Pain/Spasticity

Spasticity is defined as exaggerated muscle tone with increased tendon reflexes. Spasm describes violent reflexive muscle contraction which occurs in response to cutaneous and visceral stimulation.

Associated with SCI above level of T12 which preserves spinal reflex arc

May be exacerbated by bladder, bowels, skin, pain. Identifying and managing exacerbating factor is the first goal of treatment. This followed by physical exercise (splinting, stretching incl. passive movement, standing) and then medications (Baclofen, Tizanidine, Dantrolene, Clonidine, Diazepam, Botox Injections).

Poor management leads to contractures, pressure ulcers, pain and can impact on function, care needs and quality of life.

Pain post-SCI is commonly divided into neuropathic pain and nociceptive pain.

Nocioceptive which can either be musculoskeletal pain; often described as ‘aching’ in nature and due to muscle imbalance or visceral pain for example from bladder, bowel, gallbladder.

Neuropathic pain is pain initiated or caused by a primary lesion or dysfunction of the nervous system.

Usually identified as above level of lesion, at level or below level.

Can be exacerbated by another stimulus e.g. an infection

Treatment often empirical with use of nociceptive medications (NSAIDs and opioids) or neuropathic pain medications (e.g. antidepressants and anticonvulsants).

Non-pharmacological treatments include physical therapy (e.g TENs), psychological (coping and distraction), chemical or surgical nerve ablation.

Spasticity

Definition

Spasticity is a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflexes, as one component of the upper motoneuron syndrome.
**Pathophysiology**

Recent studies indicate that, besides changes in motoneuron activation (involuntary supraspinal descending inputs and inhibited spinal reflexes etc), changes in muscle properties also contribute to the clinical appearance of limb spasticity and rigidity, which are frequently linked symptoms.

For more information visit scireproject.com/spasticity

**Signs and symptoms**

Signs of exaggerated tendon tap reflexes associated with muscle hypertonia are generally thought to be responsible for spastic movement disorders.

**Spasticity Management**

*Determining the impact of spasticity and need for treatment*

Spasticity in SCI varies with location and degree depending on the injury pathophysiology. The goal of treatment should be to reduce pain or discomfort related to spasticity, prevent the breakdown of skin and fractures, and overcome functional impairments related to spasticity.

In some cases, increased spasticity is beneficial for transfers and mobility, and the reduction of tone may negatively impact those activities of daily living. Therefore, the decision to treat “spasticity” should not only be based on the findings gained by the examination in passive (lying bed, sitting in the wheelchair) but also in active conditions (like walking, doing transfer etc). As well, spasticity can be protective against skeletal muscle atrophy that in turn could indirectly affect functional independence, ambulation and incidence of fracture. The management of spasticity is multifaceted. initial assessment should try and identify and relieve any exacerbating factors, eg constipation, UTI. If this is not sufficient non-medical interventions should be tried, including passive mobilisation and other strategies physiotherapists can advise on. If physical measures are not sufficient, medical management can be started.

*Physical Approaches to managing spasticity*

For example, passive movement, standing, splinting, use of a motorised bike.

*Active Movement-based Approaches for Reducing Spasticity*

For example hydrotherapy and FES-assisted cycling. (Electrical stimulation applied to individual muscles may produce a short-term decrease in spasticity. However, there is also some concern that long-term use of electrical stimulation may increase spasticity).

*Afferent Stimulation for Reducing Spasticity*

For example transcutaneous electrical nerve stimulation (TENS), referral to SCIC to trial Penile Vibratory Stimulation.

*Medical management of spasticity*
Oral baclofen reduces muscle spasticity in people with SCI.

Gabapentin can be effective in reducing spasticity.

Tizanidine may be useful in treating SCI spasticity, clononidine can be beneficial if Tizanidine is effective but causing side effects.

Occasionally benzodiazepines (diazepam or clonazepam) or dantrolene are recommended.

