Spasticity is one feature of the upper motor neuron syndrome and is therefore common in disorders of the central nervous system. It is characterized by an increase in muscle tone causing resistance to movement that is velocity dependent i.e. it depends on the rate of movement, with sudden high velocity movements causing a greater increase in tone (Lance 1980). Traditionally, the features of the upper motor neuron syndrome are divided into ‘positive’ and ‘negative’ although it should be noted that the ‘positive’ features are not usually positive from the patient’s perspective (Table 1). Spasticity has a characteristic ‘feel’, which the experienced clinician can distinguish from rigidity, and resistance to movement caused by mechanical factors e.g. contracture, joint disorder. The common causes of spasticity are detailed in Table 2.

### Table 1  Features of the upper motor neuron syndrome (adapted from Greenwood 1998)

<table>
<thead>
<tr>
<th>‘Negative’ features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute hypotonic ‘shock’</td>
</tr>
<tr>
<td>Weakness due to failure of muscle activation</td>
</tr>
<tr>
<td>Loss of selective movement e.g. impaired dexterity</td>
</tr>
<tr>
<td>Fatiguability</td>
</tr>
<tr>
<td>Loss of cutaneous reflexes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>‘Positive’ features at rest in response to peripheral stimulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proprioceptive</td>
</tr>
<tr>
<td>Exaggerated tendon reflexes</td>
</tr>
<tr>
<td>Clonus</td>
</tr>
<tr>
<td>Increased muscle tone (spasticity)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Nociceptive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extensor plantar response</td>
</tr>
<tr>
<td>Extensor spasms</td>
</tr>
<tr>
<td>Flexor spasms</td>
</tr>
<tr>
<td>Mass reflexes (mass synergy patterns)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>‘Positive’ features during movement (spastic dystonias)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyssynergic patterns of co-contraction</td>
</tr>
<tr>
<td>Associated reactions</td>
</tr>
<tr>
<td>Flexor withdrawal reflexes</td>
</tr>
<tr>
<td>Positive support reaction</td>
</tr>
<tr>
<td>Extensor thrust</td>
</tr>
<tr>
<td>‘Pushing’ reactions</td>
</tr>
</tbody>
</table>

### Table 2  Commonly encountered causes of spasticity

<table>
<thead>
<tr>
<th>Cerebral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke</td>
</tr>
<tr>
<td>Acquired brain injury – traumatic, anoxic, infective</td>
</tr>
<tr>
<td>Cerebral palsy</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Spinal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Traumatic spinal cord injury</td>
</tr>
<tr>
<td>Non-traumatic spinal cord injury – cord compression, inflammatory (e.g. transverse myelitis, multiple sclerosis)</td>
</tr>
<tr>
<td>Mixed cerebral and spinal</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
</tr>
</tbody>
</table>
ents with spasticity
a practical approach

This review reflects my own clinical experience in a neurorehabilitation setting, dealing predominantly with spasticity due to acquired brain injury (traumatic and non-traumatic), multiple sclerosis, stroke and non-traumatic causes of spinal cord injury. I have tried to describe a systematic framework for the assessment and management of spasticity in routine clinical practice. I have also taken into consideration the realities of resource constraints within the UK National Health Service in which I work, so this review does not necessarily reflect absolutely ideal service provision.

PATHOPHYSIOLOGY

It is important to have some knowledge of the pathophysiology of spasticity to allow accurate medical assessment and to select appropriate pharmacological treatments when these are necessary (Sheean 1998; Brown 1994). Essentially spasticity is the consequence of loss of, or damage to, motor pathways descending from the brain to the spinal cord. These include the pyramidal (i.e. corticospinal) and parapyramidal (i.e. dorsal and ventral reticulospinal, lateral and medial vestibulospinal, rubrospinal, tectospinal and coeruleospinal) pathways which are so close together that any lesion is likely to affect more than one (Brown 1994; Greenwood 1998). While the pyramidal and dorsal reticulospinal tracts have an inhibitory influence on tone, the other parapyramidal tracts have an excitatory influence.

