

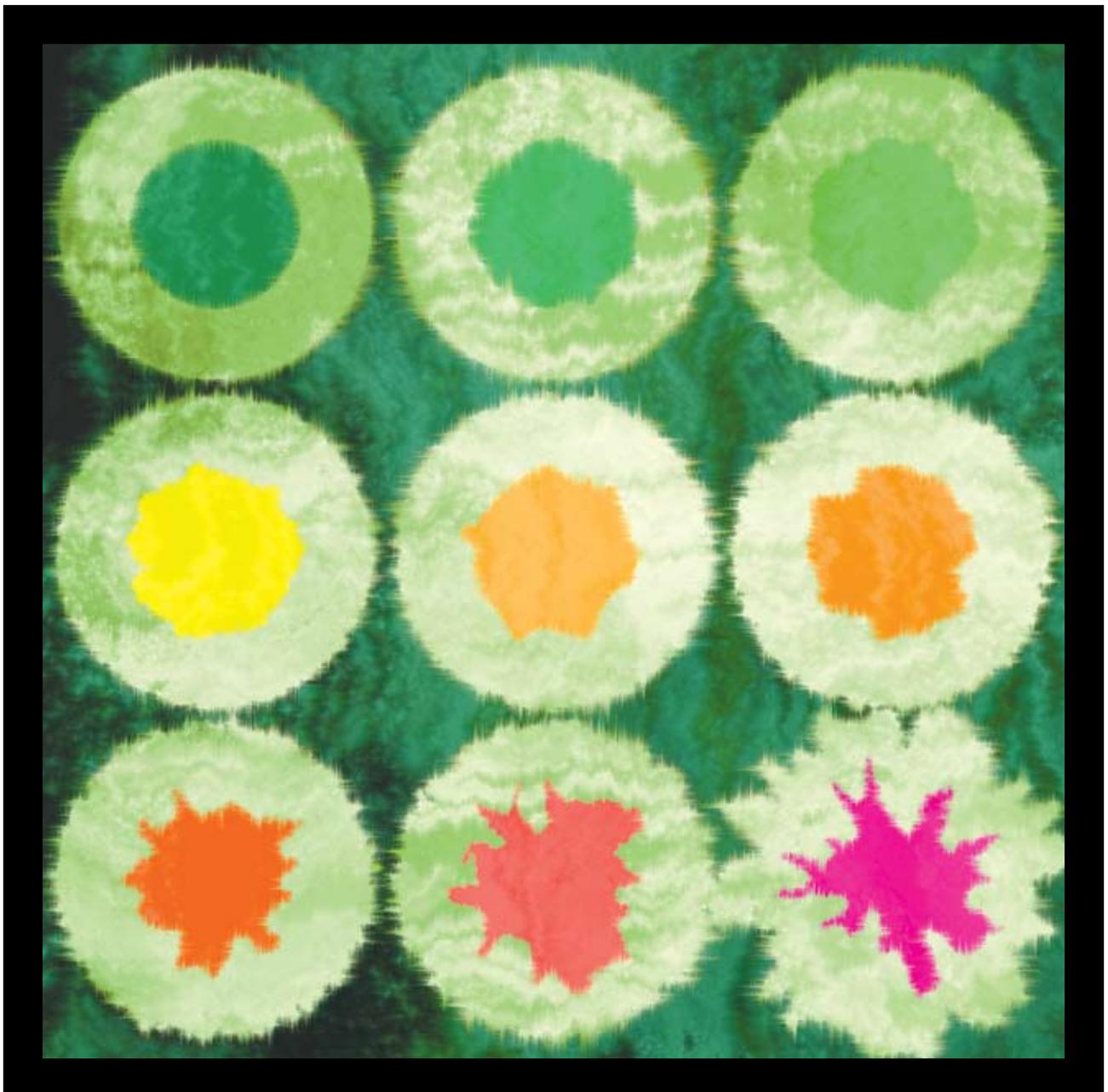
FOR HEALTH PROFESSIONALS



AUSTRALIA

PRACTICE

Spasticity and multiple sclerosis (MS)





Spasticity and multiple sclerosis (MS)

Spasticity is one of the most common physical symptoms experienced by people with MS. Because spasticity can significantly affect motor performance and the activities of daily living among people with MS, appropriate management should be implemented from the time of onset.

