Neurosurgical Management of Spastic Conditions of the Upper Extremity

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KEYWORDS

• Spasticity • Movement disorder • Upper extremity • Baclofen therapy • Neuromodulation

KEY POINTS

- Upper extremity spasticity is an unfortunate hypertonic consequence of many central pathologic conditions affecting the stretch reflex.
- Many surgical and nonsurgical approaches are used to treat upper extremity spasticity and its consequences.
- Despite treating the hypertonia associated with spasticity, surgical techniques for neuromodulation of muscle tone are unable to significantly improve the upper extremity functional impairment.

INTRODUCTION

Spasticity is best described as an uninhibited velocity-dependent activation of skeletal muscle reflexes resulting from upper motor neuron injury.[1,2] Surgical intervention for spasticity was first developed by the English neurophysiologist, Sir Charles Scott Sherrington. He experimentally induced a spastic condition in cats by separating the brainstem from the spinal cord and was able to abolish it by dividing their posterior rootlets.[3] This work, which was published in the 1890s and later awarded a Nobel Prize, was the foundation for practitioners who developed the dorsal rhizotomy (ie, sectioning of the dorsal nerve root or rootlets). Technological advancements over the past century have allowed surgeons to select abnormally firing dorsal roots with greater precision. Dorsal rhizotomy, when performed on the cervical roots, is effective in reducing spasticity in the upper extremities. However, the reduction in spasticity has not consistently resulted in functional improvement; for this reason, most cases were treated nonsurgically through the end of the twentieth century. Neurosurgical management of spasticity affecting the upper extremity is an evolving field. Despite modern technological gains, the goal of reliable functional improvement in the spastic patient remains elusive.

UPPER EXTREMITY SPASTICITY

Upper extremity spasticity may negatively affect quality of life and make activities of daily living difficult. The affected limb typically assumes a posture marked by internal rotation of the shoulder, a flexed elbow and wrist, and a clenched fist. Spasticity-related contractures may also interfere with residual function in the affected extremity. An effective treatment should relieve pain, address

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contractures, and, when possible, restore function. Because of the extensive fine motor function of the upper extremity, a satisfactory functional improvement is difficult to achieve.

Spasticity may result from any injury to the upper motor neuron, and the causes vary. There are congenital injuries, such as cerebral palsy (CP), and acquired ones, such as stroke, multiple sclerosis, and traumatic brain or spinal cord injury (SCI). The assessment of spasticity may be complicated by the presence of dystonia, a sustained series of muscle contractions that results in a writhing or twisting motion. Spasticity and dystonia often occur together and are difficult to distinguish.[4] In patients with mixed-movement disorders, reduction of spasticity may exacerbate underlying dystonia and worsen the overall functional impairment.

For the purposes of treatment, the underlying cause of spasticity is not as significant as the location and degree of the resulting symptoms. The treatment of upper extremity spasticity is typically a combination of surgical and nonsurgical; however, it always starts with conservative modalities, such as therapy and orthotics, intramuscular administration of botulinum toxin (Botox), and oral medications, such as muscle relaxants and anxiolytics.

Surgery for neuromodulation of muscle tone is reserved for cases that fail medical management. Targets have been explored throughout the neuromuscular pathway from cortex to muscle. These techniques include dorsal rhizotomy, intrathecal baclofen therapy (ITB), motor cortex stimulation, and deep brain stimulation (DBS). Musculoskeletal procedures to normalize the biomechanics of the upper extremity, such as tendon lengthening, tendon transfers, and osteotomies, may be used in combination with neurosurgical procedures. Regardless of the surgical approach, postoperative rehabilitation remains the cornerstone for the child or adult patient with spasticity. Furthermore, physical and occupational therapy are most effective when paired with a supportive home environment.

Improvements in muscle tone following Botox injections may be significant. However, studies demonstrate that without concurrent rehabilitation there is minimal to no correlated functional improvement.[5] For example, Russo and colleagues[6] found a clear functional benefit to post-Botox occupational therapy in a randomized controlled study of children with upper extremity spasticity; the improved spasticity lasted longer than Botox injections alone. This underscores the concept that integrated approaches are more successful than either therapy or Botox alone.[7]

MEDICAL TREATMENT

Therapy and orthotics are essential to prevent the development of contractures. These modalities, which can be delivered alone or in conjunction with medication, injections, or surgical treatments, are the mainstay of maintaining and improving function and accomplishing activities of daily living. There are a multitude of approaches; a full discussion is beyond the scope of this article, but a general methodology that promotes compensation strategies and active motor learning patterns have proven superior to passive manipulation and motion pattern normalization.[8–10]

Baclofen is the most commonly used medication for spasticity; nonetheless, there are other effective medications. Tizanidine, diazepam, and dantrolene are widely prescribed for spasticity, and several others, including clonidine, gabapentin, and cannabinoids, have been used to a lesser extent. Despite widespread acceptance and anecdotal reports of success, there is limited high-level evidence to guide the use of these medications.[11]

Intramuscular administration of Botox or intraneural injection of phenol for neurolysis has been described as effective nonsurgical treatments for spasticity. Botox acts by blocking the presynaptic release of acetylcholine, thereby reducing muscle contraction. It is an injectable agent with an expected duration of action of between 3 and 8 months. The medication may be administered multiple times; however, between 3% and 10% of patients develop antibodies to the toxin, which will reduce the effective duration. Botox is indicated for focal application to prevent the development of fixed contractures, to reduce spasticity compromising function, and for patient comfort.[12]

MUSCULOSKELETAL PROCEDURES

Serial casting may be used to lengthen a muscle shortened by contractures. There is robust evidence for this technique in the lower extremity, but it is not yet well accepted in the upper extremity.[13] The goal of upper extremity surgery in cases of spasticity is typically to improve function and posture of the affected upper limb. In cases of spasticity-related joint deformities, joint release, arthrodesis, tendon lengthenings, tendon transfers, and tendon rerouting may be used. Specific functional indications exist for various aspects of motor control in the elbow, wrist, fingers, and thumb. A careful assessment is required to determine which cases may benefit from this type of intervention.[14]