0022-5282/89/2905-0654$02.00/0
THE JOURNAL OF TRAUMA
Copyright © 1989 by The Williams & Wilkins Co.
Vol. 29, No. 5
Printed in U.S.A.

Spinal Cord Injury without Radiographic Abnormality in Children–The SCIWORA Syndrome

DACHLING PANG, M.D., F.R.C.S.(C), F.A.C.S., AND IAN F. POLLACK, M.D.

Spinal cord injury in children frequently occurs without fracture or dislocation. The clinical profiles of 55 children with spinal cord injury without radiographic abnormalities (SCIWORA) are reported in detail to illustrate features of this syndrome. No patient had vertebral fracture or dislocation on plain films and tomographies. There were ten upper cervical (C1–C4), 33 lower cervical (C5–C8), and 12 thoracic cord injuries; of these, 22 were complete or severe lesions and 33 were mild lesions. The mechanism of the neural injury probably relates to the inherent elasticity of the juvenile spine, which permits self-reducing but significant intersegmental displacements when subjected to flexion, extension, and distraction forces. The spinal cord is therefore vulnerable to injury even though the vertebral column is spared from disruption, and this vulnerability is most evident in children younger than 8 years. All but one of the 22 children with profound neurologic injuries were younger than 8 years ($p < 0.000001$), whereas 24 of 33 children with mild injuries were older. Younger children were also more likely to have severe upper cervical lesions ($p < 0.05$); lower cervical lesions were distributed evenly through the ages of 6 months to 16 years. Thoracic injuries most commonly resulted from distraction or crushing. Distraction invariably involved violent forces, and crush injuries were usually caused by children being run over while lying prone, when the spinal column was acutely bowed towards the spongy abdominal and thoracic cavities.

Fifteen children had delayed onset of neurologic deficits; nine of these had transient warning symptoms of paresthesia, subjective paralysis, and Lhermitte's phenomenon 30 minutes to 4 days before the onset of deterioration. Eight other children suffered a second SCIWORA 3 days to 10 weeks after the initial SCIWORA. The spines in these children were presumably rendered incipiently unstable by the initial injury and thus were susceptible to additional, often more severe, neurologic trauma. The long-term neurologic outcome in children with SCIWORA is solely determined by their admission neurologic status. Realistically, the outcome can thus only be improved by: 1) ruling out occult fractures and subluxation which will require surgical fusion; 2) identifying patients likely to have delayed deterioration; and 3) preventing recurrent SCIWORA. Our experience and recommendations in these regards are discussed.

Spinal cord injury in children younger than 16 years of age is uncommon, accounting for only 0.65% to 9.47% of all spinal cord injuries (25, 26, 33, 37, 42). Although many children with traumatic myelopathy have overt vertebral disruption, a distinct group of children suffer spinal cord injury without radiographic evidence of fracture or dislocation (2, 9, 11, 12, 16, 22, 23, 28, 29, 31, 38, 39, 40, 44, 53, 55). In 1982, one of us (DP) and Wilberger reported 24 children at Children's Hospital of Pittsburgh (CHP) with this syndrome and coined the acronym SCIWORA for spinal cord injury without radiographic abnormality (38). Observations were made on the biomechanics, neurological lesions, treatment, and outcome of these children. The neural injury, in the absence of demonstrable fracture or dislocation, was postulated to be due to nondisruptive and self-reducing intersegmental deformation of the excessively malleable juvenile spine. Between 1982 and the present, we have treated an additional 31 children with SCIWORA at CHP, and our accumulated findings further supported the previous observations and hypothesis. In addition, several subgroups of SCIWORA patients have emerged from our recent analysis that have substantially modified the management of SCIWORA in the past 3 years. This paper is a detailed analysis of this composite series of 55 children with the SCIWORA syndrome.

From the Department of Pediatric Neurosurgery, Children's Hospital of Pittsburgh, Pittsburgh, Pennsylvania.

Address for reprints: Dachling Pang, M.D., F.R.C.S.(C), F.A.C.S., Chief, Pediatric Neurosurgery, Children's Hospital of Pittsburgh, Pittsburgh, PA 15213.

MATERIALS AND METHODS

Patients. By definition, children with SCIWORA show evidence of traumatic myelopathy without vertebral subluxation or fracture on plain films, linear tomography, and computed tomography (CT). Children whose injuries resulted from birth trauma, penetrating agents, and electric shock were excluded from the series. Because our primary aim was to study the vulnerability of the normal juvenile spine to deforming forces, children with congenital spinal anomalies known to cause inherent instability were also excluded. Between 1960 and 1987, 55 of 82 children with spinal cord injury treated at CHP fit the above criteria. Their ages ranged from 6 months to 16 years. Thirty children were younger than 8 years (55%), and 25 were older than 8 years (45%). Twenty-nine were males and 22 were females. The followup period ranged from 1 to 27 years.

Clinical Evaluation. All patients were evaluated by the neurosurgical service upon arrival. If extraneural injuries were apparent, the trauma surgeons were consulted. A careful history was taken from the older children or eyewitnesses to determine the cause and mechanism of the injury. In young children and children with concomitant concussion or traumatic amnesia, a careful assessment of bone and soft-tissue injuries often gave clues to the mechanism of the spinal cord injury. For example, chin laceration, mandibular and frontal fractures, and facial injuries suggested hyperextension, whereas occipital laceration or fracture pointed to a flexion injury.

The neurological lesions were classified according to their level and type. The levels were upper cervical (C1–C4), lower cervical (C5–C8), upper thoracic (T1–T6), and lower thoracic (T7–T12). The lesion types were complete physiologic cord transection, central cord syndrome, Brown-Séquard syndrome, and partial cord syndrome. The latter category included patients with partial preservation of function below the level of the lesion but whose pattern of deficits did not fit the central cord, anterior cord, or Brown-Séquard syndrome. The three incomplete cord syndromes were further classified as "severe" and "mild." Children with severe lesions had profound hand or arm weakness and serious impairment of ambulation whereas those with mild lesions had mild to moderate hand weakness, were able to ambulate, or showed rapid improvement of functions within the first 24 hours.

Radiographic Evaluation. At the time of admission, all patients had complete cervical spine films, including an open-mouth view of the odontoid when possible. In patients with a thoracic cord lesion or multisystem trauma, a full spinal survey was obtained. Before 1979, linear tomography was used to rule out an occult fracture at any suspicious site on the plain films and at the spinal level corresponding to the neurologic lesion. Since 1979, thin-section axial CTs with bony algorithms were used instead. Metrizamide (Nyegaard & Co., AS, Oslo, Norway) or Iohexol (Winthrop-Breon Lab., Des Plaines, IL) myelography was performed if the clinical examination or CT suggested a compressive lesion on the cord such as a ruptured disc or an extraaxial hematoma. Magnetic resonance imaging (MRI) was used on one recent patient with recurrent SCIWORA.

Dynamic cervical films with the neck in voluntary flexion and extension were obtained within 1 week of the injury. If paraspinous muscle spasm prevented adequate flexion, the dynamic study was repeated 1 to 2 weeks later. Patients with a thoracic cord injury also underwent dynamic "stress" studies with the patient in the extreme fetal and then extreme lordotic positions.

Electrophysiologic Studies. Since 1981, somatosensory evoked potentials (SSEPs) using standard surface stimulating electrodes overlying the common peroneal and median nerves were routinely obtained within 24 hours of admission in all children with SCIWORA.

