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Spasticity and its management

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One of the most common medical complications associated with upper motor neuron (UMN) disease is spasticity. It is frequently seen in the context of spinal cord injury (SCI), multiple sclerosis (MS), cerebrovascular accident (CVA), cerebral palsy, and traumatic brain injury. Spasticity can potentially be a significant barrier to the functional capacity of patients and a major cause of pain. If not treated, it may result in a decline in function, development of contractures, and an increase in dependency for basic living needs. Although physiatrists and neurologists are predominantly involved in managing spasticity, some pain physicians and spine surgeons also treat it through intrathecal baclofen pumps and use of local injections. Understanding the pathophysiology of the disease and the variety of available treatments is important for appropriate patient care. This issue of *Pain Management Rounds* presents an overview of the diagnostic and treatment approaches to spasticity.

DEFINITION AND PREVALENCE

Lance described spasticity as a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes.[1] The term "velocity-dependent" means "the faster the passive movement of the limb through its available range, the greater the increase in muscle tone." This widely-accepted definition was broadened by Young to include other signs like exaggerated deep tendon reflexes, clonus, flexor/extensor spasm, the Babinski response (Babinski's sign), exaggerated phasic stretch reflexes, hyperactive cutaneous reflexes, increased autonomic reflexes, and abnormal postures. All of these signs describe manifestations of excessive involuntary motor activity.[2]

The prevalence of spasticity is unknown, but it is thought that approximately a half million people in the USA and, possibly, 12 million worldwide are affected.[3] In a spinal cord injury population, Levi et al reported that 68% experienced spasticity and 41% of these experienced pain or limitation of activities as a result of spasticity.[4] Rizzo et al analyzed a cross-sectional database of >17,000 patients with MS (the North American Research Committee On Multiple Sclerosis [NARCOMS] registry). Of these patients, 15.7% had no spasticity, 50.3% had minimal-to-mild spasticity, 17.2% had moderate spasticity, and 16.8% had severe spasticity.[5] A review of spasticity after stroke revealed that it affects <25% of stroke victims.[6]

ETIOLOGY OF SPASTICITY

There are numerous causes for spasticity. It is important to know both the location of the central nervous system (CNS) injury and the pathology involved to develop an appropriate treatment plan. The most common causes of spasticity are traumatic brain injury, stroke, MS, cerebral palsy, and SCI (Table 1). Development of spasticity is irrespective of gender, but there are gender differences in the different etiologies linked to spasticity.[7,8] Spasticity is only one component of the UMN syndrome, which results from damage to descending pathways at the level of the cerebral cortex, brainstem, or spinal cord. In the acute phase of a UMN lesion, flaccid limbs and hyporeflexia predominate in the clinical picture. In the days to weeks after a UMN injury, other clinical signs develop, including spasticity, which is often associated with clonus, the clasp-knife phenomenon, hyperreflexia, Babinski's sign, and flexor or extensor spasms. The existence of spasticity without these associated signs should lead clinicians to consider other neurologic diagnoses like dystonia and Parkinson's disease. The interval between UMN injury and the appearance of spasticity varies from days to months according to the level of the lesion and the severity of CNS damage.

PATHOPHYSIOLOGY

The mechanism for spasticity is not well understood. It is theorized that it is caused by an increase in the reaction of the stretch reflex, which becomes active when muscle is rapidly stretched out. With this reflex, muscle contracts and resists the force that is stretching it. To allow normal movements, this reflex often needs to be "turned off." Inhibitory signals, traveling from the cerebral cortex to alpha motor neurons of the spinal cord through the reticulospinal and corticospinal tract, are thought to reduce the stretch reflex. Damage to the cerebral cortex (eg, CVA, traumatic brain injury, MS, cerebral palsy, intracranial tumors) or the

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TABLE 1: Etiology of spasticity

Direct injury to motor cortex:
- Hypoxia
  - Prenatal utero-placental insufficiency
  - Perinatal asphyxia
  - Near drowning
  - Suffocation
- Cerebral palsy
- Trauma
- Tumor
- Stroke (CVA)
- Infection (Viral encephalitis)
- Brain malformation

