

REVIEW

Management of spasticity

MICHAEL P. BARNES

Academic Unit of Neurological Rehabilitation, University of Newcastle upon Tyne, Hunters Moor Regional Rehabilitation Centre, Newcastle upon Tyne NE2 4NR, UK. Fax: (+33) 191 219 5665; E-mail: m.p.barnes@ncl.ac.uk

Keywords: *physiotherapy, rehabilitation, spasticity*

Introduction

Spasticity is a major challenge to the rehabilitation team. Spasticity can prevent or hamper function, cause pain, disturb sleep, cause unnecessary complications and present major difficulties for care workers. This article reviews the variety of options available for the clinical management of spasticity. The need for clear treatment goals and robust outcome measures is emphasized. The initial management should focus on the alleviation of external exacerbating causes before specific treatment is considered. Physiotherapy is vital for correct positioning, seating, use of orthoses, splints and casts and for other anti-spastic measures such as use of heat and cold and electrical stimulation. The use of oral medication is discussed. Peripheral nerve blocks and botulinum toxin are two local treatments which are proving very useful and are under-used and undervalued. In more severe cases intrathecal medication can be helpful. Surgical procedures such as rhizotomy and orthopaedic corrections may sometimes be necessary, but usually only for the most severe cases or for those who have been poorly managed in the earlier stages. Overall, the clinical management of spasticity often depends on a variety of different approaches, necessitating the involvement of a comprehensive rehabilitation team.

Definitions of spasticity

Most physicians and therapists working with physically disabled people probably feel that they can recognize spasticity when they see or feel it. However, defining it is much more difficult. Spasticity has been narrowly defined as a motor disorder characterized by velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks [1]. This narrow definition, however, does not do justice to the additional symptoms that are often associated with increased muscle tone. Spasticity is usually accompanied by permanent or at least intermittent voluntary muscle activation, resulting in weakness and clumsiness of voluntary movements. The definition can be

broadened further to include other positive characteristics of the upper motor neuron syndrome such as flexor or extensor spasms and the 'clasp-knife' phenomenon, exaggerated cutaneous reflexes and contractures [2]. Yet even these broader definitions do not give any flavour of the bewildering variety of problems that can occur in different individuals and even in the same individual at the same time. The extent and type of spasticity can fluctuate widely according to position, fatigue, stress and drugs. One limb may have one pattern of spasticity whilst another may have a different pattern.

Goals and outcomes

The treatment of spasticity, like all rehabilitation processes, must start with the establishment of specific achievable goals and a carefully planned strategy to achieve those goals. The first question is: is it necessary to treat the spasticity at all? Spasticity can be useful for the individual. For example, spasticity in a leg may serve as a brace to support the individual's weight for transferring or walking. Some consideration also needs to be given to the side effects of some treatments, particularly the weakness and fatigue induced by anti-spastic medication.

In general terms, there are three potential aims of treatment—to improve function, to reduce the risk of unnecessary complication and to alleviate pain. Occasionally, a justifiable aim is not specifically to help the patient, who may not perceive any problems with spasticity, but to make nursing easier for the main carers and to assist with the maintenance of hygiene, dressing and transferring.

Once a goal has been established, an outcome measure should be chosen that allows goal attainment and progress to be monitored. Most measures of spasticity are measures of impairment and not measures of disability or handicap. The clinical goal should have an appropriate clinical outcome measure. For example, if the aim of treatment is to reduce pain, then little is achieved by monitoring muscle spasticity and a

pain scale should be used. There are a number of motor-orientated disability scores that can be used to monitor functional effects of anti-spastic treatment programmes, from the broad-based Barthel index [3] to the more comprehensive Functional Independence Measure [4] to more specific walking or hand function tests [5].