TREATMENT

Initial Resuscitation. All patients with cervical cord syndromes had immediate neck immobilization with a hard collar on arrival. Those with thoracic cord injuries were placed supine on a fracture board. Patients in hypovolemic shock received fluid resuscitation with crystalloid and blood. Emergency orotracheal or nasotracheal intubation and mechanical ventilation were required in children with high cervical cord transection or severe multisystem trauma. Intracranial pressure monitors were placed in children with concomitant severe head injury. Those with intra-abdominal hemorrhage and/or bowel perforation underwent emergency laparotomies. Long bone fractures were treated initially with skeletal traction.

MANAGEMENT OF THE CORD INJURY

Severe Cord Lesions. Children with complete or severe cord syndromes were admitted to the intensive care unit (ICU) and kept on a Rotorest bed (Kinetic Concepts Inc., St. Antonio, TX). After dynamic or stress films had ruled out overt instability, children with cervical injuries admitted before 1985 were immobilized in a tight fitting Philadelphia Collar (Philadelphia Cervical Collar Co., Westville, NJ) for 8 weeks. Since 1985, these children were immobilized in a Guilford Cervical Brace (G.A. Guilford and Sons Orthotic Laboratory Ltd., Cleveland, OH) for 12 weeks. Thoracic cord lesions were treated with a thoracolumbar orthosis. Active rehabilitation began in the ICU and when medically stable, patients with serious deficits were transferred to a rehabilitation hospital for long-term therapy.

Mild Cord Lesions. Children with mild cervical cord syndromes were also kept on a Philadelphia collar for 8 weeks before 1985, and since then were immobilized with a Guilford brace for 12 weeks. If they remained ambulatory or showed substantial improvement within the first 48 hours, they were discharged home after dynamic films had ruled out overt instability. Children with serious hand weakness were enrolled in an outpatient physical therapy program. Contact and noncontact sports were strictly prohibited, and the importance of activity restrictions were explained to both the patient and the family.

FOLLOWUP AND OUTCOME

Following hospital discharge, all children with SCIWORA were evaluated biweekly at the neurosurgical clinic. The bracing devices were checked for fitting and the child and family questioned firmly regarding their compliance with activity restrictions.

At the conclusion of the bracing period, dynamic films were obtained without the immobilization devices to ensure stability. SSEPs were repeated from 6 weeks to 6 months after injury.

The final outcome status of the patients was assessed at least 1 year after the injury. The neurological status was classified into: 1) complete cord syndrome; 2) severely impaired if the patient remained nonambulatory or had severe residual hand weakness, or if he or she remained ventilator dependent; 3) mildly to moderately impaired if the patient was ambulatory with good hand function; and 4) normal.

Statistical Analysis. Comparisons between relevant subgroups of children were made using Fisher's exact test with 2-tailed p-values.

RESULTS

CAUSE AND MECHANISM OF INJURY

The majority of SCIWORA injuries were related to motor vehicle accidents (Table I). Of the 28 children injured this way, 12 were passengers, seven were pedestrians, two were on a motorcycle, and seven were run over by cars. Other common causes of injury were falls (13 cases) and sporting accidents (ten cases). There was one case of child abuse.

Flexion and extension forces accounted for most of the injuries. Eighteen children suffered flexion injuries, all to the cervical region. Four others had flexion-compression injuries, three from diving accidents and one from a falling object. There were 13 hyperextension injuries, all but two involved the cervical spine. The victim of child abuse probably had repetitive flexion and extension to the neck. Five other children were injured by violent lateral bending of the neck.

Seven children sustained distraction to their spines. Three of these children were restrained by lap seat belts during high-speed collisions; all three suffered high thoracic cord transections and one also had a transverse fracture of the L2 vertebral body (Chance fracture). Four other children suffered distraction to their cervical spine when their head and neck regions were caught by moving vehicles.

Six of the seven children that were run over by vehicles had clearly visible tire marks on the back and their thoracic spines were likely hyperextended by the crushing. Only one child had tiremarks on the anterior chest. All seven suffered various degrees of lower thoracic cord damage.

ASSOCIATED EXTRANEURAL INJURIES

Eighteen children had concomitant closed head injuries; seven of these had brain contusion and 12 had skull fractures. Two children with frontal fractures also had mandibular fractures.

All seven children with thoracic crush injuries sustained some form of thoraco-abdominal trauma including pulmonary contusion, rib and pelvic fractures, and renal artery laceration. Five other children suffered intra-abdominal hemorrhage from liver laceration (one case), ruptured spleen (two cases), and mesenteric tear (two cases). Six children had extremity fractures.

NEUROLOGICAL LESIONS

Level and Type of Neurological Lesion. Ten patients had upper cervical cord (C1–C4) injuries and 33 had lower cervical cord (C5–C8) injuries. Twelve patients had thoracic cord injuries, six at the T1–T6 levels, and six at the T7–T12 levels.

Twelve children showed evidence of complete cord transection on admission (Table II). Twenty-one children presented with central cord syndrome; of these, four had profound weakness and 17 had mild to moderate weakness of the hands and legs. Sixteen children had a partial cord syndrome, which was severe in four and mild to moderate in 12. The remaining six patients had a typical Brown-Séquard syndrome with ipsilateral hemiparesis and proprioceptive impairment and contralateral hemianalgesia; only two of these were severe. Overall, 22 children (40%) sustained severe or complete cord injuries and 33 children (60%) had mild to moderate cord injuries at the time of admission.

Figure 1 relates the level to the severity of the neurological injury. Children with upper cervical SCIWORA were significantly more likely to have severe neurologic lesions than those with lower cervical injuries ($p < 0.0001$). Of the ten patients with C1–C4 injuries, nine had either complete or severe lesions; in contrast, 28 of the 33 C5–C8 lesions were mild. The upper thoracic cord likewise appeared to be more vulnerable than the lower thoracic cord. All six of the T1–T6 lesions were complete or severe, whereas only two of the six T7–T12 lesions were severe. This difference, however, was not statistically significant ($p = 0.06$).

Age and Severity of Neurological Injury. Figure 2 shows the relationship between the patient's age and the severity of the neurological lesion. Because previous studies have shown that the biomechanical properties of the juvenile spine gradually take on adult characteristics by age 8 years (10, 11), we have divided our series into younger children less than 8 years old, and older children from 8 to 16 years. The younger children were much more likely to suffer devastating neurological damage: 21 of 30 children younger than 8 suffered complete or severe lesions, whereas only one of 25 children older than 8 years suffered severe injury. Stated in another way, all but one of the 22 complete or severe injuries occurred in the younger group ($p < 0.000001$) (about two thirds of these children were less than 4 years old). This analysis strongly suggests that the spines of infants and young children were more vulnerable to deforming forces than the spines of older children.

Age and Level of Neurological Injury. Figure 3 shows the relationship between age and the level of the neurological lesion. Considering first the cervical region, nine of the ten upper cervical injuries occurred in children younger than 8 years, whereas the lower cervical injuries were more evenly distributed within the age limits of the series ($p < 0.01$). In addition, these nine upper cervical lesions were either complete or severe. This suggests that the upper cervical spine is inherently hypermobile and therefore more prone to serious injuries in infants and young children. The lower cervical segments, on the other hand, are at risk in all ages up to 16 years, although severe lower cervical lesions are still more likely to occur in the very young. In the thoracic region, nine of the 12 injuries

TABLE I
Cause of injury in 55 patients with SCIWORA

Cause of Injury	Number of Children
Automobile accident	12
Hit by vehicle	7
Run over by car (crush)	7
Motorcycle	2
Fell from height	11
Fell down steps	2
Diving	2
Gymnastics	2
Wrestling	2
Football tackle	2
Thrown from horse	1
Skating	1
Blow to face	1
Sled accident	1
Object fell on head	1
Child abuse	1

TABLE II
Types of neurologic syndromes in 55 patients with SCIWORA

Neurologic Syndrome	Number of Patients
Complete cord transection	12
Central cord, severe	4
Central cord, mild	17
Partial cord, severe	4
Partial cord, mild	12
Brown-Séquard, severe	2
Brown-Séquard, mild	4