Contents	Page
1.0 Spasticity	02
2.0 Incidence and impact of spasticity in MS	
3.0 Clinical characteristics of spasticity	
3.1 Presentation	03
4.0 Pathophysiology of spasticity in MS	
5.0 Assessment of spasticity	04
5.1 Measurement tools for spasticity	
5.2 Measuring the impact of spasticity on quality of life	
6.0 Management of spasticity	
6.1 Preventing provocative factors	05
6.2 Reducing spasticity and preventing its consequences	
6.3 Physiotherapy	
6.3.1 Stretching	
6.3.2 Strengthening	06
6.4 Medical management of spasticity	
6.4.1 Pharmacotherapy	
6.4.2 Intrathecal baclofen pump	07
6.4.3 Botulinum toxin and phenol	
6.4.4 Surgery	
7.0 Summary	

1.0 Spasticity

Spasticity is a common feature in many neurological conditions including MS, stroke, spinal cord injury, traumatic brain injury and cerebral palsy. The most commonly used definition of spasticity is probably that of Lance in 1980,¹ who defined spasticity as: “a motor disorder characterised by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks resulting from hyper-excitability of the stretch reflex as one component of upper motor neuron syndrome.”¹ However, recently, controversy has surrounded this definition with calls for a more meaningful definition.^{2,3}

Although contracture and spasticity often occur together, they are different problems. Most definitions

of contracture include the concept that a muscle has shortened sufficiently to prevent the range of movement of the joint (or joints) it spans. Contracture is most commonly seen in people with significant weakness or marked spasticity, probably because both of these conditions can cause the muscles to become ‘immobilised’ at short lengths. This handout will focus on spasticity.

2.0 Incidence and impact of spasticity in MS

Along with fatigue and impaired balance, spasticity is one of the three most common physical signs and symptoms experienced by people with MS. Some epidemiological studies indicate that spasticity is a significant problem for between 60 and 80% of people with MS,⁴ and is a major contributor to disability in this population.⁵ Spasticity can significantly affect motor performance and the activities of daily living among people with MS, such as controlling the body during functional tasks, mobilising, completing personal care tasks, or sleeping. There is an association between the degree of spasticity and level of disability. Severe spasticity can also lead to muscle shortening or contracture.

The severity of spasticity tends to increase with the natural history of the disease unless appropriate management is implemented from the time of onset.^{4,6} Lower limbs are more commonly affected than upper limbs, and lower limb spasticity may manifest initially with extensor spasms, which hold the legs in rigid

extension for a prolonged period. These are most likely to occur at night or upon waking in the morning.⁵ As the disease progresses, spasticity often begins to affect flexor tone, and over time, the flexed posture can become permanent due to muscle contractures. Some people who experience severe spasticity become bed-bound. Positioning difficulties and fixed postures can lead to bedsores, which further exacerbate muscle spasms.⁷ However, spasticity does not always negatively impact on the function of people with MS. Spasticity can also increase the stiffness of muscles that have become weak due to loss of upper motor neuron inputs, especially antigravity muscles such as the quadriceps and gastrocnemius. Spasticity can therefore help support the body against gravity. This may enhance the performance of transfers to maintain functional independence in the presence of significant lower limb weakness. It is recommended that clinicians take this into consideration prior to commencing interventions that aim to reduce spasticity.

3.0 Clinical characteristics of spasticity

A spastic muscle has the following characteristics:

- Velocity-dependent resistance to passive stretch i.e. the faster the muscle is stretched the greater resistance and more reflex activity
- Length-dependent i.e. the resistance to passive stretch depends on the length of the muscle when it is stretched (clasp-knife phenomenon).

