

Management of Childhood Spasticity: A Neurosurgical Perspective

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Spasticity is a vexing problem for both healthcare providers and patients. Cerebral palsy is the most common cause of spasticity and physical disability in children. It defines a range of nonprogressive syndromes of posture and motor impairment that results from an insult to the developing central nervous system (CNS) either in utero or within the first 2 years of life.¹ The prevalence of cerebral palsy is not known but is estimated at approximately 2 per 1,000 children, and its incidence may be increasing secondary to improved care in neonatal intensive care units and improved survival of low birth-weight infants.² The common features of cerebral palsy include movement disorders, muscle weakness, ataxia, rigidity, and spasticity.

Spasticity is defined as a velocity-dependent increased resistance to passive muscle stretch, or alternatively as inappropriate involuntary muscle activity associated with upper motor neuron paralysis.^{3,4} It is a hallmark of cerebral palsy but can occur with other genetic and metabolic diseases that cause dysfunction

or damage to the developing CNS. Spasticity is demonstrated in children as increased muscle tone, persistent primitive reflexes, and delay of normal motor skills. Spasticity inhibits effective use of motor control and strength and can lead to progressive musculoskeletal complications such as joint and muscular contractures, bony deformation, and joint subluxation or dislocation.⁵ Proper treatment for spasticity can halt progression of contractures or deformity and often can return function to affected limbs. Both prospective and retrospective studies of children treated for spasticity have demonstrated improved ease of caregiving, decreased pain, and improved quality of life.⁶

Spasticity in children can result from any disease process that affects the upper motor neuron within the CNS. Injury to the upper motor neuron decreases cortical input to the descending reticulospinal and corticospinal tracts, which causes weakness, loss of motor control, and reduction in the number of voluntarily active motor units. The reduction of these descending tracts removes the normal

inhibition of the reflex arcs within the grey matter of the spinal cord, leading to a hyperactive reflex arc and spasticity.

The diagnosis of spasticity requires a complete history and physical examination, with ancillary testing as needed. The history should inquire about possible gestational and perinatal events and motor and cognitive development. The physical examination should focus on motor power, muscle tone, active and passive range of motion of joints, sensation, deep tendon reflexes, station (pelvis and leg alignment while standing), presence of limb deformity, spinal alignment, and extent of movement disorders. Ancillary testing usually includes imaging studies such as cranial ultrasound, computed tomography, or magnetic resonance imaging to evaluate for

evidence of hemorrhage, hydrocephalus, or structural abnormalities of the CNS. Spasticity is most commonly quantified by the Ashworth spasticity scale (Table 1, see page 356).^{7,8}

MANAGEMENT

Treatment of spasticity involves multiple modalities that most commonly include observation, physical and occupational therapy, orthotics, oral medicines, intramuscular injections, and both neurosurgical and orthopedic surgery. A combination of methods is employed most often to increase the beneficial effects of each modality synergistically. A multidisciplinary team, including pediatricians, physical and occupational therapists, neurologists, orthotists, orthopedic surgeons, neurological surgeons, and

other healthcare professionals, is best suited to treat children with spasticity. Therapy should be guided by the clinical scenario and specifically targeted at treating pain, decreasing tone, and reducing muscle contractures, joint deformities, and abnormal motor control.

Spasticity should be addressed at an early age to prevent permanent contractures, joint subluxation, dislocation, and bony deformity. In general, young children generally respond well to physiotherapy, orthotics, intramuscular injections, and neurosurgical procedures. Older children benefit from these therapies as well but also may need orthopedic surgery to address musculoskeletal deformity.

An approach directed by the location and severity of the spasticity is used to identify the appropriate therapeutic in-



tervention. These therapies should be directed toward achieving goals determined in concert with caregivers, and ideally should be monitored by the use of clearly defined outcome measures.⁶

Nonpharmacologic Therapy

Physical and occupational therapy, orthotics, and casting are just a few of the nonpharmacologic and surgical therapies employed to improve joint range of motion, strengthen muscles, inhibit spastic agonist muscles, and assist with motor development. There is a lack of evidence-based information addressing these therapies for the treatment of spasticity, largely because commonly used outcome measures have not been validated or may not be functionally relevant.⁹ However, decades of clinical experience support their use for maximizing the benefit of medical and surgical intervention.

CME EDUCATIONAL OBJECTIVES

1. Recognize the importance of early identification and intervention in an infant or child with spasticity.
2. Discuss the pathophysiology of upper motor neuron lesions and their effects on muscle.
3. Describe the management of a child with spasticity and the creation of a care plan, including pharmacologic and surgical options.