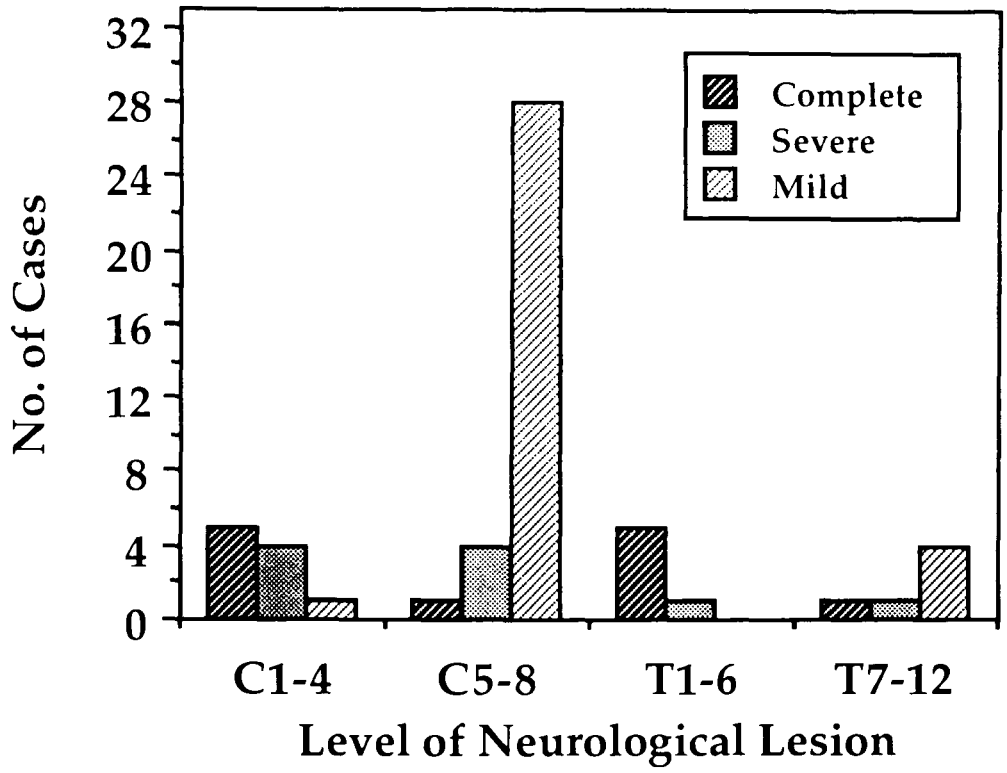

FIG. 1. Correlation between the level and the severity of the neurological injury. Patients with upper cervical SCIWORA were significantly more likely to have severe neurological lesions than patients with lower cervical SCIWORA ($p < 0.00001$).

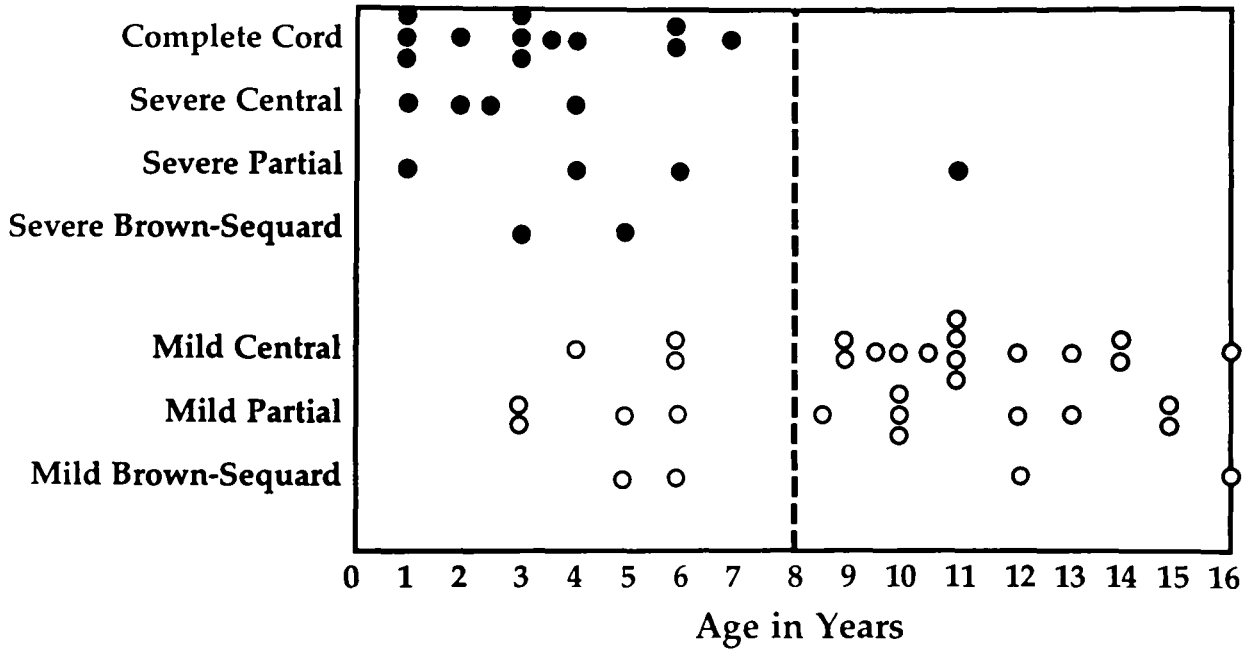

FIG. 2. Correlation between patient age and the neurological syndromes. Solid dots represent patients with complete or severe injuries and open dots represent patients with mild injuries. The vertical dotted line denotes the age (8 years) at which the physiologic properties of the juvenile spine change, thus defining a younger and an older age group. Children younger than 8 were much more likely to suffer devastating neural damage; all but one of the severe injuries occurred in children younger than 8 years. In contrast, all children older than 8 except one suffered mild injuries.

occurred in children younger than 8 years and among these nine lesions, seven were complete or severe and two were mild. Only one of the three older children had a severe lesion. The thoracic spine, like the upper cervical spine, thus also appears to be most vulnerable to deforming forces in the very young.

Mechanisms of Injury and Severity of Neurological Lesion. Table III shows that flexion, extension, and thoracic crushing produced very similar ratios of severe versus mild injuries. There was thus no consistent relationship between mechanism and severity of injury, with one possible exception. Injury due to distraction tended to be much more severe, but this may relate more to the magnitude rather than the mode of deformation since all distraction injuries were associated with high-speed accidents. Even with distraction, the age factor was

