

THE TREATMENT OF CEREBRAL ORIGIN
SPASTICITY IN CHILDREN

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Key Words: spasticity, cerebral palsy, botulinum toxin, phenol, selective dorsal rhizotomy, intrathecal baclofen pump.

ABSTRACT: As in adults, spasticity of cerebral origin in children can interfere with function, cares, positioning, and comfort. Reducing spasticity may improve overall function. Many treatment modalities have been developed to treat spasticity in children. This article reviews some of these treatment options including bracing, physical and occupational therapy, oral medications, neurolytic blocks, neurosurgical procedures, and orthopaedic surgery. Specific characteristics of children which influence treatment will also be discussed. Most children benefit from a combination of treatments and not just a single treatment modality. The optimal treatment combination usually changes over time as a child grows and develops. Once the special characteristics of children and the various treatment options are understood, the treatment of cerebral origin spasticity in children can be optimized.

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Cerebral palsy(CP), traumatic brain injury(TBI), and other encephalopathies in children may lead to neurologic deficits that interfere with motor function, cares, comfort, or positioning. These impairments include weakness, incoordination, loss of selected motor control, sensory deficits, cognitive deficits, and spasticity. Although current treatments cannot eliminate all of the problems associated with these disorders, treatments do exist to minimize the impact these impairments, especially spasticity, can have on function. According to Molnar (Molnar 1992) the goal of treating children with cerebral palsy or traumatic brain injury is to assist in acquiring functional skills and minimize complications associated with the brain injury. Spasticity reduction can help achieve these goals.

Basic management principles do not differ significantly between children and adults, but some differing characteristics do influence management decisions. To attain developmental milestones a child's brain is dependent on early stimulation, when the brain is most plastic. Early stimulation helps lay down the appropriate engrams and develop the appropriate brain architecture (Mahajan and Desiraju 1988). For example, in a child with strabismus, if the non-dominant eye does not receive early stimulation, vision will be permanently lost even if the strabismus is corrected at a later age (Traboulsi and Maumenee 1990). To maximize developmental potential, a child should be treated at any age a significant functional impairment exists.

Children with neurological impairments will have altered development because of their brain abnormality, but still make functional improvements due to brain maturation. Medical interventions intended to minimize the impact of neurological impairments can improve function. Discerning if these functional improvements in a child are due to brain maturation or medical interventions can be difficult. Although brain maturation can improve function, growth itself may adversely impact function in a child with a brain injury. For example, contractures may worsen in children during a growth spurt (O'Dwyer, Neilson et al. 1989). Adolescents are particularly susceptible to these changes and often have a deterioration of function during their pubescent growth spurt. Sometimes the skills lost are not regained. Because of these various factors, a child's function continually evolves as he matures. Thus, optimal treatment for these children can change over time.

Over the last ten years significant advances have been made in the treatment of spasticity, especially in children. Historically, spasticity in children has been managed with oral medications such as baclofen, diazepam, and dantrolene, neurolytic blocks using phenol or alcohol, range of motion exercises, bracing, (Little and Merritt 1988), or orthopaedic surgery (Chambers 1997); all with mixed success. With the recent addition of selective dorsal rhizotomies, intrathecal baclofen pumps, and botulinum toxin injections to the spasticity treatment options, the impact spasticity has on a child's function can be better managed.

When treating children with spasticity, goals need to be clearly outlined. Spasticity should not be treated because of its mere presence, but only if it adversely impacts some level of the patients functioning and if treatment would minimize this

impact. A team approach utilizing various healthcare professionals is necessary to best determine the appropriate treatment modalities to optimize outcomes.

Various factors influence the choice of a treatment modality. A patient's age, size, future risk of musculoskeletal deformities, developmental potential, and neurologic progression impact this decision making process. For example, a selective dorsal rhizotomy has an ideal age range in which the procedure should be performed and a child must be of minimal size to accommodate an intrathecal baclofen pump. If a child's spasticity is likely to lead to various musculoskeletal deformities, such as hip dislocation, contractures, or torsional deformities, spasticity treatment may be warranted even if treatment is unlikely to initially impact function. The child's developmental potential may also affect the decision to treat. For example, a 14 year old child with spastic hemiplegic cerebral palsy who has learned to function very well using only the unimpaired arm probably will not see as much improvement in function following spasticity treatment, as a one year old child might with a similar impairment. The ability to change development and function is easier in a one year old child than it would be in a 14 year old child with a long standing disorder. Rapidly progressive neurologic disorders may preclude use of a selective dorsal rhizotomy or orthopaedic surgery because of the uncertain long term benefits.

