



# Multiple sclerosis information

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## for health and social care professionals

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# Symptoms, effects and management

## Spasticity

### Spasticity

Spasticity can be a complex and challenging symptom to manage in neurological conditions and is a common symptom experienced by people with multiple sclerosis. The ongoing management of spasticity requires teamwork between the person with spasticity, their regular carers, and members of the multidisciplinary team<sup>1,2</sup>. In a survey 84% of people with MS reported symptoms of spasticity with one third rating it as moderate or severe<sup>3</sup>.

#### What is spasticity?

The true nature of spasticity is still not clearly understood. The most common definition used is: 'a motor disorder characterised by a velocity dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflex, as one component of the upper motor neurone syndrome'<sup>4</sup>. More succinctly spasticity has been defined as 'the velocity dependent increase in resistance of a passively stretched muscle'<sup>5</sup>.

More recently these definitions have been challenged by a European working group as narrow and limiting. Specifically this group identified that the term spasticity is used differently by clinical and research communities and concluded that spasticity is not a pure motor disorder, or just a result of the hyper-excitable stretch reflex or dependent on the velocity of the stretch. They suggested a new definition as, 'Disordered sensorimotor control, resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary activation of muscles'<sup>6</sup>.

The resistance to passive movement caused by spasticity is generated by abnormalities in the control of movement by the central nervous system (CNS). As well as this neural involvement of spasticity there are also biomechanical changes, which occur both in muscles and connective tissue, which through disuse and immobility can lead to reduced range of movement or contractures<sup>7</sup>. Increased resistance to passive movement felt by the clinician, often referred to as hypertonia, may be caused by a combination of spasticity, which is neurally generated, and biomechanical changes in the muscle and connective tissue. Together these changes can significantly affect function<sup>8</sup>.

Spasticity is one component of the upper motor neurone syndrome<sup>9</sup> that occurs as a result of acquired damage to any part of the CNS, including the spinal cord. It has a range of effects, which can be categorised into positive and negative features (Table 1). Most people will present with a combination of features. One or several of the positive features will influence the resistance to passive movement. Often people are described as having spasticity, but it is likely they will also have other features of the upper motor neurone syndrome.

Table 1. Features of the upper motor neurone syndrome<sup>9</sup>

Positive features	Negative features
Spasticity	Weakness
Spasms (flexor, extensor, adductor)	Loss of dexterity
Increase in tendon reflexes	Fatigability
Clonus	
Positive support reaction	
Extensor plantar responses	

#### Why does spasticity occur?

The control and regulation of normal skeletal muscle activity involves a complex combination of descending motor commands, reflexes and sensory feedback from the brain, spinal cord and peripheral sensation. During normal movement, influences from the cerebral cortex, basal ganglia, thalamus and cerebellum, travelling via upper motor neurones adjust, reinforce and regulate the lower motor neurone which connects directly via peripheral nerves to the muscle to form smooth, coordinated muscle activity and maintenance of posture.

In simple terms spasticity occurs when there is damage to these descending upper motor neurone tracts (eg a plaque in MS) which interrupts the regulation of spinal cord and lower motor neurone

activity. This can result in enhanced lower motor neurone activity and an increase in muscle activity, in response to peripheral stimuli (eg muscle stretch, a urinary tract infection or pressure ulcer)<sup>10,11</sup>.

### Consequences of spasticity

Spasticity can affect physical activities such as walking, transferring, picking up objects, washing, dressing and sexual activity. It can also have an emotional impact, on for example, mood, self-image and motivation<sup>12-14</sup>. Safety in sitting and lying can also be compromised due to spasms or persistent poor positioning<sup>15,16</sup> which can lead to the development of contractures. This can potentially lead to restricted mobility and social isolation.

Symptoms of the upper motor neurone syndrome are not always detrimental and they may even be positive in improving vascular flow and assisting in transfers and even walking<sup>17</sup>. Therefore the treatment of spasticity needs to be carefully selected and reviewed over time in order to meet the individual's aims and to maintain and promote function.

### Management and treatment of spasticity

Two core principles of spasticity management are<sup>2,15,16</sup>:

**Optimising an individual's posture and movement** through use of appropriate seating, stretching and exercise programmes<sup>2</sup>.

**Preventing or managing factors that may increase spasticity and spasms.** Primarily exacerbations can occur from cutaneous stimuli such as skin irritation, pressure sores, ingrown toenails, tight fitting orthoses. Visceral stimuli including incomplete bladder emptying, constipation, bowel impaction and infections, for example urinary tract infections, can be triggers<sup>2,10</sup>. Patterns of movement in function and sustained postures throughout the day and night can also aggravate spasticity and spasms.

These principles need to be regularly considered and reviewed over time and used in conjunction with medical treatments. Pivotal to their success is ongoing multidisciplinary teamwork across hospital and community settings working collaboratively with the person with spasticity to effectively manage their symptoms<sup>1,2</sup>.

### A multidisciplinary approach

Effective communication between disciplines is vital to enhance the management of an individual's spasticity. Each discipline can be seen to have specific expertise within the team. However this is not exclusive and teamwork is essential<sup>1,2</sup>.

**Nurses** have a significant role in educating a person on managing trigger factors and about the available treatments to manage spasticity. They can provide ongoing support and advice to a person and their family as they live with and adjust to managing spasticity and spasms over time.

**Physiotherapists** can carry out specific treatments to assist an individual to manage muscle tone particularly the biomechanical changes. Treatment may include appropriate exercise programmes that may encompass stretches, active exercises or standing. Advice can also be given regarding posture and positioning throughout the day.

