Early Diagnosis of Post-stroke Spasticity and Treatment Options

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Abstract

Stroke is a leading cause of long-term disability. As a consequence of stroke and associated upper motor neuron (UMN) syndrome, stroke survivors are often left with muscle overactivity, including spasticity. Post-stroke spasticity often has a negative impact on functional activities and daily living, and is frequently accompanied by pain and abnormal limb postures and contractures. Spasticity can be beneficial occasionally but usually is detrimental to a patient's function. Several factors need to be considered in the evaluation and management of these patients. This article discusses the various instruments and methods of assessing patients with post-stroke spasticity, as well as the spectrum of current treatment options, including the potential side effects.

Keywords

Stroke, post-stroke, spasticity, antispasticity medications, botulinum toxin, baclofen, tizanidine, upper motor neuron

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Stroke is a leading cause of long-term disability. Approximately 610,000 people suffer a new stroke each year in the US,[1] often resulting in disability due to motor impairment and consequences of the upper motor neuron (UMN) syndrome. UMN syndrome results in both positive and negative motor effects. The positive features include muscle overactivity including spasticity, clonus, flexor/extensor spasm, and hyper-reflexia. Negative features consist of weakness, loss of dexterity, and decreased co-ordination.[2] The combination of these effects often leads to complications of contractures at the involved joints.

Spasticity is characterized by a velocity-dependent increase in resistance to muscle stretch associated with attempts to flex or extend a limb.[3] It is a leading cause of morbidity and long-term post-stroke disability. Observational cohort studies have estimated that post-stroke spasticity (PSS) may affect 17–43% of the 6.5 million American stroke survivors.[4,5–9] These patients often present with characteristic antigravity postural patterns with shoulder adduction, elbow and wrist flexion, hip adduction, knee extension, ankle plantar flexion, and foot inversion. Symptoms vary in localization and severity and may include hypertonicity (increased muscle tone), clonus (a series of rapid muscle contractions), exaggerated deep tendon reflexes, muscle spasms, scissoring (involuntary crossing of the legs), and fixed joints. The degree of spasticity may vary from mild muscle stiffness to severe, painful, and uncontrollable muscle spasms.[10] Spasticity may be associated with disabling pain, and over time may lead to permanent contractures that may eventually result in posture and joint deformities. Therefore, early intervention for spasticity may be critical for the preservation of muscle reactivity.[11]

The effects of PSS can interfere with daily activities that include tasks such as walking, picking up objects, washing, dressing, and sexual activity.[12] Indeed, a recent patient/care-giver survey reported that PSS may also be a hindrance to effective post-stroke rehabilitation therapy.[13] In addition, the presence of PSS can negatively affect mood, self-image, and motivation, which in turn is a likely contributor to a post-stroke patient's psychosocial burden and depression.[12,14] Spasticity can also have an impact on the care-giver burden in patients who are dependent on others for their basic activities of daily living.[15] *Figures 1–4* show a few examples of common deformities due to PSS.

Methods of Spasticity Assessment

Assessment is critical in developing a treatment plan and gauging the progress and outcome of the treatment. On initial evaluation it is necessary to confirm that the patient has a UMN syndrome. The degree of spasticity may change according to the position and the activity being performed with the affected limb.[6] The increase in tone may be related to spasticity or intrinsic changes of the muscle.[16,17] There also is no consensus as to the number of patients developing spasticity post-stroke or the timing post-stroke. One study reported that 39% of patients with first-ever stroke are spastic after 12 months.[18] Another more recent study found that spasticity was present in 19% of patients three months post-first ever stroke.[6] Physical assessment is the single most important method in evaluating spasticity.[19] This includes observation of patient movements and gait in addition to a detailed neuromusculoskeletal