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TABLE 1.

Ashworth Scale of Muscle Tone^{4,7}

Ashworth Scale	Degree of Muscle Tone
1	No increase in tone
2	Slight increase in tone, "catch" when limb is moved
3	Marked increase in tone, passive movements difficult
4	Considerable increase in tone, passive movements difficult
5	Affected part is rigid in flexion or extension

Oral Medications

Oral medicines are used often to reduce spasticity. The most common agents are baclofen, diazepam, dantrolene, and tizanidine. These have different mechanisms of action with the common goal of reducing spasticity. Several medicines are summarized in Table 2 (see page 357). Additional novel agents that are under investigation include cannabinoids, gabapentin, and 4-aminopyridine. Although improvements in clinical measures of spasticity have been noted with several of these medications, few have shown significant functional benefit. Unfortunately, limiting side effects often are reached before a clinically significant effect of the medication can be obtained. Antispasmodic medications are very useful adjuncts to more invasive therapies and physiotherapy.¹⁰

Orthopedic Surgery

A wide variety of surgical options are available for the orthopedic surgeon to treat spasticity and its long-term consequences on the musculoskeletal system.¹ In general, orthopedic procedures have been used to improve the biomechanics of spastic patients. Some common goals of surgery include lengthening of contracted muscles, balancing of joint forces, reduction of joint subluxation, fusion of unstable joints, and diminishment of painful spasticity. The surgical techniques include tenotomy, arthrodesis, osteotomy, and tendon transfer or lengthening. The procedures used are tailored to the clinical

situation and age of the patient as the natural course of specific musculoskeletal abnormalities is factored into the decision-making process.

Neurosurgical Therapy

The role of the neurosurgeon in the treatment of spasticity is essential. A variety of surgical procedures and treatment options have a proven long-term and significant effect on spasticity in appropriately selected patients. These procedures include chemical or surgical neurotomies, botulinum toxin injections, selective dorsal rhizotomy, and chronic intrathecal baclofen therapy.

Selective neurotomy. Selective neurotomy, or surgical and chemical lesioning of peripheral nerves, has been proven to be effective in treating spasticity. This can be accomplished in three ways: through surgical exposure and transection of all or a portion of the nerve; through injection of ethanol or phenol around the nerve; or through blockade of the neuromuscular junction by botulinum toxin injection. Recently, ethanol and phenol injections have largely been set aside, as the favored technique has become botulinum injection of spastic muscles.

The objective of surgical neurotomy is to expose and isolate the nerve branches that supply the spastic muscle. A complete or partial division is then performed depending on both the particular muscle involved and a pre-operative plan designed to balance spasticity

with motor weakness. The technique involves intra-operative neurophysiologic monitoring and active stimulation of the nerves to better target destructive lesioning. Stimulation also may provide some guidance to muscle strength and how it relates to the amount of nerve lesioning that is acceptable before motor function is lost completely.

Percutaneous neurotomy is performed by injecting either alcohol or phenol to sclerose the nerve, which causes partial or complete destruction of the nerve supply to muscles. Localizing the nerve is performed with anatomic landmarks and neurophysiologic testing through the needle. The relative indications for selective denervation are spasticity, joint imbalance secondary to spasticity, and decreased function. Ethanol is used more in children because it is less caustic than phenol to surrounding tissues.

Ethanol and phenol denervation lasts about 3 to 8 months. Potential complications and side effects include pain, permanent muscle fibrosis, and dysesthesias. The obturator nerve is the most commonly targeted nerve. Lesions in the nerve can help stop or prevent progressive subluxation secondary to hip adductor spasticity.

Botulinum toxin injections. Botulinum toxin (BTX) injections have been used increasingly in place of alcohol and phenol for chemodenervation. Although

not currently approved by the Food and Drug Administration for spasticity, BTX has been used clinically since 1988 to treat spasticity associated with cerebral palsy. At least two subtypes are available in the United States, BTX-A (Botox) and BTX-B (Dysport).

The physical examination should focus on motor power, muscle tone, active and passive range of motion of joints, sensation, deep tendon reflexes, station (pelvis and leg alignment while standing), presence of limb deformity, spinal alignment, and extent of movement disorders.

