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The management of spasticity in adults

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Spasticity is a common disorder affecting people with long term neurological conditions such as stroke, multiple sclerosis, and traumatic brain and spinal cord injuries. A systematic review of 24 studies on the epidemiology of leg spasticity reported a prevalence of 28-38% in patients with stroke, 41-66% in patients with multiple sclerosis, and 13% in patients with traumatic brain injury.[1]

Spasticity varies from a subtle neurological sign to a gross increase in tone causing immobility of joints. The disorder is associated with several complications, including falls, pain, pressure ulcers, infections, and contractures,[2] although it is not clear whether these complications are caused by spasticity or co-exist independently.[1] Spasticity increases care needs and utilisation of healthcare resources,[3] and carers of patients with spasticity are more likely to experience anxiety and depression.[4] Some patients may make use of their spasticity to sit, stand, walk, or transfer. Management of spasticity requires a balanced approach, weighing the benefits of treatment against the usefulness of the spasticity. Current interventions to treat spasticity lack a robust evidence base, and guidelines often depend on expert recommendations. This review discusses the assessment and treatment of spasticity in adults.

What is spasticity?

Spasticity has been defined as "disordered sensorimotor control resulting from an upper motor neuron (UMN) lesion, presenting as intermittent or sustained involuntary activation of muscles."[5] Upper motor neurone syndrome may be associated with other clinical signs: positive features include exaggerated tendon reflexes, released reflexes, and upgoing plantars; negative features include motor weakness, slowness of movement, loss of dexterity, and loss of selective motor control. Lesions affecting the frontal motor cortex and its connections lead to increased excitation of spinal motor neurones[6] and a lower threshold to respond to stimuli such as stretch.[7] Overactivity of spinal motor neurones results in spasticity.

What are the clinical features of spasticity?

High muscle tone in spasticity characteristically affects the antigravity muscle groups. In the arms, the tone is usually high in adductors of the shoulders; flexors of elbows, wrist, and fingers; and pronators of forearm (figure). Excessive flexion of fingers and adduction of the thumb results in the characteristic clenched fist with "thumb in palm" deformity. In the legs, the high tone due to spasticity is particularly prominent in adductors of the hips, flexors of the knees, and plantar flexors and invertors of the ankle. Hyperextension of the big toe as a result of persistent high tone of extensor hallucis longus is another feature of spasticity, and patients may report difficulty in using footwear.

The high muscle tone associated with spasticity varies with the speed of the movement—that is, the faster the stretch, the greater the resistance. The resistance is felt as a catch after an initial few degrees of movement. This resistance then builds up; as the movement continues the resistance suddenly gives way. This "clasp-knife phenomenon" is evident during the initial stages of spasticity. Other clinical features of spasticity are clonus, spasms, spastic dystonia, and spastic co-contractions (box 1).

What is the differential diagnosis of spasticity?

The differential diagnosis of spasticity includes contractures, rigidity, and catatonia.

Contracture is a shortening and reduction in elasticity of muscles, tendons, ligaments, joint capsules, and skin, which is accompanied by an increase in the resistance to passive stretch.[6 8] After immobilisation, sarcomeres shorten and muscle and elastic tissue are replaced with connective tissue and fat.[6] Left unchecked, this process can result in permanent loss of motion in joints. Unlike spasticity, contractures do not show dynamic changes such as clasp knife phenomenon, catch and variation of tone with speed of movement, and position of limb. Sometimes stretching under sedation may be required to differentiate spasticity from contractures.

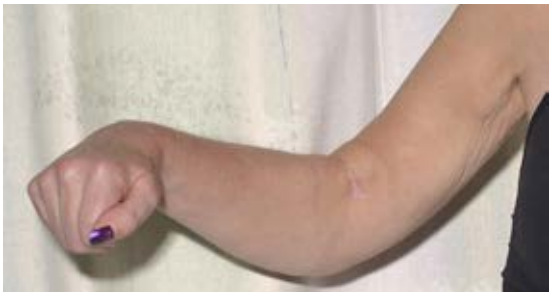
Rigidity is associated with disorders of the basal ganglia. Unlike spasticity, the high tone in rigidity is non-selective

SUMMARY POINTS

- Spasticity is a frequent and debilitating feature of common neurological conditions such as stroke, multiple sclerosis, and traumatic brain and spinal cord injuries
- The disorder is often associated with pain and discomfort and increased care needs
- Spasticity is difficult to manage and requires a collaborative approach involving multiple disciplines
- The evidence for both drug and non-drug treatments of spasticity is limited
- More research is required to determine the effectiveness of various treatments of spasticity

SOURCES AND SELECTION CRITERIA

We searched the databases PubMed, AMED, Embase, Medline, British Nursing Index, and CINAHL using the keywords "spasticity", "contracture", "upper motor neurone", and "muscle tone" from 2004 to 2014. We based this review on evidence from systematic reviews, guidelines by national and international organisations, and clinical trials published in English.