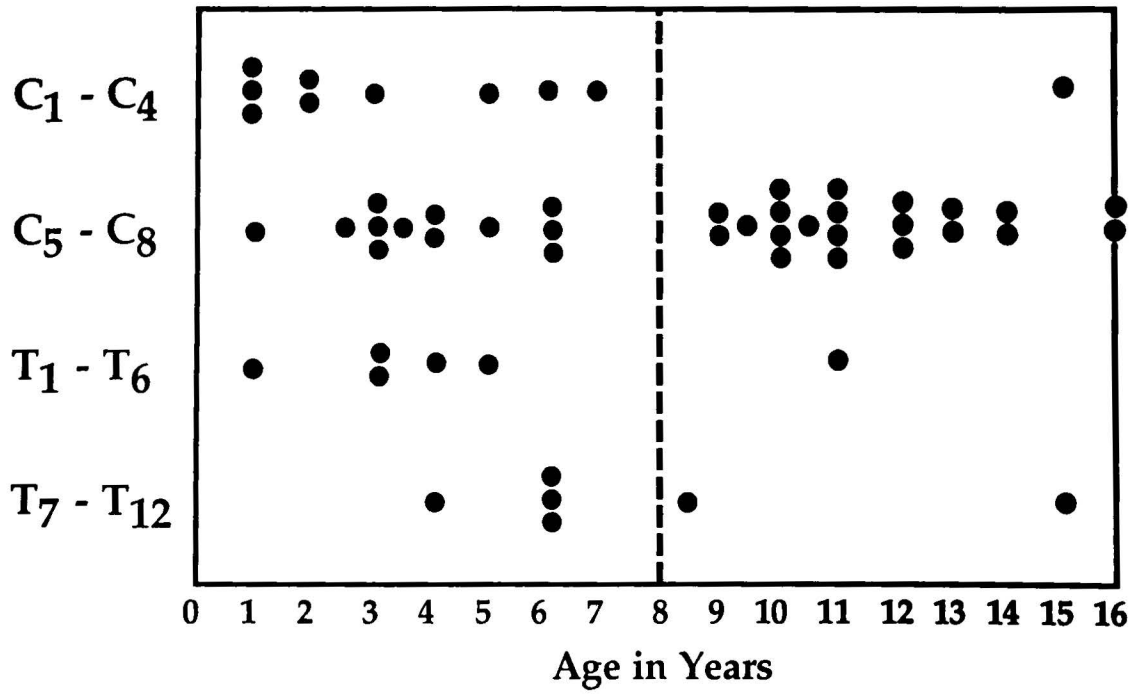

FIG. 3. Correlation between patient's age and level of neurological injury. The vertical dotted line denotes the age (8 years) at which physiologic properties of the juvenile spine change. The upper cervical spine was most prone to injury in infants and young children, whereas the lower cervical spine was at risk in all ages up to 16 years.

TABLE III
Correlation between mechanism of injury and severity of neurologic injury

Mechanism of Injury	Severity of Injury		
	Complete	Severe	Mild
Flexion	2	4	12
Hyperextension	3	1	9
Distraction	5	1	1
Flexion-lateral bending	0	2	3
Flexion-compression	0	0	4
Crush	2	1	4
Flexion-extension, repetitive	0	1	0

TABLE IV
Correlation between mechanism of injury and level of neurologic lesion

Mechanism of Injury	Level of Injury			
	C1-4	C5-8	T1-6	T7-12
Flexion	6	12	0	0
Hyperextension	0	11	2	0
Distraction	2	2	3	0
Flexion-lateral bending	2	3	0	0
Flexion-compression	0	4	0	0
Crush	0	0	1	6
Flexion-extension, repetitive	0	1	0	0

important, since all six children with severe distraction injuries were younger than 8 years, and the only child that escaped with a mild injury was 11 years old.

Mechanism of Injury and Level of Neurological Lesion. Table IV relates the types of deforming forces to the level of the neural injury. Eight of the ten upper cervical cord injuries involved some element of flexion, whereas lower cervical cord injuries were associated with a variety of mechanisms, suggesting that the upper cervical segments were most susceptible to flexion. There were too few thoracic lesions to warrant a similar comparison.

SPECIAL CLINICAL GROUPS

Delayed Neurological Deterioration. Fifteen patients in this series (27%) had delayed onset of neurological deficits following spinal trauma (Table V). The "latent period" between injury and the appearance of neurological findings ranged from 30 minutes to 4 days, with a mean of 1.4 days. There was nothing specific in their age, mechanism of injury, or radiographs to set this subgroup apart from the rest of the SCIWORA population. The only warnings that foreboded later deterioration in these 15 children were three transient symptoms experienced by nine of them immediately following trauma: paresthesia in the hands and legs (six patients); a subjective feeling of generalized weakness (three patients); and a lightning sensation down the spine associated with neck movements (two patients). In all nine patients, these warning symptoms were initially ignored and, even though all nine had significant neck pain and spasm, their spines were not initially immobilized. Once sensorimotor paralysis began, it progressed inexorably without a second documentable bout of trauma. Two children ultimately progressed to complete transection and four to severe partial or central cord syndrome, and nine suffered mild to moderate central cord syndrome.

With the institution of a regional trauma referral network 5 years ago and considerable improvement of prehospital care, the incidence of these delayed injuries has significantly decreased. While delayed neurological deterioration was documented in 13 of the initial 24 SCIWORA patients treated at CHP between 1960 and 1982, only two cases have been seen among 31 patients treated since 1983.

Recurrent SCIWORA. Eight patients ranging in age from 2 to 14 years sustained a second spinal cord injury 3 days to 10 weeks after the initial SCIWORA. The mechanisms of the

TABLE V
Delayed neurological deterioration in 15 children

Age (yrs)	Mechanism of Injury	Initial Transient Symptoms	"Latent Period"	Neurological* Status	Myelography	Final Outcome
2	Flexion	Transient paralysis	2 days	Severe central	Normal	Severly impaired
4	Distraction	Paresthesia	2 days	Severe central		Severely impaired
8½	Extension	Subjective paralysis	2 days	Mild central		Normal
10	Flexion-compression	Lightning sensation; paresthesia; subjective paralysis	1 day	Mild central		Normal
10	Flexion-compression	Paresthesia; subjective paralysis	4 hrs	Mild central		Mildly impaired
11	Extension	Paresthesia	12 hrs	T5 severe partial	Normal	Severely impaired
14	Extension	Paresthesia	12 hrs	Mild central	Normal	Normal
15	Extension	Paresthesia	6 hrs	Mild central		Normal
11	Flexion	Subjective paralysis	1 hr	Mild central	Normal	Mildly impaired
½	Flexion		4 days	Severe partial	Normal	Moderately impaired
1⅓	Crush		1 day	T6 complete	Normal	Complete
2½	Extension		4 days	Complete	Normal	Complete
9	Flexion		½ hr	Mild central		Mildly impaired
16	Extension		1 day	Mild central		Normal
15	Flexion		1 day	Mild partial	Normal	Normal

* The level of neurological injury is cervical unless otherwise stated.

initial injury included three cases of hyperextension, four cases of flexion, and one case of flexion-compression. In all eight children, the initial lesion was a mild partial cervical cord syndrome. All eight children had normal plain and dynamic cervical films, were immobilized with a hard cervical collar, and were instructed to avoid vigorous activities. Because all eight children either fully recovered or had only mild residual deficits within 2 days after the initial injury, they were discharged from the hospital on the third day.

Recurrent injury occurred within a week after the initial SCIWORA in five children, and at 3, 4, and 10 weeks in three others. In four children, re-injury involved moderate trauma during contact sports, but in the other four children, re-injury was caused by trivial trauma such as throwing a basketball or falling off a couch. Six children were not wearing their collars at the time of the second injury, and two children were re-injured with the collar on. In six children the mechanism of the second injury was identical to that of the initial injury; in the other two, the mechanism was unknown.

The neurological lesions of the recurrent SCIWORA were, without exception, more severe than the initial injury. Three children suffered profound quadriparesis or paraparesis and four of the remaining five patients had disabling sensorimotor paralysis affecting the upper and lower extremities.

At the time of the recurrent SCIWORA, plain and dynamic cervical films again failed to demonstrate fracture, dislocation or instability. Tomography (six children), myelography (three children), CT (one child), and MRI (one child) were also normal.

Thoracic SCIWORA due to Crush Injury. The ages of the seven children with thoracic SCIWORA due to crush injury ranged from 1 to 15 years (Table VI). In every case, tiremarks were clearly seen on the body of the child so that the body position and exact site of the crushing could be determined. All but one of the children were lying prone during the crush; the only exception was a 6-year-old child who was run over while lying supine over a puddle of wet mud.

