Acute Management of Spinal Cord Injury

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Abstract

Demographic trends in the occurrence of injury and improvements in the early management of spinal trauma are changing the long-term profile of patients with spinal cord injuries. More patients are surviving the initial injury, and proportionately fewer patients are sustaining complete injuries. While preventive efforts to reduce the overall incidence of spinal cord injury are important, a number of steps can be taken to minimize secondary injury once the initial trauma has occurred. Recent efforts have focused on understanding the biochemical basis of secondary injury and developing pharmacologic agents to intervene in the progression of neurologic deterioration. The Third National Acute Spinal Cord Injury Study investigators concluded that methylprednisolone improves neurologic recovery after acute spinal cord injury and recommended that patients who receive methylprednisolone within 3 hours of injury should be maintained on the treatment regimen for 24 hours. When methylprednisolone therapy is initiated 3 to 8 hours after injury, it should continue for 48 hours. In addition to the adoption of the guidelines of that study, rapid reduction and stabilization of injuries causing spinal cord compression are critical steps in optimizing patients' long-term neurologic and functional outcomes.

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Each year in the United States, between 7,600 and 10,000 individuals sustain and survive a spinal cord injury. A complex interplay of regulatory developments and social issues has influenced trends in spinal injury. Improvements in emergency medical services systems, the development of safer automobiles, more occupational safety standards, and better regulation of contact sports have had a positive impact on demographic trends. However, while the overall incidence of traumatic spinal cord injury is decreasing nationally, the percentage due to acts of domestic violence is sharply on the rise. In general, more patients are surviving the initial traumatic injury, and trends over time indicate an increase in the proportion of persons with incomplete paraplegia and a decrease in the proportion of persons with complete tetraplegia.[1]

A number of postinjury trends have developed: Advances in the rehabilitation of patients with spinal cord injuries have resulted in shorter hospital stays. Between 1974 and 1994, average acute and rehabilitation hospital stays following injury declined from 122 days to 53 days for paraplegic patients and from 150 days to 75 days for quadriplegic patients.[1] According to a 1996 study,[1] 92% of patients with spinal cord injury are discharged to independent living or residential living situations with assistance. The average life expectancy for an individual with a spinal cord injury remains below normal, but continues to increase.

These positive trends notwithstanding, the overall impact of spinal cord injury on society and on the individual patients and their families is staggering. It has been estimated that there are between 183,000 and 203,000 persons living with spinal cord injuries in the United States. Estimates of lifetime costs for health care and living expenses vary depending on severity of injury and age at the time of injury. For example, lifetime costs for a 25-year-old individual with high quadriplegia are estimated to be $1,350,000, whereas costs for a 50-year-old paraplegic patient are estimated to be $326,000.[1]

Moreover, each person who sustains a spinal cord injury undergoes a devastating transformation in quality of life, with a loss of independence and a profound impact on lifestyle, personal goals, economic security, and interpersonal relationships. For example, in a study from the National Spinal Cord Injury Statistical Center,[1] only

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about a third of persons with paraplegia and about a fourth of those with quadriplegia were employed at postinjury year 8. The likelihood of a marriage remaining intact or of getting married is far lower than in the noninjured population.

Most recent successes have been the result of efforts to decrease the incidence of primary spinal cord injury and advances in the rehabilitation phase of care. This article focuses on measures to reduce the potential for secondary mechanical injury and to address the physiologic process that ensues once the primary spinal cord injury has occurred.

Pathophysiology of Spinal Cord Injury

Mechanism of Injury

The initial traumatic injury typically involves impact, compression and contusion of the spinal cord, and resultant immediate damage to nerve cells, axonal tracts, and blood vessels. Complete severance of the spinal cord following cervical trauma (Fig. 1) is rare; however, as a result of the primary mechanical insult, the secondary physiologic processes, including hemorrhage, edema, and ischemia, rapidly extend to contiguous areas in the cord. Residual pressure on the cord from bone, ligaments, and disk material can also exacerbate the mechanical damage to the cord after the primary injury.

The secondary injury process is a complex cascade of biochemical events, the exact mechanism and sequence of which are only partially understood. After the initial impact, hemorrhage and inflammation occur in the central gray matter of the cord. On a systemic level, autonomic nervous system dysfunction, hypotension, and bradycardia contribute to impaired spinal cord perfusion, which further compounds the ischemia. Experimental studies in animal models of spinal cord injury have shown increases in tissue water content and sodium and lactate levels, along with decreases in extracellular calcium levels, tissue oxygenation, and pyruvate and adenosine triphosphate concentrations.[2] Taken together, these observations are consistent with an overall scenario of ischemia, hypoxia, uncoupling of oxidative phosphorylation, and aerobic glycolysis.

A number of theories have been proposed to explain the pathophysiology of secondary injury. Each theory provides a piece of this complex puzzle, and there is evidence of close synergism between the various mechanisms of secondary injury. The free-radical theory suggests that due to rapid depletion of antioxidants, oxygen free radicals accumulate in injured central nervous system tissue and attack membrane lipids, proteins, and nucleic acids. As a result, lipid peroxides are produced, causing the cell membrane to fail.

