

# Spinal cord injury treatment

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Management of spinal cord injury (SCI) involves several phases: immediate care, surgical intervention, rehabilitation, and long-term management.

## 1. Acute Phase Management

- **Immobilization:** [Cervical collar](#), [spine board](#) to prevent further injury.
- **Hemodynamic Support:** Maintain [mean arterial pressure](#) (MAP) between 85–90 mmHg for 5–7 days.
- **Pharmacologic Treatment:**
  - High-dose [corticosteroids](#) (e.g., [methylprednisolone](#)) are controversial and not universally recommended.
- **Respiratory Support:** Especially in high cervical injuries (C1–C4).
- **DVT Prophylaxis:** Use of anticoagulants to prevent thromboembolism.

## 2. Surgical Management

- **Goals:**
  - Decompression of the spinal cord.

- Stabilization of the spinal column (internal fixation, fusion).

- **Timing:** Early surgery (<24 hours) is associated with better outcomes in some studies.

see [Spinal cord injury surgery](#)

## 3. Rehabilitation

- **Multidisciplinary Approach:**

- Physical therapy: Maintain muscle strength, prevent contractures.
- Occupational therapy: Train for daily living activities.
- Psychological support: Address emotional and mental health.

- **Assistive Devices:** Wheelchairs, orthoses, robotic exoskeletons. - **Neuroplasticity-Based Therapies:** Functional electrical stimulation (FES), locomotor training.

## 4. Long-Term Management

- **Bladder and Bowel Care:** Intermittent catheterization, bowel programs. - **Pressure Ulcer Prevention:** Regular repositioning, skin monitoring. - **Spasticity Management:** Physical therapy, baclofen, botulinum toxin. - **Chronic Pain Management:** Multimodal strategies including medications and therapy. - **Community Reintegration:** Social, vocational, and recreational activities.

## 5. Emerging Therapies

- **Stem Cell Therapy:** Research ongoing in neuroregeneration. - **Neuroprosthetics and Brain-Computer Interfaces.** - **Neuroprotective Agents:** Clinical trials for agents that minimize secondary injury.

## Summary

SCI treatment is complex and evolves over time, requiring coordinated, patient-centered, and evolving care strategies.

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see [Spinal cord injury management](#).

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A possible therapeutic strategy that could avoid further patient deterioration is the supplementation with [Vitamin E](#) or trace elements, such as [Zinc](#), [Selenium](#), and [Copper](#), which individually promote T-

[cell](#) differentiation and proliferative responses. For this reason, the aim of the study was to evaluate whether Vitamin E, Zinc, Selenium, and Copper supplementation preserves the number of T-lymphocytes and improves their proliferative function after traumatic SCI. Sprague-Dawley female rats were subjected to moderate SCI and then randomly allocated into three groups: (1) SCI + supplements; (2) SCI + vehicle (olive oil and phosphate-buffered saline); and (3) sham-operated rats. In all rats, the intervention was initiated 15 min after SCI and then administered daily until the end of the study. Locomotor recovery was assessed at 7 and 15 days after SCI. At 15 days after supplementation, the quantification of the number of T-cells and their proliferation function were examined. Our results showed that the SCI + supplements group presented a significant improvement in motor recovery at 7 and 15 days after SCI. In addition, this group showed a better T-cell number and proliferation rate than that observed in the group with SCI + vehicle. The findings suggest that Vitamin E, Zinc, Selenium, and Copper supplementation could be part of a therapy for patients suffering from acute SCI, helping to preserve T-cell function, avoiding complications, and promoting better motor [recovery](#). All procedures were approved by the Animal Bioethics and Welfare Committee (Approval No. 201870; CSNBTBIBAJ 090812960) <sup>1)</sup>.

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There are currently no effective therapies available to ameliorate loss of function available for [spinal cord injury](#). In addition, proposed treatments which demonstrated functional recovery in animal models of acute spinal cord injury (SCI) have almost invariably failed when applied to chronic injury models. Glial scar formation in chronic injury is a likely contributor to limitation on regeneration.

Substantial heterogeneity in the patient population, their presentation and underlying pathophysiology has sparked debates along the care spectrum from initial assessment to definitive treatment.

