Management of spasticity revisited

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Abstract

Spasticity is common after stroke and other neurological conditions and causes considerable limitations of movement, activities of daily living and participation. Interaction with other components of the upper motor neurone syndrome (UMNS) and the heterogeneity of patients’ presentations together with limited tools for outcome measurement have hampered the production of randomised controlled trial data for management strategies. Specialist multi-disciplinary goal-centered management programmes are the mainstay of treatment. Pharmacological therapies have limited effect, and physical and positional management are crucial. Targeted intramuscular botulinum toxin injection is now the most popular pharmacological treatment. Intrathecal therapies also play a lesser role. A team approach and holistic assessment are essential to beneficial outcomes.

Keywords: spasticity, upper motor neurone syndrome, muscle stiffness, antispasmodics

Introduction

Spasticity is a neurological condition affecting movement which can cause muscle stiffness, pain, loss of joint range and loss of function. Difficulties for patients include problems maintaining personal hygiene and independent dressing. Comfortable seating and maintenance of adequate posture are difficult. Reduced arm function affects activities of daily living and lower limb spasticity causes compromised gait patterns, with loss of mobility and an increased risk of falls. There may be decreased mood and social interactions, resulting in an increased carer burden and a risk to family relationships.

For this review, relevant papers were identified using medline and embase. Despite the impact of the condition, the literature is limited with regard to the evidence for optimal management.

Definitions and epidemiology

It is difficult to define spasticity, as it is not seen in isolation, but occurs as part of a spectrum of symptoms in UMNS diseases. This includes muscle weakness, spasms, clonus and reduced postural responses. The interaction of these components and the complex pathophysiology of muscle tone disturbance are incompletely understood. The ability to distinguish between dynamic elements contributing to muscle stiffness, which are potentially amenable to treatment and those elements have become fixed is clinically challenging. This is reflected in descriptive definitions of the condition.

Lance’s 30-year-old landmark definition of ‘velocity-dependant intrinsic resistance to passive movement of a limb in people with upper motor neurone syndrome’ [1] has been refined by the EU spasm expert group as ‘disordered sensorimotor control resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary activation of muscles’ [2]. This definition takes into account the contribution of visco-elastic properties of soft tissue to limb stiffness (particularly pertinent in older people [3], as tissue elasticity is lost with age), and the role of proprioceptive and cutaneous neural pathways.

The exact prevalence of spasticity is not known. At least one-third of stroke survivors are affected [4] and one recent study has reported contracture development in 50% 6 months after stroke [5].

Pathophysiology

The neural mechanisms underlying normal muscle tone are complex and the contribution of nervous system components to the development of spasticity remains ill-understood [6]. Spasticity can be thought of as a breakdown in control of the spinal stretch reflex mechanisms (involving Ia and II sensory afferents and alpha motor neurones) that cause inappropriate muscle over-activity, limiting the movement of
the joints and limbs through a full range of movement. Interactions between the ‘neural’ components of these mechanisms which cause potentially reversible muscle tightness and fixed ‘non-neuronal’ stiffening and shortening of muscle fibres and supporting soft tissues form the basis of resistance to movement. When an UMN lesion occurs, the balance between these two components changes, disturbing normal resting tone and preventing muscle lengthening causing loss of joint range. The ageing process produces changes in muscle bulk (sarcopenia), strength and architecture, as well as increased tendon and soft tissue stiffening, meaning older people may be more susceptible to the shortening process and subsequent contracture.

**Clinical presentations**

There are no large studies of the natural history of spasticity and contracture development, but permanent loss of joint range has been known to occur within 3–6 weeks after stroke [7] and acute brain injury [8]. It is therefore important to identify and treat spasticity early and some small studies have shown a benefit in a proactive approach using injections of botulinum toxin [9].

