Chapter 10
Management of Spasticity

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Abstract Spasticity is one of the many components of the upper motor neurone syndrome; the other components including exaggerated reflexes, clonus, clasp-knife phenomena, flexor and extensor spasms, spastic dystonia, and Babinski's sign. Spasticity is a symptom that is not isolated and can cause pain, stiffness, and spasm, resulting in a massive impact on a person's physical and emotional lifestyle. The management of spasticity requires a multidisciplinary approach incorporating nurses, physicians, physiotherapists, occupational therapists, and orthotists working together to provide a variety of treatments tailored to the needs of the individual patient.

Keywords Spasticity • Tone • Upper motor neurone • Stroke • Post-stroke

Key Messages

- Spasticity is one component of the upper motor neurone syndrome that is characterised by increased tone, exaggerated reflexes, weakness, and contractures.
- Spasticity can cause pain, stiffness, and spasm, resulting in a massive impact on a person's physical and emotional lifestyle as well as carer burden.
- The management of spasticity involves a multidisciplinary team approach to direct treatment tailored to the needs of the individual patient.
- A variety of non-pharmacological and pharmacological treatment options for spasticity exist.
- The management of spasticity is integral to the aims of rehabilitation involving re-education of movement and promotion of independence.

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Introduction

The most common definition used for spasticity is a motor disorder characterised by "a velocity dependent increase in the tonic stretch reflex (muscle tone) with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflex, as one component of the upper motor neuron syndrome" [1]. With its increased tone and exaggerated tendon jerks, spasticity may be a significant cause of disability and pain and, if untreated, may lead to contractures. It is one component of the complex clinical picture that results from the upper motor neurone syndrome that includes weakness, loss of dexterity, fatigability, and muscle spasms [1]. The extent and type of spasticity can fluctuate widely according to position, fatigue, stress, and drug use. It is a dynamic phenomenon and requires continued multidisciplinary assessment and management.

Epidemiological studies have shown spasticity to affect 17–38 % of stroke patients and for it to occur usually within the first few weeks or months following stroke [2, 3]. However, the onset of spasticity is highly variable and can occur in the short-, medium-, or long-term post-stroke period [4]. Spasticity presents in a variety of ways depending on the size, location, and age of lesion. Epidemiological studies have demonstrated spasticity to affect primarily the elbow (79 % of patients), the wrist (66 %) and the ankle (66 %) [5]. In the upper limbs, the most frequent pattern of arm spasticity is internal rotation and adduction of the shoulder coupled with flexion at the elbow, the wrist, and the finger [6, 7]. In the lower limbs, adduction and extension of the knee with equinovarus foot is the most observed pattern.

Pathophysiology of Spasticity

Spasticity is one of the positive features of upper motor neurone syndrome and arises from upper motor neurone lesions involving the corticoreticulospinal system in the brain, brainstem (most importantly, those arising in the bulbopontine tegmentum), or spinal cord, and the clinical syndrome depends on the lesion's location, extent, and the time since it occurred [8]. These lesions disturb the balance of supraspinal inhibitory and excitatory inputs, producing a state of net disinhibition of the spinal reflexes. These include proprioceptive (stretch) and nociceptive (flexor withdrawal and extensor) reflexes [9]. The increased spinal cord excitability and impaired inter-neuronal systems result in increased muscle tone, hyper-reflexia, muscle overactivity, and antagonist muscle co-contraction.

Most of the important upper motor neurones controlling spinal reflex activity arise in the brainstem. However, the ventromedial reticular formation, the origin of the main supraspinal inhibitory tract (dorsal reticulospinal pathway), is under cortical control (Fig. 10.1). A lesion in the path of these corticobulbar fibres, either in the cortex or in the internal capsule thus results in reduced inhibitory drive and net excitation of spinal cord activity. An appreciation of this neuronal pathway explains

