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Clinical Management of Upper Limb Spasticity

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Case Report

A 67-year-old right-handed male who had suffered an ischemic left basal ganglia stroke with right hemiparesis was evaluated for post-stroke rehabilitation therapies. He had undergone comprehensive inpatient rehabilitation six months earlier. When he was discharged home, he required supervision for upper and lower body dressing but was at a “modified independent” level for ambulation. However, during his three-month follow up visit, he demonstrated increased right upper-extremity spasticity. Specifically, he reported active right hand grasp but could not voluntarily open his fist to use previously acquired skills to complete self-care tasks. When he walked, he was embarrassed by the position of the right arm, which mimicked a boxer’s posture: right elbow flexed and fist held up to his mouth.

On examination, Modified Ashworth scores in the right arm were 3/4 right shoulder, 3/4 right elbow, 1+4 right wrist, and 2/4 right fingers. Manual muscle testing showed elbow flexors to be 3+/5, right wrist extensors 4/5, and right finger flexors 4/5, but right finger extensors 1/5. Left arm exam was within normal limits. During gait evaluation, the patient developed an exaggerated right elbow flexion posture to 90 degrees with the shoulder adducted and internally rotated and the fist in a clenched position. His standing balance was uneven and a mild left trunk lean was documented.

The patient inquired if there were medications or therapies he could receive that would keep the right arm better positioned down at his side. He also wanted to use the right arm for daily tasks like opening a door handle or grasping a bottle. Lastly, he was embarrassed by how the arm appeared and was hoping that treatment would make his stroke less obvious to others.

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Key words: Spasticity; Stroke; Botulinum toxin; Phenol neurolysis; Ultrasound

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Definition of Problem

Spasticity is a clinical disorder of increased muscle activity that can occur from injury or disease to the neurons or descending pathways extending from the cerebral cortex, cerebellum, brainstem, or spinal cord. Spasticity is a general clinical term that presents in many phenotypes. Common presentations include hypertonia (increased muscle tone), co-contraction of agonist and antagonist muscles, muscle spasms, and dystonia — described as sustained muscle contractions causing twisting and repetitive movements or abnormal postures. Other clinical findings include exaggerated deep tendon reflexes, clonus, and adducted or scissoring gait.

Spasticity is observed in clinical practice with voluntary-motor weakness and joint contracture. The triad of spasticity, weakness, and contracture has been termed upper motor neuron syndrome. Spasticity management should be adjusted based on disease progression and the resulting functional deficits caused by hypertonicity. Not all spasticity is detrimental to function; for example, truncal spasms may aid sitting balance in patients with high tetraplegia. Therefore, it is very important for physicians to clearly assess and discuss the anticipated goals of treatment, since patients often focus on resolution of voluntary motor weakness rather than reduction of hypertonicity.

Assessment

When a patient presents for initial evaluation for spasticity, it is necessary to confirm that the patient has an upper motor neuron syndrome. It is not uncommon for patients to be referred to physiatrists with muscle splinting secondary to musculoskeletal pain disorders and incorrectly interpreted as spasticity. These individuals require treatment based on the underlying pain disorder. Rapid appearance of true spasticity without a clear etiology — like prior stroke — warrants immediate work-up to determine if there are new lesions in the brain or spinal cord.

In the office, resistance to passive stretch can be estimated using the Modified Ashworth Scale¹. This ordinal scale ranges from 0 to a maximal score of 4 and is judged by the resistance to passive movement across a particular joint. In the upper extremity, standardized positioning can make this evaluation more reliable. For example, when examining the elbow joint, the examiner starts with maximal elbow flexion then rapidly extends the forearm over 1 second. When examining the wrist, the elbow is kept in extension

with the forearm pronated and the fingers passively flexed into the palm to decrease tension. Then, the physician rapidly extends the wrist from a flexed starting position over 1 second. To assess the finger flexors, the elbow is placed into extension and the forearm is placed in a neutral position with the wrist in slight extension. The fingers are rapidly extended away from the palm. Of note, if limitations to full range of motion are noted, resistance to passive stretch is graded over the available range of motion.

If spasticity is documented, the physiatrist should determine if symptoms are affecting one or two body segments (regional spasticity) or if symptoms are experienced in more than two body regions (global spasticity).

Initial Management

Stretching exercises and passive range-of-motion activities are commonly used although there is no good evidence-based literature documenting efficacy². For the spastic hand, an orthosis that places the wrist in slight extension with the fingers in neutral (see Figure 1) is preferred over the typical C orthosis that does not adequately stretch spastic fingers.



Figure 1. Orthosis for spastic hand

Oral Medications

Oral medications are very effective for global spasticity management. High doses of oral medications, however, can lead to unwarranted side effects that include sedation, cognitive impairment, dry mouth, and diffuse weakness. Common oral medications include baclofen, tizanidine, dantrolene, and diazepam.