Figure 1: Shoulder Adducted, Elbow and Wrist Flexed, and Forearm Pronated

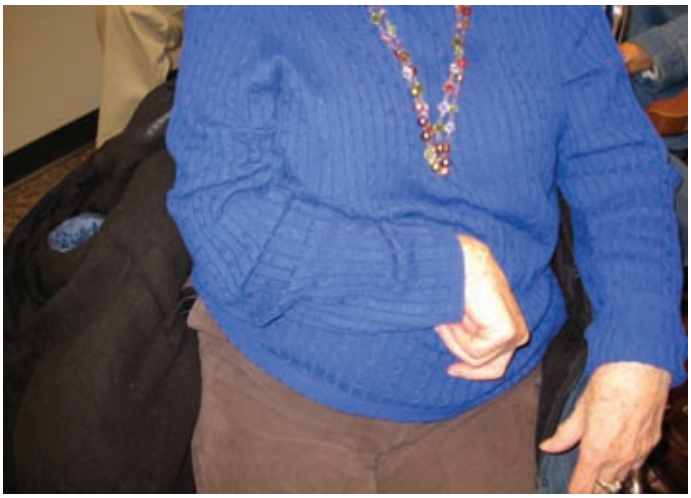
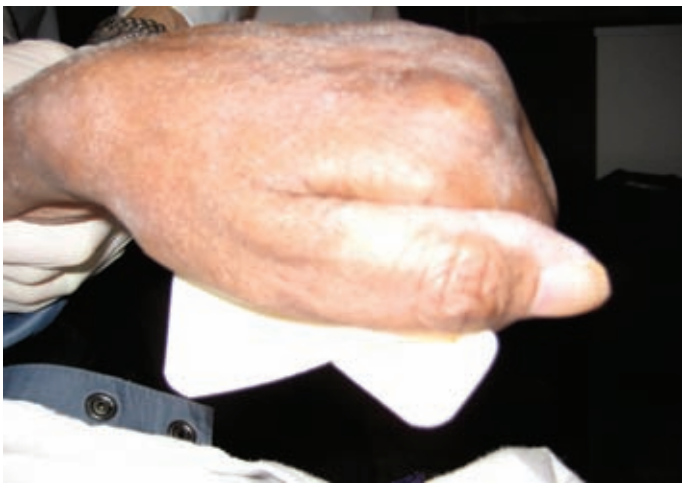


Figure 2: Clenched Fist



examination of the affected limbs. Particular attention is paid to the presence of long-tract signs, spastic catch, clonus, or clasp-knife phenomenon. The assessment should also include observing patients during functional activities (such as putting on a shirt) in order to evaluate dynamic muscle tone, which does not necessarily correlate with static muscle tone.[20]

Several rating instruments are available to evaluate various aspects of spasticity. Muscle tone can be measured using the Ashworth, modified Ashworth, and Tardieu scales.[21,22] These scales can be easily applied in the clinic setting to assess resistance to passive stretch or movement across a particular joint. In the upper limb, standardized positioning can make this evaluation more reliable. Instruments such as the Functional Independence Measure (FIM)[23] and the Disability Assessment Scale[24] can be used to evaluate the effects of spasticity on outcomes, such as the ability to dress oneself or complete personal-hygiene-related tasks. In cases where meaningful voluntary activity is retained, it can be measured with tests such as the Fugl-Meyer[25] and the Nine Hole Peg Test.[26]

Global measures of pain, patient satisfaction with treatment, and overall improvement in spasticity as rated by the physician, patient, or care-giver may also be utilized.[27] Goniometry is often used to measure joint flexibility (range of motion) and electrophysiological measurements may be useful for identifying electrical activity in agonist and antagonist muscles during movement. No single instrument is ideal to assess all aspects of spasticity, hence the choice of scales should be based on the change expected and treatment goals. While the validity and reliability of spasticity measures are important, in the clinic practical variables such as clinical relevance, ability to capture patient functioning, and time needed to complete the assessment are also important.[28] *Figure 5* shows a summary of the different assessment methods.

Spasticity Treatment

There are a variety of physical and pharmacological therapeutic options for treating PSS. Therapy typically focuses on the reduction of excessive muscle tone, and is designed to provide patients with improved range of movement and an enhanced ability to perform activities of daily living. In general, spasticity treatment regimens are multidisciplinary and include physical and occupational therapy, systemic central nervous system neurotransmitter agonists or antagonists, surgical intervention, and chemodenervation.[24,27,29]

Spasticity is not always detrimental and it is important to determine whether it is useful for an individual, for example where the spasticity provides support while the patient is standing or walking.[30] Treating the spasticity in this instance may unmask underlying weakness, making it difficult to stand or walk.

