

For the Primer, visit [doi:10.1038/nrdp.2017.18](https://doi.org/10.1038/nrdp.2017.18)

➔ Spinal cord injury (SCI) can be divided into traumatic and non-traumatic aetiologies. Traumatic SCI is caused by mechanical insults that generate the initial damage to the spinal cord, whereas non-traumatic SCI is caused by a disease process (such as tumours or degenerative disc disease). Regardless of the cause, SCI can lead to permanent and severe neurological deficits.

## PATHOPHYSIOLOGY

Different levels of the spinal cord innervate distinct muscle groups; in general, SCI results in the partial or complete loss of function below the level of injury. For example, injuries to the cervical spinal cord can cause quadriplegia (paralysis of the upper and lower limbs).

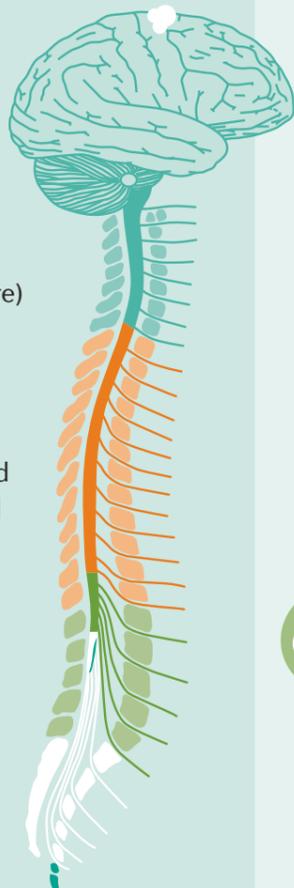
The initial trauma causes displacement or dislocation of the vertebral column, which causes compression or transection of the spinal cord

This causes a focal region of cell death and blood–spinal cord barrier dysfunction, which initiates a cascade of secondary injury mechanisms

Secondary injury mechanisms include vascular changes, inflammation, a loss of ionic homeostasis and oxidative stress, all of which increases the damage to the spinal cord

## DIAGNOSIS

Patients with suspected traumatic SCI undergo gross neurological examination to assess for neurological dysfunction and radiographic imaging to look for damage to the vertebrae and/or spinal cord. If SCI is detected, the grade of the injury is classified according to the American Spinal Injury Association (ASIA) Impairment Scale, which ranges from grade A (most severe) to grade E (least severe) and requires a complete evaluation of sensorimotor function. The clinical manifestations of SCI depend on the level of injury and the amount of spared, uninjured spinal tissue. Injuries generally result in loss of sensorimotor function, but can also affect the sympathetic nervous system, leading to severe hypotension and bradycardia. In addition, injuries that result in the loss of innervation to secondary lymphoid organs can cause secondary immunodeficiency.



POOR REGENERATION

GLIAL SCAR  
CYSTIC CAVITY

Cell death contributes to the formation of cystic cavities, which are surrounded by a glial scar (a deposition of astrocytes and molecules that inhibit neuronal regeneration), which interfere with neuronal regeneration and functional recovery

## OUTLOOK

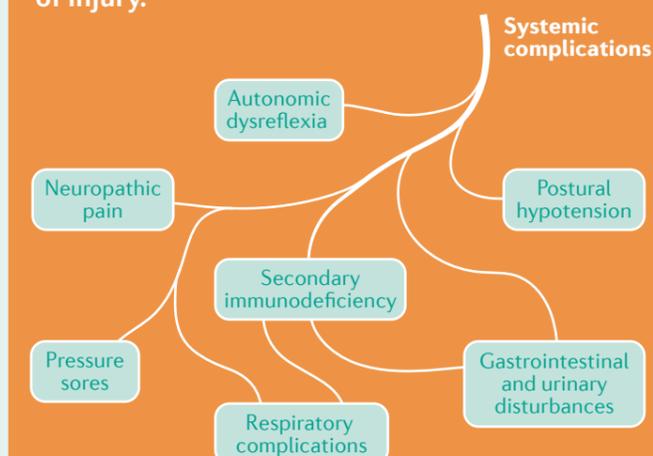
Over the past 30 years, several therapies — neuroregenerative or neuroprotective — have been translated from preclinical studies to clinical trials.

Other potential therapies include cell transplantation, neuromodulation and robotics. Indeed, a robotic exoskeleton (ReWalk) has been approved by

the US FDA for use in patients with paraplegia; this device fits around the patient's legs and back to facilitate sitting, standing and walking.

## Rx MANAGEMENT

Initially, the management of patients with SCI includes monitoring of haemodynamics and decompressive surgery to limit further damage to the spinal cord. In addition, patients can receive methylprednisolone sodium succinate — a neuroprotective agent — although its use is controversial. Long-term management includes monitoring and treating systemic complications of injury.



! Daily care of patients with SCI includes maintaining bowel and bladder function and inspecting the skin for pressure sores

## QUALITY OF LIFE

Several factors are associated with lower quality-of-life scores in SCI, including the presence of spasticity and bowel and bladder dysfunction. The financial consequences of SCI depend on the age of the patient and the severity of injury. In the United States, the lifetime cost of providing care to patients with ASIA Impairment Scale grade A–C injuries ranges from \$2.3 million for thoracic level injuries, to \$4.7 million for upper cervical level injuries.