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The Management of Spasticity in Multiple Sclerosis

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Abstract

Spasticity, defined as a velocity-dependent increase in tonic stretch reflexes, is common in patients with multiple sclerosis due to plaque formation in the brain and spinal cord. Treatment of spasticity is generally considered when the increase in tone interferes with functional activities, such as bed or wheelchair positioning, transfers, ambulation, or daily care; when it is painful; or when it leads to complications such as contractures or skin breakdown. This paper reviews the pathophysiology of spasticity and discusses treatment options, including general medical and nursing cares, physical and occupational therapy approaches, use of splints and orthoses, oral and intrathecal medications, nerve blocks, botulinum toxin injections, and orthopedic and neurosurgical procedures.

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Introduction

Spasticity, defined as an increase in resistance to passive muscle movement or a velocity-dependent increase in tonic stretch reflexes, is a common feature in patients with multiple sclerosis (MS) due to plaques located in the brain and spinal cord. It clinically manifests as "stiffness" in muscles and is often accompanied by painful muscle spasms, weakness, lack of dexterity, fatigability, and co-contraction of agonist and antagonist muscles.[1,2]

In some patients, spasticity may actually be of benefit. For example, in patients with paraparesis and marginal strength, an increase in tone in their antigravity muscles may actually facilitate transfers, standing, and ambulation. In these patients, alleviation of their spasticity could be detrimental, potentially worsening their functional abilities.

In many MS patients, however, spasticity is problematic, interfering with mobility and the ability to perform daily cares. An increase in extensor tone of the trunk musculature may impair a patient's ability to transfer safely or sit comfortably in a wheelchair, while spasticity in the upper extremities may interfere with the ability to manipulate objects with the hands. Increased disability may also result from painful spasms, and poor hygiene.[3,4] Spinal cord plaques often lead to the development of bladder-sphincter dyssynergia, with excessive spasticity in the external urethral sphincter. This can cause urinary retention and ultimately, injury to the kidneys.[5] In these patients, treatment of spasticity should be considered.

The following is a review of the pathophysiology of spasticity, and of management options that can be considered for MS patients with spasticity.

Physiology

The pathways that regulate tone are similar to those that concern voluntary and involuntary motor movements and as a final common pathway involve the spinal reflex arc.[6,7] For activation of a specific muscle, alpha motor neurons in the ventral horn of the spinal cord fire, resulting in muscle contraction. Afferent impulses from the muscle return to the spinal cord via Ia fibers; some of these fibers synapse directly on the agonist alpha motor neurons, allowing for sensory feedback necessary for motor movements. This is a monosynaptic reflex pathway. Collateral fibers from these Ia afferents also synapse on inhibitory neurons in the dorsal horn, which in turn synapse on antagonist alpha motor neurons to inhibit their contraction; this is a polysynaptic reflex.[6] These pathways allow for the coordinated action of agonist and antagonist muscles.

There are several descending central nervous system pathways that synapse directly or indirectly (via internuncial pathways, i.e., pathways with intervening neurons) on the alpha and gamma motor neurons and allow for suprasegmental control of movement. The corticospinal tract synapses directly on motor neurons and is responsible for voluntary control of the extremities, as well as inhibition of antigravity muscles of the trunk and limbs.[6,7] The pontine reticulospinal tract is excitatory to alpha motor neurons, while the medullary reticulospinal tract is inhibitory. The vestibulospinal tract is excitatory to the motor neurons of antigravity muscles.[8] MS plaques in the brain and spinal cord that affect these pathways often result in spasticity. Removal of the descending inhibitory influences due to central nervous system lesions leads to an overactivity of the spinal reflex arc. This results in an increase in muscle tone, hyperreflexia, extensor plantar response (Babinski's sign), flexor spasms, clonus, and co-contraction of agonist and antagonist muscles. Lesions that specifically involve the corticospinal tract may also cause concurrent motor weakness and decreased dexterity.[6]

A number of neurotransmitters are involved in these pathways. Glutamate, an excitatory neurotransmitter, is released by the descending corticospinal tract and from primary spinal cord Ia afferent fibers.[9] Interneurons in the dorsal and intermediate gray matter of the spinal cord are GABAergic and are involved in the polysynaptic pathways discussed above; these interneurons act to mediate presynaptic inhibition of primary Ia afferent inputs on motor neurons.[7] Presynaptic inhibition acts to suppress sensory signals from skin and muscle receptors and to decrease the amount of glutamate released by the primary afferent fibers.[9] Renshaw interneuron cells are glycinergic and are also inhibitory; these interneurons mediate postsynaptic recurrent inhibition of alpha motor neurons and reciprocal Ia fiber inhibition.[9] It is thought that these inhibitory pathways may be underactive in some patients with spasticity.