Most children with spasticity do not have one ideal spasticity treatment modality, but benefit from various treatments (See Table I). This can include a combination of bracing, stretching exercises, oral medication, and surgery. A child with spasticity should not necessarily be treated following the "pyramid" approach from most conservative treatments to the most aggressive treatments. All children should be assessed carefully

and the most effective treatment options utilized considering the relative risk and benefits.

The following is a summary of commonly used treatment modalities for children with cerebral origin spasticity.

BRACES

In rehabilitation, braces have been used to improve function. Usually they are intended to accommodate for weakness and instability. Although braces are not usually intended to reduce spasticity, they may help prevent complications of spasticity such as contractures or help prevent abnormal positioning of a joint, such as preventing excessive plantarflexion during the gait cycle. Rigid ankle-foot orthoses (AFO's) can decrease clonus at the ankle on computerized gait analysis. The AFO is thought to prevent the necessary stretch of a muscle that would to elicit a spastic response (Novacheck 1998).

Although not traditionally used to treat spasticity, AFO's in the past decade have incorporated "tone inhibitive" features in children's braces (Harris and Riffle 1986). Certain areas on the plantar surface are built up and felt to inhibit some of the nociceptive impulses that elicit spasticity. No well controlled studies have demonstrated different gait patterns in "tone reducing" AFO's versus standard AFO's and these braces have been used less over the past few years.

PHYSICAL AND OCCUPATIONAL THERAPY

Physical and occupational therapy has been a mainstay in the treatment of children with cerebral palsy and other brain injuries (Taggart and Matthews 1992). There have been no well controlled randomized trials to assess the effectiveness of therapy. Well controlled studies in CP are difficult to carry out for various reasons, including

reluctance of patients to be in the control group (without physical therapy), diversity of participants, and difficulties in measuring outcomes in a growing developing population. Therapists do provide range of motion exercises, which is felt to decrease spasticity by reducing contractures thus decreasing tension on the stretch receptor (Odeen 1981). A recent study however found no change in spasticity in children with cerebral palsy treated with range of motion exercises by a physical therapist (McLaughlin, Bjornson et al. 1996).

Although direct physical and occupational therapy may not alter spasticity, it is considered a necessary adjunct to spasticity management (Leach 1997). Therapists assist in patient selection for spasticity treatment, establishment of appropriate goals and measurement of functional changes following treatment. Therapists also provide goal directed therapy. When spasticity is reduced, it is important for a patient to take advantage of the reduction in muscle tone and work to acquire new functional skills. The therapist can be valuable in acquiring these skills.

ORAL MEDICATIONS

Oral medications are commonly used to treat spasticity in children. Oral medications are a systemic treatment, not a focal treatment. In a child that has generalized spasticity oral medications may be appropriate to help manage the spasticity. However, if a child needs only focal treatment of their spasticity then the more focal modalities would be appropriate.

Four oral medications are commonly used to treat spasticity in children: Baclofen, diazepam, dantrolene, and more recently tizanidine. Most oral medications have been poorly studied in spasticity of cerebral origin, especially in children. Oral

medications have had a mixed level of success in improving function and in some centers are used sparingly to treat children (See Table 2).

The effects of diazepam on spasticity have been studied in children with cerebral palsy. A double blind cross over study showed improvement in spasticity using doses of 8.75-20mg per day (Engle 1966). Diazepam can be very sedating in children and often not well tolerated. A tolerance to the sedating effects can occur over several days to a week. Diazepam has the advantage of being able to be given by oral, nasal, buccal, rectal, intravenous, and intramuscular routes. It can be given in liquid form in which it is very stable. The usual dose is 0.2-0.8mg. /kg of body weight per 24 hours divided every 6-8 hours (Greene 1996). Diazepam withdrawal can be severe and should be tapered slowly over several months (Gracies, P et al. 1997).

