe C-6 central cord</td>
<td>normal</td>
<td>severe deficits</td>
</tr>
<tr>
<td>2</td>
<td>4</td>
<td>longitudinal dis-traction extension</td>
<td>paresthesia, both arms &amp; legs; subjective feeling of paralysis</td>
<td>2 days</td>
<td>severe C-5 central cord</td>
<td>normal</td>
<td>severe deficits</td>
</tr>
<tr>
<td>3</td>
<td>8½</td>
<td>extension</td>
<td>paresthesia, both hands; subjective feeling of paralysis</td>
<td>24 hrs</td>
<td>mild C-5 central cord</td>
<td>normal</td>
<td>severe deficits</td>
</tr>
<tr>
<td>4</td>
<td>10</td>
<td>flexion-compres-sion</td>
<td>lightning sensation; paresthesia, both hands</td>
<td>4 hrs</td>
<td>mild C-5 central cord</td>
<td>normal</td>
<td>mild deficits</td>
</tr>
<tr>
<td>5</td>
<td>10</td>
<td>flexion-compres-sion</td>
<td>paresthesia both hands, subjective weakness</td>
<td>12 hrs</td>
<td>severe T-5 partial cord</td>
<td>normal</td>
<td>severe deficits</td>
</tr>
<tr>
<td>6</td>
<td>11</td>
<td>extension</td>
<td>paresthesia, both legs</td>
<td>12 hrs</td>
<td>mild central cord</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>7</td>
<td>14</td>
<td>extension</td>
<td>paresthesia, both hands &amp; legs</td>
<td>6 hrs</td>
<td>mild C-6 central cord</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>8</td>
<td>15</td>
<td>extension</td>
<td>paresthesia, both hands</td>
<td>4 days</td>
<td>severe C-3 partial cord</td>
<td>normal</td>
<td>moderate deficits</td>
</tr>
<tr>
<td>9</td>
<td>½</td>
<td>flexion</td>
<td>paresthesia, both hands</td>
<td>24 hrs</td>
<td>T-6 complete cord transection</td>
<td>normal</td>
<td>complete cord transection</td>
</tr>
<tr>
<td>10</td>
<td>1½</td>
<td>direct crush injury</td>
<td></td>
<td>4 days</td>
<td>C-7 complete cord transection</td>
<td>normal</td>
<td>complete cord transection mild deficits</td>
</tr>
<tr>
<td>11</td>
<td>2½</td>
<td>extension</td>
<td></td>
<td>30 min</td>
<td>mild C-5 central cord</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>12</td>
<td>9</td>
<td>flexion</td>
<td></td>
<td>24 hrs</td>
<td>mild C-5 central cord</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>13</td>
<td>16</td>
<td>extension</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

TABLE 8

Radiographic tests performed in 24 patients

<table>
<thead>
<tr>
<th>Radiography</th>
<th>Cases</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>plain spine films</td>
<td>24</td>
<td>100</td>
</tr>
<tr>
<td>tomography</td>
<td>20</td>
<td>83.3</td>
</tr>
<tr>
<td>myelography</td>
<td>12</td>
<td>50</td>
</tr>
<tr>
<td>dynamic studies</td>
<td></td>
<td></td>
</tr>
<tr>
<td>acute</td>
<td>18</td>
<td>75</td>
</tr>
<tr>
<td>delayed</td>
<td>24</td>
<td>100</td>
</tr>
<tr>
<td>computerized tomography of spine</td>
<td>1</td>
<td>4.2</td>
</tr>
</tbody>
</table>

those patients with multisystem trauma, but only the symptomatic area was studied in those with isolated neurological syndromes. All but one patient had normal studies. This child sustained a lap seat belt injury and had a transverse fracture of the L-2 vertebral body (Chance fracture). However, her level of neural injury was at T-6 and her thoracic spine film was normal.

Tomographies obtained in 83.3% of the patients were normal. Myelography with either Pantopaque or gas was performed on 50% of the patients to rule out a subarachnoid block, usually in the wake of normal plain films and tomography. Free flow of contrast medium past the region of neurological lesion was demonstrated in each case, although some cords looked slightly swollen with irregular contour.

Computerized tomography (CT) of the spine was performed on one patient who suffered a mild central cord syndrome following a hyperextension injury. Transverse imaging through the C-5 level disclosed a small hyperdense area compatible with an epidural hematoma. No cord compression or bone abnormality was appreciated. Repeat CT 2 weeks later was completely normal.

Dynamic films with the patient's neck in voluntary flexion and extension were obtained in 75% of the children after plain films in the neutral position were found to be normal. No acute instability was demonstrated, but in most cases, paraspinal muscle spasm severely restricted the range of motion and rendered these studies technically inadequate. Delayed dynamic studies were performed on all patients several days after admission, at a time when the neurological status had stabilized or after muscle spasm had subsided. Instability was demonstrated in only one patient who showed mild anterior slipping of C-4 and C-5. This patient had severe spasm during the 1st week of admission and had inadequate acute flexion-extension studies.