Corticospinal tract injury in the brain:
- Tumor
- Stroke
- Multiple sclerosis

Corticospinal tract injury in the spinal cord:
- Spinal cord traumatic injury
- Spinal ischemia
- Spinal tumor
- Tethered cord
- Epidural abscess
- Multiple sclerosis
- Syringomyelia
- Transverse myelitis
- Spina bifida

Degenerative CNS disease (rare):
- Ceroid lipofuscinosis
- Rasmussen's encephalitis
- Tay-Sachs disease
- Sialidosis
- Rett syndrome
- Pelizaeus-Merzbacher syndrome
- Tropical spastic paraparesis
- Spinocerebellar ataxias

corticospinal tract (SCI, spinal tumor, epidural abscess, spina bifida, tethered cord, MS, transverse myelitis, syringomyelia, spinal ischemia) results in a decreased inhibition of the stretch reflex, hence, spasticity.

Prolonged disinhibition of the stretch reflex results in a decreased stimulation threshold in response to triggers and physiologic shortening of the muscle fibers to the extent that complete relaxation becomes difficult and patients lose range of motion. The neurotransmitters involved in the inhibitory input to alpha motor neurons are particularly influenced by gamma aminobutyric (GABA-ergic) interneurons operating under the control of presynaptic inhibition. GABA-agonist medication mediates the inhibitory signal and decreases unwanted tone (ie, spasticity).

CLINICAL DIAGNOSIS

A physical examination is the single most important method of detecting spasticity in patients with UMN lesions. Since there is fluctuation in spasticity throughout the course of the disease, serial examinations are needed and disease status and treatment efficacy require careful follow-up. Examinations include the visual observation of patient movement and gait, palpation of affected limb(s), determination of available range of motion, evaluation of deep tendon reflexes, performance of clinical tests to elicit Babinski's and long-tract signs, and evaluation of the existence of clonus, clasp-knife phenomenon, and spastic catch.

In assessing spasticity, it is important to identify overactive muscles or muscle groups and determine the effect of spasticity on the patient's function, mobility, activities of daily living, and vocational activity. A critical and difficult part of the physical examination is isolating the spastic muscle(s). Physicians can use an electromyogram (EMG) or diagnostic blocks with anesthetics to determine the spastic muscle groups and test available range of motion and strength. Based on the response to these diagnostic blockades, the possible outcome of denervation treatments can be determined.[9]

TABLE 2: Spasticity scales

Score	Ashworth Scale[10]	Modified Ashworth Scale[11]
0	No increase in tone	No increase in tone
1	Slight increase in tone giving a catch when the limb was moved in flexion or extension	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion (ROM)
1+	N/A	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (<50%) of the available ROM
2	More marked increase in tone, but limb easily flexed.	More marked increase in muscle tone through most of the ROM (>50%)
3	Considerable increase in tone, passive movement is difficult.	Considerable increase in muscle tone, passive movement is difficult.
4	Limb rigid in flexion or extension.	Affected parts rigid in flexion or extension

Spasticity scale

Measurement scales have been developed by healthcare providers to provide a common understanding of spasticity, monitor treatment, and detect worsening disease status. Ashworth developed the most commonly used scale in 1964 and, in 1987, Bohannon and Smith developed the *Modified Ashworth Scale*, which is frequently used by practitioners (Table 2).[10,11] Other known measurement tools include the *Spasm Frequency Scale* and the *Global Assessment of Spasticity*. Functional scales such as the *Functional Independence Measure* or *Gross Motor Function Measure* may be valuable as well, but they do not directly measure spasticity.

The spasticity management team

Before treating spasticity, the treatment team needs to be identified. This usually involves a physiatrist/neurologist who is comfortable with the diagnosis and medical treatment of spasticity and can oversee different aspects of care and coordinate the teamwork. Other key players are members of the rehabilitation team, including physiotherapists and occupational therapists, who will continually work with the patient to achieve independence and decrease disability. Consultation with the neurosurgical team is indicated if neuroablation procedures are being considered. In addition, orthopedic surgeons will be involved if contracture release, tendon lengthening, osteotomies, or joint fusions are required.