Baclofen has been poorly studied in spasticity of cerebral origin with most studies evaluating efficacy in treating spasticity of spinal cord origin. Although no studies on the use of baclofen to treat children was found, it is still commonly recommended as a treatment option for children with spasticity (Stempien and Gaebler-Spira 1996). Baclofen can also be sedating (Whyte and Robinson 1990), but again tolerance may develop over several days. Baclofen is stable in liquid form, but can only be given enterally (except if given intrathecally). Baclofen can not be given intravenously and is not absorbed rectally (Kriel, Krach et al. 1997) Children can have similar withdrawal symptoms as in adults, which includes hallucinations and seizures (Reisman and BM 1997). The usually starting dose is 2.5mg a day and titrated up every 3-5 days to a maximum of 20-60mg. per day (Gracies, P et al. 1997).

Dantrolene is the anti-spasticity medication most often recommended for spasticity of cerebral origin (Whyte and Robinson 1990). Like the other oral medications, it has been poorly studied in children but it has been shown to improve spasticity in children with cerebral palsy (Haslam, Walcher et al. 1974). Although Dantrolene can be put into a suspension, it is unstable in liquid form and needs to be reformulated every few days. So, if a child must take it in liquid form, it may be too inconvenient to use. Dantrolene has a hepatotoxicity rate of 1.8% (Ward, Chaffman et al. 1986) but has not been reported in children less than 16 years of age. Dantrolene can also be sedating, but less so than diazepam and baclofen. Weakness can be a limiting side effect (Whyte and Robinson 1990). The usual dose is 0.5mg per kg of body weight per dose by mouth b.i.d. increasing to a maximum dose of 3mg per kg of body weight per dose q.i.d. up to 400mg. per day (Greene 1996). Although the risk of hepatic toxicity may low, liver function tests should be performed before treatment and periodically thereafter.

Tizanidine has recently been approved to treat spasticity of spinal cord origin in adults. Tizanidine has been poorly studied in spasticity of cerebral origin and no studies could be found on its use in children with cerebral palsy or traumatic brain injury. In spite of this, some clinicians are using it to treat spasticity in children. Sedation is the most common side effect (Lapierre, Bouchard et al. 1987). No dosing guidelines have been established in children. Anesthesiologists at our center have noted post-operative difficulties in arousal and pain management in patients on tizanidine. Children at our center are now tapered off tizanidine prior to any surgical procedure.

NEUROLYTIC BLOCKS

Neurolytic blocks utilizing phenol, or botulinum toxin can focally reduce spasticity.

Neurolytic blocks are not appropriate for patients who need generalized tone reduction unless injecting a few muscles will result in significant functional improvement.

Neurolytic blocks can be utilized at any age in children, but spasticity usually is not a significant functional problem in children until they are of one year of age. Neurolytic blocks thus can be used in very young or small children who may not yet be appropriate for more aggressive spasticity procedures such as selective dorsal rhizotomy, intrathecal baclofen pump, or orthopaedic surgery. Focal injections into select muscles can help control spasticity and its complications, until more aggressive treatments are appropriate. In some children neurolytic blocks may be used indefinitely if continued functional improvements are seen.

Phenol or carbolic acid can denature protein and cause tissue necrosis in concentrates greater than 5% (Glenn 1990). Phenol blocks have been used for decades to treat spasticity in children with good success (Easton, Ozel et al. 1984). This is a procedure where motor nerves to a spastic muscle are located using needle electrostimulation. Once localized aqueous phenol in 5-7% solution is injected. Because children often tolerate the procedure poorly, many children benefit from having the procedure performed under general anesthesia. This allows greater cooperativeness thus easing some technical difficulties. Phenol blocks are not permanent and a child who had the procedure done while awake may not return to clinic for repeat injections.

The lethal dose of injected phenol is 8.5mg. (Wood 1978). The recommended maximum dose is less than 1 gram for one treatment session (Glenn 1990). Dosing

guidelines have not been well established for children, but doses of less than 30mg. per kg. have been felt to be safe (Morrison, Matthews et al. 1991). The side effects from phenol are similar in children as in adults, with dysesthesias being the most significant problem. The incidence of dysesthesias in children is less than 5% (Glenn 1990), which is less than is reported in adults. Phenol blocks can last 3-12 months (Spira 1971).

