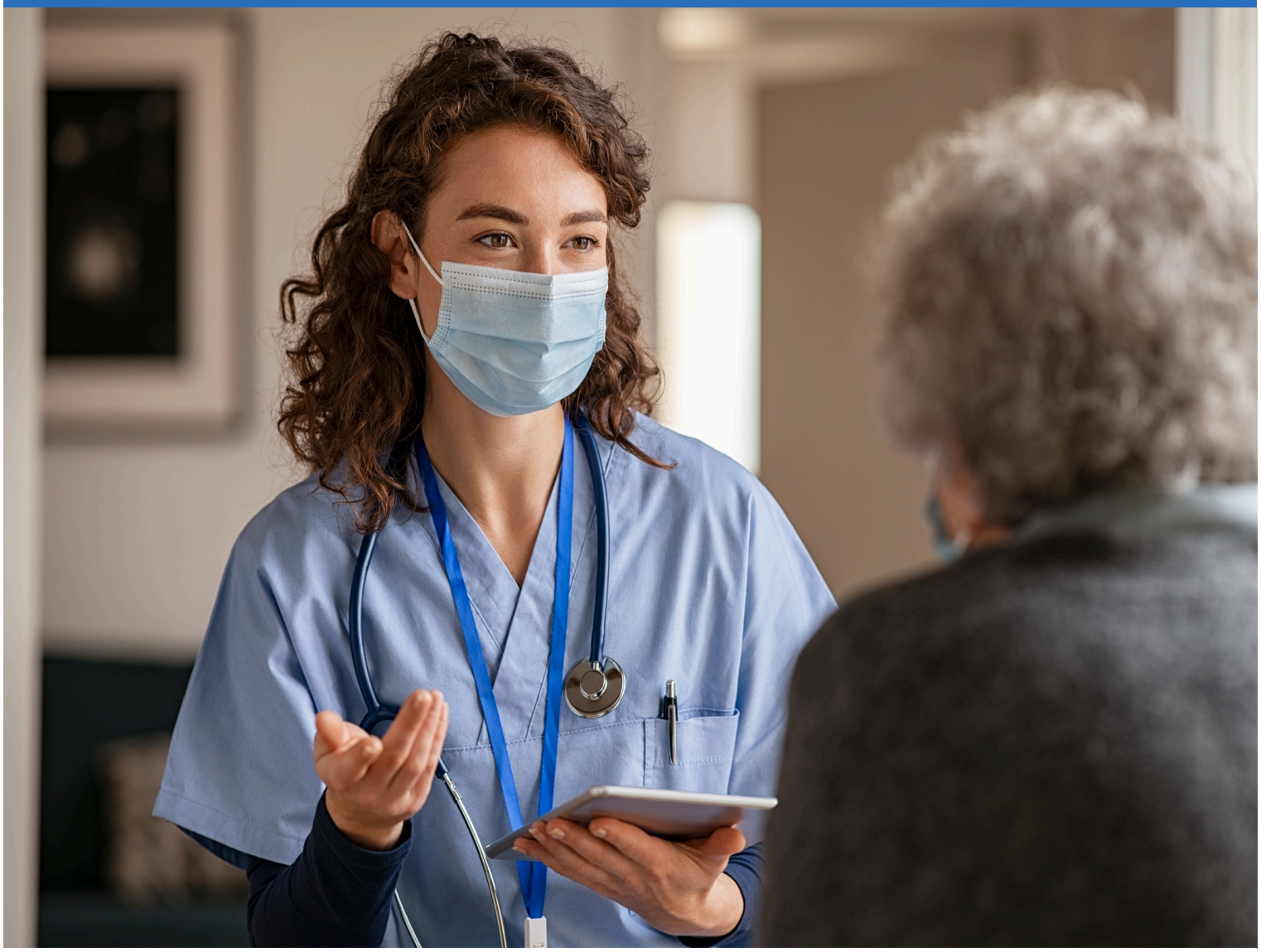
BMJ Best Practice

Chronic spinal cord injury

Straight to the point of care



Last updated: Sep 07, 2023

Table of Contents

Summary

Chronic spinal cord injury is a term generally used when elements of spinal cord injury have been present for at least 1 year.

Primary injury results from acute mechanical trauma, compression by a space-occupying lesion, infection, or a vascular insult. The injury may be exacerbated by ischaemia or inflammation.

The aim of diagnosis is to identify patients who have progressive pathology and to thoroughly assess the disability produced by the injury to guide rehabilitation.

Common problems associated with chronic spinal cord injury include bladder and/or bowel dysfunction, pain (neurogenic and musculoskeletal), gait disturbances, soft-tissue contractures, sexual dysfunction, cardiovascular dysautonomia (including autonomic dysreflexia), and development and/or progression of long-term neurological complications such as syringomyelia and spinal or foraminal stenosis.

Management involves intensive rehabilitation to optimise mobility and hand function, prevention of complications, bladder and bowel management, and analgesia. Surgical decompression may be required for progressive neurological deficits.

Full recovery of neurological function is unlikely, and outcome depends on residual function and the success of rehabilitation. Patients have a decreased life expectancy compared with the general population.

Frequently encountered complications include dysphagia, venous thrombo-embolic conditions, soft-tissue and joint contractures, pressure injuries, orthostatic hypotension, heterotopic ossification, dependent oedema, and low bone mass or osteopenia/osteoporosis.

Definition

Chronic spinal cord injury refers to a permanent and/or progressive interruption in the conduction of impulses across the neurons and tracts of the spinal cord. It may be due to a traumatic or a non-traumatic cause, such as mechanical distortion or vascular ischaemia of the spinal cord arising from trauma, tumour, infection or other space-occupying lesions. The term is generally used when elements of spinal cord injury have been present for at least 1 year. Commensurate neurological deficits occur that may be stable or progressive and lead to disability with associated spasticity, joint contractures, sensory changes, and sphincter and locomotion abnormalities.

Epidemiology

Spinal cord injury (SCI) affects over 40,000 people in the UK and approximately 300,000 in the US.[1] [2] There are an estimated 17,900 new cases of SCI in the US each year. Common causes of traumatic SCI include motor vehicle accidents, falls, sports injuries, and violence. Around three-quarters of patients with SCI are male, and the average age at injury is 43 years.[2]

The epidemiology of non-traumatic SCI is more difficult to assess and describe, and worldwide prevalence of paralysis related to SCI and spinal cord disease is very hard to quantify, but it adds a significant amount of burden to the medical and social systems.[3]

Children and older patients are most at risk of SCI. In children, the cervical spinal cord is particularly vulnerable because of their proportionally larger heads, less developed neck musculature, horizontal orientation of the facets, and relative ligamentous laxity, and because ossification of the uncinate process of the vertebra occurs at around age 7 years. In older patients, the recovery from SCI is diminished due to compromise of the vascular supply by degenerative changes in the cervical spine and atherosclerotic changes in the arteries supplying the cord. Falls are the most common cause of SCI for this population. Older patients also have a higher incidence of metastasis of tumours to the spinal column, and of some primary tumours such as myeloma.

The most frequent neurological category at discharge after acute SCI is incomplete tetraplegia, followed by incomplete paraplegia, complete paraplegia, and complete tetraplegia. [NSCISC: spinal cord injury facts & figures at a glance] (https://www.nscisc.uab.edu/Public/Facts%20and%20Figures%202020.pdf)

Aetiology

In traumatic spinal cord injury (SCI), the initial injury is usually due to compression (from a space-occupying lesion, such as a haematoma) or physical trauma in the form of distraction, shearing, or laceration. In children, the spinal cord is particularly vulnerable because it does not tolerate any stretch. Injury can occur or be exacerbated even in the absence of radiological abnormalities (a phenomenon known as SCI without radiological abnormality [SCIWORA]).[4]

Non-traumatic SCI can be due to compressive (degenerative, tumours) or non-compressive (infectious, systemic inflammatory disorder, acquired demyelinating disorder, genetic, neurodegenerative, metabolic) causes. In older patients, recovery from SCI is diminished due to compromise of the vascular supply by degenerative changes in the cervical spine and atherosclerotic changes in the arteries supplying the cord. Older patients also have a higher incidence of metastasis of tumours to the spinal column, and of some primary tumours such as myeloma.

A narrow spinal canal (a developmental abnormality) increases the risk and severity of injury following trauma or a compressive lesion.[5]

Depending on the direction and magnitude of the forces causing SCI, some neurons and tracts can be preferentially involved. The amount and location of the intact neural tissue determines the residual function. Mechanical compression of the cord, which occurs over a period, allows for gradual compensatory adjustments that preserve function; compressive lesions are tolerated better than sudden mechanical distortion of the cord such as stretch, laceration, or shear. Vascular injuries (which can be primary or secondary to a mechanical injury) are poorly tolerated and have a poorer prognosis for recovery.

Following the initial injury, secondary spinal cord damage results from ischaemia and/or inflammation.

Pathophysiology

The disability produced by spinal cord injury (SCI) depends on the level of the injury and the tracts involved. The essential tracts for movement and sensation are:[6] [7]

- Reticulospinal, vestibulospinal, and rubrospinal tracts for integration of control of voluntary movement and balance
- Corticospinal tracts for voluntary motor control and co-ordinated movements
- Dorsal column and spinothalamic tracts for proprioception, fine touch, and nociceptive function.

Involvement of motor tracts causes an upper motor neuron lesion. Cervical lesions produce tetraplegia/tetraparesis, whereas thoracic and lower lesions produce paraplegia/paraparesis. Injuries of the lumbar spine cause an injury to the nerve roots or the cauda equina. Typically the involved limbs present with spasticity, hypertonia, hyper-reflexia, a feeling of heaviness, stiffness, and an extensor plantar response in an upper motor lesion. When the injury involves the nerve roots, the affected limbs are flaccid and hypotonic (lower motor neuron type injury). Sensory changes include paraesthesias, hyperaesthesia, and proprioceptive changes commensurate with the level of the lesion.

Following the initial mechanical offence, secondary injuries such as ischaemia may occur due to damage or occlusion of the main arteries or perfusing vessels, or a compromised microcirculation. Venous congestion secondary to arterial injury or systemic hypotension produced by autonomic dysfunction can also cause ischaemia. The grey matter is especially vulnerable to ischaemic injury because it is metabolically active and highly vascular.[8] [9] Ischaemic injury, if it occurs, is poorly tolerated by the spinal cord and is associated with a worse prognosis for recovery.

The initial injury can also be exacerbated by secondary inflammation. An inflammatory response is triggered by the tissue damage and the release of cytokines. Disruption of the blood-brain barrier may trigger a systemic inflammatory response.

Compensatory adjustments in the period after the initial injury allow function to be maintained in available uninjured neural tissue. These mechanisms tend to be most effective in slowly progressing injuries produced by compression, and least effective in ischaemic injury. The final disability produced therefore depends on the level of the lesion, the amount of neural tissue spared, the extent of secondary injury from ischaemia or inflammation, and the effectiveness of the compensatory responses that preserve function.

Chronic SCI can lead to a range of complications including (but not limited to) the following:

- Disturbance in bladder function: micturition is a complex process under reflex and voluntary control. It involves the autonomic (parasympathetic and sympathetic) system, and the cortical centres that control the detrusor, internal, and external sphincters to maintain continence. Disturbance most commonly results in inappropriate bladder and sphincter activity, leading to either hyperreflexic neurogenic lower urinary tract dysfunction in the case of an upper motor neuron lesion; or a flaccid, atonic bladder in the case of a lower motor neuron lesion.
- Disturbance in bowel function: defecation and faecal continence also require complex co-ordination of the autonomic nervous system, peristalsis, spinal reflexes, and volitional input. Thus, SCI may lead to neurogenic bowel, characterised by insensate, poorly controlled bowel evacuation.

- Respiratory dysfunction: patients with cervical spine injuries can develop paralysis of ventilatory muscles, leading to a decreased breathing and coughing capability. Disruption of autonomic input can lead to bronchoconstriction and excess secretions. Reduced mobility can produce a ventilation/perfusion mismatch that can exacerbate hypoxia if there is an associated illness.
- Sexual dysfunction: disruption of the autonomic pathways can reduce the ability for erection and ejaculation in men and vaginal secretions in women. Altered sensation, impeding enjoyment of intercourse, can affect both sexes.[10] In several studies, around 60% of respondents had post-injury amenorrhoea, with the average time until menses resumption being 5 months. Although fertility is not generally affected in women following return of menses, a drop in pregnancy rates has been reported, especially in patients with tetraplegia, suggesting additional contributing factors.[11] [12] [13]
- Joint contractures: a loss of spinal cord innervation leads to muscle wasting and fibrous replacement. This, combined with spasticity, can lead to flexion contractures as these structures shorten across joints such as the elbow, hip, knee, or ankle.
- Neuropathic pain: occurs due to a heightened sensitivity of the receptors or inhibition of descending pathways.
- Autonomic dysreflexia: can occur in patients with a lesion affecting T6 or higher level due to disruption of the sympathetic chain and balance with the parasympathetic nervous system. It is caused by an excessive autonomic response to stimuli below the level of the lesion, such as a faecal impaction or blocked catheter. This produces sympathetic overactivity below, and parasympathetic overactivity above, the level of injury. This can present as a sudden uncontrolled rise in blood pressure and a range of other symptoms, including pounding headache, sweating or shivering, anxiety, chest tightness, blurred vision, nasal congestion, blotchy skin rash or blushing above the lesion level, and cold with goosebumps (cutis anserina) below the level of the injury.

Pathway	Direction	Origin	Cross-over	Destination	Function
Corticospinal	Descending	Motor cortex	90% as lateral CST; 10% as anterior CST	Alpha motor neurons	Skilled motor to extremities
Reticulospinal	Descending	Reticular formation	Variable levels	Alpha and gamma motor neurons	Cortical control of voluntary motor function
Rubrospinal	Descending	Red nucleus	Immediate	Alpha and gamma motor neurons	Facilitate flexors, inhibit extensors (for balance control)
Vestibulospinal	Descending	Vestibular nucleus	None	Alpha and gamma motor neurons	Facilitate extensors and inhibit flexors (for balance control)
Lateral spinothalamic	Ascending	Free nerve endings	Immediate	Posterior central gyrus	Pain, temperature
Anterior spinothalamic tract	Ascending	Free nerve endings	Variable levels	Posterior central gyrus	Light touch and pressure
Posterior column: fasciculus gracilis and cuneatus	Ascending	Meissner's corpuscles, Pacinian corpuscles, muscle spindle, tendon organs	None	Posterior central gyrus	Discriminative touch, vibration sense, proprioception

Neuroanatomy and functions of the spinal tracts. CST, corticospinal tracts

From personal collection of Dr Jwalant S. Mehta

Classification

American Spinal Injury Association (ASIA) International Standards for Neurological Classification of Spinal Cord Injury

The ASIA Impairment Scale classifies the injury as A, B, C, D, or E according to the degree of residual neurological function: [SCIRE: international standards for neurological classification of spinal cord injury] (https://scireproject.com/outcome/ais)

- A: no motor or sensory function
- B: preserved sensory function including S4-S5 (sacral sparing) but no motor function
- C: preserved sensory function including S4-S5 (sacral sparing); muscle flicker or full range of motion (ROM) with gravity eliminated
- D: preserved sensory function including S4-S5 (sacral sparing); full ROM against gravity or added resistance
- E: preserved sensory function including S4-S5 (sacral sparing); full ROM with normal-strength; spasticity, or long tract signs.

Lesions can also be classified as complete or incomplete:

- Complete: any type A injury
- Incomplete: any type B, C, D, or E injury.

