

Spasticity

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Introduction

Spasticity is a physiological consequence of an insult to the brain or spinal cord, that can lead to life-threatening, disabling and costly consequences (Ward 2008) It is characterised by muscle overactivity which, if left untreated, may lead to muscle and soft tissue contracture.

Definition

Many attempts have been made to define spasticity and this shows the degree of its complexity. Lance's definition of 1980 is still relevant and is widely accepted. It states "Spasticity is a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflex, as one component of the upper motoneuron syndrome."

Young (1994) broadened Lance's definition to include other signs of upper motor neuron syndrome and described spasticity as "a motor disorder characterised by a velocity dependent increase in tonic stretch reflexes that results from abnormal intra-spinal processing of primary afferent input".

Applying this definition to patients in clinical settings has been difficult because upper motor neuron lesions produce an array of responses. The pattern depends on the age and onset of the lesion, its location and size. Patients with diffuse lesions produce, for instance, different characteristics to those with localised pathology and the speed of onset changes this again (Mayer 2002) More recently, the SPASM Consortium presented its findings at a meeting in Newcastle-upon-Tyne, UK in 2006. It has tried to adapt the accepted definition to a more practical base and make it more relevant to clinical practice and to clinical research (European Thematic Network to Develop Standardised Measures of Spasticity). Its definition is thus as follows.

"Assuming that all involuntary activity involves reflexes, spasticity is an intermittent or sustained involuntary hyperactivity of a skeletal muscle associated with an upper motor neurone lesion."

There are a number of different syndromes seen following an injury to the brain or spinal cord and the spasticity is only one of the positive features of the upper motor neuron syndrome (UMNS) which consist of both positive and negative features (O'Dwyer et al. 1996)

Different impairments of the UMNS can exist independently of the other impairments of the UMNS (Canning et al. 2000). The negative features of the UMNS are often more troublesome for the patient than the positive features (Landau 1980). Severe disabilities in, for example patients with stroke, have been shown to occur without the presence of spasticity (Sommerfeld et al. 2004, Welmer et al. 2006).

Aetiology

Spasticity typically occurs in patients following stroke, brain injury (trauma and other causes, e.g. anoxia, post-neurosurgery), spinal cord injury, multiple sclerosis and other disabling neurological diseases and cerebral palsy.

Classification

Spasticity is frequently classified by its presentation and divided into generalised, regional and focal. The term, focal spasticity, is imprecise, for it is not the spasticity that is focal, but that spasticity is producing a focal problem that may be treated by local means such as with Botulinum toxin injection.

Epidemiology

There are varied figures for prevalence of spasticity in different conditions (Sommerfeld et al. 2004, Pfister et al. 2003). This may be due to the presence of many patients with mild spasticity for whom little or no treatment is required for their condition. An early brain injury study in the UK estimates that 16% and 18% of first time stroke sufferers and patients following traumatic brain injury respectively require spasticity treatment (Verplancke et al. 2005). In a Swedish study (Lundstrom et al. 2008), the observed prevalence of any spasticity one year after first ever stroke was 17% and of disabling spasticity was 4% and an American study showed a prevalence of 35% among adults living in a developmental centre.

Pathophysiology

Spasticity arises from prolonged disinhibition of spinal reflexes as a result of UMN lesion. These spinal reflexes include stretch, flexor and extensor reflexes and are under supraspinal control by inhibitory and excitatory descending pathways. Stretch reflexes are proprioceptive reflexes, and are either phasic or tonic. The tonic stretch reflex arises from a sustained muscle stretch and is the cause of spasticity (Sheean 2002). Stretch reflex is dependent on tendon lengthening and excitatory post synaptic potentials (EPSPs) carried by I-a afferents but Inhibitory post synaptic potentials (IPSPs) arising from antagonistic muscle spindles, oligosynaptic and polysynaptic pathways also have an important role in the maintenance of tone (Lance 1980, Young 1994, Nathan 1973).