Baclofen

Baclofen is a selective GABA-B agonist that hyperpolarizes the postsynaptic neuron, providing a net inhibitory effect. Baclofen affects the nervous system both centrally and peripherally. Significant side effects include central depression and lower seizure threshold. Therefore, baclofen should be used with caution in patients with increased seizure risk. Abrupt withdrawal after long-standing use should also be avoided. Starting dose is usually 10mg *b.i.d.* or *t.i.d.*, with titration as high as 120mg per day if tolerated.

Tizanidine

Tizanidine is a central alpha-2 adrenergic agonist. It acts centrally to prevent presynaptic excitatory potentials and attenuates inhibitory potentials. Significant side effects include hypotension, dry mouth, drowsiness, and elevated liver-function tests. This medication should not be used with the quinolone antibiotics like ciprofloxacin. Starting dose is usually 2mg *b.s.*, titrating up to 2mg *t.i.d.* with a maximal dose of 36mg per day.

Dantrolene Sodium

Dantrolene sodium reduces the release of calcium from the sarcoplasmic reticulum of muscles. It reduces muscle activation without depressing the central nervous system; therefore, it is often the treatment of choice for patients with traumatic brain injury. Significant side effects include hepatic toxicity and hepatonecrosis. Liver function should be monitored monthly for the first three months then decreased to three times per year if no abnormalities are detected. This is especially important given the prevalence of statin therapy, which can also negatively affect liver function. Starting dose is usually 25mg *b.i.d.* or *t.i.d.*, titrating up to a maximal safe dose of 300mg per day.

Diazepam

Diazepam reduces spasticity by attenuating the inhibitory effects of GABA-A receptors. Similar to baclofen, it can also cause central depression and care must be taken when tapering since physiological dependence can occur. Starting dose is typically 2.5mg *b.i.d.*

Regional Spasticity Management

In the patient vignette, there is a clear picture of spasticity affecting the shoulder, elbow flexors, and finger flexors, with lesser involvement of the wrist flexors. Local injections offer this patient the greatest chance of meeting his goals: namely restoring arm range of motion and improving function, especially for grasping and releasing objects in his paretic hand. If spasticity is severe and refractory to other treatments, *intraarticular baclofen pump* insertion can be an excellent treatment option, even for patients with spastic hemiparesis, since the nonaffected side is usually spared.

Spastic muscles can be selectively weakened with intramuscular injections of botulinum toxin^{3,4}. Individual nerves or separate motor branches can be neurolyzed using a denaturing agent (either 6% phenol or 95% ethyl alcohol). Focal injections, however, rely on accurate delivery of medication within the desired muscle or nerve. Inaccurate medication delivery can worsen limb posture, cause unwarranted weakness, and increase pain.

Phenol denatures the protein structure around the nerve to block both afferent and efferent nerve impulses, thus reducing spasticity. Electrical stimulation is employed to find the nerve, using currents of 1 ma or less that produce a contraction in the target muscle. Complications, in up to 20%, include dysesthesias that are typically mild and short-lasting, except when injecting nerves with large sensory territories such as the tibial nerve. It is also very important to aspirate for heme prior to injecting phenol to avoid severe tissue necrosis⁵.

A video demonstrating phenol neurolysis injection technique can be viewed on our website by typing <http://www.rehabmedicine.pitt.edu> in your Internet browser address box. On the Web page, point your mouse at the picture labeled “Phenol Neurolysis Video” and click.



Phenol neurolysis video

Perineural injection of phenol is often used in combination with botulinum toxin to achieve maximal spasticity control since phenol can induce changes in large proximal muscles and botulinum toxin can target smaller and harder-to-access muscles. Also, the combination results in injection of less botulinum toxin, which is more cost effective and less likely to produce remote side effects such as dysphagia. Since phenol works immediately, the limb can be better positioned after phenol neurolysis for more accurate injection of botulinum toxin.

Botulinum toxins block release of acetylcholine at the neuromuscular junction. The toxins are internalized into neuronal vesicles, where neurotransmitter release is inhibited by cleavage of synaptosomal-associated protein (SNAP), and vesicle-associated membrane protein (VAMP)⁶. These proteins are responsible for anchoring acetylcholine vesicles for exocytosis and their cleavage prevents acetylcholine release and subsequent muscle contraction.

There are seven different subtypes of botulinum toxins. All toxin subtypes have mechanisms of action via cleavage of either SNAP or VAMP protein at different target sites. Toxin A (Botox[®]) and Toxin B (Myobloc[®]) are commercially available in the U.S. The dosing units of botulinum toxins A and B cannot be directly compared or converted into units of one another, since there is no universally applicable safe dose conversion ratio⁷.