Good and bad spasticity

It must be emphasized that practitioners should always have functional goals in mind when treating spasticity and the reason behind treating the tone should be determined before starting treatment. Occasionally, treatment may result in a decline in function in patients with underlying weakness. Furthermore, when deciding to treat a spastic muscle, it is important to know the role of the antagonistic muscle groups. While often weak, these muscle groups themselves may be spastic. Treating the agonist muscle without the antagonist may create additional functional problems. The reasons for treating spasticity include improving gait, trans-

fers, hand function, and sleep; facilitating bracing and hygiene; alleviating pain; and normalizing appearance.

Spasticity may have some benefits for some patients, eg, compensation for weakness, retardation of atrophy, decreased risk of deep vein thrombosis (DVT) and, possibly, a decreased risk of osteoporosis. If spasticity is compensating for weakness, special attention should be given to retaining the functional capacity of the patient.

TREATMENT OF SPASTICITY

In most cases, an individualized treatment plan is required for optimal effect. Its main components include the prevention of aggravating factors, rehabilitation, bracing and casting, local injections, oral medications, an intrathecal baclofen delivery system, and surgery.

Prevention of aggravating factors

Several factors can exacerbate spasticity, such as infections (eg, urinary tract, pneumonia, etc), pain (eg, ingrown toenail, ill-fitting orthotics), occult fractures, DVT, bladder distention, bowel impaction, cold weather, and fatigue. These common conditions need to be addressed prior to adjusting spasticity treatment. Physicians should consider the primary pathology behind the patient's spasticity and the possibility of associated late medical complications, such as SCI and tethered cord syndrome, traumatic brain injury and hydrocephalus, and cerebrovascular accident and late intracranial bleed. These late complications usually result in a sudden increase in spasticity and muscle spasm. Late complications must be identified and treated before adjusting spasticity treatment.

Rehabilitation

The rehabilitation team includes physio-, occupational and, occasionally, vocational therapists using sustained stretch, thermomodalities, massage, vibration therapy (based on reciprocal inhibition), biofeedback, functional electrical stimulation, and hydrotherapy, when appropriate. Orthotics are used to maximize the effect of medications or procedures. In spastic patients undergoing orthopedic procedures, perioperative rehabilitation in combination with medical intervention markedly improves surgical outcomes. Working on vocational skills in the light of predicted disability is also important and can be especially helpful for motivated patients.

Bracing and casting

Splinting and bracing are used to prevent deformity, hold a limb in a functional position, and reduce pain. Serial casting, in combination with local injections and oral medications, may help restore muscle length, as well as range of movement. These interventions need to be modified frequently as improvements occur. Close communication between the rehabilitation team and physicians is important for making timely adjustments to orthotic devices. The pediatric population will require modification or fabrication of new braces more frequently to adjust for rapid growth.

Local injections

The advantages of local injections include selectivity, greater intensity of effect, reversibility, and avoidance of systemic side effects (eg, sedation, cognitive effects, hepatotoxicity, etc.). Disadvantages include pain with the injection, impracticality for a large number of muscles, and the anatomic inaccessibility of some muscles (eg, the subscapularis). Many agents are used for injections, but local anesthetics (for peripheral nerves), phenol/alcohol (for motor nerves), and botulinum toxin (for muscle fibers themselves) are the most common. Regardless of which local injection is used, aggressive rehabilitation during the blockade takes full advantage of the injection.

Peripheral nerve blockade with anesthetics is generally used as a diagnostic tool to assess effects on gait or to distinguish spasticity from contractures. The precise choice of anesthetic is based on the desired length of action.