Spinal cord syndromes

The pattern of weakness and loss of sensation due to the disruption of spinal tracts can additionally be described by several classic syndromes. The clinical examination may reveal a characteristic spinal cord syndrome that should be recognised. Spinal cord syndromes include:

- Brown-Sequard syndrome: produces root pain at the level of the lesion with loss of pain and temperature modalities below the lesion on the ipsilateral side, and over the lower extremity and the trunk on the contralateral side. The sensory level is 2 levels lower on the contralateral side. A spastic weakness is seen on the ipsilateral side with brisk reflexes. The posterior column may be involved on the ipsilateral side, producing loss of proprioception, and urgency may be noted.
- Complete cord transection: a bilateral sensory level at the level of the lesion affecting all the modalities, which are either absent or severely affected. The upper margin may show a zone of hyperaesthesia. Flaccid paraplegia or tetraplegia are seen. Urgency and incontinence occur due to a reflex neurogenic lower urinary tract dysfunction.
- Posterior column compression: although no longer included as a formal syndrome, this produces loss of proprioception, with an associated spastic paraparesis and brisk reflexes. There may be radicular pain along the intercostal nerve. The sensory level to pain on the trunk is 2 levels below the level of the lesion. A lesion of T12 or above produces loss of abdominal reflexes. Urgency and incontinence are seen. Lesions above T5 may produce autonomic dysfunction such as orthostatic hypotension, sweating, and bradycardia with bladder or rectal stimulatory response of distension.
- Central cord syndrome: caused by a lesion that affects the inner tracts in the cervical cord. Early in the pathology, the symptoms are predominantly sensory with a zone of pain and temperature loss 2 to 3 levels caudal to the lesion. If the lesion expands, motor involvement occurs producing bilateral spastic paraparesis of the lower extremities and asymmetric upper extremity paraparesis; neurological impairment is more severe in the upper extremities.
- Anterior cord syndrome: this is due to vascular injury involving the anterior vertebral artery. This may be due to a flexion injury involving the anterior two-thirds of the spinal cord, which injures the

anterior vertebral artery but spares the paired posterior vertebral arteries. Pain, temperature, and motor function below the level of the lesion are impaired, but proprioception is intact.

Case history

Case history #1

A 44-year-old man presents with a 3-month history of thoracic pain that has increased in severity in the previous 3 weeks. The pain eases by lying flat, but keeps him awake at night. He has noticed a weight loss of 20 kg in the previous 3 months associated with a loss of appetite and frequent night sweats. He has been through a rehabilitation programme for alcohol abuse. In the previous 3 weeks he has noticed that he has a progressively unsteady gait and has had frequent falls. He has noticed sharp burning paraesthesias in both lower extremities and the lower abdomen, which coincide with the onset of the severe back pain. On examination of his lower limbs, he has spasticity, brisk deep tendon reflexes, extensor plantars, and a sensory level of T6 affecting proprioception, light touch, and pinprick. He has a sharp tenderness over T6, with an angular kyphosis of T5 and T6. In due course, he was transferred to a spinal cord rehabilitation facility. Over the past 6 months, he has undergone extensive rehabilitation, including bladder care and prevention of joint contractures due to increasing spasticity. He is dependent on his wheelchair.

Case history #2

A 22-year-old man was involved in a motor vehicle accident 12 months earlier. He was driving under the influence of drugs and alcohol when the car crashed into a tree, and he was not wearing a seatbelt. He was taken to the accident and emergency department where he was found to have sustained a tear-drop fracture of C6. A neurological assessment determined that he had tetraplegia without sacral sparing. He underwent an emergency C6 vertebrectomy, cord decompression, and anterior stabilisation but the neurological deficit did not improve. He was transferred to a spinal cord rehabilitation facility 2 weeks later. Over the previous 12 months, he has undergone extensive rehabilitation, including bladder care and prevention of joint contractures due to increasing spasticity. He is dependent on his wheelchair.

Approach

People living with chronic spinal cord injury (SCI) may have several associated medical sequelae that can affect nearly every organ system, and these should be thoroughly assessed. An accurate assessment requires a knowledge of the anatomy and function of the tracts in the spinal cord.[28] [29]

History

Primary insult

The initial history of the inciting injury should be documented. This should include mechanism of injury, diagnostic findings such as imaging, treatments and/or surgical interventions, and any complications. Due to the strong correlation between SCI and brain injury, any loss of consciousness, altered memory of events, or cognitive changes should be noted. Most patients will have participated in an acute rehabilitation programme, and these records are invaluable in understanding their injury, recovery course, functional status, and medical sequelae.

Neurological injury

It is important to note that imaging level of injury and neurological level of injury often do not directly match: the latter is usually at a higher level. Although it is common to use the American Spinal Cord Injury Association (ASIA) classification system and International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) ASIA exam to classify neurological status after SCI, its prognostic use can only be applied to traumatic injuries.[30] If available, ASIA exam results should be maintained for reference if a neurological change is suspected. A thorough history will include the mechanism, level of injury, ASIA classification, and neurological recovery course. Routine follow-up should include a review of any changes noted by the patient, rehabilitation team, or provider.

Functional status

Any activities for which the patient needs assistance and/or use of adaptive equipment (e.g., wheelchair, transfer board, braces) should be documented. Any decline in function or independence requires an in-depth investigation into the underlying cause, such as a new infectious process, musculoskeletal injury, progression of spinal pathology, or new spinal pathology.

Bladder control and renal health

Both upper and lower urinary tract complications are common regardless of the mechanism of SCI. Urinary retention with the inability to void is the most common presentation (reflex neurogenic lower urinary tract dysfunction).[31] [32] A careful bladder history will include the ability of the patient to sense a full bladder, how they void (including frequency, typical volumes, and catheter size for intermittent catheterisation if applicable), and frequency of incontinent leakage. History of urinary tract infections, including culture results, resistance, and treatments, should be documented. Results from urodynamic testing, renal ultrasounds, kidney scans, or other urological testing should also be documented.

Bowel control

History should assess the primary goal of predictable bowel movements and social continence. The patient should be asked about their ability to recognise the need to evacuate, level of volitional sphincter control, frequency and consistency of bowel movements, constipation events, episodes of incontinence,

and history of haemorrhoids. The history should also include confidence to participate in activities outside the home. Incontinence caused by a new loss of rectal tone requires neurological assessment.

Spasticity

The patient should be asked whether and how spasticity is affecting them, such as causing pain, preventing sleep, and effects on functional status. The history should also include typical triggers for that patient, and any changes in spasticity and range of motion.

Pain

Pain is common and often complex after SCI. There are often multiple pain generators, and each requires individual attention, assessment, and treatment planning.[33] It can be helpful to group these into three categories:

- Pain above the level of injury: often musculoskeletal in nature, such as with healing traumatic injuries, shoulder overuse injuries, or focal overuse neuropathies such as ulnar or median neuropathies
- Pain below the level of injury: this is commonly neuropathic in nature with a central generator
- Pain at the level of injury: also commonly neuropathic, but it may present as having a distinctly separate character such as tightness or pressure sensation.

History should include effectiveness of medical and non-medical interventions, and impact on function and quality of life. Loss of pain due to loss of sensation should be evaluated from a neurological standpoint.

Autonomic dysfunction

Patients living with SCI may have difficulty with regulation of temperature, blood pressure, heart rate, and bronchospasm. This can lead to hyper- or hypothermia, orthostasis, and slowed cardiac response to activity demands.

Autonomic dysreflexia

Patients with a neurological level at or above T6 may experience a dysautonomic hypertensive emergency called autonomic dysreflexia. This is most commonly caused by constipation, bladder distention, or bladder irritation, but can be triggered by any noxious stimulus below the level of injury. It presents clinically as a relative increase in systolic blood pressure >20 mmHg above baseline and relative bradycardia due to a parasympathetic response. Any history of autonomic dysreflexia should be documented and discussed, including symptoms during the event such as pounding headache, facial flushing, piloerection, anxiety, sweating, stuffy nose, or crying (in the paediatric patient). A history should include triggers and the patient's emergency plan.

Orthostatic hypotension

It is common for patients to have a resting systolic blood pressure much lower than their pre-injury baseline (e.g., 90 mmHg). Orthostatic hypotension is also common due to decreased neurovascular tone and can have an impact on daily function as well as risk of syncope. The patient should be asked about symptoms and strategies used to control symptoms, such as compression stockings, abdominal binders, increased oral fluids, and any medicines.

Venous thrombosis

Risk of venous thrombosis remains elevated lifelong after SCI. Any prior thromboses should be noted, as well as any prior or current implanted devices such as a vena cava filter. All unexplained limb swelling requires investigation.

Cardiovascular disease

Cardiovascular disease is the second most common cause of death in patients living with SCI.[34] A careful cardiac history should be obtained regardless of the patient's age.

Respiratory health

Respiratory complications (e.g., atelectasis, pneumonia) are the leading cause of morbidity and mortality after SCI.[34] The impact of SCI on the respiratory system varies widely by injury severity, from lifelong ventilator dependence to normal respiratory function. Bronchospasm may be brought on by autonomic dysregulation. Respiratory symptoms and vaccinations against respiratory disease should be documented.

Bone health

There is increased risk of fragility fractures across all ages following SCI, correlated with severity of paralysis. This is due to the rapid and substantial bone density loss following paralysis, decreased physical forces across bone, and other complex neurohumoral factors.[35] [36] History should include history of prior fractures, vitamin D levels, bone densitometry results, management such as with bisphosphonates, and any family history of osteoporosis or fragility fractures.

Skin health

Altered sensation and paralysis lead to an increased risk of pressure-related injuries, and incontinence can lead to moisture-related skin breakdown. Patients should be asked about any skin problems, how often they inspect their skin, and pressure-relieving strategies.

Sexual dysfunction and fertility

Sexual function is often impacted by SCI, and this should be discussed if the patient is open to this dialogue. It should be noted that psychological factors can play a confounding role in this population as they do in the general population. Male patients should be asked about erections to include strength, duration, if an erection is possible by psychological arousal, if an erection is possible by direct stimulation, ability for orgasm, and if ejaculate is present. Any medicines and blood flow restrictive devices should be noted. Female patients should be asked about lubrication production, if arousal can be obtained through psychological means, if arousal can be obtained through direct stimulation, and ability for orgasm. As sexual interactions can produce autonomic dysreflexia events in both men and women, this should also be discussed. Fertility significantly decreases in males with SCI and an intention for children should be discussed.[37] Females living with SCI do not usually have reduced fertility following an initial phase of amenorrhoea, but this may be under-investigated.[38] [39]

Psychological comorbidities

There is increased risk of depression, anxiety disorders, substance use disorders, PTSD, and suicide in patients with SCI.[40] [41] Screenings should be conducted at each visit, to include current symptoms and lifetime history.[41]

Social integration

Following SCI, people will face personal, internal, and external barriers to their roles in the home, in work, and in social and leisure activities. Patients should be asked about time and activities outside the home, hobbies, work status, and other social activities they enjoy.

Physical examination

The neuromuscular assessment of people living with SCI uses multiple specialist evaluations. Documentation is important in order to recognise possible neurological changes and to help stratify urgency of subjective reports of functional decline. A neuralgic change found on examination always requires further assessment.

Assessment of muscle tone

Patients may have flaccidity or spasticity. Spasticity can be assessed and graded using the modified Ashworth Scale:[42]

- 0: no increase in muscle tone
- 1: slight increase in muscle tone, with resistance at the end of the range
- 1+: slight increase in muscle tone, with increased resistance throughout the range
- 2: marked increase in tone throughout the range
- 3: increase in muscle tone, passive movement difficult
- 4: joint rigidly held in flexion or extension.

Spasticity and increased muscle tone is dynamic and can fluctuate. It can lead to joint contractures, loss of function, and increased risk of skin breakdown.

Assessment of muscle strength

The assessment of specific muscles correlates with a specific spinal level. Motor strength of the following muscles is assessed:

- Elbow flexors (C5)
- Wrist extensors (C6)
- Elbow extensors (C7)
- Fingertip flexors (C8)
- Little-finger abductors (T1)
- Hip flexors (L2)
- Knee extensors (L3)
- Ankle dorsiflexors (L4)
- Great-toe extensors (L5)
- Ankle plantar flexors (S1).

The motor strength of each muscle group should be scored as follows:

- 0: no muscle contraction
- 1: palpable flicker of muscle contraction
- 2: full range of motion (ROM) with gravity eliminated
- 3: full ROM against gravity
- 4: full ROM against resistance, but less than normal strength

- 5: full ROM and normal strength following added resistance.

Obtaining an accurate score is crucial. The full ROM should be tested. A good approach is to test the full ROM against gravity and either proceed to eliminate gravity if the muscle group is too weak, or apply resistance if there is a full ROM. When testing for a flicker of muscle contraction, gravity should be eliminated in the upper extremities, or the antigravity position used in the lower extremities. If contractures are present that reduce the ROM by >50%, motor strength cannot be accurately assessed; this should be documented. It is important to ensure that the patient is not performing trick movements, which involve using other joints and muscle groups (e.g., extension of the shoulder during testing of elbow extension); if missed, these can lead to an inappropriately high score.

Assessment of sensation

Sensory deficits are produced by involvement of multiple spinal tracts. Pain and temperature are transmitted by the lateral spinothalamic tracts, and light touch by the anterior spinothalamic tract. Proprioception is transmitted by the posterior column. There may be hyperaesthesia, hyperalgesia, or numbness.

Light touch and pinprick sensation should be assessed for each dermatome on the left and right, and scored as follows:

- 0: cannot distinguish if touched or not, on light touch testing; cannot distinguish sharp from blunt ends on pinprick testing.
- 1: can appreciate if touched on light touch testing, and can distinguish sharp from blunt ends on pinprick testing, but the sensation is diminished compared with the face.
- 2: can appreciate if touched on light touch testing, and can distinguish sharp from blunt ends on pinprick testing, and the sensation is the same compared with the face.

Sacral sensation and motor function should be assessed to determine whether the injury is complete or incomplete. This is done by assessing voluntary anal contraction, anal tone, and sensation in S4 and S5 dermatomes. If there is an absence of all these sacral elements, the injury is classified as a complete injury. Sacral sparing imparts incomplete injury status.

Assessment of reflexes

In spastic paralysis, the reflexes are brisk and may be associated with clonus. Several pathological reflexes may also be elicitable. These include Babinski's sign (extensor plantar), inverted radial reflex, Hoffman's sign (finger flexor), Lhermitte's sign, scapulohumeral reflex (indicates a lesion above C4), finger escape sign, and the Oppenheim's sign. In flaccid paralysis, the reflexes are absent in the affected extremities. It is again important to document what is 'normal' for a patient in order to detect changes in the neurological examination.

Investigations

Imaging

- MRI of the spinal cord should be performed if there is an acute deterioration or new neurological symptoms.[43]

Electromyography

- Electromyography, nerve conduction studies, and somatosensory-evoked potentials can localise the site and type of lesion. Repeat studies can assist in determining if the neurological status is stable (compared with a baseline obtained 6-12 weeks after the SCI) or if there is any recovery.

Bladder and renal studies

- Bladder ultrasound to assess retention and completeness of bladder emptying, and renal ultrasound to detect renal stones, should be performed at least annually, and as needed.[44] [45] Invasive assessments that may be considered include a micturition cysto-urethrogram and urodynamic studies.[44] [46]

Bone health

- Bone densitometry (DEXA) to assess loss of bone density and response to medical management should be performed every 1-2 years depending on severity of bone loss.[47] [48]

Laboratory evaluation

- The following should be checked annually as part of health maintenance screening: full blood count; comprehensive metabolic panel with magnesium, calcium, and phosphorus; liver function panel; lipid panel; haemoglobin A1c; 25-hydroxyvitamin D level.
- Metabolic bone markers (e.g., C-telopeptide, osteocalcin, procollagen type 1 N propeptide) may be measured to assess bone turnover.

History and exam

Key diagnostic factors

presence of risk factors (common)

- Key risk factors include spinal cord trauma or ischaemia, higher level spinal cord lesion, extremes of age, and a narrow spinal canal.

motor weakness (common)

- Motor weakness is the most common, classic sign of spinal cord injury.

loss of fine motor co-ordination (common)

- Involvement of the inner corticospinal tracts can lead to a deficit in the muscles concerned with fine motor functions in the hand. This may result in difficulty in prehensile activities and in eating, and result in dropping small objects, and difficulty in writing (if there is involvement of the dominant extremity). These deficits can be very disabling.

spasticity (common)

- Can be graded using the modified Ashworth Scale.[42] Severe spasticity can be very disabling.

paraesthesia, numbness, dysaesthesia (common)

- Spontaneous abnormal sensations may be noted early in the pathology and increase in severity to a sharper, burning character (dysesthaesia) with chronic spinal cord changes. There may be associated hyperaesthesia, hyperalgesia, or numbness.

hyperreflexia and ankle clonus (common)

- Hyperexcitable state of the stretch reflex leads to brisk deep tendon jerks that can lead to clonus.
- Negotiating stairs, especially when descending, can elicit ankle clonus and lead to unsteadiness and frequent falls.

pathological reflexes (common)

- These include Babinski's sign (extensor plantar), inverted radial reflex, Hoffman's sign (finger flexor), Lhermitte's sign, scapulohumeral reflex (lesion above C4), finger escape sign, and Oppenheim's sign.

contractures (uncommon)

- Spasticity and increased muscle tone can lead to joint contractures, which classically produce elbow flexion and knee extension.
- Keeping joints supple and free of contractures is an important treatment goal to prevent secondary disability.

loss of perianal sensation, voluntary anal contraction, and anal tone (uncommon)

- A sign of a complete lesion. If perianal sensation, anal tone, or anal contraction are preserved, the patient has sacral sparing and the lesion is partial.

autonomic dysreflexia (uncommon)

- Patients develop a sudden, uncontrolled rise in blood pressure, pounding headache, sweating or shivering, anxiety, chest tightness, blurred vision, nasal congestion, blotchy skin rash or blushing above the lesion level, and cold with piloerection below the level of the lesion.
- A relative increase in systolic blood pressure >20 mmHg above baseline is diagnostic.
- The most likely causes are bladder distension due to catheter blockage (if catheter is in place), urinary retention (if catheter is not in place), or faecal impaction, but can be triggered by any noxious stimulus below the level of injury.

syrinx (uncommon)

- A syrinx may develop in some patients. This refers to the development of a cavity (hydromyelia) in the substance of the cord.
- While the true percentage of clinically silent lesions is not known, a small proportion of patients develop debilitating symptoms. These include the onset of pain and loss of sensorimotor levels. In patients this can result in loss of respiratory stability or arm function, in the case of upper cord injuries. These symptomatic lesions typically progress over time. Symptomatic lesions may need to be addressed with surgical decompression.