Damage to pyramidal tracts alone does not result in spasticity. It occurs only when the lesion involves premotor and supplementary motor areas. Clinical phenomenon are spinal in origin and arise because of hyper excitability of segmental CNS processing of sensory feedback from the periphery and they depend on location of the lesion, speed at which it has occurred and the duration since the lesion.

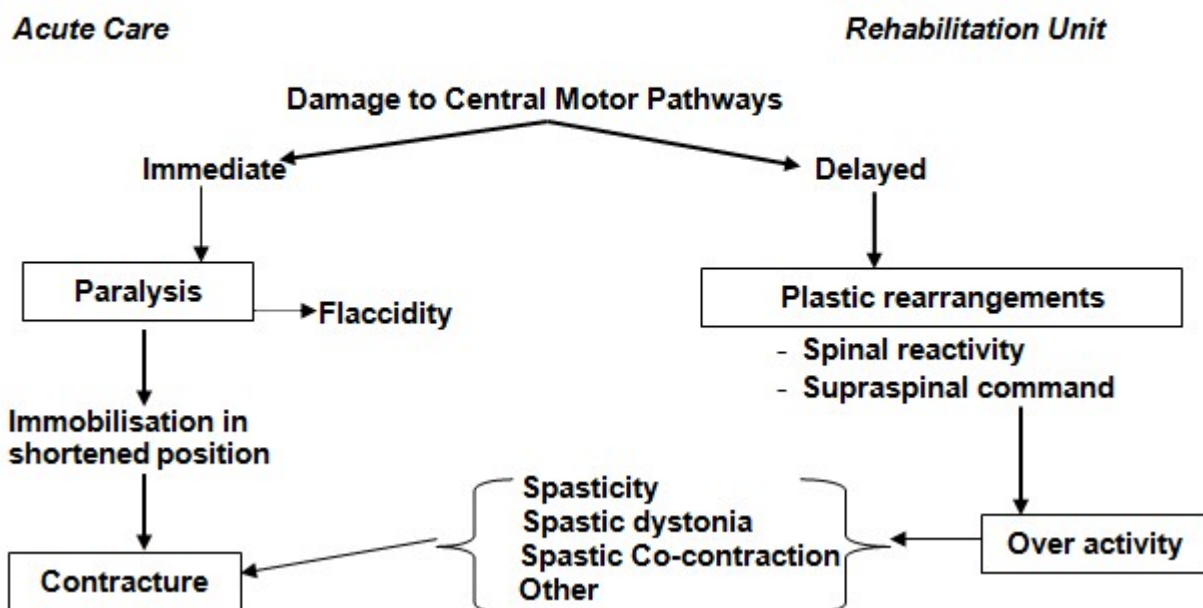
Spasticity is one of several possible impairments, resulting from a damaged upper motor neuron (O'Dwyer et al. 1996, Canning et al. 2000). The pathophysiology is complex and the actual problem of spasticity of increased resistance to passive movement is part of a bigger picture, which includes spastic dystonia, co-contraction and associated reactions in addition to spasticity

itself. Spasticity involves high -stretch sensitivity when excessive motor unit recruitment occurs with recruitment of stretch receptors and forms the stretch sensitive forms of muscle over activity, which includes spasticity itself. Spastic dystonia is dependent on efferent drives (Sheean 2002) and co-contraction is proposed to result from an activation of tonic stretch reflexes in combination with an inability to control reciprocal inhibition of agonist and antagonist muscle groups (Sheean 2002). Associated reactions are found in muscles that are not particularly stretch sensitive. They include, when there is extra-segmental co-contraction due to cutaneous or nociceptive stimuli, or inappropriate muscle recruitment during autonomic or reflex activities, such as yawning.