When BTX is injected into spastic muscle tissue, it acts at the neuromuscular junction to inhibit the release of acetylcholine and balance muscle forces across the joint.¹¹ BTX acts locally, so it is not effective in reducing global spasticity. The general indications for BTX are for temporary management of focal spasticity and to evaluate the effects of

denervating a spastic muscle. Although specific timing varies, the effects of BTX generally begin after 1 to 3 days, peak around 21 days, and usually have little effect after 3 to 4 months. The dose of BTX injected varies according to the muscle size and formulation of the toxin, but in general a dose of 2 to 6 U per kilogram of body weight is used.¹² BTX can be injected into spastic muscles with or without the use of electromyography guidance.

A number of randomized clinical trials have demonstrated the efficacy and safety of BTX injections.¹³⁻¹⁶ These trials have demonstrated a significant reduction in spasticity and improved function in both lower and upper extremities. The drug has a very good safety profile and has infrequent side effects, mostly related to an allergic reaction to the medicine.¹⁷

Selective dorsal rhizotomy. Selective dorsal rhizotomy (SDR) derives from late 19th Century procedures for spasticity, during which a complete rhizotomy was performed — the entire nerve root within the spinal canal was transected. These initial attempts effectively eliminated pathologic tone and spasticity but resulted in clinical failure because of complete loss of motor function, pain sensation, and proprioception function. The procedure was abandoned until the 1960s, but a modification of this approach has now been accepted as an effective treatment for spasticity.

TABLE 2.

Oral Medications Used in the Treatment of Spasticity

Medication	Mechanism of Action	Half-Life	Initial Dosage	Maintenance	Side Effects
Baclofen (Lioresal)	GABA _B agonist	3 to 4 hrs	2.5 to 10 mg/day	20 to 90 mg/day (in three doses per day)	Drowsiness, ataxia, confusion
Diazepam (Valium)	Benzodiazepine receptor agonist	3 to 6 hrs	0.1 to 0.2 mg/kg/day	0.1 to 0.8 mg/kg/day (in three doses per day)	Lethargy, tolerance
Dantrolene (Dantrium)	Impedes Ca ²⁺ influx into muscle	3 to 9 hrs	0.5 to 1.0 mg/kg/day	12 mg/kg/day (in four doses per day)	Weakness, diarrhea, rash, liver
Tizanidine (Zanaflex)	Alpha-2 adrenergic agent; inhibits aspartate output	2 to 3 hrs	4 to 8 mg/day	8 to 24 mg/day (in four doses per day)	Sedation, dizziness, hypotension