Spasticity of arm showing excessive flexion of elbow, wrist, and fingers

Box 1 | Clinical features of spasticity

Clonus
Involuntary rhythmic contractions triggered by stretch; these can interfere with walking, transfers, sitting, and care

Spasms
Sudden involuntary movements that often involve multiple muscle groups and joints in response to somatic or visceral stimuli

Spastic dystonia
Tonic muscle overactivity without any triggers owing to the inability of motor units to cease firing after a voluntary or reflex activity[6]; results in characteristic limb postures and contractures

Spastic co-contraction
Inappropriate activation of antagonistic muscles during voluntary activity due to lack of reciprocal inhibition causing a loss of dexterity and slowness in movements[6]

and affects all muscles acting on a joint equally. The resistance to passive movements due to rigidity is felt throughout the range of movement and does not change with speed of movement. In Parkinson's disease, rigidity associated tremors give a "cog wheel" feel on passive movement.

Catatonia is a neuropsychiatric syndrome characterised by abnormal posturing, gegenhalten, and "waxy flexibility." Gegenhalten or counterhold is an increase in muscle tone proportional to the force applied. In waxy flexibility, patients maintain their limbs in positions placed by others for a long time. When the limb is moved passively, the muscles stiffen in proportion to the force applied, as if the patient is actively opposing movement. Catatonia is caused by several psychiatric, neurological, and medical conditions. Presence of signs such as excitement, impulsivity, perseveration, combativeness, automatic obedience, stupor, mutism, staring, grimacing, stereotypical movements, echolalia, echopraxia, and withdrawal should raise a suspicion of catatonia.[9]

How is spasticity assessed?

Clinicians encounter patients with spasticity either as the initial symptom of a neurological illness or the worsening of existing spasticity due to a known long term neurological condition.

Approach to adults presenting with new onset spasticity

Spasticity could be the initial manifestation of several neurological conditions (table 1). An assessment includes history of onset and progression of symptoms such as weakness, abnormal sensations, pain, and bladder, bowel, and sexual dysfunction. An inquiry into family history, history of overseas travel, dietary preferences, and compromised immunity may be warranted. These patients require a neurological examination of muscle tone, motor power, and reflexes, and a careful search for a sensory level.

Approach to adults presenting with worsening of spasticity

Spasticity is a common feature of several long term neurological conditions such as stroke, traumatic brain and spinal cord injury, and multiple sclerosis. A long term neurological condition may be a more common reason for consultations involving spasticity compared with new onset spasticity in general practice. When spasticity worsens, these patients may experience a variety of symptoms, such as pain, stiffness, involuntary movements, deterioration of mobility, increase in care needs, and sexual dysfunction.

The worsening of spasticity could be due to:

Triggers—spasticity can be aggravated by visceral or somatic stimuli below the level of injury (box 2)[10]

Disease progression—worsening of spasticity could also be a sign of the progression of the primary neurological disease, as in progressive forms of multiple sclerosis. In people with spinal cord injury, worsening spasticity may indicate development of post-traumatic syringomyelia.

New disease—worsening of spasticity could indicate a coincidental new disease (table 1).

The first step in the assessment of patients with worsening spasticity is to look for triggers (box 2). The education of patients and carers in recognising these triggers is an important part of management. If no underlying triggers