The calcium theory implicates the influx of extracellular calcium ions into nerve cells in the propagation of secondary injury. Calcium ions activate phospholipases, proteases, and phosphatases, resulting in both interruption of mitochondrial activity and disruption of the cell membrane.

The opiate receptor theory is based on evidence that endogenous opioids may be involved in the propagation of secondary spinal cord injury. There is evidence that opiate antagonists, such as naloxone, may improve neurologic recovery in experimental models of spinal cord injury. However, different studies have reported conflicting results, and it may be that the beneficial effect of opiate antagonists is dose-responsive.

The inflammatory theory is based on the hypothesis that inflammatory substances (e.g., prostaglandins, leukotrienes, platelet-activating factor, and serotonin) accumulate in acutely injured spinal cord tissue and are mediators of secondary tissue damage.[3] Anti-inflammatory agents have been tested extensively in spinal cord injury.

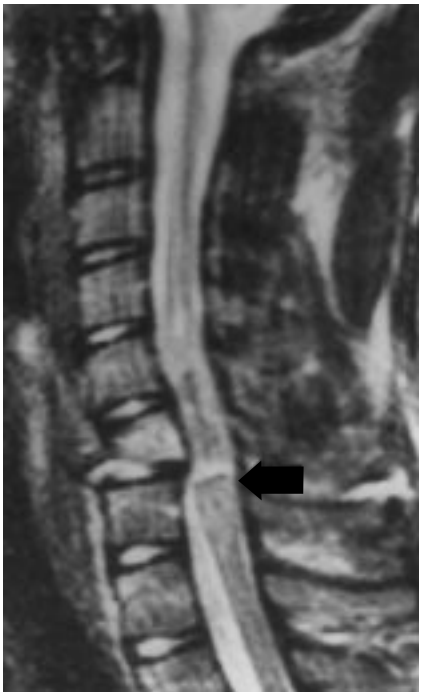

Fig. 1 Complete severance of the spinal cord after a severe C6 fracture-subluxation. The 18-year-old male patient sustained a diving injury and immediate C6 quadriplegia. This magnetic resonance image obtained 90 minutes after the injury depicts complete severance of the cord at the base of the C6 vertebra and hemorrhage into the cord cephalad to the C6 level (arrow).

Histologic manifestations of acute spinal cord injury include necrosis of central cord gray matter in the first hours after injury, followed by cystic degeneration. Over the ensuing several weeks, the development of scar tissue extends into the axonal long tracts, with disruption of axonal continuity.

Effect of Timing of Decompression

In a 1995 in vivo animal study, Delamarter et al[4] evaluated the

effect of timing of decompression of the spinal cord after acute experimental spinal cord compression injury (Fig. 2). In their canine model, 50% spinal cord compression was surgically obtained with a constriction band. Decompression was then performed immediately in 6 dogs and at 1 hour, 6 hours, 24 hours, and 1 week, respectively, in the other four groups of 6 dogs each. Data from somatosensory evoked potential monitoring, daily neurologic examinations, and histologic and electron-microscopic studies performed at autopsy were available for all animals. Initially, all 30 dogs were paraplegic. The dogs that underwent immediate decompression or decompression after 1 hour recovered the ability to walk as well as control of the bowels and bladder. When compression lasted 6 hours or more, there was no neurologic recovery, and progressive necrosis of the spinal cord was noted on histologic examination (Fig. 3). This research suggests that not all damage to the spinal cord occurs at the time of initial trauma and that the extent and persistence of damage depend in part on the duration of compression.

Pharmacologic Intervention

The development of pharmacologic agents to halt progression of secondary neurologic damage after a primary injury has been based on a growing understanding of the sequence of biochemical events. There are ongoing research efforts at the basic and preclinical levels, as well as several major clinical studies. A number of agents, including corticosteroids, 21-aminosteroids, free-radical scavengers, opiate antagonists, calcium-channel blockers, and neurotrophic factors, are being investigated. Table 1 lists a number of these agents by class. Methylprednisolone, tirilazad, and GM_1 ganglioside are each currently being evaluated in ongoing clinical trials.

