

## The Acute and Chronic Management of Spinal Cord Injury

Eric Belanger, MD, Allan DO Levi, MD, PhD

Spinal cord injury (SCI) remains a devastating injury for both patients and their families. Improvements in the quality of care over the last few decades partially reflect specialized medical centers that focus on the acute treatment and rehabilitation of patients with SCI and are best equipped to provide the magnitude of service these patients require. The prevalence of SCI is increasing steadily because of improved survival in both the acute and chronic stages of the disease. Advances in acute treatment include more sophisticated prehospital care, prompt recognition of the signs of SCI, safer transportation methods, and active resuscitation both in the field and in the emergency department. Improvements in the treatment of the chronic stages of the disease, such as the surgical management of syringomyelia, late posttraumatic deformity, and pain control, have been achieved. Increased survival has prompted the health care industry to develop strategies to enhance the quality of life, with improvements ranging from lighter wheelchairs to the development of fertility programs for patients with SCI.

The mechanism of SCI is in constant evolution. With industrialization, motor vehicle accidents have become the leading cause of spinal trauma. SCI because of violence is on a dramatic rise, as manifested by the proportion of individuals injured by assault, including penetrating injuries such as gunshot and knife wounds. Sports-related injuries, which include football, horseback riding, and hockey, have received recent media attention.<sup>1</sup> Recreational injuries from diving, snowmobile acci-

dents, and parachuting are a constant source of newly injured patients. Because of the desire for extreme speed, both on land and in the air, we expect the emergence of new injuries from recreational activities, such as with snowboarding, water craft, and others.

Preventive programs, which encourage children and young adults to modify risky behaviors, have the greatest prospect of reducing the incidence of SCI. These include, but are not limited to, the Think First program sponsored by the American Association of Neurological Surgeons and the Congress of Neurological Surgeons,<sup>2</sup> and the Feet First, First Time program initially developed in northern Florida, which encourages water enthusiasts to jump feet first into unknown waters. Driver's-education courses and police patrol, to arrest drivers in command of vehicles while under the influence of drugs or alcohol, also can help decrease these unfortunate events. Finally, regulation of handguns and assault weapons can reduce the number of intentional and accidental injuries.

Determining the prognosis for SCI patients on admission remains challenging. The clinician uses neurologic examination, age, MRI appearance of the cord, and other clinical data to counsel patients and families on the expected outcomes for a specific injury. Some recovery is the rule for most patients who enter the hospital with an incomplete SCI, but when patients present with complete injuries, the chance of regaining ambulatory function remains slim.<sup>3</sup>

SCI research is an absolute priority of the National Institutes of Health. Models of SCI, mechanisms of secondary injury, treatment of the acute phase of SCI, and the development of transplantation strategies to repair the damaged spinal cord are

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From the Department of Neurosurgery and the Miami Project to Cure Paralysis, University of Miami, Miami, FL.  
Correspondence address: Allan DO Levi, MD, PhD, Department of Neurosurgery, University of Miami, 1501 NW 9th Ave, Ste 2011, Miami, FL 33136.

ongoing research efforts across the continent and around the world. The treatment arms of the research can be divided into two broad categories: pharmacologic and transplantation strategies. Drugs that can be given during the acute phase of injury that may limit secondary injury mechanisms or promote regeneration. Two of the most promising drugs, methylprednisolone and monosialic ganglioside GM-1, have yielded only modest results. Methylprednisolone, which is used in almost all SCI centers in North America, is coming under closer scrutiny as to its effectiveness.<sup>4</sup> Drugs of the future include neurotrophins, which can promote the survival and regeneration of injured nerve cells, drugs that prevent the inflammatory response to SCI,<sup>5</sup> and drugs that prevent apoptotic cell death.<sup>6</sup> In the transplantation arena, cellular therapies to treat the chronic injury are important. Cells of interest include Schwann cells, olfactory ensheathing glia, embryonic spinal cord, and neural progenitor cells. Antibodies that neutralize the inhibitory proteins within myelin have also demonstrated promise. Strategies that combine a number of the cited treatments are most likely to have a beneficial effect in the future.

## EPIDEMIOLOGY

### Incidence

The annual incidence of SCI varies widely among countries and ranges from 6 per million<sup>7</sup> to 57.8 per million.<sup>8</sup> This wide range also reflects the inclusion in some published statistics of patients who were diagnosed with SCI but who died by the time they arrived at the hospital, which may represent as many as 16%.<sup>8</sup> On average, 10,000 new cases of SCI are diagnosed in the United States each year.<sup>9</sup> Males account for roughly 71% to 80% of patients treated with SCI. The percentages of patients with paraplegia (incomplete or complete) versus tetraplegia (incomplete or complete) have changed minimally over the last 20 years and are 62% and 38%, respectively.<sup>10</sup> On average, the age at the time of injury is in the early 30s (31.5 years). Table 1 summarizes the incidence of SCIs.

### Mechanism of injury

The most common mechanisms of injury reported among the 2,814 cases in the National Spinal Cord Injury Statistical Center database are motor vehicle

**Table 1. Epidemiology of Spinal Cord Injury**

Characteristic	Estimate
New cases in the US per year	10,000
Males	71% to 80%
Average age at injury (y)	31.5
Mechanisms	
1. Motor vehicle accidents	35.9%
2. Violence	29.5%
3. Falls	20.3%
Average cost	
First year	\$185,019–295,643
Subsequent years	\$17,275–33,439
Total annual cost	\$7.736 billion

accidents, accounting for 35.9% of injuries, followed by violence, falls, and sports-related injuries, which account for 29.5%, 20.3%, and 7.3%, respectively.<sup>9</sup> In the United States, an increasing proportion of SCIs is related to violent acts. From 1973 to 1978 until 1991 to 1995, the incidence of violent SCIs increased from 13.3% to 29.5%.<sup>9</sup> According to DeVivo,<sup>9</sup> assaults including gunshot and stab wounds are now the second most common cause of SCI in the United States.

### Survival

Age, neurologic level, and Glasgow Coma Scale (GCS) are known to be independent predictors of mortality in the first 3 months after SCI.<sup>11</sup> After the acute period, SCI patients still have a shortened life expectancy compared with their uninjured peers. Even if we exclude those who die in the first 18 months, the 25-year survival rate is 80% for paraplegic (complete or incomplete) patients and 72% for tetraplegic (complete or incomplete) patients.<sup>13</sup> In the last five decades, the causes of delayed mortality have changed significantly. In the past, urosepsis was the leading cause of death, but now this has been supplanted by respiratory problems, heart disease, and suicide.<sup>13</sup> Among longterm paraplegic survivors, the suicide rate is 10 times that of their uninjured peers.<sup>14</sup>

### Cost

The cost of treatment of patients with SCI is estimated to be between \$185,019 and \$295,643 for the first year after injury and between \$17,275 and \$33,439 per year for subsequent years. Higher costs are linked to the level of injury, with the highest costs associated with ventilator-dependent tetraple-

gic patients. The estimated lifetime costs of caring for a paraplegic and a tetraplegic are \$0.5 and \$2 million, respectively. In 1995, the total annual cost of caring for patients with SCI in the United States was estimated to be \$7.736 billion.<sup>9</sup>

## PREHOSPITAL MANAGEMENT

### Avoidance of hypoxia and hypotension

The care of an injured patient begins with airway, breathing, and circulation (ABCs). The prompt recognition of SCI is critical because it may be one of the most important determinants of the final outcomes. The primary injury to the spinal cord is sustained at the time of impact and is irreversible. The role of all intervening care givers, ranging from a passing good Samaritan to the receiving surgeon, will be to prevent and minimize secondary injury.