The definition of spasticity has been given above, but presents with muscle over activity in the absence of a volitional command (Lance 1980). It is thus measured in resting muscles. Spastic dystonia is primarily due to abnormal supraspinal descending drive, which causes a failure of muscle relaxation (despite efforts to do so) and is sensitive to the degree of tonic stretch imposed on that muscle (Denny-Brown 1966). There is inappropriate recruitment of antagonist muscles in spastic co-contraction upon triggering of the agonist under volitional command. This occurs in the absence of phasic stretch and is sensitive to the degree of tonic stretch of the co-contracting antagonist (Gracies et al. 1997). For instance, triceps will be recruited during volitional action of biceps and will lead to elbow stiffness.

The resultant pattern is determined by the age, size and location of the lesion and knowing this helps with management. Supra-bulbar lesions present predominantly with flexor patterns of spasticity, whereas spinal cord lesions produce extensor patterns predominately. Patients with partial lesions, where sensation is intact or partially intact, are typically bombarded by nociceptive inputs and display greatly increased α -motor neuron activity. Different patterns emerge early on after the neurological insult and later, when patients may find themselves in a rehabilitation unit. The following figure shows the effects of the different scenarios.

Development of Spasticity after UMN Damage



Immediately after injury, a period of neuronal shock occurs and spinal reflexes are lost, which include stretch reflexes. A flaccid weakness is seen, but even during this, the positive features of hypertonia can start to be seen. Limbs are not sufficiently stretched and may be immobilized in shortened positions. Rheological changes occur within muscles in the form of loss of proteins and sarcomeres and accumulation of connective tissue and fibroblasts (Ward 1999). Unless treated, tendon and soft tissue contracture and limb deformity are established. Altered sensory inputs such as pain, recurrent infection and poor posture, maintain a further stimulus to lead to yet further shortening, and this cycle is difficult to break.

Spasticity is set up later on, as plastic rearrangement occurs within the brain, spinal cord and muscles. This attempt at restoration of function through new neuronal circuitry creates movement patterns based on existing damaged pathways. Neuronal sprouting occurs at many levels with interneuronal endings moving into unconnected circuits from decreased supraspinal command through the vestibular, rubrospinal and reticulospinal tracts (Krenz and Weaver 1998). The end-effect is muscle over activity and exaggerated reflex responses to peripheral stimulation (Farmer et al. 1991). This process occurs at anytime, but is usually seen between one and six weeks after the insult. Muscle over activity declines over time and the following are suggested as possible causes:

- Structural and functional changes due to plastic rearrangement
- Axonal sprouting
- Increased receptor density

Measurement of Spasticity

Measurement of spasticity is essential to assess the response to treatment. Spasticity at any particular instance is dependent on several factors including presence of noxious stimuli the patient's physical and mental status and the position of the body. Therefore it is difficult to measure spasticity because of its multifactorial nature. Different methods are available for measurement but none of them is precise and reliable enough to quantify the severity of spasticity clinically.

Ashworth Scale

This scale is based on the assessment of resistance to stretch when a limb is passively moved. It was originally validated for patients with multiple sclerosis and was validated by Ashworth (1964). Its reliability is questioned by the subjectivity required by the observer to carry out the test and by the fact that it measures multiple aspects of limb stretch. However, it is in general use and has good inter-and intra-rater reliability (Ashworth 1964). The original Ashworth scale is only validated for measuring spasticity in the lower limb (Lee et al. 1989). In addition, it does not distinguish between increased neurogenic muscle tone and mechanical limb stiffness. Despite this, it has nonetheless become the measure against which all other measures are compared. The major modification (Modified Ashworth Scale) was proposed to differentiate between mild and moderate spasticity, as discrepancies appeared in clinical judgement at the lower end of the original scale. Bohannon validated the scale in elbow flexion in post-stroke patients and attempts have been made to widen the validity (Bohannon and Smith 1987). A grade 1+ was added and the top of the scale was reduced from 5 to 4.