The pattern and severity of spasticity depends on where damage has occurred within the CNS, and the combination of affected tracts. Cortical and other lesions above the brain stem tend to cause less severe spasticity than lesions affecting only the spinal cord, where there may be complete loss of supraspinal inhibitory influences. It is important to be aware of this in clinical practice because if patients with cortical causes of spasticity (e.g. stroke) develop severe spasticity, exacerbating factors (such as deep venous thrombosis, fracture, infection, etc.) have to be sought and treated. There are also implications for pharmacological interventions, which act at differing points on the motor pathway, an example being baclofen, which acts mainly at spinal level and is therefore less effective in the treatment of spasticity of cerebral origin (Young & Delwaide 1981).

**Figure 1** Consequences of damage to the motor pathways in the CNS (Gracies et al. 1997). Note the relationship between contracture and spasticity, with spasticity and other muscle overactivity further increasing contracture. The therapeutic implication is that both need to be managed.
Muscle tone is a result of excitatory and inhibitory neural influences; excitation is enhanced in the upper motor neuron syndrome. (Walton 1993). Furthermore, this balance is continuously modulated by sensory feedback from proprioceptive, visual, vestibular and nociceptive mechanisms so any imbalance in one or more of these can tip the balance in favour or against excitation. For example, in a severely brain injured patient with cognitive deficits, impaired proprioception and visual impairment, any generalized spasticity may be reduced by providing cutaneous sensory feedback by appropriate postural management.

Damage to the motor pathways causes weakness (paralysis) which in the acute stage is accompanied by hypotonia. Both hypo and hypertonia make it difficult for the patient to voluntarily move the affected parts through their normal range resulting in muscles, joints and other soft tissues being left in anatomically incorrect positions (Fig. 1). Tissues then shorten with the change in compliance of muscle and other structures (tendon, joint capsule, etc.). It is important to recognize that in many patients it is this combination of spasticity and biomechanical change that results in the increase in muscle tone felt on clinical examination. Skilled clinicians can distinguish between these in some instances, but with the increasing use of local treatments for spasticity I find this distinction is in fact difficult.

**ASSESSMENT OF THE PATIENT WITH SPASTICITY**

Although measures of spasticity may be at the level of the impairment (e.g. amount of flexion contracture), these must always be interpreted in terms of functional outcome. For example, reduction in spasticity and flexion contracture at the elbow can improve independent mobility because the patient is able to reach the controls of their powered wheelchair. Spasticity must never be treated in isolation; it is important to treat the patient. Remember, spasticity is not always harmful, but inappropriate treatment may be (Table 3).

The key to effective and appropriate treatment is comprehensive assessment with the setting of clear, meaningful and measurable goals. This generally requires an experienced multidisciplinary team, rarely is medical assessment in isolation sufficient. A team approach involving nurses and therapists, as well as doctors, gives a more comprehensive view of the situation and facilitates the planning of management. To max-

### Table 3 Possible consequences of inappropriately treated spasticity

<table>
<thead>
<tr>
<th>CLINICAL SCENARIO</th>
<th>TREATMENT GIVEN</th>
<th>ADVERSE CONSEQUENCES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spastic paraparesis due to multiple sclerosis in a young woman. She had stiff legs and difficulty walking.</td>
<td>Examination confirmed lower limb spasticity. Oral antispasticity medication prescribed in increasing doses.</td>
<td>Reduction of tone which destabilized gait because extensor tone had been facilitating mobility. This resulted in a fall and ankle fracture and the patient was unable to regain her previous level of mobility.</td>
</tr>
<tr>
<td>Severe hip adductor and knee flexor spasticity with associated spasm in an elderly lady with a cervical cord lesion</td>
<td>Oral baclofen in increasing doses to 90 mg daily because of lack of efficacy at lower dose</td>
<td>She became increasingly confused and this was attributed incorrectly to cerebrovascular disease. The baclofen was stopped because it seemed ineffective and she developed an acute confusional state on sudden withdrawal.</td>
</tr>
<tr>
<td>Patient with multiple sclerosis. Severe lower limb flexor spasm on transfers, with truncal extensor spasm. At rest low toned trunk.</td>
<td>Oral baclofen increased from 60 to 90 mg daily</td>
<td>Fall in level of consciousness, admitted acutely to hospital and found to be in ventilatory failure. This resolved with reduction in dose of baclofen</td>
</tr>
<tr>
<td>Severe painful lower limb flexor spasticity in a patient with multiple sclerosis.</td>
<td>Intrathecal phenol injected during an acute admission after failure of steroids and absence of physiotherapist</td>
<td>Full assessment of the neurological disability was not performed, the patient was not informed of the probability of sensory loss and she developed multiple pressure sores extending down to bone with associated sepsis requiring one year inpatient treatment as a consequence.</td>
</tr>
</tbody>
</table>
imizethe chance of success, the team must be involved in the setting and measuring of outcomes along with the patient and/or their carers.