Table 1 | Neurological conditions that may present initially with spasticity

Causes	Clinical features	Investigations
Spinal cord compression:		
Spinal canal stenosis, tumours affecting spinal cord	Sensory level: bladder, bowel, and sexual dysfunction	Magnetic resonance imaging
Autoimmune disorders:		
Primary progressive multiple sclerosis	Slowly progressive course without remissions and relapses	Magnetic resonance imaging, cerebrospinal fluid for oligoclonal bands
Neurodegenerative conditions:		
Motor neurone disease	Presence of lower motor neurone signs such as wasting and fasciculation	Electromyography
Infections:		
Tuberculosis of spine, Human T lymphotropic virus (HTLV1) myelopathy	Compromised immunity; contact with tuberculosis	Magnetic resonance imaging, HTLV serology
Inherited conditions:		
Hereditary spastic paraparesis, leucodystrophy	Family history of neurodegenerative conditions	Magnetic resonance imaging, genetic testing, very long chain fatty acids, white cell enzymes
Nutritional:		
Subacute combined degeneration of the cord, (vitamin B12 deficiency), copper deficiency	Nutritional history, eating disorders, use of zinc supplements	Vitamin B12, serum copper
Vascular:		
Spinal vascular malformations	Slowly progressive symptoms, combination of upper and lower motor neurone signs with sensory signs	Magnetic resonance imaging, spinal angiography

Box 2 | Triggers for worsening of spasticity

- Skin—ulcers, ingrown toenails, boils, and skin infections
- Visceral—constipation, urinary tract infections, urinary tract calculi, pain during menstruation
- Devices—improper seating, ill fitting orthotics, failure of intrathecal baclofen pump
- Drugs—rapid withdrawal of antispasticity drugs
- Others—infections, injuries, deep vein thrombosis, stress

Box 3 | Aims of treatment for spasticity

- Relief of pain and discomfort
- Improvement of posture
- Facilitation of sitting, standing, and walking
- Reduction in burden of care
- Improvement of hygiene in areas such as palm, axilla, and groin
- Improvement in body image and self esteem
- Prevention of complications such as pressure ulcers

are identified or if the spasticity continues to worsen, coincidental new disease should be excluded (table 1).

When should a patient with spasticity be referred?

Consider patients for referral to neurology services if they have new onset spasticity, spasticity that worsens rapidly without any triggers, or new neurological signs.

Referral to a rehabilitation specialist needs to be considered if patients fail to tolerate or respond to adequate doses of oral antispasticity drugs; spasticity affects posture, mobility, and care; or spasticity is associated with considerable pain and discomfort.

What is the impact of spasticity?

The impact of spasticity extends beyond the physical effects to psychological and social aspects of life.[11] Poor posture makes transfers and changes in position difficult and can contribute to pressure ulcers. Severe spasticity makes hygiene tasks, such as the cleaning of hands, axillae, elbows, and genital areas particularly difficult. Spasticity can also interfere with bowel and bladder care and sexual relationships.

Spasticity can be potentially useful for some patients. Stiffness of the weak muscles helps with tasks such as sitting, transfers, standing, and walking. Spasticity of long finger flexor muscles enables people to hold implements such as cutlery or a toothbrush. An individualised assessment looking at whether patients are making use of spasticity as a strategy to compensate for weakness is required before embarking on treatment.

People with spasticity also have additional neurological symptoms such as weakness, poor postural reactions, and sensory loss. The contribution of these symptoms to loss of function should be assessed.

What is the aim of the management of spasticity?

The aim of management is to reduce the impact of spasticity on patients and to prevent secondary complications. This requires a multidisciplinary team including doctors, physiotherapists, occupational therapists, orthotists, nurses, and wheel chair engineers. The first step is to agree on the aims of treatment with patients and carers (box 3). Interventions also need to be tailored according to the patient's activity, care needs, and access to physiotherapy.

Differentiating between spasticity and contracture is important, as this guides treatment options. Broadly, drug interventions will be effective at targeting spasticity, whereas non-drug interventions have a greater impact on contracture. Guidelines on multiple sclerosis from the National Institute for Health and Care Excellence recommend physiotherapy for the management of all patients with spasticity, and drug interventions for spasticity causing pain, discomfort, loss of independence, and limitation of activities.[12]

What are the non-drug interventions for spasticity?

Stretching and splinting

Manual stretching, often as part of a home management programme, has long been a cornerstone of the management of spasticity. However, the effect of stretching on spasticity and contractures is still largely an evidence-free zone. A meta-analysis of four trials with a total of 161 participants found no significant effect of stretching on spasticity.[13] Splinting has the advantage of providing a stretch over a more prolonged period compared with manual stretching. A before and after design study on 10 patients and a controlled trial of 15 patients showed that splinting reduced the spasticity of the fingers and wrists, measured using the modified Ashworth scale.[14 15] The modified Ashworth scale is an ordinal scale and measures the resistance of the limb to passive movements by the assessor.