Recently botulinum toxin has been used to treat focal spasticity in children. Botulinum toxin blocks the release of acetylcholine at the distal axon, paralyzing muscle thus decreasing tone (Kao, Drachman et al. 1976). Botulinum toxin not only blocks neurotransmission of the alpha motoneurons but also blocks gamma motoneuron transmission to the muscle spindle (Rosales, Arimura et al. 1996). Botulinum toxin thus weakens extrafusal muscle and directly decreases spasticity by decreasing the excitability of the muscle spindle.

Botulinum toxin has been studied in the treatment of children but not yet approved by the FDA (Koman, Mooney et al. 1993; Cosgrove, Corry et al. 1994; Sutherland, Kaufman et al. 1996). Although not approved by the FDA, botulinum toxin is a well accepted treatment in children (Russman, Tilton et al. 1997) and the American Academy of Cerebral Palsy and Developmental Medicine, and the American Academy of Physical Medicine and Rehabilitation are developing practice parameters in the use of botulinum toxin to treat childhood spasticity.

Botulinum toxin just like other neurolytic agents, is a treatment for focal spasticity and not intended to treat generalized spasticity. Only 3-4 major muscle groups can be treated at any one time, and if additional muscle groups need to be injected then other treatment modalities or combination treatment with phenol blocks should be

considered. Botulinum toxin should not be used to treat contracted muscles unless used in conjunction with adjunctive measures. Botulinum toxin will reduce spasticity that can lead to contractures but will not stretch the contracted muscle. Serial casting, splinting, range of motion exercises or other similar measures should be used to reduce the contracture. Botulinum toxin can allow the muscle to be stretched easier and since botulinum toxin's maximum effect is not reached until 7-14 days post injection, one should consider postponing these stretching measures for about one week following an injection. Botulinum toxin also may not be appropriate to treat severe spasticity since, even in large doses, its clinical effect may not be sufficient enough to satisfactorily reduce the tone of a very spastic muscle.

In children botulinum toxin is dosed by body weight. The recommended dose is 10-12 U/kg with a maximum dose of 400 U (Russman, Tilton et al. 1997). Each major muscle group should receive sufficient amount of botulinum toxin to have a good clinical effect. That dose should be at least 2 U/kg in the arms and 3 U/kg in the legs. A maximum dose per major muscle group in the arms is 4 U/kg, and 6 U/kg in the legs should not be surpassed to avoid untoward local and systemic spread. The lethal dose in adult humans extrapolated from primate studies is 2500-3000 units (Schantz and Scott 1981). In juvenile monkey studies, the TD 50 was 33 U/kg and the LD 50 was 39 U/kg (Scott and Suzuki 1988).

Dosing in children is somewhat controversial since theoretically the same number of neuromuscular junctions exist in a child's individual muscle versus an adult muscle. Given this, the same amount of botulinum toxin should be needed regardless of the size

or age of the patient. But, all current studies and dosing guidelines use body weight as the dosing standard and should be adhered to until further information is available.

Given the limitations in dosing and number of muscles groups that can be injected, using phenol blocks in conjunction with botulinum toxin can help manage spasticity in children requiring more generalized treatment (See Table 4). Phenol blocks can be utilized in this scenario to effect nerves comprised of mostly motor neurons and minimal sensory neurons such as the obturator and musculocutaneous nerves. Botulinum toxin injections can then be used to treat muscles innervated by a highly mixed sensorimotor nerve such as the median or posterior tibial nerve. Using this method, small children can be treated with neurolytic blocks as a temporary measure until they are old or large enough to be candidates for more global treatment measures such as a selective dorsal rhizotomy or intrathecal baclofen pump.

Duration of effect can vary. In the lower extremity of an ambulatory patient, duration of effect is generally 4-8 months when used in sufficient doses (Cosgrove, Corry et al. 1994; Sutherland, Kaufman et al. 1996). Duration of effect in the upper extremities is usually shorter, 3-6 months. This difference may be due to the greater ease of stretching shortened lower extremity muscles and maintaining the stretch with splints. Ambulation which leads to a consistent stretch may be another factor that promotes a longer duration of effect in treated lower extremity muscles. Factors that may decrease the duration of effect include severe spasticity, an inadequate dose, the presence of a fixed contracture, or a growth spurt.