Substance P is released by small, predominantly unmyelinated sensory afferent fibers that mediate pain. In patients with underlying spasticity, painful stimuli often result in an increase in muscle tone and painful spasms, likely triggered by an increase in Substance P release in the dorsal horn of the spinal cord.[6]

Descending pathways containing catecholamines and serotonin are also involved in the regulation of spinal cord reflexes. These pathways primarily affect the transmission of impulses from primary sensory afferent fibers and affect the excitability of interneurons.[8].

Treatment of Spasticity

Treatment of spasticity is generally considered when the increase in tone interferes with functional activities, such as positioning in bed or wheelchair, mobility, or activities of daily living; affects hygiene; or causes complications such as decubitus ulcers, urinary retention, pain, or early contractures. The patient's underlying neurologic and medical conditions should always be considered when planning treatment. For example, multiple sclerosis patients who

have fatigue or cognitive impairment may not tolerate drugs that are sedating. Furthermore, as discussed previously, patients with marginal strength may lose functional abilities if treated with medications that cause muscle weakness. Therefore, the pros and cons of the various treatments must always be considered for each individual patient.

Nonpharmacologic Interventions

Noxious stimuli, especially those involving the lower extremities, can worsen spasticity and induce painful leg spasms. This is caused by an increase in nociceptive afferent neuronal activity and heightened release of Substance P, which results in an increase in segmental spinal reflex activity. Initial evaluation of a spastic patient, or one in whom spasticity or flexor spasms suddenly worsens should always include a search for potential noxious stimuli, including urinary tract infections, bladder distension, bowel impaction, ingrown toenails, decubitus ulcers, and deep venous thrombosis.[8] Treating these underlying conditions exclusively will often improve tone and spasms and obviate the need for additional interventions.

Passive range of motion, performed at least 2 to 3 times a day, should be an integral part of any treatment regimen for spasticity. Although the reduction in tone lasts only for several hours, daily range of motion exercises may help prevent the development of contractures.[10]

There are certain physical and occupational therapy approaches, such as the neurodevelopmental technique, that stress the inhibition of spasticity as part of their treatment strategies.[11] Although studies have not demonstrated better functional outcomes in patients treated with these techniques compared to others, patients with prominent spasticity might be expected to benefit from these approaches.[12]

Electrical stimulation may also be of benefit for patients with spasticity. The most common techniques involve percutaneous or transcutaneous stimulation of spastic antagonist muscles.[13,14] This is thought to reduce tone by activation of sensory afferent fibers that activate inhibitory polysynaptic spinal cord reflex pathways, discussed above.[15] Direct electrical stimulation of the weak agonist muscles may also reduce tone by inducing reciprocal inhibition of the spastic antagonist muscles.[15] The decrease in spasticity generally lasts from 15 minutes to 3 hours, allowing the therapists to do more aggressive range of motion and strengthening exercises during this time. However, the long term value of cutaneous electrical stimulation in reducing tone and improving overall functional abilities has not been demonstrated.

Transcutaneous and epidural stimulation of the spinal cord have also been used in the treatment of spasticity.[16] It is thought that spinal cord stimulation might act to inhibit the hyperexcitable spinal cord reflexes, but the precise mechanism of action is not known.[17] There are several small studies of multiple sclerosis patients that demonstrate an improvement in spasticity, spasms, and bladder function in those treated with spinal cord stimulation.[18,19] Also, a recent study showed reduced spasticity in multiple sclerosis patients treated with magnetic stimulation of the spinal cord.[20]

Anecdotal reports suggest that other therapy modalities might help reduce muscle tone. Use of the muscle vibrator, especially when applied to antagonist muscles, may help reduce tone in a spastic limb.[21] Topical cold and anesthetics may reduce tone by decreasing the sensitivity of cutaneous receptors and slowing nerve conduction.[22,23] There are also case reports of improved spasticity following acupuncture.[24]