Once the decision has been made to treat spasticity, there may be advantages to treating sooner than later. There is some consensus that early treatment may avoid secondary maladaptation and functional impairment.[31] Specific and function-oriented treatment goals need to be established with the patient and care-giver prior to spasticity treatment.[20] Spasticity is only one of a constellation of symptoms observed in individuals post-stroke, and a multidisciplinary team approach seems to be most effective.[31,32]

Treatment Options

A number of conditions can exacerbate spasticity, and these need to be ruled out or managed before initiating other treatments. These include conditions such as a urinary tract infection, stool impaction, skin breakdown, ingrown toenail, fracture, heterotopic ossification, or any noxious stimulation.[33] The next line of treatment involves physical therapy.

Physical Therapies

Physical and occupational therapy include stretching and strengthening of the appropriate muscles, working on posture and positioning, and facilitation of movement to reduce the effect of spasticity.[34] Patients may require the use of orthotics to maintain muscle length and range at the affected joints. Modalities such as heat, ice, and electrical stimulation can help in different ways to temporarily reduce tone.[30] Both physical and occupational therapy are involved in maximizing function with a focus on mobility and activities of daily living.[35]

Medical Therapies

Medical therapies require the consideration of several factors before selecting a particular treatment. These are the patient's pattern of spasticity, the efficacy of the treatment, potential adverse effects, the reversibility of the treatment, and accessibility of treatment.[36,37] Spasticity can be classified as either focal or generalized to help determine which treatment may be more effective. It is also important to determine whether the treatment will be for a brief period or a longer period of time. Many post-stroke individuals have cognitive deficits and any additional impairment with the treatment for spasticity may be counterproductive; hence, in this situation a more focal type of treatment may be more appropriate. Several types of medical therapy are discussed in the following sections.

Oral Medications

Several different oral medications have been used for the treatment of spasticity. The most commonly used are baclofen, diazepam, tizanidine, and dantrolene. All but dantrolene act centrally. They have different mechanisms of action and side effects (see *Table 1*).

Intrathecal Baclofen

Patients who benefit from the effects of oral baclofen but cannot tolerate the side effects or are not optimally treated with the highest oral dose may benefit from intrathecal baclofen. The medication is delivered into the subarachnoid space of the spinal cord through an implanted pump and catheter. The drug level is relatively constant and well tolerated.[36] Complications related to the pump include infection, catheter dislocation or kinking, impaired wound healing, and pump malfunction. Adverse reactions can include headaches, nausea, vomiting, excessive weakness, and transient urinary retention. Despite this, treatment can be quite effective in patients with intractable spastic hemiplegia. It is more effective in treating lower-limb spasticity.[39]

Chemodenervation with Alcohol or Phenol

These treatments are administered directly into the muscle near a motor point or peri-neural region.[33] These neurolytic agents cause tissue destruction that interferes with nerve conduction. The effect is usually immediate and can last from a few weeks to years. The drawback is that it results in tissue destruction and associated complications, development of chronic dysesthesia, especially with injury to sensory nerves, and limitation to the treated areas due to the lack of motor nerve selectivity. Inaccurate medication delivery can result in unwanted weakness.[40]

Chemodenervation with Botulinum Toxin

Botulinum toxin is administered intramuscularly and inhibits muscle contraction by blocking acetylcholine release at the neuromuscular junction. It is a focal treatment and reduces the tone in hyperactive muscles, resulting in a functional response lasting for about three months.[12] As the effect wears off, repeat treatments may be required. The drawbacks of this treatment are that injection into the wrong muscles or excessive response can result in loss of temporary function, that there is potential for antibody development, and that repeated injections may be required.[41–47] The dosing units of the various botulinum toxins cannot be directly compared or converted into units of one another since there is no universally applicable safe dose conversion ratio.[48] Proper delivery of the agents in chemodenervation is crucial to optimize outcomes. Several different techniques are utilized. Phenol injections are performed with electrical stimulation to identify the nerve using small currents of <1