Treatment and Outcome

Management

All patients with cervical cord syndromes had immediate neck immobilization with cervical collars. Those with thoracic cord injuries were placed supine on a fracture board before other resuscitative measures were instituted.
The six patients in hypovolemic shock were treated with vigorous fluid and blood replacement to avoid persistent systemic hypotension. Emergency tracheostomies were performed on the five patients with upper cervical cord injuries and mechanical ventilation was begun. Corticosteroids were routinely used on all patients. In-dwelling bladder catheters were inserted into those patients with complete or severe partial cord syndromes during the acute phase of injury, and intermittent catheterization programs were set up for those infants and young children who required long-term assistance in bladder drainage.

Intracranial pressure monitors were used on two children with severe concomitant head injuries. They were subsequently managed with our intracranial hypotension protocol in the intensive care unit (ICU). Three children had laparotomies for intra-abdominal hemorrhage, and two other children had repair of the oropharynx and an occipital scalp avulsion, respectively. All long-bone fractures were managed initially with skeletal traction.

Following the radiographic establishment of the SCIWORA syndrome, all thoracic cord injuries were treated with bed rest for 1 week; stability was confirmed by follow-up films of the thoracic spine, and the patients were mobilized. One patient with a transverse lumbar fracture had posterior Harrington rod fusion. Rehabilitative measures began immediately following admission to the hospital.

Patients with high cervical cord lesions required prolonged mechanical ventilation and ICU care. They were kept in cervical collars for 1 month, had biweekly cervical spine films with delayed flexion-extension views to rule out subtle instability, and received early rehabilitative intervention. Those with severe central and partial lesions of the lower cervical cord were similarly immobilized with a cervical collar while on the neurological unit, and those without late instability were transferred to the rehabilitation unit after 1 week of bed rest. The child with the C-4 subluxation found on delayed flexion-extension studies was immobilized with the halo apparatus for 8 weeks and subsequently achieved stability. Patients with mild injuries who remained ambulatory or showed substantial clinical improvement within the first 48 hours were allowed ward privileges in the 1st week and discharged home on the following week wearing their cervical collar. This was kept on for 4 weeks, at which time a second set of dynamic films was always obtained to ensure stability.

Complications

The two most common complications during the acute treatment period were frequent respiratory and urinary tract infections. There was one case of deep vein thrombosis in a child with a complete C-6 transection.

Late complications included distressing involuntary muscle spasm below the level of the lesion and serious psychological maladjustment in the severely disabled children. No cases of delayed spinal deformity were encountered in this series.

Outcome

For children with SCIWORA, the long-term prognosis is poor (Table 9). One child with a complete cord syndrome died. This 6-year-old boy was hit by a car and sustained massive thoracoabdominal injuries. He presented with profound hypovolemic shock, a complete C-2 cord transection, and cardiorespiratory arrest. Despite heroic efforts at resuscitation, he died of progressive respiratory failure 4 days later.

The six remaining patients with complete cord syndromes remained unchanged neurologically. Two of these children with upper cervical cord injuries required long-term mechanical ventilation. One child with a C-6 complete cord syndrome spent 2 years in a rehabilitation institute but could not attain the level of self-care, partly because of troublesome muscle spasms and frequent respiratory complications.

The three children with initially complete thoracic cord transection made no neurological recovery but proved to be far better rehabilitation candidates than patients with severe cervical injuries. Five of the seven children with severe cord syndromes continue to have severe deficits. Since most of these had cervical cord injuries, they represent a severely handicapped group. Only two children in this group made a substantial recovery to the point of being ambulatory with prostheses.

The 10 children with initially mild to moderate neural damage represent the only optimistic group in the series. Seven were neurologically normal 3 months to 7 years following initial hospitalization. The three others have residual deficits but are enjoying a full psychosocial lifestyle.

It appears from these figures that the most important factor determining prognosis is the initial neurological status. Twelve of 14 children (85.7%) with initially complete or severe cord syndromes either are dead or continue to be totally disabled. Only two children (14.2%) from this group made satisfactory progress. On the other hand, seven of the 10 patients (70%) with mild to moderate initial damage to the cord have made complete recovery, and the rest are only minimally disabled.

