

# Spasticity – Pathogenesis, prevention and treatment strategies

Anju Ghai, Nidhi Garg,  
Sarla Hooda, Tushar Gupta

Department of Anaesthesiology  
and Pain Medicine, Rohtak,  
Haryana, India

## Address for correspondence:

Dr. Anju Ghai,  
H no: 19/9J, Medical campus,  
Rohtak, Haryana, India.  
E-mail: dr.wadhera@yahoo.com

## ABSTRACT

This review of the long-term management of spasticity addresses some of the clinical dilemmas in the management of patients with chronic disability. It is important for clinicians to have clear objectives in patient treatment and the available treatment strategies. The review reiterates the role of physical treatment in the management, and thereafter the maintenance of patients with spasticity. Spasticity is a physiological consequence of an injury to the nervous system. It is a complex problem which can cause profound disability, alone or in combination with the other features of an upper motor neuron syndrome, and can give rise to significant difficulties in the process of rehabilitation. This can be associated with profound restriction to activity and participation due to pain, weakness, and contractures. Optimum management is dependent on an understanding of its underlying physiology, an awareness of its natural history, an appreciation of the impact on the patient, and a comprehensive approach to minimizing that impact. The aim of this article is to highlight the importance, basic approach, and management options available to the general practitioner in such a complex condition.

**Key words:** Long-term management, outcomes, peripheral nerve blockade, pharmacological treatment, phenol, spasticity

## INTRODUCTION

Spasticity is a major challenge to the rehabilitation team. The word “spasticity” is derived from the Greek word “spasticus,” which means “to pull or to tug.” It is defined as “disordered sensory-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles.”<sup>[1]</sup> It can range from mild muscle stiffness to severe, painful, and uncontrollable muscle spasm. It is associated with some common neurological disorders: Multiple sclerosis, stroke, cerebral palsy, spinal cord and brain injuries, and neurodegenerative diseases affecting the upper motor neuron, pyramidal and extrapyramidal pathways.<sup>[2]</sup> Left untreated, it gives rise to many problems, such as pain, spasms, limb contracture, and deformity. As a result, loss of mobility and dexterity, hygiene/self-care, and an inability to wear orthoses occur,

which can lead to decreased functioning and participation, and poor self-esteem. The initial management should focus on the alleviation of external exacerbating causes before specific treatment is considered. The role of the multidisciplinary team in the management of spasticity has been well described, and good nursing care, optimal posture, and physical therapy underlie the basic principles of treatment.<sup>[3]</sup> The aims of treatment should be to improve function, to reduce the risk of unnecessary complication, to alleviate pain, and to assist with the maintenance of hygiene, dressing, and transferring. Peripheral nerve blocks and botulinum toxin are two local treatments which are proving very useful, but are undervalued and underused.<sup>[4]</sup> This article reviews the variety of options available for clinical management of spasticity.

## PATHOPHYSIOLOGY

Spasticity usually is accompanied by paresis and other signs, such as increased stretch reflexes, collectively called the upper motor neuron syndrome which results from damage to descending motor pathways at cortical, brainstem, or spinal cord levels. When the injury is acute, muscle tone is flaccid with hyporeflexia before the appearance of spasticity. The interval between injury and the appearance

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of spasticity varies from days to months according to the level of the lesion. The pathophysiologic basis of spasticity is incompletely understood. The changes in muscle tone probably result from alterations in the balance of inputs from reticulospinal and other descending pathways to the motor and interneuronal circuits of the spinal cord, and the absence of an intact corticospinal system. Loss of descending tonic or phasic excitatory and inhibitory inputs to the spinal motor apparatus, alterations in the segmental balance of excitatory and inhibitory control, denervation supersensitivity, and neuronal sprouting may be observed. Once spasticity is established, the chronically shortened muscle may develop physical changes such as shortening and contracture that further contribute to muscle stiffness [Figure 1].<sup>[5]</sup>

## ASSESSMENT OF SPASTICITY

Before any intervention is undertaken to modulate hypertonicity, it is important to attempt to assess the severity of spasticity. Measuring spasticity is difficult, as there are no direct measures. Many grading scales are used to quantify spasticity. These assess the degree of muscle tone, frequency of spontaneous spasms, and the extent of hyperreflexia.