Score	Ashworth (Ashworth 1964)	Modified Ashworth (Bohannon and Smith 1987)
0	No increase in tone	No increase in tone
1	Slight increase in tone giving a catch when the limb is moved in flexion/ extension	Slight increase in tone giving a catch, release and minimal resistance at the end of range of motion (ROM) when the limb is moved in flexion/ extension
1+		Slight increase in tone giving a catch, release and minimal resistance throughout the remainder (less than half) of ROM
2	More marked increase in tone, but the limb is easily moved through its full ROM	More marked increased in tone through most of the ROM, but limb is easily moved
3	Considerable increase in tone – passive movement difficult and ROM decreased	Considerable increase in tone – passive movement difficult
4	Limb rigid in flexion and extension	Limb rigid in flexion and extension

Tardieu Scale

The angle at the point of resistance is noted by stretching a limb passively. This is performed during as slow a movement as possible (V1), under gravitational pull (V2) and at a fast rate (V3). The examiner will feel a catch in a muscle under the influence of an overactive stretch reflex. Five levels have been described at the point of this catch to capture the quality of the muscular reaction. In essence the scale assesses dynamic and static muscle length as well as joint range of motion. The inter and intra-rater reliability is generally good (Gracies 2001), but the technique does require training to achieve this.

Stretch Velocity	Y Angle (Dynamic Range of Motion)	Quality of Muscle Reaction Course of Passive Movement
V1 Slow as possible	R2 Slow Velocity Passive joint range of motion or muscle length	0 No resistance 1 Slight resistance 2 Clear catch at precise angle, then release 3 Fatiguable clonus at precise angle 4 Unfatiguable clonus at precise angle 5 Rigid limb & joint
V2 Speed of limb falling under gravity	R1 Fast Velocity Movement through full range of motion	
V3 Fast as possible		

Wartenberg Pendulum Test

In this, the leg moves under gravity and the observer measures the pendular activity of a spastic limb as it relaxes. It is best carried out on the lower limb, for it is not so reliable for other limb segments.

Other methods for evaluating or assessing spasticity include muscle grading, deep tendon reflexes and Range of Motion measuring, bilateral adductor tone score, visual analogue scale, spasm frequency score, torque devices and electrophysiological studies (including dynamic multichannel EMG, tonic vibratory reflexes and electrical tests related to the H reflex and F wave). Most of these methods are time consuming, expensive, require specialised equipment and are used mainly in research

Why Treat Spasticity?

Spasticity in itself can be disabling and, if left untreated or sub-optimally treated, may lead to consequences, such as:

- muscle shortening,
- contractures (leading to abnormal body segment loading and sensory change)
- limb deformity and altered body mechanics, altered body image,
- the need for special wheelchairs and seating and pressure-relieving equipment,
- loading on pressure points,
- pressure sores,
- difficulty in the management of pressure sores,
- pain from muscle spasms,

- degenerative joint disease,
- loss of function, and
- mood problems and inability to participate in rehabilitation.

The misery of painful spasms or of tendon traction on bones is well known and the complications will prevent patients from achieving their optimal functioning. Deconditioning from ill-health and pain will also have a negative effect and patients and their carers may find reduced quality of life. There are therefore very good clinical, humanistic and economic reasons to treat it effectively and judiciously.

Complications that may result due to spasticity are interference with function, nursing care and hygiene, pain, deformity and disfigurement, contractures, joint subluxation and dislocation, peripheral neuropathy and pressure ulcers. Although associated with complications, spasticity is beneficial to some patients. It may help to transfer, stand and ambulate, maintain muscle bulk, prevent deep vein thrombosis and osteoporosis.

Indications of Antispastic Treatment

There has to be a guide to defining the aims of treatment, as patients have individual programmes of rehabilitation. Although there are a wide number of reasons to treat spasticity, the actual indications are quite specific and clinicians should follow these closely (Ward 2001). Non-ambulatory patients with moderate to severe weakness, hyperflexia, clonus and painful flexor spasms interfering with hygiene and nursing usually require treatment of spasticity. Patients may fulfill more than one indication, e.g. pain relief and care management.