Discussion

Because of the rarity of pediatric spinal cord injuries, comprehensive clinical and epidemiological data concerning the syndrome of spinal cord injury without radiographic abnormality (SCIWORA) are not found in the literature. However, the phenomenon of SCIWORA in children has been noted by several investigators. In 1969, Audic and Maury stated that in 21 children under the age of 16 years with spinal injuries, comprehensive clinical and epidemiological data concerning the syndrome of spinal cord injury without radiographic abnormality (SCIWORA) are not found in the literature. However, the phenomenon of SCIWORA in children has been noted by several investigators. In 1969, Audic and Maury stated that in 21 children under the age of 16 years with spinal...
Cord injury with normal radiography

<table>
<thead>
<tr>
<th>Initial Neurological Status</th>
<th>No. of Cases</th>
<th>Final Neurological Status</th>
<th>Death</th>
<th>Complete Cord Syndrome</th>
<th>Severe Deficits</th>
<th>Mild/Moderate Deficits</th>
<th>Normal</th>
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<td></td>
<td>3</td>
<td>7</td>
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cord trauma, "very often" no fracture was detected. In the same year, Melzak reported 16 similar cases among 29 children with spinal cord injury. The numbers of children with SCIWORA in other series of pediatric spinal cord trauma are as follows: Burke found 12 out of 24; Hasue, et al., one out of 10; Hachen eight out of 18; Andrews and Jung seven out of 15; Anderson and Schutt two out of 42; and Kewalramani, et al., five out of 25 children. Unfortunately, these data were obtained from records of rehabilitation facilities and lack detailed clinical information concerning the individual patients.

Some insight into this problem was provided by four additional articles which documented the clinical courses of 13 children with traumatic myelopathy without fracture or dislocation. Our own figure of 24 cases of SCIWORA among 36 children with spinal cord injury convinces us that this is a common entity in pediatric spinal trauma. Based on others' experience and the information gathered from the present study, aided by the current knowledge regarding the anatomical peculiarities of the pediatric spine and the biomechanics of spinal cord injury in general, we examined the mechanism of injury, the mode of presentation, the neurological lesions, the management, and the prognosis of this syndrome.

Mechanisms of Neural Damage Without Fracture or Dislocation

Most of the existing knowledge on the biomechanics of spinal injury is based on studies of the adult spine. Some of this work has been inspired by a need to explain the well known phenomenon of acute central cord syndrome in the elderly without evidence of vertebral fracture or dislocation. Our own figure of 24 cases of SCIWORA among 36 children with spinal cord injury convinces us that this is a common entity in pediatric spinal trauma. Based on others' experience and the information gathered from the present study, aided by the current knowledge regarding the anatomical peculiarities of the pediatric spine and the biomechanics of spinal cord injury in general, we examined the mechanism of injury, the mode of presentation, the neurological lesions, the management, and the prognosis of this syndrome.

Hypercextension Injuries

Based on cadaver study, Taylor in 1951 demonstrated that the interlaminar ligaments of the cervical spine bulged forward into the central canal during hyperextension. Hyperextension was therefore thought to be the cause of the acute central cord syndrome often seen in the elderly without evidence of fracture. This theory was supported by Alexander, et al., who found that the sagittal diameter of the cervical canal could be narrowed by over 50% during extension. They claimed that if preexisting spondylotic protrusions were present to further narrow the canal at C4–6 to less than 13 mm during hyperextension, the spinal cord would be pinched between the osteophytes and the inward bulging interlaminar ligaments. An additional aggravating factor was later found by Breig and El-Nadi to be a shortening and, therefore, thickening of the cord during hyperextension as the spinal column shortens. This would accentuate the crowding of the intraspinal contents.

In one case of hyperextension injury involving extreme violence, Taylor and Blackwood found at necropsy that the anterior longitudinal ligament had ruptured, the intervertebral disc had detached from the lower vertebral body, and the segment of the cervical column above this disc had become displaced backward to compress the cord. However, elastic recoil of the paraspinal muscles resulted in spontaneous reduction of the displacement and gave a normal radiographic appearance. They postulated that with hyperextension sprain exceeding the tensile resistance of the anterior longitudinal ligament, this structure, unattached to and hence unsupported by the anulus, will rupture and permit retrolisthesis of the upper segments. Anatomical plausibility of this mechanism was provided by Bourmer, who showed on five cadavers that if the anterior longitudinal ligament was severed and if a backward displacing force was directed at the head, the intervertebral disc promptly ruptured to allow for retrolisthesis of the body above. Radiographic confirmation of the Taylor-Blackwood mechanism came in 1974 when Marar showed subtle retrolisthesis in 11 of 45 patients suffering from quadriplegia due to hyperextension. Using postmortem stress radiography on two other patients who succumbed to this injury, he demonstrated opening of the disc space and backward displacement of the upper body. These tissue injuries were later confirmed at autopsy.
Thus, it may be assumed that with a moderate degree of hyperextension, the cervical cord can be compressed by the inward bulging of the interlaminar ligaments against preexisting spondylosis or congenital stenosis, aggravated by a thickening of its cross section area during its simultaneous shortening. Exceptionally violent hyperextension will rupture the anterior longitudinal ligament, detach the disc, and cause a retrodisplacement of the upper body. Immediate muscle action causes elastic recoil and spontaneous reduction, and reflex muscle spasm maintains the relatively stable reduction to give the normal radiographic appearance.16,18,37,61,62