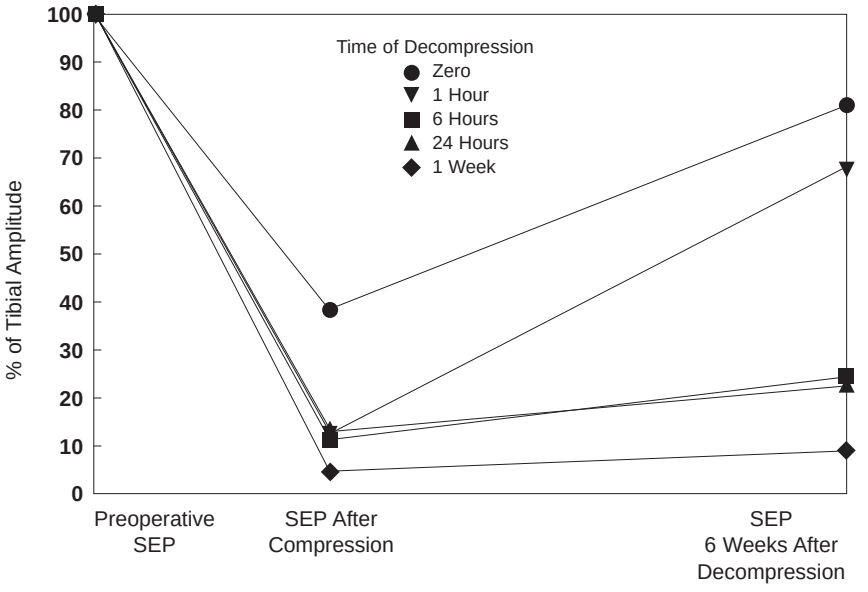

Fig. 2 Somatosensory evoked potential (SEP) recovery after decompression of experimental spinal cord injury in 30 dogs. Note the mean deterioration of the amplitude of posterior tibial SEPs, compared with preoperative values, after compression of the spinal cord and the subsequent recovery in amplitude 6 weeks after decompression. Six weeks after decompression, only the dogs in group 1 (immediate decompression) and group 2 (decompression at 1 hour) showed significant improvement ($P<0.05$) in amplitude. (Reproduced with permission from Delamarter RB, Sherman J, Carr JB: Pathophysiology of spinal cord injury: Recovery after immediate and delayed compression. *J Bone Joint Surg Am* 1995;77:1042-1049.)

Methylprednisolone

The initial rationale for use of glucocorticoids in the treatment of acute spinal cord injury was based on their efficacy in treatment of cerebral edema in patients with closed head injury and brain tumors. Subsequently, additional mechanisms have been proposed for the beneficial effects of methylprednisolone, including reduction of excitatory amino acid neurotoxicity, inhibition of lipid peroxidation, increases in spinal-tissue blood perfusion, and slowing of traumatic ion shifts.[5]

The Second National Acute Spinal Cord Injury Study (NASCIS-II), which was a prospective, randomized, placebo-controlled, double-blinded clinical trial, demonstrated that intravenous administration of high-dose methylprednisolone improved clinical outcomes.[6] Completed in January 1990, NASCIS-II was the first clinical trial to demonstrate statistically significant neurologic recovery from, or reversal of, neurologic injury. The NASCIS-II investigators evaluated the efficacy and safety of methylprednisolone and naloxone in a placebo-controlled multicenter study of 487 patients with acute spinal cord injury. Ninety-five percent of the patients were treated within 14 hours of injury. Methylprednisolone was given to 162 patients in a bolus dose of 30 mg per kilogram of body weight, followed by an infusion at the rate of 5.4 mg/kg per hour for 23 hours. Naloxone was given to 154 patients as a 5.4-mg/kg bolus injection, followed by an infusion at the

