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A Comparison of Botox Injections to Selective Dorsal Rhizotomy in the Treatment of Spasticity

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A COMPARISON OF BOTOX INJECTIONS TO SELECTIVE DORSAL RHIZOTOMY IN THE TREATMENT OF SPASTICITY

by

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This Independent Study, submitted by Katherine S. Rogers in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Faculty Preceptor, Advisor, and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.

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Signature Katherine A Rogers

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ABSTRACT

Following certain types of perinatal or adult-onset brain damage spasticity is the common feature. A persons's disability can be greatly increased secondary to spasticity, which may present a major problem in the restoration of motor function.

Depending on the cause and location of brain injury, the clinical characteristics of spasticity can vary in signs and severity. These varying signs include flexor spasms in the patient with the spinal injury, dystonic posturing in the patient with hemiplegia and spastic diplegia in the child with cerebral palsy. It is apparent that many factors are involved with augmented reflexes and the list of possible sources is incomplete. Spasticity in stroke, spinal cord injury, and traumatic brain injury often interferes with function, limits independence and can result in secondary complications such as contractures. Considering the variety of problems associated with spasticity, it is unlikely that one agent will be beneficial to all patients. Because it takes normal muscle tone and normal control of the tone to give rise to normal movement, therapeutic intervention is often necessary and the type of intervention chosen to

treat a person's spasticity is best individualized for each person.

Two treatment methods currently used are Botox injections and selective dorsal rhizotomy. The purpose of this study is to review the literature on Botox injections and selective dorsal rhizotomy in the treatment of spasticity. This information will hopefully aid therapists in making decisions as to possible candidates for each type of treatment.

CHAPTER 1

INTRODUCTION

Following certain types of perinatal or adult - onset brain damage, spasticity is the common feature. Spasticity is defined as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper- excitability of the stretch reflex, as one component of the upper motor neuron syndrome." [1]

A person's disability can be greatly increased secondary to spasticity, which may present a major problem in the restoration of motor function.[2] Depending on the cause and location of brain injury, the clinical characteristics of spasticity can vary in signs and severity.[3] These varying signs include flexor spasms in the patient who has a spinal cord injury, dystonic posturing in the patient who has hemiplegia resulting from a stroke, and spastic diplegia in the child with cerebral palsy.[4] It is apparent that many factors are involved with augmented reflexes and the list of possible sources is incomplete.

Spasticity in stroke, spinal cord injury, and traumatic brain injury often interferes with function, limits independence and can result in

secondary complications such as contractures.[2] Considering the variety of problems associated with spasticity, it is unlikely that one agent will be beneficial to all patients.[4] Because it takes normal muscle tone and normal control of the tone to give rise to normal movement, therapeutic intervention is often necessary and the type of intervention chosen to treat a person's spasticity is best individualized for each person.[5]

Two treatment methods currently used to treat spasticity are Botox injections and selective dorsal rhizotomy. The purpose of this study is to review current literature on Botox injections and selective dorsal rhizotomy in the treatment of spasticity. This review will include an examination of spasticity, a discussion of Botox injections, and a discussion of selective dorsal rhizotomy. This information will hopefully aid therapists in making decisions as to possible candidates for each type of treatment.

CHAPTER 2

SPASTICITY

Before we can understand any type of treatment for spasticity, we must first try to understand what spasticity is and some of it's causes. Spasticity is more difficult to characterize than to recognize and still more difficult to quantify.[1] One widely accepted definition of spasticity is "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome."[6]

Spasticity is caused by an injury to upper-motor neurons and diagnostically it is the hallmark of an upper-motor neuron disorder.[1,3] Spasticity is often a common feature following certain types of perinatal or adult-onset brain damage.[7] Regardless of the age of onset of damage, accentuated alpha motor neuron discharge is present. Some of the neurophysiological variables, however, seem to show age-related differences. Reflex irradiation does not appear to be present following adult-onset cerebral vascular accident but is present in individuals with

cerebral palsy.