Medical assessment of spasticity

The following should all be taken into consideration:

- The pathology causing the neurological condition, including the site of damage within the CNS (remember that lesions above the brain stem do not cause severe spasticity in their own right).
- The clinical situation – acute/subacute, or long-term/chronic neurological impairment factors which may make spasticity worse: systemic illness e.g. localized or general sepsis, metabolic upset; local nociceptive stimulus, e.g. deep venous thrombosis, fracture, heterotopic ossification, tight orthosis, catheter bag, pressure sore(s); suboptimal bladder management – recurrent infections, high intravesical pressure due to neurogenic outflow obstruction, renal tract stones; sympathetic overactivity e.g. due to anxiety; constipation.
- Poor posture secondary to spasticity and soft tissue changes will further exacerbate spasticity, worsening the situation.
- Associated neurological features e.g. impaired cognition, ataxia, underlying weakness.
- Other medical pathology which may affect management and outcome.
- Drug interactions and risk of adverse effects from systemic antispasticity agents.
- The goals of treatment and how these will be measured.

Remember that multiple factors contribute to increased muscle tone. Furthermore, the severity of spasticity in the outpatient clinic may not reflect the true situation because both spasticity and spasm are exacerbated by physical and emotional factors (Table 4).

Functional assessment of spasticity

The aims of spasticity management should always be determined within the context of maximizing function, either by prevention of permanent deformities, which could impede future function, or by reducing the consequenc-

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>POSSIBLE CAUSES</th>
</tr>
</thead>
</table>
| Anxiety                | Stress of attending an appointment  
Ambulances are rarely on time  
Inability to use the toilet if the patient needs a hoist to transfer |
| Discomfort/nociceptive stimulus | Not in usual wheelchair, ambulances do not always take powered chairs  
May have been in wheelchair longer than normal to attend clinic  
Full bladder, inadequate toileting arrangements  
Clinic attendance may have prevented usual arrangements for bowel management  
Patients with pressure sores still keeping their outpatient appointments |
| Poor posture in wheelchair | Morning clinics make it difficult to co-ordinate availability of carers and timing of the ambulance service – patients may have been positioned hurriedly, increasing spasticity  
Fatigue if sitting in wheelchair for long periods, poor trunk control will increase lower limb tone and spasm |
| Positioning adversely affecting tone | Lying on an examination couch will increase spasticity and spasm  
Inappropriate wheelchair in clinic |
| Sudden movements       | Ambulance journeys are not smooth  
Moving and handling techniques are not optimal in the outpatient setting, it is unlikely that staff will have had appropriate training unless in a neurorehabilitation clinic |
es of increased tone where these have already developed. As previously stated, spasticity should not be treated in isolation, the management of the whole patient is paramount.

It is outside the scope of this review to describe how function can be assessed formally. There are a number of validated measures used routinely in neurological rehabilitation that are applicable in the context of spasticity management, the most useful are summarized in a consensus document from the Royal College of Physicians (Turner-Stokes & Ward 2002).

AN APPROACH TO MANAGEMENT OF THE PATIENT WITH SPASTICITY

The common exacerbating factors for spasticity (see above) should first be excluded or minimized before specific treatments for spasticity are started. Medical stability with management of intercurrent infections, etc. should be achieved, and noxious stimuli eliminated. A management plan for the spasticity and its consequences can then be agreed. The first question clinicians must ask is, 'Is the spasticity harmful to this patient?' i.e. is the spasticity interfering with function (including the ability of carers to perform personal care tasks) or independence now, or might it impede the return of function in the future, or is it causing pain? To answer these questions, assessment by other team members (nurse, carer, physiotherapist, occupational therapist) may be necessary, in addition to information from the patient.