Ashworth Scale was developed in 1964. It is inadequate in measuring spasticity.<sup>[6]</sup>

- 0 = No increase in tone
- 1 = Slight increase in tone, giving a “catch” when the limb is moved in flexion or extension
- 2 = More marked increase in tone, but limb easily flexed
- 3 = Considerable increase in tone; passive movement difficult
- 4 = Limb rigid in flexion or extension.

Modified Ashworth Scale (MAS) developed in 1987 in response to concerns by the authors that the Ashworth

grade “1” was indiscrete. The MAS has an additional value of 1+ and has demonstrated inter-rater reliability of 86.7% in assessment of elbow flexor muscle spasticity.<sup>[7]</sup>

- 0 = No increase in muscle tone
- 1 = Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion (ROM) when the part is moved in flexion or extension/abduction or adduction, etc.
- 1+ = Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM
- 2 = More marked increase in muscle tone through most of the ROM, but the affected part is easily moved
- 3 = Considerable increase in muscle tone, passive movement is difficult
- 4 = Affected part is rigid in flexion or extension (abduction or adduction, etc.).

Severity of pain is assessed on a visual analog scale (VAS) where zero represents no pain and 10 represents the worst possible pain.<sup>[8]</sup>

Triple flexion of the lower limb, consecutive to the flexor muscle spasm, is assessed using a 4-point scale:<sup>[9]</sup>

- 0 = Hip and/or knee flexion  $<30^\circ$ , with or without mild gait disability
- 1 = Hip and/or knee flexion between  $30^\circ$  and  $45^\circ$ , with moderate gait disability
- 2 = Hip and/or knee flexion between  $45^\circ$  and  $60^\circ$ , with severe gait disability
- 3 = Hip and/or knee irreducible flexion with gait inability.

Range of Motion of the hip abductors is assessed passively by the abduction of the hip joint and graded as 0-3.<sup>[9]</sup>

- 0 = Ability to abduct the thigh easily to  $45^\circ$
- 1 = Ability to abduct the thigh to  $45^\circ$  with mild effort
- 2 = Ability to abduct the thigh to  $45^\circ$  with major effort
- 3 = Inability to abduct the thigh to  $45^\circ$ .

Number of spasms experienced are recorded on spasm frequency scale.<sup>[10]</sup>

- 0 = No spasm
- 1 = One spasm or fewer per day
- 2 = Between one and five spasms per day
- 3 = Between five and nine spasms per day
- 4 = Ten or more spasms per day.

Perineal hygiene is assessed using a 4-point scale, considering the ability of the patient to perform perineal hygiene care, related to the degree of adductor muscles spasticity.<sup>[9]</sup>

### Hygiene score

- 0 = Hygiene performance with relative ease
- 1 = Hygiene performance with mild difficulty

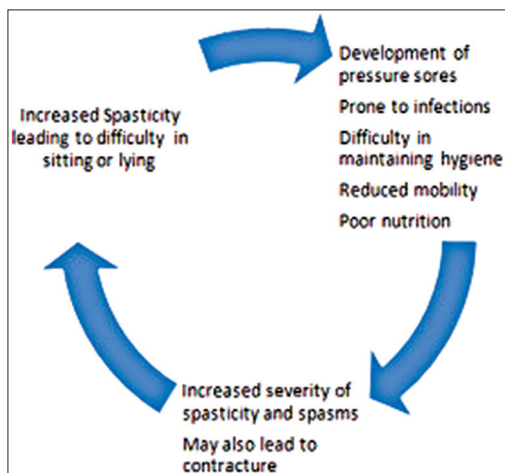


Figure 1: Vicious cycle of spasticity

- 2 = Hygiene performance with moderate difficulty  
 3 = Hygiene performance with severe difficulty.