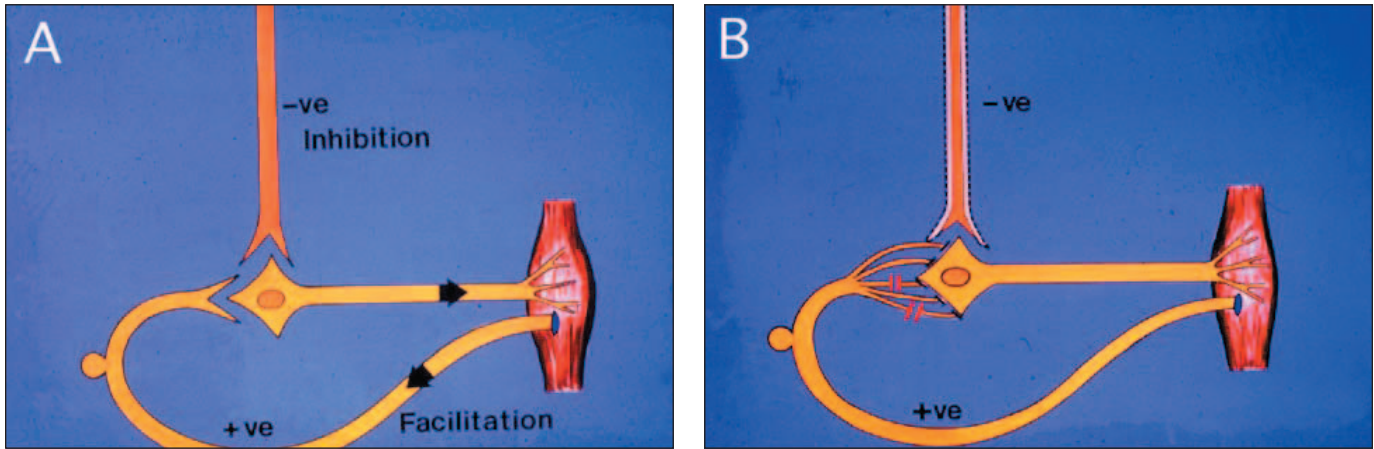


Figure 1. Schematic drawings representing the excitatory and inhibitory influences on the spinal cord alpha motor neuron, which innervates the muscle fibers. (A) Normal physiology with a balance of inhibitory influence from descending neurons and excitatory influence from the sensory spinal reflex arc. (B) In children with spasticity, injury to the upper motor neuron results in a decrease in the descending inhibitory influence, leaving a hyperactive spinal cord reflex arc. By cutting some of the dorsal rootlets, selective dorsal rhizotomy can help restore balance to the alpha motor neuron by reducing the amount of excitatory influence on the alpha motor neuron.

SDR was made possible after the exact anatomical localization of the Ia sensory input to the spinal cord at the dorsal root entry zone (DREZ) by Sindou in 1974.¹⁸ It is believed that these sensory fibers help mediate the abnormal reflex arc in spasticity. In principle, selective lesioning of these fibers could result in loss of tone without loss of other sensory input or motor control at that spinal cord level. This finding was further supported by the discovery of neurophysiological techniques that help differentiate nerve rootlets responsible for spasticity from normal rootlets uninvolved in the disease process. This method, originally described by Fasano, uses electric stimulation of dorsal sensory rootlets with 30 to 50 hertz sustained impulses to activate the hyperactive reflex arc.¹⁹ These stimulated motor responses are thought to occur in the abnormal nerve rootlets because of the loss of descending cortical inhibitory pathways.

The ability of SDR to reduce spasticity can be explained by the current pathophysiological understanding of spasticity. Motor control and tone of the muscle ultimately are controlled by the alpha motor neuron in the spinal cord. Interneurons within the spinal cord grey matter have a regulatory influence

on the activity of the alpha motor neuron. These interneurons generally have an inhibitory effect on the alpha motor neuron and are activated by descending input from cortical upper motor neurons (overall inhibitory influence to the muscle). On the other hand, interneurons are inhibited by the local spinal reflex arc, which are mediated by Ia sensory fibers (overall excitatory influence to the muscle). With damage to the brain or spinal cord, the balance of input is disrupted and the reflex arc becomes hyperactive, leading to increased limb tone and spasticity. By selectively lesioning sensory nerve rootlets, SDR reduces the amount of Ia sensory input and helps restore a more normal balance to the alpha motor neuron (Figure 1).

SDR is used primarily to treat children with lower extremity spasticity, or spastic diplegia. Decades of clinical experience suggest that the patients who ultimately benefit the most from SDR are those with pure spasticity involving the lower extremities, normal intelligence, good strength, no fixed contractures, and postural stability. The ideal patient age is still not known and is probably best determined by the individual clinical scenario. The typical age ranges from 3 to 8, but adolescents benefit as well.

Traditionally, surgery for SDR has involved a 5- to 6-inch skin incision and a five-level laminectomy or laminoplasty. More recently, SDR can be performed using a minimally invasive approach. Surgery now can be done through a small (approximately 1- to 2-inch) incision over the lower back and a single level lumbar laminectomy.²⁰ Intraoperative ultrasound is used to confirm the location just caudal to the conus, and the dura is opened to expose the conus and nerve roots. Individual dorsal nerve rootlets are then tested using electrical stimulation and neurophysiological monitoring. Sensory rootlets that result in spastic responses when stimulated are identified as “abnormal” and transected according to a pre-operative plan based on the patient’s pattern of spasticity (Figure 2, see page 359). The physical therapy team is very helpful in the operating room to manually palpate muscle groups and provide physiological feedback during stimulation.

Recovery from surgery typically takes 2 to 3 days, followed by discharge to home with intensive outpatient rehabilitation or to acute inpatient rehabilitation. Long-term physical and occupational therapy is employed to insure optimal outcomes.

