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Review Article

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Surgical and Neurointensive Management for Acute Spinal Cord Injury: A Narrative Review

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ABSTRACT

Spinal cord injury (SCI) following high-energy trauma often leads to lasting neurologic deficits and severe socioeconomic impact. Effective neurointensive care, particularly in the early stages post-injury, is essential for optimizing outcomes. This review discusses the role of neurointensive care in managing SCI, emphasizing early assessment, stabilization, and intervention strategies based on recent evidence-based practices. SCI results from primary mechanical damage to the spinal cord, triggering secondary injuries involving vascular and cellular dysfunction. Early neurointensive care focuses on stabilizing airway, breathing, and circulation while preventing further spinal damage. Imaging and neurologic assessments, including the ASIA scale, guide the management plan. Early decompressive surgery within 24 hours is widely supported for patients with spinal instability or cord compression. Pharmacologic strategies aim to reduce secondary injury, though standardization remains limited. Prophylaxis for deep vein thrombosis and pulmonary embolism, intensive pulmonary support, and monitoring for pressure sores are critical in early-phase SCI. Early neurointensive care and surgical interventions play a pivotal role in mitigating SCI progression. Optimal care requires a multifaceted approach addressing both neurologic and systemic complications, significantly influencing recovery and long-term quality of life. Further research is needed to standardize pharmacologic treatments and optimize surgical timing.

Keywords: Intensive care; Neurosurgery; Spine; Surgey

INTRODUCTION

Spinal cord injury (SCI) is one of the most severe and life-altering consequences of trauma, carrying profound effects not only for the individual but also for society. The global incidence of SCI exceeds 700 per million annually,[9)] creating a heavy burden on healthcare systems due to long-term care requirements, loss of productivity, and associated costs.[1,23)] SCI is largely irreversible, often resulting in lifelong functional impairments and psychological distress for

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patients and families. Despite advancements in medical and surgical techniques, many SCI patients still experience permanent neurological deficits that contribute to their diminished quality of life. Considering the personal and social impact of SCI on each patient, a tailored approach to individual care and careful selection of treatment modalities are of utmost importance.

Historically, SCI has been recognized as one of the most difficult injuries to treat, with references dating back 4,000 years in Egyptian medical papyri, which called it "the disease that cannot be cured."[18] Although treatment approaches have evolved dramatically over time, SCI remains a complex condition with no definitive cure. Advances in acute care and neurointensive management have, however, improved patient outcomes significantly in recent decades. Early-stage interventions, especially in neurocritical care, play a pivotal role in mitigating the progression of neurological damage and stabilizing patients. This article reviews the recommended approaches in neurocritical care for SCI, particularly during the critical early stages following trauma, with an emphasis on best practices in surgical and medical interventions that aim to optimize neurological outcomes.

PATHOPHYSIOLOGY OF SCI: PRIMARY AND SECONDARY INJURY MECHANISMS

Understanding SCI pathophysiology is crucial for developing targeted interventions. SCI can be divided into two main stages: primary injury, which occurs at the time of trauma, and secondary injury, which unfolds over hours to days after the initial event.[3,9,27]

The primary injury is the result of direct mechanical trauma to the spinal cord, often due to fractures, dislocations, or penetrating injuries. This results in immediate cell death, axonal disruption, and microvascular damage. Following the primary event, a cascade of secondary injury mechanisms is triggered, including ischemia, oxidative stress, excitotoxicity, inflammation, and apoptosis, which amplify tissue damage and neurological deficits.[24,34]

Secondary injury is a dynamic process characterized by three overlapping phases: acute, subacute, and chronic. During the acute phase, microvascular disruptions lead to ischemia, which compromises the spinal cord's blood supply and exacerbates cellular hypoxia. Increased permeability of the blood-spinal cord barrier allows for infiltration of inflammatory cells, including neutrophils and macrophages, which release pro-inflammatory cytokines such as tumor necrosis factor-α and interleukin-1β. This inflammatory response, while intended to protect, contributes to further cellular injury.[24,27,28]

Recent studies highlight the role of excitotoxicity, where excess release of neurotransmitters like glutamate overstimulates neurons, leading to calcium influx and cell death. Additionally, reactive oxygen species generated during secondary injury contribute to oxidative stress, which damages cellular proteins, lipids, and DNA, worsening neuronal loss and functional impairment.[17,19]

Given the pathophysiology of SCI, no treatment currently exists for the immediate effects of the primary injury. However, the pathomechanisms of secondary injury that follow SCI should be targeted for treatment. To maximize therapeutic outcomes, intervention at the early stages of secondary injury, both medically and surgically, is essential. Scientific

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advancements have introduced potential therapeutic targets within this secondary injury cascade, such as the use of pharmacologic agents to inhibit inflammation, reduce excitotoxicity, and limit oxidative stress. These interventions aim to protect neuronal and glial cells from further damage, ultimately preserving functional spinal cord tissue and improving outcomes.