EMG-Guided Injection

EMG-guided botulinum toxin injections confirm that the medication is delivered into muscle tissue. Accurate muscle location is determined by an increase in motor unit activity during passive joint movements, or active movements if the patient is able to do this. The injections are completed using a hollow monopolar needle electrode that allows electrical recordings and injections to occur simultaneously. Some clinicians also use nerve stimulation in place of EMG, but we find this technique less helpful except for injecting phenol solution as previously discussed. Although EMG localization is better than “blind” injections, where fat or connective tissue can be mistakenly injected, there are pitfalls to this methodology:

1. Localization of an actively contracting muscle fiber may not equate to localization of a spastic muscle fiber.
2. Synergistic motor patterns may preclude the ability to distinguish nearby muscles that mimic the targeted muscle.
3. Anatomic landmarks may not be accurate for all body types.

Surface Mapping Technique for Flexor Muscles of the Forearm

Spastic finger flexion is a very common abnormal posture in the upper limb. This posture is predominately driven by increased tone in the flexor digitorum superficialis (FDS) and profundus (FDP) muscles that are located deep to the wrist flexors and pronators, making precise delivery of neurotoxin challenging. Lumbrical spasticity can complicate this picture causing tightness at the MCP joints. In a prior study, we validated a surface mapping technique, originally developed by Bickerton⁸ and colleagues, that allows accurate localization of individual muscle bellies within the flexor digitorum superficialis⁹. This methodology distinguishes the FDS from other nearby muscles and allows dosing to be individualized if for example, the second- and third-finger flexors are more spastic than the other muscle bellies.

To use the finger flexor mapping technique, the patient is placed supine with the forearm supinated and the elbow extended. Severe elbow joint contracture is a contraindication for using this technique. A landmark line (LL) is measured in cm

from the medial epicondyle (starting point) to the pisiform bone (distal point) to record proximodistal and radial (lateral) coordinates. (See Figure 2) FDS 4 is most proximal with localizing coordinates of 50% of the LL distance then measuring 7 mm radial; FDS 3 is 55% LL and 17mm radial; FDS 2 is 70% LL and 14 mm radial; FDS 5 is most distal at 75% LL and 6 mm radial.

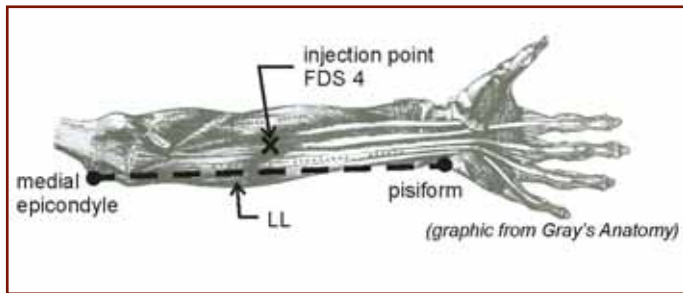


Figure 2.

Musculoskeletal Ultrasound Guidance

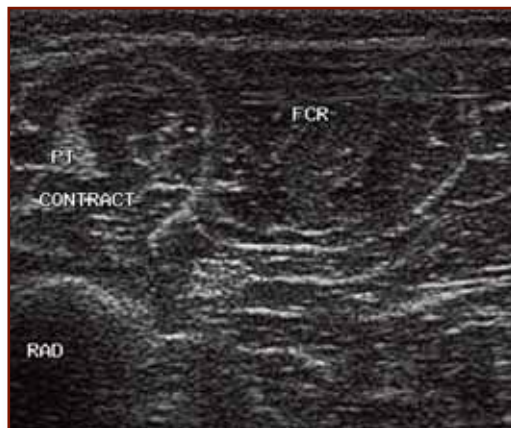
Musculoskeletal ultrasound (MU) uses pulses of sound waves that travel in a straight line then echo or reflect back to the recording transducer. Echo strengths are determined only by the reflected tissue's acoustic property. With a transverse or cross-sectional view, individual muscle bellies can be identified in the forearm while differentiated from nerves and blood vessels.

Benefits of MU localization for botulinum toxin injection include precise delivery of medication when several muscles overlap the targeted muscle, ability to avoid nerves and blood vessels in close proximity to the target muscle, and direct visualization of toxin during infusion. Drawbacks include the expense and expertise needed to operate an MU machine and temporal changes in typical muscle echogenicity that impede muscle identification¹⁰.