Indication	Example
Functional Improvement	Mobility: enhance speed, quality or endurance of gait or wheelchair propulsion Improve transfers Improve dexterity and reaching Ease sexual functioning
Symptom Relief	Relieve pain and muscle spasms Allow wearing of splints/orthoses Promote hygiene Prevent contractures
Postural Improvement	Enhance body image
Decrease Carer Burden	Help with dressing Improve care & hygiene Positioning for feeding, etc.
Enhance Service Responses	Prevent need for unnecessary medication & other treatments Facilitate therapy Delay or prevent surgery

Principles of Management

The main goal of therapy is to increase functional capacity, relieve symptoms and decrease carer burden. This should be clear to the physician, the patient and the care giver. The consequence of reduction of spasticity should be assessed. If spasticity offers stability to a joint, its reduction may decrease the patients function. But, if there is minimal weakness with significant spasticity, treatment will result in considerable improvement in the patient's function.

Spasticity requires treatment when it is causing harm and this is the sole indication. Some patients early on after their stroke or brain injury are helped by their spasticity. For example, patients may start to support their weight by using their spastic lower limb when the degree of weakness in the leg would not allow it. Clearly, for these patients, reducing muscle tone would not be helpful, but it requires treatment when it causes problems or symptoms. Successful treatment strategies have now been developed and there is good evidence of treatment effectiveness. Physical management (good nursing care, physiotherapy, occupational therapy) through postural management, exercise, stretching and strengthening of limbs, splinting and pain relief is the basis of spasticity management (British Society of Rehabilitation Medicine 1992). The aim of treatment is to reduce abnormal sensory inputs, in order to decrease excessive a-motor neuron activity (Ward 1999). All pharmacological interventions are adjunctive to a programme of physical intervention. Stretching plays an important part in physical management, but needs to be applied for several hours per day (Tardieu et al. 1998). This is of course impossible to do on a one-to-one basis with a therapist and limb casting has been developed in this field to provide a prolonged stretch. Some studies have suggested that task-specific training might be more effective (Socialstyrelsen 2006).

Patient Assessment

Spasticity is a movement disorder and patients cannot be adequately assessed unless they are observed during movement and function. Physiotherapists and occupational therapists contribute to the observation and examination process, but some patients with complex movement patterns need assessing in a gait laboratory. The assessment process highlights the differences in patterns of limb posture and movement following an upper motor neuron lesion. Where there is no movement, the assessment process is fairly straightforward, but where there is loss of motor control rather than a spastic dystonia, one has to attempt to identify the different aspects of motor impairment. Patients with longstanding problems also develop compensatory movements, which may or may not require treatment and the clinician has to be clear about the underlying pathophysiological processes.

One can then identify how function is impaired and whether the problem is generalised, focal, or more regional. This will then point to the options for treatment. The indication for pharmacological treatment therefore is when spasticity is causing the patient harm. Some patients early on in their rehabilitation following a stroke or brain injury use their spasticity to walk on, when their weakness would otherwise not allow it. Clearly, treating the spasticity here would not be helpful and physical measures to utilise the developing movement patterns would be the treatment of choice, but where the spasticity gives rise to problems for either the patient or the carer, then treatment is required.

It is sometimes quite difficult to distinguish between severe spasticity and contracture formation, but it is important to do so. The clinicians and the patient/carer can then know what anti-spastic treatment can or cannot achieve and realistic expectations can then be identified. Severe, inadequately treated spasticity will go on to develop a limb contracture through shortening the muscle and tendons. A contracture may be fixed and will require serial splinting or surgery to correct it, but before it becomes fixed, the spasticity contributes to a dynamic contracture and treating the underlying spasticity may allow easier treatment of the contracture. One way to do that is examination under sedation. It is advisable to use a general anaesthetic for children. This relaxes spastic muscles and allows the range of passive joint movement to be assessed. One particular use is in assessing patients, who externally rotate their leg during walking. The adductor muscles can compensate for weak hip flexors and the patient rotates the leg

accordingly. Blocking the obturator nerve reduces the function of the adductors and it is then possible to see the degree of hip flexor weakness, so that a programme of muscle strengthening can be started rather than of BTX injections to weaken the adductors.