Phenol block: Phenol is a carbolic acid and in different concentrations has different effects ranging from antiseptic, to anesthetic, to neurolytic. The latter is used to treat spasticity. A 5% to 6.5% concentration is most commonly used. When phenol is in contact with motor nerves, it produces demyelination, destruction of axons (axonotmesis), and an immediate decrease in spasticity. It takes 3-6 months for valerian regeneration and during this period, spasticity is controlled. A neurostimulator is used to localize the targeted nerve(s). Local pain, fibrosis at injection site, and swelling are undesirable sequelae of phenol block. However, the worse side effect is probably dysesthesias, which can be prevented by choosing nerves that have "motor only" function (eg, musculocutaneous nerve for forearm flexor spasticity).

Botulinum toxin: This is the newest addition to local injections and has been used in many patients. It results in a desirable, prolonged, yet reversible, control of spasticity.[12] Multiple serotypes have been developed, but the most commonly used (FDA approved) are serotypes A and B. Botulinum toxin is internalized through the presynaptic membrane and by cleaving the SNAP-25 protein (synaptosome-associated protein of 25 kd), inhibits the attachment and excretion of acetylcholine vesicles. The end result is decreased acetylcholine release at the neuromuscular junction and relaxed muscle.[13,14]

Once botulinum toxin is injected into the targeted muscle, a decrease in spasticity becomes obvious in 1-2 weeks. The effect may last anywhere from 3-4 months. Recurrence of spasticity depends on the rate of new axon terminal growth, which is the result of re-establishing neuromuscular transmission. Since it is a foreign substance, antibodies to botulinum toxin may form. To prevent this, prolonged intervals between exposures are recommended (minimum, 3 months). Once antibodies develop, that serotype is no longer effective and an alternative serotype may be used.

Side effects and complications of botulinum toxin, although infrequent, include allergic reactions, infection, muscle atrophy, diffusion of toxin into adjacent muscles resulting in unwanted effects (eg, dysphagia in treatment of cervical dystonia),[15] and transient flu-like symptoms. Comparisons between botulinum injection and phenol block are listed in Table 3.

Oral medications

Antispasmodics are the most widely used treatment for spasticity in clinical practice; however, their use is often challenging because of significant side effects, This is particularly the case when treating children with baseline cognitive deficits and spasticity. The most common side effects are drowsiness, cognitive impairment, and mood alteration. If the adverse effects are mild and tolerated by the patient, antispasmodics tend to be very effective in treating spasticity. Polypharmacy in treating spasticity may result in amplification of side effects. The choice of antispasmodic depends on the level of injury and the site of action for that particular medication. Each agent has a different mechanism of action for producing relaxation of hyperactive muscle(s). In general, antispasmodics are GABA analogs, α-2 adrenoreceptor

TABLE 3: Comparison of botulinum toxin injection to phenol block

Botulinum toxin	Phenol
Targets single muscle	Targets single nerve innervating a muscle(s)
Easy to administer	Technically more challenging to administer
Multiple injection sites at the same time	Single injection site
Lasts 3 months	Lasts 3-6 months
Delayed effect	Immediate effect
Loss of nerve terminals	Axonotmesis and scarring
Reversible	Usually reversible
No trial drug	Trial with anesthetics to predetermine efficacy
Reduces strength	May reduce strength
No dysesthesias	Possible dysesthesias
Occasionally painful to inject	Could be painful to inject
Expensive – 500 US$/100 units	Cheap – 2 US$/1 gm

agonists, direct muscle relaxing agents, or central acting relaxants.

***Benzodiazepines*:** These are commonly used for treating spasticity in SCI and MS. Benzodiazepines bind at the brain stem and spinal cord levels and increase the affinity of GABA (inhibitory neurotransmitter) to the GABA-A receptor complex, reesulting in an increase in presynaptic inhibition and a reduction in monosynaptic and polysynaptic reflexes.[16] In general, benzodiazepines have a long half-life (18-20 hours) and a small starting dose, gradual increases, and divided dosing are recommended. As with many muscle relaxants, sedation, hypotension, weakness, incoordination, memory impairment, ataxia, confusion, adverse gastrointestinal effects, and depression may occur. Problems of tolerance, dependency, abuse, and potentially dangerous withdrawal (eg, seizures) also need to be considered when starting this group of medications.