3.1 Presentation

Clinically, spasticity can present in the following ways:

- Muscle tone increases with rapid movement of an arm or leg or quick change of body position
- Sudden contraction of a muscle that causes an arm or leg to either flex or extend
- Abnormal posturing of the arms or legs
- A series of rapid muscle contractions such as bouncing of the foot (clonus) when the ankle is dorsiflexed
- This phenomenon is often more pronounced when the spastic muscle is tired after exercise
- Scissoring or crossing of the legs when attempting to stand, transfer or walk
- Exaggerated tendon jerk responses.

People with symptoms of spasticity may report a mild feeling of stiffness or tightness in certain muscles, or the symptoms may be more severe, such as painful uncontrollable spasms of the extremities (usually of the legs). Spasticity may also produce feelings of pain or tightness in and around joints, and can cause low back pain.

4.0 Pathophysiology of spasticity in MS

MS is characterised by disseminated lesions in the myelin sheaths of neurons in the central nervous system (CNS). The signs and symptoms experienced by a person with MS depend on the sites of the lesions, which tend to increase and spread during the natural history of the disease. If the lesions involve those parts of the CNS associated with motor pathways, the patient will have clinical signs and symptoms of an upper motor neuron (UMN) syndrome. UMN syndrome has two classical distinctions – negative features characterised by weakness and loss of dexterity, and positive features characterised by muscle over-activity. Spasticity is only one of the positive features.⁸ Other positive features of UMN syndrome are hyperactive tendon reflexes, clonus and flexor spasms. Clinically however, spasticity has been used as a generic term for all these clinical features.

Over the past decade several pathophysiological mechanisms have been proposed to explain the development of spasticity; these explanations are more theoretical than empirical. Thus, the underlying mechanism for spasticity remains controversial. A detailed discussion of these mechanisms is beyond the scope of this handout.

The definition of spasticity proposed by Lance¹ states that spasticity is a form of muscle over-activity due to a velocity-dependent increase in tonic stretch reflexes.

The increase in stretch reflex is thought to result from abnormal spinal processing of proprioceptive inputs. One view, based predominantly on animal studies, suggests that the abnormal spinal processing is due

to prolonged loss of inhibitions from descending supra-spinal inputs, such as reticulo-spinal, rubro-spinal and vestibule spinal tracts, and inhibitory Renshaw interneurons.^{9,10}

Another view suggests that spasticity is purely a spinal phenomenon; the tonic stretch reflex is enhanced within the spinal cord, rather than due to disinhibited normal stretch reflex.⁸ According to this theory, tonic stretch reflex is mediated by Ia afferent fibres predominantly in the muscle spindles, which are not more sensitive to stretch in a person with spasticity than they are in a person without spasticity. This suggests that the same amount of stretch (fast or slow) in both a person with and without spasticity produces the same amount of spindle activity feeding back to the spinal cord. Thus, it is argued that the increased stretch reflex is due to the increased excitability occurring in the spinal cord.

Additionally, Gracies^{11,12} recently proposed that contracture is one of the factors that causes excessive responsiveness to stretch, which in turn aggravates contracture.

In summary, the mechanisms underlying spasticity in general and particularly in MS are complex and not fully understood.

5.0 Assessment of spasticity

5.1 Measurement tools for spasticity

A range of measurement tools have been proposed for the assessment of spasticity in both clinical and laboratory settings.² The Ashworth Scale,¹³ and the Modified Ashworth Scale¹⁴ are the scales most commonly used by clinicians. The five-point Ashworth scale was originally proposed by Ashworth for use with people with MS. Subsequently, the Modified Ashworth Scale was proposed by Bohanon and Smith to differentiate more specifically between the lower levels of spasticity by adding a sixth point.¹⁴