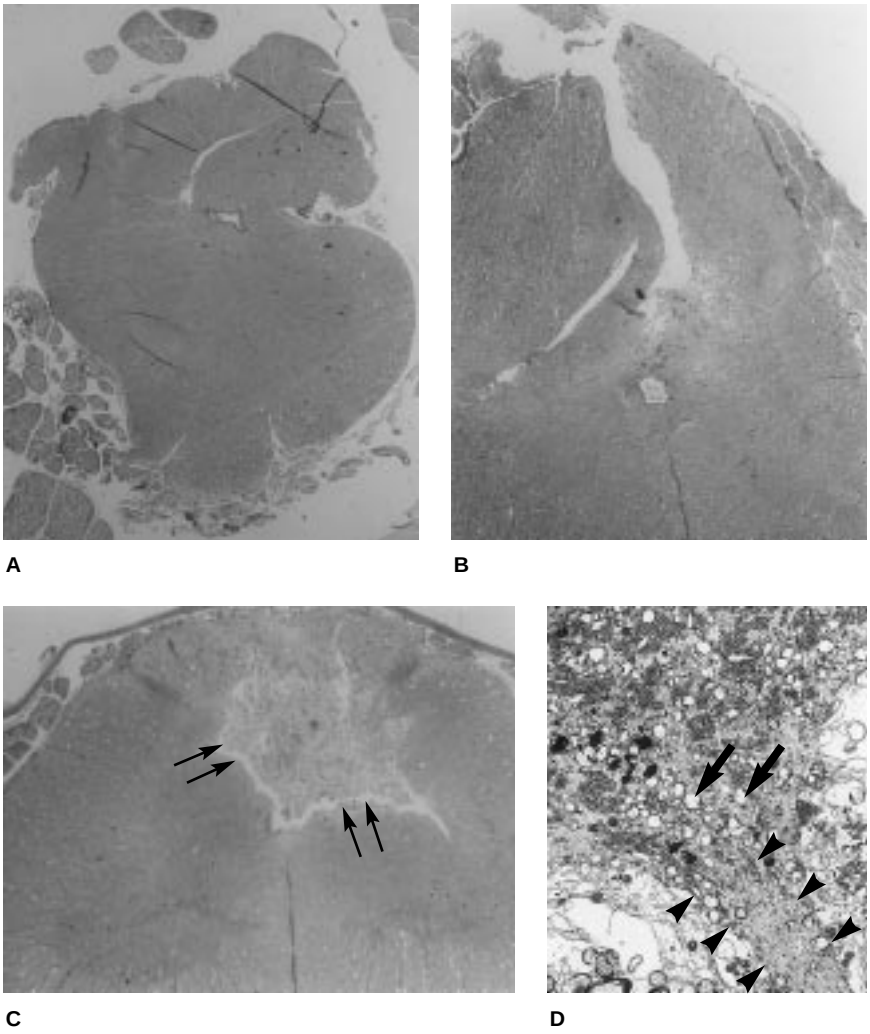

Fig. 3 Histologic findings in an experimental model of spinal cord injury in dogs. **A,** Section of spinal cord approximately 1 cm cephalad to spinal cord injury after immediate decompression. Note the mild deformity of the spinal cord but only minimal histologic damage (hematoxylin-eosin staining). **B,** Higher-power view of a similar section from a dog after 1 hour of constriction. Note the mild to moderate cord deformity, the early degeneration in the central cord, and mild peripheral destructive changes. **C,** Spinal cord section from a dog with decompression after 6 hours of compression (hematoxylin-eosin, original magnification ×6. Note the severe degeneration in the central cord (arrows) and the posterior columns. Spinal cord damage was significantly related to the duration of compression. **D,** Electron-microscopic view showing neural tissue and exiting dendrite. Section was taken 5 mm caudad to the level of compression from a dog after 6 hours of compression. Note the severe degenerative changes in the mitochondria (arrows) and disorganization on both sides of the exiting dendrite (arrowheads) (original magnification ×6,000). (Parts C and D reproduced with permission from Delamarter RB, Sherman J, Carr JB: Pathophysiology of spinal cord injury: Recovery after immediate and delayed compression. *J Bone Joint Surg Am* 1995;77:1042-1049.)

rate of 4.0 mg/kg per hour for 23 hours. Placebo was given to 171 patients.

The NASCIS-II data demonstrated that patients who received a high-dose methylprednisolone infusion within 8 hours of spinal cord injury had better recovery of neurologic function at 6 weeks, 6 months, and 1 year after injury, compared with patients treated with placebo or naloxone.[6] Although the degree of neurologic recovery was strongly related to the completeness of injury, patients with complete injuries as well as those with incomplete injuries improved more after treatment with methylprednisolone than after placebo administration.

There were no statistically significant differences in mortality and morbidity in the methylprednisolone group in comparison to the placebo group. However, patients with incomplete spinal cord injuries treated with methylprednisolone beyond 8 hours postinjury had significantly less neurologic recovery than similar patients treated with placebo, indicating that there may be a detrimental effect to late administration of methylprednisolone. Treatment with naloxone in the doses used in NASCIS-II did not significantly improve neurologic recovery in comparison to placebo.[6]

The NASCIS-II study has been criticized for deficiencies in experimental design and incomplete data. Detailed medical and surgical protocols, as well as radiologic descriptions of the injuries, were not reported. Description of the initial severity of neurologic injuries within each of the treatment groups was not provided in detail. The scheme for grading neurologic improvement in NASCIS-II did not employ functional measures of outcome; therefore, it was not possible to assess clinically useful degrees of recovery.[7,8]

The Third National Acute Spinal Cord Injury Study (NASCIS-III) was a multicenter, randomized, double-blinded prospective study reported in May 1997.[9] Because NASCIS-II showed greater neurologic recovery with methylprednisolone, the investigators felt an obligation to include methylprednisolone in the treatment of all

Table 1
Pharmacologic Agents Under Investigation for Use in Treatment of Acute Spinal Cord Injury

Agent	Class
Naloxone	μ-Opiate receptor antagonist
Methylprednisolone	Corticosteroid
Nimodipine	Calcium-channel blocker
4-Aminopyridine	Potassium-channel blocker
GM_1 Ganglioside	Glycolipid (neurotrophic factor)
Tirilazad (lazeroid)	Lipid peroxidase inhibitor
Vitamin E	Free-radical scavenger

patients in NASCIS-III and all subsequent clinical trials. Therefore, the three groups of patients in NASCIS-III all received an initial 30-mg/kg bolus dose of methylprednisolone before randomization.

The first group of NASCIS-III patients (n = 166) received an infusion of methylprednisolone at a rate of 5.4 mg/kg per hour for 23 hours after the bolus dose. The second group (n = 166) received the methylprednisolone infusion for a total of 48 hours after the bolus dose. The third group (n = 167) received a bolus dose of methylprednisolone, followed by a 2.5-mg/kg bolus of tirilazad every 6 hours for 48 hours.

Neurologic function was assessed at the time of initial presentation and at 6 weeks and 6 months after spinal cord injury. At the time of the 6-month follow-up, 94.7% of surviving patients were available for evaluation. Examinations were conducted by NASCIS-trained physicians and nurses and included quantitative scoring of motor and sensory function, as well as functional independence measures.