Splints and orthotic devices are often used in the treatment of spasticity, to reduce tone, prevent contractures, and reduce pain. Splints act mechanically by reducing muscle spindles' reaction to stretch and are generally applied to position affected joints in opposition to the tonal pattern. Although clinical observations suggest tone to be reduced with splints, studies have not determined the most effective design.[25–27] Orthotic devices control joint instability and may

alter the loading of a limb to prevent stretch reflex activity in antagonist muscles resulting in tone reduction.[28,29] Both static and dynamic orthoses provide prolonged muscle stretch and can be used to prevent or correct early contractures that result from chronic spasticity. As well, devices such as ankle-foot orthoses (AFO's) may help improve functional abilities, such as ambulation, if focal muscle weakness or spasticity interferes with these tasks.[28]

Plaster casting, an extension of splinting, acts to reduce tone by inhibiting the response of Ib afferent fibers arising in the Golgi tendon organs.[30,31] Prolonged immobilization through serial casting appears to be most effective for managing mild soft tissue contractures resulting from spasticity.[32] With serial casting, an extremity is positioned in full stretch and casted in that position; the cast is left in place for 7 to 10 days and is then removed. The patient undergoes range of motion therapy between castings and is then recasted at the new (reduced) angle; this is repeated 4 to 5 times. After the cast is removed, further therapy and splinting is often needed to maintain the improved range of motion.[32,33] It is suggested that serial casting is most effective within 6 months of an acute injury, while neurologic recovery is still possible.

Pharmacologic Interventions

A number of medications are beneficial in the treatment of spasticity. All of these drugs act to decrease the excitability of spinal reflexes.[9] In choosing a medication, one must account for a variety of factors, including the underlying neurologic disease, concurrent symptoms, medical problems, and medications.

Baclofen is a GABA agonist, active primarily on type $GABA_B$ receptors, and inhibits both monosynaptic extensor and polysynaptic flexor reflexes.[9] Baclofen acts presynaptically to reduce the release of excitatory neurotransmitters from the descending corticospinal tracts and primary spinal cord Ia afferent fibers, and possibly of Substance P from afferent nociceptive fibers.[7] At high concentrations, baclofen also acts postsynaptically to decrease the effects of the excitatory neurotransmitters, thus inhibiting activation of motor neurons in the ventral horn.[6]

Baclofen is effective for the treatment of spasticity secondary to both brain and spinal cord diseases, including multiple sclerosis.[34] In addition to reducing tone, baclofen is effective in diminishing painful flexor and extensor spasms.[35] The use of baclofen is limited by its sedative side effects and tendency to cause muscle weakness, especially at high doses.[36] Less common side effects include ataxia, confusion, headache, hallucinations, dyskinesias, and respiratory and cardiovascular depression.[7,37] Baclofen can lower seizure threshold and should be used cautiously in epileptic patients.[8] The drug is generally started at a low dose (5-10 mg qd, bid) and titrated upward to a maximum of 120 to 200 mg/day (divided tid-qid). Abrupt withdrawal should be avoided as this can precipitate rebound flexor spasms and hallucinations.[7]

Tizanidine, an imidazoline derivative, is the newest of drugs approved for the management of spasticity. It is an $alpha_2$-adrenergic agonist active at both $alpha_2$-adrenergic and imidazoline receptors in the spinal cord.[38,39] It is thought to have several mechanisms of action resulting in a decrease in polysynaptic spinal cord reflex activity, including inhibition of the release of excitatory neurotransmitters from presynaptic sites and of Substance P from nociceptive sensory afferents.[40–42] Tizanidine also decreases neuronal firing at the level of the locus ceruleus.[39] Tizanidine has been shown to be effective in reducing spasticity and spasms in patients with multiple sclerosis, and other brain and spinal cord diseases with efficacy similar to baclofen.[39,43] It is currently under investigation for the treatment of neuropathic pain, and detrusor-sphincter dyssynergia, which are often seen in multiple sclerosis patients. The major advantage of tizanidine over baclofen is that tizanidine does not cause muscle weakness. Therefore, tizanidine may be of particular benefit, and may be the drug of first choice, in patients with marginal strength.

The sedative side effects of tizanidine are somewhat equivalent to that of baclofen, but less than benzodiazepines.[43] Other side effects include dry mouth and hypotension, which is generally dose related.[44] The side effects are minimized by slow titration of dosage. Slight elevation of liver enzymes have been noted in 5% of patients; these generally normalize with a decrease in dosage or discontinuation of medication.[43] It is recommended that liver enzymes be monitored during the first 6 months of treatment and that the drug be avoided in patients with liver disease. Initial dosage is 2 to 4 mg/d and is slowly titrated upwards in 2 to 4-mg increments every 3 to 4 days to a maximum of 36 mg/d divided tid-qid.[35] Peak effect is in 1 to 2 hours with 3- to 6-hour duration of action.[33] A sustained release formulation of tizanidine is currently under investigation for the management of spasticity in multiple sclerosis patients.