Owing to the drawbacks of the Ashworth Scale and its modified version,¹⁵ clinicians have been looking for more useful instruments to measure spasticity. It has been suggested that the Tardieu Scale may be a more appropriate clinical measure of spasticity than the Ashworth or Modified Ashworth Scales on the basis that it adheres more closely to Lance's definition.¹⁶ The Tardieu Scale incorporates velocity control and has been shown to be more reliable than the Ashworth Scale. Furthermore, the Tardieu Scale can differentiate between spasticity and contracture in people who have had a stroke.¹⁷ However, the validity and reliability of the Tardieu Scale has not been established. There are other proposed techniques for measuring spasticity, such as the Wartenberg pendulum test and the Powered oscillation system, but these methods are only applicable to certain joints and may not be applicable in cases of severe spasticity.¹⁸

Thus, clinicians to date still do not have a valid instrument for the clinical measurement of spasticity. Further studies are needed to identify more valid and reliable measurement tools for spasticity.

5.2 Measuring the impact of spasticity on quality of life

Recently, Hobart *et al.* developed the Multiple Sclerosis Spasticity Scale 88 for measuring the impact of

spasticity on quality of life in people with MS.¹⁹ The scale contain a total of 88 questions divided into 8 sections. These questions use interval level scores to measure subjective perception of the impact of spasticity on different clinical areas, including both physical and psychological aspects. In developing this scale the authors attempted to conceptualise and measure the impact of spasticity from the patient's perspective. Although it has the potential to advance outcome measures of spasticity in clinical trials, the scale requires further research to understand the meaning of the score and score changes.

Notwithstanding the drawbacks of current measurement tools, it is necessary in clinical work to 'measure' spasticity, or the impact of spasticity on functions, in order to evaluate the effectiveness of interventions. Clinicians may choose a preferred method to assess spasticity. Ashworth or Modified Ashworth Scales are still preferable in clinical settings despite their limitations since they are easy to apply and not time consuming. In addition, functional assessments such as walking gait and walking speed can provide clinicians with information on how effective the interventions are. One precaution is that the degree of spasticity can vary throughout the day and depend on other aggravating factors such as fatigue. Therefore, it is best to assess spasticity in a reproducible environment. For an assessment of spasticity to be reliable over time for the same person, measurement should be carried out at the same time of the day, in the same position and with the same degree of fatigue.²⁰ Measurement of spasticity shortly after an intervention should be avoided unless the aim is to determine the effectiveness of this therapy.

6.0 Management of spasticity

The aims of spasticity management are to prevent or minimise provocative factors that may give rise to spasticity, and to reduce spasticity and prevent its consequences.²¹ The achievement of these aims requires a multidisciplinary approach involving the

person with MS, as well as their family or carer. Education and management strategies should be implemented as early as possible to prevent or reduce the disabling impact of spasticity in people with MS.

6.1 Preventing provocative factors

Conditions such as urinary tract infection, pressure sores, constipation, limb pain and the use of some medications (e.g., antidepressants and immunological modulators) can induce or worsen spasticity in people with MS.^{22,23} These provocative factors need to be identified and removed (if possible), or modified before further interventions are implemented. The person with MS, their family or carer should be kept informed so that they can assist health professionals in identifying potential factors that may provoke or intensify spasticity.

6.2 Reducing spasticity and preventing its consequences

Before making treatment decisions clinicians must consider a functional evaluation. This will determine whether or not spasticity is causing clinical symptoms and signs that are considered disabling. For example, a person may complain of difficulty when beginning to walk in the morning after waking up or after sitting still for a long time due to stiffness in the legs. In this case a stretching technique to temporarily reduce stiffness may be useful (refer to 6.3.1 Stretching). In contrast, a non-ambulant person may use the stiffness of the legs to weight-bear transfer. People who can use stiffness to their advantage, and for whom spasticity is not affecting other functions, may not need treatment. Thus, functional evaluation is important for making decisions regarding appropriate treatments.