Minimizing secondary injury begins in the field with the initial resuscitation. Patients should receive oxygen supplementation. If the airway is compromised, options for intubation include nasotracheal, orotracheal with in-line traction, or cricothyroidotomy, if these two fail. Injuries above T6 effectively reduce sympathetic tone, so unopposed vagal effects include hypotension and bradycardia. Initiating an IV line will permit a fluid bolus and counteract the frequent hypotension associated with SCI.

Vale and colleagues<sup>15</sup> reported that an aggressive medical protocol, which should be followed in every SCI patient, is to maintain the mean arterial BP above 85 mm Hg. This protocol "enhanced neurologic outcome."<sup>15</sup>

### Immobilization of the cervical spine

Over the years, spinal immobilization in the field has become a standard of care in the United States.<sup>16,17</sup> This includes placement of a hard cervical collar, transportation on a rigid spine board, and log rolling of the patient. All trauma victims, including patients with an altered mental status, head injury, neurologic deficits, back or neck pain, intoxication, multisystem trauma, or suspicion of spinal injury, should be immobilized. Improper immobilization may cause fracture displacement, which can further compromise spinal cord function and convert a good-prognosis injury into a devastating and a life-threatening one. Approximately 10% of patients with spinal fractures will have a fracture at

**Table 2. Summary of the Prehospital Management for Spinal Cord Injury**

Objective	Management
Avoid hypoxia	Supplement with O <sub>2</sub> Intubate as necessary
Avoid hypotension	Control blood loss Maintain mean arterial BP > 85 mm Hg
Immobilize	Cervical collar Spine board
Transport	To a hospital with spinal cord injury expertise

another level, so careful immobilization is required for all spinal levels until x-rays confirm the diagnosis.

### Transfer to an SCI center

Ideally, patients with SCI should be transferred to a level I trauma center affiliated with a unit that cares for a large volume of SCI patients. DeVivo and coworkers<sup>18</sup> studied the implications of admitting directly to a general hospital, with later referral to a center with expertise in managing SCI, versus direct admission to an SCI center. They found a significant reduction in pressure sores and shorter lengths of stay for patients admitted directly to centers with expertise in SCI. A larger volume of patients in a specialized SCI unit promises increased expertise from physicians (eg, surgeons, anesthesiologists, intensivists, physiatrists), nursing personnel, and paramedical professionals. Table 2 summarizes the prehospital management.

## EMERGENCY ROOM MANAGEMENT

On arrival at the emergency room, the ABCs are again a priority. Simultaneous treatment and evaluation should proceed, which includes a trauma and a complete neurologic examination. The physical examination should include an assessment of the neck and back. The physician can remove the hard collar and palpate the back and the neck to examine for deformity or pain. Patients with blunt trauma who are alert (GCS 14 or 15) and do not complain of neck pain have a low incidence of cervical fractures. Gonzalez and colleagues<sup>20</sup> clinically examined 2,176 consecutive patients with blunt trauma with a GCS of 14 or 15 and found a low incidence (1.6%) of spine fractures.

### Neurologic examination

A carefully done initial neurologic examination is critical. It allows the admitting physician to direct subsequent radiologic investigations to the appropriate level and will serve as a baseline for further comparison. It is imperative to repeat the neurologic examination hourly and after subjecting the patient to traction or a bed transfer. Up to 4.9%<sup>20</sup> of patients will deteriorate after admission. One of the indications for operation that most surgeons agree upon is for cord decompression in a patient with neurologic deterioration. The neurologic examination on admission remains one of the best predictors of outcomes. An accurate knowledge of sensory dermatomes and muscle innervation (Fig. 1) permits one to determine the level of the SCI.

### American Spinal Injury Association standards

Over the last decade, the American Spinal Injury Association (ASIA) and the International Medical Society of Paraplegia have developed and revised a scale that has facilitated communication among clinicians about the neurologic status of the patient.<sup>21</sup> In an attempt to standardize assessments of the SCI patient, ASIA has published international standards.<sup>21</sup> In this classification, the term *tetraplegia* is used rather than quadriplegia to define "impairment or loss of motor and/or sensory function in the cervical segments of the spinal cord."<sup>21</sup> *Paraplegia* refers to "impairment or loss of motor and/or sensory function in the thoracic lumbar or sacral segments of the spinal cord."<sup>21</sup> *Incomplete injury* is used to describe the SCI patient with preservation of motor or sensory function below the level of injury. When all function is lost, including loss of rectal, motor, and sensory function, the injury is deemed complete. In the patient with a complete injury, the dermatomes and myotomes caudal to the neurologic level that remain partially innervated are named the "zone of partial preservation."

The sensory examination should be performed by assessment of light touch and pinprick in 28 defined areas (Fig. 1). The motor examination should be performed by testing 10 key muscle groups bilaterally on a scale of 0 (complete loss of function) to 5 (normal strength) (Table 3). ASIA defines the motor level as the level with at least grade 3/5 strength, provided that the next most rostral level key muscle has 5/5 strength. At the com-

pletion of the neurologic examination, the patient is classified according to the ASIA system (Table 4). Sometimes, the SCI can be further classified according to a specific syndrome. This subsequent classification has some prognostic importance (Table 5).<sup>22-25</sup> Figure 2 illustrates an uncomplicated case of a complete C5 quadriplegia.

### IMAGING IN SPINAL TRAUMA

There is controversy as to which investigations are required to detect a clinically significant SCI or how many and what kind of x-rays are required to "clear" the spine. Plain x-rays, CT scans, and MRIs should be used in combination as directed by the patient's clinical condition and by the resources available at the treating institution.

#### X-rays

The initial radiologic investigation should include cervical spine x-rays, including anteroposterior and lateral films to include the C7 to T1 junction and an open-mouth view demonstrating the odontoid process of C2 and lateral masses of C1. Swimmer's views may be required if the C7 to T1 junction is not visualized on the initial lateral x-ray. This collection of x-rays is frequently called a "trauma series." The cervical spine x-rays remain the first line of investigation for evaluation of the cervical spine in a trauma patient. They are recommended for any trauma patient except for the alert, nonintoxicated, and neurologically intact patient without neck pain.

A single lateral cervical spine x-ray to rule out injury has been shown to be less than acceptable. The sensitivity reported by MacDonald and associates<sup>26</sup> for lateral views of the cervical spine, including the swimmer's view, was 83%. In another study, Woodring and Lee<sup>27</sup> found that a cross-table lateral film and trauma series detected only 68% and 77% of the patients with cervical fractures, respectively. Most of the missed fractures were at C2 and the C7 to T1 junction. Although many of the fractures that are missed using plain films do not necessarily lead to delayed neurologic deficit, we need to improve our diagnostic sensitivity by using additional techniques.