If the answer is 'no', then treatment of the spasticity is not necessary. In some circumstances the spasticity may even be beneficial, for example extensor lower limb tone may enable standing and walking, and so reduction in tone could impair mobility. If the answer is 'yes', then the following should be considered:

- Will patient and/or carer education suffice? For example, simple passive exercises, positioning to minimize triggers to spasm, etc.
- Physical treatments should come before pharmacological treatments, which are a potentially dangerous substitute for poor service provision.
- Pharmacological treatments can be harmful, all the systemic antispasticity agents may cause significant adverse effects.
- Factors that might influence treatment and outcome e.g. underlying weakness, ataxia, impaired cognition and insight, unrealistic expectations.

It is important to treat disabling spasticity for the following reasons:

- Spasticity can cause pain and lead to deformity which increase disability, reducing functional activities by impacting on mobility, self care tasks, and the ability of carers to attend to hygiene.
- Spasticity can increase the risk of pressure sores.
- Severe elbow or knee flexor spasticity and contracture can impede tissue viability in the elbow crease and behind the knee, further increasing spasticity and spasm.

If the decision is that treatment (be this physical or pharmacological) is necessary, the goals must be agreed with the patient and/or carers (Fig. 2). These outcomes should be agreed before starting treatment, and measured before and after any interventions. But the treatment goals themselves depend on the clinical situa-

### Treatment goals

#### Pain reduction
- Spasticity per se
- Spasms
- Musculo-skeletal pain

#### Prevent/treat complications
- Pressure sores
- Contractures

#### Improve function
- Mobility (walking or wheelchair)
- Activities of daily living – for patient or carers
- Facilitate rehabilitation

#### Improve quality of life
- Independence
- Pain reduction
- Reduced care needs
- Participation in social/leisure activities

Figure 2 Possible treatment goals in spasticity management.
Spasticity management in the acute/subacute situation

An example is following acute brain injury (e.g. traumatic, anoxic, stroke). Here the goals of early spasticity management are the prevention of contractures with the aim of maximizing function should neurological recovery occur. Failure to manage spasticity appropriately in this situation can result in fixed contractures causing impairment of function, prolonged hospital stay, and increased costs of equipment and care. Depending on the nature and location of the injury, the pattern of spasticity may be focal or generalized.

Decisions may need to be taken about prioritization of treatments because it is rarely possible to treat the whole problem where there is multifocal or generalized spasticity. Spasticity due to brain injury tends to be less responsive to systemic antispasticity drugs than spasticity secondary to spinal cord lesions, with an increased risk of adverse effects. I now rarely prescribe systemic treatments for spasticity in patients with acquired brain injury (including stroke) because in my experience they are minimally effective when compared to good medical and physical management combined with focal treatments.

Spasticity management in the chronic situation

The goals of treatment are to improve, or prevent deterioration in function, and to reduce the impact of spasticity on personal care tasks performed by carers. Again careful assessment is crucial, taking multiple factors into consideration when planning treatment interventions. In patients with very severe neurological impairments, the goal may be to facilitate care, an example being the patient with severe hip adductor spasm requiring three carers to attend to perineal hygiene and to replace a urethral catheter. The treatment goal here is to reduce the adductor spasticity and spasm in order to minimize pain and facilitate care with a reduced number of carers.

Pharmacological treatment options

Before considering any pharmacological options, remember these should complement physical and therapeutic interventions, not replace them. Knowledge of the pathophysiol-

CASE HISTORY 1 – A DISASTROUS DELAY IN TREATMENT

A 45-year-old lady had a subarachnoid haemorrhage, complicated by obstructive hydrocephalus and ventriculitis. Subacute management was on an acute medical ward. Severe lower limb spasticity developed but treatment was difficult because of pain. A neurorehabilitation bed was available but only four months following onset. On transfer she had severe cognitive and physical neurological impairments – spastic quadriplegia with painful hip adductor and knee flexor spasticity. She was very anxious, particularly about any moving, handling and personal care interventions. Lower limb spasticity and spasm were treated with physiotherapy, botulinum toxin injections to the hip adductors and knee flexors, and this facilitated positioning in bed with a T roll to enable prolonged stretch on the contracted muscles. Pain and therefore anxiety levels were reduced to enable better care and therapy. She had underlying fixed contracture at her knees and hips, of approximately 90 degrees. Six months after transfer to the neurorehabilitation unit she had good return of muscle power to both legs, no obvious spasticity, and her cognition was only slightly impaired. She was mobile in a wheelchair, and transferred using a sliding board with assistance. Unfortunately there were major delays in discharge because of her need for adaptations to enable access to her home. The total hospital stay was 11 months, which could have been only six months with appropriate management in the subacute stage. Eighteen months after the brain injury, she is awaiting corrective surgery to both legs, because it is felt that she has the potential to walk. The delay in neurorehabilitation was a disaster.
be acknowledged that in some cases the ‘whole’ problem may not be amenable to pharmacological treatment, and that some impairments have to be accommodated e.g. with the use of specialized seating and postural management.