Historically, the treatment approach for spasticity has involved physical management followed by oral medication.²¹ When these interventions fail to improve function, and spasticity becomes quite disabling (e.g., where the person is unable to maintain balanced sitting due to excessive knee flexion), more invasive treatments may be used, such as intrathecal baclofen, nerve block, and neurosurgery. Presently, the approach to treatment of spasticity is multidisciplinary. A person may at some point receive physiotherapy interventions such as exercise programs, take an anti-spastic agent that is evaluated and titrated in light of their specific needs, and use assistive devices to improve daily functioning of the spastic limb.

6.3 Physiotherapy

The physiotherapist has a key role in providing education about spasticity for both the person with MS and their carer, as well as commencing physiotherapeutic interventions aimed at promoting

optimal movements to prevent contractures, reduce pain, and improve functions (e.g., walking gait).

The physiotherapist also has a role in referring the person to their doctor or neurologist for further advice regarding initiating or adjusting the dosage of oral medications. It is important for the person with MS, physiotherapist, and doctor to communicate in order to obtain the best balance of medication and exercise so that spasticity is minimised and physical function is maximised. For example, consider the oral agent baclofen which is commonly prescribed for spasticity. Baclofen has a short half-life (the time the half of the medication is eliminated from the body) taking approximately 1.5–2 hours to reach maximum effect. Given this, the dose should be timed to address problems caused by spasticity, such as getting up and getting dressed in the morning. For some people it may be preferable to take medication at night only, while others may be better suited to a daytime dose. Taking baclofen with meals three times a day (as is often prescribed for people with MS) may not be the most appropriate regimen.

Many physical modalities and methods have been used by physiotherapists in the management of spasticity, including electrical stimulation,²⁴ massage, cooling, hydrotherapy,²⁵ stretching and strengthening. Among these methods, stretching and strengthening are perhaps the most common methods that have been used extensively in clinics.

6.3.1 Stretching

The key aims of stretching in spasticity are to improve muscle extensibility, reduce muscle stiffness, and improve function. Clinically, a range of stretching techniques is used including static, dynamic, Proprioceptive Neuromuscular Facilitation (PNF), as well as prolonged and ballistic stretching. Although it has been well-documented that these stretch-based techniques can temporarily change viscoelastic properties and excitability of the muscles,²⁶ there is no direct clinical evidence that these effects can become permanent and result in a reduction in spasticity. A recent review of investigating the effects of stretching on spasticity showed the clinical benefits of stretching to be inconclusive.²⁶ However, it is noted that most of



the studies cited in this particular review were of low quality, and the clinical outcome measures used often failed to measure spasticity.²⁶ Controlled studies of the effects of stretching on spasticity are urgently needed. In the meantime, clinical physiotherapists should consider continuing with stretching intervention for spasticity where it is warranted.

6.3.2 Strengthening

Strength training is another physiotherapy intervention often used clinically for the management of spasticity. It was previously thought that strength training might worsen muscle spasticity, but evidence has not shown this to be the case.²⁸ Strength training is now a commonly accepted intervention. Studies involving a population of people with stroke have demonstrated that as time progresses, reduced strength (rather than spasticity) becomes the primary activity-limiting factor.²⁹ Therefore, strengthening muscles may not have direct effect on spasticity, but may improve functions that are compromised by spasticity and/or weakness. In addition, it is suggested that when there is less effort (spastic muscle is stronger), there will be less increase in tone; so it becomes a cycle.³⁰

To date, there is limited evidence regarding the effectiveness of physiotherapy in treating spasticity; however these interventions are considered critical to the success of medical and surgical interventions for spasticity. For example, it has been shown that stretching may enhance the benefits of baclofen,³¹ or botulinum toxin injections used for focal spasticity.³² Rochester *et al.*³³ have suggested that stretching following eccentric contractions can decrease motor neuron excitability. This may therefore be beneficial in decreasing spasticity whilst strengthening muscle.