Baclofen (oral): Commonly used in spasticity of spinal cord origin (SCI and MS), baclofen binds to GABA-B receptors and blocks release of substance P, an excitatory neurotransmitter from primary afferent terminals.[17] Both monosynaptic and polysynaptic reflexes are affected by its action, resulting in a decrease in clinical manifestations such as clonus, spasm, and decrease in tone.[18] Baclofen has a 4-hour half-life.

A recent large study by Rizzo et al demonstrated that baclofen is the most commonly used antispasmodic after agents like tizanidine and the benzodiazepines. Its use in intrathecal delivery systems results in improved spasticity with decreased side effect profiles. A comparison between 198 patients using intrathecal baclofen and >315 patients using oral medications demonstrated that those receiving the intrathecal delivery had less spasticity, leg stiffness, pain, and spasms.[5] Sedation, ataxia, weakness, and fatigue are common side effects of baclofen and it must be used with care in patients with renal insufficiency since its clearance is primarily renal. When the decision is made to taper the patient off baclofen, a slow taper is crucial. Complications of abrupt stoppage include seizure, rhabdomyolysis, and multiorgan failure that may result in death.

Tizanidine: This drug belongs to the imidazoline family and is a good choice to treat spasticity associated with traumatic brain injury, SCI, and MS.[19,20] Data also support its use in stroke patients to preserve muscle strength and decrease tone, motor reflexes, and painful spasms.[21] Tizanidine is an α-2 adrenergic agonist that directly impairs excitatory amino acid release from spinal interneurons and inhibits facilitator cerebrospinal pathways.[22] This results in presynaptic inhibition of motor neurons and decreased muscle stimulation, motor reflexes, and tone. The drug primarily affects the polysynaptic reflexes.[23] Tizanidine has a 2-hour half-life and a high first-pass metabolism. In patients with hepatic and renal impairment, meticulous medical monitoring, as well as dose adjustment, is required. Side effects include asthenia (fatigue and weakness), drowsiness, dizziness, xerostomia (dry mouth), sweating, and hypotension.[22,24]

Clonidine: This is another member of the imidazoline family and is effective for spasticity associated with stroke, MS, and traumatic brain injury. Like tizanidine, it is a selective α-2 agonist and a centrally acting medication. The biggest drawback is its tendency to cause hypotension. It is available as a patch for convenient use and is generally utilized as a second agent for spasticity management.

Dantrolene: This is a good choice for spasticity of cerebral cortex origin (traumatic brain injury, cerebral palsy) because it acts at the level of the muscle cell itself. It affects the release of calcium from the sarcoplasmic reticulum in skeletal muscle and, therefore, reduces muscle contraction. Dantrolene decreases muscle tone, clonus, and muscle spasm with minimal CNS side effects. Dantrolene affects all skeletal muscle, regardless of spasticity and, therefore, may cause generalized weakness, including respiratory muscle weakness. Other side effects include fatigue and diarrhea. The half-life of dantrolene is 6-8 hours with peak effects in 4 hours. Dantrolene should not be used in combination with other agents known to cause hepatotoxicity, including tizanidine. Prolonged use is not recommended.[18]

Intrathecal baclofen pump (ITB)

If oral agents are ineffective or the patient cannot tolerate escalating doses of baclofen due to side effects, the intrathecal delivery of baclofen should be considered. The adverse effects of baclofen are minimized during intrathecal infusion because the concentration gradient favors higher levels of action at the spinal cord level versus the brain. The FDA has approved this system for both spinal cord and cerebral spasticity. In practice, some functional gain is seen with ITB and recent studies reveal that ITB therapy improves walking speed, functional mobility ratings, and spasticity in stroke patients, while maintaining the muscle force in the uninvolved extremities.[25]

When assessing a patient for an ITB, a test dose of baclofen is administered under sterile technique via lumbar puncture. Prior to medication administration and every 2-hours post-procedure, the scale of spasticity is documented. Based on the amount of improvement, the efficacy of this drug delivery method can be assessed. If proven effective, arrangements for device and catheter implantation are made. Although monotherapy is preferred, occasionally pain medications can be combined with antispasmodics and delivered intrathecally for pain control.