Other diagnostic factors

central (midline) pain (common)

- Midline pain may arise from a variety of pain generators, ranging from dura to osseous nociceptive nerve endings.[33]
- The pain is typically axial, constant, and relieved with rest.
- Spinal cord tumours produce a different pattern in which the pain is present at rest, even when recumbent, and is nocturnal.

girdle pain (common)

- Radiation from a thoracic lesion can present as a girdle pain if it involves a nerve root and is noted in the dermatome or myotome supplied by the root or the intercostal nerve.[33] It is aggravated by coughing and sneezing. Bilateral involvement can present as a 'tight-belt' constriction.

musculoskeletal or visceral pain (common)

- The pain is usually positional and clearly distinguishable in character and distribution from neurogenic pain.

unsteady gait (common)

- Unsteady gait can be due to a combination of weakness of the anti-gravity lower limb muscles, loss of proprioceptive sensations, and spasticity of the muscles. This can lead to heaviness of the legs, frequent trips and falls around the home, and difficulty in negotiating stairs.

urinary incontinence or retention (common)

- Both upper and lower urinary tract complications are common regardless of the mechanism of SCI. Urinary retention with the inability to void is the most common presentation (reflex neurogenic lower urinary tract dysfunction); alternatively, incontinence may be present.[31] [32] Spinal imaging and neurological status are not accurate estimates of the sphincteric status; urodynamic studies are required for proper assessment.[46]

constipation (common)

- Constipation is common following SCI and can lead to faecal impaction with overflow incontinence.

sexual dysfunction (common)

- Controlled by complex autonomic pathways with cortical input. Parasympathetics control erection whereas sympathetics control erection and ejaculation. Potency affects males. Reduced sensation can lead to reduced pleasure in both sexes. Lesions of the cord can lead to priapism.

non-specific malaise (uncommon)

- SCI substantially alters the symptomatology of many conditions. In particular, symptoms and signs of pain and focal tenderness do not occur below the level of the lesion. Patients presenting with an intercurrent illness may only report a non-specific feeling of malaise.

radicular pain (uncommon)

- The pain may radiate in the dermatome or myotome of any root and cause pain in the extremities. If involving the dorsal root it may cause pain in relation to the posterior musculature. It is increased by coughing or sneezing.

Risk factors

Strong

spinal cord trauma or ischaemia

- The initial spinal cord injury (SCI) is usually due to compression (from a space-occupying lesion) or physical trauma in the form of distraction, shearing, or laceration. Common causes of traumatic SCI

include motor vehicle accidents, falls, sports injuries, and violence. [NSCISC: spinal cord injury facts & figures at a glance] (https://www.nscisc.uab.edu/Public/Facts%20and%20Figures%202020.pdf)

- Depending on the direction and magnitude of the forces causing the injury, some neurons and tracts can be preferentially involved. The amount and location of the intact neural tissue determines the residual function. Mechanical compression of the cord, which occurs over a period, allows for gradual compensatory adjustments that preserve function; compressive lesions are tolerated better than sudden mechanical distortion of the cord such as stretch, laceration, or shear. Vascular injuries (which can be primary or secondary to a mechanical injury) are poorly tolerated and have a poorer prognosis for recovery.

higher-level spinal cord lesion

- Injury to higher levels of the spinal cord causes interruption of the conduction of large-diameter fibres. Subaxial cervical lesions cause tetraplegia and thoracic lesions cause paresis of the lower extremities. Injuries of the lumbar spine cause an injury to the nerve roots or the cauda equina. High cervical lesions result in a profound neurological injury that can require ventilatory support.

Weak

extremes of age

- In children, the spinal cord is particularly vulnerable because it does not tolerate any stretch. Injury can occur or be exacerbated even in the absence of radiological abnormalities (a phenomenon known as SCI without radiological abnormality [SCIWORA]).
- In older patients, recovery from SCI is diminished, due to compromise of the vascular supply by degenerative changes in the cervical spine and atherosclerotic changes in the arteries supplying the cord. Falls are the most common cause of SCI for this population. Older patients also have a higher incidence of metastasis of tumours to the spinal column, and of some primary tumours such as myeloma.

narrow spinal canal

- This is a developmental abnormality that increases the risk and severity of injury following trauma or a compressive lesion.

male sex

- Around three-quarters of patients with SCI are male; this is likely due to the stronger preponderance of risk-taking behaviour in males.[2]

Investigations

Other tests to consider

Test	Result
MRI spine • Recommended if there are any new acute neurological or functional changes.[43] • Perform as needed.	**vertebral body or spinal cord lesions**
EMG • Can be used if a focal compression neuropathy is suspected. • Can help differentiate symptoms with a peripheral source from those with a central source such as the cord. • Perform as needed.	**aetiology and localisation of neuromuscular pathology**
urodynamic studies • Required for proper assessment of co-ordinated urinary bladder and sphincter function.[46] • Follow-up studies can be used to monitor the evolution of the dysfunction and detect response to medicines.[44] • Perform as needed.	**urinary bladder and sphincter function; bladder pressures; bladder capacity and compliance; bladder spasticity**
bladder ultrasound • Used to assess retention and completeness of bladder emptying. • Used in conjunction with a renal ultrasound to detect urinary tract stones. • Perform as needed and annually with a renal ultrasound.[44] [45]	**incomplete bladder emptying; urinary tract stones**
renal ultrasound • Used in conjunction with a bladder ultrasound to assess the health of the urinary system. • Perform annually with a bladder ultrasound.[45]	**identifies renal stones, hydronephrosis, pyelonephritis, and scarring**
micturition cysto-urethrogram • A dynamic and invasive assessment of the flow mechanism. • Used to visualise bladder hypertrophy and other pathology. • Perform as needed.	**incomplete bladder emptying; obstruction; bladder reflux to the kidney; bladder hypertrophy**
bone densitometry (DEXA) • Used to assess loss of bone density and response to medical management. • Perform every 1-2 years depending on severity of bone loss.[47] [48]	**bone density changes**
laboratory evaluation • Should be checked annually as part of health maintenance screening: full blood count; comprehensive metabolic panel with magnesium, calcium, and phosphorus; liver function panel; lipid panel; haemoglobin A1c; 25-hydroxy vitamin D level. • Consider metabolic bone markers (e.g., C-telopeptide, osteocalcin, procollagen type 1 N propeptide) in conjunction with endocrine specialty to assess bone turnover.	**changes in health, organ system, cardiovascular disease risk factors, bone metabolism**

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Non-compressive myelopathy	• Similar neurological picture. No history of trauma or other acute event. • Examples include multiple sclerosis, neuromyelitis optica spectrum disorder, idiopathic transverse myelitis, Guillain-Barré syndrome, motor neuron disease.	• MRI for evidence of cord parenchymal disruption and pattern suggestive of an aetiology. Electrodiagnostic studies additionally for Guillain-Barré syndrome and motor neuron disease.
Compressive myelopathy	• Similar neurological picture. No history of trauma or other acute event. May be signs/ symptoms of infection or malignancy. • Examples include vertebral compression fractures, intervertebral disc disease, malignancy, infection (spinal epidural abscess), vascular malformation or haematoma.	• MRI demonstrates parenchymal lesion and source of spinal cord compression. CT scans better evaluate boney architecture. • FBC, CRP, and erythrocyte sedimentation rate usually elevated in infection.

Approach

The approach to care for patients with chronic spinal cord injury (SCI) comprises management of secondary complications, identifying progression of spinal pathology, and ongoing rehabilitation. The aim is to minimise further complications and maximise functional independence. Due to the breadth and complexity of medical problems and risks following SCI, it is common for multiple medical specialties to be part of a patient's multidisciplinary care; these may include urology, nephrology, orthopaedics, endocrinology, cardiology, and psychiatry.[49]

Surgical intervention for progressive neurological deficit

Progressive neurological deficit does not necessarily relate to the original SCI - it may indicate cord compression by metastases, primary spinal cord tumours, extradural haematoma or abscess, or intervertebral disc prolapse, and requires investigation. If any of these conditions is diagnosed, timely surgical decompression and stabilisation of the involved spinal column is required.[15] Patients require reassessment of their neurological function post-surgery, and require rehabilitation if there is residual neurological deficit.

If progressive neurological deficit indicates secondary ischaemia or inflammation following the acute injury, there is no role for surgery.

Therapeutic interventions

Many interventions that are started after acute SCI are ongoing for patients with chronic SCI. There has been a shift regarding the role of therapy in the context of SCI-related paralysis. While traditionally therapy emphasised compensatory approaches, with focus on strengthening and shifting performance of most function to the unaffected muscles above injury level, emerging activity-based therapies aim to optimise neurological function through neuroplastic interventions.[50] [51]

The goal for any rehabilitation programme for SCI-related paralysis is to maximise day-to-day functioning to achieve levels similar to before the injury through compensatory and restorative approaches. Compensatory approaches involve maximising the strength and functionality of the intact, non-affected parts of the body, while restorative approaches aim to improve mobility and daily activities by optimising neuro-recovery.[51] [52] [53] [54]

Physiotherapy is used to maintain joint range of motion (ROM) and mobility. This stimulates circulation and aims to prevent deformities, muscle and secondary joint contractures, and osteoporosis.[55] Activity-based therapies can promote neurological recovery and thus enhance sensory-motor and autonomic function.[56] [57] [58] [59] [60]

Specific components include the following:

- Mobility and transfers: techniques are taught in order to facilitate position changes for pressure relief, dressing, daily self-care activities, sleeping, and transfers to and from a wheelchair. Different sitting mechanisms, supports, and methods of rising from the supine to the upright position are explored and taught.[61]
- Wheelchair mobility: effective use of power or manual wheelchairs requires training. A variety of different options are available, and control can be customised based on the level of functioning of the patient. Chin, mouth, and hand controls are available for power wheelchairs; power-assisted technology can be used to improve ability to navigate different spaces using manual

wheelchairs.[62] [63] Advanced manoeuvring techniques can be taught to enable patients to turn in tight spaces and negotiate ramps, slopes, and kerbs.

- Walking: there are multiple interventions that can be used to enable a patient with SCI-related paralysis to practise gait, but neurological status is the primary predictor of the ability of the patient to walk over ground independently. Patients with at least full ROM of the lower limbs with gravity eliminated are more likely to be able to walk with aids. Locomotor training uses interventions such as treadmill body weight-supported walking or robot-assisted walking.[64] [65] Orthoses for joint support or a reciprocating gait orthosis can be used for aiding ambulation. In patients with more severe motor involvement, standing may be possible with a tilt table, frames, upright wheelchairs, or parallel bars. Walking may be difficult if there is concomitant upper limb paralysis, lack of pelvic control, loss of proprioception, obesity, joint contracture, or spasticity.[54] [66]
- Hand function: the aim is to improve function using neuro-restorative and/or compensatory interventions.[67] [68] The compensatory rehabilitative model builds on available function using training and orthoses to maximise dexterity. Splints hold joints in functional positions and can be adapted depending on the function required. Regular hand therapy can be performed out of the splint to maintain flexibility and full passive ROM. The tenodesis effect (passive finger flexion in response to wrist extension) can be taught to some patients to allow them to use the basic pinch, grasp, and 3-point grips. The activity-based therapy model utilises task-specific training and massed practice in order to improve upper limb function.[69] [70] Surgical reconstructions using arthrodesis, tenodesis, and tendon and nerve transfers may be considered in suitable patients.
- Exercise: a variety of exercise interventions, including passive ROM, strengthening and conditioning, functional electrical stimulation, and electrically stimulated resistance exercise, may improve arterial function and help neuro-recovery in patients with SCI.[71] [72] [73]

Supportive care

Respiratory function

Maintenance of good respiratory function is vital. Regular airway clearance techniques and clinical assessment and ongoing monitoring of pulmonary function are recommended to ensure adequate airway clearance.[74] A regular change in position and posture and regular deployment of assisted cough and regular breathing exercises (incentive spirometry) are useful in preventing secondary respiratory problems.[75] In patients requiring ongoing ventilatory support, non-invasive approaches appear to be associated with fewer complications than invasive ventilation.[74] Resistive inspiratory muscle training has been found to have a positive short-term effect on inspiratory muscle function in patients with SCI who have impaired pulmonary function.[76]

Pressure ulcers

Pressure ulcers typically occur under the sacrum (lying supine), ischial tuberosities (sitting), or trochanters (lying on one side). They are prevented by good nursing, regular change in position, padding of prominences, maintaining cleanliness, and regular checking of the skin. Surgical management is required in the presence of necrotic tissue.[77] See Pressure ulcers .

Prevention of venous thromboembolism (VTE)

Patients at increased risk of thrombosis (e.g., immobilised for bed rest, or admitted for medical illness or surgery) should be given prophylaxis to prevent VTE and possible pulmonary embolism.[78] Pharmacological prophylaxis should be used unless contraindicated; non-pharmacological measures

(e.g., graduated compression stockings, intermittent pneumatic compression devices) may be used for patients at high risk for bleeding. See Venous thromboembolism (VTE) prophylaxis .

Bladder management

Most patients with SCI have impairments in bladder function, be it storage, evacuation, or both.[79] Neurogenic lower urinary tract dysfunction can be characterised as upper motor neuron type, lower motor neuron type, or mixed.[80] [81]

The preferred method of management is intermittent self-catheterisation. Use of an indwelling catheter with an external collection system may also be considered; however, this increases the risk of urinary contracture and infection. It is important that the method used maintains a low-pressure system, prevents bladder overdistension, and ensures complete emptying.

Management strategies can be developed based on bladder studies (post-void ultrasound, urodynamic assessments, and micturition cysto-urethrogram).[82] [83] Pharmacological management is aimed at optimising storage and elimination by using agents that decrease detrusor hyper-reflexia, improve bladder compliance, and address detrusor-sphincter dyssynergia (e.g., anticholinergics, beta agonists, alpha-blockers, botulinum toxin injection, and sometimes cholinergic agents).[84]

Bowel management

Constipation is the most common problem associated with chronic SCI, and it can lead to impaction and overflow incontinence if not managed appropriately. A bowel management programme should be developed and customised for each patient.[85] [86] [87]

Components of the programme include:

- Dietary management: consumption of a balanced diet, including fibre and stimulant foods, with fluid intake >2 litres/day.
- Regular routine: take meals and aim to have bowel movements at the same time each day; use the same location for bowel movements.
- Physical manoeuvres and positioning: these include stimulation of the gastrocolic reflex by a hot dietary trigger, abdominal massage, and physical activity to promote defecation. The patient should sit on a commode or toilet if possible.
- Local triggers for defecation such as digital stimulation, suppositories, or manual evacuation.
- Pharmacological treatments: stool softeners are preferred. Stimulant or osmotic laxatives are indicated only if constipation persists despite optimisation of all other components of the bowel management programme. Bowel obstruction should be excluded before administration of laxatives.

Management of pain

Nociceptive pain is amenable to physiotherapy and simple analgesia.[88]

Neuropathic pain is difficult to treat.[89] [90] First-line agents are neurostabilising anticonvulsants (e.g., gabapentin, pregabalin), serotonin-noradrenaline reuptake inhibitors (e.g., duloxetine), and tricyclic antidepressants such as amitriptyline.[91] [92] [93] Opioid analgesics such as tramadol or oxycodone may be considered as a last resort once other options have been tried, but only if expected benefits outweigh risks and after a full discussion with the patient.[94] [95][96] Neuromodulation therapies for treating pain, such as transcutaneous electrical stimulation, spinal cord stimulation, and brain stimulation, have

mixed outcomes.[97] [98] [99] Patients with nerve root compression should be considered for surgical decompression.