Occasionally, a specific measure of spasticity is useful, particularly in a research environment. Unfortunately there are very few properly validated and reliable clinical assessments. The modified Ashworth scale [6] is the best known but there have been only two studies of its reliability [6, 7]. The original paper by Bohannon and Smith [6] was only concerned with the elbow joint; the second paper by Sloan and co-workers [7] confirmed reliability for the elbow, but showed a rather poor inter-rater reliability for the knee flexors. The validity and reliability of this scale for other joints are not known. A variety of other assessment procedures are available. These tend to be either biomechanical or neurophysiological. In the former category, attempts have been made to use mechanical techniques to measure torque/angle relationships with spastic joints during passive flexion and extension [8-12]. A valid technique for the measurement of spasticity of the knee is the pendulum test [13], which is a more sensitive measure of spasticity than the Ashworth scale. The pendulum test involves the appropriate joint, such as the knee, being held in extension and released, with spasticity measured as the rate of decay of oscillation of the limb. Obviously this is a difficult test to perform in smaller joints or in severe spasticity. Quantitative neurophysiological measurements of spasticity have largely been developed as research tools. Measurements mainly focus on characteristics of the H reflex, particularly suppression of the reflex by vibration and by reciprocal inhibition. These techniques are well reviewed by Shahani and Cross [14].

The problem with biomechanical or neurophysiological measurement of spasticity is that such techniques are cumbersome and impractical in a clinical setting. They can also provide a false estimation of the functional effects of spasticity, as measurements are usually taken in a static rather than dynamic situation. Practising clinicians, for the moment, will have to rely on the modified Ashworth scale in combination with practical, simple and preferably quick measures of disability related to the goals of treatment.

An approach to treatment

Alleviation and exacerbating factors

Spasticity should be seen as a sign that has a variety of underlying causes. This is particularly important for people who are comatose, cognitively impaired or

unable to communicate. Common causes for the onset or exacerbation of spasticity are urinary retention or infection, severe constipation, skin irritation such as pressure sores or increased sensory stimuli from external causes such as ill-fitting orthotic appliances and catheter leg bags. Occasionally, exacerbation of spasticity can indicate an underlying abdominal emergency or lower limb fracture, particularly in those who are unable to appreciate pain and not able to localize their problem.

Positioning and seating

Correct positioning, certainly for the immobile patient, is an important aspect of management. Incorrect positioning in bed, particularly in the early stages after stroke or brain injury, is a major cause of unnecessary spasticity. The supine position easily exacerbate extensor spasm by facilitation of the tonic labyrinthine supine reflex [15]. Similarly, a number of patients exhibit an asymmetric tonic neck reflex after brain injury which, in the supine position, will encourage a windswept posture characterized by asymmetric position of the pelvis, with one hip assuming a flexed position in abduction and external rotation whilst the other assumes an adducted and internally rotated posture. This is a common cause of later orthopaedic problems, particularly subluxation of the hip on the adducted side [16]. It is sometimes a matter of trial and error to find a posture that reduces spasticity. Side lying, sitting and standing can all be helpful in different circumstances, with the primary aims of reducing spasticity and producing stretch on the spastic muscles, as well as facilitating the use of antagonistic muscle groups [17]. Unfortunately, we do not know how long muscles need to be stretched in order to prevent contractures, although guidelines have been produced which suggest that each joint should be put through a full range of movement for at least 2 h in every 24 [18].

Proper seating is vital. The fundamental principle of seating is that the body should be contained in a balanced, symmetrical and stable posture which is both comfortable and maximizes function. There are many different types of seating system. All should have the ultimate aim of stabilization of the pelvis without lateral tilt or rotation, but with a slight anterior tilt so the spine adopts a normal lumbar lordosis, thoracic kyphosis and cervical lordosis. The hip should be maintained at an angle of slightly more than 90°, which is often facilitated by a seat cushion with a slight backward slope. Knees and ankles should be at 90°. In people with severe spasticity, this posture may not be possible or may require a variety of seating adjustments such as foot straps, knee blocks, adductor pommels, lumbar supports, lateral trunk supports and a variety of head and neck support systems. An adaptable and adjustable system is useful, particularly in people with

complicated disabilities and in those with varied and changeable conditions such as multiple sclerosis [19].

Splinting and casting

The application of splints and casts can prevent the formation of contractures in the spastic limb and serial casting can improve the range of movement in a joint that is already contracted — a new cast being applied every few days as the range improves [20–22]. It is not known whether this is purely a mechanical effect or whether splinting actually reduces spasticity. Unfortunately, there is no clear agreement on the most appropriate design nor the length of time a splint should be applied to give the desired effect. It is a field that requires much research [23, 24].