Gait is assessed, when possible, using a 4-point score, representing the effect of obturator neurolysis on spasm and leg crossing:<sup>[9]</sup>

- 0 = Patient able to walk with mild difficulty  
 1 = Patient able to walk with moderate difficulty  
 2 = Patient able to walk with severe difficulty  
 3 = Patient unable to walk.

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## AIMS OF SPASTICITY MANAGEMENT

When the patient presents a pattern of spasticity that implies on functional or postural loss, management of spasticity must be considered within a progressive approach – from the most conservative to the most invasive therapy.<sup>[11]</sup> The aims of treatment should be the following:

1. Improve function - mobility, dexterity
2. Symptom relief - Ease pain-muscle shortening, tendon pain, postural effects
  - Decrease spasms
  - Orthotic wearing
3. Postural - body image
4. Decrease care burden - care and hygiene, positioning, dressing
5. Optimize service responses - to avoid unnecessary treatments, facilitate other therapy, delay/prevent surgery.

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## PRINCIPLES OF MANAGEMENT

### Physical

#### *Positioning and seating*

Correct positioning, for the immobile patient, is an important aspect of management. Incorrect positioning in bed, particularly in the early stages after stroke or brain injury, is a major cause of unnecessary spasticity. Side lying, sitting, and standing are of help in different circumstances, with the primary aim of producing stretch on the spastic muscles, thereby reducing spasticity as well as facilitating the use of antagonistic muscle groups.<sup>[12]</sup> Guidelines have been made to put each joint through a full range of movement for at least 2 h in every 24 h. The fundamental principle of seating is that the body should be contained in a balanced, symmetrical, and stable posture which is both comfortable and maximizes function. A proper seating system should aim at stabilization of pelvis without lateral tilt or rotation, but with a slight anterior tilt, so that the spine adopts its normal curves.<sup>[13]</sup>

#### *Stretching*

Maintaining muscle length through passive or active exercise and stretching regimens including standing or

splinting can be key to managing spasticity both in the short and the long term.

#### *Cooling of muscles*

This inhibits monosynaptic stretch reflex and lowers the receptor's sensitivity, thus inhibits spastic muscles, but the effect is short lived. Different techniques such as quick icing and evaporating spray like ethyl chloride are occasionally used.<sup>[14]</sup>

#### *Heat*

Heat may increase the elasticity of the muscles, causing relaxation of spastic muscles. Techniques used include ultrasound, fluidotherapy, paraffin, and superficial heat. These techniques should be combined with stretching and exercise.<sup>[15]</sup>

#### *Orthosis/equipment/aids*

An orthosis or splint is an external device designed to apply, distribute, or remove forces to or from the body in a controlled manner to control body motions and/or alter the shape of body tissues, e.g., ankle foot orthosis, insoles, ankle supports, wrist/hand/elbow splints, knee splints, spinal brace, hip brace, neck collar. The application of splints and casts can prevent the formation of contractures in the spastic limb and serial casting can improve the range of movement in a joint that is already contracted - a new cast being applied every few days as the range improves. Some equipment can also aid positioning, e.g., T roles, wedges, cushions, and foot straps. These are usually used in combination with other modalities like botox therapy.<sup>[16,17]</sup>

### Electrical therapy

#### *Functional electrical stimulation*

This is an adjunct to physiotherapy that can be of benefit to selected individuals predominantly affected by upper motor neuron pathologies resulting in foot drop.<sup>[18]</sup>

#### *Transcutaneous electrical nerve stimulation*

This has been found to reduce spasticity through its nociceptive action and reduction of pain.

### Pharmacological

Medication should always be used as an adjunct to good general management and education though the effect is short lived. Aims should be to improve function and relieve troublesome symptoms.