Benzodiazepines enhance presynaptic and postsynaptic inhibition in the spinal cord by enhancing the affinity of GABA receptors for endogenous GABA.[6,8] Similar to baclofen and tizanidine, benzodiazepines are effective in reducing spasticity from both spinal cord and cerebral injuries.[45] Although studies suggest that benzodiazepines have similar efficacy to baclofen,[36] their use is limited by side effects, including habituation and tachyphylaxis, sedation, and fatigue.[6] Benzodiazepines are probably most appropriate for patients with nocturnal spasms or for those who can benefit from the sedative/anxiolytic side effects. The usual dosage of diazepam is 2 to 10 mg bid-qid, and 0.5 to 3 mg at bedtime for clonazepam.

Dantrolene interferes with the excitation-coupling reaction in skeletal muscle by inhibiting depolarization-induced release of calcium from the sarcoplasmic reticulum.[46] Because dantrolene acts directly on the muscle it is effective in reducing spasticity of both cerebral and spinal origin. The biggest limiting feature of dantrolene is that it tends to cause marked weakness in addition to reduction of tone. Because of this, dantrolene is probably best used to manage spasticity in severely affected quadriplegic patients in whom an increase in weakness will not worsen their functional abilities. The most serious potential side effect of dantrolene is a 1% incidence of hepatotoxicity and 0.1% incidence of fatal hepatitis.[6] Liver function tests should be monitored regularly with the dosage decreased or the medication discontinued if the liver enzymes are elevated. Dantrolene is typically started at 25 to50 mg/d and slowly increased to a maximum of 100 mg qid.

There are other medications that can be considered in patients with refractory spasticity. Clonidine is an alpha$_2$-adrenergic agonist similar to tizanidine, but is limited in its use by orthostatic hypotension. It is started orally at 0.05 mg bid and increased by 0.1 mg/d weekly to a maximum of 0.4 mg/d.[47] It may also be administered transdermally 0.1 to 0.3 mg.[48] Phenothiazines, such as chlorpromazine, have been shown to decrease spasticity, probably due to their alpha-adrenergic blocking properties. They reduce gamma motor neuron excitability and antagonize the postsynaptic actions of dopamine.[8,9] Their use is limited by sedation and development of extrapyramidal side effects, including tardive dyskinesia.[6] Gabapentin, given adjunctively at doses of at least 400 mg tid, has been shown to be effective in reducing spasticity in several series of multiple sclerosis and spinal cord injured patients.[49–51] Cyprohepatadine also has antispasticity effects, at doses of 4 to 16 mg per day.[52,53] Its major side effects include sedation, dry mouth, and an increase in appetite. Finally, there are several studies that report benefit with valproic acid given at doses of 250 mg tid.[54,55] The benefits need to be confirmed in large clinical trials.

Intrathecal Medications

Approximately 30% of patients do not achieve adequate control of their spasticity with oral medications, or are unable to tolerate them due to side effects.[56] In these patients, intrathecal administration of baclofen via an implantable pump may be an alternative. Intrathecal baclofen has been shown to be effective in the management of spasticity in multiple sclerosis and other central nervous system disorders, including cerebral palsy, traumatic brain injury, and spinal cord injury.[28,56–66]

As an initial evaluation for the pump, patients undergo a screening trial. A lumbar puncture is performed and 50 mcg of baclofen is administered intrathecally. The onset of action is within 30 to 60 minutes and peaks at 2 to 4 hours. An Ashworth Scale score is recorded for lower extremity muscles (hip abductors and flexors, knee flexors, and ankle dorsiflexors) at baseline and at 1, 2, 4, and 8 hours post-administration;[67] a positive response is defined as an average of a 2-point drop in the score sustained for 4 to 8 hours.[56] If there is no improvement with the 50-mcg test dose, a 75- or 100-mcg dose can be given the following day. If there is no response to a 100-mcg test dose, the pump should not be implanted and alternative treatments should be sought. On the other hand, should any of the trial doses be effective, surgery for implantation of the intrathecal pump is warranted.