NEUROSURGICAL PROCEDURES

Various neurosurgical procedures have been used in attempts to reduce spasticity in children with cerebral palsy (CP). Selective dorsal rhizotomies (SDR) (Peacock and Staudt 1990) and intrathecal baclofen pumps (Albright, Barron et al. 1993) are currently being used with good success. Cerebellar stimulations are no longer used because of poor results and pallidotomies are still in the development stages but show promise (Chambers 1997). All these procedures are intended to treat spasticity in a more global manner.

Children with spasticity from cerebral palsy have lost the cerebral inhibitory influences on the monosynaptic reflex arc at the spinal cord level. This results in hyperreflexia or spasticity. If this hyperactive monosynaptic reflex arc is disrupted then spasticity can be reduced. Neurolytic blocks work at the efferent nerve level, and baclofen and diazepam work at the spinal cord level to disrupt this reflex arc. Neurosurgeons in the past have tried to disrupt the reflex arc by transecting the afferent sensory fibers. Forster (Forster 1908) in the early 1900's transected the sensory fiber at the dorsal root level in patients with spasticity and found muscle tone significantly reduced. Since he carried out a complete rhizotomy these patients had no sensation and had problems with pressure sores and bowel and bladder control. This procedure was abandoned. In the 1970's Fasano (Fasano, Barolat-Romana et al. 1976) developed a technique which selectively cut some of the dorsal rootlets which resulted in spasticity reduction but preservation of sensation. Peacock (Peacock and Arens 1982) in the 1980's further refined the procedure and popularized it in the United States.

Selective dorsal rhizotomy is intended to only reduce spasticity in the legs. In the current procedure, the dorsal roots at the L1-S2 level are identified then divided into rootlets. These rootlets are then stimulated and the resultant motor or reflex response monitored by electromyography of selected lower extremity muscles. If an abnormal response is seen then the rootlet is cut. A response is considered abnormal if a sustained response occurs instead of a single muscle twitch or if a muscle not typically innervated at that root level responds. Typically 25-40% of the dorsal rootlets are transected. The percentage of rootlets cut does not necessarily correlate with the level of spasticity or neurologic involvement. The abnormal response criteria and the need to electrically monitor patients during a selective dorsal rhizotomy is controversial and some clinicians see no value in monitoring patients (Logigian, Wolinsky et al. 1994).

Following a SDR there is a marked decrease in spasticity and patients are weak for several weeks. Their strength does return to its baseline. Following a rhizotomy, because of the marked change in tone, there is an opportunity to change motor patterning. This requires aggressive daily physical therapy for several weeks slowly tapering the intensity of therapy over several months. Many gross motor activities are restricted to in therapy only until patients have mastered their new motor patterns.

Because a child must be able to cooperate with the intensity of therapy a minimal age or maturity level is necessary. Also, because one of the main purposes of carrying out a SDR is to change motor patterns, a child undergoing a SDR must be young when a significant amount of brain plasticity exists. For these reasons the ideal age range for a SDR is 4-8 years although patients up to adolescence can be appropriately treated with a

SDR. Because of the intensity of therapy, the child must have a strong social structure so they can follow through with their rehabilitation program.

Because SDR only affects spasticity, children with dystonia do not respond well to a SDR. (See Table 3.) Children with cerebral palsy often have a mixture of spasticity and dystonia especially patients with quadriplegic CP. If a patient has a significant amount of dystonia and only treating the spasticity changes function little, these patients should not have a SDR. Virtually all patients with spastic hemiplegic CP walk and historically because they are all ambulatory, spastic hemiplegic patients do not have SDR's. Patients who use their spasticity for functional reasons especially to stand and have poor strength underlying their spasticity may become less functional following a SDR. Patients who have had multiple tendon releases can have excess weakness following a SDR and are also not considered good candidates for a SDR.

The functional improvements following a SDR have been debated. McLaughlin, et.al. found no significant improvement in patients who had a SDR with aggressive physical therapy versus patients who received physical therapy alone. McLaughlin used the Gross Motor Function Measure™(GMFM) to assess functional changes. Steinbok, et.al. (Steinbok, Reiner et al. 1997) also using the GMFM showed significantly better improvement in the rhizotomy plus physical therapy patients versus the physical therapy alone group. In our center we looked at 79 patients with CP and measured their oxygen consumption before SDR and one year following an SDR. Oxygen cost diminished by an average of 27% after a SDR (Stout, Gormley et al. 1997). Although we did not have a control group who received physical therapy alone, the oxygen cost improvement in our group far exceeded the expected improvement following physical therapy alone.