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## DELIVERY OF MEDICATIONS

- Enteral - orally, e.g., baclofen, benzodiazepines, dantrolene, clonidine, tizanidine, gabapentin
- Intrathecal - baclofen pump (other drugs used alone)

or in combination intrathecally include clonidine, morphine, fentanyl, midazolam, lidocaine)

- Intramuscular/focal injection, e.g., botulinum toxin
- Nerve blocks, e.g., phenol, ethanol.

### The oral agents

Oral antispastic medications are helpful for milder cases. However, in more severe cases and for focal spasticity, the side effects, commonly drowsiness and weakness, can significantly restrict the usefulness of these drugs. The most commonly used drugs are baclofen, tizanidine, dantrolene, benzodiazepines, and gabapentin [Table 1].<sup>[19,20]</sup>

**Baclofen:** It is GABA B receptor agonist that has presynaptic inhibitory effects on the release of excitatory neurotransmitters such as glutamate, aspartate, and substance P. It is interesting to note that there is no convincing evidence of efficacy in spasticity of cerebral origin.<sup>[21]</sup> In double-blind, crossover, placebo-controlled trials, baclofen was reported to be effective, producing statistically significant improvements in spasticity.<sup>[22]</sup> The main adverse effects of oral baclofen include sedation or somnolence, excessive weakness, vertigo, and psychological disturbances. The incidence of adverse effects ranges from 10 to 75%. The majority of adverse effects are not severe; most are dose related and reversible. The main risks of oral baclofen administration are related to withdrawal; seizures, psychic symptoms, and hyperthermia.<sup>[2]</sup>

**Tizanidine:** It acts by preferential inhibition of polysynaptic spinal excitatory pathways by stimulating alpha 2 receptors. There is large evidence for the effective use of tizanidine monotherapy in the management of spasticity.<sup>[23]</sup> It has better tolerability; in particular, weakness was reported to occur less frequently with tizanidine than with baclofen. It is comparable to baclofen and may even be superior.<sup>[24]</sup>

**Dantrolene sodium:** It has a peripheral mode of action via a direct effect on suppression of release of calcium ions from the sarcoplasmic reticulum of muscle, with

consequent inhibition of excitation, contraction, and coupling.<sup>[25]</sup> The recommended dosage is 25-400 mg daily with a slow titration. Doses higher than this can result in severe hepatotoxicity.<sup>[26]</sup>

**Benzodiazepines:** Diazepam is a GABA A agonist and it increases presynaptic inhibition of polysynaptic and monosynaptic reflexes.<sup>[27]</sup>

### Nerve block

Peripheral nerve blockade using neurolytic agents is one of the therapeutic possibilities in the treatment of spasticity. Nerve block refers to the application of chemical agents to a nerve to impair, either temporarily or permanently, the conduction along that nerve. The agents most frequently used are phenol, alcohol, and local anesthetics. This can be done with the help of fluoroscopy or nerve stimulation [Table 2]. Khalili and co-workers were the first to describe the use of phenol for nerve block.<sup>[37]</sup> The obturator nerve is the commonest and most accessible nerve blocked for adductor spasticity. The posterior tibial nerve is the second common nerve blocked for calf muscle spasticity.<sup>[4]</sup> The main indication is debilitating or painful spasticity. Peripheral blocks with local anesthetics are used as tests to mimic the effects of motor blocks and determine their potential adverse effects. They are easy to perform, effective, and inexpensive.

## INDICATIONS FOR CHEMICAL NEUROLYSIS

1. Management of chronic, intractable, non-terminal pain that are not responsive to other modalities<sup>[38]</sup>
2. Alternative to treat spasticity in order to improve balance, gait, self-care, and global rehabilitation. An important difference between neurolytic blocks for pain relief versus spasticity is that motor or mixed nerves are targeted preferentially in the management of spastic disorders.<sup>[38]</sup>

