Poststroke Spasticity Management

Gerard E. Francisco, MD; John R. McGuire, MD

Poststroke spasticity (PSS) is a common complication associated with other signs and symptoms of the upper motor neuron syndrome, including agonist/antagonist co-contraction, weakness, and lack of coordination. Together, they result in impairments and functional problems that can predispose to costly complications. The goal of PSS management should take into consideration not only reduction of muscle hypertonia but also the impact of PSS on function and well-being. Therapeutic interventions focus on peripheral and central strategies, such as physical techniques to increase muscle length through stretching and pharmacological modulation. Although there are few comparative studies on the superiority of one method over another, it appears that optimal management of PSS involves a combined and coordinated compendium of therapies that encompass cost-effective pharmacological and surgical interventions, along with rehabilitative efforts.

What is PSS and How Common Is It?
Spasticity, commonly defined as “a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome,” is a common complication of stroke. It contributes to the impairments and disabilities that negatively impact functional recovery. Consequently, PSS, along with weakness and lack of coordination, result in gait abnormalities and problems with arm use. In addition to functional limitations, spasticity, when inappropriately treated, may lead to reduced quality of life, increased pain, and joint contractures.

Three community-based studies that followed-up stroke survivors for 3 to 12 months reported an incidence of PSS between 17% and 43%. Certain factors are recognized as predictors of PSS: stroke lesions in the brain stem; hemorrhagic stroke and younger age; and severe paresis and hemihypotonia at stroke onset.

How Is PSS Measured?
To quantify the full impact of PSS, assessment should include a measure of passive stretch, volitional movement, and active/passive function. The benefit of using multiple measures in the evaluation of PSS is to differentiate the various components of the upper motor neuron syndrome, such as spasticity, spastic co-contraction, spastic dystonia, synergistic limb patterns, weakness, soft tissue contractures, and the functional implications of these impairments.

The most commonly used clinical measure of spasticity is the Ashworth Scale or its modified version. This ordinal scale is a simple clinical test of resistance to passive stretch and is limited by poor inter-rater reliability. The Tardieu Scale has advantages over the Ashworth Scale because it is an interval scale and takes into consideration the velocity-dependent nature of spasticity. Tardieu Scale measures the spasticity angle, which is the difference between the angle at the end of passive range of motion at slow stretch and the angle of catch at fast stretch. This estimates the relative contribution of neural mechanisms (ie, spasticity) and mechanical restraint attributable to rheological changes in soft tissues.

Electrophysiological tests, such as the H-reflex and H/M ratio, have been used to quantify PSS but tend to correlate poorly with the degree of spasticity. Biomechanical measurements of spasticity using a servo-controlled motor-driven device can provide a more objective measure of resistance to passive stretch.

Measures of passive function, such as the Carer Burden Scale, assess the impact of upper limb spasticity on the physical ability of caregivers to provide assistance. Measures of active function of the upper limb, such as the Composite Functional Index and Disability Assessment Scale, show a positive correlation with reduction of arm spasticity. Timed ambulation tests are clinically useful measures of the ability to walk. The Goal Attainment Scale, although not a measure of spasticity, can identify symptoms that a specific therapeutic intervention is meant to change.

When and Why Should PSS Be Treated?
Some argue that the importance of spasticity may be overstated, but it is widely acknowledged that treatment of disabling PSS is beneficial. In many instances PSS is a significant contributor to the multitude of impairments and disabilities and decreased quality of life that challenge stroke
survivors. For example, PSS resulting in an adducted internally rotated shoulder can limit overhead reaching activities and make underarm cleaning more difficult. When it involves wrist and finger flexors, PSS interferes with object grasp and release. The “clenched fist” deformity can lead to skin breakdown and nail bed infections. Lower extremity spasticity can limit ambulation, such as when an equinovarus foot prevents the foot flat position during the stance phase of gait, resulting in instability, and resulting in knee pain attributable to excessive recurvatum when ankle plantar flexor spasticity overwhelms. Severe spasticity of hip adductor muscles can make perineal care and toileting difficult. There are instances when even “mild” PSS causes significant limitations and warrants attention to enhance function or comfort. An example is mild spasticity of finger flexors that limits typing on a keyboard or object manipulation, or mild spasticity of the great toe extensor that causes pain attributable to friction against footwear when walking.