EMERGENCY ROOM (ER) NEUROINTENSIVE CARE FOR SCI PATIENTS

In the ER, SCI patients require immediate and systematic evaluation to prevent secondary injury and stabilize vital functions. The initial approach follows the Advanced Trauma Life Support protocol, focusing on airway, breathing, and circulation (ABC). The ABC assessment is particularly critical in high cervical spine injuries, which can compromise respiratory function due to phrenic nerve involvement, leading to respiratory distress and failure.

For patients with Glasgow Coma Scale scores of 8 or lower, endotracheal intubation is typically required to secure the airway. Spinal cord injuries at the level of C5 or above are at great risk of alteration of respiratory function and are vulnerable to risk of dyspnea, fatigue of breathing muscles or even pulmonary functions.[24,34] Therefore, in cases of suspected high cervical injury, airway management must be conducted with minimal cervical spine movement to prevent further damage to an unstable spinal segment. Rapid sequence intubation with manual in-line stabilization is recommended to reduce the risk of secondary neurological injury during intubation.

Once stabilized, a neurological assessment is performed, beginning with the American Spinal Injury Association (ASIA) Impairment Scale, which classifies SCI severity based on motor and sensory function. This classification assists in predicting recovery potential and determining appropriate treatment options.

Diagnostic imaging is essential in SCI evaluation. Initially, plain radiographs are used to assess alignment and detect bony injuries, but more advanced imaging modalities are necessary to visualize soft tissue and neural structures. It is crucial to evaluate the entire spine, as 10%–15% of multiple trauma patients have non-contiguous spinal injuries. Therefore, the whole spine should be included in the initial evaluation.[29] High-resolution computed tomography scans provide a detailed view of vertebral injuries and aid in identifying fractures, dislocations, and other structural damage. Magnetic resonance imaging (MRI) is invaluable for assessing soft tissue injuries, such as ligamentous damage, disc herniation, and neural compression, as well as for detecting spinal cord edema, hemorrhage, and ischemic injury. Evidence suggests that MRI findings, particularly the presence of cord hemorrhage, correlate with the extent of SCI and the likelihood of functional recovery.

SURGICAL INTERVENTION AND NEUROINTENSIVE MANAGEMENT

Optimizing treatment and care for SCI patients is essential to minimize the risk of neurological function loss. The therapeutic approach to SCI involves a complex combination of multimodal neurocritical care and treatments. Neurocritical care for SCI includes several

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key steps, such as early pre-hospital immobilization, acute phase medical management, administration of neuroprotective agents, and, importantly, surgical decompression and stabilization. Surgical intervention is often required in SCI management to decompress the spinal cord, stabilize the spine, and prevent further neurological deterioration. Early decompression within the first 24 hours post-injury is widely supported by evidence, particularly for patients with incomplete SCI. The Surgical Timing in Acute Spinal Cord Injury Study (STASCIS) found that early decompression improved motor recovery outcomes in patients with incomplete SCI,[9] reinforcing the importance of timely surgical intervention.

Decompression techniques

Surgical techniques vary according to the location and nature of the injury. For cervical SCI, anterior approaches, such as anterior cervical discectomy and fusion, are commonly used to remove disc fragments and decompress the spinal cord. Posterior approaches, including laminectomy or laminoplasty, may be required for multi-level decompression or when significant posterior ligamentous disruption is present.[22] In thoracolumbar injuries, posterior fixation with rods and screws is the preferred method for stabilizing fractures and restoring spinal alignment.

Classification of spinal injuries and surgical indications

Several factors must be carefully considered when evaluating an SCI patient as a candidate for surgery. These include bony fracture morphology, the integrity of the posterior ligamentous complex, the trauma mechanism, and, of course, the patient's neurological status. Before discussing surgical indications, spinal injuries must be properly classified to ensure accurate patient assessment and to determine the appropriate treatment modalities.[33] In this regard, various classification systems have been developed to assist physicians in their decision-making. Recently, the Thoracolumbar Injury Classification and Severity Score (TLICS)[13,31] and the Subaxial Injury Classification System (SLICS)[30] have become widely used for classifying thoracolumbar and cervical spine injuries, respectively (**TABLES 1** & **2**). Unlike previous systems that focused primarily on injury classification, TLICS and SLICS offer additional advantages, such as simplicity, ease of application, and guidance for surgical decision-making.