**Systemic treatments**

Table 5 details the commonly prescribed systemic antispasticity agents, their site of action, commonly encountered adverse effects which restrict their use in clinical practice, and their dosage regimes. These drugs tend to be most effective in spasticity of spinal origin. Because dantrolene acts peripherally on muscle, it should be effective in all causes of spasticity but it is severely restricted by generally poor tolerance and adverse effects. In my experience

<table>
<thead>
<tr>
<th>DRUG</th>
<th>SITE OF ACTION</th>
<th>LIMITING FACTORS IN CLINICAL PRACTICE</th>
<th>SUGGESTED DOSES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baclofen</td>
<td>Spinal cord</td>
<td>Non-selective reduction in tone and muscle weakness</td>
<td>Start low: 5 mg bd or tds Increase every 3–4 days by 5 mg at each dose</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Limited effect in spasticity of cerebral origin</td>
<td>Careful titration necessary to balance efficacy &amp; potential adverse effects</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sedation and cognitive impairment</td>
<td>Many patients unable to tolerate the maximum recommended dose of 100 mg daily</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lowers seizure threshold</td>
<td>Usual tolerated effective daily dose 30–60 mg split 3 to 4 times daily</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Withdrawal problems – seizures, hallucinations</td>
<td></td>
</tr>
<tr>
<td>Dantrolene</td>
<td>Peripherally at the level of the muscle fibres</td>
<td>Generally poorly tolerated – sedation, malaise, nausea, dizziness, diarrhoea Non-selective muscle weakness Hepatotoxicity – need to monitor liver function</td>
<td>Start at 25 mg daily Increase by 25 mg every 3–4 days Usually given in split doses 3–4 times daily Few patients need or tolerate the maximum recommended daily dose of 400 mg Most patients find doses of 100–200 mg effective, especially when given in combination with other medication</td>
</tr>
<tr>
<td>Tizanidine</td>
<td>Mainly spinal, but also at supraspinal level</td>
<td>Said to preferentially affect spastic muscles but still can cause generalized weakness Sedation Hypotension Bradycardia Need to monitor liver function</td>
<td>Start with 2 mg daily Increase by 2 mg every 3–4 days, slower if problems with tolerance Give in split doses 3–4 times daily Usually effective once 16 mg daily reached Maximum daily recommended dose 36 mg</td>
</tr>
<tr>
<td>Diazepam</td>
<td>Spinal cord and brain stem. Also effective as an anxiolytic where anxiety contributes to spasticity</td>
<td>Sedation, confusion, memory impairment Development of tolerance necessitating increasing doses</td>
<td>Start low with 2 mg initially Increase slowly every 3–4 days to avoid sedation Effective dose varies greatly between individuals, may be between 5 and 60 mg daily. May only need a night time dose for painful nocturnal spasms</td>
</tr>
</tbody>
</table>
patients with cerebral damage are least tolerant of all the systemic agents, which are either minimally effective, or not effective at all when assessed objectively in this context. When dealing with severely neurologically impaired patients, the logistics of haematological monitoring also need to be considered – is there easy venous access, does the patient have to attend a clinic regularly, are there transport issues, can the blood be taken at home?

Baclofen remains the systemic antispasticity agent of first choice. Although the maximum recommended daily dose is 100 mg split three or four times a day, few patients in my experience can tolerate so much. Most take 30–40 mg daily. Patients with isolated spinal cord damage appear to tolerate higher doses than those with cerebral damage. Tolerance is variable between individuals, especially in patients with multiple sclerosis, with some being unable to manage even low doses (e.g. 5 mg three times daily).

