Most often when orthopedic surgeons discuss spinal cord injury (SCI) the emphasis is on vertebral fracture patterns, mechanism of injury, and definitions of stability. The purpose of this chapter is to focus more on the spinal cord itself to better understand its reaction to traumatic injury. By understanding the pathophysiology of SCI, the surgeon is better prepared to manage those factors that affect the course of the resultant neurologic deficit.

An estimated 30 new SCIs occur per million population each year in the United States. Fifty percent of these injuries are complete SCIs and more than two thirds occur in the region of the cervical spine. This translates to an overall prevalence of 906 patients with SCIs per million population. According to the National Spinal Cord Injury database, the majority of these patients are between the ages of 15 and 20. Males are four times as likely to sustain a severe SCI compared to females. Nearly half of all SCIs are sustained as a result of motor vehicle accidents. Falls from a height account for 20.8%, while penetrating injury including gunshot wounds or stabings account for nearly 15%. Sports related injuries account for 14% of all SCI.

With regard to neurologic level, Thomas in 1982 reported that approximately 28% of all patients with SCI are incomplete quadriplegics. Twenty-six percent are complete paraplegics while 24% are complete quadriplegics. These percentages are actually in decline as a result of improved management in the field. One aspect of SCI that is not usually apparent is that a decrease in life expectancy is associated with increased age or increased magnitude of neurologic deficit at the time of injury. For instance, a 20-year-old patient who is a complete quadriplegic may have an estimated 20-year life expectancy, while a patient of the same age who is a complete quadriplegic may only have an estimated 20-year life expectancy. Within the first month of SCI, mortality is most often associated with pulmonary embolus, septicemia, and pneumonia.

Philosophical differences have existed between the orthopedic and neurosurgical management of SCI. The orthopedic literature has focused primarily on defining what constitutes vertebral column stability and how best to surgically restore it. The literature on the management of SCI lacks well-designed, prospective studies, and as such is quite confusing. Most of these studies include small numbers of patients with limited follow-up. In many of these studies, the prospective neurologic status of the patient's is unclear, and relatively few consistent grading systems are employed. The interval between the injury and the intervention also varied from study to study. The role of stabilization seems well accepted in the orthopedic literature as it is believed to prevent further cord damage and provides the optimal environment for recovery. The role of decompression with respect to the timing of surgery, however, remains somewhat fatalistic and unclear, as pathologic changes that occur during the initial insult have previously been considered irreversible.

**HISTORY**

The first natural history study on SCI was reported by Sir Ludwig Guttman in 1963. He reported the 20-year experience of the National Spinal Injury Center at the Stokes Mandiville Hospital in England. One hundred forty-two patients with cervical spine injuries were followed for 20 years during which approximately 50% of the patients had some neurologic improvement with nonoperative treatment. Improvement was defined as any increase in grade of strength.
or return of nerve root function. In 1969, Frankel reported the natural history data from the same institution after following 123 patients with complete SCI's. Over time, 34% of the patients had some degree of functional recovery, while 2% experienced some degree of deterioration. It was unclear whether these patients deteriorated as the result of intervention or as the result of the natural progression of their injury. In a prospective study of 283 patients with SCIs Marshall later defined the incidence of neurologic deterioration. He noted that approximately 5% of patients deteriorated during the acute management phase of their treatment. The majority of these patients deteriorated as a result of a specific event such as surgery, traction, halo application, or striker frame rotation.

In 1971, Bosch and Stauffer reported the natural history of various SCI patterns. They noted that in 60 patients with incomplete spinal cord lesions, 90% of those patients with Brown-Sequard syndrome experienced improvement, 50% of those with a central cord syndrome experienced improvement, and only 16% of those with an anterior cord syndrome experienced improvement. When they looked at patients with complete injury patterns, they found that as many as 80% can expect to recover at least one nerve root level after a complete spinal cord lesion. The only consistently poor results with respect to the recovery of nerve root function was in those patients who had unreduced dislocated facets. Over the next decade, Stauffer went on to further describe several observations regarding the natural history of SCI. Some of these include the following:

1. Complete lesions do not recover motor control.
2. Complete lesions recover one nerve root level of function.
4. Less injury allows greater recovery.
5. Patients with a Brown-Sequard syndrome have a higher rate of recovery compared with patients with a central cord syndrome, who in turn had better prognoses than patients with an anterior cord syndrome.
6. Reduction of dislocated facets facilitates neurologic recovery regardless of the time interval to reduction.