In summary, the ideal candidate for a SDR is a child 4-8 years old, born prematurely (these children usually have pure spasticity and their legs are more involved than their arms), ambulatory, cognitively intact, and with a good social structure. (See Table 4.)

Since SDR has been utilized only for about the last ten years, long-term outcomes need to be assessed to better determine the value of a SDR.

Another recently used treatment modality for children with cerebral origin spasticity is the intrathecal baclofen(ITB) pump(Albright, Barron et al. 1993). For several years ITB has been approved to treat spasticity of spinal cord origin, but in June 1996, the ITB pump was also approved to treat spasticity of cerebral origin in children. ITB is intended to treat spasticity of the lower extremities. Baclofen crosses the blood brain barrier poorly, often leading to minimal clinical improvement in spasticity when take orally. However, if delivered intrathecally via a catheter attached to a subcutaneously implanted computerized pump, spasticity can be markedly reduced. An ITB is not a permanent procedures and if the clinical effects do not significantly improve function the pump can be removed without residual effects. The ITB pump does need to be refilled every three months and replaced when the battery loses power, which is after 4-5 years.

Intrathecally delivered baclofen can also reduce dystonia(Albright, Barry et al. 1996). So, children with mixed spasticity and dystonia can have significant tone reduction. This is particularly advantageous for children with quadriplegia who have few other treatment options. Because the pump is computerized, the baclofen dose can be

regulated via radio transmitted signals from a laptop computer, thus optimizing the spasticity reduction.

The complication risk from a SDR is minimal and less than 1%, but the complications from ITB can approach 20% and includes meningitis, cellulitis, and catheter malfunction (Albright, Barry et al. 1996). All of these complications may require hospitalization for intravenous antibiotics or surgical intervention.

In children with predominantly dystonia and little spasticity stereotactic encephalotomy has improved dystonia or dyskinesia, but had little effect on spasticity (Speelman and van Manen 1989). In this procedure a probe is placed within the globus pallidus or ventrolateral thalamic nuclei and electrocoagulated. This can result in the reduction of dyskinetic movements but has little effect on spasticity. Children with dyskinetic CP and not spastic CP may benefit from this procedure, but more studies need to be done to better delineate appropriate target organ, patient selection and efficacy.

In the 1970's Cooper (Cooper, Riklan et al. 1976) and Davis (Davis, Barolat-Romana et al. 1980) reported significant reduction in spasticity among cerebral palsy patients who had an electrostimulator placed on the surface of the cerebellum. A large percentage of these stimulators failed mechanically. A double-blind placebo controlled study of the cerebellar stimulators by Gohm failed to show significant clinical benefit. Other studies showed poor results (Bensman and Szegho 1978; Ivan, Ventureyra et al. 1981) and this procedure has since been abandoned.

ORTHOPAEDIC SURGERY

Orthopaedic surgery has been a mainstay of treatment for children with CP for many years (Chambers 1997). Many of the musculoskeletal deformities seen can only be

corrected by orthopaedic intervention. These include bony deformities such as: coxa valga, femoral anteversion, vertical talus, tibial torsion, hip subluxation and dislocation, and severe contractures. Orthopaedic surgery treats the end organ involved in spasticity, the muscle and tendons, but does not alter the central process of spasticity. By lengthening tendons however the tension on the muscle spindle is less, thus reducing spasticity. This spasticity reduction is only temporary and eventually the spasticity and contractures return. Orthopaedic surgery is an excellent treatment for the majority of children with cerebral palsy to correct contractures refractory to other measures, bony deformities, and temporarily can reduce spasticity.