The efficacy of psychological interventions in the treatment of chronic neuropathic pain has been insufficiently studied.

Management of spasticity

Spasticity management may involve both pharmacological agents (e.g., oral baclofen or tizanidine; some neuropathic pain medications such as gabapentin; dopaminergic drugs such as levodopa/carbidopa; chemodenervation with botulinum toxin or phenol/alcohol; intrathecal baclofen) and surgical procedures (orthopaedic contracture release; nerve transfers; dorsal rhizotomy).[49] Evidence for the effectiveness of non-pharmacological interventions (such as electro-neuromuscular stimulation, stretching, splinting, repetitive magnetic stimulation, transcranial magnetic stimulation, transcranial direct current stimulation, vibration therapy) is limited.[100]

Management of bone health

Bone loss starts early after paralysis onset and, while most pronounced in the first 12 months, continues for years after SCI.[101] Assessment of bone mineral density should be done as soon as the patient is medically stable after paralysis, and repeated after at least 12 months of medical therapy and then at 1- to 2-year intervals.[47]

The low bone density and secondary osteoporosis associated with SCI-related paralysis leads to a high incidence of low impact fractures, which often result in hospitalisation.[102] [103] Several commonly prescribed medications for patients with SCI (antidepressants, anticonvulsants, opioids, proton-pump inhibitors, anticoagulants) may have a negative effect on bone density.[104]

No therapeutic intervention has been shown to decrease fracture risk, but ambulation, standing, and electrical stimulation may increase bone mineral density in patients with SCI.[48] There is evidence that bisphosphonates, anti-RANKL monoclonal antibodies (e.g., denosumab), and teriparatide (parathyroid hormone analogue) can increase bone density of the spine, hip, and knee in patients with SCI.[48]

Management of autonomic dysreflexia

Autonomic dysreflexia can occur in patients with a lesion affecting T6 or higher.[105] [106] [107] It is caused by an excessive autonomic response to stimuli below the level of the lesion, such as a faecal impaction or blocked catheter.

Addressing the underlying cause is first-line treatment. Bladder distension should be excluded first. If the patient has a catheter, the tubing should be checked for blockage or kinking, and replaced if needed. If there is no catheter, but clinical signs of urinary retention, catheterisation is indicated. If there is no bladder distension, a rectal examination should be performed to check for and remove rectal faecal impaction. Pressure ulcers or ingrown toenails are rarer causes.

If symptoms persist or no cause is identified, patients should be treated with sublingual nifedipine or glyceryl trinitrate to lower their blood pressure.[105] [108] If the response remains inadequate after 2 doses, an intravenous hypotensive agent (e.g., hydralazine, diazoxide, or nitroprusside) should be given.[108] [109] [110] [111] Blood pressure should be monitored regularly, and efforts to find the underlying cause should be continued.

Management of psychological comorbidities

Patients with SCI who screen positive for a psychological or substance use disorder should be referred to a mental health professional for further assessment, and initiation of treatment if indicated. Pharmacological and/or non-pharmacological interventions should be considered, with treatment decisions based on clinical considerations and patient preference.[41]

Management of sexual dysfunction, fertility, pregnancy, and birth

Patients with sexual dysfunction should be provided with information, and offered non-pharmacological and pharmacological treatments as appropriate.[112]

Assisted fertility treatments should be offered as required.[38] [39]

Pregnancy, labour, and birth for female patients living with SCI require specialty care by a multidisciplinary team. Patients considering pregnancy should have a pre-pregnancy evaluation. Autonomic dysreflexia can mimic pre-eclampsia, and labour can trigger severe autonomic dysreflexia; neuraxial anaesthesia is preferred to reduce the risk of autonomic dysreflexia. Women with SCIs can give birth vaginally. For caesarean birth, spinal or epidural anaesthesia is preferred. Clinicians should be aware that patients with SCI may have delayed wound healing.[113]

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute (summary)		
progressive neurological deficit		
	1st	urgent assessment ± surgical decompression
with autonomic dysreflexia	plus	treatment of underlying cause
	adjunct	vasodilator
stable neurological status		
	1st	therapeutic interventions
	plus	supportive care
	plus	bladder management
	plus	customised bowel management programme
	plus	management of bone health
	adjunct	pain management
	adjunct	management of spasticity
	adjunct	management of psychological comorbidities
	adjunct	management of sexual dysfunction, fertility, pregnancy, and birth
with autonomic dysreflexia	plus	treatment of underlying cause
	adjunct	vasodilator

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute

progressive neurological deficit

progressive neurological deficit — **1st** — **urgent assessment ± surgical decompression**

» Progressive neurological deficit does not necessarily relate to the original spinal cord injury (SCI) - it may indicate cord compression by metastases, primary spinal cord tumours, extradural haematoma or abscess, or intervertebral disc prolapse, and requires investigation. If any of these conditions are diagnosed, timely surgical decompression and stabilisation of the involved spinal column is required.[15]

» Patients require reassessment of their neurological function post-surgery, and require rehabilitation if there is any residual neurological deficit.

» If progressive neurological deficit indicates secondary ischaemia or inflammation following the acute injury, there is no role for surgery.

with autonomic dysreflexia — **plus** — **treatment of underlying cause**

Treatment recommended for ALL patients in selected patient group

» Autonomic dysreflexia can occur in patients with a lesion affecting T6 or higher.[105] [106] It is caused by an excessive autonomic response to stimuli below the level of the lesion, such as a faecal impaction or blocked catheter. The abnormal response produces autonomic imbalance with sympathetic overactivity.

» Addressing the underlying cause is first-line treatment. Bladder distension should be excluded first.[105] If the patient has a catheter, the tubing should be checked for blockage or kinking, and replaced if needed. If there is no catheter but clinical signs of urinary retention, catheterisation is indicated.

» If there is no bladder distension, a rectal examination should be performed to check for and remove rectal faecal impaction.

» Other noxious stimuli such as pressure ulcers or an ingrowing toenail are rarer causes.

adjunct — **vasodilator**

Acute

Treatment recommended for SOME patients in selected patient group

Primary options

» nifedipine: consult specialist for guidance on dose

OR

» glyceryl trinitrate: consult specialist for guidance on dose

Secondary options

» hydralazine: consult specialist for guidance on dose

OR

» diazoxide: consult specialist for guidance on dose

OR

» nitroprusside: consult specialist for guidance on dose

» If symptoms persist despite treatment of the underlying cause, or no cause is identified, patients should be treated with sublingual nifedipine or glyceryl trinitrate to lower their blood pressure.[105] [108]

» If the response remains inadequate after 2 doses, an intravenous hypotensive agent (e.g., hydralazine, diazoxide, or nitroprusside) should be given.[108] [109] [110] [111]

» Blood pressure should be monitored regularly, and efforts to find the underlying cause should be continued.

stable neurological status

1st **therapeutic interventions**

» The goal for any rehabilitation programme for spinal cord injury (SCI)-related paralysis is to maximise day-to-day functioning to achieve levels similar to before the injury through compensatory and restorative approaches. Compensatory approaches involve maximising the strength and functionality of the intact, non-affected parts of the body, while restorative approaches aim to improve mobility and daily activities by optimising neuro-recovery.[51] [52] [53] [54]

Acute

» Physiotherapy is used to maintain joint range of motion (ROM) and mobility. This stimulates circulation and aims to prevent deformities, muscle and secondary joint contractures, and osteoporosis.[55] Activity-based therapies can promote neurological recovery and thus enhance sensory-motor and autonomic function.[56] [57] [58] [59] [60]

» Specific components of rehabilitation include the following.

» Mobility and transfers: techniques are taught in order to facilitate position changes for pressure relief, dressing, daily self-care activities, sleeping, and transfers to and from a wheelchair. Different sitting mechanisms, supports, and methods of rising from the supine to the upright position are explored and taught.[61]

» Wheelchair mobility: effective use of power or manual wheelchairs requires training. A variety of different options are available, and control can be customised based on the level of functioning of the patient. Chin, mouth, and hand controls are available for power wheelchairs; power-assisted technology can be used to improve ability to navigate different spaces using manual wheelchairs.[62] [63] Advanced manoeuvring techniques can be taught to enable patients to turn in tight spaces and negotiate ramps, slopes, and kerbs.

» Walking: there are multiple interventions that can be used to enable a patient with SCI-related paralysis to practise gait, but neurological status is the primary predictor of the ability of the patient to walk over ground independently. Patients with at least full ROM of the lower limbs with gravity eliminated are more likely to be able to walk with aids. Locomotor training uses interventions such as treadmill body weight-supported walking or robot-assisted walking.[64] [65] Orthoses for joint support or a reciprocating gait orthosis can be used for aiding ambulation. In patients with more severe motor involvement, standing may be possible with a tilt table, frames, upright wheelchairs, or parallel bars. Walking may be difficult if there is concomitant upper limb paralysis, lack of pelvic control, loss of proprioception, obesity, joint contracture, or spasticity.[54] [66]

» Hand function: the aim is to improve function using neuro-restorative and/or compensatory interventions.[67] [68] The compensatory rehabilitative model builds on available function using training and orthoses to maximise

Acute

dexterity. Splints hold joints in functional positions and can be adapted depending on the function required. Regular hand therapy can be performed out of the splint to maintain flexibility and full passive ROM. The tenodesis effect (passive finger flexion in response to wrist extension) can be taught to some patients to allow them to use the basic pinch, grasp, and 3-point grips. The activity-based therapy model utilises task-specific training and massed practice in order to improve upper limb function.[69] [70] Surgical reconstructions using arthrodesis, tenodesis, and tendon and nerve transfers may be considered in suitable patients.

» Exercise: a variety of exercise interventions, including passive ROM, strengthening and conditioning, functional electrical stimulation, and electrically stimulated resistance exercise, may improve arterial function and help neuro-recovery in patients with SCI.[71] [72] [73]

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

» Maintenance of good respiratory function is vital. Regular airway clearance techniques and clinical assessment and ongoing monitoring of pulmonary function are recommended to ensure adequate airway clearance.[74] A regular change in position and posture and regular deployment of assisted cough and regular breathing exercises (incentive spirometry) are useful in preventing secondary respiratory problems.[75] In patients requiring ongoing ventilatory support, non-invasive approaches are associated with fewer complications than invasive ventilation.[74] Resistive inspiratory muscle training has been found to have a positive short-term effect on inspiratory muscle function in patients with SCI who have impaired pulmonary function.[76]

» Pressure ulcers typically occur under the sacrum (lying supine), ischial tuberosities (sitting), or trochanters (lying on one side). They are prevented by good nursing, regular change in position, padding prominences, maintaining cleanliness, and regular checking of the skin. Surgical management is required in the presence of necrotic tissue.[77] See Pressure ulcer .

» Patients at increased risk of thrombosis (e.g., immobilised for bed rest, or admitted for medical illness or surgery) should be given prophylaxis to prevent venous thromboembolism

Acute

and possible pulmonary embolism.[78] Pharmacological prophylaxis should be used unless contraindicated; non-pharmacological measures (e.g., graduated compression stockings, intermittent pneumatic compression devices) may be used for patients at high risk for bleeding. See Venous thromboembolism (VTE) prophylaxis .

plus **bladder management**

Treatment recommended for ALL patients in selected patient group

» Most patients with SCI have impairments in bladder function, be it storage, evacuation, or both.[79]

» The preferred method of management is intermittent self-catheterisation. Use of an indwelling catheter with an external collection system may also be considered; however, this increases the risk of urinary contracture and infection. It is important that the method used maintains a low-pressure system, prevents bladder overdistension, and ensures complete emptying.

» Management strategies can be developed based on bladder studies (post-void ultrasound, urodynamic assessments, and micturition cysto-urethrogram).[83] Pharmacological management is aimed at optimising storage and elimination by using agents that decrease detrusor hyper-reflexia, improve bladder compliance, and address detrusor-sphincter dyssynergia (e.g., anticholinergics, beta agonists, alpha-blockers, botulinum toxin injection, and sometimes even cholinergic agents).[84] Consult a specialist for guidance on choice of therapy.

plus **customised bowel management programme**

Treatment recommended for ALL patients in selected patient group

» A bowel management programme should be developed and customised for each patient.[86] [87] [114]

» Dietary management should include consumption of a balanced diet, including fibre and stimulant foods, with fluid intake >2 litres/day.

» Regular routine: meals and bowel movements, if possible, should occur at the same time each day; the same location should be used for bowel movements.

Acute

» Physical manoeuvres include stimulation of the gastrocolic reflex by a hot dietary trigger, abdominal massage, and physical activity to promote defecation. The patient should sit on a commode or toilet if possible.

» Local triggers for defecation can be used, including digital stimulation, suppositories, or manual evacuation.

» Pharmacological treatments: stool softeners are preferred. Stimulant or osmotic laxatives are indicated only if constipation persists despite optimisation of all other components of the bowel management programme. Bowel obstruction should be excluded before administration of laxatives.

plus **management of bone health**

Treatment recommended for ALL patients in selected patient group

» Bone loss starts early after paralysis onset and, while most pronounced in the first 12 months, continues for years after SCI.[101] Assessment of bone mineral density should be done as soon as the patient is medically stable after paralysis, and should be repeated after at least 12 months of medical therapy and then at 1- to 2-year intervals.[47]

» The low bone density and secondary osteoporosis associated with SCI-related paralysis leads to a high incidence of low impact fractures, which often result in hospitalisation.[102] [103] Several commonly prescribed medications for patients with SCI (antidepressants, anticonvulsants, opioids, proton-pump inhibitors, anticoagulants) may have a negative effect on bone density.[104]

» No therapeutic intervention has been shown to decrease fracture risk, but ambulation, standing, and electrical stimulation may increase bone mineral density in patients with SCI.[48] There is evidence that bisphosphonates, anti-RANKL monoclonal antibodies (e.g., denosumab), and teriparatide (parathyroid hormone analogue) can increase bone density of the spine, hip, and knee in patients with SCI.[48] Consult a specialist for guidance on choice of therapy.

adjunct **pain management**

Treatment recommended for SOME patients in selected patient group

Primary options

Acute

» gabapentin: 300 mg orally once daily on day 1, followed by 300 mg twice daily on day 2, followed by 300 mg three times daily on day 3, then increase gradually according to response, maximum 3600 mg/day

OR

» pregabalin: 75-150 mg orally twice daily

OR

» duloxetine: 60 mg orally once daily

OR

» amitriptyline: 25-50 mg orally once daily at night

Secondary options

» oxycodone: 5-10 mg orally (immediate-release) every 4-6 hours when required; 10 mg orally (controlled-release) twice daily when required

OR

» tramadol: 50-100 mg orally (immediate-release) every 4-6 hours when required, maximum 400 mg/day

» Nociceptive pain is amenable to physiotherapy and simple analgesia.[88]

» Neuropathic pain is difficult to treat.[89] [90] First-line agents are neurostabilising anticonvulsants (e.g., gabapentin, pregabalin), serotonin-noradrenaline reuptake inhibitors (e.g., duloxetine), and tricyclic antidepressants (e.g., amitriptyline).[92] [93] Opioid analgesics such as tramadol or oxycodone may be considered as a last resort once other options have been tried, but only if expected benefits outweigh risks and after a full discussion with the patient.[94] [95] [96]

» Neuromodulation therapies for treating pain, such as transcutaneous electrical stimulation, spinal cord stimulation, and brain stimulation, have mixed outcomes.[97] [98] [99] Patients with nerve root compression should be considered for surgical decompression.

Acute

» The efficacy of psychological interventions in the treatment of chronic neuropathic pain has been insufficiently studied.

adjunct **management of spasticity**

Treatment recommended for SOME patients in selected patient group

» Spasticity management may involve both pharmacological agents (e.g., oral baclofen and tizanidine; some neuropathic pain medications such as gabapentin; dopaminergic drugs such as levodopa/carbidopa; chemodenervation with botulinum toxin or phenol/alcohol; intrathecal baclofen) and surgical procedures (orthopaedic contracture release; nerve transfer; dorsal rhizotomy).[49] Consult a specialist for guidance on choice of therapy.