#### CT

CT scans in the setting of spinal trauma can significantly enhance the diagnostic yield for fracture and

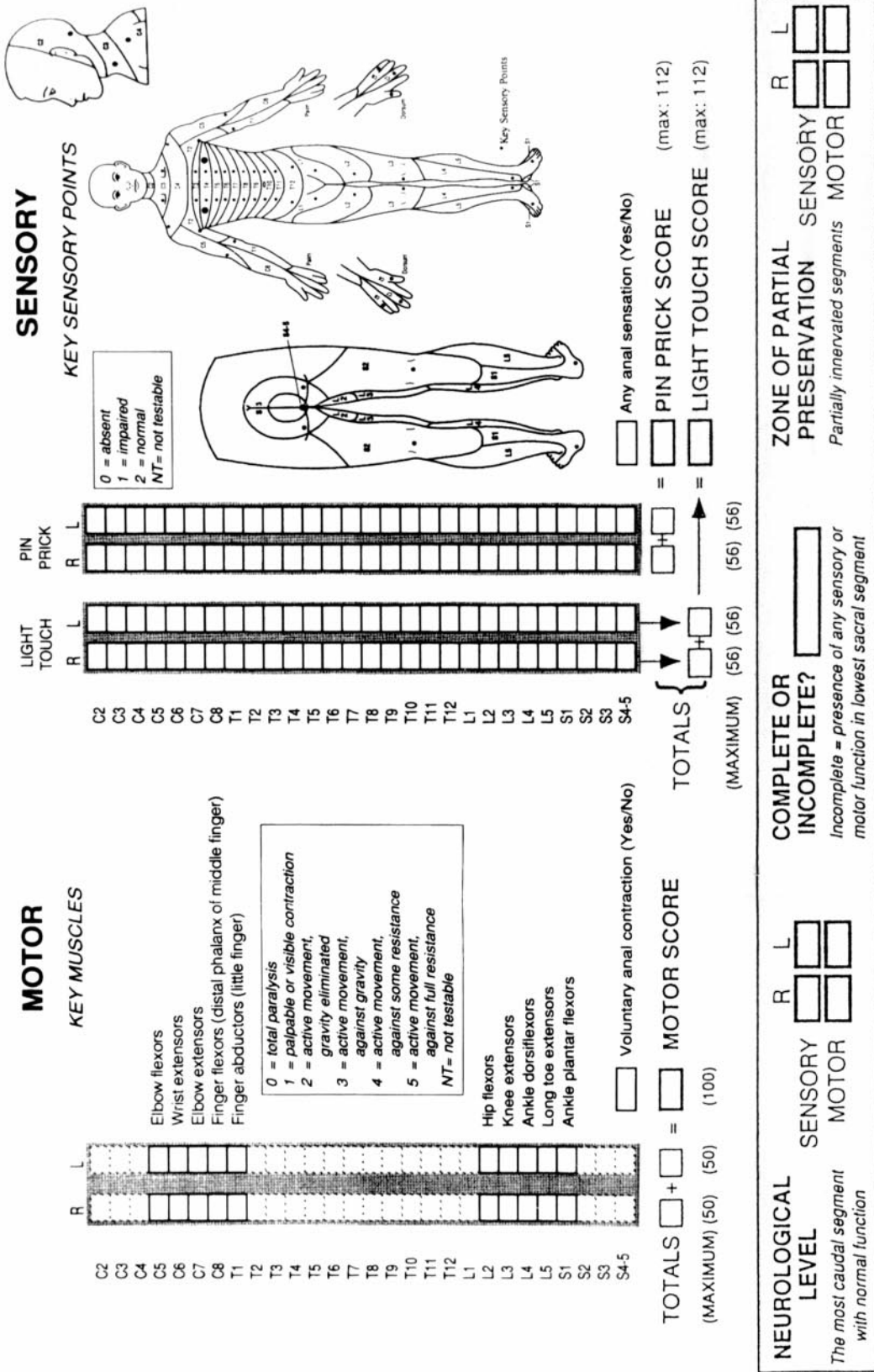


Figure 1. American Spinal Injury Association (ASIA) "Standard Neurological Classification of Spinal Cord Injury." Image may be copied freely but should not be altered without permission from the ASIA.

Table 3. Key Muscles According to the American Spinal Injury Association

Key muscle groups	Roots	Movement	Muscles
1	C5	Elbow flexion	Biceps, brachialis
2	C6	Wrist extension	Extensor carpi radialis longus and brevis
3	C7	Elbow extension	Triceps
4	C8	Finger flexion, middle finger	Flexor digitorum profundus
5	T1	Small finger abductors	Abductor digiti minimi
6	L2	Hip flexor	Iliopsoas
7	L3	Knee extensors	Quadriceps
8	L4	Ankle dorsiflexion	Tibialis anterior
9	L5	Long toe extensors	Extensor hallucis longus
10	S1	Ankle plantar flexion	Gastrocnemius, soleus

have largely supplanted tomography in fracture detection. With the advent of helical CT, the time required for imaging has been reduced. In a study by Nunez and coauthors,<sup>28</sup> the addition of CT scanning in the evaluation of trauma patients resulted in more rapid clearance of the cervical spine and detection of some fractures that would have been missed by plain x-ray. In their retrospective study, they found that 32 of 88 patients had cervical fractures that were not revealed or were incompletely demonstrated without helical CT.

### MRI

In a patient with a concomitant severe head injury, the cervical spine becomes more difficult to clear. If a fracture is not detected by plain x-rays or cervical CT scans, ligamentous injuries alone may be present and result in cervical instability. In the comatose, sedated, or uncooperative patient, flexion-extension x-rays of the cervical spine may provide insight into ligamentous stability but are risky, and their use is discouraged. MRI can be used to assess ligamentous injury. Recently, D'Alise and colleagues<sup>29</sup> used MRIs of the cervical spine in 121

comatose or obtunded trauma patients. D'Alise and colleagues<sup>29</sup> found that 25.6% of the patients had pronounced soft-tissue injuries and 6.6% of the patients required surgical treatment. None of these injuries was identified by the trauma series. In contrast, Klein and associates<sup>30</sup> found that MRI could miss up to 90% of posterior-element fractures detected by CT scan. The time, cost, and the manipulation required for transport to MRI are limiting factors in some critical-care patients.

MRI can be useful both as a diagnostic and prognostic tool. It is clearly the gold standard for imaging any spinal cord pathology. Hematomas, traumatic disc herniation, or associated soft tissue injury can significantly influence the treatment plans for patients with SCI, and MRI is required in most of these patients.

### PATHOPHYSIOLOGY

The "primary" injury is defined as the one that occurs immediately at the scene of the accident. If the forces are of sufficient magnitude to overcome the resistance of the osteoligamentous structures of the spine, the energy is transmitted to the spinal cord, creating the primary injury. These forces may result in contusion, hemorrhage, shear, or laceration of the spinal cord.<sup>31</sup>

"Secondary" injury processes follow the primary injury and include disruption of the microcirculation, loss of autoregulation, edema, anoxia, and ischemia. These may be followed by calcium toxicity, lipid peroxidation, or free-radical activation. Some of the cells submitted to these processes may ultimately die, potentially contributing to the pa-

Table 4. Injury Classification According to the American Spinal Injury Association

Class	Explanation
A	Complete motor and sensory loss
B	Incomplete sensory preservation, complete motor loss
C	Incomplete motor loss with more than 50% of muscles with strength < 3/5
D	Incomplete motor loss with more than 50% of muscles with strength > 3/5
E	Normal

**Table 5. Spinal Cord Injury Syndromes**

Syndrome	Description
Acute traumatic central cord syndrome and cruciate paralysis <sup>22</sup>	Disproportionate weakness of both arms and hands with relative preservation of leg function
Brown-Séquard syndrome <sup>23</sup>	Physiologic hemisection of the spinal cord Ipsilateral reduction or loss of proprioception and motor function Contralateral reduction or loss of pain and temperature
Anterior spinal cord syndrome	Preservation of proprioception with bilateral loss of motor and other sensory function below the level of injury
Conus medullaris syndrome	Mixture of upper and lower motor neuron findings with prominent sphincter disturbance
Cauda equina syndrome	Injury to nerve roots (lower motor neuron) arising from the conus with distal lower-extremity weakness
Spinal cord concussion <sup>24,25</sup>	Transient motor and sensory impairment with complete spontaneous recovery in less than 48 h. These injuries are often sports related and often resolve in less than 15 min.
SCIWORA	A condition observed mainly in children and young adults. Traditional radiographs are negative for fracture or subluxation, but the MRI may show signal change in the spinal cord or spinal ligaments and soft tissues. The prognosis for these injuries is good.