Figure 3: Flexed Elbow

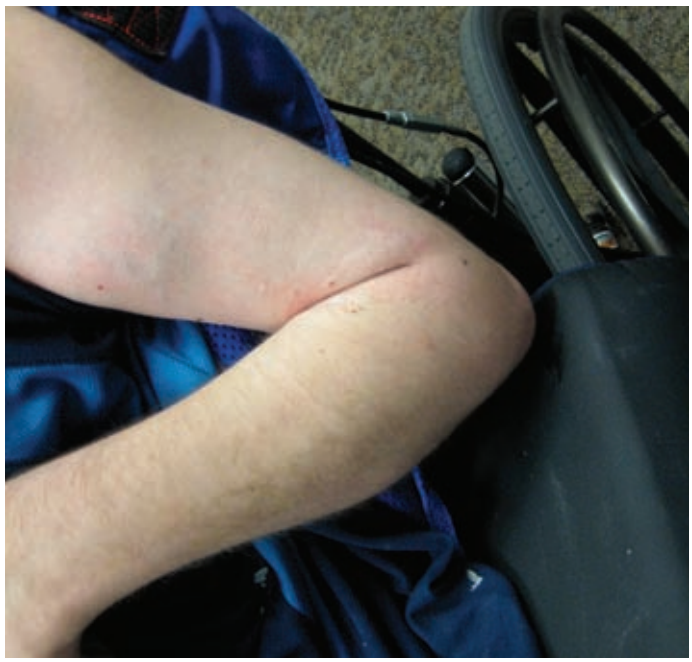


Figure 4: Clenched Fist and Equinovarus Foot Deformity

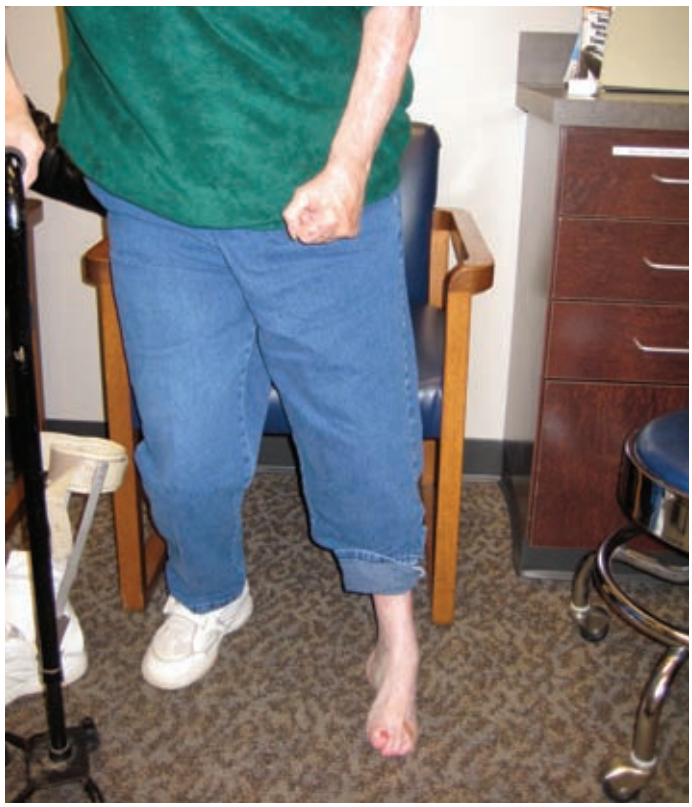


Figure 5: Summary of Spasticity Assessment Methods

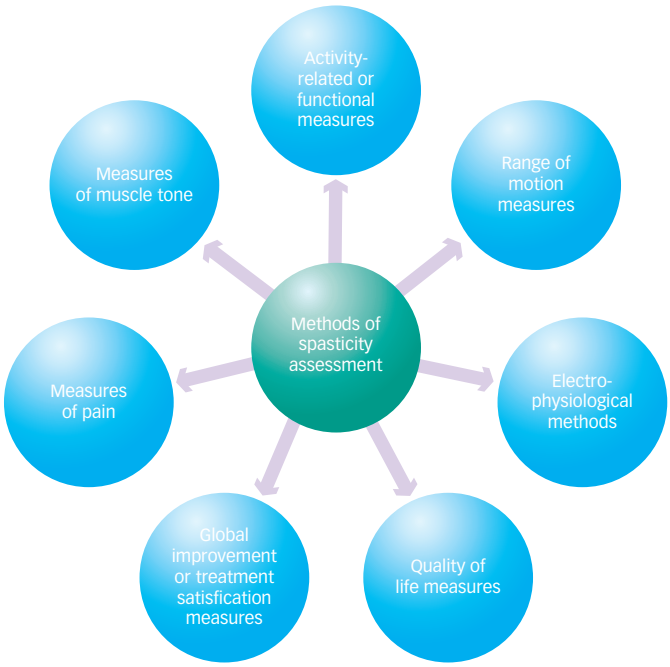


Table 1: Oral Medications for the Treatment of Spasticity

Medication	Mechanism of Action and Side Effects
Baclofen	Baclofen is a selective GABA-B agonist providing a net inhibitory effect and reducing spasticity by decreasing the activity of alpha motor neurons. Significant side effects include central depression and lower seizure threshold. It can also cause drowsiness and lethargy.[36]
Diazepam	Diazepam reduces spasticity by its inhibitory effects on GABA-B receptors in the central neurons. Its use is limited by central side effects such as sedation, fatigue, and reduced cognitive function.[36]
Tizanidine	Tizanidine is a central alpha-2 adrenergic agonist and acts centrally to prevent pre-synaptic excitation of alpha motor neurons. Side effects include drowsiness, dizziness, dry mouth, and sedation.[36]
Dantrolene sodium	Dantrolene sodium acts peripherally by reducing the release of calcium from the sarcoplasmic reticulum in skeletal muscles. This results in the reduction of muscle contraction force and phasic stretch reflexes.[38] It does not have central side effects, but other side effects include generalized weakness and hepatotoxicity.

GABA = gamma-aminobutyric acid.

milliamp that produce contractions in the target muscle. Electromyographic guidance is used for botulinum toxin injections to confirm delivery into muscle tissue. Anatomical landmarks and joint movement can help better identify the proper muscles; however, synergistic motor patterns may preclude the ability to distinguish nearby muscles that mimic the targeted muscles.

Nerve stimulation can be employed for botulinum injections, similar to that used for phenol injections. The drawback is that this is more technically challenging and it may still be difficult to distinguish nearby muscles that mimic the targeted muscle. A newer technique with some promise of more accurate muscle identification is musculoskeletal ultrasound. It can help sort out specific portions of target muscles and avoid vital structures such as blood vessels and nerves.[49]

Surgical Treatments

Selective dorsal rhizotomy involves the transection of selected posterior rootlets in the lumbosacral region of the spinal cord. This interrupts the sensory input for stretch reflexes, resulting in reduced spasticity.[30] Complications of this procedure include infections, altered sensory responses, transient bladder incontinence, and adverse muscle atrophy.[50]