The clinical characteristics of spasticity can vary in signs and severity depending on the location and the cause of brain injury.[3] There are four signs of spasticity. Two of the signs, hypertonicity and hyperactive deep tendon reflexes, must be present in order to diagnose spasticity while the remaining two signs, clonus and muscle spasms, are usually, but not always, present in a person experiencing spasticity.

Hypertonicity, also known as increased muscle tone, is an involuntary resistance to a quick movement or stretch felt in an affected limb by an examiner.[3] The tension between the origin and insertion of each muscle is referred to as muscle tone.[8] Muscle tone can be defined as "the sensation of resistance felt as one manipulates a joint through a range of motion, with the subject attempting to relax."[1] Muscle tone consists of several distinct components: 1) physical inertia of the extremity, 2) mechanical-elastic characteristics of muscular and connective tissues, and 3) reflex muscle contraction (tonic stretch reflexes). Hyperactive tonic stretch reflexes are classic components of spasticity.[8] Normal postural tone can be defined as "a state of readiness."[5] Muscle tone must be sufficiently high to provide support against gravity yet low enough to allow a person to move freely.

Normal movement requires normal muscle tone and normal control of the tone.

Hyperactive deep tendon reflexes, resulting in a condition referred to as hyperreflexia, can be seen in individuals with spastic-type cerebral palsy as an accentuated myotatic (stretch) reflexes.[7] This hyperactive reflex can also exhibit reflex irradiation where by one muscle group receives a tendon tap (e.g. quadriceps) and EMG potentials are seen in that muscle group as well as it's antagonist (i.e. hamstrings). Hyperactive reflexes can also be seen in muscles at sites distant from the point of the tendon tap. When one has a loss of descending inhibition from higher centers due to some type of injury, the result can be reflex irradiation.[7] To be considered as a positive sign of hyperactive deep tendon reflex, there must be a decrease in the amount of stimulus needed to elicit a knee jerk or an exaggerated muscle response.[3]

According to Howe and Oldham,[5] "clonus is a series of repetitive muscle contractions elicited by a rapidly applied, but maintained, stretch."[5] Clonus can often be elicited in the patient with spasticity by quickly initiating and then holding the foot in dorsiflexion. One will see a rhythmic contraction and partial relaxation of affected muscles in response to the sustained stretch.[3] Clonus is believed to be the result of alteration of the normal pattern of motor neuron discharge.[7]

Spasms, according to Webster,[9] are "an involuntary and abnormal contractions of muscle or muscle fibers." Spasms are often in response to a noxious stimulus.[3] The triggering stimulus is from a visceral or cutaneous source making spasms unlike the other signs of spasticity which are triggered only by the stretch of skeletal muscle fibers. It is important to consider spasms as a sign of spasticity due to the fact that both spasticity, which is triggered by a muscle stretch, and spasms, proceeded by noxious stimulus, are often present in the same patient, although one or the other is usually dominant.[3] The spasm can take the form of sudden flexion or extension. The form demonstrated depends on several factors, one being the site of the triggering stimulus. Whether flexion or extension is elicited depends greatly on which would be most protective to the body. The reflex becomes exaggerated due to lack of modification of the reflex by higher motor centers. Katz and Rymer[1] proposed the idea that changes in the intrinsic muscle mechanical properties (rather than stretch reflex enhancement) are largely responsible for spastic hypertonia. These possible neural mechanisms for spastic hypertonia include increased motor neuronal excitability via three ways. The first two ways are: 1) excitatory synaptic input is enhanced and 2) inhibitory synaptic input is reduced. The third way is by a change in the intrinsic electrical properties of the neuron or secondly

enhanced stretch-evoked synaptic excitation of motor neurons through 1) hyperactivity of gamma efferent or 2) excitatory interneurons more sensitive to muscle afferent input. St. George[3] proposed that spasticity occurs when there is lack of control of spinal reflex activity because of the upper motor neuron damage. It is apparent that many factors are involved regarding augmented reflexes and the list of possible sources is incomplete.[1]