The presence of PSS is not an indication for treatment because it may not have a negative impact on the stroke survivor’s well-being. PSS can aid in some functional tasks, such as using increased knee extensor tone for standing and transfers, and possibly can preserve muscle bulk and retard osteoporosis. The most reasonable approach is to treat PSS whenever it becomes disabling or problematic. The treatment goals can be active, ie, tasks performed by the patient, and for the lower-level patient they tend to be passive, ie, performed for the patient. Frequently, however, treatment goals are a combination of passive and active. For example, treatment of elbow flexor spasticity is expected to facilitate hygiene and prevent contractures (passive goal) and at the same time is anticipated to improve limb movement (active goal). Table 1 lists common passive and active goals.

### Table 1. Treatment Goals for Problematic Poststroke Spasticity

<table>
<thead>
<tr>
<th>Level Patient: Passive Function</th>
<th>Upper extremity</th>
<th>Increased independence in performance</th>
<th>Decreased time spent on exercise program</th>
</tr>
</thead>
<tbody>
<tr>
<td>Improved prehension and grasp and release</td>
<td>Improved reaching and overhead activities</td>
<td>Reduced shoulder pain during movement</td>
<td></td>
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<tr>
<td>Decreased time to accomplish ADL</td>
<td>Mobility</td>
<td>Prevent long-term injury attributable to alteration in joint biomechanics</td>
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<tr>
<td>Improved speed, balance, quality, and safety of gait</td>
<td>Discontinuation of oral spasmyotic drugs to decrease risk for adverse events</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Others</td>
<td>Decreased time spent on exercise program</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADL</td>
<td>ADL indicates activities of daily living.</td>
<td></td>
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### Table 2. Rehabilitation Techniques for Poststroke Spasticity

<table>
<thead>
<tr>
<th>Potentiate medication</th>
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<tbody>
<tr>
<td>Neuromuscular electrical stimulation after BoNT injections</td>
</tr>
<tr>
<td>Restore biomechanics</td>
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<tr>
<td>Orthotics</td>
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<tr>
<td>Stretching, including serial casting</td>
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<tr>
<td>Improve motor control</td>
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<tr>
<td>Body weight–supported treadmill training</td>
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<tr>
<td>Robot-assisted</td>
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<tr>
<td>Neurofacilitatory techniques</td>
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<tr>
<td>Functional electrical stimulation</td>
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<tr>
<td>Strengthen weak muscles</td>
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<tr>
<td>Resistance training programs</td>
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<tr>
<td>Aquatic therapy</td>
</tr>
<tr>
<td>Integrate into functional tasks</td>
</tr>
<tr>
<td>Constraint-induced movement therapy</td>
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<tr>
<td>Neural prosthesis</td>
</tr>
<tr>
<td>Improve endurance</td>
</tr>
<tr>
<td>Aquatic therapy</td>
</tr>
<tr>
<td>Treadmill exercises</td>
</tr>
<tr>
<td>Circuit training</td>
</tr>
<tr>
<td>Others</td>
</tr>
<tr>
<td>Electroacupuncture</td>
</tr>
<tr>
<td>Biofeedback</td>
</tr>
<tr>
<td>Physical modalities (ultrasound, vibration, thermotherapy)</td>
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</tbody>
</table>

BoNT indicates botulinum neurotoxins.

### Rehabilitation Techniques

Reducing spasticity alone without addressing the negative components of the upper motor neuron syndrome will limit meaningful recovery. A combination of rehabilitation techniques is needed to facilitate functional improvements. Most rehabilitation studies for spasticity management are limited to small, randomized, controlled trials. There is emerging evidence of effectiveness of electric stimulation delivered to muscles after botulinum toxin injections, serial casting of ankle and elbow joints, partial body weight support gait training, and EMG-triggered electronic stimulation of wrist and finger extensors (Table 2). Studies on the use of an upper limb neuroprosthesis are limited, but there is more evidence that lower limb neuroprosthesis can improve gait speed.¹⁹

### Pharmacological Management

The choice of drugs to treat PSS depends on severity, anatomic distribution (see Figure), presence of comorbidities, and drug cost, among others. Many stroke survivors have cognitive deficits that may be worsened by the central effects exerted by oral medications, or they are receiving other drugs that are relatively contraindicated for use with certain antispasticity drugs (eg, clonidine and tizanidine act in synergy, resulting in hypotension; dantrolene sodium used concurrently with statins may induce hepatotoxicity.)