» Evidence for the effectiveness of non-pharmacological interventions (such as electro-neuromuscular stimulation, stretching, splinting, repetitive magnetic stimulation, transcranial magnetic stimulation, transcranial direct current stimulation, vibration therapy) is limited.[100]

adjunct **management of psychological comorbidities**

Treatment recommended for SOME patients in selected patient group

» Patients with SCI who screen positive for a psychological or substance use disorder should be referred to a mental health professional for further assessment, and initiation of treatment if indicated. Pharmacological and/or non-pharmacological interventions should be considered, with treatment decisions based on clinical considerations and patient preference.[41]

adjunct **management of sexual dysfunction, fertility, pregnancy, and birth**

Treatment recommended for SOME patients in selected patient group

» Patients with sexual dysfunction should be provided with information, and offered non-pharmacological and pharmacological treatments as appropriate.[112]

» Assisted fertility treatments should be offered as required.[38] [39]

» Pregnancy, labour, and birth for female patients living with SCI require specialty care by a multidisciplinary team. Patients considering pregnancy should have a pre-pregnancy evaluation. Autonomic dysreflexia can mimic

Acute

pre-eclampsia, and labour can trigger severe autonomic dysreflexia; neuraxial anaesthesia is preferred to reduce the risk of autonomic dysreflexia.[38] Women with SCIs can give birth vaginally. For caesarean birth, spinal or epidural anaesthesia is preferred. Clinicians should be aware that patients with SCI may have delayed wound healing.[113]

with autonomic dysreflexia

plus **treatment of underlying cause**

Treatment recommended for ALL patients in selected patient group

» Autonomic dysreflexia can occur in patients with a lesion affecting T6 or higher.[105] [106] It is caused by an excessive autonomic response to stimuli below the level of the lesion, such as a faecal impaction or blocked catheter. The abnormal response produces autonomic imbalance with sympathetic overactivity.

» Addressing the underlying cause is first-line treatment. Bladder distension should be excluded first.[105] If the patient has a catheter, the tubing should be checked for blockage or kinking, and replaced if needed. If there is no catheter but clinical signs of urinary retention, catheterisation is indicated.

» If there is no bladder distension, a rectal examination should be performed to check for and remove rectal faecal impaction.

» Other noxious stimuli such as pressure ulcers or an ingrowing toenail are rarer causes.

adjunct **vasodilator**

Treatment recommended for SOME patients in selected patient group

Primary options

» nifedipine: consult specialist for guidance on dose

OR

» glyceryl trinitrate: consult specialist for guidance on dose

Secondary options

» hydralazine: consult specialist for guidance on dose

OR

MANAGEMENT

Acute

» diazoxide: consult specialist for guidance on dose

OR

» nitroprusside: consult specialist for guidance on dose

» If symptoms persist despite treatment of the underlying cause, or no cause is identified, patients should be treated with sublingual nifedipine or glycerin trinitrate to lower their blood pressure.[105] [108]

» If the response remains inadequate after 2 doses, an intravenous hypotensive agent (e.g., hydralazine, diazoxide, or nitroprusside) should be given.[108] [109] [110] [111]

» Blood pressure should be monitored regularly, and efforts to find the underlying cause should be continued.

Emerging

Neuroplasticity modulating strategies

Nervous system stimulation (central, peripheral, and mixed) utilising transcutaneous, epidural, or magnetic approaches allows for motor and non-motor neuronal activation to be used in the context of therapeutic interventions.[115] [116] [117]

Acute hypoxia

Acute intermittent hypoxia has been consistently shown to improve function in patients with incomplete motor injuries.[118]

Transplantation strategies

The transplantation of several cell types has been investigated as potential therapy for spinal cord injury (SCI); these include Schwann cells, neural stem cells or progenitor cells, olfactory ensheathing cells, oligodendrocyte precursor cells, and mesenchymal stem cells. Strategies are focused on neuroprotection, immunomodulation, and neural reconstruction.[119] [120] However, despite demonstration of safety and effectiveness in animal models, sufficient evidence of efficacy in a clinical setting is still lacking.[120]

Chondroitinase ABC

Studies in animals revealed that chondroitinase-induced plasticity improved the dexterity associated with trained skills, but had adverse effects on non-trained motor activities.[121] [122] There are no studies in humans at this time.

Electrical stimulation

Simultaneous chemical and electrical stimulation of receptors has a synergistic effect. This has led to the development of combined electrical and pharmacological strategies for improving locomotor function.[123] [124] Preliminary results from a trial of combined locomotor treadmill training, weight support, and epidural electrical stimulation showed an increase in voluntary movement among participants.[125] One systematic review concluded that functional electrical stimulation cycling exercise improves lower-body muscle health in adults with SCI, and may increase power output and aerobic fitness.[126] Cranial electrotherapy stimulation improved pain intensity and pain interference with daily life, with few side effects, in patients with SCI.[127]

Botulinum toxin

Preliminary evidence indicates that botulinum toxin type A (known as onabotulinumtoxinA, incabotulinumtoxinA, or abobotulinumtoxinA in the US) may reduce intractable chronic neuropathic pain in patients with SCI. Further study is warranted, given the difficulty of treating neuropathic pain.[128] [129] [130]

Primary prevention

In the face of an evolving compressive lesion, early diagnosis and management, including cord decompression, can prevent or reduce the severity of a chronic spinal cord injury (SCI). In acute trauma, early and aggressive resuscitation and correction of hypoxia and hypotension maintains cord vascularity. Minimal handling of the cord, meticulous surgical techniques, early cord decompression, and rigid stabilisation of the involved motion segments can prevent, or reduce the severity of, a chronic injury.[14] [15]

For traumatic SCI, prevention of injuries due to vehicle accidents, falls, violence, and sports can be achieved by combining individual and societal interventions that aim to increase research in and awareness of effective prevention initiatives and enhance education.[16] [17] Law enforcement measures (e.g., alcohol driving limits, laws requiring safety belt and helmet usage), use of safety equipment, and community programmes have also been shown to be effective.[18] [19]

Primary prevention strategies for non-traumatic myelopathies could include:

- Identifying risk factors such as congenitally narrow canals and anomalies that predispose to development of myelopathies (e.g., Down's syndrome, Klippel-Feil syndrome)[20]
- Exploring genetic and environmental factors (e.g., Epstein-Barr virus [EBV] infection, exposure to silica, cigarette smoking, oral contraceptives, and/or post-menopausal hormone therapy) for multiple sclerosis- or systemic lupus erythematosus-related myelopathy[21] [22]
- Preventing and treating nutritional deficits like vitamin D (immunomodulatory effect) in patients with autoimmune diseases or those related to inborn errors of metabolism, dietary restriction, or malabsorption (vitamin B12, folate, biotin, vitamin E, or copper)[23] [24]
- Vaccination and initiation of physical protective barriers to prevent viral or infectious myelopathies such as acute flaccid myelitis, herpesviruses-related myelopathies (cytomegalovirus, human herpes virus 6 and 7, herpes simplex virus 1 and 2, varicella zoster virus, and EBV) and maybe acquired demyelinating disorders (acute disseminated encephalomyelitis, neuromyelitis optica spectrum disorders, and myelin oligodendrocyte glycoprotein antibody-associated disease)
- Addressing hypotension during surgical procedures; minimising cardioembolic risk associated with arrhythmias, atherothrombotic disease, endocarditis, and decompression sickness; and managing hypercoagulable states (sickle cell, malignancies, positive antiphospholipid antibody) for vascular myelopathies[25]
- Minimising spine trauma (fibrocartilaginous embolism) and exposure to radiation (radiation myelopathy)
- Monitoring for signs of progressive myelopathy in metabolic storage disorders[26]
- Increasing awareness and education about the dangers of nitrous oxide inhalation that could induce subacute degeneration of the spinal cord and would help prevent the onset of possible neurological deficit.[27]

Secondary prevention

- Normal thromboprophylaxis is required for medical illness or surgery. In one study, only 38% of patients admitted to inpatient rehabilitation sites were receiving thromboprophylaxis.[153] [154] See Venous thromboembolism (VTE) prophylaxis .
- Good nursing care, regular changes in position, and padding can be used to prevent pressure ulcers if the patient is immobile for long periods. See Pressure ulcer .
- Maintenance of physiotherapy and use of stents helps to prevent contractures.

Patient discussions

Patients, families, and carers require full education on all aspects of spinal cord injury management.[149] [150] [151] [152] A written management plan should be agreed with the patient, and shared with the patient and all carers. This should include full details of management of autonomic dysreflexia, starting thromboembolic prophylaxis (if immobilised for bed rest or admitted for medical illness or surgery), pressure ulcer prevention, nutritional requirements, bowel management, bladder management, physiotherapy, and stenting.

If opioid treatment is being considered for intractable chronic pain that has not responded to non-pharmacological and non-opioid pharmacological therapies, discuss with the patient the realistic benefits and known risks of opioid therapy, work with them to establish treatment goals for pain and function, and explain that opioid therapy will be discontinued if benefits do not outweigh risks.[94]

Patients with chronic spinal cord injury are often keen to try new treatments, and guidelines are available to patients who are considering participation in clinical trials for any emerging therapy. [ICCP clinical trials information for patients] (http://icord.org/research/iccp-clinical-trials-information)

FOLLOW UP

Monitoring

Monitoring

Patients require daily assessments of calf and thigh circumference to allow early detection of deep vein thrombosis and checking of skin and pressure areas for pressure ulcers. The consistency of stools and the frequency of bowel action and interventions should be monitored regularly to allow appropriate adjustments of the bowel management plan. Respiratory and neurological function should be assessed regularly.[74]

For patients taking opioids, benefits and risks should be evaluated within 1 to 4 weeks of initiation or dosage escalation, and regularly thereafter. Clinicians should also periodically evaluate risks for opioid-related harms and discuss these with the patient.[94]

Patients should also be monitored for psychological aspects of the chronic disease, such as depression.[147] A meta-analysis has shown that cognitive behavioural therapy may have a significant positive impact on short-term psychological outcomes following spinal cord injury, but further research is needed regarding its long-term benefits.[148]

Complications

Complications	Timeframe	Likelihood
dysphagia	**short term**	**medium**
This is common in patients with lesions involving C4 to C6. Other factors that are predictive of dysphagia include age >60 years, surgical approach to treatment, severity of associated neurological deficit, and the need for a tracheostomy.[141]		
orthostatic hypotension	**short term**	**medium**
A dramatic drop in blood pressure is noted when the patient's position is changed from supine to upright sitting. It is more common in the early stages and in patients with lesions above T5 due to disruption of the autonomic pathways. This condition limits the use of upright apparatus during rehabilitation.[142] [143]		
heterotopic ossification	**short term**	**medium**
A painful condition that is very disabling when fully evolved. It occurs due to the spontaneous formation of abnormal osseous tissue in the periarticular soft tissues. This is seen commonly around the hip, knee, elbow, and shoulders. The incidence is higher in patients with complete lesions (50%). The cause is not clear, although overstretching in the acute stages has been suggested as a contributory factor. The condition presents with pain, localised redness, and joint swelling initially, and, when fully evolved, results in a complete ankylosis of the affected joint. Radiographic findings reflect the stage of the process, ranging from speckled calcification in the soft tissues to a bony bridge across the joint. Surgical excision in the fully evolved stage (1-2 years after onset) may help in the rehabilitation process.[144]		
contractures	**long term**	**high**
Muscles that have lost the innervations from the spinal cord rapidly waste. If passive mobility is not maintained aggressively, the muscles become fibrous and contract, leading to contractures across a non-functioning joint. Secondary effects on the joint capsule lead to very disabling contractures that are resistant to therapy. Early, aggressive, and regular physiotherapy, and stretching and splinting of all affected joints in a functional position, help to prevent contractures. Tizanidine can be used to manage spasticity in combination with physiotherapy.[137]		
pressure ulcers	**long term**	**high**
Insensate skin is at a high risk of breaking down when subjected to high-point loads.[138] [139] This typically occurs under a bony prominence such as the sacrum (lying supine), ischial tuberosities (sitting), or trochanters (lying on one side). The clinical presentation is typically as a red area that does not blanch, in the initial stages. Continued pressure could lead to ischaemic necrosis of the skin and the underlying tissues. The skin breakdown occurs later in the process. Other factors that contribute to the formation of pressure ulcers are spasticity, sphincteric incontinence, age, loss of sympathetic tone, poor circulation, oedema, tight garments, infection, and poor nutrition.[140] Good nursing care, regular changes in position, and padding over the affected areas could prevent further damage. Surgical treatment may be required if tissue necrosis occurs.[77]		
osteoporosis	**long term**	**medium**
Occurs due to a lack of axial loading and a range of systemic factors associated with spinal cord injury that increase bone loss. Bone loss is managed by a combination of medications and physiotherapy.[55] [145] [146]		

FOLLOW UP

Complications	Timeframe	Likelihood
deep vein thrombosis (DVT)	**variable**	**high**
May be triggered due to hypercoagulability, loss of venous muscle pump, smoking, and obesity. A regular and intensive therapy protocol ensures a good circulation. Prophylaxis is recommended to prevent DVT. Pulmonary embolism due to DVT is a leading cause of death in patients with spinal cord injuries.[136]		
dependent oedema	**variable**	**medium**
A bilateral pitting oedema in the gravitationally dependent position is noted that may resolve with a change in position. This occurs due to a combination of poor vasomotor control and a lack of normal muscle function-related venous return.		

Prognosis

The outlook for people with chronic spinal cord injury (SCI) is linked directly to the severity of the neurological injury, and associated impairment. If the neurological deficit is progressive, surgical intervention may be required to achieve damage control. This can lead to some improvement in neurological status. With established lesions, full recovery of neurological function is unlikely, and the outcome depends on the residual function and the ability of rehabilitation to maximise it.

SCI is associated with significant challenges to overall wellbeing, including a risk of mental health problems, a high risk of secondary chronic illnesses, financial insecurity, and social isolation. One review showed that poor social participation can lead to problems in re-integration into society following discharge from inpatient rehabilitation.[131] Several factors predicted poor social participation: older age when injured, medical complications, cognitive deficits, poor perceptions of control or self-efficacy, and poor social support.[131] Social support may lead to better health and functioning in people with SCI.[132] Return to work is higher in patients who were injured younger, with less severe injuries, and with higher functional independence, although employment rates are low after SCI.[133]

Patients with SCI have a decreased life expectancy compared with the general population.[134] [135]

Treatment guidelines

United Kingdom

Rehabilitation after traumatic injury (https://www.nice.org.uk/guidance/ng211)

Published by: National Institute for Health and Care Excellence **Last published:** 2022

Guidelines for management of neurogenic bowel dysfunction in individuals with a spinal cord injury and other central neurological conditions (https://www.mascip.co.uk/best-practice/mascip-best-practice)

Published by: Multidisciplinary Association of Spinal Cord Injury Professionals **Last published:** 2021

Neuropathic pain in adults: pharmacological management in non-specialist settings (https://www.nice.org.uk/guidance/cg173)

Published by: National Institute for Health and Care Excellence **Last published:** 2020

Management of chronic pain (https://www.sign.ac.uk/our-guidelines)

Published by: Scottish Intercollegiate Guidelines Network **Last published:** 2019

Guidelines for the management of neuropathic pain in adults following spinal cord injury (https://www.mascip.co.uk/wp-content/uploads/2015/02/MASCIP-Neuropathic-Pain-Management-Guidelines-v2-2008.pdf)

Published by: Multidisciplinary Association of Spinal Cord Injury Professionals **Last published:** 2008

Chronic spinal cord injury: management of patients in acute hospital settings (https://www.rcplondon.ac.uk/guidelines-policy/chronic-spinal-cord-injury)

Published by: Royal College of Physicians **Last published:** 2008

North America

Obstetric management of patients with spinal cord injuries (https://www.acog.org/clinical/clinical-guidance/committee-opinion)

Published by: American College of Obstetricians and Gynecologists **Last published:** 2020 (re-affirmed 2023)

Best practice guidelines: spine injury (https://www.facs.org/quality-programs/trauma/quality/best-practices-guidelines)

Published by: American College of Surgeons; American Congress of Rehabilitation Medicine **Last published:** 2022

CDC clinical practice guideline for prescribing opioids for pain: United States, 2022 (https://www.cdc.gov/mmwr/indrr_2022.html)

Published by: Centers for Disease Control and Prevention **Last published:** 2022

Spinal cord injury: rehabilitation practices (https://scireproject.com/evidence)

Published by: Spinal Cord Injury Research Evidence, Canada **Last published:** 2021

Management of mental health disorders, substance use disorders, and suicide in adults with spinal cord injury (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8152173)

Published by: Consortium for Spinal Cord Medicine; Paralyzed Veterans of America **Last published:** 2021

Clinical practice guidelines for spinal cord injury (https://www.pva.org/publications/clinical-practice-guidelines)

Published by: Consortium for Spinal Cord Medicine; Paralyzed Veterans of America **Last published:** 2018