According to Howe and Oldham,[5] authors have attempted to grade spasticity but the quantification of spasticity is a challenging and difficult problem.[1,2] Classification of spasticity has been based primarily on highly observer-dependent measurements with a concentration on tabulation of functional activities, biomechanical analysis of limb resistance to mechanical displacement, purified surface electromyographic responses to agitation, voluntary movement, gait analysis and other clinical measurements.[1,2] Several systems have been composed to grade spasticity such as the Modified Ashworth Scale[3] and the Clinical Scale for Spastic Hypertonia.[1] Spasticity is ranked from mild to severe and the criteria for grading the severity of spasticity are not standardized, therefore there is no precise method for grading the severity of spasticity.[3] The ability to quantify the presence and severity of spasticity can be essential to the understanding and treatment of this

disorder.[2] However, since no standardized test of spasticity exists and the effect of abnormal tone on movement can be clearly seen, it may be appropriate to substitute a measurement of the 'quality' of movement.[5] Measuring the quality of movement can also be valuable in assessing the effects of a therapeutic interventions such as drug treatments or surgical procedures.[2] A person's disability can be greatly increased secondary to spasticity, especially in an incompletely paralyzed person.[8] Spasticity is always present as part of a movement but is never confined to one muscle group.[5] Flexor or adductor spasms may result from a release of reflexes from descending inhibitory control.[1] For example, a person with paraplegia may require the use of restraints in his wheelchair due to severe flexor spasms.

Spasticity presents major problems in the restoration of motor function and because its presence is so predictable, it is reasonable to base therapies on the appearance of spasticity.[5] "Scissoring" of the lower extremities may be the result of hip adductor spasms and may limit a patient's ability to ambulate effectively.[1] It is also important to realize when spasticity is beneficial to the patient in carrying out basic activities of daily living such as transferring. A person with mild spasticity can trigger extensor spasms in their legs that enables them to stand straight

while transferring into bed, thus making the transfer easier.[3] Therefore it is important to note that reduction of spasticity does not necessarily result in functional gains.[7] For therapeutic interventions, functional gains must be the goal. It is unlikely that one agent will be beneficial to all patients considering the variety of problems associated with spasticity. "The functional impairment due to spasticity must be carefully assessed before any treatment is considered."[4] Therapeutic intervention should be individualized for each patient and therefore it is important to know the different options for intervention.

CHAPTER 3

BOTOX INJECTIONS

"Clostridium botulinum" is a bacterium that causes a serious form of food poisoning.[10] The commercial name for this bacterium is "Botox". The paralytic neurotoxin has become widely investigated as a potential therapeutic treatment for a variety or neuromuscular disorders[11] and due to the fact that in purified form, it is the first bacterial toxin to be used in medicine.[12] Botox was initially used in the early 1980s to treat strabismus[12,13] (crossed eye) and blepharospasm[1-17] (squinting) and the Food and Drug Administration (FDA) licensed botulinum toxin as an oculinum in December 1989 for these two specific eye conditions.[12] Since 1986[18] Botox has also been used to treat spasmodic dysphonia,[11,14,15,17,19,20,21] and is presently used to treat many other conditions which include spasmodic torticollis,[11,15,17,22] cerebral palsy,[17,23] dystonia,[12,15,24-27] myofascial pain syndrome,[26] achalasia,[28,29] hemifacial spasm,[11,15,24] and progressive supranuclear palsy.[30] Epperson[19] reported Scott as pioneering the use of botulinum toxin for use as a chemical denervation of extraocular muscles, resulting in a new area in neurology

called "interventional neurology."