### Oral Medications

Antispastic oral medications (baclofen, tizanidine, dantrolene, and benzodiazepines) can effectively reduce PSS, but their use is
limited by many adverse effects. Because of this, and because of the limited evidence of efficacy attributable to inadequate sample size and lack of quality-of-life measures, it is best to limit the use of these drugs in PSS. However, oral drugs may be cost-effective choices for individuals who achieve adequate spasticity reduction without experiencing adverse events. The somnolent effect of oral medications may benefit a stroke survivor who has difficulty sleeping because of muscle spasms. Antispasticity drugs are discussed in greater detail in other reviews.

Neurolysis

Nerve blocks are effective in managing focal PSS. Depending on the dose and concentration used, these agents (phenol and alcohol) denature proteins in axons and membranes in afferent and efferent nerve fibers, leading to denervation and degeneration of muscle spindles. Adverse effects include postinjection dysesthesia, localized swelling, and excessive weakness. Although nerve blocks are widely used to manage PSS, there is paucity of evidence of efficacy and safety based on randomized controlled studies.

Botulinum Neurotoxins

Recent consensus articles support the use of botulinum neurotoxins (BoNT) for focal spastic conditions in adults with PSS, as do individual trials involving the upper and lower limbs. It has been suggested that proper choice of muscles and individualized doses of BoNT type A can improve function in selected poststroke patients. Advantages of BoNT over oral medications is target specificity (ie, exerting significant changes only in injected muscles) and better adverse event profile. Drowsiness and sedation, which are commonly associated with oral spasmyotics, are practically nonexistent with BoNT. BoNT is superior over tizanidine in terms of efficacy and safety in treating PSS. Many clinicians also appear to favor BoNT over phenol and alcohol neurolysis, which are more technically challenging and have a higher incidence of complications, among them dysesthesia.

Clinical Issues in the Use of BoNT

Unlike in other countries where payors dictate the total dose of BoNT that can be used, dosing in the United States is not standardized and is empirical or largely based on expert recommendation and consensus. A few injection cycles using different doses may be needed before determining the optimal dose, defined by these authors as the BoNT units required to achieve a predetermined outcome without causing adverse event unique to an individual with PSS. It is unclear where the common practice of administering BoNT not more frequently than every 3 months was based, because the early studies of immunoresistance were in the cervical dystonia, not stroke, population. Although no prospective investigation of antibody formation to repeated BoNT injection has been reported, it is estimated that the incidence of antibody to BoNT in the spastic hypertonia population is <1%, which is lower than in cervical dystonia. Repeated BONT injections appear to result in sustained reduction of spastic hypertonia. Most studies on BoNT efficacy and safety reported improvement in muscle tone and reduction of impairment, but only a few use functional improvement as the primary outcome measure. A randomized, controlled, multicenter trial found that BoNT type A treatment was safe and efficacious in reducing upper limb spasticity but did not result in a change in quality of life using standardized measures. However, the treatment appeared to result in achievement of patient-centered goals.

Intrathecal Baclofen Therapy

In the past decade, intrathecal baclofen (ITB) has been increasingly used to treat generalized or regional PSS recalcitrant to oral medications or injection therapy. Previous concerns that an intrathecally administered medication is not selective and, thus, will result in weakness of the noninvolved side were unfounded. ITB therapy has been shown to be effective in managing PSS, potentially enhancing functional recovery of gait and upper limb use, and improving quality of life. A consensus statement by a panel of experts recommended that ITB therapy be considered as early as 3 to 6 months poststroke, whenever it causes significant functional impact or hinders progress in rehabilitation. Common side effects of ITB are similar to the oral form but occur less frequently, largely because much lower intrathecal (and, hence, not required to cross the blood–brain barrier) doses are needed to exert therapeutic effects. Additional potential adverse effects are procedure-related or device-related, such as surgical infection, pump malfunction, or catheter interruption.