In hemiplegia, the lower limb pattern is planter flexion and inversion at the ankle with hamstring tightness limiting range of movement at the knee adductor spasticity, causing the knees to be pulled together, has implications for personal hygiene, sexual function and seating. Spasticity can cause painful spasms, and the nature and pattern of these movements needs to be fully explored to establish positional trigger points which may be potential targets for physical therapy interventions or intramuscular botulinum toxin injection.

**Management**

A thorough specialist multi-disciplinary assessment, including patient and carer education and physical management, is always the first step in spasticity management [9]. The aim is to establish a goal-based treatment plan with patient and carer involvement.

**Principles of assessment**

A holistic assessment should be carried out to form a list of problems caused by spasticity and the effects on function [10].

Spasticity does not always need treatment, particularly if there is no realistic proposition of functional gain. For example, where concurrent upper limb weakness limits hand use even if tone can be reduced, use of botulinum toxin to forearm flexors is only indicated if hand hygiene is compromised.

**History and examination**

The effects of pain, muscle and joint stiffness, spasms and clonus need to be explored in the context of each patient’s individual health, family and social circumstances.

Effect on sleep patterns and the ability to do self-care tasks, including bladder and bowel management and mobility, should be explored. Previous pharmacological and physical therapies and side effects experienced should also be noted. A history of falls should be taken and fractures actively looked for and treated.

**Assessment for aggravating factors**

It is important to identify and remove any irritant which may lead to increase in tone (Table 1).

Physical examination should include full documentation of the pattern of neurology and consideration as to whether this fits with the history. The presence of soft tissue shortening and limitations of joint range of movement should also be noted. Testing of power in the affected limb is important in identifying whether spasticity treatment will be likely to improve function. Further acute investigations (such as CT scan to exclude further stroke or hydrocephalus) may be required if there is a change in neurological signs. Skin inspection is important, both to identify triggering factors and to protect hygiene in compromised skin creases (for example, claw hand and elbow flexor pattern after stroke). Mobile patients should have an examination of barefoot gait.

**Outcome measures**

Standardised measures before and after treatment are a crucial component of the assessment process and should ideally include measures of impairment, activity limitation and participation, as well as completion of the patient’s own goals which can be formally measured using goal attainment scaling [11].

**Impairment measures**

Spasticity is most often measured using the Modified Ashworth scale or Tardieu scales, although both have drawbacks. Ashworth scales are quick to administer but have limited validity and reliability, whereas the Tardieu scale is too complex and time-consuming to use in day-to-day clinical practice [12].

Goniometry is used to measure the angle of range of movement across a joint, or tape measurements can record

<table>
<thead>
<tr>
<th>Table 1. Factors which aggravate spasticity</th>
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<tbody>
<tr>
<td><strong>Skin lesions</strong></td>
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<tr>
<td>Pressure sores</td>
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<tr>
<td>Local skin infection or irritation</td>
</tr>
<tr>
<td>Ingrowing toe nails</td>
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<tr>
<td><strong>Urinary tract dysfunction</strong></td>
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<td>Infection</td>
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<td>Urinary tract stones</td>
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<td>Incomplete bladder emptying/retention</td>
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<td><strong>Gastro-intestinal dysfunction</strong></td>
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<td>Constipation</td>
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<td>Diarrhoea</td>
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<tr>
<td>Overflow incontinence</td>
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<tr>
<td><strong>CNS</strong></td>
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<tr>
<td>Cerebrovascular disease, further stroke/TIA</td>
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<tr>
<td>Hydrocephalus</td>
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<td>Syrinx development</td>
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<tr>
<td><strong>Systemic illness</strong></td>
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<tr>
<td>Generalised infection</td>
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<td>Deep Venous Thrombosis</td>
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changes. Visual analogue scales for pain and stiffness, including vertical or symbol-based scales which may be more appropriate for those with communication difficulties or cognitive impairment, are also useful.