CONCLUSION

Cerebral origin spasticity in children can interfere with function, cares, positioning, and comfort. Different treatment modalities can reduce the adverse effects of spasticity and these can include bracing, stretching exercises, oral medications, neurolytic blocks, selective dorsal rhizotomies, intrathecal baclofen, and orthopaedic surgery. No single best treatment for spasticity exists. Many factors influence the choice of a treatment modality including a child's age and size, severity of spasticity and functional level. Each child should be assessed individually and the most effective treatment to improve function chosen. Almost all children will benefit from a combination of interventions and not one single intervention. The appropriateness of these interventions may change as a child grows and develops. Specific goals need to be well outlined utilizing a comprehensive team approach. With a good understanding of childhood spasticity and the treatment options available, treatment of the patient can be optimized. (See Table 5.)

CASE STUDIES

Case 1:

A 4 year old girl with spastic diplegic cerebral palsy secondary to prematurity can walk community distances with crutches. She has good function of her arms and is cognitively normal. She has no athetosis but significant spasticity of her legs. She walks with her feet plantarflexed, in a crouched stance, and has co-contractions of her muscles with walking. This child receives no therapy, has not had any surgeries, and does not wear braces. She does have some moderate torsional deformities.

This child could benefit from a number of interventions. Range of motion exercises could help reduce contractures and physical therapy could improve her gross motor skills. Ankle foot orthoses would help stabilize her ankles during gait.

Orthopaedic surgery may help reduce her contractures, temporarily reduce her spasticity, and correct her bony deformities. Given her age however, her contractures, spasticity, and bony deformities are likely to return before she is fully grown if orthopaedic surgery is performed now.

Neurolytic blocks would help reduce her lower extremity spasticity, but since her spasticity involves her plantarflexors, hamstrings, hip adductors, rectus femoris, and hip flexors; it is difficult to treat all of these muscles with neurolytic blocks, especially on a repeat basis potentially over many years. This patient could benefit functionally from focal spasticity treatment to her plantar flexors, hamstrings, or other muscle groups with botulinum toxin or phenol blocks but may benefit more from global spasticity management.

A SDR or ITB would be the most appropriate initial treatment since a significant reduction in spasticity would occur. The patient is at an age where significant brain plasticity remains and developmental patterns could change significantly if spasticity is reduced. Since the patient meets all the ideal criteria for a SDR it may be the most appropriate treatment as it is a one time treatment with a low complication rate and high rate of functional improvement. An ITB would also be appropriate but carries a higher complication risk and needs maintenance every three months. Because of this girl's torsional deformities she may benefit from derotational osteotomies in the future to correct bony alignment.

Case 2:

A 14 year old boy with severe spastic and dystonic quadriplegia secondary to a traumatic brain injury at 4 years of age is non-ambulatory. He has had multiple orthopaedic surgeries in the past but still has significantly increased muscle tone and spasticity. He has a difficult time being positioned in this wheelchair, poorly tolerates his braces, is difficult to care for, and generally uncomfortable. This patient is particularly tight in his hip adductors, hip flexors, hamstrings, and plantarflexors. He has been tried on oral dantrolene, diazepam, and baclofen with minimal success. He regularly receives range of motion exercises, but has still developed contractures.

This patient would be a candidate for more aggressive spasticity treatment measures. He has already had multiple orthopaedic surgical procedures and probably is not a good candidate for repeat orthopaedic procedures. He has some dystonic features to

his abnormal muscle tone and is 14 years of age, both of which make him as a poor candidate for a SDR.

This patient may be a good candidate for neurolytic blocks. He may benefit from botulinum toxin injections into his plantarflexors and medial hamstrings in conjunction with phenol blocks to the obturator nerves. Following these injections he should have aggressive stretching which may include serial casting of his ankles and splinting of his knees. Hopefully, this would improve his positioning, comfort, and care.

Depending on the severity of the spasticity and effect of the neurolytic blocks, this patient may be a good candidate for an ITB pump. ITB would significantly reduce his spasticity in his legs, more so than neurolytic blocks alone. The dose could also be titrated for maximum benefit. With the tone in the legs reduced, if spasticity is a problem in his arms, botulinum toxin and phenol not utilized in the legs could be used to treat the spasticity in the arms.

Case 3

A 2 year old boy with spastic diplegia cerebral palsy secondary to prematurity is non-ambulatory, but can crawl and cruise around furniture. He has significant problems with scissoring of his legs, long sitting, and standing with his feet plantarflexed. Ankle foot orthoses control his ankles but he tolerates them poorly. He is actively involved in a physical therapy program one time per week. He was tried on oral baclofen and diazepam to reduce his tone, but tolerated them poorly because of sedation.