Several anatomical characteristics of the pediatric spine increase its susceptibility to hyperextension injury. In children, the ligaments, posterior joint capsules, and cartilaginous structures are more elastic than the rigid adult spine and, therefore, more deformable.9,30,69,74,76,78 The planes of the facet joints in children are also more horizontal and thus possess greater mobility but less stability.9,30,69,76,78 In addition, Aufdermaur7 found that in the young spine, a locus of weakness exists within the growth zone of the cartilaginous end plate at its junction with the primary centrum, where the fibrous lamellae are loosely arranged, being segregated by columns of cartilage cells. Separation of the end plate from the centrum readily occurs at this region with even a moderate degree of shearing, as evidenced in 12 autopsy studies. Two patients described by Cheshire31 and one by Ahmann, et al.,2 probably had hyperextension. Hyperextension is implicated in 10 children in the present series. Scher44 showed that hyperextension was maximum at the C5–6 junction, and suggested that hyperextension injury to the cord most likely occurred in this area. It is noteworthy that in all our eight cases of hyperextension of the cervical spine, the lower cervical cord segments were indeed involved.

**Flexion Injuries**

In 1907, Lloyd44 was the first to suggest flexion instead of extension as the principal mechanism involved in cord compression without vertebral fracture or deformity. In his “flexion-recoil” theory, he reasoned that after forward displacement, producing the neural injury, the upper cervical segment sprang back as a result of muscle action to give a normal appearance on x-ray films. Barnes in 194841 categorically refuted Lloyd’s flexion-recoil theory: his studies on adult cadavers showed that for flexion dislocation to damage the cord, there would have to be either fracture of the facet joints or unilateral locking of at least one facet, which would make the forward displacement irreducible spontaneously. In either case, the x-ray film would be abnormal.

Since Barnes’ report, a number of researchers have noted flexion as the culpable mechanism in cases of pediatric spinal cord injury without vertebral damage. The four cases of Burke,24 two cases of Glasauer and Cares,41,42 and one case of Cheshire31 all had complete cord transection following a flexion injury. Moreover, Teng and Papatheodorou,79 Dunlap, et al.,34 and Papavasiliou60 have all described the syndrome of traumatic flexion subluxation of the cervical spine during childhood with normal radiography. Although Barnes’ refutation of Lloyd’s “flexion-recoil” theory might be entirely justified in the adult, it is conceivable that flexion dislocation could spontaneously reduce itself without facet fracture or locking in the highly supple and mobile spine of a child.

The following anatomical features in the pediatric cervical spine account for its increased physiologic mobility as well as its susceptibility to flexion injury:

1) The interspinous ligaments, posterior joint capsule, and cartilaginous end plates are elastic and often redundant.9,59,76
2) The articulating surfaces of the facet joints are more horizontally oriented.9,30,76,78
3) The anterior portion of the vertebral bodies is wedged forward so that anterior slipping between adjacent bodies is facilitated.9,69
4) In the mature spine, the two uncinate processes project upward and outward to articulate with the corresponding lower borders of the body above at the uncovertebral joints. The characteristic orientation of the fully developed uncinate processes normally limits lateral and rotational movements between adjacent bodies. In infants and children under 10 years, the uncinate processes are flat and therefore ineffective in withstand flexion-rotation forces.19,74
5) The proportionately heavy head and relatively underdeveloped musculature of the infant neck constitute an unusual susceptibility for flexion-extension injuries.27,28,68,75

Large-scale anatomical studies of normal children reveal that the horizontal orientation of the facet joints as well as the anterior wedging of the vertebral bodies is much more prominent in the upper three to four segments of the cervical spine.9,69 Also, according to Townsend and Rowe,76 Baker and Berdon,10 and Braakman and Penning,18 the fulcrum for maximum flexion in young children is at C2–3 and C3–4, whereas the fulcrum for maximum flexion in the adult is at C5–6. It is therefore not surprising that the upper cervical segments in children display the greatest physiological mobility.30,76 and that flexion subluxation in infants and younger children most often involves the upper segments of the cervical cord.47,69,73,74

In the present series, eight children had flexion compression of the cord and in five the cord segments injured were between C-1 and C-4. In only three children was the lower cervical cord involved.

According to Bailey9 and von Torklus and Gehle,78 the characteristic anatomical features of the young spine gradually approach adult status by 8 years of age. Thus, the anterior wedging of the bodies disappears, the articulating planes of the facets become more vertical, the uncinate processes gain height, and the ligaments and capsules increase in tensile strength. It follows that the cervical spine, especially its upper
Cord injury with normal radiography

portion, is at maximum risk for forward slipping against flexion forces during the first few years of life. As the child develops beyond 8 years, the cervical spine gains resistance against flexion insults, and by 16 to 18 years, when the adult status is reached, flexion forces will more likely produce the well known syndrome of fracture or fracture-subluxation. This is, indeed, borne out by our data: seven out of eight cases of flexion injuries involved children under 8 years (Table 2), with five involving the C1–4 segments (Table 6). Cord injuries due to flexion are also more serious in the younger children, again reflecting the inherent instability of the spine at this age: the six children with severe or complete cord syndromes were all under 8 years of age (Table 5 and Fig. 1).