In patients who were treated less than 3 hours after injury, essentially identical rates of motor recovery were observed in all three treatment groups. In patients in whom treatment was initiated between 3 and 8 hours after injury, the 48-hour methylprednisolone group recovered significantly more motor function than the 24-hour methylprednisolone group. The 48-hour tirilazad group recovered at a rate slightly faster than the 24-hour methylprednisolone group, but the difference was not statistically significant. Patterns of recovery of sensory function paralleled those for recovery of motor function. However, differences in sensory function improvement between the groups were smaller. Greater improvement in functional independence measures at 6 months was observed in the 48-hour methylprednisolone group than in the 24-hour group. The 48-hour tirilazad group improved at rates between those for the two methylprednisolone groups.

Small differences in complication rates were noted between the groups, with higher rates of severe sepsis and severe pneumonia in the 48-hour methylprednisolone group. These complications did not affect overall mortality. Although the NASCIS-II investigators did not report a statistically significant difference in mortality and morbidity between treatment and control groups, the first NASCIS study demonstrated that 10 days of glucocorticoid treatment was associated with an increased risk of complications.[7] Other authors have associated the use of high-dose glucocorticoids in the treatment of acute spinal cord injury with increased risk of pneumonia and wound infections and prolongation of hospital stay.[10]

On the basis of the results of the NASCIS-III trial, the investigators recommended that patients with acute spinal cord injury who receive methylprednisolone within 3 hours of injury should be maintained on the treatment regimen for 24 hours. They further recommended that when methylprednisolone therapy is initiated 3 to 8 hours after injury, it should be continued for 48 hours.[9]

Tirilazad

Tirilazad is a lazeroid (synthetic 21-aminosteroid). Lazeroids are extremely potent antioxidants and exhibit neuroprotective effects by a variety of other mechanisms as well, such as improving spinal cord blood flow and membrane stabilization. Because lazeroids have none of the glucocorticoid properties of methylprednisolone, tirilazad may have fewer side effects.

GM_1 Ganglioside

Gangliosides are complex acidic glycolipids found in high concentrations in central nervous system tissue as a major component of the cell membrane. In animal studies, gangliosides have been shown to stimulate the growth of nerve cells in damaged tissue.[11] Their mechanism of action involves enhancing survival of residual axonal tracts passing through the site of injury, thereby facilitating the recovery of useful motor function distally. Gangliosides also act to limit cell destruction by excitatory amino acids.

In a 1991 randomized, prospective clinical trial, Geisler et al[12] demonstrated statistically significant neurologic improvement in patients given a parenteral GM_1

ganglioside sodium salt, compared with patients given placebo. At follow-up 1 year after injury, significant improvement was noted on the basis of both the American Spinal Injury Association motor score and the Frankel classification grade. Analysis of the data indicated that improved function in patients treated with GM_1 ganglioside occurred in initially paralyzed, rather than paretic, muscles.

Currently, a large multicenter study is in progress to validate the initial clinical results seen with GM_1 ganglioside treatment.[13] The study also seeks to establish the safety and efficacy of two dose regimens of GM_1 ganglioside.

4-Aminopyridine

4-Aminopyridine is a fast potassium-channel blocker, which has been shown in experimental models of spinal cord injury to enhance nerve conduction through demyelinated nerve fibers by prolonging the duration of action potentials. When 4-aminopyridine was given in limited clinical trials to patients with incomplete injuries, it produced temporary neurologic improvements, which persisted for as long as several days after administration of the drug.[14]

Spinal Cord Regeneration

A number of studies to investigate the regeneration of axonal tracts after traumatic spinal cord injury are currently underway. For example, researchers at the University of Zurich administered antibodies to neutralize myelin-associated neurite growth inhibitory factor to young adult rats that had undergone partial transection of the midthoracic spinal cord. The treatment resulted in growth of corticospinal axons around the site of injury and into spinal cord levels caudal to the injury.[15]

Recently, Cheng et al[16] reported on a study in which they completely transected a 5-mm section of spinal cord at the T8 level in adult rats. This was followed by grafting of peripheral nerve implants from individual axonal tracts to areas of neuronal cell bodies to bridge the gap. Acidic fibroblast growth factor, a constituent of normal spinal cord tissue, was mixed with fibrin glue and then used to stabilize the grafts. Rat hind-limb function improved progressively over a 6-month period, compared with controls. Although this study is far removed from clinical application to traumatic spinal cord injury in humans, it represents the first evidence that regeneration can occur in a completely transected spinal cord of an adult animal and suggests that therapies will eventually be discovered for regeneration of the spinal cord after traumatic injury.

Management of Acute Spinal Cord Injury

Evaluation and Medical Management

Although current understanding of the pathophysiology of acute spinal cord injury is limited, the recommended treatment protocol (Table 2) is based on three major objectives. First is prevention of secondary injury by pharmacologic intervention, such as administration of methylprednisolone within 8 hours after injury, in accordance with the guidelines established in NASCIS-III. Patients should be given a 30-mg/kg bolus dose of methylprednisolone, followed by either a 23-hour or a 48-hour infusion at the rate of 5.4 mg/kg per hour.[6]

Second, hypoxia and ischemia at the local site of spinal cord injury should be minimized by controlling hemodynamic status and oxygenation. All patients should receive supplemental oxygen sufficient to achieve an oxygen saturation approaching 100%. This should be initiated as soon as the diagnosis of spinal cord injury is made. Patients with high cervical injuries may require intubation to reach this level.