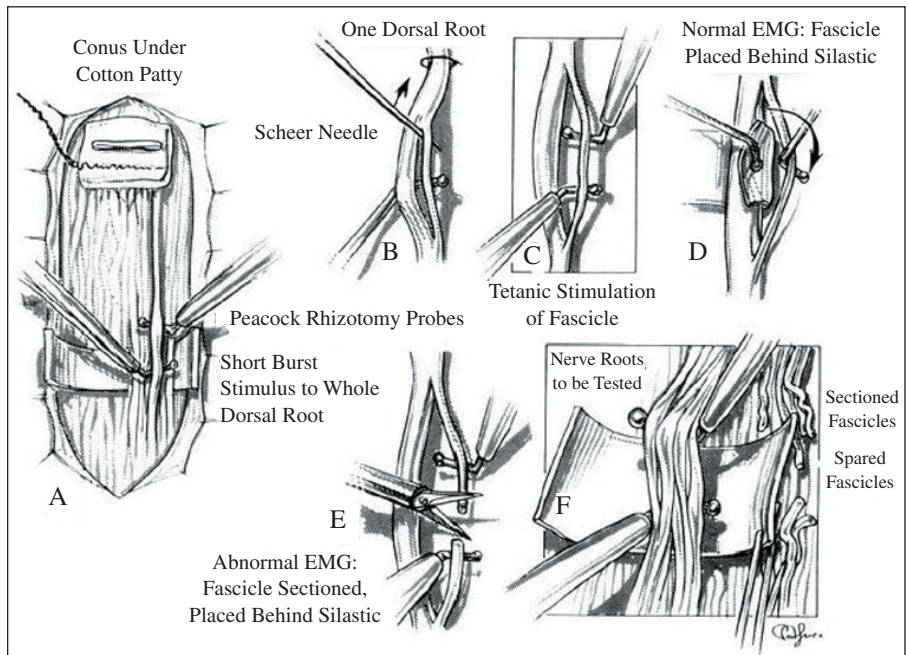


Figure 2. Illustration of steps shows sparing and sectioning the dorsal root fascicles during selective dorsal rhizotomy. (A) An innervation pattern of each dorsal root is examined by electromyographic (EMG) responses to electrical stimulation with a threshold voltage. (B) A dorsal root is subdivided into four to seven smaller rootlets of equal size. (C) The rootlets are tested for EMG responses. (D) Rootlets spared from sectioning are placed behind and covered by the Silastic sheet. (E) Rootlets producing 3+ or 4+ responses are sectioned. Bipolar cautery is seldom used for hemostasis. (F) Dorsal roots remaining over the Silastic sheet are those requiring EMG examination. Spared rootlets are placed behind the Silastic, and sectioned rootlets are left on the side of the Silastic. No dorsal rootlet is left over the Silastic after the EMG testing and the sectioning of the dorsal rootlets are completed.²⁹

There have been a number of excellent long-term outcome studies for SDR. The outcome measures examined include muscle tone, flexibility, gait pattern, functional positioning, and the ability of the child to deal with his or her environment. Nearly all studies investigating SDR have demonstrated a significant and persistent decrease in spasticity without a return of hypertonicity over time. Improved function and ambulation are commonly seen regardless of the pre-operative abilities. Despite the impressive decrease in spasticity after the procedure, some patients still suffer from loss of joint mobility and require subsequent orthopedic surgery for tendon lengthening or transfer.

McLaughlin et al. reported a comparative analysis and meta-analysis of three randomized clinical trials in 2002.^{21,22} Eighty-two children with spastic diplegia received either SDR and physiotherapy

or physiotherapy alone. Outcome measures were used for spasticity (Ashworth scale) and function (Gross Motor Function Measure) and applied at a 12-month follow-up visit. As shown in Figure 3 (see page 360), selective dorsal rhizotomy with physical therapy was more effective than physical therapy alone in reducing spasticity and improving overall

function in children with spastic diplegia. Interestingly, multivariate analysis in the selective dorsal rhizotomy group also revealed a direct relationship between percentage of dorsal root tissue transected and functional improvement. A review of the literature supports the findings summarized in Table 3.

Complications occasionally can be seen following SDR, the most common being pain or transient neurologic dysfunction including weakness, sensory loss, or bladder dysfunction. The majority of these are temporary, with permanent dysfunction occurring in less than 5% of patients. Retrospective studies have drawn attention to the possibility of an increased incidence of scoliosis and hip subluxation after SDR. Because these conditions are common in children with spasticity who do not undergo SDR, it is unclear what role SDR plays in the development of scoliosis or hip subluxation.²³⁻²⁵ It remains to be seen if minimally invasive SDR through a single level laminectomy rather than a multilevel laminectomy reduces the incidence of these conditions.

Baclofen. Baclofen was first used parentally for spasticity in the late 1960s. Baclofen works as an agonist of gamma aminobutyric acid (GABA) at GABA-B receptors within the dorsal horn of the spinal cord. Activation of these receptors is thought to inhibit the excitatory input to the alpha motor neuron. The effective-

TABLE 3.

