



Government of **Western Australia**
Department of **Health**

Spasticity Management following Spinal Cord Injury

A Resource for Health Service Providers

WA State Spinal Injury Unit

Version 1 October 2013

Review Date October 2016

What is this document?

This document has been developed to provide health service providers with a sound understanding of the physiology and management of spasticity following spinal cord injury.

Key Points

- Spasticity is a common complication post spinal cord injury (SCI).
- Changes in spasticity may indicate alterations in other body systems or alterations in neurology and should be discussed with a spinal physician.
- Spasticity causes changes to function which can create issues with mobility, posture, upper limb function or fitting of splints
- Spasticity only requires intervention if it is bothersome to the patient, interferes with function or results in unwanted limb contractures
- Assessment is commonly completed using either Tardieu or modified Ashworth scale (these scales provide a guide to compare treatment regimes and the impact of these and also to identify the presence of contracture)
- Treatment options include :
 - Pharmacological Intervention
 - Splinting
 - Casting
 - Surgery
 - Ranging
 - Botulinum Toxin

Introduction

Spasticity is reported in 53% to 78% individuals following SCI. ¹ “Spasticity is a motor disorder that is characterized by a velocity dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex as one component of the upper motor neuron syndrome.”^{2,3} and occurs in patients with an upper motor neurone injury (above T12/L1).¹

SCI leads to a loss of descending cortico-spinal pathways which control movement and changes in motor neuron activation which create a reflex like response to stimulus such as stretch or attempted movement. This response to stimuli cannot be inhibited and thus there is a reflex contraction and a stronger excitatory effect on muscles following stretch.

Spasticity can lead to secondary changes in involved musculoskeletal tissues including contractures, as well as changes to mechanical muscle fibre, collagen tissue and tendon properties.^{1,4} Contractures can prevent full functional capacity, inhibit hygiene and result in abnormal positioning.

For further information on the anatomy and physiology of spasticity – refer to <http://elearnSCI.org> within the section for doctors regarding spasticity management. This provides general background and information about treatment options which maybe useful.

Assessment of Spasticity

It is critical to assess and regularly review the impact and severity of spasticity on the person with SCI. Not all spasticity is negative or harmful for someone with SCI, in fact it can help to maintain joint position for function, improve circulation and preserve muscle bulk.

Careful assessment includes review of:

- resistance to passive movement⁵
- limbs for signs of contractures⁵
- limb position at rest⁵
- posture during activity⁵
- weakness or paralysis in some muscle groups⁵
- muscle balance between muscle groups⁵
 - decreased co-activation
 - reduced stability and mobility of limb during active movement
- use of compensatory movement patterns to aid function⁵
- fixed contractures due to prolonged posturing and changes to joints, muscles and other soft tissue⁵

The most widely utilised assessments for spasticity are the Modified Ashworth Scale (MAS) ⁶ and the Tardieu scale⁷.

The MAS is a qualitative scale for the assessment of spasticity in particular tone. However it measures only resistance to passive movement and has been criticised for its inability to

differentiate between the reflex and peripheral components of spasticity and its inability to measure the functional effects of intervention.

Table 1-- Modified Ashworth Scale⁸

Score	Description
0	No increase in muscle tone
1	Slight increase in muscle tone manifested by a catch and release at end range of motion
1+	Slight increase in muscle tone, manifested by a catch followed by minimal resistance throughout the remainder of range of motion
2	More marked increase in tone through most of range of motion but joint easily moved
3	Considerable increase in muscle tone; passive movement is difficult
4	Affected part is rigid in flexion or extension

Table 2-- Tardieu Scale⁸

Score	Description
0	No resistance throughout the course of passive movement
1	Slight resistance throughout the course of passive movement with no clear catch at a precise angle
2	Clear catch at a precise angle, interrupting passive movement followed by release
3	Fatiguable clonus, less than 10 seconds when maintaining pressure, appearing at a precise angle
4	Nonfatiguable clonus, more than 10 seconds when maintaining pressure, at a precise angle

The Tardieu Scale compares the differences when a muscle is stretched at different velocities and comparing the angles at which the catch is noted including recording of:

- 1) strength and duration of the stretch reflex
- 2) the angle at which the stretch reflex is activated
- 3) the speed necessary to trigger the stretch reflex.

The Tardieu scale as discussed in Haugh⁷ has limited research into its reliability and validity.