The bacteria of Clostridium botulinum produces seven[12,19] or eight[15,17,23] serologically distinct toxins that are potent neuroparalytic agents. The strains of toxin have been identified and labeled A through G[12,15,17,19,23] with the eighth strain distinguished as C-2.[15] The bacteria and spores are harmless, but the toxin produced by the bacteria, when they grow, are very dangerous.[12] Botulinum toxins are considered the most poisonous substances known to humankind.[12,15] This anaerobic organism, sometimes found in improperly canned food is a potentially lethal toxin.[20] The food poisoning, due to botulism, blocks the transmission of nerve impulse to a muscle and this results in muscle weakness, paralysis[10] and ultimately death.[12] Death occurs by suffocation as the botulism paralyzes the diaphragm and prevents the lungs from functioning.[28] Type A toxin, the most potent known biologic toxin, is easily crystallized and relatively stable.[17] The toxin is supplied in vials in small amounts which make the administration of a fatal dose extremely difficult.

In the clinic, when used in minute doses, Botox becomes a very effective means for relieving spasmodic symptoms.[20] When small doses of the toxin are injected into the affected muscle,[12] it acts presynaptically at the nerve terminals to prevent calcium dependent [20,30] release of vesicle bound acetycholine. The process is not fully

understood but the toxin prevents exocytosis of neurotransmitter vesicles.[15] There are three hypothesized mechanisms a) inhibition of calcium influx during nerve stimulation, b) induction of calcium ion efflux and c) direct action of the toxin on acetycholine release at the nerve terminals.[17] The final outcome is that effective neuromuscular control is blocked and a functionally denervated muscle results. Focal weakening and atrophy of the injected muscle is the final result of local Botox intramuscular injections.

The most effective Botox dose per muscle is not known.[31] For the treatment of blepharospasm, the recommended dose is 10-30U/eye whereas for Dysport, another trade name for botulinum toxin, it is 100-200 U/eye.[11] The lethal dose for an adult human is estimated at 5,000U.[17] In a study done by Koman et al,[17] initial dosage was determined on the basis of patient body weight and its distribution was based on the relative mass of the muscle causing deformity. Comella et al[31] showed improvement was more significant in patients who received EMG-assisted Botox injections versus clinically assisted injections. The reason for greater improvements with EMG assistance is believed to be due to the increased accuracy in identifying overactive muscles. Koman et al[17] also reported on a protocal where pediatric patients

received small initial dosages of 1-2U/kg and dosages were increased at each subsequent follow-up evaluation until the target threshold for the desired muscle was reached. Threshold was defined as the dose of Botox necessary to a) eliminate dynamic deformity, b) allow antagonist muscle(s) to correct the dynamic deformity either partially or completely, or c) allow effective bracing.

In the literature, there are a number of studies showing the positive effects of Botox Injections. Polo and Jabbari[30] reported a patient with progressive supranuclear palsy was significantly handicapped by the degree of his rigidity. After receiving local injections of botulinum toxin in the upper limb muscles, the patient showed marked improvement and was able to move his frozen limb. There were a number of studies on the treatment of dystonia.[12,15,24-27] Dystonia is a neurological disorder characterized by involuntary, sustained, intermittent or repetitive muscle contractions resulting in abnormal twisting or deviations of posture.[26] After receiving Botox injections, patients with many types of dystonia, such as blepharospasm, spasmodic torticollis and spasmodic dysphonia, felt relief when the abnormally contracting muscles were made to relax. Metzer and Jenkins[22] reviewed several studies and reported significant benefit in 53-90 % of patients with cervical dystonia after Botox injections.