Online resources

1. SCIRE: international standards for neurological classification of spinal cord injury (https://scireproject.com/outcome/ais) *(external link)*

2. NSCISC: spinal cord injury facts & figures at a glance (https://www.nscisc.uab.edu/Public/Facts%20and%20Figures%202020.pdf) *(external link)*

3. ICCP clinical trials information for patients (http://icord.org/research/iccp-clinical-trials-information) *(external link)*

REFERENCES

Key articles

- Royal College of Physicians. Chronic spinal cord injury: management of patients in acute hospital settings. Feb 2008 [internet publication]. Full text (https://www.rcplondon.ac.uk/guidelines-policy/chronic-spinal-cord-injury)
- Consortium for Spinal Cord Medicine; Paralyzed Veterans of America. Clinical practice guidelines (CPG) for spinal cord injury. 2022 [internet publication]. Full text (https://www.pva.org/publications/clinical-practice-guidelines)
- Obstetric management of patients with spinal cord injuries: ACOG Committee opinion summary, number 808. Obstet Gynecol. 2020 May;135(5):1247-49. Full text (https://www.acog.org/clinical/clinical-guidance/committee-opinion/articles/2020/05/obstetric-management-of-patients-with-spinal-cord-injuries) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32332412?tool=bestpractice.bmj.com)

References

1. Royal College of Physicians. Chronic spinal cord injury: management of patients in acute hospital settings. Feb 2008 [internet publication]. Full text (https://www.rcplondon.ac.uk/guidelines-policy/chronic-spinal-cord-injury)
2. National Spinal Cord Injury Statistical Center. Spinal cord injury model system. 2022 [internet publication]. Full text (https://www.nscisc.uab.edu)
3. New PW, Biering-Sørensen F. Review of the history of non-traumatic spinal cord dysfunction. Top Spinal Cord Inj Rehabil. 2017 Fall;23(4):285-98. Full text (https://www.doi.org/10.1310/sci2304-285) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29339905?tool=bestpractice.bmj.com)
4. Pang D, Wilberger JE Jr. Spinal cord injury without radiographic abnormalities in children. J Neurosurg. 2010 Dec;28(6):545-52. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/7086488?tool=bestpractice.bmj.com)
5. Kang JD, Figgie MP, Bohlman HH. Sagittal measurements of the cervical spine in subaxial fractures and dislocations. An analysis of two hundred and eighty-eight patients with and without neurological deficits. J Bone Joint Surg Am. 1994 Nov;76(11):1617-28. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/7962021?tool=bestpractice.bmj.com)
6. Steeves JD, Jordan LM. Localization of a descending pathway in the spinal cord which is necessary for controlled treadmill locomotion. Neurosci Lett. 1980 Dec;20(3):283-8. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/7443078?tool=bestpractice.bmj.com)
7. Lawrence DG, Kuypers HG. The functional organization of the motor system in the monkey. I. The effects of bilateral pyramidal lesions. Brain. 1968 Mar;91(1):1-14. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/4966862?tool=bestpractice.bmj.com)

8. Hayashi N, Green BA, Gonzalez-Carvajal M, et al. Local blood flow, oxygen tension, and oxygen consumption in the rat spinal cord. Part 2: relation to segmental level. J Neurosurg. 1983 Apr;58(4):526-30. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/6827348?tool=bestpractice.bmj.com)

9. Kobrine AI, Doyle TF, Rizzoli HV. Spinal cord blood flow as affected by changes in systemic arterial blood pressure. J Neurosurg. 1976 Jan;44(1):12-5. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/811767?tool=bestpractice.bmj.com)

10. Everaert K, de Waard WI, Van Hoof T, et al. Neuroanatomy and neurophysiology related to sexual dysfunction in male neurogenic patients with lesions to the spinal cord or peripheral nerves. Spinal Cord. 2010 Mar;48(3):182-91. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/20048757?tool=bestpractice.bmj.com)

11. Bughi S, Shaw SJ, Mahmood G, et al. Amenorrhea, pregnancy, and pregnancy outcomes in women following spinal cord injury: a retrospective cross-sectional study. Endocr Pract. May-Jun 2008;14(4):437-41. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18558596?tool=bestpractice.bmj.com)

12. Jackson AB, Wadley V. A multicenter study of women's self-reported reproductive health after spinal cord injury. Arch Phys Med Rehabil. 1999 Nov;80(11):1420-8. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/10569436?tool=bestpractice.bmj.com)

13. Ditunno JF Jr, Formal CS. Chronic spinal cord injury. N Engl J Med. 1994 Feb 24;330(8):550-6 Abstract (http://www.ncbi.nlm.nih.gov/pubmed/8302323?tool=bestpractice.bmj.com)

14. Morawietz C, Moffat F. Effects of locomotor training after incomplete spinal cord injury: a systematic review. Arch Phys Med Rehabil. 2013 Nov;94(11):2297-308. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23850614?tool=bestpractice.bmj.com)

15. Fehlings MG, Vaccaro A, Wilson JR, et al. Early versus delayed decompression for traumatic cervical spinal cord injury: results of the Surgical Timing in Acute Spinal Cord Injury Study (STASCIS). PLoS One. 2012 Feb 23;7(2):e32037 Full text (https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0032037) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22384132?tool=bestpractice.bmj.com)

16. Orton E, Whitehead J, Mhizha-Murira J, et al. School-based education programmes for the prevention of unintentional injuries in children and young people. Cochrane Database Syst Rev. 2016 Dec 27;12:CD010246. Full text (https://www.doi.org/10.1002/14651858.CD010246.pub2) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28026877?tool=bestpractice.bmj.com)

17. Tupetz A, Friedman K, Zhao D, et al. Prevention of childhood unintentional injuries in low- and middle-income countries: a systematic review. PLoS One. 2020;15(12):e0243464. Full text (https://www.doi.org/10.1371/journal.pone.0243464) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/33373371?tool=bestpractice.bmj.com)

18. Foroutan A, Heydari ST, Karvar M, et al. Results of a campaign for motorcycle helmets advocacy in a city in southwest of Iran; a population-based intervention study. Bull Emerg Trauma.

2019 Oct;7(4):404-10. Full text (https://www.doi.org/10.29252/beat-070410) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31858004?tool=bestpractice.bmj.com)

19. Zimmerman K, Jinadasa D, Maegga B, et al. Road traffic injury on rural roads in Tanzania: measuring the effectiveness of a road safety program. Traffic Inj Prev. 2015;16(5):456-60. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25356935?tool=bestpractice.bmj.com)

20. Bajwa NS, Toy JO, Young EY, et al. Establishment of parameters for congenital stenosis of the cervical spine: an anatomic descriptive analysis of 1,066 cadaveric specimens. Eur Spine J. 2012 Dec;21(12):2467-74. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22829425?tool=bestpractice.bmj.com)

21. Ascherio A, Munger KL, Lünemann JD. The initiation and prevention of multiple sclerosis. Nat Rev Neurol. 2012 Nov 5;8(11):602-12. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23045241?tool=bestpractice.bmj.com)

22. Barbhaiya M, Costenbader KH. Environmental exposures and the development of systemic lupus erythematosus. Curr Opin Rheumatol. 2016 Sep;28(5):497-505. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/27428889?tool=bestpractice.bmj.com)

23. Arnson Y, Amital H, Shoenfeld Y. Vitamin D and autoimmunity: new aetiological and therapeutic considerations. Ann Rheum Dis. 2007 Sep;66(9):1137-42. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/17557889?tool=bestpractice.bmj.com)

24. Canda E, Kalkan Uçar S, Çoker M. Biotinidase deficiency: prevalence, impact and management strategies. Pediatric Health Med Ther. 2020;11:127-33. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7211084) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32440248?tool=bestpractice.bmj.com)

25. McEntire CR, Dowd RS, Orru' E, et al. Acute myelopathy: vascular and infectious diseases. Neurol Clin. 2021 May;39(2):489-512. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/33896530?tool=bestpractice.bmj.com)

26. Khan SA, Tomatsu SC. Mucolipidoses overview: past, present, and future. Int J Mol Sci. 2020 Sep 17;21(18):6812. Full text (https://www.doi.org/10.3390/ijms21186812) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32957425?tool=bestpractice.bmj.com)

27. Keddie S, Adams A, Kelso ARC, et al. No laughing matter: subacute degeneration of the spinal cord due to nitrous oxide inhalation. J Neurol. 2018 May;265(5):1089-95. Full text (https://www.doi.org/10.1007/s00415-018-8801-3) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29502317?tool=bestpractice.bmj.com)

28. Critchley E, Eisen A. Spinal cord disease: basic science, diagnosis and management. London: Springer Verlag London; 1997.

29. Snell RS. Clinical neuroanatomy. 7th ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2009.

30. ASIA and ISCoS International Standards Committee. The 2019 revision of the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI)-What's new? Spinal Cord. 2019 Oct;57(10):815-17. Full text (https://www.doi.org/10.1038/s41393-019-0350-9) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31530900?tool=bestpractice.bmj.com)

31. Hamid R, Averbeck MA, Chiang H, et al. Epidemiology and pathophysiology of neurogenic bladder after spinal cord injury. World J Urol. 2018 Oct;36(10):1517-27. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29752515?tool=bestpractice.bmj.com)

32. Rabadi MH, Aston C. Complications and urologic risks of neurogenic bladder in veterans with traumatic spinal cord injury. Spinal Cord. 2015 Mar;53(3):200-3. Full text (https://www.doi.org/10.1038/sc.2014.205) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25403501?tool=bestpractice.bmj.com)

33. de Miguel M, Kraychete DC. Pain in patients with spinal cord injury: a review. Rev Bras Anestesiol. 2009 May-Jun;59(3):350-7. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19488549?tool=bestpractice.bmj.com)

34. Savic G, DeVivo MJ, Frankel HL, et al. Causes of death after traumatic spinal cord injury-a 70-year British study. Spinal Cord. 2017 Oct;55(10):891-7. Full text (https://www.doi.org/10.1038/sc.2017.64) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28631749?tool=bestpractice.bmj.com)

35. Haider IT, Lobos SM, Simonian N, et al. Bone fragility after spinal cord injury: reductions in stiffness and bone mineral at the distal femur and proximal tibia as a function of time. Osteoporos Int. 2018 Dec;29(12):2703-15. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30334093?tool=bestpractice.bmj.com)

36. Shams R, Drasites KP, Zaman V, et al. The pathophysiology of osteoporosis after spinal cord injury. Int J Mol Sci. 2021 Mar 17;22(6):3057. Full text (https://www.doi.org/10.3390/ijms22063057) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/33802713?tool=bestpractice.bmj.com)

37. Ibrahim E, Lynne CM, Brackett NL. Male fertility following spinal cord injury: an update. Andrology. 2016 Jan;4(1):13-26. Full text (https://www.doi.org/10.1111/andr.12119) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26536656?tool=bestpractice.bmj.com)

38. Stoffel JT, Van der Aa F, Wittmann D, et al. Fertility and sexuality in the spinal cord injury patient. World J Urol. 2018 Oct;36(10):1577-85. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29948051?tool=bestpractice.bmj.com)

39. DeForge D, Blackmer J, Garritty C, et al. Fertility following spinal cord injury: a systematic review. Spinal Cord. 2005 Dec;43(12):693-703. Full text (https://www.doi.org/10.1038/sj.sc.3101769) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/15951744?tool=bestpractice.bmj.com)

40. Cao Y, Massaro JF, Krause JS, et al. Suicide mortality after spinal cord injury in the United States: injury cohorts analysis. Arch Phys Med Rehabil. 2014 Feb;95(2):230-5. Full text (https://www.doi.org/10.1016/j.apmr.2013.10.007) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24161272?tool=bestpractice.bmj.com)

41. Bombardier CH, Azuero CB, Fann JR, et al. Management of mental health disorders, substance use disorders, and suicide in adults with spinal cord injury: clinical practice guideline for

healthcare providers. Top Spinal Cord Inj Rehabil. Spring 2021;27(2):152-224. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8152173) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/34108836?tool=bestpractice.bmj.com)

42. Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. Phys Ther. 1987 Feb;67(2):206-7. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/3809245?tool=bestpractice.bmj.com)

43. Cadotte DW, Wilson JR, Mikulis D, et al. Conventional MRI as a diagnostic and prognostic tool in spinal cord injury: a systemic review of its application to date and an overview on emerging MRI methods. Expert Opin Med Diagn. 2011 Mar;5(2):121-33. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23480586?tool=bestpractice.bmj.com)

44. Cameron AP, Rodriguez GM, Schomer KG. Systematic review of urological followup after spinal cord injury. J Urol. 2012 Feb;187(2):391-7. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22177149?tool=bestpractice.bmj.com)

45. Kreydin E, Welk B, Chung D, et al. Surveillance and management of urologic complications after spinal cord injury. World J Urol. 2018 Oct;36(10):1545-53. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29845320?tool=bestpractice.bmj.com)

46. Erol B, Kocak T, Kadioglu A, et al. The relationship between level of injury and bladder behavior in patients with post-traumatic spinal cord injury [in Turkish]. Ulus Travma Acil Cerrahi Derg. 2009 Jul;15(4):377-82. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19669969?tool=bestpractice.bmj.com)

47. Morse LR, Biering-Soerensen F, Carbone LD, et al. Bone mineral density testing in spinal cord injury: 2019 ISCD official position. J Clin Densitom. 2019 Oct-Dec;22(4):554-66. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31501005?tool=bestpractice.bmj.com)

48. Sadowsky CL, Mingioni N, Zinski J. A primary care provider's guide to bone health in spinal cord-related paralysis. Top Spinal Cord Inj Rehabil. 2020 Spring;26(2):128-33. Full text (https://www.doi.org/10.46292/sci2602-128) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32760192?tool=bestpractice.bmj.com)

49. Ong B, Wilson JR, Henzel MK. Management of the patient with chronic spinal cord injury. Med Clin North Am. 2020 Mar;104(2):263-78. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32035568?tool=bestpractice.bmj.com)

50. Hamid S, Hayek R. Role of electrical stimulation for rehabilitation and regeneration after spinal cord injury: an overview. Eur Spine J. 2008 Sep;17(9):1256-69. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18677518?tool=bestpractice.bmj.com)

51. Dolbow DR, Gorgey AS, Recio AC, et al. Activity-based restorative therapies after spinal cord injury: inter-institutional conceptions and perceptions. Aging Dis. 2015 Aug;6(4):254-61. Full text (https://www.doi.org/10.14336/AD.2014.1105) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26236547?tool=bestpractice.bmj.com)

52. Harvey L. Management of spinal cord injuries: a guide for physiotherapists. Philadelphia, PA: Churchill Livingstone; 2008.