**PATHOPHYSIOLOGY**

It has been well documented that mechanical injury rarely transects the spinal cord even when the SCI is complete. This raises the question as to what changes occur in the structure of the spinal cord that makes the resultant neurologic deficit irreversible. This question was first addressed by Allen in 1911 when he described the phenomenon of posttraumatic autodestruction. Using a weight dropped from a height to impart an SCI in a dog, Allen performed postinjury myelotomies to remove the hematomyelia and found subsequent improvement in neurologic function. From this, he concluded that there existed some biochemical factor in the necrotic hemorrhage that perpetuated the injury over time. This observation led to the development of the concept of primary versus secondary mechanisms of SCI. Primary injury mechanisms traditionally have been considered mechanical phenomenon, and most often include acute compression or impact, distraction, laceration, or shear injuries.

In a prospective study examining the effects of primary injury mechanisms, Delamarter inflicted a compressive injury to the spinal cord in a dog using a band constriction technique. In this model, a 50% constriction was applied to the spinal cord at the L4 level using a nylon band. Initially, this resulted in paraplegia in all dogs studied. The dogs were then divided into five groups each undergoing release of the constriction at various times from immediately through 1 week. Only those dogs who had release of the constriction band within 1 hour had return of motor function demonstrated by the ability to walk, as well as return of bowel or bladder function. All dogs subjected to greater than 6 hours of spinal compression had no recovery of motor function or bowel and bladder function. The spinal cords of the dogs were then subjected to histologic exam.

While those spinal cords subjected to 1 hour or less of constriction experienced variable degrees of Wallerian degeneration and demyelination, those spinal cords subjected to greater than 6 hours had evidence of severe necrosis extending 2 cm cephalad to the level of the constriction. Caudal to the level of the constriction, there was loss of cellular architecture, axonotmesis, and increased fibrosis. In addition to primary mechanisms of injury, several secondary mechanisms of injury have been proposed. These include vascular insults to the spinal cord sustained by hypotension associated with neurogenic shock, hemorrhage, and decreased blood flow as a result of vasospasm and thrombosis. Electrolyte changes include increased intracellular calcium and increased extracellular potassium. Biochemical changes include neurotransmitter accumulation (noradrenaline, dopamine, and glutamate), free radical production, and lipid peroxidation. Secondary spinal cord edema also has been associated with progressive damage to the spinal cord. In 1991, Tator described a posttraumatic ischemic cascade in which hemorrhage and edema resulted in axonal and neuronal necrosis. This was followed by demyelination, aseptic degeneration, and ultimately infarction of the central portion of the spinal cord. This cascade reflects similar pathologic changes previously observed by Allen in 1914 after using his weight-drop technique on the spinal cords of dogs. Allen demonstrated that within 15 minutes after the acute compression,
microhemorrhages developed within the gray matter, while edema occurred in the white matter. By 2 hours, there was increased hemorrhage in the gray matter. At 4 hours post injury, axial degeneration ensued and myelin sheaths were disrupted. Central cord necrosis could be identified within 1 to 2 days after acute injury.

By defining these secondary mechanisms of SCI, specific treatment interventions directed at each component of the ischemic cascade may be proposed. Volume expansion increases blood pressure and can thereby rapidly improve spinal cord perfusion. Opiate antagonists, such as naloxone, improve blood pressure and spinal cord blood flow. Calcium channel blockers, including nimodipine, which exerts selective action on cerebral vasculature, prevents the intracellular influx of calcium responsible for toxic cell death. These calcium channel blockers also dilate blood vessels increasing spinal cord blood flow.

Based on these observations, with respect to the pathophysiology of SCI, certain conclusions can be made. First, the initial contusion is not solely responsible for the pathologic changes observed histologically in SCI. Progressive cellular degeneration seems to interfere with the ability to regenerate. The degree of demyelination is proportional to the duration of compression; if the decompression can occur within 1 hour, the spinal cord would have the best chance for recovery. Decompression beyond 6 hours of compression may preclude the recovery of function. As Delamarter stated, "Although the time available for intervention is short, there is a period when complete injury may be partially reversible." Although ischemia, with or without compression, is related to loss of neurologic function, degree of pathologic change is related to the duration of compression.