While there is broad consensus that surgical decompression and stabilization play a significant role in improving outcomes for SCI patients, there remains some debate regarding the specific

TABLE 1. Thoracolumbar Injury Classification and Severity Score system

Contents	Points
Injury morphology	
Compression	1
Burst	2
Translation/rotation	3
Distraction	4
Neurologic status	
Intact	0
Nerve root injury	2
Complete cord injury	2
Incomplete cord injury	3
Posterior ligamentous complex integrity	
Intact	0
Suspected/intermediate injury	2
Definite injury	3

Non-surgical management for total score ≤3, surgeons decision for total score = 4, and surgical intervention for total score ≥5.

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TABLE 2. Subaxial Injury Classification System

Contents	Points
Injury morphology	
No abnormality	0
Compression/burst	2
Distraction	3
Rotation/translation	4
Neurologic status	
Intact	0
Nerve root injury	2
Complete cord injury	2
Incomplete cord injury	3
Continuous cord compression	+1
Discoligamentous complex integrity	
Intact	0
Suspected/intermediate injury	1
Definite injury	2

Non-surgical management for total score ≤3, surgeons decision for total score = 4, and surgical intervention for total score ≥5.

indications for surgery. The key indications for surgical intervention include: 1) Clinically and radiologically confirmed spinal cord compressive lesions, with or without fractures; 2) SCI patients with progressive neurological deterioration; and 3) Significant spinal instability, as demonstrated by imaging studies.[23,33]

Optimal timing for surgical intervention

The optimal timing for surgery to achieve the best clinical outcomes in SCI patients remains a topic of debate. However, recent evidence indicates that early decompression, ideally within 24 hours, results in better outcomes compared to delayed surgery.[14] A meta-analysis by Liu et al.[20] showed that the early decompression group experienced significantly better neurological recovery, earlier discharge, and a lower complication rate than those who underwent surgery later. In 2012, Fehlings et al.[9] published the STASCIS, a large multicenter prospective cohort study that examined the timing of surgery after SCI. This study, which involved 313 patients, compared clinical outcomes between those who received surgery within 24 hours of trauma and those who had surgery later. Six months post-operatively, the early decompression group had 2.8 times higher odds of showing a two or more grade improvement in ASIA impairment scores. Since the STASCIS study, early decompression surgery has been widely accepted as the standard for determining surgical timing, with many subsequent studies confirming these findings.[4,12,16,25]

The advantages of early decompression are not only supported by clinical evidence but also widely endorsed by spinal surgeons. In a 2010 survey by Fehlings et al.,[8] more than 80% of over 900 active spinal surgeons reported they would operate on ASIA B or C patients within 24 hours, highlighting the general consensus among spinal surgeons. As such, medically stable patients without other major risk factors should be considered candidates for early surgery.

Recently, some have suggested that ultra-early decompression, within 6 to 8 hours post-injury, might offer even better outcomes. However, more research is needed to confirm these findings.

On the other hand, the favorable outcomes of early decompression after SCI appear to be limited to patients with ASIA B or C injuries. ASIA D patients with complete neurological

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deficits did not show any significant improvement, even with early surgery.[16] Additionally, patients with central cord syndrome seem to benefit less from early surgical decompression. Studies have shown no significant clinical differences in this group based on the timing of surgery.[25]

HEMODYNAMIC MANAGEMENT FOR SCI

Hemodynamic management in acute SCI is a critical and complex aspect of neurointensive care that requires a sophisticated and multifaceted approach to patient stabilization and treatment. Patients with acute SCI often experience neurogenic shock, a unique physiological state characterized by profound hypotension and bradycardia resulting from the sudden disruption of sympathetic nervous system function, which fundamentally alters the body's normal cardiovascular regulatory mechanisms and necessitates aggressive fluid resuscitation and potentially vasopressor support to maintain adequate perfusion of the injured spinal cord.[6,15,26] Maintaining an optimal mean arterial pressure (MAP) is crucial because it directly influences spinal cord perfusion, with inadequate MAP potentially leading to secondary ischemic injury, reduced neural tissue oxygenation, and lesser chance of improved neurological deficits.[15] The primary therapeutic objective is to preserve MAP between 85–90 mmHg during the initial 5–7 days post-injury, as extensive clinical research has demonstrated that maintaining this specific hemodynamic target can significantly improve neurological outcomes and potentially reduce secondary spinal cord damage caused by compromised blood flow and microvascular insufficiency.[15] It is important to balance the critical need for hemodynamic stability with the potential risks associated with excessive fluid administration, which can contribute to pulmonary edema, increased intracranial pressure, and further neurological compromise, requiring a nuanced and individualized approach to each patient's specific physiological presentation and underlying pathology. Comprehensive monitoring includes frequent and detailed assessment of cardiovascular parameters, careful and precise titration of intravenous fluids, judicious use of vasopressors to maintain optimal tissue perfusion if needed, and continuous neurological evaluation to detect and mitigate potential complications associated with neurogenic shock, ultimately aiming to create the most favorable conditions for potential neurological recovery and minimizing long-term functional impairment.