Surgically, a catheter is placed into the intrathecal space at the L3-4 level with the tip threaded rostrally to approximately the T10 level. The other end of the catheter is connected to a medication pump, which is implanted in the peritoneal cavity and filled with baclofen. The starting pump dosage is twice that of the test bolus that produced a positive effect (e.g., if 50-mcg test bolus was effective, then the pump is started at 100 mcg/d). The dosage can then be titrated upwards by 5% to 15% daily until a satisfactory response is obtained. The pump can be programmed to administer baclofen on a continuous basis, and/or to provide boluses at specific times during the day. The pump is refilled percutaneously every 4 to 12 weeks depending on the type of reservoir used.

The most common side effects of intrathecal baclofen include local infection, orthostatic hypotension, sedation, loss of erections, and postsurgical pseudomeningoceles.[57] In patients who develop sudden worsening of their spasticity, one must always consider the possibility of pump failure, exhaustion of the pump reservoir, or disruption of the catheter. Baclofen overdoses can result in respiratory depression and coma; in these instances physostigmine may reverse the symptoms.

Morphine may also be administered intrathecally via an implantable pump. It is effective in reducing spasticity by inhibition of polysynaptic reflexes in the spinal cord through its action at opiate receptors.[6] It has been shown to improve pain and spasticity associated with spinal cord injuries.[68] Because of the potential development of habituation, use of intrathecal morphine should be considered only for patients refractory to other forms of treatment.

Nerve Blocks and Botulinum Toxin Injections

Patients with focal spasticity, i.e., an increase in tone that is particularly severe in isolated muscle groups, often benefit from locally directed treatments, used alone or in combination with the management strategies described above. Focal treatment options include use of nerve blocks and botulinum toxin; these injections selectively weaken spastic muscles. Clinical indications would include, for example, a quadriplegic multiple sclerosis patient in whom excessive hip adductor spasticity interferes with positioning and the ability to perform adequate perineal hygiene. Such a patient might specifically benefit from an obturator nerve block or botulinum toxin injections in the adductor muscles. Similarly, a patient with severe ankle plantar flexor and invertor tone that impairs the ability to wear an ankle foot orthosis, or one who has excessive wrist and finger flexor spasticity that cannot be adequately managed with a resting hand splint might benefit from focally directed interventions. Focal reduction of tone will also allow therapists to perform more complete range of motion exercises and help prevent contractures that might necessitate the need for more definitive surgical procedures, such as tendon releases.

Phenol is the most commonly used agent for nerve blocks. Phenol is neurolytic; when applied locally it destroys myelin and axons but spares the endoneurial tubes.[69] The clinical effect lasts 1 to 12 months. Generally, strength returns as regenerating axons eventually reinnervate the motor endplates, although occasionally permanent muscle weakness develops. Ethanol,

although not used as commonly as phenol, is also a neurolytic agent and has been shown to be effective in reducing focal spasticity.[70] Local anesthetics, such as lidocaine, block nerve transmission by decreasing sodium channel permeability. These agents cause a short-lived reduction in tone (hours) and can be used to help predict the effect of a nerve block or distinguish between spasticity and contracture before a longer-acting drug like phenol is administered.[71] The most common side effect of nerve blocks is pain; however, local infection, lightheadedness, nausea and vomiting, and thrombophlebitis occasionally occur.[71]

There are different nerve block techniques. Originally, closed perineural injections of nerve trunks were commonly performed. However, because mixed (sensory and motor) nerves are injected, this technique is often complicated by development of painful paresthesias and has largely been abandoned.[72] Development of dysesthesias can be avoided by doing closed motor branch blocks. With this technique a nerve's motor branch is identified with a neurostimulator to ensure appropriate position for the phenol injection. Commonly blocked nerves using this technique include the recurrent branch of the median, obturator, and musculocutaneous nerves. Although this technique is effective, the response is generally not as long lasting or predictable as an open motor point block.[73]

Open motor point blocks require surgical isolation of motor nerve branches. Nerves often blocked with this method include the motor branches of the median and ulnar nerves in the forearm, deep motor branch of the ulnar nerve at the wrist, obturator nerve, sciatic motor branch in the posterior thigh, and posterior tibial nerve. The clinical effect with this technique lasts 2 to 8 months.[74] The obvious drawback to this procedure is that surgery is required.