Cannabinoids are not licenced for managing spasticity after SCI.

*For some patients, more invasive management is required, by the SCIC.*

**Intrathecal Baclofen**

Bolus or long-term intrathecal baclofen decreases spasticity and may improve functional outcomes with low complication rates and is a cost-effective intervention.

*More information on [Baclofen withdrawal](#)*

**Focal Neurolysis**

Botulinum neurotoxin appears to improve focal muscle spasticity in people with SCI. For more information on managing spasticity using botulinum toxin please refer to [RCP guidelines](#)

Phenol block may improve pain, the range of motion and function related to shoulder spasticity in individuals with tetraplegia.

Phenol block may reduce hip adductor spasticity in individuals with paraplegia and tetraplegia.

**Pain**

**Definition**

Pain post SCI has a significant effect on quality of life. The most common types of pain post SCI are:

1) a burning pain (likely neuropathic) usually localized to the front of torso, buttock or legs. This can be experienced at the level of injury or below the level of injury.

Individuals with SCI frequently experience a band of pain and hyperalgesia at the border zone between diminished or abnormal and preserved sensation an aching pain (likely musculoskeletal) usually localised to neck, shoulders and back.

**Pathophysiology**

**Pain**

Musculoskeletal pain is often due to muscle imbalance and compensation of 'working' muscle groups.
Visceral pain can be from bladder, bowel, gallbladder, diaphragm.

Neuropathic pain is poorly understood and various competing theories. For more information visit SCIRE

**Signs and symptoms**

**Pain**

Pain attributable to nerve root damage is suggested by features of neuropathic pain (i.e. burning, stabbing, shooting, electric-like pain, allodynia) and increased pain with spinal movement.

Radicular pain is described as stimulus-independent, often accompanied by troublesome allodynia or hyperalgesia and thought to arise from segmental deafferentation.

Muscular pain usually 'aching' in nature, located above the level of SCI usually in the neck, shoulders and back.

**Neuropathic pain management**

**Non-pharmacological neuropathic pain management**

Acupuncture, regular exercise and hypnosis may all reduce post-SCI pain.

Cognitive behavioural therapy combined with pharmacological treatment results in short-term improvement in chronic pain.

Transcutaneous electrical nerve stimulation may reduce pain at the site of injury in patients with thoracic but not cervical injury.

Transcranial magnetic stimulation reduces post-SCI pain.

**Pharmacological pain management**

Gabapentin and pregabalin improve neuropathic pain post SCI.

Lamotrigine may improve neuropathic pain in patients with incomplete SCI.

Duloxetine may improve neuropathic pain post SCI

Amitriptyline is effective in reducing pain in depressed SCI individuals.

Tramadol reduces neuropathic pain.

Topical capsaicin reduces post-SCI radicular pain.

Lidocaine patches can improve localised neuropathic pain.

Cannabinoids are a potential new treatment for post-SCI pain in need of further study.
For some patients, more invasive management is required, by the SCIC or local pain team

Lidocaine through a subarachnoid lumbar catheter and intravenous Ketamine improve post SCI pain short term.

Intrathecal Baclofen improves musculoskeletal pain post SCI and may help dysethetic pain related to spasticity.

Motor point phenol block reduces spastic shoulder pain.

Botulinum toxin injections for treatment of focal spasticity improves pain.

Intravenous morphine reduces mechanical allodynia.

Intrathecal Clonidine may be helpful in combination with Intrathecal Morphine.

**Surgical interventions**

Spinal cord stimulation may improve post-SCI pain.

Dorsal longitudinal T-myelotomy procedures reduce pain post SCI.

DREZ surgical procedure reduces pain post SCI.

**References**

Spinal Cord Injury Rehabilitation Evidence (SCIRE) Project. Spasticity Review. scireproject.com/spasticity


© Royal National Orthopaedic Hospital Trust, Brockley Hill, Stanmore, Middlesex HA7 4LP. Tel: 020 3947 0100