This child is too young and small for a SDR, ITB, and orthopaedic surgery. He has tried many conservative treatments but still has functional mobility problems because

of his spasticity. He would be a good candidate for botulinum toxin injections. Injections into the plantarflexor and medial hamstrings are the muscles most likely to be interfering with function. His scissoring is caused by both his spasticity in his hip adductors and medial hamstrings. Since his hamstrings also cause problems with sitting and crouching while standing, injecting these muscles are more likely to improve his function. Injections into the plantarflexors would probably improve stance stability and brace tolerance. Phenol blocks into the obturator nerves could be considered but in this child would likely require general anesthesia and may want to be avoided initially.

As this child grows and develops he will likely be a candidate for a SDR or ITB and probably orthopaedic surgery. Neurolytic blocks will help reduce complications from spasticity and improve development until he is appropriate for more aggressive treatment measures.

FIGURE # 1

4 year old with hemiparesis following a stroke. The foot is in equinus even after serial casting. The patient could not tolerate AFO's nor stand.

FIGURE # 2

One month following botulinum toxin into the gastrocnemius and repeated serial casting.

The equinus is corrected allowing weight bearing and use of an AFO.

TABLE # 1

TREATMENT OPTIONS FOR CEREBRAL PALSY

	Type of tone problem	Age	Advantage	Disadvantage	Cost
PT/OT	all types	any	improves development	expensive	\$\$\$
Oral Meds	diffuse	> 1 year	works systemically	sedating	\$
Bracing/Casts	all types	any	improves joint position + ROM	doesn't reduce spasticity	\$
Orthopedic Surgery	all types	5 years to adolescent	corrects alignment	temporarily reduces spasticity	\$\$\$
Neurolytic blocks	focal	any	reduces spasticity	temporary	\$\$
SDR	diplegia	4 -8 years	eliminates spasticity	irreversible	\$\$\$\$
ITB	lower extremity	> 34 pounds	adjustable tone reduction	20% adverse effects	\$\$\$\$

TABLE # 2

ORAL MEDICATIONS

Medication	Dose	Side effects
baclofen	20-60 mg/d divided qid	Sedation, withdrawal risks
diazepam	0.2-0.8mg/kg/d divided 6-8h	Sedation
dantrolene	3mg/kg qid, max 400mg/d	Weakness, hepatotoxicity
tizanidine	not determined	sedation, increased liver enzymes

TABLE # 3

PHENOL VS. BOTOX COMPARATIVE

Blocking Agent	Administered	Effectiveness	Advantages	Drawbacks	Complications
Botulinum Type A Toxin	Injected into the muscle	Lasts 12-30 weeks	<ul style="list-style-type: none"> • Easy to administer • Diffuses readily into the muscle • Painless • Can be administered without anesthesia 	<ul style="list-style-type: none"> • Effects are always transient • Lasts only 12-30 weeks • Limited approval 	No significant complications reported
Phenol Block	Injected into the motor points of the involved muscle	Lasts 4-12 months	<ul style="list-style-type: none"> • Use is widely approved • Lasts longer than botulinum toxin • Cumulative effects often occur 	<ul style="list-style-type: none"> • Can be painful • May require general anesthesia during administration • Takes more skill to administer 	<ul style="list-style-type: none"> • Transient dysesthesias and numbness • Hematomas may occur, which negate the effects of the treatment • If a large intravascular injection occurs, Phenol can cause systemic effects such as muscle tremors and convulsions, as well as depressed cardiac activity, blood pressure and respiration

TABLE # 4

DYSTONIA VS. SPASTICITY

	Dystonia	Spasticity
CNS influence	Extrapyramidal	Pyramidal
I _a afferents influence	Little	Significant
Variability	Significant	Little
Clinical characteristics	Through whole ROM Lead pipe resistance	Initial catch Velocity dependent

TABLE # 5

SELECTIVE DORSAL RHIZOTOMY

Indications	Contraindications
cerebral palsy	spinal cord origin spasticity
spasticity	athetosis, rigidity
good selective motor control	previous tendon releases/lengthening
ages 4-8 years	poor trunk control
intelligent	severe weakness
good social structure	

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