Neurogenic shock results from the disruption of sympathetic outflow by cord injury. It is clinically manifested by hypotension due to vasodilatation and bradycardia secondary to unopposed vagal influence on the heart. Patients in neurogenic shock typically have a heart rate between 50 and 70 beats per minute and a systolic pressure 30 to 50 mm Hg below normal. Neurogenic shock must be differentiated from hypovolemic shock,

Table 2
Acute Management of Cervical Spinal Cord Injury

1. Maintenance of perfusion systolic blood pressure >90 mm Hg
2. 100% O_2 saturation via nasal cannula
3. Early diagnosis by plain radiography
4. Methylprednisolone therapy (loading dose of 30 mg/kg followed by infusion at rate of 5.4 mg/kg per hour for 23 or 48 hours)
5. Immediate traction reduction for cervical fracture and dislocation
6. Spinal imaging (MR imaging and/or computed tomography)
7. Surgery if indicated for residual cord compression or fracture instability

which presents with a combination of tachycardia and hypotension, generally due to blood loss from abdominal or pelvic injury.[17] Treatment of neurogenic shock includes an initial fluid challenge, Trendelenburg positioning (10 to 20 degrees), vasopressors (e.g., dopamine and phenylephrine hydrochloride) after central line placement, and atropine for treatment of bradyarrhythmias. Systolic blood pressure should be restored to normal as quickly as possible.

Third, once a spinal cord injury is suspected, the spine should be immobilized to prevent further neurologic injury. Currently, most spinal cord injury patients are transported to trauma centers by emergency medical services personnel and arrive immobilized on a trauma board with a collar. Effective management requires the assumption that every polytraumatized or unconscious patient has a spinal cord injury until proven otherwise.

Early recognition and appropriate acute management of spinal cord injuries is critical to improving overall patient outcome. For example, the incidence of complete neurologic injury in patients with traumatic spinal insults admitted to one regional spinal cord injury system in 1972 was 81%; by 1992, this had dropped to 57%.[18] In another study,[19] the proportion of complete spinal cord injuries decreased from 64% to 46% after the establishment of a regional spinal cord injury unit.

Spinal cord injury is frequently accompanied by other injuries, many of which can be life-threatening. For example, of patients with spinal cord injury secondary to motor-vehicle accidents, 40% have associated fractures, 42.5% experience loss of consciousness, and 16.6% have a traumatic pneumothorax or hemothorax.[20] The initiation of evaluation and treatment of acute spinal cord injuries may be delayed by the need to treat more life-threatening injuries. Nevertheless, during the acute resuscitation and evaluation of the polytrauma patient, the spine should be stabilized and protected from further injury at all times.

Accurate radiologic (Fig. 4) and neurologic assessment of the patient with a spinal cord injury should be part of the secondary trauma survey. When feasible, malaligned vertebral fractures or dislocations should be reduced concurrently with ongoing trauma resuscitation measures. Early intervention is essential to limit the secondary spinal cord injury. If the patient survives the life-threatening injuries, the outcome of the spinal injury will be a predominant factor influencing the future quality of life.

Patients presenting with either a neurologic deficit or evidence of cervical spine instability should be placed in cervical traction with tongs or a halo ring. Contraindications to cervical traction include distraction injuries at any level in the cervical spine and type IIA hangman's fractures. The objectives of application of halo or tong traction are spinal stabilization and, when possible, rapid decompression through realignment of the spinal canal.

A lateral cervical spine film showing C1 to T1 should be available before the application of traction and should be repeated after the initial application of 10 to 15 lb. Weight can then be added in 5- and 10-lb increments, followed by serial neurologic evaluations and repeat radiographs until evidence of alignment is seen. Intravenous administration of 1 to 4 mg of midazolam hydrochloride as an adjunct to achieve muscle relaxation and use of fluoroscopy can facilitate a more rapid, controlled reduction of cervical facet dislocations. Contraindications to continued attempts at reduction using traction include worsening neurologic deficits and evidence of distraction by more than 1.0 cm in a disk space. Reduction is typically obtained with 40 to 70 lb of traction, although use of more than 100 lb has been reported.[21]

For initial immobilization, cervical tongs and the halo ring each have advantages. In some centers, cervical tongs are preferred because of the rapidity and ease with which they can be applied by one person in an emergency room. Halo application takes somewhat longer and generally requires two persons, but has the advantage of control of alignment in three planes and can facilitate the reduction of unilateral and bilateral facet dislocations. Availability of traction equipment is important; delays in application of traction are common due to the necessity of obtaining a halo from another location or due to ongoing radiologic or trauma evaluation. Ideally, the halo or tongs should be compatible with magnetic resonance (MR) imaging. However, the application of cervical traction should not be delayed in order to first obtain a diagnostic study, such as MR imaging or computed tomography/myelography.