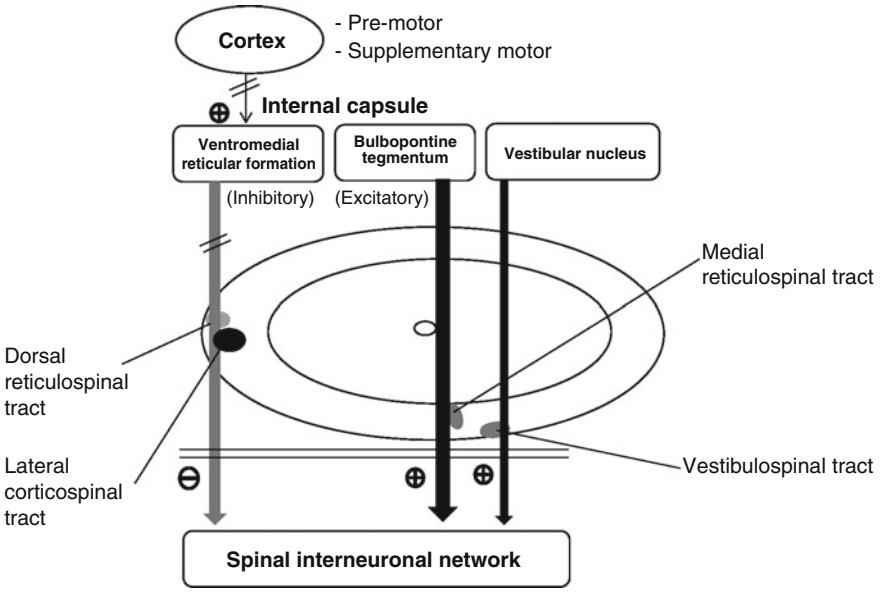

Fig. 10.1 The majority of descending pathways controlling spinal reflex excitability. (Inhibitory fibres are shown in grey and excitatory fibres are shown in *black*)

why spasticity secondary to stroke is usually less marked than that due to a spinal cord lesion, with less severe upper motor neurone features [10].

There are two main contributory factors to resistance to movement in the context of post-stroke limb spasticity: a neurogenic component (overactive muscle contraction) and a biomechanical component (stiffening and shortening of the muscle and soft tissues). If left untreated, a vicious cycle occurs in which unopposed contraction due to spasticity of affected muscle groups leads to abnormal limb posture, resulting in soft tissue shortening and further biomechanical changes in the contracted muscles. This, in turn, prevents muscle lengthening and perpetuates further tonicity and formation of contractures [11].

Effect on Lifestyle

People with spasticity often feel embarrassed and frustrated with its limiting effect on daily activities [10, 12]. Severe pain and stiffness, in addition to loss of function, can have a devastating effect on the patient, and problems with sleep due to spasms can lead to fatigue and depression. Spasms in the limbs may also result in problems with positioning and pain that may, in addition, affect sexual relationships. Maintaining hygiene may prove difficult adjacent to areas with increased tone, and patients with spasticity are also at a high risk of developing pressure ulcerations [13]. The patient's emotional and psychological state can be in constant turmoil,

with a strain on their social life, and referral to appropriate specialist agencies may be of benefit [14]. For some patients, spasticity may not only be distressing and painful, but an expensive cause of disability in terms of increased carer burden and reduced rehabilitative progress.

It may be seen, therefore, that secondary complications arising due to spasticity include impaired movement, hygiene, and self-care; poor self esteem, body-image, and sleep patterns; low mood; deformity; weakness; pain; contractures; and pressure ulcers. Patients with spasticity are also more likely to live in institutional care than in their own home, and are significantly more functionally impaired than those without spasticity.

Assessment

Spasticity assessment includes both identifying which muscles or muscle groups are overactive, and also determining the effect of spasticity on all aspects of patient function, including mobility, employment, and activities of daily living. Furthermore, factors such as cognition and deficits of sensation, attention, and vision (that may exacerbate spasticity) need to be evaluated. A systematic approach to assessment is required by a multidisciplinary team involving medical specialists (often from rehabilitation medicine or neurology disciplines), nurses, and allied healthcare professionals including physiotherapists, occupational therapists, orthotists, and rehabilitation engineers.