Physiotherapy

A physiotherapist has a vital role to play in the assessment and management of positioning, seating, splinting and casting, and the use of orthotic devices. However, do other physiotherapy techniques have an anti-spastic effect? Cold inhibits spastic muscles, but the effect is short-lived, perhaps outlasting the application of the cold by about half an hour [25]. Paradoxically, heat is also used for relaxation of a spastic muscle [26]. Unfortunately, the anti-spastic effect is relatively short-lived. Electrical stimulation has been used in some centres. Alfieri [27] found that 10 min of stimulation to the finger extensors produced a decrease in spasticity and an improved range of movement lasting for up to 3 h. Seib and colleagues [28] have recently found that surface electrical stimulation of the tibialis anterior muscle has an anti-spastic effect that lasts for up to 24 h. Potissk and colleagues [29] have confirmed similar findings with the use of a transcutaneous electrical nerve stimulation machine, but the effects only persisted for up to 45 min. Unfortunately, the role of electrical stimulation and other related techniques, such as electromyographic biofeedback and electrical vibration, is still not clear. None of these appear to have much long-term benefit but can have useful short-term effects, particularly when used as an adjunctive treatment in combination with other measures, such as the fitting of orthoses.

There are a number of different dynamic physiotherapy techniques, including the Bobath technique [30], proprioceptive neuromuscular facilitation [31], the Brunnstrom technique [32, 33] and the techniques proposed by Carr and Shepherd [34]. All claim an anti-spastic effect. There is, however, little evidence that any particular technique is better than the other for the management of spasticity. Larger-scale prospective and controlled studies or single case studies are urgently needed to address this question.

Oral medication

Oral anti-spastic medication can be helpful. Occasionally oral medication can be all that is required, particularly for milder cases. However, in more severe cases and for focal spasticity, the side effects, commonly drowsiness and weakness, can significantly restrict the usefulness of these drugs. The most widely used anti-spastic drug is baclofen, a GABA B receptor agonist that probably has other pre-synaptic inhibitory effects on the release of excitatory neurotransmitters such as glutamate, aspartate and substance P. It is a commonly used drug, but it is interesting to note that there is no convincing evidence of efficacy in spasticity of cerebral origin [35]. It should be administered in divided doses as it has a short half-life. A total daily dosage of up to 80 mg is standard. There appears to be little benefit in increasing the daily dose beyond this level. In addition to the usual problems of drowsiness and weakness it can occasionally induce hallucinations and even convulsions on discontinuation.

An alternative agent is dantrolene sodium, which has a peripheral mode of action via a direct effect on suppression of release of calcium ions from the sarcoplasmic reticulum of muscle, with consequent inhibition of excitation, contraction and coupling [36]. It is an effective anti-spastic agent if introduced in slow incremental stages up to a maximum of 400 mg daily in divided doses. As well as the usual problems of drowsiness, weakness and fatigue, there is the additional potential complication of impairment of liver function which necessitates monitoring liver function tests.

Diazepam is the earliest anti-spastic agent introduced and, whilst effective, it has serious problems of drowsiness and fatigue and is rarely of benefit to the patient [37]. A new agent, currently available in parts of Europe and soon to be available in the UK, is Tizanidine. It appears to take effect by preferential inhibition of poly-synaptic spinal excitatory pathways. The drug also has an effect on stimulation of α_2 receptors [38]. It is certainly comparable to baclofen as an anti-spastic agent and indeed may even be slightly superior [39, 40]. Whilst sedation, weakness and dry mouth can be problematic, Tizanidine may exhibit slightly fewer side effects than alternative agents.

A number of other anti-spastic agents have been the subject of small-scale studies. None has really stood the test of time or at least has not been subjected to larger-scale evaluation. Clonidine [41], glycine [42], threonine [43], tetrahydrocannabinol [44], divalproex-sodium [45] and orphenadine [46] are a few examples of drugs that probably do have an anti-spastic effect, but whose place in the overall management of spasticity has yet to be determined.

Nerve blocks

Khalili and co-workers [47] were the first to describe the use of phenol for selective peripheral nerve block,

by a percutaneous approach. A surface electrode is normally used to locate the peripheral nerve. A needle with insulated shaft is then used as an exploratory electrode and the needle tip manipulated until a good muscle contractile response is observed. At this stage the phenol is injected.