SCIWORA, spinal cord injury without radiographic abnormality.

thology of the initial injury or explaining incomplete neurologic recovery after injury (Fig. 3).

Some of the initially surviving cells may activate intracellular proteases, ultimately leading to self-destruction. Apoptosis<sup>6</sup> is known to occur in a variety of neurologic disorders, including human SCI. In the future, protease inhibitors may be able to halt apoptotic cell death and may have the potential to be used in the clinical arena.

## MEDICAL TREATMENT

### Steroids

Steroids have proved beneficial in acute SCI in at least two randomized, prospective, blinded, multicenter trials: the National Acute Spinal Cord Injury Study (NASCIS) II<sup>32</sup> and III.<sup>33</sup> In the subgroup of patients treated within 8 hours of injury, NASCIS II demonstrated statistically significant improvements in neurologic recovery. Methylprednisolone was administered at 30 mg/kg as a loading dose over the first 15 to 30 minutes, followed by 5.4 mg/kg for the next 23 hours. No benefit was observed for those who received their first dose of steroid later than 8 hours. This study led to the widespread use of steroids for the treatment of SCI patients in North America. In 1997, NASCIS published their third study.<sup>33</sup> Their recommendation for patients who could receive the drug within 3 hours of injury was to give the NASCIS II protocol (24 hours of treatment). Patients who began treatment between

3 and 8 hours obtained better recovery if they received the drug for 48 hours. The slight gains in neurologic recovery were not without risk; for instance, the 48-hour treatment group had significantly higher rates of sepsis (2.6%) and pneumonia (5.6%) compared with the 24-hour group (0.6% and 2.6%, respectively).

The two NASCIS studies have been and will continue to be highly criticized. Nesathurai<sup>4</sup> has argued that the studies are statistically flawed, contain ambiguous results, and have poor definition of end points. In his article, he describes how a patient can appear to be improved with the motor scale system used in the study without having any clinically significant improvement. Nesathurai<sup>4</sup> has demanded that the medical community organize an "impartial blue-ribbon panel of clinicians and statisticians to reanalyze the NASCIS II and III primary data."

### Monosialic Ganglioside GM-1

Gangliosides have proved to enhance recovery after SCI.<sup>34,35</sup> In a randomized, prospective, placebo-controlled trial on GM-1 done in Maryland, the recovery rate was significantly improved compared with the control group.<sup>35</sup> The recovery rate to strength of 3/5 or 4/5 for muscles that were initially paralyzed was 51.7% in the treated group and 25.3% in the control group. In contrast, weak, non-paralyzed muscles recovered to the same extent in



**Figure 2.** Case illustration: A 30-year-old man sustained a severe fracture dislocation of C5 on C6. On admission, the strength of his deltoids was 4/5 and his biceps were 2/5, with no voluntary movement below that level. Sensory examination demonstrated bilateral sensation to pinprick and light touch on the lateral part of the shoulder (C5) and on his thumb (C6). He had no sensation below. He had no rectal tone and had lack of sparing of sacral sensation. This patient sustained a complete C5 spinal cord injury (American Spinal Injury Association class A).

both groups. The study included only 34 patients and, in the next few months, the results of the multicenter trial should be available.

## SURGICAL TREATMENT

### Options

The shortterm goal of the surgeon who treats SCI patients is to place the spinal cord and nerves in an optimal milieu for recovery. This may be accomplished by decompressing neurologic structures or correcting bony deformities. To accomplish these tasks, strategies range from an external orthosis to surgical intervention. The best treatment for each fracture type must be considered on an individual basis. Factors influencing the decision include the

experience of the surgeon, the chance of fusion with external orthosis versus operation, the degree of spinal canal compromise or deformity, and the individual's compliance with recommendations. An individual fracture may have more than one surgical solution. For example, bilateral cervical facet fracture dislocations can be managed with posterior fixation using lateral mass plates or interspinous cables<sup>36</sup> or with an anterior approach using a bone graft and unicortical anterior plates.<sup>37</sup> The details of managing individual spine fractures are beyond the scope of this article.

If x-rays demonstrate spinal malalignment in the cervical area, traction can be applied for both stabilization and reduction of the fracture. There is evidence from animal studies that early decompression can lead to substantial neurologic recovery.<sup>38</sup> For most cervical fractures presenting with malalignment and cord compression (Fig. 4), an initial attempt at closed reduction should be pursued. Traction is applied to the head with Gardner-Wells tongs, and reduction is accomplished either manually with fluoroscopy or by successively adding more weight to the traction. One has to be very careful to avoid overdistraction. Frequent followup radiographic and neurologic examinations are necessary. The classic teaching of a maximum of 5 lb of traction per cervical level sometimes underestimates the weight required to achieve reduction. Cotler and associates<sup>39</sup> used up to 140 lb of traction to achieve reduction of some cervical dislocations. They did not document any complications related to this practice. Occasionally, closed reduction with traction is not possible and an open reduction and fixation is required. In many patients, closed reduction is followed by internal fixation and by bone grafting.

Once the malalignment is externally reduced, the timing of subsequent interventions is also important. If the injury is judged to have a good potential for healing without operation, the SCI patient may be placed in a hard collar or a halo vest and will benefit from aggressive early mobilization. If surgical treatment is elected, one must rely on the scarce retrospective data available on that subject to plan the proper timing of the intervention. This issue is controversial and is reviewed in the paragraphs below.