Tizanidine tends to be given to patients who are unable to tolerate baclofen, or where it is ineffective. Tolerance is variable, especially in multiple sclerosis where in my experience the risk of adverse effects is higher than in patients with isolated spinal cord damage.

The use of dantrolene is limited by poor tolerance, adverse effects (especially the effects on cognition) and the need to monitor liver function. With the introduction of tizanidine, I rarely now prescribe dantrolene.

Diazepam is an effective antispastic agent but is limited by sedation and effects on cognition. It is particularly effective for nocturnal spasms when sedation is not a concern. Effective doses are variable, between 2 mg and 60 mg. It is also useful where anxiety is a trigger to spasticity/spasm. Tolerance, rather than dependency, may develop, requiring increasing doses to maintain the optimum effect. The decision to use diazepam will depend on the clinical situation, e.g. with patients in the late stages of multiple sclerosis I would not be concerned about the development of tolerance and the need for higher doses.

If one systemic antispasticity agent is ineffective, it should be gradually stopped before starting an alternative. If there is a partial effect, or full effect is limited by poor tolerance, a second drug can be introduced, but cautiously as the likelihood of adverse effects is increased. Logically, a combination of a centrally active and a peripherally active drug should be more effective than two centrally active agents (e.g. baclofen or tizanidine combined with dantrolene). As with any other treatment, combinations should be evaluated using objective measures.

Before starting treatment clinicians must ask about any diurnal variations in the spasticity/spasm, and any triggers. For example, some patients have an increase at night and/or on waking, but no specific problems with spasticity/spasm during the day so medication may then only be necessary at these times.

Other drugs (e.g. gabapentin, clonidine) are used in the pharmacological management of spasticity, as are illicit drugs such as cannabis. As yet there is no robust evidence for their routine use.

Focal treatments
These should be used in conjunction with other therapeutic interventions, and include:
- physical measures such as splinting/casting;
- pharmacological treatments
  - botulinum toxin
  - chemical neurolysis with phenol
  - intrathecal baclofen for severe lower limb spasticity
  - intrathecal phenol in specific situations.

Because dantrolene acts peripherally on muscle, it should be effective in all causes of spasticity but it is severely restricted by generally poor tolerance and adverse effects.
CASE HISTORY 2 – SO MUCH BACLOFEN THAT HE COULDN'T LIVE AT HOME

This 56-year-old man had had multiple sclerosis for 16 years and was in the secondary progressive stage with a spastic paraplegia. He had minimal upper limb involvement, and normal cognition. He managed at home. Following hospital admission for neurosurgical treatment of severe trigeminal neuralgia he developed lower limb flexor spasm affecting his ability to transfer and care for himself. He was prescribed an increased dose of oral baclofen, resulting in some reduction in spasm but not in the underlying spasticity. The baclofen also lowered truncal tone, adversely affecting sitting balance. The combination of knee flexor spasticity/spasm and reduced truncal tone required review of his seating requirements and a wheelchair was issued that accommodated his knee flexion of > 90 degrees. The reduced truncal tone and power required him to be reclined backwards. Because of his inability to manage in his own home, he was admitted to a nursing home and 18 months later to a neurorehabilitation unit. At this time he was unable to function from his wheelchair in a semi reclined position, found it difficult to self-propel his wheelchair because his arms were in a compromised position, and he had to use a hoist for all transfers. He had no independent sitting balance. In physiotherapy sessions he was able to achieve 90 degrees flexion at both knees, also achievable at night with a T roll. Whilst sitting he still had flexor spasm so he was unable to sit in a functional position. Botulinum toxin injections were given to his hamstrings bilaterally and these eliminated flexor spasm and some of the spasticity, and he became able to extend both knees to about 50 degrees short of full extension. Reduction in the oral baclofen dose was possible further improving trunk stability. He was then able to manage kitchen tasks – cooking from his wheelchair. Sliding board transfers reduced his care needs. It became possible for him to live in his own house, close to friends and family with carer support from social services.
All these treatments are effective in spasticity of both cerebral and spinal origin but, as with all management strategies, careful clinical assessment is essential to ensure the optimum outcome.