The case of the abused child with severe central cord syndrome represents a special instance of repetitive flexion and hyperextension injury. During the paroxysms of shaking in the anteroposterior plane, the heavy infantile head supported poorly by weak neck muscles courses through a two-phase cycle of rapid, repetitive flexion of the head until the chin strikes the anterior chest, alternating with extensions of the head until the occiput strikes the back. Although the elastic spinal column escapes fracture-dislocation, the underlying cord necessarily suffers multiple battering.

**Longitudinal Distraction Injuries**

In 1974, Burke described autopsy findings of a constricted segment of the cervical cord 4 cm in length in an 11-month-old infant. In another infant with a T-4 paraplegia, he found during laminectomy a similarly narrowed and elongated segment of traumatized cord unlike the usual discrete, short segment seen after compression injury. Glasauer and Cares reported similar findings in two other infants, one at autopsy and the other at surgery. They postulated that longitudinal distraction rather than flexion or extension-compression was the mechanism involved in these infants.

In this context, Leventhal’s cadaver study of the longitudinal compliance of the neonatal spine is most revealing. He found that the elastic spinal column of the neonate can be stretched 2 in. without signs of structural disruption, but the spinal cord, devoid of elastic elements, can only stretch 1/2 in. before rupturing. During a forceful breech extraction, the spinal cord can be ruptured by longitudinal distraction, along with its investing dura and leptomeninges, whereas the vertebral column will remain completely intact. None of Leventhal’s six cases had any evidence of radiographic abnormalities.

It is reasonable to assume that the younger the victim, and therefore, the more elastic the spine, the more serious the myelopathy will be with longitudinal distraction. In our series, there were two cases of longitudinal distraction, both involving very young children. Both received serious damage to the cord caused by extremely violent forces; one had a lower cervical cord injury and the other a complete lesion at T-3.

**Ischemic Injuries**

At least two cases of spinal cord infarction following minor trauma in children can be found in the literature. Ahmann, et al., described the autopsy findings in a 4-year-old child who sustained a relatively trivial hyperextension injury to the neck and who presented with a high cervical cord lesion of delayed onset. Bilateral gray matter infarction was found from C-3 to T-2. A 22-month-old child, also with a delayed cervical cord lesion following minor hyperextension, was also found to have infarction of the dorsolateral columns from the cervicomedullary junction to T-5. Both children had normal plain films and myelograms. Ahmann, et al., postulated that the vertebral arteries could have been temporarily occluded or thrown into spasm at the time of hyperextension, and longitudinal watershed infarction would result if collateral flow from thoracic and lower cervical medullary arteries was insufficient. He added that if surface vessels in the dorsal coronary plexus were compressed at multiple levels during hyperextension, infarction could occur within the ventrodorsal watershed area between terminal supplies from the dorsal plexus and the anterior sulcal arteries.

Gilles, et al., made a detailed anatomical study of the infantile atlanto-occipital junction that has shed much light on the subject of the vulnerability of the infantile vertebral arteries during trauma. They found the infantile atlanto-occipital joint to be inherently unstable. The small arch of C-1 resting against a large foramen magnum, the weak and redundant alar, apical, and atlanto-occipital ligaments, the elastic and lax joint capsules investing the occipital condyles, and the condyles’ own flattened surface, all contribute to an inherent instability in this region where flexion and extension customarily induce horizontal sliding movements. Moreover, the lateral mass of C-1 and the posterior portion of the occipital condyle, which normally form the protective groove for the vertebral artery as it curls behind the lateral mass to enter the skull, are both stunted in height. This exposes the artery to ready compression by the to-and-fro movements between the condyle and the C-1 arch during hyperextension. Gilles, et al., actually demonstrated bilateral occlusion of the vertebral arteries with the neck in extension at postmortem angiography. If this were an antemortem event, the upper cervical cord could be rendered ischemic.

A final important point concerning ischemic injury is that six patients in our series had severe hypotension on admission. A suboptimal perfusion pressure to the traumatized cord with impaired autoregulation of blood flow could have contributed to the final insult.
Other Hypotheses

In 1915, Holmes postulated that a direct blow to the vertebral column insufficient to cause skeletal damage or deformity could set up shock-wave oscillations in the subjacent cord at a frequency different from that in the column of bone, causing "slapping damage" to the cord against the bone wall of the central canal. This has never been substantiated.

Cramer and McGowan in 1944 suggested that during flexion, the intervertebral disc protrudes backward by means of a hydraulic piston-like action of the nucleus pulposus, which then spontaneously retracts back inside the anulus. We now know that traumatic disc extrusion with rupture of the anulus does occur rarely in adults but usually is associated with fracture of the adjacent vertebral bodies. The relevance of this to the pediatric syndrome of SCIWORA is uncertain, but myelography may be helpful in its exclusion in doubtful situations.