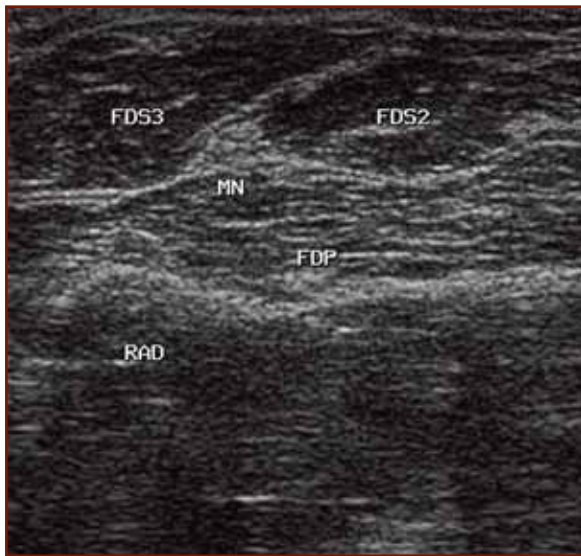


Scan A: Standard approach

We recently completed a study to determine whether or not standard surface localization techniques accurately target smaller forearm muscles for botulinum toxin injection. We compared standard EMG descriptions from Delagi¹¹ to MU localization using a 12 MHz linear transducer and passive movements. The skin was marked at the best point for injection where muscle movement occurred during real-time MU. Our results showed that MU was more accurate for localizing the pronator teres (PT) and flexor carpi radialis (FCR) muscles. See scans showing the difficulty of localizing the pronator teres and flexor carpi radialis in standard Delagi approach (Scan A) vs. the ultrasound-guided approach near the radius (RAD) (Scan B).



Scan B: Ultrasound-guided approach



Scan C.

Surface mapping for the FDS was accurate although MU showed that FDS 3 was more radial than FDS 2 compared to the median nerve (MN). Scan C illustrates the relationship of FDS bellies and the median nerve in the mid forearm.

Post-Injection Rehabilitation Protocols

Robust evidence suggests that task-oriented practice, where individuals engage in intensive practice using the upper limb in goal-oriented activities, is the most effective intervention for improving upper limb function after stroke¹². Task-oriented practice is more effective than simple strengthening exercises because it facilitates active recruitment of multiple muscle groups in a meaningful context (e.g., retrieving a glass from the tabletop), rather than facilitating recruitment of a single muscle group in isolation (e.g., bending an elbow). However, the application of task-oriented practice may vary based on the severity of upper limb impairment. For individuals who have minimal to moderate impairment, but are still able to actively recruit some wrist and finger movement, constraint-induced movement therapy is an option. Individuals with more-severe impairment, including spasticity, may benefit from neurolytic injections combined with rehabilitation devices (e.g., electrical stimulation or electromyography-driven devices) in order to facilitate task-oriented practice.

Case Vignette: Injection Protocol

Before a physician initiates injection therapy for regional spasticity, relevant anatomy and pathokinesiology must be fully understood. For the case study, outflow from the musculocutaneous nerve, in combination with activity in the brachioradialis, is keeping the elbow hyperflexed. Along with the tonic pull from the pectoralis and latissimus muscles that keep the shoulder adducted to the chest wall, these muscle groups promote the “boxer” posture described in the vignette. The fisted muscles are due to spastic overpull of both FDS and FDP muscles.

Treatment began with injecting 5ml of 6% aqueous phenol perineurally into the right musculocutaneous nerve. Once the phenol injection was completed, we injected a total of 400 units of botulinum toxin A, using a 1:1 dilution of preservative-free saline, into the right arm as listed in the table. Specific guidance technique for each muscle is described below.

Right Upper Limb Muscles Injected	Units
Pectoralis	100
Latissimus	70
Brachialis	30
Brachioradialis	40
Pronator teres with ultrasound guidance	20
Flexor carpi radialis with ultrasound guidance	20
Flexor digitorum superficialis with surface mapping	70
Flexor digitorum profundus with ultrasound guidance	50

Case Vignette: Clinical Outcomes

Before the injections, the patient had active right-hand grasp without any ability to voluntarily open his fist. He could not use the right arm functionally. After the injections, he could grasp and release objects like a small cup. He also demonstrated improved proximal range of motion that allowed him to grasp and release a dowel rod.

A video showing the patient discussed in this case three and a half weeks post treatment can be viewed on our website by typing <http://www.rehabmedicine.pitt.edu> in your Internet browser address box. Point your mouse at the picture labeled “3.5 weeks post treatment video” and click.



Post-injection video

Summary

Spasticity occurs commonly after stroke and other upper motor neuron disorders and can cause significant morbidity that impedes functional recovery. For regional spasticity affecting the upper limb, targeted injection therapy using phenol neurolysis and botulinum toxin injections in combination with post-injection therapy can offer patients significant improvement^{13,14}. Physicians should be aware of localizing techniques such as surface mapping, EMG, and ultrasound to accurately deliver the neurolytic agents into affected muscles and nerves.

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