The programming possibilities (simple continuous, complex programming, and boluses) are useful features. The pump can be programmed to infuse more medication during the night hours (for more tone control and improved sleep) and/or less medication during the day (for

functional tasks and mobility). The pump needs to be refilled as frequently as 3-5 times per year depending on the amount used per day, the reservoir volume, and concentration of baclofen used. The pump battery will deplete in about 5 years, requiring a small surgical revision. The advantages/disadvantages of this system are outlined in Table 4. Treating physicians, however, should consider intrathecal delivery as a last resort, given the complexity of management and potential iatrogenic complications. Baclofen overdose causes symptoms (eg, drowsiness, lightheadedness, respiratory depression and apnea, seizures, cardiac arrhythmias, and coma) that may be caused by pump malfunction, errors in ITB programming and concentration, and subcutaneous baclofen deposition. Errors in programming may result in withdrawal symptoms as well, which will present with increase in tone, dysreflexia symptoms (in some SCI patients), and potentially rhabdomyolysis, multiorgan failure, and death. As a result, proximity to medical centers capable of handling intrathecal pumps and 24-hour physician coverage for these devices is mandatory.

Surgical considerations in spasticity

There is rarely the need for surgical correction in patients with spasticity, but it may occasionally be indicated and is aimed at 4 different levels: brain, spinal cord, peripheral nerves, and muscle. Procedures are predominantly neurosurgical or orthopedic.

Neurosurgical procedures

• *Selective dorsal rhizotomy (SDR)* aims to manage severe intractable lower extremity spasticity by cutting the sensory (dorsal) nerve rootlets, from the upper lumbar (L2) level to the upper sacral root (S2) level. Normally these fibers carry excitatory signals from the muscle to the spinal cord and descending inhibitory input from the brain to counterbalance them for control of muscle tone. If this delicate balance is lost due to CNS injury, one way to avoid excess sensory input is to cut sensory input via rhizotomy. The candidate nerve rootlets are stimulated electrically (intraoperative EMG) and those producing abnormal responses are cut. SDR has been performed mostly on children with spastic diplegic cerebral palsy. SDR combined with physical and occupational therapy leads to significantly greater functional motor improvement at 1-year following surgery compared with physical and occupational therapy alone.[26]

• *Sterotactic neurosurgery, cerebellar stimulation and longitudinal myelotomy, and neurectomy* are procedures used to reduce spasticity, but outcomes are poor.[27]

Orthopedic procedures

These are more prevalent in the management of spasticity compared with neurosurgical procedures. Target tissues are bones, joints, ligaments, tendons, and muscles. The goals include reducing spasticity, increasing active or passive range of motion, improving access for hygiene, improving fit of orthotics, and reducing pain.

• *Contracture release* is the most common orthopedic procedure for spasticity. By cutting the tendon of a contracted muscle, the surgeon can reposition the joint in a normal angle and cast over it. In a few weeks when the tendon re-grows, the cast is removed and serial casting is done followed by rehabilitation for many months. The result should be a more natural joint position and a better orthotics fit and gait. Hamstring and achilles tendon release are common.[28]

• *Tendon transfer* moves the insertion site of the spastic muscle to a new location; thus, the spastic muscle no longer pulls the joint into a deformed position. After this surgery, joints will generally lose active function, but will maintain passive range and have better anatomical alignment. Split anterior tibial tendon transfer (SPLATT) is a common procedure for correction of equinovarus deformity.[29]

• *Osteotomy* is a procedure where part of the bone is removed (wedge shape) to reshape or reposition the main bony structure and is commonly done in hip displacement and foot deformity.