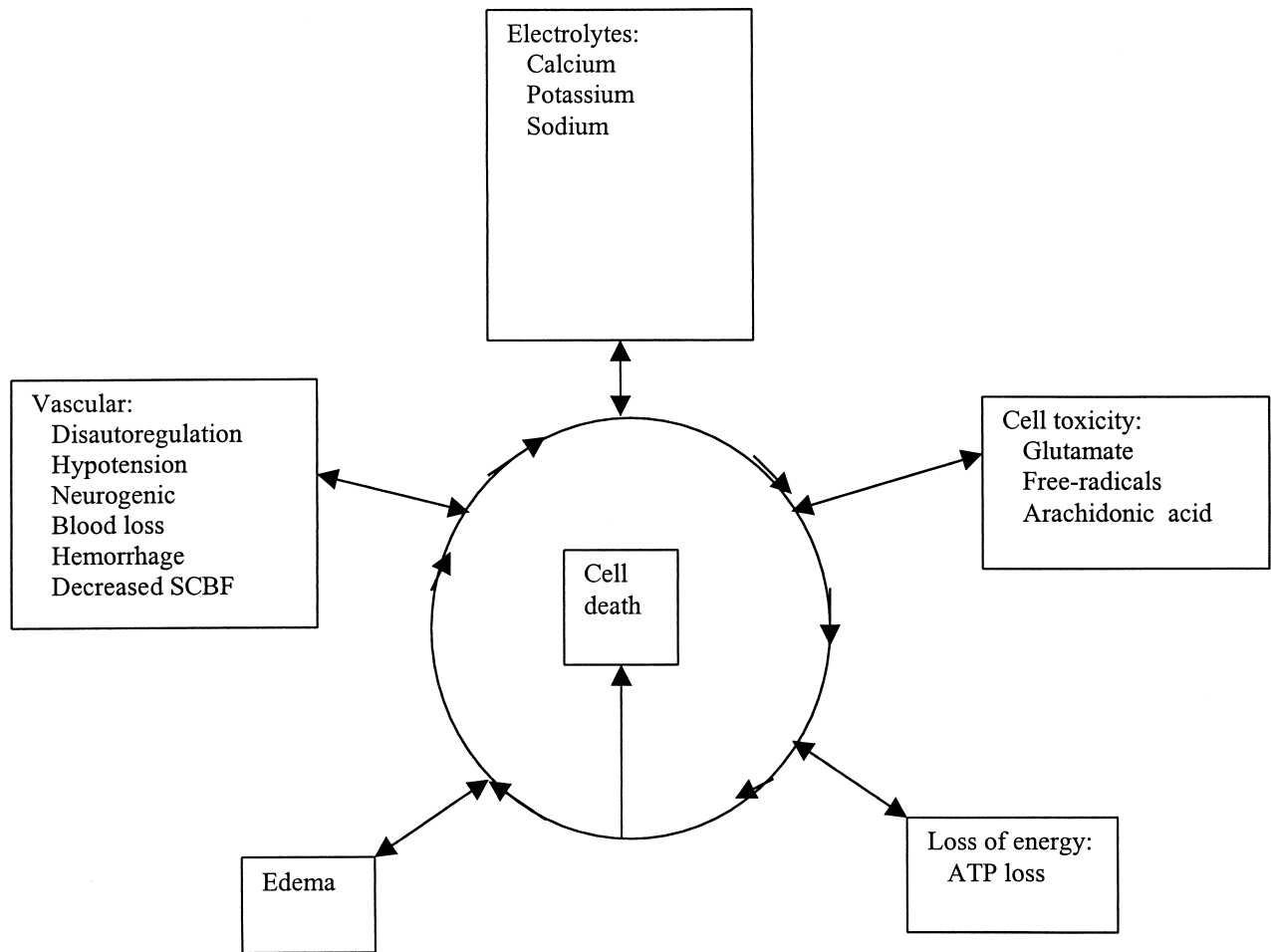


Figure 3. Secondary injury mechanisms. ATP, adenosine triphosphate; SCBF, spinal cord blood flow.

### Timing of surgery

**Human data.** In a recent review of the literature, Fehlings and Tator<sup>40</sup> were unable to draw any definite conclusions on the correct timing of surgical intervention for SCI. They found only class II evidence that operations earlier than 25 hours or later than 200 hours are equally safe and effective. Duh and coworkers<sup>41</sup> examined the data from NASCIS II and found a suggestion of worse outcomes for patients operated on between 25 and 200 hours. It was hypothesized that the spinal cord may be maximally edematous between day 2 and day 4 and more susceptible to damage during surgical manipulation. A randomized, prospective study was conducted by Vaccaro and colleagues<sup>42</sup> on the timing of operations for SCI. They randomized their acute-injury group to either early (less than 72 hours) or

late (later than 5 days) operation. They did not find any differences in neurologic outcomes between the groups.

**Animal data.** Animal studies have demonstrated benefit from early decompression in incomplete SCI.<sup>43</sup> Delamarter and associates<sup>43</sup> used a spinal cord compression model in dogs to study the recovery rate after decompression at different times after a compressive insult. Dogs decompressed immediately and at 1 hour had significantly better recovery than those decompressed at 6 hours or later. The animals treated later did not achieve any notable recovery and underwent progressive spinal cord necrosis. If we extrapolate from the animal data, the window of opportunity for decompression appears to be very short (a few hours). To achieve decompression either operatively or nonoperatively



**Figure 4.** Sagittal proton density MRI of the cervical spine after acute trauma. Severe cord compression resulting from the fracture dislocation is evident. This patient has a severe spinal cord injury (American Spinal Injury Association class A).

in the first hours after injury is difficult. One should add the time needed for transportation to the hospital, initial systemic stabilization, radiologic investigations, and the time required for attaining the decompression.

## PREDICTING OUTCOMES

### Acute mortality rate

The acute mortality rate for cervical SCI within the first 3 months is approximately 21%.<sup>11</sup> In the acute setting, age, neurologic level, and GCS are known to be independent predictors of mortality.

### Clinical examination as a predictor of outcomes

The 3-day neurologic examination appears to be one of the most accurate and practical predictors of outcomes.<sup>44,45</sup> In the emergency room, the neurologic examination can be tainted by alcohol, drugs, medications, shock, and other factors. It is not uncommon to see marked improvement in the first 24 hours after injury. In general, in complete tetraplegia (ASIA class A), we can expect the recovery of one root with muscle strength graded as 1/5 or 2/5, and some patients will recover to 3/5.<sup>46</sup> In a study by Maynard and associates,<sup>45</sup> none of the cognitively intact patients without sensory or motor function at 72 hours was ambulating at 1 year. Motor-complete and sensory-incomplete tetraplegia (ASIA class B) have been divided into two groups by Crozier and colleagues.<sup>47</sup> In the group of patients with pin appreciation and light touch, eight of nine patients became ambulatory. In the group with only light touch appreciation, only 2 of 18 became ambulatory. Within the incomplete injury group (ASIA class C), Burns<sup>48</sup> found a 91% and 42% ambulatory rate for patients younger and older than 50 years, respectively. In this latter study,<sup>48</sup> all ASIA class D patients became ambulatory at the time of discharge from rehabilitation. Maynard and coworkers<sup>45</sup> reported ambulatory rates 1 year after injury as predicted by the 72-hour neurologic examination; they were 0% for complete injuries, 47% for sensory incomplete injuries, and 87% for the motor incomplete injuries (Table 6).

### Age and SCI syndrome as predictors of outcomes

Age is also strongly associated with recovery. As a rule, for the same injury, younger patients recover significantly more often than older individuals, with a significantly lower general complication rate. DeVivo and coworkers<sup>49</sup> found that patients older than 61 years were 2.1, 2.7, and 5.6 times more likely to have pneumonia, gastrointestinal hemor-

**Table 6.** Predicting Outcomes Based on the American Spinal Injury Association Class

Class	Expected average 1-y recovery
A	One root level and muscle with 1/5 or 2/5 strength on admission will recover to 3/5 or greater.
B	37% will regain 3/5 strength in one or both lower limbs. Unlikely to become ambulatory.
C	Up to 76% of patients with "motor useless" strength recover to motor useful or normal.
D	All patients become ambulatory by the time of discharge from rehabilitation.
E	Intact (by definition)

rhage, and pulmonary embolus, respectively. They also found an inverse linear relation between age and the number of individuals able to take care of themselves at discharge.

Among patients with central cord syndrome, Penrod and coworkers<sup>50</sup> found that 97% of patients younger than 50 years and 41% of patients older than 50 years were ambulatory. Waters and coauthors<sup>51</sup> reported on patients with SCI presenting with spondylosis without spinal fracture. They found that patients on average doubled their initial ASIA motor score at 1 year. Central cord syndrome in young patients seems to have a relatively good prognosis.

### MRI as a predictor

In a recent study, Selden and colleagues<sup>52</sup> evaluated early MRI as a prognostic indicator in acute SCI. They found that hematoma in the spinal cord, the extent of the spinal cord hematoma, the extent of spinal cord edema, and spinal cord compression were associated with poorer outcomes. In a similar study, Flanders and coworkers<sup>53</sup> found that hematoma and the length of edema predicted worse outcomes. In the former study, the best predictor remained the initial clinical examination.