The disadvantage is that splinting often holds the joint in a fixed position rather than applying a constant torque. The use of lycra based garments (dynamic fabricated orthoses) is another way of applying a stretch over longer periods while not rigidly fixing a joint. Customised, reinforced panels can direct the stretch appropriately, although they need adjustment for this to be maintained throughout the day. An open labelled crossover trial of 16 patients and a case series of six patients with chronic stroke suggest that these garments increase the range of movements at the wrist and fingers.[16 17] However, an open labelled trial of 13 patients showed no significant benefit with this intervention.[18]

Postural management and standing

Postural management involves managing postural alignment to prevent or reduce contracture and spasticity in people with severe symptoms. Management includes systems to maintain alignment when sitting, standing, and in bed. Standing frames allow people to remain upright for prolonged periods by providing the required support at the ankle, knee, or hip.

A crossover study on six patients with multiple sclerosis noted an increase in the range of movement at the ankle after the use of a standing frame for 30 minutes daily for three weeks.[19] Another small trial on eight patients with spinal cord injury showed that standing reduced spasticity, measured using the modified Ashworth scale.[20]

Exercises

Strengthening exercises improve motor control and function and, contrary to conventional beliefs, do not worsen

Table 2 | Details of oral antispasticity drugs

Drug	Indication	Dose	Side effects
Baclofen	Preferred oral antispasticity drug	Starting dose 5 mg three times daily; titration 5-10 mg weekly; maximum dose 90-120 mg/day divided into 3 doses; lower doses in renal failure; taper by up to 15 mg/week	Weakness, drowsiness, dizziness, sexual dysfunction, urinary incontinence. Sudden withdrawal may cause rebound spasticity, seizures, and hallucinations. Should be used with caution during pregnancy
Tizanidine	Second line treatment if baclofen is not tolerated or not effective	Starting dose 2 mg at bedtime; titration, increased by 2 mg weekly; maximum dose 36 mg/day divided into 3 or 4 doses; taper by 4 mg/week	Dry mouth, gastrointestinal disturbance, hypotension, and acute hepatitis. Monitor liver enzymes
Dantrolene	Second line treatment if baclofen is not tolerated or not effective	Starting dose 25 mg/day; titration, increase in steps of 25 mg/week; maximum dose 100 mg 3 or 4 times daily, taper by 25 mg/week	Hepatotoxicity, weakness, dizziness, and diarrhoea. Monitor liver enzyme levels
Gabapentin	Spasticity associated with pain	300 mg once daily on day 1, then 300 mg twice daily on day 2, then 300 mg 3 times daily on day 3, then increased according to response in steps of 300 mg every 2 or 3 days, up to maximum of 3600 mg daily	Weight gain, gastrointestinal disturbance, confusion, depression, hostility, and sleep disturbances. Need electrocardiography to monitor for QT interval prolongation

spasticity. A meta-analysis of 15 randomised controlled trials showed that strengthening exercises improved strength and activity after stroke and did not worsen spasticity. The evidence of the effect on spasticity was based on three studies with a total of 61 participants.[21] Strong trunk, pelvic, and shoulder girdle muscles provide the stability required for accurate control of the distal movements. Exercises may be avoided in patients who are otherwise not fit, are in the immediate postoperative period, have osteoporosis, or are severely limited in their passive range of movement.

Constraint induced therapy is a technique of training the affected arm while restraining the unaffected arm. A trial of this technique on 10 patients showed a reduction in spasticity, measured using the modified Ashworth scale.[22] In a small trial of seven patients with chronic spinal cord injury, treadmill training of walking with body weight support showed a reduction in spasticity, measured using the same scale.[23]

Other physical modalities

Several physical modalities have been tried to reduce spasticity, such as extracorporeal shock wave therapy, whole body vibration, transcutaneous electrical stimulation, repetitive transcranial magnetic stimulation, transcranial direct current stimulation, and electromagnetic therapy. A Cochrane review of nine randomised controlled trials with 341 participants using these non-drug modalities for the treatment of spasticity in multiple sclerosis found "low level" evidence only for repetitive transcranial magnetic stimulation.[24] All these techniques require further evidence before they can be recommended for the treatment of spasticity.

What are the drug interventions for spasticity?