**Occupational therapists** can play a key role in assessing and recommending appropriate adaptations to an individual's environment and advising on how to maximise activities of daily living within the context of spasticity. Appropriate seating is of particular importance in spasticity management.

Occasionally the expertise of speech and language therapists can be sought when spasticity affects neck and facial muscles<sup>18</sup>.

Medical management is important in terms of assessing, prescribing and evaluating the use of antispasticity drugs. In conjunction with other members of the team, doctors can decide the appropriate timing and selection of more invasive treatments.

Inpatient rehabilitation may be appropriate to provide a more thorough assessment of an individual's spasticity throughout a 24 hour period and to allow a more detailed management programme to be developed.

Sometimes despite optimal physical management programmes and optimisation of trigger factors pharmacological measures are necessary. Depending on the pattern of spasticity these can be generalised or focal.

# Symptoms, effects and management

## Spasticity

### Generalised treatments<sup>19</sup>

**Baclofen** acts on the CNS and is the most commonly used antispasticity drug. To avoid side effects it needs to be started at low doses, slowly increased and stopped at a dose that does not cause unwanted side effects. The effect of an oral baclofen dose can last between 4-6 hours so doses need to be taken regularly to ensure adequate control of symptoms. Side effects can include weakness, drowsiness and dizziness.

**Gabapentin** is useful for treating spasticity and spasms. It is particularly helpful in managing spasticity when pain is associated with it. Side effects can include drowsiness, dizziness and fatigue.

The NICE clinical guideline states that the following should only be given if treatment with baclofen or gabapentin is unsuccessful or side effects are unmanageable.

**Tizanidine** also works on the CNS and needs to be introduced slowly to avoid side effects. Regular blood tests should be performed to ensure there is no adverse effect on liver function. Side effects can include weakness, drowsiness and dry mouth.

**Diazepam or clonazepam** can be used alone or in combination with other drugs. Their daytime use is limited by sedative side effects, but if taken prior to sleep they can be very useful in managing nocturnal spasms. Side effects can include drowsiness and dizziness.

**Dantrolene** is the only antispasmodic drug that works directly on the muscles rather than on the CNS. It can be used in combination with other drugs. Often it is not well tolerated and can cause nausea, vomiting, diarrhoea and weakness. Regular blood tests need to be completed to ensure no adverse effect on liver function.

**Sativex** is a cannabis extract which works on the cannabinoid receptors in the brain and spinal cord. It is licensed in MS as an add-on therapy for those people whose spasticity and spasm has not responded to the other available drugs<sup>20,21</sup>. It is available as an oral spray. Side effects can include dizziness, sleepiness and feelings of light headedness. Occasionally the spray can cause soreness in the mouth so it is important to change the spray site regularly. About half of people with

MS will respond to Sativex; whether someone is a responder can be identified after a four week trial of the drug. The dose of Sativex is then controlled by varying the number of sprays taken each day.

### Focal treatments

**Botulinum toxin** can be injected into muscles and acts as a neuromuscular block which causes the targeted muscle to become temporarily weak. It can take 10-14 days for the full effect to be felt. It must be used in conjunction with physiotherapy/occupational therapy and an exercise programme to maximise effect and to promote an ongoing change in the spasticity once the toxin has worn off (approx. three months)<sup>2, 22,23</sup>.

**Phenol or alcohol motor point injections.** The injection permanently destroys nerve fibres in the injected muscle. Some nerves may partially regrow, causing the effect to wear off after several weeks or months. Injections can however be repeated if necessary.

### Intrathecal therapies

**Intrathecal baclofen** acts by binding to gamma aminobutyric acid (GABA) receptors and results in inhibition of mono and polysynaptic spinal reflexes<sup>24</sup> with associated reduction in spasm, clonus and pain. A concentration of GABA receptors is situated in the intrathecal space of the spinal cord. Delivering baclofen intrathecally accentuates its antispasticity effect whilst minimising the troublesome systemic side effects associated with oral intake.

An implanted pump can deliver baclofen directly to this area and can be used to treat generalised lower limb spasticity<sup>25-27</sup>. It requires commitment from the person with MS, not only during the trial and implant phase, but also for its ongoing maintenance of regular reservoir refills and pump replacements<sup>2</sup>. It is however an extremely effective treatment and is being used earlier in people with MS to improve their walking<sup>28</sup>.

**Intrathecal phenol** is a permanent destructive procedure<sup>29</sup>. It can be helpful for some people, to treat very severe spasms that do not respond to other drug treatments. The effects of an injection can sometimes wear off but can be repeated if necessary.

Negative effects on lower limb sensation, sexual function, bladder and bowel management can occur so appropriate patient selection is critical to ensure effective strategies are in place to manage these<sup>30</sup>.

Intrathecal treatments require a detailed clinical governance framework to ensure safety of administration, an example of guidelines and nursing care plans from one service have been published<sup>2</sup>.

## Surgery

Occasionally orthopaedic or neurosurgical procedures may be recommended. These can include myelotomy (severing of tracts in the spinal cord) and rhizotomy (resection of posterior roots)<sup>31,32</sup>.

## Complementary therapies

Some individuals with spasticity report that complementary therapies such as acupuncture can help relieve symptoms.

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### MS Trust resources

Spasticity and spasms factsheet

Care pathway: the role of the health care professional in the management of spasticity.



We hope you find the information in this book helpful. If you would like to speak with someone about any aspect of MS, contact the MS Trust information team and they will help find answers to your questions.

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