Six of the seven children had a lower thoracic cord lesion whereas one 1-year-old infant had a T6 lesion. Two lesions were complete, one was severe, and the other four were mild. Plain and stress thoracolumbar X-rays showed no fracture or dislocation. The three children with complete or near-complete transection also had CT myelography which showed swelling of the cord without evidence of cord compression.

TABLE VI
Summary of 7 children with thoracic SCIWORA due to crush injury

< 8 Years			≥ 8 Years		
Age (yrs)	Tiremark	Severity of Cord Lesion	Age	Tiremark	Severity of Cord Lesion
1	Back	T6-complete	8 yr	Back	T10-mild
6	Front	T8-mild	15 yr	Back	T10-mild
4	Back	T10-mild			
6	Back	T8-severe			
6	Back	T10-complete			

All seven children had significant non-neural injuries, the most common being pulmonary contusion and rib fractures (five cases). Two children also had pelvic fractures. One child had traumatic asphyxia, and a 6-year-old girl had renal artery thrombosis, duodenal contusion, mesenteric artery avulsion, and jejunal infarction.

RADIOGRAPHIC FINDINGS

All SCIWORA patients had normal plain spine films at the time of admission, with one exception. This child sustained a lap seat belt injury and had a Chance fracture of the L2 vertebral body. However, her thoracic spine at the level of her neurological lesion (T6) was normal. Linear tomography (24 children) and CT (11 children) of the relevant spinal segments showed no fracture, dislocation, or malalignment. Myelograms performed on 17 patients were unremarkable in 14 except for nonspecific cord swelling. In one 3-year-old child with a complete high cervical cord lesion, myelography suggested an intradural hematoma at C1, and surgical exploration revealed complete disruption of the cord. In two 4-year-old children with

complete T4 lesions due to distraction, the chest X-rays showed widening of the mediastinum and myelography revealed rupture of the spinal cord and adjacent dura, with free extravasation of contrast into the posterior mediastinum and extrapleural space.

Dynamic cervical films obtained during the first week in all children with cervical lesions demonstrated no instability. However, many such studies were unacceptable due to severe paraspinous muscle spasm which prevented adequate flexion. Dynamic films repeated on these patients after spasm had subsided showed late instability in only one patient who had anterior subluxation of C4 on C5 that was masked by spasm on previous studies.

None of the thoracic stress films performed on patients with thoracic myelopathy showed any instability.

ELECTROPHYSIOLOGIC STUDIES

Abnormalities in the SSEPs were noted in 17 of 22 patients who had the test. These included unilateral or bilateral prolongation of peroneal and/or median nerve latencies, often associated with decreased wave amplitudes. The five patients with normal SSEPs all had relatively mild weakness and minor alteration in pinprick sensation, without loss of proprioceptive or vibratory sensation. Their dorsal columns were presumably uninjured.

SSEPs were particularly useful in detecting spinal cord dysfunction in children with combined craniospinal trauma. In one child with cerebral contusion and hemiparesis, significant lengthening of the early latencies of the evoked responses indicated that some of the motor weakness was in fact due to a concomitant cord lesion. SSEPs were also useful in evaluating young children and infants with questionable weakness on clinical examination. In addition, early SSEPs serve as a baseline against which subsequent recovery could be gauged. In the children who ultimately regained normal neurological function, SSEPs likewise normalized. In three of the recurrent SCIWORA cases, SSEP abnormalities which resolved during the recovery phase after the initial injury were again evident following the second SCIWORA.

OUTCOME

Six patients with complete or severe high cervical cord injuries underwent tracheostomy to facilitate long-term mechanical ventilation. The patient with the transverse L2 body fracture and a midthoracic cord lesion underwent successful posterior lumbar fusion with Harrington rods. The two children with high thoracic cord rupture and CSF-mediastinal fistulae were treated successfully with serial thoracenteses.

Two children with high cervical cord lesions, including the child who underwent an exploratory C1–C2 laminectomy, died from progressive respiratory failure during the first 2 weeks of treatment. The child with the C4 subluxation found on delayed flexion-extension films was fitted with a halo apparatus for 8 weeks and subsequently achieved stability. The eight children with recurrent SCIWORA were begun on a second course of immobilization. The Philadelphia collar was used on the first six patients, but the two most recent patients were given the Guilford brace for more secure stabilization. Because one recurrent injury occurred 10 weeks following the first injury, suggesting inadequate ligamentous healing during that length of time, the two recent patients were kept in the brace for 12 weeks, counting from the day of the second injury. All eight "recurrent" patients ultimately completed their second course of treatment without further complications. The remaining 44 patients also completed their prescribed course of immobilization without complication. Subsequent dynamic films on all 53 survivors showed no late instability or deformity.

The most reliable predictor of neurological outcome was the initial neurological status (Table VII). An initially severe neural injury was almost always associated with a poor prognosis, whereas an initially mild to moderate injury was compatible with good recovery. Of the ten children with complete cord transection who survived, none had any neurological improvement. All three surviving children with complete upper cervical injury remained ventilator dependent. Another child with C6 transection, while not requiring permanent ventilatory support, has been unable to assume self care because of frequent respiratory complications and distressing muscle spasm. The six children with complete thoracic injuries, although severely handicapped, have all attained some degree of self care.

Of the ten patients with initially severe lesions, six remained severely impaired and four improved to the point of independent ambulation with prostheses. Again, the children with thoracic injuries tended to be better rehabilitation candidates and had a higher rate of community re-entry than those with severe cervical injuries.

In contrast to the dismal outlook of severe SCIWORA, most of the 33 children with mild to moderate injuries have shown significant improvement in neurologic function. Twenty-six children became normal and five had mild to moderate residual deficits that did not preclude a full psychosocial lifestyle. Among these 33 children with initially mild injuries were the eight patients with recurrent SCIWORA; two of the eight remained permanently and severely disabled, two had mild to moderate weakness, and four enjoyed complete neurological recovery. The devastating effect of the second SCIWORA is evidenced by the fact that all eight children were either completely or nearly normal just before the recurrent injury.

DISCUSSION

The incidence of SCIWORA among all pediatric spinal cord injuries varies between 5% to 55% in the literature (4, 5, 17, 23, 29, 31, 33). The 67% incidence in our series probably reflects our particular interest in this syndrome, although other recent series (30, 41) also emphasized the common occurrence of SCIWORA in pediatric spinal trauma. In comparison, spinal cord injury without skeletal disruption is rare between the ages of 16 and 50 years (34, 43). The mechanical properties of the adult spine in this age range are such that vertebral fracture and/or dislocation invariably occurs in association with the neural damage. The immature and inherently elastic pediatric spine differs from the adult spine in that it is much more deformable, so that momentary intersegmental displacement caused by external forces may endanger the spinal cord without disrupting bone or ligaments. Four mechanisms may be involved in the pathogenesis of SCIWORA in children: 1) hyperextension, 2) flexion, 3) distraction, and 4) spinal cord ischemia.