In seeking a cure, these patients often undergo treatments that lack scientific and methodological rigor.

Ahuja et al. reviews spinal cord injury (SCI) management followed by a discussion of the salient controversies in the field. Current care practices modeled on the [American Association of Neurological Surgeons/Congress of Neurological Surgeons](#) joint section guidelines are highlighted including key recommendations regarding immobilization, avoidance of hypotension, early International Standards for Neurological Classification of SCI examination and intensive care unit treatment. From a diagnostic perspective, the evolving roles of CT, MRI, and leading-edge microstructural MRI techniques are discussed with descriptions of the relevant clinical literature for each. Controversies in management relevant to clinicians including the timing of surgical decompression, [methylprednisolone](#) administration, blood pressure augmentation, intraoperative electrophysiological monitoring, and the role of surgery in central cord syndrome and pediatric SCI are also covered in detail. Finally, the article concludes with a reflection on clinical trial design tailored to the heterogeneous population of individuals with SCI <sup>2)</sup>.

## Bone-marrow mesenchymal stem cells for spinal cord injury treatment

see [Bone-marrow mesenchymal stem cells for spinal cord injury treatment](#).

## Perfusion

Increased [spinal cord perfusion](#) and [blood pressure](#) goals have been recommended for [spinal cord injury](#) (SCI).

Treatment consists of restoration of CSF flow, typically via arachnoidolysis and syrinx decompression. Research into treatments for spinal cord injuries includes controlled hypothermia and stem cells, though many treatments have not been studied thoroughly and very little new research has been implemented in standard care.

Treatment of spinal cord injuries starts with restraining the spine and controlling inflammation to prevent further damage. The actual treatment can vary widely depending on the location and extent of the injury.

Acute spinal cord injury (SCI) is commonly treated by elevating the [mean arterial pressure](#) (MAP). Other potential interventions include [cerebrospinal fluid drainage](#) (CSFD).

Both MAP elevation alone and CSFD alone led to only short-term improvement of SCBF. The combination of MAP elevation and CSFD significantly and sustainably improved SCBF and spinal cord perfusion pressure. Although laser Doppler flowmetry can provide flow measurements to a tissue depth of only 1.5 mm, these results may represent pattern of blood flow changes in the entire spinal cord after injury <sup>3)</sup>.

Lumbar cerebrospinal fluid drainage after spinal cord injury, as used in the pig study by Martirosyan et al would reduce intrathecal pressure at the injury site only if the spinal cord is not compressed against the surrounding dura. Unfortunately, in most patients with severe spinal cord injury, the spinal cord is compressed against the surrounding dura; therefore, drainage of cerebrospinal fluid from the lumbar region will not reduce intrathecal pressure at the injury site <sup>4)</sup>.

Unfortunately, no data correlate the severity of spinal cord injury, the degree of spinal cord swelling, and persistent CSF flow across an injured segment in the human spinal cord. The physiological observations in animals and humans alike indicate that CSF drainage and induced hypertension warrant further investigation as a potential treatment for acute spinal cord injury <sup>5)</sup>.

## Rehabilitation

In many cases, spinal cord injuries require substantial physical therapy and rehabilitation, especially if the patient's injury interferes with activities of daily life.

## Pharmacological Therapy

Despite a degree of theoretical progress, there is a lack of effective drugs that are able to improve the motor function of patients following [spinal cord injury](#) (SCI) <sup>6) 7) 8) 9)</sup>.

see [Methylprednisolone for Spinal cord injury](#).

[Dexamethasone](#) acetate (DA) produces neuroprotective effects by inhibiting [lipid peroxidation](#) and inflammation by reducing cytokine release and expression. However, its clinical application is limited by its hydrophobicity, low biocompatibility and numerous side effects when using large dosage.