Management

Prevention of worsening of spasticity is very important in the management. It can be prevented from becoming severe by the avoidance of noxious stimuli such as pressure ulcers, urinary retention, constipation, infection and pain, patient and carer education regarding proper positioning, regular skin inspection and a good management of bladder and bowel, proper positioning, daily stretching to maintain range of motion, splinting (Pizzi et al. 2005, Turner-Stokes and Ashford 2007), serial casting, functional electrical stimulation, motor re-education and biofeedback.

Medical

All medical interventions are adjunctive to a programme of physical treatment, removal of exacerbating stimuli and patient and carer education.

Oral medications

Oral agents are useful in treating mild to moderate spasticity. The use of baclofen and dantrolene sodium has not changed much over the years (Tardieu et al. 1988, Cracies et al. 2002), but some newer products have emerged. Forty percent of patients are unable either to tolerate oral agents because of side-effects or unable to produce an adequate antispastic effect before side-effects occur.

Baclofen

Baclofen is a structural analogue of gamma-aminobutyric acid (GABA) and binds to GABA-B receptors both pre- and post-synaptically (Hwang and Wilcox 1989, Prince et al. 1984). Baclofen has been used as an anti-spastic drug for over 30 years and most of the clinical trials in several countries involving patients mostly with multiple sclerosis and spinal cord lesions, had proved that Baclofen is quite effective in reducing spasticity and for sudden painful flexor spasms (Hudgson et al. 1971).

Dantrolene Sodium

Dantrolene acts peripherally on muscle fibres. By suppressing the release of calcium ions from the sarcoplasmic reticulum, it dissociates excitation-contraction coupling and diminishes the force of muscle contraction (Pinder et al. 1977). It tends to be generally preferred for spasticity due to supraspinal lesions such as stroke, traumatic brain injury or cerebral palsy and some workers have suggested that stroke patients are more likely to improve with dantrolene (Chyatte et al 1971, Ketel and Kolb 1984). It was reported that patients with spinal cord injury also responded well to dantrolene (Weiser et al. 1978) , but was somewhat less effective in patients with multiple sclerosis (Gelenberg and Poskanzer 1973).

Dantrolene is associated with idiosyncratic symptomatic hepatitis, which may rarely be fatal in 0.1 to 0.2 % patients (Utili et al 1977, Wilkinson et al 1979). Hence, liver function tests should be checked periodically during dantrolene therapy.

Benzodiazepines

The antispastic effect of benzodiazepines is mediated via GABAA receptors. Among benzodiazepines, Diazepam was the earliest anti-spasticity medication used in clinical practice,

but is not much used now because of its daytime sedation. It is effective and compares well to baclofen in multiple sclerosis and spinal cord injured patients (Ketelaer and Ketelaer 1972). Other benzodiazepine analogues such as clonazepam are used in epilepsy and have been compared to baclofen mainly in multiple sclerosis patients (Cendrowski et al. 1977). It was found to be equally effective as diazepam, but it was less well tolerated due to adverse effects such as sedation, confusion and fatigue, resulting in more frequent discontinuation of the drug. It is thus used mainly for suppression of nocturnal painful spasms.

Tizanidine

It is an imidazoline derivative and has as an agonistic action at central alpha-2 adrenergic receptor sites. A number of studies have clearly demonstrated its benefit in spasticity due to multiple sclerosis and spinal cord injured patients, but definite functional improvements have not been shown (Smith et al. 1994, Nance et al. 1994, United Kingdom Tizanidine Trial Group 1994). It is also comparable to baclofen in efficacy in multiple sclerosis or spinal cord injured patient (Hassan and McLellan 1980, Smolenski et al. 1981, Newman et al 1982, Stein et al. 1987). It was similarly efficacious in comparison with diazepam in hemiplegia due to stroke and traumatic brain injury and allowed significantly better walking distance ability (Bes et al. 1988). Tizanidine also had a favourable adverse effects profile, although sedation remained a prominent side effect (Wagstaff and Bryson 1997).