A useful assessment tool has been developed by the Queensland Spinal Cord Injuries service (<http://www.health.qld.gov.au/qscis/html/spasm>) which incorporates patient goals, impact on function as well as above assessments.

Overview of spasticity management options

There is general agreement that management decisions regarding spasticity need to be based on achieving balance between the useful and detrimental effects of spasticity on an individual's quality of life. Such decisions should address both functional problems and 'passive' problems such as facilitating splint wearing, reducing pain and preventing contracture.⁴

With diffuse spasticity affecting a number of large muscles or muscle groups and impacting on an individual's function, a medical review through the State Spinal Injury Service or with a GP or Allied Health professional familiar with SCI management is required. This would facilitate optimal management including pharmacological options.

Medications for Spasticity

Pharmacologic management of spasticity is often required in spinal cord injury. Medications commonly used and available in Australia for generalised spasticity are: baclofen, benzodiazepines and dantrolene.

Baclofen binds to and activates the presynaptic GABA_B receptor (i.e. acts as an agonist) on spinal cord neurons.⁹ This alters potassium conductance resulting in net membrane hyperpolarisation and a reduction in endogenous transmitter release.^{10,11} Baclofen also activates receptors postsynaptically inhibiting calcium conductance resulting in inhibition of δ -motor neuron activity, reduced drive to intrafusal muscle fibres and reduced muscle spindle sensitivity.¹⁰ Overall, baclofen reduces sensory and motor neuron activation. The therapeutic half-life ranges from 2-6 hours and oral dosing is usually initiated at 5mg TDS.¹⁰ The maximum recommended dose of oral Baclofen is 75mg per day, but is sometimes exceeded under the guidance of a spinal rehabilitation physician.

Baclofen is also available as a liquid for delivery via a pump sited in the lower abdominal wall with a catheter to the spine and intrathecal space at approximately the T10 spinal cord level. This allows medication delivery to spinal cord neurons responsible for spasticity at a 100-fold increase in potency compared to oral dosing.¹⁰ Prior to siting a pump for baclofen, test dose/s of baclofen are given via lumbar puncture (LP), with evaluation by the physiotherapy team before and after LP administration to document effectiveness.

Side effects of baclofen include drowsiness, motor incoordination, nausea and occasionally behavioural effects.

Benzodiazepines act as agonists at the GABA_A receptor both presynaptically and postsynaptically⁽³⁾ and increase the opening frequency of the chloride channels thus increasing the affinity of GABA for the receptor.¹² This decreases polysynaptic reflexes and has muscle relaxant, sedation and antispasticity effects.¹⁰

Diazepam is a commonly used benzodiazepine for spasticity with a commencing dose of 2mg orally TDS.¹³ Diazepam can be used as a bedtime dose of 5mg increasing to 10mg as needed.(2) Clonazepam is a further option, in particular for night time spasms; it causes less sedation than diazepam and has a slightly lower risk for dependence.⁴ The usual dosage of clonazepam is 0.5-2mg 2-3 times daily.¹⁴

For spasticity resistant to baclofen and benzodiazepines, **dantrolene**, which acts peripherally at the muscle tissue, is a therapeutic option.^{4,14} The primary pharmacologic effect of dantrolene is reduction of calcium flux across the sarcoplasmic reticulum of skeletal muscle,¹⁵ uncoupling motor nerve excitation and skeletal muscle contraction. The peak blood concentration of dantrolene occurs in 3 to 6 hours post ingestion.¹⁰ The commencing dose is 25 mg once daily increasing to 25 mg two, three or four times daily and then by increments of 25 mg up to as high as 50 mg two, three or four times daily if necessary. The maximum recommended dose is 200 mg/day. Adverse effects include drowsiness, dizziness, weakness, general malaise, fatigue, and diarrhoea. The adverse effects are generally transient and occur early in treatment, and can often be obviated by commencing with a low dose with gradual dose increases until an optimal regimen is established. The use of dantrolene has been associated with hepatotoxicity and death in 1.8% and 0.3%, respectively, of users in one trial.^{10,15}

Nerve blocks or Botulinum Toxin Type A may be used to target individual muscles or muscle groups and is discussed below. Other chemodenervation injectable agents for focal spasticity can include phenol and ethanol.

Further discussion about other medications and treatment options can be found in 'Spasticity Following Spinal Cord Injury' from SCIRE¹⁶.

For spasticity in specific muscle groups for people with SCI a regular intervention programme is recommended to maintain the affected muscle in a stretched position. This may be achieved using splinting and/or daily ranging.