The Missed Occult Fracture

On rare occasions, the physician may be misled into making the diagnosis of SCIWORA if an occult fracture is missed on conventional radiography. This can be hazardous, for, as the physician is busy searching for esoteric mechanisms to explain the spinal cord injury in the absence of fracture-dislocation, the patient's spine remains dangerously unstable. Marar demonstrated in 12 autopsy cases that a horizontal fracture of the vertebral body below the pedicles can occur in the cervical spine with hyperextension. The anterior longitudinal ligament is always torn, but reflex muscle action may temporarily reduce this fracture so perfectly that visualization with conventional plain films may be impossible. None of these 12 patients had abnormal antemortem x-ray films. Reduction in such cases is temporary; instability will become obvious when reflex muscle spasm subsides. Vines encountered lateral mass fractures not extending to the facet surface missed by routine radiographic views. Thus, multiplane tomographic studies or CT should be employed to rule out these occult fractures.

The Neurological Lesions

It is obvious from our figures that the neurological lesion in the pediatric SCIWORA syndrome is more serious than the type of neurological lesions found in adult patients with spinal cord injury and normal x-ray films. Most authors agree that the mechanism for the adult patients is one of hyperextension superimposed on preexisting cervical spondylosis, and the neurological picture is predominantly that of acute central cord syndrome. Bedbrook mentioned only two cases of anterior cord syndrome, two cases of Brown-Séquard syndrome, and four cases of complete cord lesions in a group of 63 adults with hyperextension injury and normal radiography. This probably explains the generally favorable long-term prognosis for these patients, for the central cord syndrome is usually associated with good recovery, whereas complete cord transection and anterior cord syndromes have a more sinister prognosis. In our series of pediatric SCIWORA, seven children had complete transections and seven others had severe incomplete cord syndromes, representing an incidence of 58.3% with serious spinal cord damage. The data from others, although scanty, reflect the same distressing outlook for children with SCIWORA.

Although children with SCIWORA have worse prognoses than adult patients with hyperextension injury and normal radiography, they fare considerably better than those children whose spinal cord injuries are associated with fractures or dislocations. Twelve such children were treated at our institution from 1960 to 1980; two died, eight had complete transections, one had a severe incomplete lesion, and only one had mild neurological injury. None of the children with initially complete transections made any significant neurological recovery. These figures concur with other series on pediatric spinal cord trauma with bone injuries: Burke reported 86% of cases with complete cord lesions; Kwalrman, et al., 60%; Hubbard 57.1%; Hachen 88.9%; and Scher 85.7%.

Our data also indicate several important differences in the neurological status between children younger than 8 years and those over 8 years. The neurological injuries encountered in children younger than 8 years are much more serious than those in the older children. All the complete transections and all but one of the severe incomplete lesions were found in children 6 months to 8 years, whereas all but one older child had mild lesions (Fig. 1). The same age influence is also apparent within the subgroup with central cord syndrome: the children with more severe lesions are clearly younger than those with mild lesions (Fig. 1). This supports the observation that the inherent instability of the pediatric spine is maximum in infancy and decreases as the child reaches the second decade of life. The pattern of neurological lesions in children from 8 to 16 years is not unlike that seen in the adult counterpart of SCIWORA.

Upper cervical cord injuries are also more common in the younger age group. In contrast, lower cervical cord injuries occur in equal frequency throughout the age range of the series (Fig. 2). This is due to the fact that the physiological hypermobility peculiar to infants and young children predominantly involves the upper two to three cervical segments and not the cervicothoracic junction as much as in adults. Most of the age-related changes in anatomy and biomechanics occur in the upper segments, while the lower segments...
Cord injury with normal radiography

go through a much more subtle transition between the childhood and adult status.

Marar 57 concluded from his study of 126 patients with cervical spine injuries and cord damage that the mechanism of the injury can be predicted by the neurological lesions. The suggested mechanisms are: 1) flexion dislocation for complete cord transection; 2) hyperextension for central cord syndrome; 3) burst fracture or disc retropulsion for anterior cord syndrome; and 4) unilateral facet subluxation for Brown-Séquard syndrome. Unlike Marar, we found no correlation between the mechanism of injury and the type or completeness of the neurological lesions in children with SCIWORA. Certainly, the central cord syndrome characteristically associated with hyperextension in the adult is as often caused by flexion as by hyperextension in these children. Also, complete cord transection can be a result of hyperextension, flexion, longitudinal distraction, or direct crush injury. Our one case of Brown-Séquard syndrome was caused by hyperextension. Similarly, the unclassifiable partial syndrome can be caused by either flexion or hyperextension. Although it appears from Table 5 that flexion more often causes severe or complete lesions and that hyperextension more often causes mild lesions, the distinction is spurious, for the more serious neurological injuries all involved infants or young children. The younger the child, the more deformable the spine and the worse the neural insult, regardless of the mode or the direction of deformation.