## CHRONIC SPINAL CORD INJURY

### Posttraumatic syringomyelia

Syringomyelia represents an accumulation of fluid in the spinal cord lined by reactive gliosis. Hydro-myelia is defined as dilation of the central canal and is lined by ependyma cells. The two conditions are pathologically distinct but less easily distinguishable using clinical criteria. Most clinicians use the terms hydrosyringomyelia, syringomyelia, and hydromyelia interchangeably to describe an abnormal fluid collection in the spinal cord. In this article, we use the most prevalent term in the literature: syringomyelia.

The incidence of posttraumatic syringomyelia depends on the level of injury and its severity. Rossier and associates<sup>54</sup> reported an incidence of 3.2% among 951 SCI patients. They reported a rate of 7.9% and 4.5% for tetraplegic and paraplegic patients, respectively, and a rate of 3.9% and 2.4% for complete and incomplete injuries, respectively. The symptoms can appear as early as 3 weeks<sup>55</sup> after

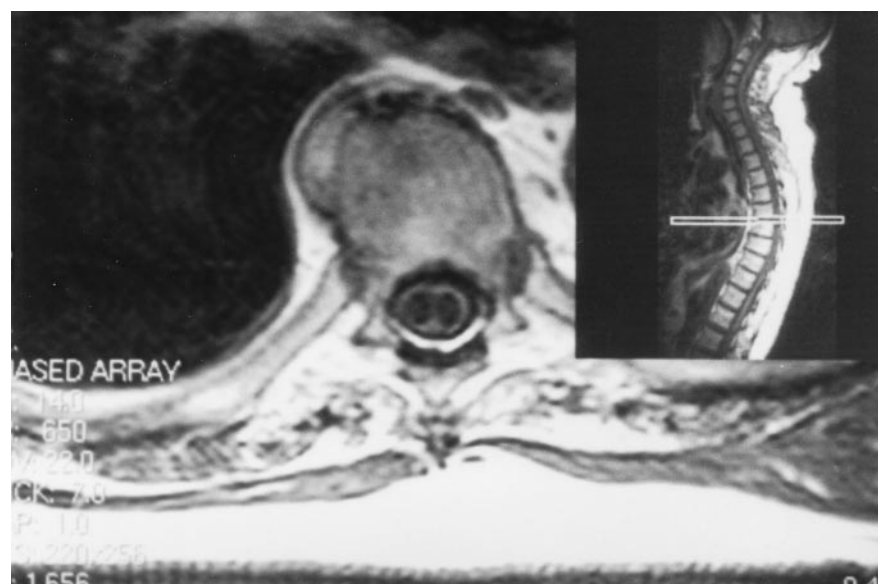
injury but may be delayed by as much as 36 years.<sup>56</sup> The diagnosis is delayed an average of 2 to 3 years after the appearance of the first symptoms.<sup>57</sup> The natural history of untreated syringomyelia is difficult to predict. In the prospective study by Schurch and associates,<sup>57</sup> 3 of 13 patients reported new deficits and worsening of existing symptoms after the diagnosis was established.

One of the most common and disturbing presenting features is pain. Classic symptoms include dissociated sensory loss (loss of pain and temperature with preserved proprioception) in a suspended territory, progressive motor deterioration, and other sensory disturbances. With further progression, Charcot's arthropathy, muscle wasting, and loss of reflexes can supervene. Patients tend to present with a slow, chronic loss of function, dominated by pain.

The pathophysiology of posttraumatic syrinx formation remains debated. One of the modern theories proposes that cyst formation is caused by local abnormalities of cerebrospinal fluid (CSF) flow. The initiating factor can be an increase in the resistance of the CSF flow locally caused by fibrosis, arachnoiditis, dural compression, or spinal deformity. Transudation of CSF from the high-resistance subarachnoid space occurs through perivascular spaces (Virchow-Robin spaces) to a dilated central spinal cord cavity.<sup>59</sup>

When syringomyelia is clinically suspected, the imaging modality of choice is MRI (Fig. 5). The syrinx can be defined in multiple planes. The causative factor, such as arachnoiditis or deformity, can be further delineated. The classic MRI finding is an increased diameter of the spinal cord caused by the fluid collection. The signal parallels the CSF signal. A variable number of septations may be present throughout the syrinx. A CSF flow dynamic study (cine MRI) can be done to assess the fluid flow surrounding the syrinx. The rostral and caudal syrinx's extension varies from a few spinal segments to involvement of the entire spinal cord.

When MRI is contraindicated or when spinal instrumentation would create artifact in the area of interest, a CT myelogram may be performed as an alternative method of investigation. The syrinx appears as a dilatation of the spinal cord, which becomes hyperdense on delayed films because of the



A



B

**Figure 5.** Axial (A) and sagittal (B) T1-weighted images demonstrating a posttraumatic syrinx. The spinal cord is expanded and has a cyst within it containing fluid with signal characteristics similar to those of cerebrospinal fluid.

delayed migration of the intrathecal contrast into the syrinx.

When symptomatic and progressive clinical deterioration occurs, one should strongly consider a

surgical approach with the goal to reverse some of the symptoms and to prevent further deterioration. Most neurosurgeons agree on the surgical indications for treatment, but there is a discrepancy of opinion on the best treatment modality. The options include simple cyst aspiration,<sup>56,59</sup> which is prone to a high recurrence rate; a CSF diversion procedure, such as from the syrinx to the subarachnoid space, pleura, or to the peritoneal cavity; spinal cord untethering and duraplasty; and any combination of these treatments. Spinal cord transection at the level of a syrinx, but below an area of complete neurologic dysfunction (eg, paraplegia), has also been reported.<sup>60</sup>

Failure rates of operations consisting only of shunting from the syrinx to the subarachnoid, pleural, or peritoneal space have been reported to be as high as 50% to 92%.<sup>61</sup> Spinal cord untethering, deformity correction, and correction of residual spinal cord compression address directly the cause of the syrinx—the CSF flow abnormality. A wide laminectomy should be performed, followed by spinal cord untethering and deformity correction supplemented by a syringo-subarachnoid shunt. The dura is closed in a watertight fashion with an expansile cadaveric dural or synthetic graft patch. We try to create an increased space around the cord to reestablish free CSF flow. Correction of spinal deformity is critical in preventing cord retethering and syrinx recurrence.



A



B



C

**Figure 6.** Case illustration of a patient with a posttraumatic kyphotic cervical deformity secondary to a failed posterior fusion. (A) CT scan with sagittal reconstructions of the cervical spine demonstrates a kyphotic deformity associated with a marked reduction of the size of the canal at the apex of the deformity. (B) MRI T2-weighted image demonstrating the posttraumatic deformity, spinal cord compression, spinal cord tethering, and a post-traumatic syrinx. Note also some artifacts produced by the wires used in the failed operation. (C) Postoperative plain x-rays showing correction of the deformity using both anterior and posterior instrumentation. The patient also underwent spinal cord untethering and a syngo-subarachnoid shunt. (Courtesy of Dr J Guest, University of Miami, Miami, FL.)

### Spinal cord tethering

As a result of the primary and secondary injury, the spinal cord and the surrounding arachnoid are subjected to inflammation. Hemorrhage in the vicinity of the injured cord probably aggravates this process. The inflammation may cause some localized arachnoiditis, which could impair CSF flow or create adhesions between the cord and the dura (spinal cord tethering). The pulsatility of the flow on the adherent cord and the loss of the slight normal upward and downward movement of the cord, which occurs in flexion and extension, progressively contribute to the spinal dysfunction seen in these patients.

This subset of SCI patients present with progressive motor dysfunction and marked pain. They are sometimes indistinguishable from patients who have syringomyelia but who lack the dissociated sensory loss and loss of reflexes. On imaging studies, the spinal cord is tethered but cyst formation is lacking.<sup>62</sup> As for syringomyelia, the imaging modality of choice is MRI.