Oral antispasticity drugs

Commonly used oral antispasticity drugs (table 2) are those acting on the γ-aminobutyric acid (GABA)ergic system (baclofen, gabapentin, and benzodiazepines) and α_2 adrenergic system (tizanidine) and those that block calcium release into the muscles (dantrolene). Although these drugs have been used for several decades, evidence for their efficacy is poor. A systematic review of 101 studies on oral antispasticity drugs noted a "dearth" of good quality trials and found limited evidence for efficacy of baclofen, tizanidine, and dantrolene in spasticity.[25] A Cochrane review on the management of spasticity after spinal cord injury found nine trials with a total of 218 participants,[26] Only tizanidine resulted in a significant reduction in the Ashworth score. Patients taking tizanidine also had significantly more adverse events than those taking placebo (81% *v* 53%). None of the oral antispasticity drugs resulted in improvement of function.[26] In a systematic review of 12 randomised control trials of 469 patients with non-progressive neurological disorders, the evidence on the efficacy of oral antispasticity drugs was "scarce and weak." Among all oral antispasticity drugs, tizanidine resulted in a reduction in the Ashworth score but did not improve function.[27]

Baclofen is the most preferred oral antispasticity drug. NICE guidelines recommend baclofen as a first line drug for the treatment of spasticity in multiple sclerosis and in children and young adults.[12 28] The second line options are tizanidine and dantrolene. Benzodiazepines have a similar efficacy to antispasticity drugs but more troublesome side effects. Drowsiness and behavioural side effects limit the use of benzodiazepines during the day time. Clonazepam is particularly useful for the treatment of nocturnal spasms. The usual starting dose is 250 µg at night, and the maximum dose is 1 mg.

Patients who do not respond to one type of oral antispasticity drug may respond to another. Evidence on the use of a combination of oral antispasticity agents is lacking. A combination of two drugs may be tried only in special circumstances: when spasticity is not responding to any single agent and in patients who could tolerate only small doses of antispasticity drugs.

Only around 50% of adults comply with treatment with oral antispasticity drugs.[29] Reduction of spasticity often unmasks underlying weakness due to the upper motor neurone syndrome or reliance on spasticity for postural stability and function, or both. It is important to mention this undesirable effect of treatment to patients. They should be advised to report any increase in weakness or falls after starting treatment. These drugs should be started at a small dose and increased in small increments.[30] It is also important to review the effects of oral antispasticity drugs periodically and to taper and stop them if they are not effective.[30] Even if drugs are apparently ineffective, it is better to taper the dose before stopping to avoid a rebound increase in spasticity from sudden discontinuation.

The timing of administering oral spasticity drugs and the dose should be tailored to the patient's lifestyle. Patients who are walking often require lower doses of antispasticity drugs during the day time as spasticity frequently facilitates standing and walking. A dose may be required before going to bed as spasticity often increases with change in posture. Furthermore, a dose immediately after awakening to reduce high tone may facilitate care in the morning.

TIPS FOR NON-SPECIALISTS

- Spasticity is often associated with other signs and symptoms such as weakness, poor postural reactions, and sensory loss; these should also be looked for and addressed
- Look for triggers or independent neurological causes, or both, in people presenting with worsening of pre-existing spasticity
- Involve patients and carers while formulating aims of management of spasticity
- Consider non-pharmacological interventions such as exercises, stretching, splinting, and posture management in all patients with spasticity
- Oral antispasticity drugs should be started at a low dose and gradually titrated up; they should be withdrawn in small decrements
- All antispasticity drugs cause muscle weakness
- Consider referral for botulinum toxin injections for focal spasticity
- Consider referral for intrathecal baclofen pump or chemical neurolysis for severe, treatment resistant spasticity

Spasticity can worsen during pregnancy.[31] No data are available about the effects of antispasticity drugs during pregnancy in humans. Neonatal baclofen withdrawal syndrome has been reported in babies born to mothers taking the drug during pregnancy.[32] Antispasticity drugs are also secreted in human milk. During pregnancy these drugs should be prescribed judiciously and only if clearly indicated.