Formal assessment of tone may be measured using clinical scales such as the modified Ashworth and Tardieu scales (Table 10.1), or using techniques such as

Table 10.1 Modified Ashworth and Tardieu scales

Modified Ashworth scale	
0	No increase in muscle tone
1	Slight increase in muscle tone, manifested by a catch and release, or by minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension
1+	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder of the range of movement
2	More marked increase in muscle tone through most of the range of movement, but affected parts easily moved
3	Considerable increase in muscle tone, passive movement difficult
4	Affected parts rigid in flexion or extension
Modified Tardieu scale	
0	No resistance throughout the course of the passive movement
1	Slight resistance throughout the course of the passive movement, with no clear catch at precise angle
2	Clear catch at precise angle, interrupting the passive movement, followed by release
3	Fatiguable clonus (<10 s when maintaining pressure) occurring at precise angle
4	Infatiguable clonus (>10 s when maintaining pressure) occurring at precise angle

electrogoniometry to measure range of movement across a joint or quantitative neurophysiology [15, 16]. Whilst the latter two techniques are not widely available or practical in routine clinical practice, the clinical scales require no instrumentation, are quick to carry out, and have good inter- and intra-observer reliability [17–19]. The modified Ashworth scale is most widely used but its validity, reliability, and sensitivity are acknowledged to have limitations [20]. A number of scales also exist to assess patients' self-reported health status including pain, comfort, mobility, continence, and fatigue [21, 22]. Since pain and stiffness are important and troublesome symptoms in relation to spasticity, visual analogue, and verbal rating scales are often usefully employed to record objective change following treatment. In those who have communication deficits or who lack numerical skills, pictoral rating scales may be used. In people with severe cognitive deficits and problems with communication, tools such as the AbilityQ may be used to test an individual's ability to use different types of scales and thus help present questions in an appropriate format [23].

In patients with selective, underlying, voluntary movement in limbs with increased tone limiting "active" function, functional assessments are helpful tools to guide rehabilitative progress. Examples include the Action Research Arm, Frenchay, and nine-hole peg tests for the upper limbs; the Functional Ambulation Category, 10-m walking time, and 6-min walking distance tests for lower limbs [11]. Passive function can also be assessed using verbal or visual analogue ratings of "ease of care", timed-care tasks (for example, the time taken for washing and dressing), or formal scales that measure dependency or carer burden (such as the Barthel's index of activities of daily living).

First introduced in the 1960s, goal setting and its attainment has developed into a crucial element of spasticity assessment [24]. The attainment of goals following interventions varies amongst patients, and a single outcome measure is not always able to capture all domains. The Goal Attainment Scale (that involves simple recording of treatment goals achieved) has proved useful in terms of being suitable for patients with health problems, who need a multidimensional but individualised approach to treatment planning and outcome [25].

Purpose of Treatment

The management of muscle tone is an integral part of therapy for patients suffering from spasticity. Muscle tone is a dynamic, complex process that is part of an overall pattern of posture and movement. Appropriate management of tone is one of the fundamental principles of the Bobath method of facilitative physiotherapy, which gives priority to normalisation of tone and improving symmetry even at the cost of postponing standing or walking. However, this pre-occupation with normalisation of tone is not supported by evidence, and there are several other approaches which combine early mobilisation with active muscle tone management during rehabilitation [26].

The management of abnormal tone and spasticity is difficult, as it depends on achieving the right balance between hypo- and hypertonia between different muscle

groups. The problem is compounded by the fact that spasticity varies between different groups of muscles and times of the day, and is affected by the emotional state of the patient, activity being undertaken, limb posture, and the timing of medication. Inappropriate exercise can result in inappropriate tone patterns, to the ultimate detriment of the patient. If not managed correctly, spasticity leads to poor gait patterns, contractures, and loss of function.

Spasticity should be considered in relation to other impairments, and in the context of therapy goals, because interventions directed solely at reduction of spasticity are unlikely to result in significant functional gains. The therapeutic management of spasticity is closely related to the aims of rehabilitation; these include avoidance of complications, restoration of movement, re-education of movement and gait, development of self-dependency, and social integration, improving self-esteem and overall body image, as well as promoting new neurophysiological dynamics and neural plasticity. A further aim of treating spasticity is to relieve pain and other distressing symptoms that have a detrimental effect on quality of life.

There should be a multidisciplinary team approach to spasticity management, through which realistic goals and expectations of the patients, families, and caregivers can be established. It is important that treatment be tailored to the individual patient, and factors that may aggravate spasticity, including inter-current medical illness, medications that increase muscle tone, and emotional stressors, should be managed in the first instance [27, 28]. It should also be borne in mind that some patients may be able to use their increased tone to aid with function and maintaining postural control and ambulation, and so global reduction of tone may be destabilising. Appreciation of the differing treatment options for focal versus global spasticity is important, as is the awareness that treatment of spasticity may ameliorate weakness in affected limbs.