With intramuscular motor point blocks, specific motor points within muscle are injected. The motor points, areas of high concentration of motor endplates, are identified using a needle stimulator. One can titrate the degree of tone reduction and weakness desired by the number of motor points that are blocked. Although this technique results in decreased spasticity of shorter duration than open branch blocks, the major advantage is the ease of administration.[75] In adults these injections are performed while awake; however, in children deep conscious sedation is generally recommended.

Botulinum toxin is a thermolabile exotoxin produced by the Clostridium botulinum bacteria. There are 7 serotypes of the toxin, A through G. Type A (BTX-A) is the most potent and the only form commercially available at present; however, type B is currently under investigation. The site of action of botulinum toxin is at the neuromuscular junction where it inhibits the release of acetylcholine from presynaptic nerve terminals. This results in functional denervation of muscle fibers, which causes focal muscle weakness and a decrease in muscle tone.[76]

Currently BTX-A is FDA approved only for the treatment of strabismus, blepharospasm, and hemifacial spasm but is used commonly to treat other conditions, including focal dystonias and spasticity. A number of studies have shown BTX-A to be effective in reducing upper and lower limb spasticity and painful spasms in patients with multiple sclerosis and other disorders, including stroke, cerebral palsy, spinal cord injury, and traumatic brain injury.[77–92] It has also been advocated for the treatment of external urethral sphincter dyssynergia.[93]

The specific spastic muscles that are to be injected are identified by electromyogram (EMG) guidance.[94] The number of BTX-A units injected depends on the particular muscle and degree of weakness desired. After intramuscular injection, there is a delay in the onset of clinical effect for 24 to 72 hours. The duration of action averages 2 to 6 months; there is a gradual wearing off of the effect due to nerve sprouting, which eventually reinnervates the muscle fibers. It is recommended that repeat injections be spaced at least 12 weeks apart as neutralizing antibodies are more likely to develop if injections are more frequent.[95] Side effects of BTX-A

include local skin reactions and pain at the site of injection. Weakness can occur both in injected muscles and adjacent noninjected muscles.

Advantages to use of phenol injections include an immediate response (in contrast to botulinum toxin), low cost, and ease of sterilization and preparation. The major advantages of BTX-A are the relative ease of administration and availability of this procedure at many local centers.

Orthopedic Procedures

There are several orthopedic procedures that can be considered in the management of focal spasticity and contractures, including tenotomies, tendon transfers, and tendon lengthenings. Tenotomies involve the release of muscle tendons of severely spastic muscles, and are generally performed on muscles without voluntary movement. Tendon lengthenings are performed to weaken spastic muscles and position joints at a more natural and functional angle. Tendon transfers are done to allow partially functional muscles to perform more useful movements.

In the lower extremity, the most common procedures are: hamstring tendon lengthening to correct knee flexion contractures; Achilles tenotomy or lengthening to correct equinus deformity; adductor tenotomy to reduce hip adductor spasticity; iliopsoas tenotomy to alleviate painful flexor spasms of the hip; toe flexor tenotomies to correct claw foot deformities; and split anterior and posterior tibialis tendon transfers to correct foot and ankle posture and improve gait.[96,97] Similar procedures may be considered for upper extremity deformities, such as thumb-in-palm posturing or wrist contractures, although generally they do not improve function to the degree that the lower extremity surgeries do.[98,99]

Neurosurgical Procedures

Although not generally used for the management of spasticity due to multiple sclerosis, selective dorsal rhizotomy is commonly used in the management of children with cerebral palsy. Studies have shown that surgically ablating specific dorsal rootlets (levels L2-S2) that are hyperexcitable are effective in improving lower extremity spasticity, standing, sitting, and ambulation with benefit persisting for at least 10 years.[100–103]

For extreme refractory cases of spasticity, invasive surgical procedures can be considered. Spasticity can be reduced by a myelotomy, which involves severing the segmental reflex arc in the gray matter. Recent modifications of the procedure allow for preservation of lateral column and white matter tract function.[104] This procedure is irreversible and can cause permanent loss of bowel and bladder function. A cordectomy, which involves sectioning part of the cord, is also an irreversible procedure. This procedure results in flaccid paraplegia and permanent bowel and bladder dysfunction.[8]

Conclusions

Spasticity and painful spasms are common in patients with multiple sclerosis and can cause or worsen disability. Treatment is recommended for patients in whom an increase in tone worsens function or causes medical complications. Available therapeutic options include basic nursing cares, physical and occupational therapy approaches, orthotic devices, oral and intrathecal medications, and surgical interventions. As with any form of treatment, management strategies should be considered on an individual basis.

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