6.4 Medical management of spasticity

When the muscle overactivity is distributed diffusely and physiotherapy interventions are unable to adequately manage the symptoms of spasticity, medical interventions are considered. Non-invasive medical management of spasticity relies on the use of anti-spastic medications. Invasive treatment involves the use of intrathecal baclofen pump, botulinum toxin injection, and surgical procedures.

6.4.1 Pharmacotherapy

Anti-spastic medications are usually used to treat mild, moderate, or general spasticity. Four common

oral anti-spastic drugs include baclofen, diazepam, dantrolene, and tizanidine; tizanidine is not currently available in Australia. These agents are indicated to promote tone reduction, improve range of motion, and facilitate rehabilitation via the central mechanisms of either enhanced pre-synaptic inhibition (e.g., diazepam and baclofen) or decreased activities of excitatory interneurons (e.g., tizanidine). Dantrolene, on the other hand, acts outside the CNS. It inhibits calcium release from the sarcoplasmic reticulum, thus decreasing the excitation–coupling reaction.³⁴ Of these agents, baclofen is probably the most common drug used for the management of spasticity in people with MS. Some studies have shown that baclofen and tizanidine are more effective than diazepam and dantrolene. However a recent systematic review demonstrated that the efficacy and tolerability of these drugs is still poorly documented and no prescribing advice can be recommended.³⁵

All anti-spastic medications produce adverse side effects. The most common side effect is muscle weakness, while other effects include drowsiness, dizziness, dry mouth, nausea, increased urinary frequency, light-headedness, and withdrawal effects. Each of these side effects can be troublesome for people with MS who may already be experiencing symptoms of weakness, fatigue and/or incontinence.³⁶ Prescription of these drugs requires close monitoring through collaboration between the person with MS, their doctor, and physiotherapist. It can be difficult to differentiate between weakness caused by disease progression or weakness that is a side effect of an anti-spastic drug. It is not uncommon to see people with MS take these drugs for many years without review. In many cases they may be taking doses that are too low to adequately manage their spasticity, or so high that they cause functional or quality-of-life impairments. During assessment, if there is doubt about the signs and symptoms of side effects from anti-spastic medications, physiotherapist should refer clients back to their doctor. In addition, thorough education during the initial prescription of these medications can assist people to gain the most value from anti-spastic medications.



In recent years there have been reports that cannabis and its major components, cannabinoids, have beneficial effects on MS-related symptoms such as pain, tremor, and spasticity.³⁷ However, cannabis-based medications have not been shown to have a beneficial effect on spasticity as measured with the Ashworth scale.³⁸ Further research is required to identify more objective evidence for the use of cannabinoids in the treatment of spasticity and other symptoms of MS.

6.4.2 Intrathecal baclofen pump

Some people may have chronic and severe spasticity that is unresponsive to therapeutic doses of the aforementioned anti-spastic drugs, or experience intolerable side effects from these drugs. In these cases the use of an intrathecal baclofen pump (the invasive approach) may be recommended. Intrathecal baclofen therapy involves direct long-term delivery of baclofen to the intrathecal space using an implanted programmable pump. The pump is usually implanted subcutaneously near the waistline.

Studies show that the benefits of intrathecal baclofen therapy for managing severe spasticity may include a reduction in spasticity, improvement in the ability to sit in a wheelchair, as well as stand and walk, and improved nursing care.^{39,40} Limitations include its probative cost (as it is an expensive initial procedure that requires ongoing maintenance, extensive screening, and selection process for suitable candidates) as well as the risk of complications, such as infection or pump dysfunction.

6.4.3 Botulinum toxin and phenol

When spasticity is focal, botulinum toxin injections may be indicated. Botulinum toxin is a neurotoxin (that blocks nerve conduction) produced by bacteria *Clostridium botulinum*. There are several types of botulinum toxin, but only type A and B are used for medical treatment. It has been reported that botulinum toxin type A can reduce focal spasticity in people with MS or after stroke.⁴¹ The clinical benefits from

this reduction are clearer in the upper limb than in the lower limb.⁴² There is little data on the use of botulinum toxin type B in spasticity. The disadvantages of botulinum injections include the need for repeat injections (as the effects last for approximately three months), being limited to treating focal and not general spasticity, and the possibility of significant side effects – such as sensory damage. The clinical use of botulinum toxin type A for addressing severe spasticity is becoming increasingly popular.