Hyperextension of the cervical spine forces the interlaminar ligaments to bulge forward into the spinal canal and may cause up to 50% narrowing of the canal diameter (3, 46). In addition, the cord also thickens as it shortens during hyperextension (15). Thus the space availability for the cervical cord within the canal is normally reduced during moderate hyperextension. During violent hyperextension, Taylor and Blackwood (47) and Bourmer (12) demonstrated rupture of the anterior longitudinal ligament, shearing of the intervertebral disc from the end plates, and sufficient retrodisplacement of the upper vertebral body to compromise the spinal cord. Dynamic

TABLE VII
Outcome in 55 patients

Initial Neurological Status	No.	Final Neurological Status				
		Death	Complete Cord Syndrome	Severely Impaired	Mildly to Moderately Impaired	Normal
Complete cord syndrome	12	2	10			
Severe cord syndrome	10			6	4	
Mild cord syndrome	33			2*	5†	26‡

* Recurrent SCIWORA patients. †Includes 2 recurrent SCIWORA patients with moderate deficits.‡ Includes 4 recurrent SCIWORA patients with no deficit.

radiography on fresh cadavers further showed that even in the adult spine, elastic recoil of the displaced segment could effect perfect spontaneous reduction so as to give a normal radiographic appearance (36). With its elastic osseoligamentous elements, the juvenile spine is especially susceptible to this sequence of momentary dislocation and spontaneous reduction. In fact, intersegmental movement in children is facilitated by their horizontally oriented facet joints which normally permit greater mobility at the expense of stability (8, 21, 45, 49, 51, 52). Furthermore, detachment of the vertebral body from its adjacent disc can occur readily within the brittle growth zone of the immature end plate, which splits easily from the primary centrum with even moderate hyperextension (6). Our experience confirms that hyperextension is a common mechanism of SCIWORA.

Although cadaver studies had shown that flexion forces could not damage the cord of adults without causing fracture or dislocation of the facets (10), more recent studies in children demonstrated that flexion injury to the juvenile spine could and often did produce sufficient subluxation to cause myelopathy without concomitant bony and ligamentous damage (17, 22, 24, 28, 38, 39, 48). As mentioned, the ligaments and joint capsules in children are elastic and redundant enough to undergo significant stretching without tearing (8, 45, 51), and the horizontal facets also permit considerable interarticular motions (8, 21, 51, 52). The wedge-shaped vertebral bodies in children also facilitate forward slipping between adjacent vertebrae (8, 45). Furthermore, the uncinate processes of the mature vertebra, which normally restrict lateral and rotational movements between bodies, are virtually absent in children less than 10 years old (14, 49). Finally, the proportionately large head and underdeveloped nuchal musculature of infants make then unusually susceptible to flexion and extension injuries (18, 50).

Population studies among normal infants and children revealed that the horizontal orientation of the facets as well as the anterior wedging of the vertebral bodies are particularly prominent in the upper four segments of the cervical spine (8, 45). Dynamic radiographs have also determined that the fulcrum for maximal flexion in the cervical spine is at C2–C3 in infants and young children, at C3–C4 around age 5 or 6, and at C5–C6 in adults (9, 13, 51). These findings suggest that the upper cervical segments in young children have the greatest degree of physiologic mobility but also are most susceptible to flexion injuries. Our results fully support this view, in that nine of our ten children who suffered upper cervical cord injuries were younger than 8 years, and all nine had either complete or very severe cord lesions. Moreover, eight of these injuries involved some component of flexion.

Bailey (8) and von Torklus and Gehle (52) found that the characteristic anatomic features of the juvenile cervical spine gradually assume adult appearances around the age of 8 years, when the wedge-shaped bodies become rectangular, the facets become more vertical, the uncinate processes begin protruding, and the ligaments and capsules gain in tensile strength. Our data suggest that this maturation takes place much more abruptly and successfully in the upper segments, so that the upper neck, while very vulnerable to flexion injuries under the age of 8, abruptly becomes resistant to injuries shortly afterwards. Only one upper cervical lesion in our series occurred in the older age group, and this was a mild injury. The lower cervical spine, on the other hand, seems to mature more gradually, since lower cervical injuries were evenly distributed over the entire span of 16 years. However, even for the lower cervical spine, the serious lesions still tended to congregate in the younger age group, as all of the five severe lower cervical lesions occurred in children younger than 8 years.

Besides hyperextension and flexion, the pediatric spine is also uniquely vulnerable to distraction injuries (17, 28). Leventhal found that while the elastic spinal column of neonatal cadavers could be stretched up to 2 inches without structural damage, the relatively inelastic spinal cord would rupture if stretched more than ¼-inch (35). The clinical counterpart of Leventhal's study is the finding of frank rupture of the cord and meninges within a completely intact vertebral column in infants having undergone forceful breech extraction (1, 51). Among reported cases of nonobstetrical SCIWORA, our two cases of lap-belt injuries with CSF-mediastinal fistulae probably provide the best evidence of distraction. It is well known that the thoracic spine is forcefully distracted in lap-belt injuries when the upper body is hurled forward by the collision while the pelvis is fixed to the seat by the belt. The dural and spinal cord rupture in these two patients confirmed the mechanism of distraction. That

six of our seven cases of distraction injuries resulted in severe cord lesions may simply reflect the violent forces involved in high-speed accidents, but the age factor also may play a part in that all six victims were very young and presumably had very elastic spinal columns.

Finally, the pediatric spine also possesses biomechanical properties that predispose the upper cervical cord to ischemic necrosis (2). Gilles et al. (27) found the infantile atlanto-occipital joint inherently unstable due to: 1) an incompatibly small C1 arch against a large foramen magnum; 2) redundant atlanto-occipital ligaments; 3) lax condylar capsules; and 4) flat condylar articulating surfaces. A great deal of sliding movement occurs between the occipital condyle and the lateral mass of C1 during normal activity which may cause stretch injury to the vertebral arteries. Moreover, the bony protective groove for the vertebral artery where it curls behind the lateral mass to enter the skull is so shallow in infants that the artery may be crushed between the occipital condyle and the lateral mass during hyperextension. Gilles et al. (27) have demonstrated bilateral vertebral artery occlusion during hyperextension on postmortem angiography.

In general, thoracic SCIWORA shares with cervical SCIWORA the same relationship between age and severity of lesion; there were eight complete or severe lesions out of a total of 12 thoracic lesions and seven of these were in children younger than 8 years old. The splinting effect of the rib cage normally protects the thoracic spine against flexion and extension forces, and a great deal of violence is necessary to produce intersegmental displacement. However, since the sternocostal joints normally permit a fair amount of "bucket-handle" movements, the rib cage provides little protection against severe longitudinal distraction. The fact that all three thoracic distraction injuries resulted in complete cord rupture lends support to this contention.

In order to elucidate the mechanism of cord injury in the seven cases of thoracic crushing, we analyzed a parallel series of seven cases of crush injuries to the abdomen and thorax without spinal cord injury (author's unpublished data). In the group without cord injury, the tire marks were invariably on the front, whereas in the crush SCIWORA group, six of seven cases had tire marks on the back. The incidence of pulmonary contusion and rib fractures is comparable in both groups, but the SCIWORA group had more retroperitoneal soft-tissue injuries while the other group had more intraperitoneal injuries such as liver and spleen laceration and bowel perforation. We postulate that for SCIWORA to result from crush injury, the patient must be lying prone so that the thoracic spine may be acutely bowed ventrally (hyperextended) past physiologic limits into the spongy abdominal and thoracic cavities. This ventral bowing also tears retroperitoneal structures but the spine shields the intraperitoneal organs from direct crushing. If the crushing occurs with the child supine, the spine is lying directly against and hence firmly supported by the ground; the normal curvatures of the thoracic and thoracolumbar segments merely straighten out with very little bowing, no matter how heavy the crush. The abdominal contents are directly vulnerable in this case but the spinal cord is not. It is interesting that the one exception to this hypothesis is the 6-year-old with SCIWORA who was crushed from the front, but his back happened to be supported loosely by a puddle of soft mud which presumably allowed the spine to bend sharply backwards.