For individuals who have ongoing issues with spasticity, available options may include the following, based on the assessment and preference of the person with SCI in liaison with their spinal physician.

Splinting

The use of splinting in upper limbs for individuals with SCI can maintain existing range.^{17,18} This option is suitable for those individuals who identify the issues early or are proactive in considering the potential impact of their spastic muscles on passive or active range. Splints for lower limbs are not routinely utilized, however can be considered if function is deteriorating due to loss of range of movement.

Treatment benefits

- maintains existing passive range if worn in regular regime
- can be applied as care allows
- does not worsen function
- can be used with other treatment modalities ie casting if care support allows
- splints worn at night therefore not restricting daily activity

Rationale for treatment selection

- to maintain independence during the day for mobility and general activities
- limited access to care assistance to support of other options

Treatment protocol

- construction of a resting pan splint or palmar/dorsal splint in a sub maximal position at wrist (refer to Splinting Information on this website)
- Contact State Spinal Injury Service if lower limb splinting is being considered

Casting

Casting is used for patients who present with contracture. Contracture can be assessed through the techniques described above and is demonstrated where both passive and active range are restricted and there is no evidence of active spasticity.⁵ Patient compliance and care assistance is required for effective and safe use of this treatment technique.

Treatment benefits

- able to resolve contractures
- no surgical intervention required
- splinting follow-up maintains improved passive range
- to address maceration of skin (i.e. in palm) creating hygiene concerns and improved cosmesis

Rationale for treatment selection

- patient able to manage with limb being immobilised for at least 3 weeks
- requires appropriate care support
- patient able to attend regular hospital appointments
- ability to monitor for skin breakdown, i.e some sensation in UL present

Treatment protocol

- series of casts over both involved joints wrist, fingers and thumb
- wrist positioned in submaximal stretch for all casts
- oedema has previously been noted on removal of cast which resolved with passive ranging and mobilization⁵

Botulinum Toxin

Botulinum Toxin can be used in conjunction with all other techniques. It provides an opportunity to position the affected limb effectively to allow the fitting of splints or ranging of muscles without working against muscle contraction.

Botulinum Toxin is used to chemically denervate a specific muscle by blocking neurotransmitters.¹⁶ inhibits the release of acetylcholine from presynaptic motor axons. The chemical denervation develops slowly over 24-72 hours with peak effect occurring after about 2-6 weeks.⁷ The duration of clinical benefit is generally about 2-4 months.⁶

The degree and length of effectiveness of this intervention varies from person to person.

Treatment benefits

- paralyses specific muscle groups temporarily - re-inervation after 3 months on average

Rationale for treatment selection

- preserves some functional movement as treatment focuses on one specific muscle e.g. one of three elbow flexors : brachialis, brachioradialis and biceps

Treatment protocol

- injection using Functional Electrical Stimulation (FES) to identify desired muscle belly
- ongoing repeat dose may be required

Ranging

Ranging involves the movement of an affected joint within pain limits, whilst limiting other moveable parts. The effectiveness of ranging as a technique to resolve or limit the impact of spasticity has not been demonstrated in randomised control trials¹⁹ however it is supported by clinical consensus. Ranging can work effectively to increase range of movement whilst stretch is applied, but typically it has short lasting effect on the spastic muscle.

SCIRE 2012¹⁶ reports that there is some evidence to support prolonged standing as an activity to reduce lower limb spasticity.

Treatment benefits

- can maintain passive range if performed as part of regular regime
- does not impact on skin integrity
- does not increase range therefore reducing range continues to impact on function

Rationale for treatment selection

- splinting unsuccessful or not appropriate due to skin breakdown
- there is available care support to perform ranging
- the person with SCI is reluctant to trial other options

Treatment protocol

- ranging programme given to carers
- regular ranging at each joint using prolonged stretch ie 30 secs encouraged

Surgery

Surgery is usually considered by a person with SCI if pain is a major complicating factor, or all other treatment modalities have been trialled without success.²⁰

Treatment benefits

- permanent reduction of pain at affected joint
- resolution of positioning issues
- splints able to be applied easily and worn to maintain position achieved

Rationale for treatment selection

- tried everything else and ongoing issues relating to spasticity persist

Treatment protocol

- surgical follow-up would included OT provision of splints to support positioning