**Table 1: Drugs used in spasticity**

Drug	Dosage		Doses per day	Mechanism of action	Common side effects
	Initial dosage	Maximum dosage			
Baclofen	5 mg×3	90 mg	4	GABA-ergic	Seizure, sedation, dizziness, GI disturbances, psychosis, muscle weakness
Baclofen (intrathecal)	25 µg	500-1000 µg	Infusion		Decreased ambulation speed, muscle weakness
Tizanidine	2-4 mg	36 mg	2-3	Agonist at α2 adrenoreceptors	Liver dysfunction, dry mouth
Diazepam	5 mg or 2 mg×2	60 mg		GABA agonist	Dizziness somnolence, muscle weakness, addiction
Dantrolene	25 mg	400 mg	4	Inhibits release of intramuscular calcium stores	Hepatotoxicity, Decreased ambulation speed, muscle weakness
Clonazepam	0.5 mg	3 mg			Sedation, muscle weakness
Gabapentin	100 mg		400 mg×3	GABA agonist	Sedation, dizziness

GABA – Gamma Amino Butyric Acid

**Table 2: Efficacy of peripheral nerve blocks in clinical trials**

Investigator	No. of patients	No. of peripheral nerve blocks	Nerves blocked	Neurolytic agent used	Duration of effect	Complication
Khalili and Betts <sup>[28]</sup> (1967)	68	126	Median 32 Ulnar 7 Radial 2 Musculocutaneous 3 Femoral 5 Obturator 7 Medial popliteal 55 Common peroneal 5 Sciatic 7 Perineal 1 L=2 root 1 L=3 root 1	5 ml of 2-3% phenol	2-743 days	Burning sensation and hyperesthesia to light touch- common complaint Paresthesia in 10 patients
Spira <sup>[29]</sup> (1971)	61	136	38 Obturator nerve 80 Tibial nerve 5 Sciatic nerve 5 Median nerve 3 Musculocutaneous nerve 2 Common peroneal Nerve 2 Femoral nerve 1 Brachial plexus	2-5 ml 5% phenol 0.5-0.75 ml of 5% phenol	5-29 months (average 14 months)	Reduction of motor power in 17 patients Pain and paresthesia in seven patients
Trainer <i>et al.</i> <sup>[30]</sup> (1986)	3		Obturator nerve	1-5 ml of 6% phenol	6 weeks	—
Gunduz <i>et al.</i> <sup>[31]</sup> (1992)	36	50	34 Obturator nerves 11 Sciatic nerves 5 Femoral nerves	2-3 ml of 5% phenol	2-3 months	Cutaneous anesthesia in one patient
Yadav <i>et al.</i> <sup>[32]</sup> (1994)	116	246	110 Obturator nerves 134 Posterior tibial 2 Median nerves	6% Aqueous phenol	3-18 months (average 13 months)	Paresthesias in five patients with posterior tibial nerve block, pain at the site of injection or in the distribution of nerve in one patient, motor weakness in two patients, complete loss of sensation in one patient
Kong and Chua (1999) <sup>[33]</sup>	13			Absolute alcohol	Follow-up up to 6 months	Local swelling over the injection site Pain in eight patients
Kong and Chua (2000) <sup>[34]</sup>	8		Sciatic nerve	50-100% alcohol	Follow-up up to 6 months	None
Viel <i>et al.</i> <sup>[9]</sup> (2002)	23	27	Obturator nerve	8-9 ml of 65% ethanol	Up to 4 months	Local pain lasting for 2 months in one patient, obturator neuralgia for 3 weeks
Kumar <i>et al.</i> <sup>[35]</sup> (2008)	20		Obturator nerve Posterior tibial nerve	6% phenol	Follow-up up to 21 days	None
Akkaya <i>et al.</i> <sup>[36]</sup> (2010)	62	80	Obturator nerve	5-10 ml of 6% phenol	Follow-up up to 3 months	None

The success and duration of the block may vary anywhere from partial to excellent pain relief lasting from weeks to months depending upon the type of block and the skill of the physician. The most common cause of an unsuccessful block is incorrect placement of the neurolytic agent.