• *Arthrodesis* is used when joint fusion limits the ability of a spastic muscle to pull the joint into an abnormal position; it is most commonly performed on bones in the ankle and foot.

TABLE 4: Advantages and disadvantages of a baclofen pump

Good reasons to consider a pump
Improved tone and comfort
Reduced side effects for patients
Reduced consumption of systemic medication
Improved ability to perform ADLs
Increased patient productivity
Possible return to work
Enhanced quality of life
Long-term cost effectiveness
Reasons not to consider a pump
Potential for errors in programming the pump
Lack of 24/7/365 medical coverage
Lack of expertise in local community hospitals to manage the pumps
Lack of social support for patients with low cognitive function
Discomfort with refill
Consideration for following potential ITB complications:
• Surgical complications
– Disseminated infection
– Spinal abscess
– Bleeding
– Spinal cord injury
– CSF leak
• Pump malfunction
• Catheter issues
– Breakage
– Disconnect
– Leak
– Migration

ADL = activities of daily living

CONCLUSION

Spasticity is a challenging aspect of managing patients with UMN disorders. An understanding of the pathophysiology and available diagnostic tools, as well as the ability to provide state-of-the-art treatment are essential for the treating physician. It is likely that no single treatment modality will result in a satisfactory outcome and that a combination of available treatments is indicated. Major goals of treatment are improved quality of life, functional gains, decreased pain, and improved rehabilitation potential. It is important to recognize that a dedicated multi-

disciplinary team – including the treating physician, surgeons, rehabilitation therapists, and the patient – is needed to manage spasticity.