**Chemical neurolysis.** Phenol or alcohol may have prolonged or even permanent effects, so this technique should be used when recovery of motor function is unlikely (Barnes 1993; Botte et al. 1995; Bakheit et al. 1996). Most peripheral nerves are mixed motor and sensory and so injection of phenol can cause painful causalgia. To avoid this, only motor nerves should generally be injected, e.g. the obturator nerve for hip adductor spasticity. Although phenol itself is inexpensive, consideration needs to be given to the time and other resources required to perform these procedures - peripheral nerves need to be accurately located using a nerve stimulator, or motor end points using EMG.

**Botulinum toxin** injected directly into the affected muscle(s) is now widely used to treat muscle overactivity, including that due to spasticity (Brashear et al. 2002; Burbaud et al. 1996; Simpson et al. 1996; Bhakta et al. 2000; Reichel 2001). The Royal College of Physicians of London has published guidelines for its use in spasticity and these include guidance on functional assessment and review which can be applied to any treatment for spasticity (Turner-Stokes & Ward 2002). Specific guidance on dosages can be obtained from Brin (1997). It is important to note that the currently available botulinum toxin preparations (type A - Botox, Dysport, and type B - Neurobloc) are not dose equivalent.

Botulinum toxin blocks cholinergic transmission at the neuromuscular junction. It is injected directly into spastic muscles to reduce overactivity. The selection of muscles for injection requires a team approach, including therapists, nurses and doctors, followed by appropriate follow-up therapy (this may include splinting/casting, physiotherapy, muscle stretches). The principles of spasticity management described earlier apply, with comprehensive assessment being the key to success, clear documentation of treatment goals, and objective measurement before and after treatment to ascertain whether the treatment is effective.
The advantages of botulinum toxin over other focal treatments include:

- ease and convenience of injection – electrical localization of muscles is required in some, but not all, instances e.g. accurate localization of wrist and finger flexors;
- no sensory disturbance;
- the effect lasts up to 12 weeks, so it can be used in the subacute situation to prevent the development of contractures, where neurological recovery may occur;
- it can be an adjunct to splinting or surgery;
- the ability to titrate the dose in individual patients requiring repeated injections for specific functional effects;
- it can be used in conjunction with other pharmacological treatments to achieve the desired total effect e.g. with systemic antispasticity agents, with phenol neurolysis, and with intrathecal baclofen;
- low risk of adverse effects;
- non-toxic to the person giving it.

**Intrathecal treatments.** Baclofen can be delivered intrathecally via an implantable programmable pump. This can be very effective in severe lower limb spasticity in highly selected cases (Coffey et al. 1993). But because intrathecal baclofen is very expensive and can have serious complications (some life threatening),
and successfully managed pro-actively. Intrathecal baclofen should only be given in specialist centres with the infrastructure to deal with emergencies should they arise.

There is still a role for intrathecal phenol in specific situations (e.g. end stage multiple sclerosis) and it can be very effective in experienced hands. If sensation is intact, dysaesthesiae can be very painful. Bladder and bowel function will also be affected. Patients must therefore have had all aspects of their neurological disability addressed prior to consideration of intrathecal phenol. I have not personally referred any patients for this procedure for many years, perhaps reflecting the existence of a good neuromusculoskeletal rehabilitation service where problems are anticipated and successfully managed pro-actively.

CONCLUSIONS

• The effective management of spasticity requires a multidisciplinary team approach.
• Comprehensive assessment is the key to success.
• Spasticity is treated because of its harmful effects, which include pain and deformity, resulting in loss of function and increased disability.
• It is essential to use objective measures of function and outcome to ascertain whether treatment interventions are beneficial for the individual patient.
• Although some patients can tolerate systemic antispasticity drugs, many cannot.
• Patients with cerebral causes of spasticity are less likely to benefit from systemic antispasticity drugs, and are more likely to develop adverse cognitive effects if they are used.
• If the treatable problem is focal, a systemic treatment may not be a logical choice.
• Always consider physical treatments first, pharmacology is not a substitute for deficiencies in therapy or rehabilitation services, and they are potentially dangerous.
• Of the focal treatments, botulinum toxin has many advantages over chemical neurolysis, although both can be used together in some circumstances.
• Physical, pharmacological and electrical treatments (such as functional electrical stimulation) can be used together.
• Expertise and planning are essential for the best outcomes.

REFERENCES