Incomplete spinal cord injury

Any residual motor or sensory function is more than 3 segments below the level of the injury.

Look for signs of preserved long-tract function.

Signs of incomplete lesion:

1. sensation (including position sense) or voluntary movement in the LEs in the presence of a cervical or thoracic spinal cord injury

2. “sacral sparing”: preserved sensation around the anus, voluntary rectal sphincter contraction, or voluntary toe flexion

3. an injury does not qualify as incomplete with preserved sacral reflexes alone (e.g. bulbocavernosus)

An incomplete spinal cord injury is the term used to describe damage to the spinal cord that is not absolute. The incomplete injury will vary enormously from person to person and will be entirely dependant on the way the spinal cord has been compromised.

An “incomplete” spinal cord injury involves preservation of motor or sensory function below the level of injury in the spinal cord.

If the patient has the ability to contract the anal sphincter voluntarily or to feel a pinprick or touch around the anus, the injury is considered to be incomplete. The nerves in this area are connected to the very lowest region of the spine, the sacral region, and retaining sensation and function in these parts of the body indicates that the spinal cord is only partially damaged. This includes a phenomenon known as sacral sparing.

The true extent of many incomplete injuries isn't fully known until 6-8 weeks post injury. The spinal cord normally goes into what is called spinal shock after it has been damaged. The swelling and fluid masses showing on any resultant X-ray, MRI or CT scans, may well mask the true nature of the underlying injury. It is not uncommon for someone who is completely paralysed at the time of injury to get a partial or very near full recovery from their injuries after spinal shock has subsided.

Types

Central cord syndrome

Central cord syndrome.

Anterior cord syndrome

Anterior cord syndrome.

Brown-Séquard syndrome

Brown-Séquard syndrome.

Posterior cord syndrome

Posterior cord syndrome: rare

Treatment

The results of kinesiotherapy treatment in patients after incomplete spinal cord injury (iSCI) are inconclusive, mostly due to different, subjective evaluation methods. A study aimed to evaluate the range of functional regeneration in long-term 13 months follow-up using comparative neurophysiological tests after uniform kinesiotherapy in patients with thoracic iSCI.

Material and methods: Comparative tests were performed of sensory perception in dermatomes Th1-S1, electromyography (at rest-rEMG and during maximal contraction-mcEMG) in the muscles of the trunk and lower extremities, electroneurography (ENG) of the motor fibers of the lower extremities, and motor-evoked potential induced transcranially (MEP) before and after treatment in 25 iSCI patients. All subjects were treated with the same kinesiotherapeutic procedures.

A moderate increase was found in amplitudes in rEMG and mcEMG recordings from the rectus abdominis and rectus femoris muscles, MEPs amplitudes, and amplitudes after peroneal nerve stimulations in ENG studies. There was no improvement in sensory perception.

Following the proposed kinesiotherapy algorithm, patients after thoracic iSCI presented a moderate more motor than sensory functions improvement. Applied neurorehabilitation evoked normalization of muscle tension, moderate improvement of rectus abdominis and rectus femoris muscles motor units activity, and motor central and peripheral neural impulses transmission. The comparative neurophysiological assessment provides more precise and objective insight into the functional status of afferent and efferent systems than the classical clinical approach in iSCI patients [1)].

Outcome

Following incomplete spinal cord injuries, neonatal mammals display a remarkable degree of behavioral recovery.

Previously, it has been demonstrated in neonatal mice a wholesale re-establishment and reorganization of synaptic connections from some descending axon tracts (Boulland et al., 2013).

To assess the potential cellular mechanisms contributing to this recovery, Chawla et al., have

characterized a variety of cellular sequelae following thoracic compression injuries, focusing particularly on cell loss and proliferation, inflammation and reactive gliosis, and the dynamics of specific types of synaptic terminals. Early during the period of recovery, regressive events dominated. Tissue loss near the injury was severe, with about 80% loss of neurons and a similar loss of axons that later make up the white matter. There was no sign of neurogenesis, no substantial astroglial or microglial proliferation, no change in the ratio of M1 and M2 microglia and no appreciable generation of the terminal complement peptide C5a. One day after injury the number of synaptic terminals on lumbar motoneurons had dropped by a factor of 2, but normalized by 6 days. The ratio of VGLUT1/2+ to VGAT+ terminals remained similar in injured and uninjured spinal cords during this period. By 24 days after injury, when functional recovery is nearly complete, the density of 5HT+ fibers below the injury site had increased by a factor of 2.5. Altogether this study shows that cellular reactions are diverse and dynamic. Pronounced recovery of both excitatory and inhibitory terminals and an increase in serotonergic innervation below the injury, coupled with a general lack of inflammation and reactive gliosis, are likely to contribute to the recovery [2].

1)

Wincek A, Huber J, Leszczyńska K, Fortuna W, Okurowski S, Tabakow P. Results of a long-term uniform system of neurorehabilitation in patients with incomplete thoracic spinal cord injury. Ann Agric Environ Med. 2022 Mar 21;29(1):94-102. doi: 10.26444/aaem/135554. Epub 2021 Apr 15. PMID: 35352911.

2)

Chawla RS, Züchner M, Mastrangelopoulou M, Lambert FM, Glover JC, Boulland JL. Cellular reactions and compensatory tissue re-organization during spontaneous recovery after spinal cord injury in neonatal mice. Dev Neurobiol. 2016 Dec 29. doi: 10.1002/dneu.22479. [Epub ahead of print] PubMed PMID: 28033684.