Medical management and neuroprotective strategies

Beyond surgical intervention, medical management plays a critical role in mitigating secondary injury and supporting neurological recovery. Various pharmacological agents have been studied for their neuroprotective effects in SCI, although few have achieved widespread adoption due to limited efficacy in clinical trials.

Pharmacologic interventions

The use of high-dose corticosteroids, specifically methylprednisolone, in acute SCI remains a highly controversial topic in clinical practice, with a complex history of evolving medical recommendations. Historically, methylprednisolone was used to reduce inflammation and oxidative stress, with the National Acute Spinal Cord Injury Study trials initially supporting its administration as a potential treatment to mitigate secondary neurological damage.[11,21,35] However, more recent meta-analyses have failed to demonstrate a clear clinical benefit, raising substantial concerns about the potential adverse effects of the treatment, including increased infection rates, gastrointestinal bleeding, and other long-term complications.[2]

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These findings have led many current clinical guidelines to recommend against routine steroid administration in acute spinal cord trauma, reflecting a significant shift from earlier clinical practices and highlighting the ongoing debate about the appropriate management of inflammatory responses in acute SCI. As a result, clinicians now approach steroid use with considerable caution, carefully weighing the potential risks against any marginal therapeutic benefits.

Other pharmacological agents are being explored for neuroprotection in SCI, including riluzole, a sodium-channel blocker that may reduce glutamate-induced excitotoxicity, and GM1 ganglioside, which has shown potential in promoting axonal regeneration in preclinical studies.[17] Although promising, these agents require further research to determine their safety and effectiveness in human SCI.

Additional neurointensive care considerations

Comprehensive neurointensive care in SCI extends beyond surgical and pharmacological interventions. Preventing complications such as deep vein thrombosis (DVT), pulmonary embolism (PE), respiratory infections, and pressure ulcers is essential to optimize outcomes and reduce morbidity.

DVT and PE

SCI patients are at high risk of thromboembolic events due to immobility and altered blood flow. Acute SCI patients specifically have significantly higher risk of developing DVT or PE,[7] and if proper preventions treatments are not done the risk gets up to 47%–100% of these patients.[10] Low molecular weight heparin (LMWH) is recommended for prophylaxis, and mechanical compression devices may be used adjunctively. Evidence shows that thromboprophylaxis with LMWH significantly reduces DVT and PE rates, although there is variability in protocols regarding dosage and duration.

Respiratory complications

Respiratory failure is common in patients with high cervical injuries. The phrenic nerve, which innervates the diaphragm, originates from the C3–C5 spinal nerves. Therefore, injuries to the more proximal regions of the spinal cord can impair breathing. While the impact varies greatly from case to case and remains highly unpredictable, many patients with high cervical spine injuries are unable to wean off ventilation support, even after the acute phase of neurointensive care.[32] Mechanical ventilation support, frequent suctioning, and early tracheostomy for prolonged ventilation cases are critical. Prophylactic antibiotics and chest physiotherapy reduce the risk of pneumonia, a leading cause of mortality in SCI. Studies indicate that early weaning from mechanical ventilation, when possible, is beneficial for reducing respiratory complications.

Pressure ulcers

Immobility and sensory loss in SCI patients lead to a high risk of pressure ulcers, which can result in infections and delayed recovery. If proper care has not been taken for the SCI patient, up to 45% are known to develop pressure sores during their neurointensive care.[5] Regular repositioning every 2 hours, use of pressure-relieving mattresses, and attention to nutrition and hydration are recommended. Evidence supports early nutritional support as a preventive strategy against pressure ulcers, as malnutrition exacerbates skin breakdown and impairs wound healing.

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CONCLUSION

Acute SCI is a complex condition that requires a multidisciplinary approach for optimal management. While surgical decompression within 24 hours and early neurointensive care are generally recommended to improve neurological outcomes, the management of SCI is continually evolving. Evidence-based approaches that address both primary and secondary injury processes, combined with vigilant monitoring for complications, are essential for maximizing recovery potential. Ongoing research into neuroprotective agents and regenerative therapies offers hope for future advancements in SCI care, and continued efforts are needed to establish standardized protocols for SCI management.

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