Visual hallucinations and liver function test abnormalities also occur with clinically significant increases in liver enzymes in 5 to 7% of patients (Wallace 1994). An assessment of liver function test is therefore recommended before starting tizanidine and then after a month of treatment.

Gabapentin

Gabapentin is useful when there is pain and particularly when there is cortical dysaesthesia giving rise to abnormal sensory inputs. Like other oral agents, it is poorly tolerated in a significant proportion of patients and its use is therefore limited.

Cannabis

Convincing evidence that cannabinoids are effective in MS is still lacking (Killestein et al. 2004). Much of the evidence that cannabinoids could help spasticity symptoms is anecdotal. The recent CAMS study in multiple sclerosis patients compared oral cannabis extract and delta 9-tetrahydrocannabinol with placebo in 667 patients with stable multiple sclerosis and muscle spasticity in 33 UK centres over a 15 week period. The primary outcome measure was a change the Ashworth scale. Treatment with cannabinoids did not have a beneficial effect on spasticity, but there was evidence of a treatment effect on patient-reported spasticity and pain (Killestein et al. 2004).

Intrathecal medications

Intrathecal Baclofen

The treatment consists of the surgical fitting of a programmable electronic pump in the anterior abdominal wall attached to a subcutaneous catheter tunneled around the trunk and inserted into the spine canal at about the L2/3 level. The catheter is then placed up to a level between D8 and D10. This allows baclofen to be delivered at its site of action in the spinal cord, at higher concentrations than would be possible with oral administration and without the expected CNS side-effects (Pen and Kroin 1985).

The main indication is for people with paraplegia and tetraplegia, who are unable to tolerate or respond adequately to oral antispastic drugs. It is particularly useful in both brain and spinal cord injured patients, who do not have residual functioning, but the pump settings can also deliver doses in a highly specific manner to allow ambulant people to balance the weakening effect of baclofen against the spasticity required for weight support and joint mobility.

Intrathecal Phenol

Five percent intrathecal phenol in glycerine is given on infrequent occasions for the management of paraplegia. This is only indicated for people with progressive disease, who are refractory to other antispastic treatments and who have no ambulatory function and are incontinent. (For example terminally ill multiple sclerosis patients). The block is usually painless, as the phenol exerts a local anaesthetic effect and the procedure can be repeated as required.

Chemodenervation

Chemical neurolysis describes a destructive process of a nerve. Perineural injection of motor nerves using 3-6% aqueous solution blocks groups of muscles. This provides an initial local anaesthetic effect, which is later followed by blockade one hour later, as protein coagulation and inflammation occur (Kelly and Gautier-Smith 1959). Wallerian degeneration occurs later on before healing by fibrosis. This leaves the nerve with about 25% less function than before, but does not disadvantage people with little or no residual function, as a mild progressive denervation can be beneficial in reducing spasticity (Burkel and McPhee 1970). The effect can last for 4-6 months, and the renewal of muscle over activity is probably due to nerve regeneration (Bodine-Fowler et al. 1996). The indications for use are as an alternative to BTX or surgery in the treatment of focal problems (Kirazli et al. 1998). Disadvantages are, it takes relatively more time to perform the injection and can cause dysaesthesia if the phenol is placed in proximity to sensory nerve fibres.