Even when head control was not dramatically improved, pain significantly decreased. In the patient who is in the advanced stage of Parkinson's disease, foot dystonia is frequently observed.[25] The foot dystonia is often painful and resembles a common muscle spasm. The pain is probably due to sustained muscle contraction and often interferes with daily activities. Significant improvement of pain and dystonia after Botox injections was observed in the study by Pacchetti et al.[25] Botox injections to lower extremity adductor muscles led to pronounced improvements in daily toilet care for a patient with multiple sclerosis and severe adductor spasticity.[14]

In cerebral palsy, the lesion in the central nervous system often results in spasticity of various muscle groups.[23] Due to the increase in tone, functional problems develop. The increased tone may also be responsible for the failure of longitudinal muscle growth which is characteristic of the condition. Botulinum Toxin-A may have a twofold benefit to patients with cerebral palsy: 1) reduced levels of hypertonicity would allow more control of movement for the duration of the toxin's effect and 2) increased stretch of the relaxed muscle would promote longitudinal growth, and reduce the incidence of degree of fixed contractures. In the study done by Miller and Dabney,[23] parents reported that tone was clinically decreased in most patients, with the

exception of one child who had fixed contractures. Some parents even reported a gain of maximum range of motion. Koman et al[17] reported that children with cerebral palsy, who were treated with Botox, showed noticeable improvements in easing patient care, positioning, and function. The literature reflects various response times to the Botox injections with initial change in spasticity seen in as little as three[23] days and effects lasting up to six months.[17,19,20-22]

Some of the drawbacks of the use of Botox injection include the side effects that can occur. The side effects are infrequent and transient. When used in the treatment of blepharospasm, side effects have included drooping of the eyelid, blurred vision and double vision, which all recovered spontaneously.[16] Weakness in the injected muscle is the principle side effect and can impair useful motor function.[26] Transient weakness can also occur in nearby muscle due to regional diffusion. For example, swallowing difficulties have been reported by those patients being treated for cervical dystonia.[32]

Injections have to be repeated as symptomatology returns due to the temporary effect of Botox injections.[20] Muscle recovery occurs when the muscle develops new extra-junctional acetylcholine receptors.[17,19] New synaptic contacts on the adjacent muscle fiber receptors are formed when the terminal axon begins to sprout new branches.

Fast-twitch muscle is functionally denervated longer than slow-twitch muscle.[17] Effective dosages tend to decrease and the interval between necessary injections tends to increase with repeated therapy.[20] Because of the repeated injections, 10%[12] of patients will develop antibodies to the toxin.[22,26]

There are several advantages of Botox injections over surgical therapy in the management of intractable diseases.[19] These advantages include 1) varied degrees of weakening can be achieved by varying the injected dosages and 2) the patient is awake and therefore you don't have the risk of anesthesia or postoperative infection.[19,23]

Because Botox has only been used briefly in the clinic, long term effects are unknown but no lasting side effects have been reported.[13] However, due to the fact that functional nerve sprouts form after each injection, altering the architecture of muscle innervation, more study is needed on the effect of repeated injections.[15]

CHAPTER 4

SELECTIVE DORSAL RHIZOTOMY

A surgical procedure designed to reduce spasticity is selective dorsal rhizotomy.[33] The term selective refers to the selective sectioning of segmental rootlets or fascicles with abnormal neurophysiologic characteristics and sparing of a variable proportion of rootlets with normal responses. The term rhizotomy is derived from the word rhizomes which is another term for nerve rootlet.[34]

According to Walker,[34] in 1898, Sherrington rendered cats spastic by transecting their midbrains. He then severed the posterior nerve roots of the spinal nerves and the spasticity disappeared, an introduction of the procedure referred to as selective dorsal rhizotomy. Fifteen years later, Foerster used the technique to reduce tone in patients with congenital spastic paraplegia.[35]

The reasoning behind selective dorsal rhizotomy is to get a neurolytic reduction in afferent facilitatory influences on the anterior horn cell in the patient who demonstrates spasticity and has diminished inhibitory influences from descending tracts.[36]

According to Sweetser et al,[37] a revised technique was introduced into the United States in 1986 which had decreased complications and has increased the prominence of selective dorsal rhizotomy as a treatment for spastic cerebral palsy. The revisements have improved the safety and efficacy of selective dorsal rhizotomy. Peacock and Staudt[38] suggested that the principle goal of the surgery is to either improve functional performance or to ease the daily care routines of the person with spasticity.