When progressive symptoms occur, surgical intervention may be offered to the patient. The highlights of these procedures are wide laminectomies, lysis of arachnoidal adhesions without creating any new deficits, and closure with an expansive duraplasty. We aim to create a space around the cord so that CSF can flow freely, and the cord is away from the dura to prevent retethering. With this technique, up to 79% of patients improve with respect to motor function and pain postoperatively.<sup>63</sup>

### Progressive spinal deformity

Unfortunately, despite good initial treatment of the primary injury, the occasional patient may suffer from progressive spinal deformity. This can present with progressive loss of neurologic function (both motor and sensory), pain, and visible deformity. In the face of a progressive functional loss, increasing pain, or increasing deformity, the physician should consider an operation to halt the deterioration (Fig. 6).

The goals of the operation are to protect or increase the neurologic function, to decrease or abolish the pain, and to restore spinal alignment. Obviously, the optimal treatment must be individualized according to the specific presentation and x-ray appearance. Often this requires an initial anterior surgical approach for spinal cord decompression

and loosening of the bone and ligament, anterior correction of the deformity with graft, and instrumentation followed by a posterior instrumented fusion. In certain patients, correction of deformity can be accomplished with only a posterior approach.<sup>63</sup> Spinal cord monitoring with either somatosensory evoked potentials or motor evoked potentials is useful in patients with residual spinal cord function.

### Spasticity

Spasticity is a motor disorder characterized by a velocity-dependent increase in muscle tone secondary to upper motor neuron injury. The failure of inhibitory input from descending brain stem centers to reach their target in the spinal cord allows exaggeration of the excitatory afferent input from proprioceptive pathways.<sup>64</sup> Troublesome spasticity is reported to occur in more than 25% of SCI patients.<sup>65</sup> Spasticity impairs rehabilitation, self-care, and sleep, and can also cause pain. The treatment begins with stretching exercise and physical therapy. If this alone is ineffective, oral medication can be tried. The drug of choice is baclofen, which is a  $\gamma$ -aminobutyric analogue.<sup>66</sup> Other useful medications include diazepam, dantrolene sodium, and clonidine. For the symptomatic patient in whom oral agents are ineffective or poorly tolerated, one should consider delivering the drug directly into the thecal sac with an implantable pump. This form of treatment has been effective in decreasing spasms, with improvement in activities of daily living. Botulinum toxin has also been used with success in SCI patients.<sup>67</sup> The drawbacks of this approach are the high cost and short duration of benefit. When all conservative treatments have failed, the rare patient may benefit from an ablative procedure such as neurectomy, spinal cord tractotomy, and other destructive procedures.

### References

1. Tator CH, Carson JD, Edmonds VE. Spinal injuries in ice hockey. *Clin Sports Med* 1998;17:183-194.
2. Watts C, Eyster EF. National Head and Spinal Cord Injury Prevention Program of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons. *J Neurotrauma* 1992;9:S307-312.
3. Levi L, Wolf A, Rigamonti D, et al. Anterior decompression in cervical spine trauma: does the timing of surgery affect the outcome? *Neurosurgery* 1991;29:216-222.
4. Nesathurai S. Steroids and spinal cord injury: revisiting the NASCIS 2 and NASCIS 3 trials. *J Trauma* 1998;45:1088-1093.

5. Bethea JR, Castro M, Keane RW, et al. Traumatic spinal cord injury induces nuclear factor-kappa  $\beta$  activation. *J Neurosci* 1998;18:3251–3260.
6. Emery E, Aldana P, Bunge MB, et al. Apoptosis after traumatic human spinal cord injury. *J Neurosurg* 1998;86:911–920.
7. Knutsdottir S. Spinal cord injuries in Iceland 1973–1989. A follow up study. *Paraplegia* 1993;31:68–72.
8. Martins F, Freitas F, Martins L, et al. Spinal cord injuries—epidemiology in Portugal's central region. *Spinal Cord* 1998;36:574–578.
9. DeVivo MJ. Causes and cost of spinal cord injury in the United States. *Spinal Cord* 1997;35:809–813.
10. Exner G, Meinecke FW. Trends in the treatment of patients with spinal cord lesions seen within a period of 20 years in German centers. *Spinal Cord* 1997;35:415–419.
11. Claxton AR, Wong DT, Chung F, Fehlings MG. Predictors of hospital mortality and mechanical ventilation in patients with cervical spinal cord injury. *Can J Anaesth* 1998;45:144–149.
12. Yeo JD, Walsh J, Rutkowski S, et al. Mortality following spinal cord injury. *Spinal Cord* 1998;36:329–336.
13. Frankel HL, Coll JR, Charlifue SW, et al. Long-term survival in spinal cord injury: a fifty year investigation. *Spinal Cord* 1998;36:266–274.
14. Rish BL, Dilustro JF, Salazar AM, et al. Spinal cord injury: a 25-year morbidity and mortality study. *Mil Med* 1997;162:141–148.
15. Vale FL, Burns J, Jackson AB, Hadley MN. Combined medical and surgical treatment after spinal cord injury: result of a prospective pilot study to assess the merits of aggressive medical resuscitation and blood pressure management. *J Neurosurg* 1997;87:129–146.
16. Orledge JD, Pepe PE. Out-of-hospital spinal immobilization: is it really necessary? *Acad Emerg Med* 1998;5:203–204.
17. Green BA, Eismont FJ, O'Heir JT. Pre-hospital management of spinal cord injuries. *Paraplegia* 1987;25:229–238.
18. DeVivo MJ, Kartus PL, Stover SL, Fine PR. Benefit of early admission to an organized spinal cord injury care system. *Paraplegia* 1998;28:545–555.
19. Gonzalez RP, Fried PO, Bukhalo M, et al. Role of clinical examination in screening for blunt cervical spine injury. *J Am Coll Surg* 1999;189:152–157.
20. Marshall LF, Knowlton S, Garfin SR, et al. Deterioration following spinal cord injury. A multicenter study. *J Neurosurg* 1987;66:400–404.
21. Maynard FM, Bracken MB, Creasey G, et al. International standards for neurological and functional classification of spinal cord injury. *Spinal Cord* 1997;35:266–274.
22. Levi ADO, Tator CH, Bunge RP. Clinical syndromes associated with disproportionate weakness of the upper versus the lower extremities after cervical spinal cord injury. *Neurosurgery* 1996;38:179–183.
23. Roth EJ, Park T, Pang T, et al. Traumatic cervical Brown-Sequard and Brown-Sequard-plus syndromes: the spectrum of presentations and outcomes. *Paraplegia* 1991;29:582–589.
24. Del Bigio MR, Jonhson GE. Clinical presentation of spinal cord concussion. *Spine* 1989;14:37–40.
25. Torg JS, Corcoran TA, Thibault LE, et al. Cervical cord neuropraxia: classification, pathomechanics, morbidity, and management. *J Neurosurg* 1997;87:843–850.
26. MacDonald RL, Schwartz ML, Mirich D, et al. Diagnosis of cervical spine injury in motor vehicle crash victims: how many x-rays are enough? *J Trauma* 1990;30:392–397.
27. Woodring JH, Lee C. Limitation of cervical radiography in the evaluation of acute cervical trauma. *J Trauma* 1993;34:32–39.
28. Nunez DB, Zuluaga A, Fuentes-Bernardo DA, et al. Cervical spine trauma: how much more do we learn by routinely using helical CT? *Radiographics* 1996;16:1307–1318.
29. D'Alise MD, Benzel EC, Hart BL. Magnetic resonance imaging evaluation of the cervical spine in comatose or obtunded trauma patients. *J Neurosurg (Spine)* 1999;91:54–59.
30. Klein GR, Vaccaro AR, Albert TJ, et al. Efficacy of magnetic resonance imaging in the evaluation of posterior cervical spine fractures. *Spine* 1999;24:771–774.
31. Tator CH, Fehlings MG. Review of the secondary injury theory of acute spinal cord trauma with emphasis on vascular mechanisms. *J Neurosurg* 1991;75:15–26.
32. Bracken MB, Shepard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the Second National Acute Spinal Cord Injury Study. *N Engl J Med* 1990;322:1405–1411.
33. Bracken MB, Shepard MJ, Holdford TR, et al. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. *JAMA* 1997;277:1597–1604.
34. Geisler FH, Dorsey FC, Coleman WP. Past and current clinical studies with GM-1 ganglioside in acute spinal cord injury. *Ann Emerg Med* 1993;22:1041–1047.
35. Geisler FH, Dorsey FC, Coleman WP. Recovery of motor function after spinal-cord injury—a randomized, placebo-controlled trial with GM-1 ganglioside. *N Engl J Med* 1991;324:1829–1838.
36. Fehlings MG, Cooper PR, Errico TJ. Posterior plates in the management of cervical instability: long-term results in 44 patients. *J Neurosurg* 1994;81:341–349.
37. Razack N, Green BA, Levi ADO. The treatment of bilateral facet fracture dislocations using unicortical anterior plates. *J Spinal Disord (In press)*.
38. Guha A, Tator CH, Endrenyi L, Piper I. Decompression of the spinal cord improves recovery after acute experimental spinal cord compression injury. *Paraplegia* 1987;25:324–339.
39. Cotler JM, Herbison GJ, Nasuti JF, et al. Closed reduction of traumatic cervical spine dislocation using traction weights up to 140 pounds. *Spine* 1993;18:386–390.
40. Fehlings MG, Tator CH. An evidence-based review of decompressive surgery in acute spinal cord injury: rationale, indication, and timing based on experimental and clinical studies. *J Neurosurg (Spine)* 1999;91:1–11.
41. Duh MS, Shepard MJ, Wilberger JE, et al. The effectiveness of surgery on the treatment of acute spinal cord injury and its relation to pharmacological treatment. *Neurosurgery* 1994;35:240–249.
42. Vaccaro AR, Daugherty RJ, Sheehan TP, et al. Neurologic outcome of early versus late surgery for cervical spinal cord injury. *Spine* 1997;22:2609–2613.
43. Delamarter RB, Sherman J, Carr JB. Pathophysiology of spinal cord injury. Recovery after immediate and delayed decompression. *J Bone Joint Surg Am* 1995;77:1042–1049.
44. Herbison GJ, Zerby SA, Cohen ME, et al. Motor power differences within the first two weeks post-SCI in cervical spinal cord-injured quadriplegic subjects. *J Neurotrauma* 1992;9:373–380.
45. Maynard FM, Reynolds GG, Fountain S, et al. Neurological prognosis after traumatic quadriplegia. Three-year experience of California Regional Spinal Cord Injury Care System. *J Neurosurg* 1979;50:611–616.
46. Kirshblum SC, O'Connor KC. Predicting neurologic recovery in traumatic cervical spinal cord injury. *Arch Phys Med Rehabil* 1998;79:1456–1466.
47. Crozier KS, Graziani V, Ditunno JF Jr, Herbison GJ. Spinal cord injury: prognosis for ambulation based on sensory examination in patients who are initially motor complete. *Arch Phys Med Rehabil* 1991;72:119–121.
48. Burns SP, Golding DG, Rolfe WA, et al. Recovery of ambulation in motor-incomplete tetraplegia. *Arch Phys Med Rehabil* 1997;78:1169–1172.