Neuromuscular blockade

Botulinum toxin (BoNT) is injected into the overactive target muscles, which are responsible for the clinical picture. It is a potent neurotoxin, that inhibits the release of neurotransmitter chemicals by disrupting the functioning of the SNARE complex required for exocytosis of synaptic vesicles (Tardieu et al. 1988). It is suitable for long term blocking of neuromuscular transmission through the inhibition of release of acetyl choline. This leads to muscle paralysis over three to four months, but this can be extended by a programme of physical activity. The toxin will cross about four to five sarcomeres to get to the neuromuscular junction and can be seen there after about 12 hours. The toxin's clinical effect is seen at about 4 days and is certainly working at seven days. It works optimally at one month and will go to produce a clinical effect for three to four months. The end effect is weakening and relaxation of muscle over activity in people suffering the effects of the upper motor neurone syndrome. This results in a biomechanical change in the muscle's function and makes it amenable to stretching and lengthening. In addition, the weakening allows an opportunity for strengthening of antagonist muscles and thereby it is possible to restore some of the balance between the two. EMG guidance can be used to locate the smaller muscles precisely. Contraindications for BoNT injection include known sensitivity to BoNT, Patients receiving aminoglycoside antibiotics, myasthenia gravis, Lambert-Eaton syndrome, motor neurone disease and upper eyelid apraxia

Surgery

Surgical procedures include rhizotomy, peripheral neurectomy, neuroablative procedures, central electrical stimulators, cordotomy, cordectomy, myelotomy, tenotomy, tendon lengthening and tendon transfers.

Outcome Measures

Outcome measurement in spasticity is controversial because of the huge array of available tools. Most clinicians do not actually measure the outcomes of their interventions in terms of the change to the neurogenic component of the upper motor neuron lesion. They more often measure the change in either the biomechanical consequence of the spastic limb (at impairment level) or the functional change (activity) of the goal of treatment. The main problem here is that the accepted measure of spasticity, the Ashworth score, does not actually measure what it purports to do. It does not follow Lance's definition and measures limb stiffness rather than velocity-dependent resistance (Pandyan et al 1999). The Tardieu Scale (Tardieu et al. 1954) and the Wartenburg Pendulum Test (Wartenburg 1951), on the other hand, do a better job, but are more unwieldy to use in clinical practice.

In clinical practice, measures of disability are the most useful to quantify and relate to the patient's rehabilitation aims. Spasticity is but one component that has to be dealt with and the outcomes of rehabilitation depend on issues relating to other impairments, to activity and to participation. An easy-to-measure tool is needed, whereas in research a standardised testing protocol is required to follow the definition of the condition as closely as possible. The Ashworth scale fails in this and to measure clinically important changes in spasticity but remains a useful bedside clinical measure. For research purposes, the Wartenburg Pendulum Test follows the definition and gets around the complex variables that occur in the alpha motor neurones of agonist and antagonist muscles during passive movements. Rymer & Katz conclude, however, that biomechanical measures correlate most closely with the clinical state, as extending a limb against passive resistance may be related more to the visco-elastic properties of the soft tissues than to spasticity (Gracies 2001). EMG activity and the motor unit magnitude correlate well with the torque and ramp and hold displacement around the elbow (Katz et al. 1994).

Functional aspects are important to measure (Francis et al. 2004), but one of the problems is that functional change with treatment may be dependent on factors other than the spasticity. Few studies have shown a global correlation with the Ashworth score and the measurement of function, as in the Rivermead or Fugl-Meyer Motor Assessment scores (Wade 1992), is best correlated with other impairment measures, like the spasm frequency score, adductor tone, pain score, etc. Therein lies the dilemma. We will probably have to keep on using the Ashworth scale in the clinical setting, but realise its limitations and always combine management of the patient with a functional outcome measure in relation to the rehabilitation goal.

Other measures have a particular use in physiotherapy practice and contribute to the overall picture of change following treatment. The walking speed (measured by a 10 metre walking time), the stride length and joint goniometry are useful in measuring change in hip and thigh spasticity in spastic diplegics (Ward 1999). Pain has been addressed above and the Jebsen Taylor Hand test demonstrates improvement in dexterity and isolated finger movement, whereas the Berg Balance scale evaluates what it suggests (Wade 1992). The final thought is that clinicians tend to measure what they feel is the most relevant aspect of treatment. Just as we need to ask the patient and family their view of the goal, we probably ought to involve more in

the measurement process too. The patient satisfaction score on a 10cm visual analogue scale is very useful identifying whether the targets were met in the patient's perspective and is useful when everyone is sure on expectations. The patient and physician global scores also address this aspect.

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