Selective dorsal rhizotomy has been used for both adults and children with various diagnoses.[39] Some of these diagnoses include patients with multiple sclerosis, spinal cord injuries, myelomeningocele with tethered cord, and traumatic brain injuries.[33] When selective dorsal rhizotomy has been applied to pediatric populations, it is most commonly used to treat children with cerebral palsy.

The procedure involves a lumbosacral laminectomy and dural incision to expose lumbar and sacral spinal nerve roots.[40] Intra operative electromyography is used to isolate dorsal rootlet bundles associated with sustained muscular contraction or with diffusion of contractions of muscle groups not belonging to that nerve's segmental distribution. Rootlet bundles (containing 3 -5 rootlets) that do not have normal inhibitory responses to electrical stimulation are sectioned. The rhizotomy

of the dorsal roots is performed between L2 - S2 but care is taken to avoid cutting more than two adjacent nerve roots which can cause excess loss of sensation.[37] Fifty percent or more of rootlets may be sectioned at a specific root with no detectable loss of cutaneous sensation in corresponding dermatomes. The S3 and S4 roots are always avoided at all costs to spare bladder innervation.[33,41]

The success of selective dorsal rhizotomy depends on the careful selection of patients.[36] Children with spastic diplegia, who are able to walk independently and willing to cooperate with the rigorous physical therapy that will follow the surgery, are considered ideal candidates for selective dorsal rhizotomy.[40] Children with other problems such as rigidity, dyskinesia or ataxia do not benefit and children with hemiplegia are considered too mildly involved to benefit.[36] Children who have had insufficient anti-gravity strength to maintain their limb function without the spasticity in their legs would not be selected as candidates for selective dorsal rhizotomy for obvious reasons.[42] According to Hendricks,[43] selection criteria at the St. Louis Children's Hospital included diagnoses of spastic diplegia or quadriplegia in patients between the age of two and twenty-three years of age and no evidence of significant damage to basal ganglia on magnetic resonance imaging.

Other selection factors included good muscle strength in the legs and trunk, history of delayed motor development and motivation and ability to cooperate in therapy. As mentioned earlier, not all children benefit from selective dorsal rhizotomy. Individuals who are not candidates for the procedure include those with a history of meningitis, congenital infection, head trauma, severe scoliosis and those who would not be expected to make functional gains after surgery.

According to Goldstein,[44] selective dorsal rhizotomy is not usually performed until a child is at least three years old and more commonly, seven years old or older. Because the brain cells and the connections in the brain may change as the child matures, the clinicians often wait until a child is three years old before identifying the type and severity of his or her cerebral palsy. In contrast, according to McDonald,[33] maximal functional benefit was shown to occur in preschool age children with spastic diplegia. This was attributed to the fact that younger patients may have fewer abnormal movement patterns developed and they tend to have fewer musculoskeletal deformities.

Surgery always involves some risk, such as infection, unexpected bleeding, or side effects of anesthesia.[40] The risk that the wrong nerves or too many nerves will be cut are additional risks of selective dorsal

rhizotomy. This could result in further complications and can cause new problems. Intraoperative complications can occur with each child who undergoes selective dorsal rhizotomy.[43] Such complications include impaired skin integrity related to the surgical incision, immobility or improper positioning, alteration in comfort related to the surgical incision, alteration in urinary elimination and neuromuscular changes related to improper positioning. Even when appropriate nerve rootlets are cut, some children experience sensory abnormalities, such as numbness or tingling in some areas of the body.[40] Serious peri-operative complications which included bronchospasms, aspiration pneumonia, urinary retention, sensory loss, cerebrospinal fluid leakage, temporary loss of bowel and bladder function, spinal subdural hematoma and urinary tract infection where experienced by some patients.[33,37,41,42,44] Post-operatively, the patient will experience a great deal of pain for several days.[34,40,41,44] There are several components to the post-operative pain in this patient population: somatic pain at the operative site, dysaethesia and hyperaesthesia of the lower extremities, and distress and discomfort resulting from intermittent muscle spasms of the lower extremities.[40] According to McDonald and Hays,[41] severe post-operative pain was reported in 58% of patients. The effect of dorsal rhizotomy documented in the literature provides evidence that, immediately after surgery