Despite the potential benefits of ITB, <1% of stroke patients with severe disabling spasticity are treated with ITB. Possible
reasons for ITB underutilization include surgical risks, excessive weakness, less effect on upper limbs, and limited functional improvement. These concerns are alleviated by recent studies that conclude that the benefits of ITB therapy outweigh the risks. ITB therapy should be considered as a safe and effective treatment for PSS when less invasive treatments fail to provide optimum reduction in problematic spasticity.

Role of Surgical Intervention
In those patients with PSS complicated by muscle or tendon shortening and who have not had success with the less invasive procedures, surgical interventions are considered. Most studies are limited to case reports or case series. These include split anterior tibial tendon transfer and tendon Achilles lengthening for spastic equinovarus foot. A retrospective review of a mixed patient population (stroke, cerebral palsy, and brain injury) demonstrated improved ability to ambulate, decreased need to wear orthosis, and increased ability to wear normal shoes postsurgery. Poor surgical outcomes were associated with nonambulatory status. Upper limb surgery, such as tendon transfer of the brachioradialis-to-extensor digitorum communis, tendon lengthening of the flexor pollicis longus, and release of the flexor-pronator tendons have favorable outcomes in a small case series. One investigation reported superiority of tibial neurotomy over botulinum toxin therapy in improving spasticity, range of motion, foot position, and gait characteristics, including velocity.

Gaps in PSS Research and Clinical Practice
Despite the advances in the treatment of PSS, there are several gaps in PSS research and clinical practice, foremost of which is the relative deficiency of knowledge of the pathophysiology of PSS. That its clinical presentation varies in terms of onset, severity, and regional distribution suggests that there may be >1 mechanism that underlies PSS. A better understanding of these mechanisms would help clinicians determine the type, timing, degree, and duration of the most effective combination of treatments. Although some predictors of disabling spasticity have been suggested, they are limited in their ability to forecast the onset of disabling spasticity during the poststroke recovery period, severity and regional distribution of spasticity, and response to therapies. If the characteristics of PSS evolve during the course of recovery, then it might be possible that a particular treatment will be effective only during a certain phase of spasticity evolution. An important question that needs to be answered is whether spasticity is a constraint on recovery, and would early intervention improve long-term outcomes?

There is a dearth of research on the comparative efficacy, safety, and cost-effectiveness of various therapies, both pharmacological and nonpharmacologic. With the introduction of newer BoNT, there are now three different type A and one type B toxins available in the United States, but how these toxins measure up against each other is unknown. The same is true of the effects of combination therapies, eg, different drugs or drug plus exercise. Outcomes of long-term use of antispasticity drug therapy rarely have been reported.

Conclusions
Selecting the appropriate treatment strategy and goals for the management of PSS should result in favorable functional outcomes. Inappropriate PSS management can interfere with functional recovery and increase complications. Function is a complex phenomenon that relies not only on muscle tone but also on strength, coordination, endurance, and sensation. In many instances, spasticity is incorrectly blamed as the main cause of dysfunction, when in fact the negative components of the upper motor neuron syndrome are most problematic. Thus, PSS management should be guided by its potential impact on function and well-being, rather than merely on the difficulty with passive muscle stretch or loss of range of motion. Other factors to consider before management include duration of condition, previous response to therapies, topographical involvement, response to medication, potential side effects, and cost. Therapeutic efforts have focused on peripheral (ie, altering muscle properties through physical techniques) and central (ie, influencing neurotransmission through GABA-mediated medications and modifying reciprocal inhibition) strategies. The optimal combination of rehabilitation techniques along with cost-effective medical and neurological management may provide the most favorable outcomes for PSS treatment.

Disclosures
Dr Francisco is consultant to Allergan, Merz, and Ipsen, and has received speaking honoraria from Allergan. Dr McGuire is consultant and member of the speakers’ bureau for Allergan, Ipsen, and Medtronic, and has received research grants from these companies.

References


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