CLOSED MANAGEMENT

Early administration of pharmacologic agents such as high-dose methylprednisolone has been shown to alter the course of neurologic recovery. Bracken reported in 1992 that methylprednisolone administered in a loading dose of 30 mg/kg over 15 minutes followed by a maintenance dose of 5.4 mg/kg/hr over the subsequent 23 hours improved neurologic recovery rate compared to naloxone or placebo if the methylprednisolone was administered within 8 hours after injury. When administered beyond 8 hours, either methylprednisolone or naloxone resulted in a worse outcome when compared with placebo administration alone. Methyldprednisolone exerts its effect by inhibiting lipid peroxidation while increasing spinal cord blood flow, extracellular calcium, and overall cell metabolism. Closed reduction of cervical spine dislocations have been considered the earliest method of neurologic decompression. Cotler in 1987 reported results of early high-weight closed reduction of patients with unilateral and bilateral facet dislocations. In his group of 24 patients, 9 of 10 patients with incomplete SCI improved at least one Frankel grade. Those reductions attempted within 24 hours were far more successful. Nearly half of the unreducible dislocations were those that were attempted after an average of 12 days. He recommended that patients with unilateral facet dislocations should undergo early closed reduction. If this procedure were done successfully, elective open reduction with internal fixation should be performed. When a bilateral facet dislocation with a neurologic deficit was identified, urgent reduction was recommended. If the surgeon was only able to convert the bilateral facet dislocation into a unilateral facet dislocation, early posterior open reduction and internal fixation should be carried out. If the patient with the bilateral facet dislocation and neurologic deficit is unreducible, the patient should be brought to the operating room for an immediate posterior open reduction with internal fixation. Findings from the Regional Spinal Cord Injury Center of the Delaware Valley suggested that urgent reduction of flexion distraction injuries (under 8 hours) improved recovery. These studies suggest that high-weight closed reductions were safe and effective in improving neurologic function if the patient was awake and cooperative.

The criticism of acute closed reductions of cervical dislocations stems from a documented incidence of herniated cervical discs as high as 54% in those patients with facet dislocations. The concern is that the spinal canal volume is lessened by the disk herniation and that during the reduction, the cord is brought under direct compression by the disk herniation. In our institution, we have not observed any cases of neurologic deterioration associated with closed reduction when the patient was awake and cooperative. We believe that, if the patient is indeed awake and cooperative and has a documented neurologic deficit, obtaining an MRI scan before an attempt at closed reduction delays the decompressive effects of an early reduction and adds additional risk to the patient incurred during transport. An MRI scan should be obtained before reduction if the patient is unconscious, uncooperative, or if the first attempt at closed reduction is unsuccessful.

SURGICAL INTERVENTION

The controversy regarding early surgical intervention arises from the question of whether or not improvement or deterioration in the postoperative period is the result of surgery, or if it reflects the natural course of the injury. As such, surgical intervention has been controversial and is usually reserved for those patients with incomplete SCI. Early surgical intervention had traditionally been associated with a high rate of
complication. Verbiest in 1962 reported results of nine patients with complete quadriplegia who underwent early anterior decompression to remove anterior bone fragments creating compression on the spinal cord. Although four patients had some improvement in upper extremity strength, five patients died within 3 weeks. Verbiest therefore, abandoned surgery within the acute phase of SCI. Other studies have suggested that early intervention might be detrimental as well. Herbison described a period of initial spontaneous worsening after SCI and advocated delaying surgery until this period was over. This concept was partially based on the work of Tator and Fehlings with their description of the secondary mechanism of injury. Herbison noted that 27% of SCI patients had initial worsening within the first 72 hours when followed with serial neurologic examinations. These changes were noted to peak at approximately 1 week. In 1987, Marshall reviewed the outcome of 134 patients with SCI who underwent surgical intervention at various intervals from the time of injury. Overall, 3% of the 134 patients experienced some degree of neurologic worsening. Interestingly, all patients with neurologic worsening had their operation within 5 days of their injury. Similarly, in a review of 1,031 patients with SCI treated at the Thomas Jefferson University Hospital, 2% experienced deterioration overall. None of the patients operated on after 5 days of injury experienced neurologic deterioration. In 1982, Boihman and Anderson studied a cohort of patients with complete quadriplegia who underwent late surgical decompression with anterior cervical discectomies and fusions. In this group of patients, the average time from injury to surgical intervention was 15 months. At an average follow-up of 5 years, “root return” was noted to be at least one level in 18 patients, and at least two to three grades of improvement in six patients. Two levels of neurologic recovery were noted in seven patients. From this study, the authors concluded that the time to surgery was not the crucial factor contributing to functional recovery.