Delayed Onset of Neurological Manifestation

The phenomenon of delayed onset of neurological manifestation in children with SCIWORA has been described previously. Cheshire 31 reported three patients with this delay, Burke 23 reported one, and Ahmann, et al., 2 two infants with similar delay and slow evolution of neurological signs. The paralysis developed rapidly once it began in all six children and culminated in complete cord lesions. In our series, delayed onset seemed to be the predominant mode of clinical presentation.

We have no ready explanation for this delay, but several points are worthy of note. Seven patients vividly recalled transient neurological symptoms at the time of injury. These include paresthesia, numbness, lightning sensation, and a subjective feeling of “total body paralysis.” If the spinal cord injury in SCIWORA is caused by some form of bone compression secondary to self-reducing subluxation, these symptoms must arise at the exact time of contact of bone with neural tissues. Because of rapid spontaneous reduction, complete neural destruction does not occur immediately. The severe neurological damage following the “latent period” must be a result of one of two occurrences: 1) incipient instability developed at the time of the original subluxation, which was then reactivated repeatedly by continued movements of the spine, thereby causing multiple repetitive insults to the cord; or 2) the original injury to the cord set off slow but progressive destruction of neural tissues.

If the first tenet is correct, the original subluxation must have resulted in partial tearing of crucial ligaments normally responsible for stability. Although immediate muscle action resulted in spontaneous reduction, such ligamentous injury will permit significant but not easily demonstrable displacements with each normal cycle of flexion-extension, causing a form of “punch drunk” trauma to the cord. Scher, 62 Evans, 60 and Webb, et al., 79 all alluded to this type of damage to the posterior ligaments in their so-called “hidden flexion injury of the cervical spine.” This mechanism is strongly suggested by the case of the child (Case 3 in Table 7) who experienced the initial “feeling of paralysis” but remained well until he played basketball 2 days following his neck injury. It may also be involved in the child (Case 4 in Table 7) who began recovering from his central cord syndrome while wearing a cervical collar but then experienced recurrence of paralysis after he removed his collar during play. These two were the only children with reported incidents involving strenuous neck movements during the latent period. Radiographic confirmation of this incipient instability is lacking since none of these 12 children had abnormal dynamic films. However, these films were made after, and not before, the onset of neurological signs, and the attendant spasm could have masked whatever incipient instability that was present before the cord was permanently injured. All of these patients had late (1 to 2 years) x-ray films to rule out delayed spinal deformities, but none was ever found.

The second tenet implies a slow but relentlessly progressive insult to the cord following a single contact or stretch injury. This insult may be ischemia. Ahmann, et al., 2 reported two infants with delayed onset; both had extensive watershed infarctions of the cord at autopsy, and the authors linked this lesion to the syndrome of progressive or evolving cerebral stroke. We know of no other case of proven cord infarction secondary to SCIWORA. A slowly enlarging epidural hematoma may cause gradual compression, but Bedbrook 13 categorically denied the existence of such an entity following spinal injury. Certainly, none of the myelograms performed on our patients, including six cases with delayed evolution of signs (Table 7), showed a subarachnoid block. Other possibilities are delayed traumatic hematomyelia, progressive edema, or the central hemorrhagic necrosis claimed by Osterholm 59 to be caused by accumulation of putative amines. None of these hypotheses have been proven in our patients.

We were unable to detect any unique clues in the age distribution, mechanisms of injury, or radiographic features of this subgroup to distinguish it from those patients with no delay in onset. The pattern of neurological outcome in this subgroup is also comparable to that of the main series: the complete lesions...
remained complete; those with initially severe lesions remained severely disabled; and those with initially mild lesions made generally satisfactory recovery. It is noteworthy that once sensorimotor paralysis began at the end of the latent period, it progressed inexorably into its established state within hours. The importance of this subgroup, therefore, rests on the hope that if the early warning signs of transient symptoms could be recognized and promptly acted upon before the onset of neurological signs, the tragic fate of some of these children might be duly averted.

Complications
The early and late complications of the pediatric SCIWORA syndrome are the same as those found in cord-injured children with fracture-dislocation, with one exception: delayed and progressive spinal deformities such as scoliosis, kyphosis, and lordosis were unknown in our series through a follow-up period of 18 years. Such deformities are common among children with skeletal injuries of the spine. Campbell and Bonnett reported an incidence of 91% in their children and Burke reported a 55.2% incidence. Half of Hubbard's series had some degree of spinal deformity, especially those with initially unstable spines. Babcock suggested that the late deformities are a result of a combination of factors, including destruction of the growth centers in the centrum, ischemic necrosis of epiphyseal growth plates, unilateral bone loss or wedging, unilateral fusion between fractured vertebral segments, and imbalance of the paraspinous muscle. It may be assumed that, since the first four factors all involve bone injuries in the premature spine and are therefore not relevant to the SCIWORA muscle. It may be assumed that, since the first four factors all involve bone injuries in the premature spine and are therefore not relevant to the SCIWORA syndrome, paraspinous muscle imbalance alone is not sufficient to cause delayed deformities.