Cannabinoids

Cannabinoids are the pharmacologically active compounds in marijuana.[33] They act on cannabinoid receptors (CB-1 and CB-2), which are widely distributed in brain and spinal cord. Tetrahydrocannabinol, an agonist of both receptors, reduces spasticity but causes sedation and psychotropic side effects. Cannabidiol has lower affinity to both receptors and reduces the psychotropic and sedative effects of tetrahydrocannabinol. A systematic review for the American Academy of Neurology concluded that oral cannabis extracts containing both these compounds are effective in improving patient reported outcomes in those with multiple sclerosis but not in changing objective measures of spasticity.[33] Nabiximols (Sativex; oromucosal spray, a 1:1 mixture of 9-δ-tetrahydrocannabinol and cannabidiol) is licensed for use to treat spasticity in people with multiple sclerosis in the United Kingdom. A meta-analysis of the original data pooled from 666 patients from three trials on nabiximols reported that it is well tolerated; 35% of the participants in the treatment arm showed a reduction of spasticity only on the patient reported outcome measures.[34] A further systematic review of six trials using a combination of tetrahydrocannabinol and cannabidiol reported no significant change in objective outcome measures but noted an improvement in patient reported outcome measures.[35] Side effects include taste disturbance, dry mouth, oral ulcers, dizziness, depression, mood changes, cognitive impairment, drowsiness, dysarthria, and blurred vision. As only 30-40% of people show a response to treatment, the use of cannabinoids should be reviewed and discontinued if there is no improvement after 4-6 weeks. The long term effects on cognition, behaviour, and mental health are unclear.

Botulinum toxin

When injected into skeletal muscle, botulinum toxin causes selective weakness of the target muscle by blocking the release of acetylcholine at the neuromuscular junction. It results in a focal reduction in spasticity without side effects of global weakness or sedation. Two randomised controlled trials, one on 96 and another on 333 adults with arm spasticity after stroke, showed a reduction in the modified Ashworth score but no significant improvement in function.[36 37] Currently, botulinum toxin is recommended for the treatment of patients with focal spasticity of the arms after stroke in the United Kingdom, United States, and Europe.[38-40] The botulinum toxins licensed for this purpose are onabotulinum toxin A (Botox; Allergan), abobotulinum toxin A (Dysport; Ipsen, Biopharm), and incobotulinum toxin A (Xeomin; Merz Pharma).

Therapeutic effects are seen in 7-10 days, peak in 4-6 weeks, and wane by 12 weeks. Patients should be reassessed in about four weeks after the initial injections to determine their efficacy and whether treatment goals have been attained. If required, further injections should be planned once every 12 weeks.[39]

Adverse events of botulinum toxin include dry mouth, injection site pain, respiratory tract infections, muscle weakness, urinary incontinence, falls, fever, and pain. Rarely, the toxin can cause transient dysphagia, which can sometimes result in the need for nasogastric feeding. Patients should be counselled about these adverse events and advised to seek medical help if they develop signs and symptoms of toxin spread.

Botulinum toxin injections for spasticity should be considered as part of a neurorehabilitation programme. Adjunctive interventions such as serial casting may help to maximise the outcome.[41]

Intrathecal baclofen

A relatively small dose of baclofen administered intrathecally achieves a high concentration of the drug within

ADDITIONAL EDUCATIONAL RESOURCES

Resources for healthcare professionals

- Spasticity in adults: management using botulinum toxin (www.rcplondon.ac.uk/spasticity-in-adults-management-botulinum-tox)—gives evidence based guidance on patient selection, assessment, setting of treatment goals, and follow-up for use of botulinum toxin injections for the management of arm spasticity
- Spasticity in children and young people with non- progressive brain disorders (http://publications.nice.org.uk/spasticity-in-children-and-young-people-with-non-progressive-brain-disorders-cg145)—provides advice on management of spasticity and associated motor disorders in children and young adults up to age 19 years
- Management of spasticity and associated features (www.ebrainjnc.com/learning/course/view.php?id=530)—this e-learning module describes the mechanism, assessment, and management of spasticity (free but requires registration)

Resources for patients

- Resources for people living with spasticity (www.spinalcord.org/resources-for-people-living-with-spasticity)—a compilation of resources on spasticity for patients and care givers
- A to Z of MS: spasticity (www.mstrust.org.uk/atoz/spasticity.jsp)—information on spasticity for patients with multiple sclerosis (site has links to webcast and fact sheets on spasticity)
- Spasms and stiffness (www.mssociety.org.uk/what-is-ms/signs-and-symptoms/spasms-and-stiffness)—describes causes, effects, and treatment options for spasticity affecting people with multiple sclerosis

the spinal cord resulting in good muscle relaxation without systemic side effects. The intrathecal baclofen pump both stores and delivers programmable doses of baclofen through a catheter into the spinal subarachnoid space. The pump can be programmed to administer a selected dose of baclofen. The drug is effective in the treatment of spasticity secondary to spinal cord disorders, stroke, and multiple sclerosis.[42-44] A systematic review of eight non-randomised controlled trials on intrathecal baclofen in 162 people with spinal cord injury found a significant reduction in spasticity measured using the Ashworth scale. The reviewers could not identify any randomised controlled trial that met their inclusion criteria.[45] The intrathecal baclofen pump is indicated for carefully selected patients with significant limb spasticity despite adequate treatment with oral antispasticity drugs.