53. Devillard X, Rimaud D, Roche F, et al. Effects of training programs for spinal cord injury. Ann Readapt Med Phys. 2007 Jul;50(6):490-8, 480-9. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/17482709?tool=bestpractice.bmj.com)

54. Lam T, Eng JJ, Wolfe DL, et al; SCIRE Research Team. A systematic review of the efficacy of gait rehabilitation strategies for spinal cord injury. Top Spinal Cord Inj Rehabil. 2007 Summer;13(1):32-57. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3423445) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22915835?tool=bestpractice.bmj.com)

55. Charmetant C, Phaner V, Condemine A, et al. Diagnosis and treatment of osteoporosis in spinal cord injury patients: a literature review. Ann Phys Rehabil Med. 2010 Dec;53(10):655-68. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21094110?tool=bestpractice.bmj.com)

56. Jones ML, Evans N, Tefertiller C, et al. Activity-based therapy for recovery of walking in individuals with chronic spinal cord injury: results from a randomized clinical trial. Arch Phys Med Rehabil. 2014 Dec;95(12):2239-46.e2. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25102384?tool=bestpractice.bmj.com)

57. Sadowsky CL, Hammond ER, Strohl AB, et al. Lower extremity functional electrical stimulation cycling promotes physical and functional recovery in chronic spinal cord injury. J Spinal Cord Med. 2013 Nov;36(6):623-31. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24094120?tool=bestpractice.bmj.com)

58. Behrman AL, Ardolino EM, Harkema SJ. Activity-based therapy: from basic science to clinical application for recovery after spinal cord injury. J Neurol Phys Ther. 2017 Jul;41(3 suppl):S39-45. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28628595?tool=bestpractice.bmj.com)

59. Quel de Oliveira C, Refshauge K, Middleton J, et al. Effects of activity-based therapy interventions on mobility, independence, and quality of life for people with spinal cord injuries: a systematic review and meta-analysis. J Neurotrauma. 2017 May 1;34(9):1726-43. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/27809702?tool=bestpractice.bmj.com)

60. Duan R, Qu M, Yuan Y, et al. Clinical benefit of rehabilitation training in spinal cord injury: a systematic review and meta-analysis. Spine (Phila Pa 1976). 2021 Mar 15;46(6):E398-410. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/33620185?tool=bestpractice.bmj.com)

61. Boswell-Ruys CL, Harvey LA, Barker JJ, et al. Training unsupported sitting in people with chronic spinal cord injuries: a randomized controlled trial. Spinal Cord. 2010 Feb;48(2):138-43. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19597520?tool=bestpractice.bmj.com)

62. Flockhart EW, Miller WC, Campbell JA, et al. Evaluation of two power assist systems for manual wheelchairs for usability, performance and mobility: a pilot study. Disabil Rehabil Assist Technol. 2021 Nov 22:1-13. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/34807781?tool=bestpractice.bmj.com)

63. Kloosterman MG, Snoek GJ, van der Woude LH, et al. A systematic review on the pros and cons of using a pushrim-activated power-assisted wheelchair. Clin Rehabil. 2013 Apr;27(4):299-313. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22952307?tool=bestpractice.bmj.com)

64. Mehrholz J, Harvey LA, Thomas S, et al. Is body-weight-supported treadmill training or robotic-assisted gait training superior to overground gait training and other forms of physiotherapy in people with spinal cord injury? A systematic review. Spinal Cord. 2017 Aug;55(8):722-9. Full text (https://www.doi.org/10.1038/sc.2017.31) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28398300?tool=bestpractice.bmj.com)

65. Holanda LJ, Silva PMM, Amorim TC, et al. Robotic assisted gait as a tool for rehabilitation of individuals with spinal cord injury: a systematic review. J Neuroeng Rehabil. 2017 Dec 4;14(1):126. Full text (https://www.doi.org/10.1186/s12984-017-0338-7) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29202845?tool=bestpractice.bmj.com)

66. Musselman KE, Fouad K, Misiaszek JE, et al. Training of walking skills overground and on the treadmill: case series on individuals with incomplete spinal cord injury. Phys Ther. 2010 Feb;48(2):138-43. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19423643?tool=bestpractice.bmj.com)

67. Inanici F, Brighton LN, Samejima S, et al. Transcutaneous spinal cord stimulation restores hand and arm function after spinal cord injury. IEEE Trans Neural Syst Rehabil Eng. 2021;29:310-9. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/33400652?tool=bestpractice.bmj.com)

68. Lu X, Battistuzzo CR, Zoghi M, et al. Effects of training on upper limb function after cervical spinal cord injury: a systematic review. Clin Rehabil. 2015 Jan;29(1):3-13. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25575932?tool=bestpractice.bmj.com)

69. Zoghi M, Galea M. Brain motor control assessment post early intensive hand rehabilitation after spinal cord injury. Top Spinal Cord Inj Rehabil. 2018 Spring;24(2):157-66. Full text (https://www.doi.org/10.1310/sci17-00008) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29706760?tool=bestpractice.bmj.com)

70. Beekhuizen KS, Field-Fote EC. Massed practice versus massed practice with stimulation: effects on upper extremity function and cortical plasticity in individuals with incomplete cervical spinal cord injury. Neurorehabil Neural Repair. 2005 Mar;19(1):33-45. Full text (https://www.doi.org/10.1177/1545968305274517) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/15673842?tool=bestpractice.bmj.com)

71. Phillips AA, Cote AT, Warburton DE. A systematic review of exercise as a therapeutic intervention to improve arterial function in persons living with spinal cord injury. Spinal Cord. 2011 Jun;49(6):702-14. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21339761?tool=bestpractice.bmj.com)

72. Martin Ginis KA, van der Scheer JW, Latimer-Cheung AE, et al. Evidence-based scientific exercise guidelines for adults with spinal cord injury: an update and a new guideline. Spinal Cord. 2018 Apr;56(4):308-21. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29070812?tool=bestpractice.bmj.com)

73. Sandrow-Feinberg HR, Houlé JD. Exercise after spinal cord injury as an agent for neuroprotection, regeneration and rehabilitation. Brain Res. 2015 Sep 4;1619:12-21. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25866284?tool=bestpractice.bmj.com)

74. McKim DA, Road J, Avendano M, et al. Home mechanical ventilation: a Canadian Thoracic Society clinical practice guideline. Can Respir J. 2011 Jul-Aug;18(4):197-215. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3205101) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22059178?tool=bestpractice.bmj.com)

75. Michael SM, Porter D, Pountney TE. Tilted seat position for non-ambulant individuals with neurological and neuromuscular impairment: a systematic review. Clin Rehabil. 2007 Dec;21(12):1063-74. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2630001) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18042602?tool=bestpractice.bmj.com)

76. Postma K, Haisma JA, Hopman MT, et al. Resistive inspiratory muscle training in people with spinal cord injury during inpatient rehabilitation: a randomized controlled trial. Phys Ther. 2014 Dec;94(12):1709-19. Full text (https://academic.oup.com/ptj/article/94/12/1709/2741898/Resistive-Inspiratory-Muscle-Training-in-People) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25082923?tool=bestpractice.bmj.com)

77. Srivastava A, Gupta A, Taly AB, et al. Surgical management of pressure ulcers during inpatient neurologic rehabilitation: outcomes for patients with spinal cord disease. J Spinal Cord Med. 2009 Apr;32(2):125-31. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2678283) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19569459?tool=bestpractice.bmj.com)

78. Kahn SR, Lim W, Dunn AS, et al. Prevention of VTE in nonsurgical patients: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians Evidence-based clinical practice guidelines. Chest. 2012 Feb;141(2 suppl):e195S-226S. Full text (https://www.doi.org/10.1378/chest.11-2296) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22315261?tool=bestpractice.bmj.com)

79. Consortium for Spinal Cord Medicine. Bladder management for adults with spinal cord injury: a clinical practice guideline for health-care providers. J Spinal Cord Med. 2006;29(5):527-73. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1949036) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/17274492?tool=bestpractice.bmj.com)

80. de Groat WC, Griffiths D, Yoshimura N. Neural control of the lower urinary tract. Compr Physiol. 2015 Jan;5(1):327-96. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25589273?tool=bestpractice.bmj.com)

81. Consortium for Spinal Cord Medicine; Paralyzed Veterans of America. Clinical practice guidelines (CPG) for spinal cord injury. 2022 [internet publication]. Full text (https://www.pva.org/publications/clinical-practice-guidelines)

82. Li L, Ye W, Ruan H, et al. Impact of hydrophilic catheters on urinary tract infections in people with spinal cord injury: systematic review and meta-analysis of randomized controlled trials. Arch Phys Med Rehabil. 2013 Apr;94(4):782-7. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23168400?tool=bestpractice.bmj.com)

83. Holston D, Stroope J, Cater M, et al. Implementing policy, systems, and environmental change through community coalitions and extension partnerships to address obesity in rural Louisiana. Prev Chronic Dis. 2020 Feb 27;17:E18. Full text (https://www.doi.org/10.5888/pcd17.190284) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32105589?tool=bestpractice.bmj.com)

84. del Popolo G, Mencarini M, Nelli F, et al. Controversy over the pharmacological treatments of storage symptoms in spinal cord injury patients: a literature overview. Spinal Cord. 2012 Jan;50(1):8-13. Full text (https://www.doi.org/10.1038/sc.2011.110) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22042300?tool=bestpractice.bmj.com)

85. Krassioukov A, Eng JJ, Claxton G, et al. Neurogenic bowel management after spinal cord injury: a systematic review of the evidence. Spinal Cord. 2010 Oct;48(10):718-33. Full text (https://www.nature.com/sc/journal/v48/n10/full/sc201014a.html) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/20212501?tool=bestpractice.bmj.com)

86. Emmanuel A. Neurogenic bowel dysfunction. 2019 Oct 28;8:F1000 Faculty Rev-1800. Full text (https://www.doi.org/10.12688/f1000research.20529.1) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31700610?tool=bestpractice.bmj.com)

87. Johns J, Krogh K, Rodriguez GM, et al. Management of neurogenic bowel dysfunction in adults after spinal cord injury: clinical practice guideline for health care providers. Top Spinal Cord Inj Rehabil. Spring 2021;27(2):75-151. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/34108835?tool=bestpractice.bmj.com)

88. Scottish Intercollegiate Guidelines Network. Management of chronic pain: a national clinical guideline. Aug 2019 [internet publication]. Full text (https://www.sign.ac.uk/media/1108/sign136_2019.pdf)

89. Wyndaele JJ. Pain in individuals who suffered a spinal cord injury. How does pain interfere? Hope for improved management. Spinal Cord. 2009 May;47(5):351. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19421196?tool=bestpractice.bmj.com)

90. Mehta S, McIntyre A, Janzen S, et al; Spinal Cord Injury Rehabilitation Evidence Team. Systematic review of pharmacologic treatments of pain after spinal cord injury: an update. Arch Phys Med Rehabil. 2016 Aug;97(8):1381-91. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26797114?tool=bestpractice.bmj.com)

91. National Institute for Health and Care Excellence. Neuropathic pain in adults: pharmacological management in non-specialist settings. Jul 2019 [internet publication]. Full text (https://www.nice.org.uk/guidance/cg173)

92. Mehta S, McIntyre A, Dijkers M, et al. Gabapentinoids are effective in decreasing neuropathic pain and other secondary outcomes after spinal cord injury: a meta-analysis. Arch Phys Med Rehabil. 2014 Nov;95(11):2180-6. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24992021?tool=bestpractice.bmj.com)

93. Moore RA, Derry S, Aldington D, et al. Amitriptyline for neuropathic pain in adults. Cochrane Database Syst Rev. 2015 Jul 6;2015(7):CD008242. Full text (https://onlinelibrary.wiley.com/

doi/10.1002/14651858.CD008242.pub3/full) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26146793?tool=bestpractice.bmj.com)

94. Dowell D, Ragan KR, Jones CM, et al. CDC clinical practice guideline for prescribing opioids for pain - United States, 2022. MMWR Recomm Rep. 2022 Nov 4;71(3):1-95. Full text (https://www.cdc.gov/mmwr/volumes/71/rr/rr7103a1.htm) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/36327391?tool=bestpractice.bmj.com)

95. Mehta S, Janzen, S, Loh E, et al. Poster 96 Opioid treatment for chronic pain following spinal cord injury: a systematic review. Arch Phys Med Rehab. 2012 Oct 1;93(10):e42. Full text (https://www.archives-pmr.org/article/S0003-9993%2812%2900728-9/fulltext)

96. Rekand T, Hagen EM, Grønning M. Chronic pain following spinal cord injury [in Norwegian]. Tidsskr Nor Laegeforen. 2012 Apr 30;132(8):974-9. Full text (https://www.doi.org/10.4045/tidsskr.11.0794) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22562333?tool=bestpractice.bmj.com)

97. Cruccu G, Garcia-Larrea L, Hansson P, et al. EAN guidelines on central neurostimulation therapy in chronic pain conditions. Eur J Neurol. 2016 Oct;23(10):1489-99. Full text (https://www.doi.org/10.1111/ene.13103) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/27511815?tool=bestpractice.bmj.com)

98. O'Connell NE, Marston L, Spencer S, et al. Non-invasive brain stimulation techniques for chronic pain. Cochrane Database Syst Rev. 2018 Mar 16;3:CD008208. Full text (https://www.doi.org/10.1002/14651858.CD008208.pub4) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29547226?tool=bestpractice.bmj.com)

99. Knotkova H, Hamani C, Sivanesan E, et al. Neuromodulation for chronic pain. Lancet. 2021 May 29;397(10289):2111-24. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/34062145?tool=bestpractice.bmj.com)

100. Khan F, Amatya B, Bensmail D, et al. Non-pharmacological interventions for spasticity in adults: an overview of systematic reviews. Ann Phys Rehabil Med. 2019 Jul;62(4):265-73. Full text (https://www.doi.org/10.1016/j.rehab.2017.10.001) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29042299?tool=bestpractice.bmj.com)

101. Bauman WA, Cardozo CP. Osteoporosis in individuals with spinal cord injury. PM R. 2015 Feb;7(2):188-201; quiz 201. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25171878?tool=bestpractice.bmj.com)

102. Gifre L, Vidal J, Carrasco J, et al. Incidence of skeletal fractures after traumatic spinal cord injury: a 10-year follow-up study. Clin Rehabil. 2014 Apr;28(4):361-9. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24096543?tool=bestpractice.bmj.com)

103. Morse LR, Battaglino RA, Stolzmann KL, et al. Osteoporotic fractures and hospitalization risk in chronic spinal cord injury. Osteoporos Int. 2009 Mar;20(3):385-92. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18581033?tool=bestpractice.bmj.com)

104. Kokorelis C, Gonzalez-Fernandez M, Morgan M, et al. Effects of drugs on bone metabolism in a cohort of individuals with traumatic spinal cord injury. Spinal Cord Ser Cases. 2019;5:3. Full text (https://

www.doi.org/10.1038/s41394-018-0146-8) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30675387?tool=bestpractice.bmj.com)

105. Krassioukov A, Warburton DE, Teasell R, et al. A systematic review of the management of autonomic dysreflexia after spinal cord injury. Arch Phys Med Rehabil. 2009 Apr;90(4):682-95. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19345787?tool=bestpractice.bmj.com)

106. Prévinaire JG, Mathias CJ, El Masri W, et al. The isolated sympathetic spinal cord: cardiovascular and sudomotor assessment in spinal cord injury patients: a literature survey. Ann Phys Rehabil Med. 2010 Oct;53(8):520-32. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/20797928?tool=bestpractice.bmj.com)

107. Courtois F, Rodrigue X, Côté I, et al. Sexual function and autonomic dysreflexia in men with spinal cord injuries: how should we treat? Spinal Cord. 2012 Dec;50(12):869-77. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22869221?tool=bestpractice.bmj.com)

108. Eldahan KC, Rabchevsky AG. Autonomic dysreflexia after spinal cord injury: systemic pathophysiology and methods of management. Auton Neurosci. 2018 Jan;209:59-70. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28506502?tool=bestpractice.bmj.com)

109. Squair JW, Phillips AA, Harmon M, et al. Emergency management of autonomic dysreflexia with neurologic complications. CMAJ. 2016 Oct 18;188(15):1100-3. Full text (https://www.doi.org/10.1503/cmaj.151311) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/27221275?tool=bestpractice.bmj.com)

110. Braddom RL, Rocco JF. Autonomic dysreflexia. A survey of current treatment. Am J Phys Med Rehabil. 1991 Oct;70(5):234-41. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/1910647?tool=bestpractice.bmj.com)

111. Consortium for Spinal Cord Medicine. Acute management of autonomic dysreflexia: individuals with spinal cord injury presenting to health-care facilities. J Spinal Cord Med. 2002 Spring;25(1 suppl):S67-88. Full text (https://pva.org/wp-content/uploads/2021/09/cpg_autonomic-dysreflexia.pdf) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/12051242?tool=bestpractice.bmj.com)

112. Consortium for Spinal Cord Medicine. Sexuality and reproductive health in adults with spinal cord injury: a clinical practice guideline for health-care professionals. J Spinal Cord Med. 2010;33(3):281-336. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/20737805?tool=bestpractice.bmj.com)

113. Obstetric management of patients with spinal cord injuries: ACOG Committee opinion summary, number 808. Obstet Gynecol. 2020 May;135(5):1247-49. Full text (https://www.acog.org/clinical/clinical-guidance/committee-opinion/articles/2020/05/obstetric-management-of-patients-with-spinal-cord-injuries) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32332412?tool=bestpractice.bmj.com)

114. Coggrave M, Norton C, Cody JD. Management of faecal incontinence and constipation in adults with central neurological diseases. Cochrane Database Syst Rev. 2014 Jan 13;(1):CD002115. Full text (https://onlinelibrary.wiley.com/doi/10.1002/14651858.CD002115.pub5/full) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24420006?tool=bestpractice.bmj.com)

115. Rossini PM, Burke D, Chen R, et al. Non-invasive electrical and magnetic stimulation of the brain, spinal cord, roots and peripheral nerves: basic principles and procedures for routine clinical and research application. An updated report from an I.F.C.N. Committee. Clin Neurophysiol. 2015 Jun;126(6):1071-107. Full text (https://www.doi.org/10.1016/j.clinph.2015.02.001) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/25797650?tool=bestpractice.bmj.com)

116. Wagner FB, Mignardot JB, Le Goff-Mignardot CG, et al. Targeted neurotechnology restores walking in humans with spinal cord injury. Nature. 2018 Nov;563(7729):65-71. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30382197?tool=bestpractice.bmj.com)