### Neurolytic agents

#### Phenol

Phenol is a chemical composite containing carbolic acid, phenic acid, phenylic acid, phenyl hydroxide, hydroxybenzene, and oxybenzene. It is available as colorless crystal and can be prepared to a maximum concentration of 6.7% solution in water. This substance is highly soluble in organic solvents such as alcohol and glycerol. The solution can be diluted with saline and is compatible when mixed with radiocontrast dye to allow fluoroscopic guidance during injection of the agent and to monitor spread of the solution. When mixed with glycerol, it slowly diffuses from the solution and is an advantage when injected intrathecally because it allows for limited spread and is localized in the area that needs to be destroyed. When mixed with water, it has a wider spread, causing a bigger area of nerve destruction. Aqueous solution of phenol is more potent than the glycerine solution.<sup>[39]</sup> The choice of diluent is dependent upon the extent of neurolysis desired. The use of higher concentrations of phenol might predispose to a higher incidence of vascular injury.

Shelf life exceeds 1 year when the solution is refrigerated and not exposed to light. Phenol turns red on exposure to sunlight and air because of oxidation. It is metabolized in the liver by conjugation to glucuronides and oxidation to equinol compounds or to carbon dioxide and water. Excretion of metabolites is via the kidneys. Systemic doses of more than 5 g can cause convulsive seizures and central nervous system depression. Doses less than 100 mg are less likely to cause serious side effects.<sup>[40]</sup>

Phenol causes nerve destruction by inducing protein precipitation. There is loss of cellular fatty elements, separation of the myelin sheath from the axon, and axonal edema. At a concentration of 2%-3% in saline, phenol seems to spare motor function. The efficacy of 3% phenol in saline is comparable to that of 40% alcohol. Protein denaturation occurs at concentrations less than 5%, and concentrations higher than 5% produce protein coagulation, nonspecific segmental demyelination, and orthograde degeneration. Wallerian degeneration occurs approximately 2 weeks following the injection. The degree of damage correlates directly with the concentration and the total amount used. Concentrations of 3.3% and less are ineffective. Pain transmission is blocked at a concentration of 5%. Touch, proprioception, and nociceptive fibers are blocked at concentrations above 5%. The effect reaches

maximum in 2 weeks, followed by maximum recovery in 14 weeks because eventually there is regrowth of most of the axons.<sup>[39]</sup> It has an immediate local anesthetic effect due to its immediate selective effect on smaller nerve fibers. This differential blocking ability is due to small vessel destruction that initially spares large fibers. However, the effects of the block cannot be evaluated until after 24-48 h, to allow time for the local anesthetic effect to dissipate. The neurolytic effect may be clinically evident only after 3-7 days. If inadequate pain relief is obtained after 2 weeks, this may indicate incomplete neurolysis and requires repetition of the procedure.<sup>[41,42]</sup> The subsequent fibrosis that occurs following phenol injection makes nerve regeneration more difficult, but not impossible. Nerve regeneration occurs as long as the nerve cell body is intact, at a rate of 1-3 mm/day. Nerve arborization and neuroma formation can also occur at the site of nerve disruption and can be a focus of neuropathic pain.

Ethyl alcohol has similar destructive effect as phenol and is more efficient in destroying nerve cell bodies. Its mechanism of nerve destruction is similar to that of phenol. It extracts phospholipids, cholesterol, and cerebroside from neural tissues and precipitates mucoprotein and lipoprotein.<sup>[43]</sup> Although 50%-100% alcohol is used as a neurolytic agent, the minimum concentration required for neurolysis has not been established. A concentration of 95% will reliably lyse sympathetic, sensory, and motor components of the nerve. It is hypobaric to the CSF, is readily soluble in body tissues, and produces severe burning pain on injection. It spreads quite rapidly from the injection site and requires 12-24 h before the effects of the injection can be assessed.<sup>[40]</sup>

## COMPLICATIONS OF CHEMICAL NEUROLYSIS

### Skin and other non-target tissue necrosis and sloughing

This is due to damage of the vascular supply to the skin, causing ischemia, and chronic trauma to denervated tissue. Necrosis of muscles, blood vessels, and other soft tissues has also been reported.<sup>[44]</sup>

### Neuritis

The reported incidence of neuritis is up to 10%. It is caused by partial destruction of somatic nerve and subsequent regeneration. Neuritis would occur only if the nerve cell body is not destroyed. It is clinically manifested as hyperesthesia and dysesthesia that may be worse than the original pain problem. It is one of the limiting factors in the use of chemical neurolysis.