Outcome
For children with SCIWORA the long-term prognosis is poor. Neither age nor treatment affect the final outcome. The only prognosticating factor is the initial neurological status. The trend of neurological recovery follows a grim but consistent pattern: useful recovery does not occur in those with initially complete lesions and seldom occurs in those with severe incomplete lesions. The best outcome is in those patients with initially mild deficits; most will make a complete recovery, and the rest will be only minimally disabled. The level of the neurological lesion, however, does influence the rehabilitation potential of the victim. Those with higher lesions generally do poorly compared to those with thoracic lesions. All of the high cervical lesions in this series occurred in infants or young children. Since quadriplegic children who are also ventilator-dependent have very limited rehabilitative potentials, these young children with SCIWORA represent a group with a very low rate of re-entry to the community.

Management
The diagnosis of SCIWORA should only be made after occult fractures have been excluded by polytomography or CT scanning. Occasionally, myelography may be necessary to rule out traumatic disc extrusion or extradural hematoma, but none of the 12 myelograms in our series showed a subarachnoid block.

Furthermore, gross instability must be ruled out by flexion-extension films. Although the initial subluxation had been reduced by spontaneous muscle action, there could have been sufficient stretch injury to the ligaments to render the spine unstable. Cheshire cited one case of instability missed by plain films and tomography but discovered by dynamic radiographic studies. Such studies must be executed under careful control. Ideally, the patient should be awake and cooperative so that continued neurological examination can be done to monitor the status of the cord. Fluoroscopy should be used, and troublesome paraspinal muscle spasm may be partly neutralized by muscle relaxant. The spasm, although serving an important protective function, interferes with obtaining good flexion-extension films. In some of the cases of Webb, et al., the spasm virtually precluded any active flexion, and subtle instability could not be ruled out until several days later. An inadequate dynamic study should never be accepted as proof of stability.

If immediate instability is revealed by the flexion-extension study, either surgical fusion or halo fixation is recommended. There are often objections to using only external immobilization for pure ligamentous injuries for fear of nonhealing, but since in the pediatric spine the ligaments are probably not ruptured but stretched, we think that halo fixation is a viable option.

If no immediate instability is demonstrated, or if spasm prevents adequate flexion, the cervical spine should be immobilized by a well fitting stiff collar. We find this adequate immobilization for this group of children with ligamentous sprain. In 1 to 7 days, or when the initial spasm has subsided, delayed dynamic films should be obtained. Both Webb, et al., and Scher have mentioned patients whose instability was discovered only on delayed dynamic films. One child in our series was discovered to have delayed anterior subluxation on the late dynamic films.

We urge that children who present with seemingly trivial head and neck injuries be questioned specifically for transient neurological symptoms. If these are present, tomography and dynamic films should be obtained, followed by admission for observation.

Summary and Conclusions
1. The pediatric syndrome of spinal cord injury without radiographic abnormality (SCIWORA) includes children with traumatic myelopathy who have
no radiographic evidence of fracture or dislocation on initial examination.

2. The mechanisms include hyperextension, flexion, repetitive flexion-extension, longitudinal distraction, and direct crush injury. Anatomical features peculiar to children permit transient subluxation without bone injury. Reflex muscle action causes spontaneous reduction which gives the normal radiographic appearance.

3. The neurological lesions encountered included complete cord syndrome, Brown-Séquier syndrome, central cord syndrome, and partial cord syndrome that did not fit the central or anterior cord patterns. Fifty-eight percent of these lesions were severe or complete.

4. There were differences in the neurological presentations of children younger than 8 years compared to children over 8 years. The younger children had more serious neural injuries and a higher incidence of upper cervical cord and thoracic cord injuries. Within the subgroup of central cord syndrome, the younger children also suffered more severe injuries.

5. Fifty-two percent of children had delayed onset of paralysis, and among these many recalled transient symptoms including paresthesia, numbness, and a subjective feeling of paralysis. All children with head and neck injuries complaining of symptoms must be fully investigated.

6. Initial radiographic examination should include tomography to rule out occult fractures and occasionally myelography to rule out traumatic disc extrusion. Immediate dynamic films should be obtained to select those children with incipient instability, who may then require surgical fusion or immobilization with halo fixation. Delayed dynamic films should be obtained to rule out late instability.

7. Long-term prognosis of SCIWORA is poor. Children with complete lesions and most of those with severe lesions do not recover. Only those with initially mild lesions have hope for satisfactory recovery. The initial neurological status is the major factor that determines the extent of long-term recovery.

References

Cord injury with normal radiography


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