In summary, those advocates of delayed surgical intervention feel that true “early” intervention may not be practical. Delaying surgery until the patient has reached a neurologic plateau is preferable and allows the surgeon to carry out a well-planned procedure on a medically stable patient. According to Boihman’s work, many patients have experienced some degree of neurologic recovery even after late decompression. Recently, with the advent of improved stabilization techniques and implants, renewed interest in early surgical intervention has surfaced. Advocates for early intervention suggest that the surgeon can maximize the environment for neurologic recovery by providing immediate decompression while simultaneously restoring alignment and stability. Early intervention may also allow earlier mobilization, thereby avoiding the systemic and psychiatric complications associated with prolonged immobilization. This would allow earlier transfer to rehabilitation facilities with an associated decrease in overall medical expenditure.

Wilberger demonstrated that systemic complications in patients with SCIs do indeed decrease if surgery is performed within 24 hours of injury. The incidence of pneumonia has been shown to decrease from 21% to 10%, while the rate of decubitis decreases from 16% to 10%.

If one considers the findings by Bracken regarding the early administration of methylprednisolone with an associated improvement in neurologic recovery rates, then one must suspect that there does exist a therapeutic window during which surgical intervention also may affect the course of the neurologic deficit. This concept of a therapeutic window is consistent with the findings of Delamarter, who, as previously discussed, demonstrated that decompression of constricted spinal cords within 6 hours of injury provided for the ultimate complete return of motor and bowel and bladder function.

In a recent prospective randomized blinded trial looking at the effects of urgent surgical decompression with or without methylprednisolone, Rabinowitz and colleagues demonstrated that early decompression within 6 hours, with or without methylprednisolone, resulted in significantly better neurologic function than with methylprednisolone alone. Whether or not methylprednisolone administration improves outcome when combined with decompression remains unclear.

Other studies have been somewhat more ambivalent. Levi and coworkers compared the outcomes of 103 patients with SCIs treated in under 24 hours compared with those treated surgically after 24 hours. They noted that most patients undergoing surgical intervention within 24 hours experienced no difference in mortality, motor improvement, respiratory care, or duration of hospitalization. From this they concluded that there was no statistical difference in outcome between early and delayed surgery. There were no increased complications associated with early surgery. In conclusion, they advocated early surgery simply because it was easier to care for the stabilized patients and was subsequently easier to transfer them to their rehabilitation facilities.

In summary, advocates for early surgical intervention suggest that fewer systemic complications are experienced by the patient and that earlier transfer to rehabilitation may be possible. With recent improvements in surgical technique, early intervention is not only safer but may be better for possible neurologic improvement. Lastly, studies advocating the use of methylprednisolone within 8 hours of injury suggest that there may indeed exist an early therapeutic window during which time surgical intervention may provide for improved neurologic recovery. Surgery within 6 hours of injury, realistically is impossible in all but a select few patients with minimal multisystem trauma.
who have the injury close enough to an emergency center where diagnosis and surgical team assembly can take place.

**SUMMARY**

The surgical approach to the patient with SCI requires an understanding of primary versus secondary mechanisms of injury. Primary mechanisms of injury include mechanical trauma such as compression and may exert their effects in a time-dependent fashion. Secondary mechanisms of injury may be vascular in origin and may result in an ischemic cascade of injury at the cellular level within the spinal cord. Subsequent biochemical, electrolyte, and inflammatory changes may contribute to the propagation of the SCI. Like the primary mechanisms of injury, these secondary mechanisms exert their effects in a time-dependent fashion as well. In the decision-making process regarding surgical intervention, the surgeon must first ensure that the patient is medically stabilized. He must then thoughtfully evaluate the mechanism and extent of the SCI. Attention should then turn to decompression of the spinal cord, whether through early closed reduction or through early or delayed surgical intervention after which some form of mechanical stability to the spine is restored. Advocates for early, aggressive surgical intervention suggest that fewer systemic complications are sustained by the patient. Further, there may exist an early, albeit short, therapeutic window during which the neurologic deficit may be partially or even completely reversible. Advocates for delayed surgical intervention suggest that true early surgery within the theoretic therapeutic window of 6 to 8 hours may not be realistic. Accordingly, surgery after a neurologic plateau is reached may be safer. Delayed surgery allows the surgeon to carry out a well-planned procedure in a medically stable patient. Several studies show patients with SCIs have variable degrees of recovery even after late decompression.

**REFERENCES**