**Activity limitation**

Measures may be used which reflect the pattern of limb involvement. The Frenchay arm test, Action research arm test or nine hole PEG test are suggested tools for assessing upper limb spasticity [10]. The Arm Activity Measure [10] is a useful practical tool which reflects daily activities and can be easily used in clinic. Simple standard lower limb measures include the 10-m or 6-min walking distance, or video recording of gait. Detailed gait analysis if available may be appropriate for more complex cases [10].

**Participation restriction**

Formal measures of participation and quality of life have not been validated specifically for spasticity due to the heterogeneity of individualised goals and the patient populations. Achievement of specific goals relevant to the patient provide a measure of success—for example, being able to sit comfortably in a wheelchair for longer, allowing social outings or maintaining independent transfers.

**Management**

**Physical therapies**

Before considering pharmacological agents, the mainstay of spasticity management depends on adequate 24-h positioning and consideration of trunk, head and limb posture [13].

The contribution of compensatory muscle actions in combating postural weakness leading to global increases in tone is now well recognised [14]. The concept of restoring 'good' normal movement patterns after neurological injury versus quick mobilisation using abnormal muscle patterns which increase tone further and potentially limit long-term rehabilitation outcomes is a challenge to neurophysiotherapists aiming to meet early hospital discharge targets.

The incorporation of an active or passive movement programme into the patient’s daily care package is crucial to the maintenance of range of joint movement preventing contracture.

Good posture may be maintained in bed using a range of positioning tools including ‘TBar’ or trunk wedge to aid trunk stability and combat adductor spasticity (Figure 1). The principles of adequate pelvic and trunk support, allowing lowering of resting tone, also apply to providing adequate head and neck support whilst sitting. Satisfactory seating systems may include using a tilt in space wheelchair and suitable head rest or cervical collar (Figure 2). These postural aids allow trunk stability, alleviating compensatory limb spasticity and maximising motor control.

Standing (using a standing frame) or weight-bearing on a tilt table (with blood pressure monitoring) allows stretch of the ankle joint complex using body weight and helps to combat contracture development.

Thermoplastic splinting and serial plaster casting are both used as adjuncts to passive stretching to maintain range of movement, but there is insufficient high-quality evidence to support any specific method [15]. The potential for skin pressure damage may be a particular issue for older people. A combined approach using casting with botulinum toxin injections to specific muscle groups has makes clinical sense and has shown some benefit in post-stroke spasticity [16].

**Pharmacological treatments**

The evidence base for benefits of drug treatment for spasticity has not had significant expansion in terms of high-quality randomised controlled trials of particular relevance to functional outcomes since Professor Mike Barnes’ review in this journal 15 years ago [17]. The rationale for the use of oral medication relies on practical expert experience.

**Oral medication**

Baclofen is the commonest systemic agent used to treat spasticity. Side effects occur in half of all cases and are more common in older people and those with cognitive impairment. Drowsiness, weakness, parasthesia, nausea and vomiting, and dry mouth limit its usefulness. More cautious commencement and a lower dosage are required. Lowering of the seizure threshold also occurs. Sudden withdrawal may precipitate seizures, confusion, anxiety and hallucinations.

Baclofen’s mechanism of action is incompletely understood, but it works at the spinal level to inhibit monosynaptic and polysynaptic reflexes. Functional benefit has not been studied in detail. In stroke patients, a benefit in Ashworth scores was shown but no change on the incapacity status scale [18]. Baclofen’s half life is 3–4 h, requiring dosing three times daily to retain effectiveness. The recommended maximum dose is 120 mg per day but decreased dosage is required in those with renal impairment.

Tizanidine acts as a potent selective alpha two adrenergic receptor agonist which acts to reduce stretch reflexes and co-contraction. Studies in stroke patients have shown a reduction in tone and spasms compared with placebo and equal to baclofen [19]. Side effects are similar to baclofen and more likely to affect older people. Liver function tests need to be monitored as fulminant hepatic necrosis can occur. Like baclofen, tizanidine has a short half-life, requiring three or four times daily dosing to a maximum of 36 mg.