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References

1. Lance JW. Symposium synopsis. In: Feldman FG, Young KK, Koella WPs, eds. *Spasticity: Disorder of Motor Control.* Chicago, Ill: Year Book Medical; 1980:485-494.
2. Young RR. Treatment of spastic paresis (editorial) *N Engl J Med* 1989;320: 1553-5.
3. Mayer H, Brin MF. Preface. Clinicophysiologic concepts of spasticity and motor dysfunction in adults with an upper motoneuron lesion. *Muscle Nerve Suppl* 1997; 6:S1-13.
4. Levi R, Hultling C, Nash MS, Seiger A. The Stockholm spinal cord injury study: Medical problems in a regional SCI population. *Paraplegia* 1995;33: 308-15.
5. Rizzo MA, Hadjimichael OC, Preiningerova J, Vollmer TL. Prevalence and treatment of spasticity reported by multiple sclerosis patients. *Mult Scler* 2004; 0(5):589-95.
6. Sommerfeld DK, Eek EU, Svensson AK, et al. Spasticity after stroke: its occurrence and association with motor impairments and activity limitations. *Stroke* 2004;35(1):134-9.
7. Maynard FM, Karunas RS, Waring WP 3rd. Epidemiology of spasticity following traumatic spinal cord injury. *Arch Phys Med Rehabil* 1990;71(8):566-9.
8. National Multiple Sclerosis Society, Successful Initiative on Gender Differences in MS.
9. Mayer NH. Clinicophysiologic concepts of spasticity and motor dysfunction in adults with an upper motoneuron lesion. *Muscle Nerve Suppl* 1997; 6: S1-13
10. Ashworth B. Preliminary trial of carisoprodol in multiple sclerosis. *Practitioner* 1964;192:540-2.
11. Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. *Phys Ther* 1987;67(2):206-7.
12. Brin MF. Dosing, administration, and a treatment algorithm for use of botulinum toxin A for adult-onset spasticity. Spasticity Study Group. *Muscle Nerve Suppl* 1997;6:S208-20.
13. Ahnert-Hilger G, Bigalke H. Molecular aspects of tetanus and botulinum neurotoxin poisoning. *Prog Neurobiol* 1995;46(1): 83-96.
14. Blasi J, Chapman ER, Link E, et al. Botulinum neurotoxin A selectively cleaves the synaptic protein SNAP-25. *Nature* 1993;365:160-163.
15. Comella CL, Tanner CM, DeFoor-Hill L, Smith C. Dysphagia after botulinum toxin injections for spasmodic torticollis: clinical and radiologic findings. *Neurology* 1992;42(7): 1307-1310.
16. Young RR, Delwaide PJ. Drug therapy: spasticity. *N Engl J Med* 1981;304:28-33, 96-99.
17. Davidoff RA. Antispasticity drugs: mechanisms of action. *Ann Neurol* 1985; 17:107-116.
18. Gracies MJ, Elovic E, McGuire J, Simpson D, Brin MF. Traditional pharmacological treatments for spasticity, Part II: general and regional treatments *Muscle Nerve* 1997;20(suppl 6):S92-S120.
19. Meythaler JM, Guin-Renfroe S, Johnson A, Brunner RM. Prospective assessment of tizanidine for spasticity due to acquired brain injury. *Arch Phys Med Rehabil* 2001;82(9):1155-63.
20. Smith C, Birnbaum G, Carter JL, et al. Tizanidine treatment of spasticity caused by multiple sclerosis: results of a double-blind, placebo-controlled trial. US Tizanidine Study Group. *Neurology* 1994;44(11suppl 9):S34-S43.
21. Gelber DA, Good DC, Dromerick A, Sergay S, Richardson M. Open-label dose-titration safety and efficacy study of tizanidine hydrochloride in the treatment of spasticity associated with chronic stroke. *Stroke* 2001;32(8): 1841-6.
22. Delwaide PJ, Pennisi G. Tizanidine and electrophysiologic analysis of spinal control mechanisms in humans with spasticity. *Neurology* 1994 Nov; 44(11 Suppl 9): S21-7; discussion S27-8
23. Coward DM. Tizanidine: neuropharmacology and mechanism of action. *Neurology* 1994;44(11 Suppl 9):S6-10; discussion S10-1.
24. Nance PW, Bugaresti J, Shellenberger K, et al. Efficacy and safety of tizanidine in the treatment of spasticity in patients with spinal cord injury. North American Tizanidine Study Group. *Neurology* 1994;44(11 suppl 9): S44-S52.
25. Francisco GE, Boake C. Improvement in walking speed in poststroke spastic hemiplegia after intrathecal baclofen therapy: a preliminary study. *Arch Phys Med Rehabil* 2003;84:1194-1199.
26. Wright FV, Sheil EM, Drake JM, Wedge JH, Naumann S. Evaluation of selective dorsal rhizotomy for the reduction of spasticity in cerebral palsy: a randomized controlled trial. *Dev Med Child Neurol* 1998;40(4):239-47.
27. Chambers H. The surgical treatment of spasticity. *Muscle Nerve Suppl* 1997;6: S121-8.
28. Keenan MAE, Kozin SH, Berlet AC. *Manual of Orthopedic Surgery for Spasticity*. New York: Raven Press; 1993.
29. Edwards P, Hsu J. SPLATT combined with tendo achilles lengthening for spastic equinovarus in adults: results and predictors of surgical outcome *Foot Ankle* 1993;14(6):335-8.

Upcoming Scientific Meetings

11-14 January 2006
The 7th International Conference on Pain and Chemical Dependency
New York Marriott at the Brooklyn Bridge
Brooklyn, New York
CONTACT: www.painandchemicaldependency.org/

3-5 February 2006
New England Pain Association Annual Meeting
Woodstock, Vermont
CONTACT: 804-282-4011

22-25 February 2006
American Academy of Pain Medicine 22nd Annual Meeting
San Diego, California
CONTACT: http://www.painmed.org/

7-8 April 2006
The First Annual American Conference on Pain Medicine
Jointly sponsored by the Institute of Applied Science and Medicine and The Johns Hopkins University School of Medicine
New York Marriot Marquis, New York, NY
CONTACT: www.iasmconference.com/pain2006/index.asp

3-6 May 2006
The 25th Annual Scientific Meeting of the American Pain Society
Henry B. Gonzalez Convention Center and Marriott River Center
San Antonio, Texas
CONTACT: www.ampainsoc.org/

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