The frequency of complications from using the intrathecal baclofen pump varies from zero to 2.24 per implant.[46] Procedure related complications include infection, skin erosions, and cerebrospinal fluid leak and seroma formation around the pump. Abrupt withdrawal of intrathecal baclofen due to pump failure, battery failure, catheter block, or patient non-compliance to treatment can cause a clinical emergency, with features similar to the neuroleptic malignant syndrome.[47] Signs of acute baclofen withdrawal include high fever, confusion, rebound spasticity, and muscle rigidity. Therefore patients need to comply with treatment and attend the hospital regularly for monitoring and pump refills. The treatment of acute intrathecal baclofen withdrawal is intrathecal baclofen through a temporary external catheter. Pending this, patients should be started on oral baclofen 90-120 mg/day.

Chemical neurolysis

Chemical neurolysis involves an intraneural injection of phenol, or alcohol destruction of peripheral nerves by protein coagulation. Chemical neurolysis is effective in treating spasticity in large, powerful muscle groups close to the trunk, such as adductors of the thighs. A randomised controlled trial of 20 patients found that an intraneural injection of either 5% phenol or 50% alcohol resulted in a significant reduction in the spasticity of plantar flexors at the ankle.[48] A retrospective case review of 20 patients with spasticity due to spinal cord injuries showed that chemical neurolysis of obturator nerve with phenol resulted in a significant reduction in the adductor spasticity.[49] Side effects include skin sloughing, wound infection, necrosis of muscle near the injection site, and pain. Injecting phenol into the lumbar intrathecal space results in chemical neurolysis of cauda equina. This is an option for people with severe spasticity of the legs who have lost bladder and bowel functions and have no functional movement and no sensation in their legs. A retrospective review of 40 patients who received intrathecal phenol showed that 31 had good improvement and seven experienced side effects.[50]

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ANSWERS TO ENDGAMES, p 36 For long answers go to the Education channel on thebmj.com

ANATOMY QUIZ

Axial post-contrast computed tomogram at the level of the aortic arch

A: Oesophagus
B: Superior vena cava
C: Left brachiocephalic vein
D: Trachea
E: Aortic arch
F: Aberrant right subclavian artery

STATISTICAL QUESTION

Understanding why "absence of evidence is not evidence of absence"

Statement *b* is true, whereas *a* and *c* are false.

PICTURE QUIZ

More than just a simple fracture

1 This anterioposterior radiograph of the left proximal humerus shows a fracture in the proximal metaphysis. Minimal displacement is seen and there is no intra-articular involvement. However the bone has a lytic appearance. This is a pathological fracture.
2 In this case, the low energy mechanism of injury (a fall from the patient's own height or less) in a normally fit and well child raises the possibility of an atypical fracture. The combination of abnormal bone and the fracture after low impact trauma confirms that this as a pathological fracture.
3 To narrow down the many possible causes, they are usually grouped by age and whether the underlying lesion is benign or malignant. The differential diagnosis includes benign lesions such as aneurysmal bone cysts or unicameral bone cysts. Malignant lesions such as osteosarcoma and rare giant cell tumours must also be considered. Osteosarcoma is the most common primary bone cancer in children and adolescents. Its incidence increases with age, most dramatically during adolescence, consistent with the rate of skeletal bone growth.
4 Because this is a pathological lesion, it is important to rule out underlying malignancy. Further tests could include magnetic resonance imaging, but the diagnosis is usually made histocytologically. An orthopaedic surgeon specialising in bone cancer can make the correct diagnosis in these cases and obviate the need for further investigation. However, if malignancy is a possibility or the underlying disease is uncertain, it is reasonable to seek help from a specialist bone cancer unit. If a biopsy is needed to confirm the diagnosis this should be performed by a specialist.
5 The goal of treatment for simple bone cysts is the prevention of pathological fractures. If a fracture has occurred, the aim of treatment is to assist healing. The optimal treatment option is unclear. Currently, the management of these patients includes observation with serial radiographs and intralesional steroid injections. If these two options are unsuccessful, surgical intervention should be considered.