Dantrolene works directly on skeletal muscle to reducing calcium ion release to reduce muscle fibre excitation. Moderate beneficial effects are seen in stroke and it may be used as an adjunct to centrally acting agents [20]. No studies have demonstrated improvement in function. The risk of hepatotoxicity requires on-going monitoring of liver function tests and severely limits its use.
Benzodiazepines act to reduce spasticity through modulation of GABAergic transmission. Diazepam and clonazepam are the most common agents, but their usefulness is limited by side effects [21]. Clonazepam may be particularly helpful in low dose (0.25–1 mg) for nocturnal spasm.

Gabapentin, a GABAergic drug-modulating intracellular calcium channels, was introduced as an anti-epilepsy drug in the 1990s and found to be beneficial for neuropathic pain. Randomised-controlled trial data support a beneficial effect [22]. Gabapentin is generally well tolerated. Side effects of drowsiness, somnolence and dizziness may be avoided by using a starting dose of 100 mg a day, gradually increasing to three times daily and increasing in 100 mg increments to a maximum dose of 2400 mg.

Cannabis has been noted to relieve pain and spasticity, particularly in MS and spinal cord injury for many years. In the UK an oromucosal spray—‘Sativex’ (Delta 9-tetrahydrocannabinol THC) is now available, but reserved for patients with severe spasticity in MS, refractory to other treatments [23].

**Focal treatments**

Focal therapies have the advantage of targeting specific muscle groups or patterns of spasticity without the risk of systemic side effects such as drowsiness, cognitive impairment and
Botulinum toxins

Botulinum toxin is a powerful neurotoxin produced by *Clostridium botulinum*. There are seven distinct subtypes—Types A and B are used for medical purposes [24]. Acetyl choline release is blocked at the neuromuscular junction, causing temporary paralysis and allowing stretching of muscle fibres. Three Types of botulinum Type A (Dysport®<sup>®</sup>, Botox®<sup>®</sup> and Xeomin<sup>®</sup>) and one of Type B (Neurobloc<sup>®</sup>) are currently available in the UK. These drugs are not interchangeable, having different dosing ranges. Adverse effects are related to anticholinergic effects and local injection site irritation. Swallowing muscles may be affected and disabling generalised weakness may occur rarely, especially in the frail with small muscle bulk, where reduced dosage should be considered. Side effects are transient, as are the treatment effects, which last for 3–4 months. An advantage over oral agents is the lack of systemic side effects, in particular generalised CNS effects which may limit active movement and cognition.

The pattern of injections depends on the degree and dynamic nature of spasticity. The muscle groups targeted and dosages depend on the outcome of multi-disciplinary assessment focused on the patient’s functional goals (Table 2). Identification of muscle groups is carried out using anatomical landmarks and palpation, or by using EMG guidance [25]. There are no randomised-controlled trials comparing these methods. Use of ultrasound is becoming more common, but whether this improves outcomes is not yet clear.

After stroke, there is evidence that botulinum toxin reduces muscle tone, but evidence for functional gain is limited. The recent BoTULs trial [26] showed significant improvement in Ashworth scores (as well as improved facilitation of dressing and hand hygiene) in stroke patients, but no benefit in active upper limb function. The methodology of available trials is diverse and differs from current clinical practice. Variable doses and different preparations of botulinum toxin are used and consideration of the need for repeat injections as well as how to measure meaningful improvement is some of the challenges in the literature. A Cochrane systematic review of botulinum toxin for adult spasticity after stroke is on-going [27].

For patients with lower limb spasticity after stroke, there is an even smaller body of evidence which suggests there is improvement of gait speed. Combination treatments with functional electrical stimulation and targeted botulinum injections have not yet shown convincing benefit.