selective dorsal rhizotomy reduces spasticity and increases joint range of movement, apparently limited by abnormal muscle tone.[35,40] Gaskill and Steinbok[45] reported decreased spasticity and improved ease of care taking in non-ambulatory children and improved ambulation in ambulatory children. Selective dorsal rhizotomy can permanently reduce spasticity so that the child with spastic cerebral palsy experiences little or no stiffness, has more normal muscle tone, and experiences greater ease of movement.[43] Abbott et al[42] noted that all post selective dorsal rhizotomy groups studied had statistically significant improvements in the tone of every muscle tested. Goniometric examination was significantly improved in all joints evaluated. Children participating in this study experienced dramatic decreases in leg muscle tone and corresponding improvements in passive range of motion in their lower extremities one year after surgery. Staudt and Peacock[39] stated that functional improvements have been more difficult to quantify, but rating of gross motor skills has revealed improvements in developmental postures, transitional movements, and walking abilities.

When improvement occurs, it may be different for each child.[44] Children with spastic diplegia may be able to stand with their feet flat on the floor, and may also make gains in walking, climbing stairs or self-care

tasks following selective dorsal rhizotomy. According to Goldstein,[44] some report improvement in skills that include upper body movements which are probably related to an improvement in overall balance and stability. Improvement in sitting or in the ability to transfer from one seated position to another has been seen in children with spastic quadriplegia following selective dorsal rhizotomy. Children may also improve in self-care skills and be less dependent on the assistance of others.

According to Goldstein,[44] not all children improve after selective dorsal rhizotomy and some even lose ground. Giuliani[35] reviewed several studies and reported a decrease in strength in the lower limbs initially following selective dorsal rhizotomy. Strength levels, however, gradually increased resulting in improved function. Abbott et al[42] reported significant deterioration in plantar flexor ranges, for some subjects, between six and twelve month examinations. Dudgeon et al[36] stated that some children, who lacked voluntary muscle control or strength, might have learned to use their spasticity to support upright stance, sitting or hand placement. After selective dorsal rhizotomy, retraining may be unsuccessful and therefore result in a loss of function or difficulty and delay in resuming previous levels of function. McDonald and Hays[41] reported that after three years, 5% of patients had

recurrence of spasticity and an additional 2.5% had recurrence when followed fifteen years after selective dorsal rhizotomy.

Children with cerebral palsy are at risk of developing a symptomatic neurogenic bladder.[42] Symptoms suggestive of neurogenic bladder include enuresis, stress incontinence, and dribbling. The reduction in spasticity following selective dorsal rhizotomy can also result in a hypotonic bladder requiring intermittent catheterization on either a temporary or permanent basis. Selective dorsal rhizotomy reduces spasticity but does not have a direct effect on balance, abnormal movement patterns, or persistent tonic reflexes.[43] Perhaps the most important factors influencing outcomes are the child's motivation, cooperation, and intelligence and the level of family support available as the child gains new skills. Objective evaluation of treatment outcome in cerebral palsy remains a challenge.[33] To date, most of the evaluation of progress after selective dorsal rhizotomy has been subjective in nature.

Following selective dorsal rhizotomy, weakness, primitive movement patterns, and impaired motor control persist.[33] For this reason, intensive therapy appears necessary to maximize ultimate functional gains. McDonald and Hays[41] suggested that intensive and consistent physical therapy, tailored to the needs of the individual child, is essential

to ensure maximum physical progress and functional gain during the postoperative period.