Therefore, improving DA's water solubility, biocompatibility and reducing its side effects are important goals that will improve its clinical utility. The objective of this study is to use a biodegradable polymer as the delivery vehicle for DA to achieve the synergism between inhibiting lipid peroxidation and inflammation effects of the hydrophobic-loaded drugs and the amphipathic delivery vehicle. Wang et al., successfully prepared DA-loaded polymeric micelles (DA/MPEG-PCL micelles) with monodispersed and approximately 25 nm in diameter, and released DA over an extended period in vitro. Additionally, in the hemisection spinal cord injury (SCI) model, DA micelles were more effective in promoting hindlimb functional recover, reducing glial scar and cyst formation in injured site, decreasing neuron lose and promoting axon regeneration. Therefore, data suggest that DA/MPEG-PCL micelles have the potential to be applied clinically in SCI therapy <sup>10)</sup>.

## Internal decompression

Refers to pial opening, allowing spontaneous extrusion and irrigation of fluid necrotic debris relieving pressure and resulting in a space for biomaterial scaffold insertion. After thoracic contusions, rats were randomized to: contusion only, contusion + ID and contusion + ID + PLGA-PLL scaffold implantation, to test for neuroprotection and endogenous repair over 3 months. ID alone reduced inflammatory activity, cavity volume, and increased tissue sparing. Scaffold biodegradation produced delayed ingrowth of inflammatory and other cells resulting in endogenously derived laminin-rich tissue, marked reduction in cavitation and presence of tissue remodeling macrophages. Extensive recruitment of Schwann cells into adjacent spared white matter occurred, greatest in scaffold-implanted animals. Despite tissue preservation with myelin repair, no groups differed significantly in open field locomotion. However, across all rats, spared epicenter tissue and locomotor outcomes were correlated. Scaffold-implanted animals showed no obvious toxicity. To study the clinical feasibility, timing and indications for scaffold implantation, Göttingen minipigs underwent ID and were implanted with scaffolds 4, 6, and 24 h after T10 contusion. High intra-spinal tissue pressures fell to pre-injury levels after ID and scaffold implantation. Extrusion of necrotic debris left sufficient space for a sized scaffold. These results provided the preclinical rationale for a current clinical study of biomaterial scaffold implantation into the human injured spinal cord <sup>11)</sup>.

## Near-infrared light-triggered NO release

Jiang et al. suggested a “pleiotropic messenger” strategy based on near-infrared (NIR)-triggered on-demand NO release at the lesion area for traumatic SCI recovery via the concurrent neuroregeneration and neuroprotection processing. This NO delivery system was constructed as upconversion nanoparticle (UCNP) core coated by zeolitic imidazolate framework-8 (ZIF-8) with NO donor (CysNO). This combined strategy substantial promotes the repair of SCI in vertebrates, ascribable to the pleiotropic effects of NO including the suppression of gliosis and inflammation, the promotion of neuroregeneration, and the protection of neurons from apoptosis, which opens intriguing perspectives not only in nerve repair but also in neurological research and [tissue engineering](#) <sup>12)</sup>.

## Experimental spinal cord injury treatment

### [Experimental spinal cord injury treatment](#)

<sup>1)</sup>  
Garcia E, Hernández-Ayvar F, Rodríguez-Barrera R, Flores-Romero A, Borlongan C, Ibarra A.

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Fu ES, Tummala RP. Neuroprotection in brain and spinal cord trauma. *Curr Opin Anaesthesiol*. 2005;18:181-187. doi: 10.1097/01.aco.0000162838.56344.88.

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Guest JD, Moore SW, Aimetti AA, Kutikov AB, Santamaria AJ, Hofstetter CP, Ropper AE, Theodore N, Ulich TR, Layer RT. Internal decompression of the acutely contused spinal cord: Differential effects of irrigation only versus biodegradable scaffold implantation. *Biomaterials*. 2018 Sep 19;185:284-300. doi: 10.1016/j.biomaterials.2018.09.025. [Epub ahead of print] PubMed PMID: 30265898.

12)

Jiang Y, Fu P, Liu Y, Wang C, Zhao P, Chu X, Jiang X, Yang W, Wu Y, Wang Y, Xu G, Hu J, Bu W. Near-infrared light-triggered NO release for spinal cord injury repair. *Sci Adv*. 2020 Sep 25;6(39):eabc3513. doi: 10.1126/sciadv.abc3513. PMID: 32978153.

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