Slucky and Eismont[19] recommend MR imaging for assessment of the degree of spinal cord compression in patients with complete or incomplete neurologic deficit, as well as in patients whose neurologic status has deteriorated and those in whom disk retropulsion with canal compromise or posterior ligament injury is suspected. The MR images should be obtained after application of traction; reduction of a dislocation in a patient with a severe incomplete or complete neurologic deficit should not be delayed for completion of an MR study.

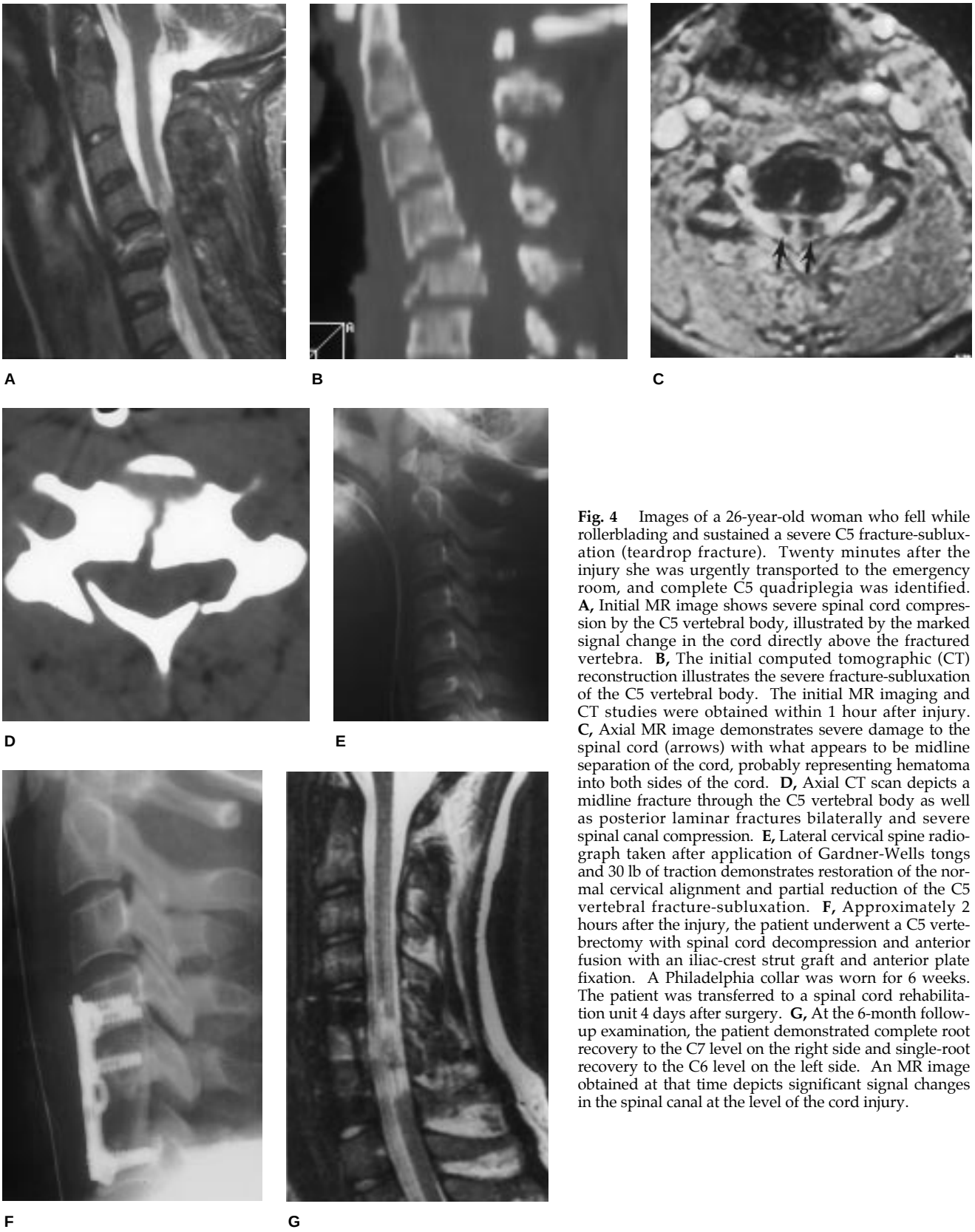

Fig. 4 Images of a 26-year-old woman who fell while rollerblading and sustained a severe C5 fracture-subluxation (teardrop fracture). Twenty minutes after the injury she was urgently transported to the emergency room, and complete C5 quadriplegia was identified. **A,** Initial MR image shows severe spinal cord compression by the C5 vertebral body, illustrated by the marked signal change in the cord directly above the fractured vertebra. **B,** The initial computed tomographic (CT) reconstruction illustrates the severe fracture-subluxation of the C5 vertebral body. The initial MR imaging and CT studies were obtained within 1 hour after injury. **C,** Axial MR image demonstrates severe damage to the spinal cord (arrows) with what appears to be midline separation of the cord, probably representing hematoma into both sides of the cord. **D,** Axial CT scan depicts a midline fracture through the C5 vertebral body as well as posterior laminar fractures bilaterally and severe spinal canal compression. **E,** Lateral cervical spine radiograph taken after application of Gardner-Wells tongs and 30 lb of traction demonstrates restoration of the normal cervical alignment and partial reduction of the C5 vertebral fracture-subluxation. **F,** Approximately 2 hours after the injury, the patient underwent a C5 vertebrectomy with spinal cord decompression and anterior fusion with an iliac-crest strut graft and anterior plate fixation. A Philadelphia collar was worn for 6 weeks. The patient was transferred to a spinal cord rehabilitation unit 4 days after surgery. **G,** At the 6-month follow-up examination, the patient demonstrated complete root recovery to the C7 level on the right side and single-root recovery to the C6 level on the left side. An MR image obtained at that time depicts significant signal changes in the spinal canal at the level of the cord injury.