### Anesthesia dolorosa

The patient complains of distressing numbness caused by an imbalance in afferent input. It is caused by long-term loss of afferent input and the resultant CNS changes. A local

anesthetic block done a few hours prior to the performance of the neurolytic block seems to prevent the development of this complication. Management of this problem is pharmacotherapy with the use of tricyclic antidepressants, major antipsychotic tranquilizers, and anticonvulsants.

### Prolonged motor paralysis

This can be a major complication and is greatly feared by physicians, patients, and family. It occurs infrequently and is usually temporary.

### Systemic complications

These include hypotension secondary to sympathetic block and systemic toxic reactions, heart rate, and rhythm disturbances, blood pressure changes, and CNS excitation and depression.

### Botulinum toxin injection

Botulinum toxin is the most widely used treatment for focal spasticity. The effect of the toxin is to inhibit the release of acetylcholine at the neuromuscular junction.<sup>[45]</sup> The clinical effect of injecting botulinum toxin is reversible due to nerve sprouting and muscle reinnervation, leading to functional recovery of the muscle in a few months. It is essential that botulinum toxin injections are given in conjunction with physiotherapy in order to obtain the maximum benefit. The toxin is injected directly into the targeted muscle and an effect is noticed in 2-3 days with a maximum effect seen by about 3 weeks, lasting at least 3 months. As it is not a permanent treatment, it may have to be repeated after a few months.<sup>[46]</sup>

### Intrathecal pump

#### Baclofen

If oral drug treatment is inadequate in controlling lower limb spasticity or is not tolerated, intrathecal delivery of baclofen should be considered.<sup>[47]</sup> The main risks of intrathecal baclofen infusion are symptoms related to overdose or withdrawal. These are mostly related to catheter disruption, failure to refill the pump reservoir, or failure of the pump's power source. Abrupt disruption of intrathecal baclofen can be a serious scenario with continuous spasms, tremors, temperature elevation, seizure, and death having been reported.<sup>[2]</sup>

#### Phenol

Use of phenol is reserved for those individuals who do not have any functional movement in the legs, who have lost bladder and bowel function, and who have impaired leg sensation. Intrathecal phenol can be an effective treatment which requires expert administration and does not have the long-term maintenance or cost issues that are associated with intrathecal baclofen treatment. The effect of a single injection often lasts many months and can be repeated if necessary.<sup>[48]</sup>

### Surgical technique

#### Neurosurgical techniques

Anterior and posterior rhizotomy, peripheral neurotomy,<sup>[49]</sup> percutaneous radiofrequency rhizotomy, spinal cord and deep cerebellar stimulation of the superior cerebellar peduncle,<sup>[50]</sup> functional neurosurgery<sup>[51]</sup>

#### Orthopedic procedures

Directly act on muscles and tendons, e.g., lengthening operation, tenotomy, neurectomies, and transfer of tendons.

## CONCLUSION

The management of spasticity is complex involving contribution from physiotherapist, orthopedician, and pain physician. Most individuals, even with quite severe spasticity, can be managed by a combination of physiotherapy and local nerve block or botulinum toxin injection, sometimes combined with relatively low dose oral medication. Neurolytic block with phenol, when adequately indicated, is a tool that presents excellent cost-benefit relation, high margin of safety and rare complications, especially when administered by well-qualified professionals. More advanced intrathecal and surgical techniques are rarely needed. A multidisciplinary approach helps to deal with the problem and improve the quality of life.

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