49. DeVivo MJ, Kartus PL, Rutt RD, et al. The influence of age at time of spinal cord injury on rehabilitation outcome. *Arch Neurol* 1990;47:687-691.
50. Penrod LE, Hegde SK, Ditunno JF Jr. Age effect on prognosis for functional recovery in acute, traumatic central cord syndrome. *Arch Phys Med Rehabil* 1990;71:963-968.
51. Waters RL, Adkins RH, Sie IH, Yakura J. Motor recovery following spinal cord injury associated with cervical spondylosis: a collaborative study. *Spinal Cord* 1996;34:711-715.
52. Selden NR, Quint DJ, Patel N, et al. Emergency magnetic resonance imaging of cervical spinal cord injuries: clinical correlation and prognosis. *Neurosurgery* 1999;44:785-792.
53. Flanders AE, Spettell CM, Friedman DP, et al. The relationship between the functional abilities of patients with cervical spinal cord injury and the severity of damage revealed by MR imaging. *Am J Neuroradiol* 1999;20:926-934.
54. Rossier AB, Foo D, Shillito J, Dyro FM. Posttraumatic cervical syringomyelia. Incidence, clinical presentation, electrophysiological studies, syrinx protein and results of conservative and operative treatment. *Brain* 1985;108:439-461.
55. Milhorat TH, Johnson WD, Miller JJ, et al. Surgical treatment of syringomyelia based on magnetic resonance imaging criteria. *Neurosurgery* 1992;31:231-244.
56. Williams B. Post-traumatic syringomyelia, an update. *Paraplegia* 1990;28:296-313.
57. Schurch B, Wichmann W, Rossier AB. Post-traumatic syringomyelia (cystic myelopathy): a prospective study of 449 patients with spinal cord injury. *J Neurol Neurosurg Psychiatry* 1996;60:61-67.
58. Cho KH, Iwasaki Y, Imamura H, et al. Experimental model of posttraumatic syringomyelia: the role of adhesive arachnoiditis in syrinx formation. *J Neurosurg* 1994;80:133-139.
59. Levy R, Rosenblatt S, Russel E. Percutaneous drainage and serial magnetic resonance imaging in the diagnosis of symptomatic posttraumatic syringomyelia: case report and review of the literature. *Neurosurgery* 1991;29:429-433.
60. Durward QJ, Rice GP, Ball MJ, et al. Selective spinal cordectomy: clinicopathological correlation. *J Neurosurg* 1982;56:359-367.
61. Batzdorf U, Klekamp J, Johnson P. A critical appraisal of syrinx cavity shunting procedures. *J Neurosurg* 1998;89:382-388.
62. Lee TT, Arias JM, Andrus HL, et al. Progressive posttraumatic myelomalacic myelopathy: treatment with untethering and expansive duraplasty. *J Neurosurg* 1997;86:624-628.
63. Wu SS, Hwa SY, Lin LC, et al. Management of rigid post traumatic kyphosis. *Spine* 1996;21:2260-2267.
64. Ordia JJ, Fischer E, Adamski E, Spatz EL. Chronic intrathecal delivery of baclofen by a programmable pump for the treatment of severe spasticity. *J Neurosurg* 1996;65:452-457.
65. Johnson RL, Gerhart KA, McCray J, et al. Secondary conditions following spinal cord injury in a population-based sample. *Spinal Cord* 1998;36:45-50.
66. Middleton JW, Siddal PJ, Walker S, et al. Intrathecal clonidine and baclofen in the management of spasticity and neuropathic pain following spinal cord injury: a case study. *Arch Phys Med Rehabil* 1996;77:824-836.
67. Al-Khodairy AT, Gobelet C, Rossier AB. Has botulinum toxin type A a place in the treatment of spasticity in spinal cord injury patients? *Spinal Cord* 1998;36:854-858.