Any accessible peripheral nerve can be blocked in this manner. The obturator is probably the commonest and most accessible nerve and gives rise to very satisfactory reduction in adductor spasticity. The posterior tibial nerve is also a useful injection site for the relief of calf muscle spasticity and often abolishes troublesome clonus or facilitates the fitting of an ankle-foot orthosis. Hamstring spasticity can be alleviated by blocking the sciatic nerve or possibly the branches to the hamstring muscles themselves. It is less easy to block the many branches of the femoral nerve to the quadriceps muscle and equally difficult to locate the nerve supply to the iliopsoas for the relief of hip flexor spasticity, although some authors have described a paravertebral approach using radiological control. It is also possible to block the median and ulnar nerves as well as the musculo-cutaneous nerve for the relief of flexion spasticity at the elbow.

The side effects of this technique depend on whether a mixed motor-sensory nerve is blocked, with the consequent risk of dysaesthesiae, or whether the block is confined to motor nerve or motor end points. The most common problem is obviously loss of motor function, but if there is any doubt as to the potential functional effect of the nerve block then bupivacaine should be used before definitive block with phenol or alcohol. The duration of effect is variable, ranging from a few days to long-term—presumably dependent on the proximity of the needle to the nerve at the time of injection. The incidence of dysaesthesiae is highly variable and reported as 3–32%. Fortunately this complication usually consists of no more than a transient burning sensation lasting a few days, although occasionally there is more persistent dysaesthetic pain. Damage to the local structures is possible and local pain, oedema and infection have been reported, but fortunately only rarely. Overall, phenol and alcohol nerve blocks are useful for focal spasticity either as definitive procedures or as an adjunct to other techniques [48, 49].

Botulinum toxin

Botulinum toxin type A produces dose-related weakness of skeletal muscle by impairing the release of acetylcholine at the neuromuscular junction. It is now an established first-line treatment for focal dystonia. It is important to note that there are two commercial manufacturers of the toxin and that different units are used by each manufacturer. A number of studies have now shown that botulinum toxin is useful for the management of spasticity, although this is still an

unlicensed indication [40–53]. It is particularly helpful for spasticity in the leg adductors, calf muscles and the upper limb flexors.

The technique is simple: botulinum toxin is diluted in normal saline and is injected intramuscularly. In readily identifiable and palpable muscles no EMG identification of the muscle is normally required. The dose varies considerably depending on the bulk of the muscle and the number of muscles to be injected. An average dose in unilateral leg adductors is approximately 4–600 Dysport units or about 100–150 Allergan units.

Botulinum toxin injection is a safe and effective technique with very few side effects reported in the literature. Occasionally, weakness of the injected muscle or of neighbouring muscles can be a problem, but no general systemic weakness has been reported. There are some disadvantages to the technique, including the cost of the toxin, the need for repeat injections every 2–3 months and a risk, albeit a small one, of developing antibodies. Fortunately, there are seven types of botulinum toxin and at least two other types are now being developed for commercial use.

Overall, botulinum toxin is now an established adjunctive treatment for the management of focal spasticity in the adult. It probably also has an important role to play in the management of spasticity in children with cerebral palsy. It has been shown to improve gait and reduce the need for multiple surgical procedures [54].

Intrathecal techniques

There has been much interest in recent years in the use of intrathecal baclofen for the treatment of more resistant spasticity in the lower limbs. The technique was first described by Penn and Kroin in 1984 [55]. Surgical details of the technique can vary but would normally involve implantation of a sub-cutaneous pump to allow programmable intrathecal delivery of baclofen via a silastic catheter. The baclofen can either be administered in regular doses or by continuous infusion. A initial test dose is usually administered. The daily dose is adjusted according to clinical effect but would normally range from 50 to 1000 µg per day. Both short- and long-term efficacy have been confirmed in a number of studies [56, 57]. For example, a recent study demonstrated complete abolition of spasticity in 28 patients who were previously unresponsive to oral baclofen and other anti-spastic medications [58]. The follow-up period of this study was up to 2 years but averaged 8 months. The only significant complications were related to technical problems with the pump device and included one pump failure and two catheter replacements. There is a risk of the pump delivering an overdose of baclofen and a risk of respiratory depression. Tolerance is a possible but rare occurrence. A similar technique using intrathecal morphine injections has been described and this remains an alternative.