Phenol is another substance that had been used widely in controlling focal spasticity before the introduction of botulinum type A. Although phenol injections are much less costly than botulinum toxin, they are used infrequently due to the inherent difficulties associated with performing injections, their sensory complications, and unpredictable side effects.

6.4.4 Surgery

Surgery is only considered for the treatment of people with severe spasticity after conducting a thorough evaluation of their current level of function, trying all less invasive management options, and setting realistic expectations for surgical outcomes. As with any other spasticity treatment, surgical intervention must be incorporated into an overall spasticity management plan. An ongoing physiotherapy program following surgery is important to maximise the benefits of surgery. Only when musculoskeletal deformities significantly impair a person's personal care functions are surgical options such as tenotomy, osteotomy, tendon transfer or selective dorsal rhizotomy to alleviate focal spasticity and its secondary consequences (such as contracture) considered. A detailed description of surgical procedures is beyond the scope of this document.

7.0 Summary

- Spasticity significantly affects 60% of people with MS and tends to increase in severity as the disease progresses. Spasticity can impair a range of activities including mobility, personal care and sleeping.
- The mechanisms underlying spasticity in MS are still poorly understood



- The commonly used assessment scales for measuring spasticity in a clinical setting are the Ashworth Scale and Modified Ashworth Scale; these scales have not been appropriately validated for use in people with MS. Functional testing is recommended during assessment of spasticity.
- Management of spasticity aims to prevent and minimise provocative factors, reduce spasticity, and prevent its consequences. This requires a multidisciplinary approach and involvement of the person with MS and their family or carer.
- Spasticity interventions should be initiated as soon as possible after symptom onset.
- Mild-to-moderate spasticity should be treated with a combination of physiotherapy interventions (such as stretching and strengthening) and a titrated dose of oral anti-spastic medication, such as baclofen.
- Severe spasticity that is unresponsive to physiotherapy interventions and oral anti-spastic drugs may benefit from intrathecal baclofen therapy.
- Botulinum injection is effective in reducing focal spasticity but not general spasticity. It is an expensive therapy.
- Surgical interventions are not commonly used to manage spasticity in people with MS; they may be indicated when spasticity is severe and causes significant impairment of personal care activities.
- Further research is required to investigate the effectiveness of physiotherapy interventions as well as new approaches for the management of spasticity.

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MS Practice//For Health Professionals

MS Practice is an initiative of MS Australia (MSA). MS Practice is an online resource designed to support allied health professionals in the symptom management of people with multiple sclerosis (MS). The series addresses the various symptoms associated with MS, providing health professionals with evidence-based information and clinical practice recommendations to enhance the quality of care and outcomes for people with MS. The MS Practice topics were identified by the MSA Physiotherapy Network.

Spasticity and multiple sclerosis

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Credits

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MS Australia

MS Australia is a not-for-profit organisation that has been supporting people with MS since 1956. Through state-based MS Societies, MS Australia strives for a world without MS through quality research and service excellence for people with multiple sclerosis, their family and friends, and healthcare professionals.

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This handout is intended to provide information to support current best practice for the management and treatment of physical impairments in people with MS. While the information is available to all health professionals, there are details that are most relevant to physiotherapists, exercise physiologists, and people who are qualified to provide exercise opportunities for people with MS. MS Australia has made every effort to ensure that the information in this publication is correct. MSA does not accept legal responsibility or liability for any errors or omissions, or for any physical or financial loss incurred whilst participating in the exercises or activities outlined in the MS Practice handouts. Be sure to seek advice from the sources listed.



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