The phenomenon of delayed onset of neurological deficits has been previously reported in children with SCIWORA (2, 16, 17, 22, 23, 53). The majority of these cases did not have a slowly expanding mass lesion to account for the late deterioration. In our series, no cord compression was found in the eight children with delayed deficits who had myelography. The transient but definite neurologic symptoms of subjective paralysis, distal paresthesia, and the Lhermitte phenomenon experienced by nine children with delayed deficits suggest that contact had occurred between the cord and the bony spine at the time of the injury as a result of intersegmental displacement. Although this displacement had self reduced by elastic recoil, we believe that it had resulted in overstretching or partial tearing of crucial stabilizing ligaments, thus rendering the injured segments incipiently unstable to even minor stresses induced by casual neck movements during the "latent period." The cord was, in effect, at risk for re-injury during this period, and ultimately did succumb to that one displacement that exceeded physiologic limits. In this regard, our original series of SCIWORA patients treated from 1960 to 1982 consisted of 13 children with delayed deficits (38). Since 1983, we have become much more aware of this phenomenon and have widely propagated to local communities the practice of immediate neck immobilization and admission for children with neck injury who complained of neck pain and transient neurologic symptoms. Since then, we have only encountered two children with delayed deficits.

Even more convincing evidence for the existence of incipient instability in SCIWORA is the phenomenon of recurrent injury. The fact that four children sustained their second SCIWORA after trivial trauma several days to weeks following the first injury meant that this incipient instability probably persisted for some time. It may seem paradoxical that the only children who suffered a recurrent SCIWORA were those who sustained a mild first SCIWORA, and not those with initially serious injuries who presumably had more ligamentous disruption. The explanation is that children with initially serious injuries were all confined to bed with neck immobilization and could not have sustained further stress to their necks. The mildly injured children, in contrast, were granted early activity and because of rapid recovery, were also more likely to ignore warnings against vigorous activities or removing their collars.

The fact that dynamic X-rays failed to disclose abnormal movements in the SCIWORA patients does not necessarily argue against the existence of incipient instability. Immediately after the injury, relatively severe instability can be masked by muscle spasm (54). Even after spasm has subsided, dynamic studies involve cautiously executed deliberate flexion and extension which can in no way simulate the stresses of sudden and random neck movements in real-life activities. It is therefore not surprising that these studies were all normal before the recurrent SCIWORA, and yet seemingly tame sports precipitated the recurrent cord injury. It should be emphasized that dynamic studies in pediatric neck trauma serve the sole purpose of separating patients with overt instability (who require surgical fusion) from SCIWORA patients. Normal dynamic studies do not predict which SCIWORA patients are susceptible to recurrent injury.

Delayed spinal deformities such as scoliosis and kyphosis were not found in our SCIWORA patients through a followup period of 26 years, whereas such deformities are common among children with spinal column injuries (17, 20, 32). Babcock (7) suggested that the late deformities are a result of several factors, including destruction of growth centers, ischemic necrosis of epiphyseal growth plates, unilateral bone loss, asymmetric fusion between adjacent vertebrae, and paraspinous muscle imbalance. Since the first four factors all involve bone injuries, it may be assumed that paraspinous muscle imbalance alone in SCIWORA is not sufficient to cause delayed deformities.

Our data suggest that standard treatment does not affect the final outcome of SCIWORA once serious neurological damage has occurred. The overall outcome can be improved only if the incidence of serious neurological damage can be reduced. Since the proneness of children to falls and road accidents is unlikely to change, only three ways are available to improve outcome once the injury has occurred: 1) rule out overt spinal instability; 2) identify patients likely to have delayed neurological deterioration before it happens; and 3) prevent recurrent injury.

It has already been emphasized that occult fractures and vertebral malalignment generally imply overt instability and must be categorically ruled out by CT before the diagnosis of SCIWORA can be made. While obtaining dynamic films, inadequate flexion due to spasm must never be accepted as proof of stability. If abnormal intersegmental movement is identified, the patient should undergo surgical fusion or halo immobilization.

Although lacking direct proof, our study also suggests that increased awareness in our community of the phenomenon of delayed neurological deterioration has reduced its recent occurrence. We urge that children who present with neck and head injuries be questioned specifically for transient neurological symptoms even if they are neurologically normal. If the symptoms are convincing, CT and dynamic X-rays should be obtained, followed by admission for observation, and immobilization with a hard collar for 6 weeks. Arguably, a number of children may be "overtreated" by this regimen, but we are consoled by the fact that only two patients manifested delayed deterioration since our initial interpretation of this phenomenon 5 years ago. The benefit of preventing a potentially serious neurological injury, however remote the possibility, probably still outweighs the inconvenience of treatment.

In addition to establishing better prehospital care for potential neck injuries, we have also modified our immobilization protocol for mild to moderate SCIWORA patients in order to prevent recurrent injuries. Before 1985, we maintained all SCIWORA children in Philadelphia collars for 8 weeks and proscribed contact sports for the same period. We have found this protocol inadequate for several reasons. First, when children return to an often unsupervised home environment, compliance with neck immobilization and activity restrictions diminishes. The better the neurological status on discharge, the more likely it is for the child to discard the collar (since it is easily removable) and engage in vigorous play. Second, recurrent injury occurred in two children with the collar in place, suggesting that the collar was an inadequate immobilizer. Also, Philadelphia collars do not come in a size suitable for children younger than 2 years. Finally, re-injury in one child occurred 10 weeks after the initial injury, implying that 8 weeks of immobilization was insufficient. Since 1985, we immobilize all children with SCIWORA in a Guilford brace, which has adjustable chin and chest plates with occipital and back supports. The unit is usually well tolerated and can be tailored to conform to the child's size down to that of an infant. In addition to imparting greater limitation of neck movements than the Philadelphia collar, the brace is more difficult for the child to remove without assistance, and its cumbersome configuration also impedes vigorous activities. Bracing is maintained for 3 months instead of 2, and during this period, both non-contact as well as contact sports are strictly prohibited. All that aside, the most important aspect of this revised protocol is probably the stern discussions with the family and child about the serious nature of the injury and its potentially devastating complications. The child is followed frequently in the clinic where his neurological recovery and compliance with treatment are closely monitored. At the end of 3 months, spinal stability is again confirmed by yet another dynamic study before bracing is discontinued. With this stringent protocol, no recurrent injury has been encountered among 12 children with SCIWORA treated during the last 21 months.

REFERENCES

1. Abroms, I. F., Bresnan, M. J., Zuckerman, J. E., et al.: Cervical cord injuries secondary to hyperextension of the head in breech presentations. *Obstet. Gynecol.*, **41**: 369–378, 1973.
2. Ahmann, P. A., Smith, S. A., Schwartz, J. F., et al.: Spinal cord