References

1. Burchiel, KJ and Hsu, FP 2001. Pain and Spasticity after Spinal Cord Injury, Mechanisms and Treatment. *SPINE* Vol 26 (24S) S146-160
2. Lance, JW. Symposium Synopsis. In Feldman R. et al 1990 Spasticity: disordered motor control. Chicago: Yearbook Medical
3. Nance, P et al in Braddom, RL (Ed) 2011. Physical Medicine and Rehabilitation 4th Ed. Philadelphia : Elsevier Saunders p 641- 59.
4. Adams MM and Hicks AL. 2005. Spasticity after spinal cord injury. *Spinal Cord* 43:577-586
5. Copley, J & Kuipers, K. 1999. Management of Upper Limb Hypertonicity. San Antonio. Therapy Skills Builders.
6. Richard W Bohannon and Melissa B Smith 1987 Interrater Reliability of a Modified Ashworth Scale of Muscle Spasticity *Physical Therapy* February 67:206-207
7. Haugh, AB and Pandyan, AD. Johnson GR. 2006. A systematic review of the Tardieu Scale for the measurement of spasticity. *Disability & Rehabilitation*. 28(15):899-907
8. Wagner, AK et al . 2011 Traumatic Brain Injury. In : Braddom RL (Ed) Physical Medicine and Rehabilitation. 4th Ed. Philadelphia: Elsevier Saunders p 641-59
9. Rang H, Dale M, Ritter J, Flower R. 2007 .Antiepileptic drugs. Rang and Dale's Pharmacology. 6th ed. Philadelphia: Churchill Livingstone;. p. 575-87.
10. Nance P, Satkunam L, Ethans K. 2011. Spasticity management. In: Braddom RL, editor Physical Medicine and Rehabilitation. 4th ed ed. Philadelphia: Elsevier Saunders p. 641-59.
11. Dietz V, Sinkjaer T. 2012. Spasticity. In: Verhaagen J, McDonald III JW, editors. Handbook of Clinical Neurology. Edinburgh: Elsevier; p. 197-211.
12. Rang H, Dale M, Ritter J, Flower R. 2007. Anxiolytic and hypnotic drugs. Pharmacology (Rang & Dale's). 6th ed. Philadelphia: Churchill Livingstone Elsevier; p. 535-44.
13. Therapeutic Guidelines Limited. 2011. Therapy directed to symptoms of multiple sclerosis/Spasticity. 4 ed. Melbourne: Therapeutic Guidelines.
14. Gelber DA, Jozefczyk PB. 1999. Therapeutics in the management of spasticity. *Neurorehabilitation and neural repair*. 13:5-14.

15. Krach LE. 2001. Pharmacotherapy of spasticity: oral medications and intrathecal baclofen. *Journal of child neurology*. 16(1):31-6. Epub 2001/02/28.
16. Spasticity Following Spinal Cord Injury. Spinal Cord Injury Rehabilitation Evidence (SCIRE) (2012) <http://www.scireproject.com/rehabilitation-evidence/spasticity>
17. Krajnik, S R & Bridle M J. 1992. Hand Splinting in Quadriplegia. Current Practice. *American Journal of Occupational Therapy* 46(2) 149-155.
18. Lannin N et al. 2003. Splinting the Hand in the Functional Position After Brain Impairment. A Randomized Control Trial. *Archives of Physical Medicine and Rehabilitation*. 84: 297-302.
19. Harvey, L A & Herbert R D. 2002. Muscle stretching for treatment and prevention of contracture in people with spinal cord injury. *Spinal Cord* 40: 1-9.
20. Van Heest, A E 2003. Surgical management of wrist and finger deformity. *Hand Clinics* 19 657-665.

Useful resources

The online educational tool from the International Spinal Cord Society contains learning modules directed at each discipline.

- www.elearnsoci.org

Spinal Injuries Unit Spasticity Assessment Form developed by the Princess Alexandra Hospital in Queensland.

- <http://www.health.qld.gov.au/qscis/html/spasm.asp>

Compiled by

Nicky Hunter, Acting Senior Occupational Therapist, State Spinal Unit, Royal Perth Hospital

Contributors

- Carly Hartshorn, Clinical Nurse Consultant Spinal Urology, State Spinal Unit, Royal Perth Hospital
- Dr Colin Crawford, Spinal Registrar, State Spinal Unit, Royal Perth Hospital
- Christine Rimmer, Senior Physiotherapist State Spinal Unit, Royal Perth Hospital

The development of this document has been supported by the
TRACS WA Subacute Learning Fund project funding



Delivering a **Healthy WA**