The literature suggests that selective dorsal rhizotomy can be useful for spasticity in cerebral palsy when patients are carefully selected.[41] Younger children with spastic diplegia, who are seriously impaired by spasticity but have retained good selective motor control and some degree of forward locomotion appear to make the most functional gains in uncontrolled studies. Pre-operatively, physical therapists also rule out candidates who have motor impairment due to dystonia, athetosis, ataxia, and abnormal reflex posturing,[34] who would not be good candidates for the surgical procedure. It is, however, very difficult to make decisions with absolute confidence regarding who should receive selective dorsal rhizotomy and therapy.[36] Further clarification is needed to determine which children have the most to gain from the reduction of spasticity. The long term effects of rhizotomy have not been documented and questions remain as to whether the weakening that inherently accompanies rhizotomy will be counterproductive as age and weight advance.[45]

CHAPTER 5

CONCLUSION

Spasticity is very complex and not completely understood. Physical therapists are often faced with this phenomenon that is observed in many types of patients. Some patients who often show signs of spasticity include those who have suffered from spinal cord injuries, strokes and cerebral palsy. A person's disability may be greatly increased secondary to spasticity. Depending on the cause, clinical characteristics may vary in signs and severity. Spasticity may interfere with function, limit a person's independence and may cause secondary complications such as contractures.

Although spasticity presents major problems in the restoration of motor function, it is important to realize that spasticity may be beneficial in some patients in carrying out basic activities of daily living. Physical therapists play a vital role in the treatment of spasticity. They will not be the ones to prescribe Botox injections or selective dorsal rhizotomy but a therapist's observation can help determine if in fact the spasticity is disabling or of benefit to the patient.

When it has been determined that the patient's spasticity is so severe and is causing multiple complications that interfere with activities of daily living, medical management will become necessary. Two options for the treatment of spasticity include Botox injections and selective dorsal rhizotomy. The choice of treatment will depend on the severity of the spasticity, the goals to be achieved with spasticity reduction and criteria for patient selection.

Botox injection therapy is a more conservative treatment form than selective dorsal rhizotomy. Botox is injected directly into the affected muscle(s) whereas selective dorsal rhizotomy is a surgical procedure that requires the cutting of sacral spinal nerve roots.

Unlike Botox, in which effects last temporarily unless use is continued and/or repeated, selective dorsal rhizotomy is permanent and irreversible due to nerve rootlet severing. Botox can be used in patients who exhibit many types of dystonia whereas selective dorsal rhizotomy is not recommended. Botox can be used in spastic muscles of the upper and lower extremity and selective dorsal rhizotomy is used when lower extremity spasticity is problematic.

There are documented side effects with the use of Botox but these are infrequent and transient. There are also advantages of using Botox injections over surgical intervention, one of which is that there is no risk

secondary to the use of anesthesia which is required in selective dorsal rhizotomy but not with the use of Botox injection.

With selective dorsal rhizotomy, there is risk of cutting too many or the wrong nerve rootlets, resulting in permanent secondary complications such as an alteration in urinary elimination. With Botox injections the most effective dose per muscle is not known and there is risk of transient weakness in nearby muscles due to regional diffusion. However, this weakness is only temporary.

Botox use in the clinic has been very limited and long term effects are unknown. New nerve sprouts form after injection and these sprouts can alter the architecture of muscle enervation. The consequences from this require more study. Selective dorsal rhizotomy has been around for many years and revised techniques have improved upon its safety and efficacy. However, not all patients improve functionally and the question remains if the procedure is counterproductive as the age and weight of the patient advances.

In conclusion, compelling evidence has been presented in the literature showing that even when spasticity is reduced there may be no improvement of function. Therapists need to be aware of this and realize that reducing the tone may increase the range of motion, but may unmask underlying weakness rather than underlying control.

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