infarction due to minor trauma in children. *Neurology*, **25:** 301–307, 1975.
3. Alexander, E., Jr., Davis, C. H., Jr., Field, C. H.: Hyperextension injuries of the cervical spine. *Arch. Neurol. Psychiat.*, **79:** 146–150, 1958.
4. Anderson, M. J., Schutt, A. H.: Spinal injury in children: A review of 156 cases seen from 1950 through 1978. *Mayo Clin. Proc.*, **55:** 499–504, 1980.
5. Andrews, L. G., Jung, S. K.: Spinal cord injuries in children in British Columbia. *Paraplegia*, **17:** 442–451, 1979.
6. Aufdermaur, M.: Spinal injuries in juveniles: Necropsy findings in twelve cases. *J. Bone Jt. Surg.*, **56-B:** 513–519, 1974.
7. Babcock J. L: Spinal injuries in children. *Pediat. Clin. No. Amer.*, **22:** 487–500, 1975.
8. Bailey, D. K.: The normal cervical spine in infants and children. *Radiology*, **59:** 712–719, 1952.
9. Baker D. H. Berdon, W. E.: Special trauma problems in children. *Radiol. Clin. No. Amer.*, **4:** 289–305, 1966.
10. Barnes, R.: Paraplegia in cervical spine injuries. *J. Bone Jt. Surg.*, **30-B:** 234–244, 1948.
11. Boltshauser, E., Isler, W., Bucher, H. U., et al.: Permanent flaccid paraplegia in children with thoracic spinal cord injury. *Paraplegia*, **19:** 227–234, 1981.
12. Bourmer, H. R.: Zur Frage der Halsmarkschadigung bei Hyperextensionsverletzungen der Wirbelsaule. *Arch. Klin. Chir.*, **268:** 409–416, 1951.
13. Braakman, R., Penning, L.: The hyperflexion sprain of the cervical spine. *Radiol. Clin.*, **37:** 309–320, 1968.
14. Braakman, R., Penning, L.: *Injuries of the Cervical Spine.* Amsterdam, Excerpta Medica, 1971.
15. Breig, A., El-Nadi, A. F.: Biomechanics of the cervical spinal cord: Relief of contact pressure on and over-stretching of the spinal cord. *Acta Radiol. (Diag.)*, **4:** 602–624, 1966.
16. Burke, D. C.: Spinal cord trauma in children. *Paraplegia*, **9:** 1–14, 1971.
17. Burke, D. C.: Traumatic spinal paralysis in children. *Paraplegia*, **11:** 268–276, 1974.
18. Caffey, J.: On the theory and practice of shaking infants: Its potential residual effects of permanent brain damage and mental retardation. *Am. J. Dis. Child.*, **124:** 161–169, 1972.
19. Caffey, J.: The whiplash shaken infant syndrome: Manual shaking by the extremities with whiplash-induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation. *Pediatrics*, **54:** 396–403, 1974.
20. Campbell, J., Bonnett, C.: Spinal cord injury in children. *Clin. Orthop.*, **112:** 114–123, 1975.
21. Cattell, H. S., Filtzer, D. L.: Pseudosubluxation and other normal variations in the cervical spine in children: A study of one hundred and sixty children. *J. Bone Jt. Surg.*, **47-A:** 1295–1309, 1965.
22. Cheshire, D. J. E.: The pediatric syndrome of traumatic myelopathy without demonstrable vertebral injury. *Paraplegia*, **15:** 74–85, 1977.
23. Choi, J.-U., Hoffman, J. H., Hendrick, E. B., et al.: Traumatic infarction of the spinal cord in children. *J. Neurosurg.*, **65:** 608–610, 1986.
24. Dunlap, J. P., Morris, M., Thompson, R. G.: Cervical-spine injuries in children. *J. Bone Jt. Surg.*, **40-A:** 681–686, 1958.
25. Forni, I.: Le fratture del rachide nel bambino. *Chir. Org. Movimento*, **31:** 347–361, 1947.
26. Gelehrter, G.: Fracture of the vertebrae in children and adolescents. *Arch. Orthop. Unfallchir.*, **49:** 253–263, 1957.
27. Gilles, F. H., Bina, M., Sotrel, A.: Infantile atlantoccipital instability: The potential danger of extreme extension. *Am J. Dis. Child.*, **133:** 30–37, 1979.
28. Glasauer, F. E., Cares, H. L.: Biomechanical features of traumatic paraplegia in infancy. *J. Trauma*, **13:** 166–170, 1973.
29. Hachen, J. H.: Spinal cord injury in children and adolescents: Diagnostic pitfalls and therapeutic considerations in the acute stage. *Paraplegia*, **15:** 55–64, 1977.
30. Hadley, M. N., Zabramski, J. M., Browner, C. M., et al: Pediatric spinal trauma: Review of 122 cases of spinal cord and vertebral column injuries. *J. Neurosurg.*, **68:** 18–24, 1988.
31. Hasue, M., Hoshino, R., Omata, S., et al.: Cervical spine injuries in children. *Fukushima J. Med. Sci.*, **20:** 115–123, 1974.
32. Hubbard, D. D.: Injuries of the spine in children and adolescents. *Clin. Orthop.*, **100:** 56–65, 1974.
33. Kewalramani, L. S., Kraus, J. F., Sterling, H. M.: Acute spinal cord lesions in a pediatric population: Epidemiological and clinical features. *Paraplegia*, **18:** 206–219, 1980.
34. Kraus, J. F.: Epidemiological features of head and spinal cord injury. *In* Schoenberg, S. (ed): *Neurological Epidemiology: Principles and Clinical Applications.* New York, Raven Press, 1978, pp. 261–279.
35. Leventhal, H. R.: Birth injuries of the spinal cord. *J. Pediat.*, **56:** 447–453, 1960.
36. Marar, B. C.: Hyperextension injuries of the cervical spine: The pathogenesis of damage to the spinal cord. *J. Bone Jt. Surg.*, **56-A:** 1655–1662, 1974.
37. Melzak, J.: Paraplegia among children. *Lancet*, **2:** 45–48, 1969.
38. Pang, D., Wilberger, J. E.: Spinal cord injury without radiographic abnormalities in children. *J. Neurosurg.*, **57:** 114–129, 1982.
39. Papavasiliou, V.: Traumatic subluxation of the cervical spine during childhood. *Orthop. Clin. No. Amer.*, **9:** 945–954, 1978.
40. Robson, P. N.: Hyperextension and haematomyelia. *Br. Med. J.*, **2:** 848–852, 1956.
41. Ruge, J. R., Sinson, G. P., McLone, D. G., et al.: Pediatric spinal injury: The very young. *J. Neurosurg*, **68:** 25–30, 1988.
42. Scher, A. T.: Trauma of the spinal cord in children. *S. Afr. Med. J.*, **50:** 2023–2025, 1976.
43. Scher, A. T.: Anterior cervical subluxation: An unstable position. *AJR*, **133:** 275–280, 1979.
44. Sherk, H. H., Schut, L., Lane, J. M.: Fractures and dislocations of the cervical spine in children. *Orthop. Clin. No. Amer.*, **7:** 593–604, 1976.
45. Sullivan, C. R., Bruwer, A. J., Harris, E.: Hypermobility of the cervical spine in children: A pitfall in the diagnosis of cervical dislocation. *Am. J. Surg.*, **95:** 636–640, 1958.
46. Taylor, A. R.: The mechanism of injury to the spinal cord in the neck without damage to the vertebral column. *J. Bone Jt. Surg.*, **33-B:** 543–547, 1951.
47. Taylor, A. R., Blackwood, W.: Paraplegia in hyperextension cervical injuries with normal radiographic appearances. *J. Bone Jt. Surg.*, **30-B:** 245–248, 1948.
48. Teng, P., Papatheodorou, C.: Traumatic subluxation of C2 in young children. *Bull. Los Angeles Neurol. Soc.*, **32:** 197–202, 1967.
49. Tondury, G.: The cervical spine: Its development and changes during life. *Acta Orthop. Belg.*, **25:** 602–607, 1959.
50. Towbin, A.: Spinal injury related to the syndrome of sudden death ("crib-death") in infants. *Am. J. Clin. Pathol.*, **49:** 562–567, 1968.
51. Townsend, E. H., Jr., Rowe, M. L.: Mobility of the upper cervical spine in health and disease. *Pediatrics*, **10:** 567–573, 1952.
52. von Torklus, D., Gehle, W.: *The Upper Cervical Spine.* New York, Grune & Stratton, 1972, pp. 10–94.
53. Walsh, J. W., Stevens, D. B., Young, A. B.: Traumatic paraplegia in children without contiguous spinal fracture of dislocation. *Neurosurgery*, **12:** 439–445, 1983.
54. Webb, J. K., Broughton, R. B. K., McSweeney, T., et al.: Hidden flexion injury of the cervical spine. *J. Bone Jt. Surg.*, **58-B:** 322–327, 1976.
55. Wilberger, J. E., Alba, A., Maroon, J. C.: Burning hands syndrome revisited. *Neurosurgery*, **19:** 1038–1040, 1986.