Orthopaedic procedures to help patients with joint deformities and issues of positioning secondary to increased tone include tendon lengthening, tendon transfer, osteotomy, and joint arthrodesis.[51] These types of procedure can also help with pain reduction, prevention of further joint deformity, and increased range of joint motion. The potential risks include unintended weakening of the agonist muscle, complications of infection, and improper placement of tendon attachment.

Conclusions

Spasticity is a common problem in post-stroke patients and can have a significant impact on function. Proper assessment of the patient with spasticity is crucial for treatment guidance and monitoring of the outcomes. Hence, clinicians should be aware of the common assessment instruments. Many different medical therapies are available for spasticity and its sequelae. Oral medications and intrathecal baclofen are reversible in that the drugs can be discontinued. Additionally, these drugs are most useful for general spasticity given that they are distributed systemically.

Chemodenervation agents have reversible actions and are most useful for focal or multifocal spasticity. These agents include alcohol, phenol, and botulinum toxin. Physicians should be aware of localizing techniques such as electromyography, motor point or nerve stimulation, and ultrasound to accurately deliver the neurolytic agents.

Surgeries for spasticity and its orthopaedic sequelae are generally irreversible. Selective dorsal rhizotomy is most often used for generalized or diffuse spasticity of the lower limbs. Local surgeries such as tendon lengthening are often used to improve orthopaedic deformities.

Like all medical therapies, the treatments described here have the potential for adverse effects. When selecting a medical therapy, the adverse effects of each treatment must be considered along with treatment efficacy and applicability to the patient's particular pattern of spasticity. The selection of a therapy for spasticity should be guided by the treatment goals. Other factors such as the chronicity, severity, and cause of the spasticity may play a role in treatment selection, in addition to concomitant medical conditions, concurrent medications, and treatment cost. Tangible benefits are often achieved by matching the right treatment to the right patient, which offers the potential to dramatically improve the lives of patients and their care-givers. However, it must be emphasized that the treatment of spasticity is undertaken as part of a multidisciplinary management

program that includes numerous professionals, as well as the patient and his/her family or care-giver.

Just as important as selecting the appropriate medical treatment for the right patient is the adjunct and combination treatment. In most cases, physical and/or occupational therapy is crucial in achieving the full benefit of the medical and/or surgical treatments. In patients with generalized spasticity, the optimal treatment often involves both systemic and focal treatments. Finally, most patients require some type of maintenance treatment. ■

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