Non-pharmacological Approaches

Prevention of Aggravating Factors

In addition to causing pain and discomfort, pain and discomfort themselves and other nociceptive stimuli aggravate the symptoms from spasticity. As such, a multidisciplinary approach to identifying any aggravating factors and treating them is crucial to management of spasticity. Besides pain and discomfort, the other common aggravating factors are constipation, infection, tight clothing, and poor postural management.

Education and Psychological Support

All members of the multidisciplinary team should provide education to patients and carers about the causes and nature of spasticity and, if needed, strategies should be employed to reduce emotional stress. Patients and carers should be provided with

verbal and written information, including information leaflets, to help them understand how spasticity affects day-to-day function and how to avoid any triggers. Patients need to be made aware of how visceral and cutaneous stimuli may affect their spasticity.

Involvement of Physiotherapy/Occupational Therapy

Treatment of abnormal tone is initiated by physiotherapists, who can offer a range of interventions including physical therapy, attention to posture and seating, and orthotic devices [29–31]. Correct positioning is a critical aspect of management in order that the patient is in a balanced and stable posture that is comfortable and maximises function. Optimal seating is planned and implemented by occupational therapists and physiotherapists, and this may involve the use of a variety of seating adjustments such as foot straps; knee blocks; and head, neck, and trunk supports [32]. Occupational therapists and physiotherapists also are responsible for application of casts and splints to minimise spasticity and prevent contractures [33]. Implementation of planned seating and positioning strategies by nurses and carers throughout the day and night is crucial to management of spasticity and prevention of its complications.

Physiotherapists and occupational therapists should complete their assessments over a period of time and in conjunction with other members of the patient's multidisciplinary team, including the patient's carers and nurses, in order to optimise management strategies. It is essential that such strategies attain the correct balance between movement and positioning and continuity of care, particularly across the interfaces of primary and secondary care, involving community rehabilitation teams and care agencies, facilitates the appropriate choice and timing of any management intervention [21].

Pharmacological Treatments

Drug therapy is generally initiated at low dosages and then gradually increased in an attempt to avoid adverse effects [21, 28]. Optimal therapy is the lowest effective dosage. Drug treatments should be contemplated early in severe cases of spasticity, where secondary problems often develop and combination therapy using oral medications and focal injections of botulinum toxin or other chemodenervating agents may allow for the best control of spasticity with the least side effects [34].

Oral Medication for Treatment of Global Spasticity

Baclofen, tizanidine, diazepam (that act centrally) and dantrolene (that acts peripherally on skeletal muscle) are the most widely used drugs in patients with global spasticity. Other agents such as gabapentin, clonazepam, clonidine, and cyproheptadine have also been used for the management of spasticity, but in fewer patients.

Baclofen

This is the most widely used anti-spastic drug whose clinical benefits mainly relate to reducing muscle spasms and hyper-reflexia [35]. Baclofen is structurally similar to the inhibitory neurotransmitter GABA (gamma-aminobutyric acid) and increases inhibition both pre-synaptically and post-synaptically by selectively binding to GABA-B receptors [36]. The common starting dose is 5 mg three times daily that then may be titrated up to a maximum daily dose of 60–100 mg in divided doses. Side effects are predominantly from central depressant properties including sedation, ataxia, weakness, and fatigue [35]. Tolerance to the medication may develop, and baclofen must be slowly weaned to prevent withdrawal effects such as seizures, hallucinations, and increased spasticity. Limitations of baclofen use include its lowering of seizure threshold and patients' intolerance of side effects at higher doses.

Tizanidine

This is an imidazoline central alpha-adrenoceptor agonist that has been confirmed to be a useful anti-spastic agent. It is a short-acting drug with dose-dependent linear pharmacokinetics and larger inter-patient variability compared with other anti-spastic agents [37]. Patients report less muscle weakness from tizanidine than baclofen or diazepam, but side effects include drowsiness, fatiguability, dizziness, dry mouth, and gastrointestinal disturbance [37–39]. There is a small incidence of abnormal liver function tests and these should be monitored at intervals during therapy [40]. Tizanidine may be combined with baclofen, presenting the opportunity to reduce the dosage of both drugs, but additive adverse effects, including sedation, may occur.