117. Martin R. Utility and feasibility of transcutaneous spinal cord stimulation for patients with incomplete SCI in therapeutic settings: a review of topic. Front. Rehabilit. Sci. 2021;2:724003. Full text (https://www.frontiersin.org/articles/10.3389/fresc.2021.724003/full)

118. Hayes HB, Jayaraman A, Herrmann M, et al. Daily intermittent hypoxia enhances walking after chronic spinal cord injury: a randomized trial. Neurology. 2014 Jan 14;82(2):104-13. Full text (https://www.doi.org/10.1212/01.WNL.0000437416.34298.43) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/24285617?tool=bestpractice.bmj.com)

119. Assinck P, Duncan GJ, Hilton BJ, et al. Cell transplantation therapy for spinal cord injury. Nat Neurosci. 2017 Apr 25;20(5):637-47. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28440805?tool=bestpractice.bmj.com)

120. Hu XC, Lu YB, Yang YN, et al. Progress in clinical trials of cell transplantation for the treatment of spinal cord injury: how many questions remain unanswered? Neural Regen Res. 2021 Mar;16(3):405-13. Full text (https://www.doi.org/10.4103/1673-5374.293130) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/32985458?tool=bestpractice.bmj.com)

121. Garcia-Alias G, Barkhuysen S, Buckle M, et al. Chondroitinase ABC treatment opens a window of opportunity for task-specific rehabilitation. Nat Neurosci. 2009 Sep;12(9):1145-51. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19668200?tool=bestpractice.bmj.com)

122. Zhao RR, Fawcett JW. Combination treatment with chondroitinase ABC in spinal cord injury--breaking the barrier. Neurosci Bull. 2013 Aug;29(4):477-83. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23839053?tool=bestpractice.bmj.com)

123. Canadian Agency for Drugs and Technologies in Health. Functional electrical stimulation for children with spinal cord injuries or cerebral palsy: Update of review of clinical effectiveness. August 2015 [internet publication]. Full text (https://www.cadth.ca/functional-electrical-stimulation-fes-children-spinal-cord-injuries-or-cerebral-palsy-update-review)

124. Sadowsky CL, Sayenko DG. Editorial: harnessing neuroplasticity in the injured central nervous system using spinal neuromodulation. Front. Rehabilit. Sci. 2022;3:841014. Full text (https://www.frontiersin.org/articles/10.3389/fresc.2022.841014/full#B2)

125. Angeli CA, Boakye M, Morton RA, et al. Recovery of over-ground walking after chronic motor complete spinal cord injury. N Engl J Med. 2018 Sep 27;379(13):1244-50. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/30247091?tool=bestpractice.bmj.com)

126. van der Scheer JW, Goosey-Tolfrey VL, Valentino SE, et al. Functional electrical stimulation cycling exercise after spinal cord injury: a systematic review of health and fitness-related outcomes. J Neuroeng Rehabil. 2021 Jun 12;18(1):99. Full text (https://www.doi.org/10.1186/s12984-021-00882-8) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/34118958?tool=bestpractice.bmj.com)

127. Tan G, Rintala DH, Jensen MP, et al. Efficacy of cranial electrotherapy stimulation for neuropathic pain following spinal cord injury: a multi-site randomized controlled trial with a secondary 6-month open-label phase. J Spinal Cord Med. 2011;34(3):285-96. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3127367) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/21756567?tool=bestpractice.bmj.com)

128. Han ZA, Song DH, Oh HM, et al. Botulinum toxin type A for neuropathic pain in patients with spinal cord injury. Ann Neurol. 2016 Apr;79(4):569-78. Full text (https://onlinelibrary.wiley.com/doi/10.1002/ana.24605/full) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26814620?tool=bestpractice.bmj.com)

129. Chun A, Levy I, Yang A, et al. Treatment of at-level spinal cord injury pain with botulinum toxin A. Spinal Cord Ser Cases. 2019;5:77. Full text (https://www.doi.org/10.1038/s41394-019-0221-9) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/31632735?tool=bestpractice.bmj.com)

130. Ling HQ, Chen ZH, He L, et al. Comparative efficacy and safety of 11 drugs as therapies for adults with neuropathic pain after spinal cord injury: a Bayesian network analysis based on 20 randomized controlled trials. Front Neurol. 2022;13:818522. Full text (https://www.doi.org/10.3389/fneur.2022.818522) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/35386408?tool=bestpractice.bmj.com)

131. Craig A, Nicholson Perry K, Guest R, et al. Adjustment following chronic spinal cord injury: determining factors that contribute to social participation. Br J Health Psychol. 2015 Nov;20(4):807-23. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26037456?tool=bestpractice.bmj.com)

132. Müller R, Peter C, Cieza A, et al. The role of social support and social skills in people with spinal cord injury: a systematic review of the literature. Spinal Cord. 2012 Feb;50(2):94-106. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22006079?tool=bestpractice.bmj.com)

133. Lidal IB, Huynh TK, Biering-Sørensen F. Return to work following spinal cord injury: a review. Disabil Rehabil. 2007 Sep 15;29(17):1341-75. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/17729082?tool=bestpractice.bmj.com)

134. National Spinal Cord Injury Statistical Centre. The 2013 annual statistical report for the spinal cord injury model systems. 2013 [internet publication]. Full text (https://www.nscisc.uab.edu/PublicDocuments/reports/pdf/2013%20NSCISC%20Annual%20Statistical%20Report%20Complete%20Public%20Version.pdf)

135. Wilson JR, Cadotte DW, Fehlings MG. Clinical predictors of neurological outcome, functional status, and survival after traumatic spinal cord injury: a systematic review. J Neurosurg Spine. 2012 Sep;17(1 Suppl):11-26. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22985366?tool=bestpractice.bmj.com)

136. Merli GJ, Herbison GJ, Ditunno JF, et al. Deep vein thrombosis: prophylaxis in acute spinal cord injured patients. Arch Phys Med Rehabil. 1988 Sep;69(9):661-4. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/3262334?tool=bestpractice.bmj.com)

137. Kamen L, Henney HR III, Runyan JD. A practical overview of tizanidine use for spasticity secondary to multiple sclerosis, stroke, and spinal cord injury. Curr Med Res Opin. 2008 Feb;24(2):425-39. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18167175?tool=bestpractice.bmj.com)

138. Gelis A, Dupeyron A, Legros P, et al. Pressure ulcer risk factors in persons with spinal cord injury. Part 2: the chronic stage. Spinal Cord. 2009 Sep;47(9):651-61. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19350047?tool=bestpractice.bmj.com)

139. Gelis A, Dupeyron A, Legros P, et al. Pressure ulcer risk factors in persons with SCI. Part 1: acute and rehabilitation stages. Spinal Cord. 2009 Feb;47(2):99-107. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18762807?tool=bestpractice.bmj.com)

140. De Groot PC, van Kuppevelt DH, Pons C, et al. Time course of arterial vascular adaptations to inactivity and paralyses in humans. Med Sci Sports Exerc. 2003 Dec;35(12):1977-85. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/14652491?tool=bestpractice.bmj.com)

141. Seidl RO, Nusser-Müller-Busch R, Kurzweil M, et al. Dysphagia in acute tetraplegics: a retrospective study. Spinal Cord. 2010 Mar;48(3):197-201. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19668256?tool=bestpractice.bmj.com)

142. Chelvarajah R, Knight SL, Craggs MD, et al. Orthostatic hypotension following spinal cord injury: impact on the use of standing apparatus. NeuroRehabilitation. 2009;24(3):237-42. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19458431?tool=bestpractice.bmj.com)

143. Gillis DJ, Wouda M, Hjeltnes N. Non-pharmacological management of orthostatic hypotension after spinal cord injury: a critical review of the literature. Spinal Cord. 2008 Oct;46(10):652-9. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18542098?tool=bestpractice.bmj.com)

144. Teasell RW, Mehta S, Aubut JL, et al; SCIRE Research Team. A systematic review of the therapeutic interventions for heterotopic ossification after spinal cord injury. Spinal Cord. 2010 Jul;48(7):512-21. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/20048753?tool=bestpractice.bmj.com)

145. Ashe MC, Craven C, Eng JJ, et al; SCIRE Research Team. Prevention and treatment of bone loss after a spinal cord injury: a systematic review. Top Spinal Cord Inj Rehabil. Summer 2007;13(1):123-45. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3389041) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/22767990?tool=bestpractice.bmj.com)

146. Biering-Sørensen F, Hansen B, Lee BS. Non-pharmacological treatment and prevention of bone loss after spinal cord injury: a systematic review. Spinal Cord. 2009 Jul;47(7):508-18. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19172152?tool=bestpractice.bmj.com)

147. Kalpakjian CZ, Bombardier CH, Schomer K, et al. Measuring depression in persons with spinal cord injury: a systematic review. J Spinal Cord Med. 2009 Feb;32(1):6-24. Full text (https://

www.ncbi.nlm.nih.gov/pmc/articles/PMC2647502) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19264045?tool=bestpractice.bmj.com)

148. Dorstyn D, Mathias J, Denson L. Efficacy of cognitive behavior therapy for the management of psychological outcomes following spinal cord injury: a meta-analysis. J Health Psychol. 2011 Mar;16(2):374-91. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/20978150?tool=bestpractice.bmj.com)

149. Craig A, Tran Y, Middleton J. Psychological morbidity and spinal cord injury: a systematic review. Spinal Cord. 2009 Feb;47(2):108-14. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/18779835?tool=bestpractice.bmj.com)

150. Courtois F, Charvier K, Leriche A, et al. Sexual and climactic responses in men with traumatic spinal injury: a model for rehabilitation. Sexologies. 2009 Apr-Jun;18(2):79-82.

151. Chevalier Z, Kennedy P, Sherlock O. Spinal cord injury, coping and psychological adjustment: a literature review. Spinal Cord. 2009 Nov;47(11):778-82. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/19506568?tool=bestpractice.bmj.com)

152. Krause JS. Risk and protective factors for mortality after spinal cord injury. Future Neurol. 2010 Sep 21;5(5):757-64.

153. Powell M, Kirschblum S, O'Connor KC. Duplex ultrasound screening for deep vein thrombosis in spinal cord injured patients at rehabilitation admission. Arch Phys Med Rehabil. 1999 Sep;80(9):1044-6. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/10489006?tool=bestpractice.bmj.com)

154. Merli GJ, Crabbe S, Paluzzi RG, et al. Etiology, incidence, and prevention of deep vein thrombosis in acute spinal cord injury. Arch Phys Med Rehabil. 1993 Nov;74(11):1199-205. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/8239962?tool=bestpractice.bmj.com)

Images

Pathway	Direction	Origin	Cross-over	Destination	Function
Corticospinal	Descending	Motor cortex	90% as lateral CST; 10% as anterior CST	Alpha motor neurons	Skilled motor to extremities
Reticulospinal	Descending	Reticular formation	Variable levels	Alpha and gamma motor neurons	Cortical control of voluntary motor function
Rubrospinal	Descending	Red nucleus	Immediate	Alpha and gamma motor neurons	Facilitate flexors, inhibit extensors (for balance control)
Vestibulospinal	Descending	Vestibular nucleus	None	Alpha and gamma motor neurons	Facilitate extensors and inhibit flexors (for balance control)
Lateral spinothalamic	Ascending	Free nerve endings	Immediate	Posterior central gyrus	Pain, temperature
Anterior spinothalamic tract	Ascending	Free nerve endings	Variable levels	Posterior central gyrus	Light touch and pressure
Posterior column: fasciculus gracilis and cuneatus	Ascending	Meissner's corpuscles, Pacinian corpuscles, muscle spindle, tendon organs	None	Posterior central gyrus	Discriminative touch, vibration sense, proprioception

Figure 1: Neuroanatomy and functions of the spinal tracts. CST, corticospinal tracts

From personal collection of Dr Jwalant S. Mehta

Disclaimer

BMJ Best Practice is intended for licensed medical professionals. BMJ Publishing Group Ltd (BMJ) does not advocate or endorse the use of any drug or therapy contained within this publication nor does it diagnose patients. As a medical professional you retain full responsibility for the care and treatment of your patients and you should use your own clinical judgement and expertise when using this product.

This content is not intended to cover all possible diagnosis methods, treatments, follow up, drugs and any contraindications or side effects. In addition, since such standards and practices in medicine change as new data become available, you should consult a variety of sources. We strongly recommend that you independently verify specified diagnosis, treatments and follow-up and ensure it is appropriate for your patient within your region. In addition, with respect to prescription medication, you are advised to check the product information sheet accompanying each drug to verify conditions of use and identify any changes in dosage schedule or contraindications, particularly if the drug to be administered is new, infrequently used, or has a narrow therapeutic range. You must always check that drugs referenced are licensed for the specified use and at the specified doses in your region.

Information included in BMJ Best Practice is provided on an “as is” basis without any representations, conditions or warranties that it is accurate and up to date. BMJ and its licensors and licensees assume no responsibility for any aspect of treatment administered to any patients with the aid of this information. To the fullest extent permitted by law, BMJ and its licensors and licensees shall not incur any liability, including without limitation, liability for damages, arising from the content. All conditions, warranties and other terms which might otherwise be implied by the law including, without limitation, the warranties of satisfactory quality, fitness for a particular purpose, use of reasonable care and skill and non-infringement of proprietary rights are excluded.

Where BMJ Best Practice has been translated into a language other than English, BMJ does not warrant the accuracy and reliability of the translations or the content provided by third parties (including but not limited to local regulations, clinical guidelines, terminology, drug names and drug dosages). BMJ is not responsible for any errors and omissions arising from translation and adaptation or otherwise.Where BMJ Best Practice lists drug names, it does so by recommended International Nonproprietary Names (rINNs) only. It is possible that certain drug formularies might refer to the same drugs using different names.

Please note that recommended formulations and doses may differ between drug databases drug names and brands, drug formularies, or locations. A local drug formulary should always be consulted for full prescribing information.

Treatment recommendations in BMJ Best Practice are specific to patient groups. Care is advised when selecting the integrated drug formulary as some treatment recommendations are for adults only, and external links to a paediatric formulary do not necessarily advocate use in children (and vice-versa). Always check that you have selected the correct drug formulary for your patient.

Where your version of BMJ Best Practice does not integrate with a local drug formulary, you should consult a local pharmaceutical database for comprehensive drug information including contraindications, drug interactions, and alternative dosing before prescribing.

Interpretation of numbers

Regardless of the language in which the content is displayed, numerals are displayed according to the original English-language numerical separator standard. For example 4 digit numbers shall not include a comma nor a decimal point; numbers of 5 or more digits shall include commas; and numbers stated to be less than 1 shall be depicted using decimal points. See Figure 1 below for an explanatory table.

BMJ accepts no responsibility for misinterpretation of numbers which comply with this stated numerical separator standard.

This approach is in line with the guidance of the International Bureau of Weights and Measures Service.

Figure 1 – BMJ Best Practice Numeral Style

5-digit numerals: 10,000

4-digit numerals: 1000

numerals < 1: 0.25

Our full website and application terms and conditions can be found here: Website Terms and Conditions.

Contact us

+ 44 (0) 207 111 1105
support@bmj.com

BMJ
BMA House
Tavistock Square
London
WC1H 9JR
UK

Contributors:

// Authors:

Cristina Sadowsky, MD

Clinical Director
International Center for Spinal Cord Injury, Kennedy Krieger Institute, Associate Professor Physical Medicine and Rehabilitation, Johns Hopkins School of Medicine, Baltimore, MD
DISCLOSURES: CS is an author of a number of references cited in this topic.

Travis Edmiston, MD

Assistant Professor for Physical Medicine and Rehabilitation
International Center for Spinal Cord Injury, Kennedy Krieger Institute, Baltimore, MD
DISCLOSURES: TE declares that he has no competing interests.

// Acknowledgements:

Dr Cristina Sadowsky and Dr Travis Edmiston would like to gratefully acknowledge Dr Kenneth Casey and Dr Jwalant Mehta, previous contributors to this topic.
DISCLOSURES: KC and JM declare that they have no competing interests.

// Peer Reviewers:

Steven Kirshblum, MD

Medical Director and Director of Spinal Cord Injury Services
Kessler Institute for Rehabilitation, West Orange, Professor, University of Medicine and Dentistry of New Jersey/New Jersey Medical School, Newark, NJ
DISCLOSURES: SK declares that he has no competing interests.

Deborah Short, MRCP

Consultant in Rehabilitation Medicine
Robert Jones and Agnes Hunt Hospital, Oswestry, UK
DISCLOSURES: DS declares that she has no competing interests.