Spasticity and its management

By S. Ali Mostoufi, MD, F.A.P.M.&R.

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 tracts, 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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**CLINICAL DIAGNOSIS**

A physical examination is the single most important method
detector 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 Babin-
ski’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
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**Spasticity scale**

Measurement scales have been developed by healthcare
providers to provide a common understanding of spasticity, mon-
tor 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 spastic-
ity and can oversee different aspects of care and coordinate the

**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 func-
tion in patients with underlying weakness. Furthermore, when
deciding to treat a spastic muscle, it is important to know the role
of the antagonist 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, thermodalities, 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 modifications 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
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.\(^1\) 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 α2 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, dizzi-ness, 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 α2 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 severe spasticity.
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

- **Stereotactic 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-grow, 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.

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-

TABLE 4: Advantages and disadvantages of a baclofen pump

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<tr>
<th>Good reasons to consider a pump</th>
<th>Reasons not to consider a pump</th>
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<tbody>
<tr>
<td>Improved tone and comfort</td>
<td>Potential for errors in programming the pump</td>
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<td>Reduced side effects for patients</td>
<td>Lack of medical coverage</td>
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<td>Reduced consumption of systemic medication</td>
<td>Lack of expertise in local community hospitals to manage the pumps</td>
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<tr>
<td>Improved ability to perform ADLs</td>
<td>Lack of social support for patients with low cognitive function</td>
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<td>Increased patient productivity</td>
<td>Discomfort with refill</td>
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<td>Possible return to work</td>
<td>Consideration for following potential ITB complications:</td>
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<tr>
<td>Enhanced quality of life</td>
<td>• Surgical complications</td>
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<td>Long-term cost effectiveness</td>
<td>– Disseminated infection</td>
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<td>– Spinal abscess</td>
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<td>– Bleeding</td>
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<td>– Spinal cord injury</td>
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<td>– CSF leak</td>
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<td>• Pump malfunction</td>
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<td>• Catheter issues</td>
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<td>– Breakage</td>
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<td>– Disconnect</td>
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<td>– Migration</td>
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<td>ADL = activities of daily living</td>
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disciplinary team – including the treating physician, surgeons, rehabilitation therapists, and the patient – is needed to manage spasticity.

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8. National Multiple Sclerosis Society. Successful Initiative on Gender Differences in MS.

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/
3-6 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.amapainsoc.org/

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