The first description of intrathecal injections for the relief of spasticity were made in 1959 by Kelly and Gautier-Smith [59] who used phenol and glycerine injection. Although this technique is now largely unnecessary, it is worth remembering for people with severe and resistant lower limb spasticity. It is effective but is likely to damage sacral nerves and should therefore be restricted to those who already have irreversible faecal and urinary incontinence. Sensation is also likely to be abolished with the consequent increased risk of pressure sores. However, for those already paraplegic and incontinent it can be a useful technique both to relieve spasticity and, more particularly, to relieve pain from spasticity [60].

Surgical and orthopaedic procedures

There is rarely a need to resort to surgical procedures for the management of spasticity, except in the occasional severe and resistant case and for the management of fixed contractures. A few techniques, however, are still useful and should be mentioned.

Anterior and posterior rhizotomy have been performed for many years for the treatment of severe and resistant spasticity [61, 62]. A more refined technique has been pioneered by Sindou and Jean Monod [63, 64]—a microsurgical lesion in the dorsal root entry zone ('DREZ-otomy'). The procedure can be used for both upper and lower limbs and the authors have reported consistently good results with minimal morbidity. The less invasive technique of percutaneous radiofrequency rhizotomy [65] has also been described as a relatively simple procedure with an apparently high rate of efficacy but with a small risk of recurrence. Although I have not had to resort to surgical advice for the management of spasticity for a number of years, these techniques should be borne in mind for the severe and resistant case.

Spinal cord and cerebellar stimulation have been reported to be effective in reducing spasticity but unfortunately the effect tends to be weak and relatively short-lived. The treatment is also time-consuming and expensive, with some risk of equipment failure and electrode movement. However, it is a technique that should be recalled if there is resistant pain as a result of spasticity [66].

Occasionally, surgical repositioning of joints and limbs can facilitate proper seating and ease positioning and the application of orthoses. One of the more common orthopaedic interventions is the use of one of the various Achilles tendon lengthening operations for a fixed equinus deformity, often with associated correction of a varus deformity. Hindfoot varus is normally caused by spasticity of the tibialis posterior whilst mid-foot varus is normally caused by tibialis anterior spasticity. Often equino-varus deformity needs the combination of Achilles tendon lengthening,

tibialis posterior lengthening and split anterior tibialis transfer procedures, sometimes in combination with lengthening of the toe flexors. Similar lengthening procedures can be undertaken on the hamstring muscles, although some surgeons prefer hamstring tenotomy and transposition [67]. Hip adduction deformities are relieved by obturator phenol nerve blocks or botulinum toxin injections but sometimes obturator neurectomy or adductor tenotomy can be carried out in more resistant cases [68]. Hip flexion deformities are a problem that is not readily amenable to non-invasive techniques and, although not always successful, ileopsoas procedures can be performed.

Surgery in the upper limb is not generally as successful as in the leg but various tenotomy or tendon-lengthening procedures are possible, including lengthening of the biceps and brachio-radialis, lengthening of the flexor carpi ulnaris and flexor carpi radialis tendons for wrist flexor spasticity and transfer of flexor pollicis longus to the radial side of the thumb for isolated thumb-in-palm deformities [69–71].

Conclusions

The management of spasticity is complex. Most individuals, even with quite severe spasticity, can be managed by a combination of physiotherapy and local nerve block or botulinum toxin injection, sometimes combined with relatively low-dose oral medication. The use of more advanced intrathecal and surgical techniques is rarely needed unless complications have arisen, often due to inappropriate early management. Spasticity requires the input of a full rehabilitation team, involving in particular a physician, orthotist and physiotherapist. Despite the complexities, the management of spasticity can often yield rewarding results and lead to major improvements in quality of life.

Key points

- Spasticity is a cause of major disability and handicap and is one of the commonest problems in those with neurological disease.
 - The initial management depends on identification of suitable and achievable goals and explanation of the aims to the patient and carer.
 - Physiotherapy is vital for correct positioning, seating and appropriate use of splints, casts and orthoses.
 - Medical management has centred on drug use but more recently focal treatment methods including phenol blocks and botulinum toxin have been introduced.
 - Surgery, including intrathecal baclofen, should now be the last resort in difficult, resistant cases.
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