Serial Examinations

The objectives of the initial neurologic examination conducted during the secondary trauma survey are to establish the level and type of neurologic deficit and to determine whether there is any motor or sensory sparing distal to the level of injury. The initial evaluation is the most valuable from a prognostic standpoint, as it guides treatment decisions and serves as a baseline for subsequent evaluations. Follow-up examinations should be performed at regular intervals and also whenever the patient is transferred or undergoes traction adjustments or surgical procedures. In a multicenter study of deterioration of neurologic status after spinal cord injury, Marshall et al[22] prospectively evaluated 283 patients admitted to five trauma centers. Fourteen of these patients deteriorated neurologically during acute hospital management. In 12 of the patients, deterioration could be specifically associated with a management intervention, such as traction or halo-vest application, surgery, or Stryker frame or rotating bed rotation.

The use of the American Spinal Injury Association scoring diagram for spinal cord injury helps examiners obtain accurate, complete, and reproducible neurologic assessments. If examinations are recorded each time in the same format and with use of the same data points, they can be easily compared with one another.

Timing of Operative Treatment

The timing of surgery remains a controversial issue. There is little debate that emergency surgical decompression is indicated for a progressive neurologic deficit in the presence of persistent spinal cord compression. Operative intervention in other clinical circumstances can be done on an acute or urgent basis or can be delayed. Ducker et al[23] advocated acute operative intervention for patients with cervical spinal cord injury who require open reduction or decompression for persistent spinal cord compression, instability at the occipital cervical junction, or atlantoaxial instability. Other authors recommend treating nonprogressive neurologic deficits on a semiurgent basis, when the patient is medically stable.[24]

In a multicenter study, Marshall et al[22] had three patients with cervical spinal cord injuries whose neurologic condition deteriorated after surgery. Each patient had been operated on within 5 days of injury. No such deterioration was noted when surgery was performed after 5 days. On the basis of these observations in a very small sample of patients, they recommended that early surgical intervention should be performed only to avoid further deterioration in neurologic function.

There have been other reports of marked neurologic recovery in patients who presented initially with complete deficits and canal compromise and were treated with rapid closed reduction and restoration of alignment. In one of the earliest retrospective reviews, Frankel et al[25] evaluated the data on 682 patients who underwent postural reduction at the National Spinal Injuries Centre in England between 1951 and 1968. On detailed analysis of the neurologic results, the authors noted that a small number of patients with complete neurologic lesions initially and a larger number of patients with incomplete lesions improved. No mention was made of a correlation between timing of the reduction and degree of recovery. Furthermore, the authors could not correlate the severity of the neurologic lesion or the degree of reduction achieved with the neurologic recovery.

Hadley et al[26] presented the data on a series of 68 patients with acute traumatic cervical-facet fracture-dislocations. One patient, who presented initially with a unilateral dislocation and a complete deficit, improved neurologically after reduction to the point that he could ambulate with arm braces. Another patient, who presented with a complete neurologic deficit due to a bilateral facet dislocation, underwent closed reduction with cervical traction within 4 hours of injury and was neurologically intact at last follow-up (54 months after injury).

In patients with incomplete neurologic function, the results of very rapid reduction are more promising. In a series of 100 surgically treated cervical spine injuries, Aebi et al[27] noted neurologic improvement after manual or surgical reduction in 31 patients. Of these patients, 75% underwent reduction within 6 hours of the injury. In contrast, 85% of the 69 patients who had no neurologic recovery underwent reduction more than 6 hours after injury.

These clinical observations are consistent with the previously cited experimental conclusions drawn by Delamarter et al[4] regarding the effect of timing of decompression of the spinal cord after acute experimental spinal cord compression injury. The findings in that study suggest that not all damage to the spinal cord occurs at the time of initial trauma and that the extent and persistence of damage depend in part on the duration of compression. It therefore appears that a window of opportunity may exist in many spinal cord injuries. Although the time available for intervention is short, there is a period when complete injury may be partially reversible.

Other authors have considered both the force of the initial injury and the timing of decompression in

the prognosis for recovery.[28] Although the force of the initial injury may be the predominant factor, the timing of decompression or reduction and medical management are the only factors over which the spine surgeon has control.

Summary

Recent advances in understanding of the pathogenesis of spinal cord injury hold promise for future improvement in clinical outcomes. In the meantime, early management in accordance with the NASCIS-III protocol, along with rapid reduction and stabilization, affords the best opportunity for optimization of the long-term outcome in patients with spinal cord injuries.

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