**Chemical neurolysis**

Local injection of ethanol or phenol results in irreversible destruction of neural tissue by protein coagulation. The procedure is carried out percutaneously, using EMG for nerve identification [28]. Targets include the tibial nerve (correction of equinus deformity), obturator nerve (adductor spasticity aiding personal hygiene and catheter care) and the musculo-cutaneous nerve to reduce elbow spasticity. The procedure has largely been superseded by botulinum toxin injection where botulinum is available.

**Intrathecal therapies**

These interventions are usually reserved for severe widespread spasticity that does not respond to oral or focal treatments.

**Intrathecal baclofen**

Intrathecal baclofen directly acts on the GABA receptors in the lumbar spinal cord where a high concentration of receptors allows small doses to be used to good effect without systemic side effects. Originally used in spinal injury patients, it has also been successful in treating MS and hemiplegia, including post-stroke spasticity [29]. Patients are selected after thorough multi-disciplinary assessment and inpatient trial via lumbar puncture. Regular outpatient review by the specialist team is required for pump refills. Pump failure or catheter fracture, kinking or displacement may result in under-dosage and withdrawal syndrome which is potentially life-threatening.

**Intrathecal phenol**

This therapy is appropriate for a small number of patients with severe painful spasticity and may be a highly effective treatment [30]. Application is limited because it is neuro-destructive and not reversible. Painful parasthesia, incontinence and loss of sexual function may occur. The aim is to improve seating, hygiene and personal care and pain.

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**Table 2.** Muscle pattern for botulinum toxin injections in the upper limb for post-stroke spasticity [10].

<table>
<thead>
<tr>
<th>Treatment goals</th>
<th>Muscle</th>
<th>Rationale for injection</th>
<th>Pectoralis major</th>
<th>Reduces adduction and medial rotation of arm at shoulder/upper humerus, allowing arm to move away from body</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aid hygiene in palmar and elbow creases</td>
<td>Biceps brachii</td>
<td>Reduces flexion and supination at the elbow</td>
<td>Breachialdialis brachialis</td>
<td></td>
</tr>
<tr>
<td>Allow movement of arm for dressing</td>
<td>Flexor carpi radialis</td>
<td>Reduces flexion at the wrist and elbow</td>
<td>Breachialdiais radialis</td>
<td></td>
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<tr>
<td>Reduce pain</td>
<td>Flexor carpi ulnaris</td>
<td>Reduces flexion at the wrist and adduction of the hand</td>
<td>Breachialdiais ulnaris</td>
<td></td>
</tr>
<tr>
<td>Reduce associated reaction to aid balance when walking</td>
<td>Flexor digitorum superficialis</td>
<td>Reduces flexion of PIP and MCP joints</td>
<td>Breachialdiais profundus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Flexor digitorum profundus</td>
<td>Reduces flexion of all finger joints</td>
<td></td>
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</tr>
</tbody>
</table>
Conclusion

Spasticity is common after neurological injury and causes limitations to function and quality of life as well as a range of distressing symptoms. Physical measures to correct tonal changes are pivotal in management and aggravating factors should be actively sought and treated. There is a lack of RCT evidence for pharmaceutical interventions, but the usefulness of individual agents and botulinum toxin in particular in managing the problems created by increased tone is becoming clearer. A specialist multi-disciplinary medical and therapy approach to management with clear goals is the mainstay of all spasticity management programmes.

Key points

- Spasticity management should be a proactive multidisciplinary process as postural and physical interventions are the mainstay of treatment.
- There is limited evidence for the effectiveness of oral antispasmodic agents.
- There is increasing evidence for the effectiveness of intramuscular botulinum toxin type A for focal spasticity after stroke.

Conflict of interest

Laura Graham has received sponsorship for the organising and attending postgraduate teaching from the manufacturers of all 3 Botulinum Type A products and uses these products in clinical practice.

Dedication

This article is dedicated to the memory of Dr. A. B. Field, Rehabilitation Consultant and previously doctor in Elderly Care.

References


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