Diazepam

This was one of the first anti-spastic agents, but in view of its potential to cause significant fatigue and drowsiness, is only recommended for relieving painful nocturnal spasms [35, 41]. Midazolam, another benzodiazepine, is sometimes useful to help distinguish between patients with active spasticity and contractures.

Dantrolene

This is a useful anti-spastic agent that has a similar range of side effects to baclofen, but is less likely than the other agents to cause drowsiness, confusion, and other central effects because of its mechanism of action. Dantrolene has been shown to decrease muscle tone, clonus, and muscle spasm, but since its action is not selective for spastic muscles, it may cause generalised weakness, including weakness of the respiratory muscles [36]. It can also cause hepatitis, and so periodic monitoring of liver function tests is advised [42, 43].

Gabapentin

Gabapentin interacts with voltage-sensitive calcium channels in cortical neurons and increases the synaptic concentration of GABA, enhances GABA responses at non-synaptic sites in neuronal tissues, and reduces the release of mono-amine neurotransmitters. It is generally used as an anticonvulsant and analgesic for neuropathic pain. The most common side effects include dizziness, fatigue, drowsiness, weight gain, and peripheral oedema. In a randomised, double-blind, placebo-controlled study involving 30 patients with upper motor neurone spasticity secondary to a pyramidal tract lesion, Formica et al. showed Gabapentin in doses of 2,700–3,600 mg/day to provide significant improvement in Ashworth scores but no change in spasm frequency [44].

Focal Treatments, Intrathecal Baclofen, and Surgical Techniques

Patients who are unresponsive or intolerant to conservative spasticity treatments may benefit from referral to a specialist service for consideration of other therapeutic modalities [45].

Focal Treatments

Spasticity is often focal in origin and it may be more appropriate only to reduce spasticity in the affected muscles [34]. Focal pharmacological treatments should be combined with non-pharmacological therapies, including stretching programmes and physiotherapy assessments, in order to obtain optimal benefits. The aim is to improve function, mobility, and dexterity; ease pain and decrease spasms; allow orthotic wearing; and improve body image in terms of cosmesis.

Botulinum Toxin

Botulinum toxin is a powerful neurotoxin produced by Clostridium botulinum. There are seven distinct subtypes (A–G) and the most commonly used one in spasticity is Type A botulinum toxin. Botulinum toxin prevents presynaptic release of acetylcholine resulting in neuromuscular blockade. The multicentre, randomised, controlled BoTULS (Botulinum Toxin for the Upper Limb after Stroke) trial, involving 333 stroke patients with upper limb spasticity and reduced arm function, demonstrated botulinum toxin injection (in addition to a 4-week therapy programme) to improve muscle tone, upper limb strength, basic arm functional tasks of hand hygiene and facilitation of dressing, and pain, compared with therapy alone [46]. There was no significant difference, however, in achievement of improved arm

function between groups using the Action Research Arm Test at 1 month as a primary outcome measure. The ongoing PrOMBIS (Predicting Outcome and Measuring benefit from Botulinum therapy In Stroke) trial may provide more information regarding the potential for botulinum toxin to improve the functional ability of stroke patients with spasticity.

Botulinum toxin is injected intramuscularly with an onset of action within 12 h and its clinical effect, in terms of reduction in spasticity, is visible over a course of 4–7 days from the time of injection. The total duration of the effect lasts for approximately 10–12 weeks with the maximal effect seen at 3–4 weeks. Repeat injections may be necessary but are not recommended within 3 months. Some patients may become resistant to botulinum toxin as a result of antibody formation [11]. Side effects are uncommon with licensed and recommended doses, but induction of excessive weakness of the injected muscle, pain, flu-like symptoms, and rash exist. If larger doses are employed, neuritis, dysphagia, and respiratory compromise may occur [11, 47].

Post-botulinum toxin injection, it is important for the multidisciplinary team to review the ongoing care of the injected muscle, the achievement of goals, and the measurement of functional outcomes with the patient and their carers. Splinting and orthosis usage, in addition to botulinum toxin, provides prolonged stretch to the muscle injected and aims to improve muscle length, correct and prevent contractures, and maximise function. Pre-existing splints should be reviewed and, if required, new ones applied 7–14 days post-injection when maximal clinical effects of botulinum toxin are clinically apparent [11]. At the same time, it is important to make sure that the weakened muscles are not overstretched, as that can end up in tearing of the stiffened muscle fibres, resulting in intramuscular haematoma. In addition, ongoing patient education on stretching regimens and guidance on participating in activities is useful. Functional electrical stimulation may also be combined with botulinum toxin therapy to improve symptoms and function [48, 49].

Phenol Nerve Block

Phenol (carbolic acid), in concentrations more than 3 %, acts as a neurolytic agent and it is this neurolytic effect that is responsible for reducing spastic muscle innervations, and hence spasticity. In addition, phenol has a local muscle relaxant property and patients experience a transient muscle relaxation within an hour of phenol nerve blocks. Phenol nerve blocks produce a dramatic and instant effect, but the technique may be time-consuming, provide variable duration of symptomatic relief, and there is a risk of painful dysaesthesia and neural damage following the procedure [29, 50, 51].

Phenol injections are generally used for regional lower limb spasticity in individuals who are intolerant of systemic muscle relaxant therapies. They are also occasionally used for large muscles of the lower limbs (e.g. the quadriceps and hamstrings) that may require doses of botulinum toxin too high to be safely used for the individual. Side effects are not very common, but include those local to the

injection site such as erythema, pain, discomfort, and sometimes local haematoma, infection, abscess formation, muscle fibrosis, or nerve causalgia. Very rare side effects include vascular injury and systemic side effects of arrhythmia, pulmonary fibrosis, confusion, and renal impairment [52].

Intrathecal Baclofen Therapy

This consists of long-term delivery of baclofen to the intrathecal space from a programmable pump surgically placed just below the skin in the abdomen [35]. Meythaler et al. showed this to be an effective treatment modality in a randomised study of 21 stroke patients with "intractable" spasticity for more than 6 months with significant reductions in Ashworth scores [53]. The ongoing multicentre, randomised, controlled SISTERS trial of intrathecal baclofen versus best medical treatment in patients with generalised spasticity post-stroke, who have not reached their therapy goals with currently available treatment options, will help to identify further benefits of intrathecal baclofen for patients with post-stroke severe spasticity. Side effects are less common when baclofen is administered intrathecally, because the drug does not circulate throughout the body, but it may still be associated with drowsiness, nausea, and headache.

Surgery

This is the last option to treat spasticity, and surgical interventions can be divided into peripheral ablative procedures, such as rhizotomy or peripheral neurectomy, more central ablative procedures such as cordectomy, myelotomy, and stereotactic procedures [54], or procedures like tendon release, lengthening and transfer, tenotomy and myotomy that require referral to orthopaedic surgeons. These procedures are considered in individuals who are refractory to medical treatments, and the benefits of surgery always need to be weighed carefully against its risks. Bollens et al. recently completed a randomised controlled trial of selective neurotomy versus botulinum toxin in 16 patients with spastic equinovarus of the foot after stroke and showed tibial neurotomy to produce a higher reduction in ankle stiffness, but no difference in ankle kinematics during gait, muscle weakening, or patient activity or quality of life [55].

Conclusion

Assessment for spasticity needs to be individualised towards a person's needs. Care should be managed in a multidisciplinary format allowing for treatment options to be considered and chosen regularly. Effective management should be seamless, incorporating continuous education, support, and treatment in both primary and

secondary care, and involvement of rehabilitation teams and care agencies in the community. The effects of spasticity are likely to change over time, and therefore continuous assessment and review is integral to the successful management of spasticity.

Patient Questions

Q. How should post-stroke spasticity best be managed?

A. The management of spasticity requires a multidisciplinary approach incorporating nurses, physicians, physiotherapists, and occupational therapists working together to provide a variety of treatments tailored to the needs of the individual patient. There should be arrangements for targeted therapy and this should include a programme of stretching and physical therapy intervention. Therapists, along with carers and relatives, help in planning 24-h postural management programme.

Q. Will botulinum toxin injection to my "spastic" fingers make them work again?

A. No. If the multidisciplinary team consider it to be appropriate treatment, its aim is to relieve the increased stiffness and associated symptoms, but it will not have an effect on the already decreased power. In fact, the muscles into which the injection is undertaken may have less apparent use by virtue of the botulinum toxin induction of decreased tone. It is important to have ongoing therapy-directed stretching exercises in addition to optimisation of splinting to maintain muscle and soft tissue length across joints.

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