Summary of Reported Outcomes Following Selective Dorsal Rhizotomy

Class I	Decrease in lower limb spasticity (Ashworth scale); up to 12 years Increase in lower extremity range of motion; up to 5 years Improvement in motor function (Gross Motor Function Measure)
Class II	Improvement in disability (Pediatric Evaluation of Disability Inventory) and activities of daily living performance Improvement in gait including increased stride length and velocity Improvement in suprasegmental effects including upper limb function and cognition
Class III	Reduce the need for future orthopedic procedures

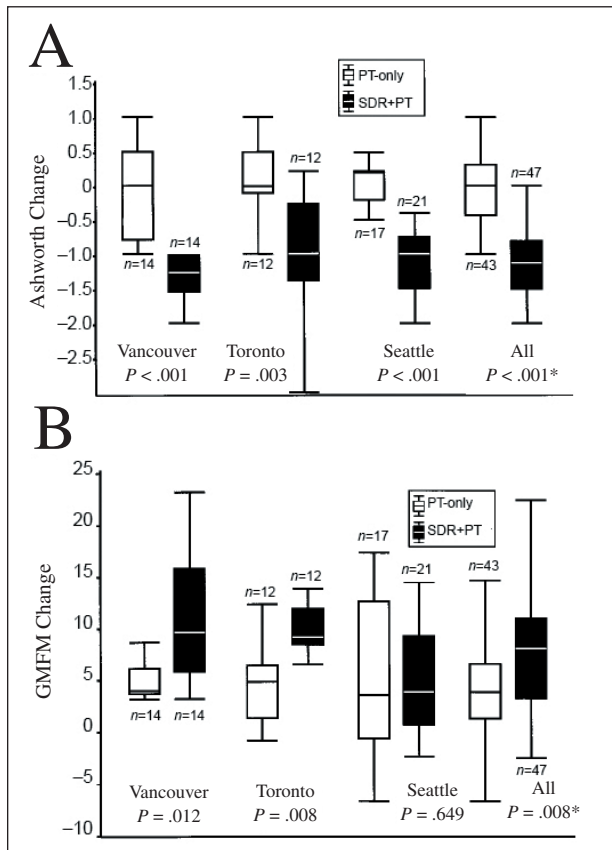


Figure 3. Summary of meta-analysis data after selective dorsal rhizotomy. Ashworth change score (A) and Gross Motor Function Measure (GMFM) change score (B) are shown for those receiving physical therapy only and selective dorsal rhizotomy plus physical therapy group data for individual studies and pooled meta-analysis. Interval is from baseline to 12 months after beginning treatment (9 months for Vancouver). Boxes represent the 25th, 50th, and 75th percentiles. Whiskers represent minimum and maximum values excluding outliers beyond 1.5 times the interquartile range. P values are based on Wilcoxon's tests, with blocking on site for combined tests.²¹

ness of oral baclofen may be limited by dose-related side effects such as sedation, respiratory depression, confusion, and hallucinations. Baclofen penetration into the central nervous system is also limited by the blood-brain barrier.

Impressive results from intrathecal administration of baclofen (ITB) were first demonstrated in adult patients with spasticity from spinal cord injury or multiple sclerosis. A dramatic reduction of tone was achieved with dosages that were many orders of magnitude less than oral or parental doses. Penn et al.²⁶ first reported a trial of 20 patients in 1989 who received baclofen or placebo for three consecutive days followed by

long term therapy with baclofen. All patients experienced decreased muscle tone, and there were minimal complications. The safety and efficacy of chronic intrathecal therapy in adult patients was reported in 1993.²⁷ A total of 93 patients with intractable spasticity due to either spinal cord injury, multiple sclerosis, or other spinal pathology were entered into a randomized, double-blind, placebo-controlled screening protocol of (ITB) test injections, and 75 underwent implantation of a programmable pump system for chronic therapy. Patients were followed for 5 to 41 months after surgery (mean = 19 months). The results of this study indicated that intrathecal baclofen infusion was safe and effective for the long-term treatment of intractable spasticity. The first trial

reported in children was by Albright et al.²⁸ in 1991, which also demonstrated significantly reduced spasticity with an acceptable complication rate.

In the clinical setting, it often is difficult to determine which patients are better suited for SDR and which are better for ITB therapy. Two main groups of children generally have a better outcome if treated with ITB therapy. The first group includes those who have a severe spastic quadriplegia, are functionally debilitated, and are completely dependent for care. These children often respond well to ITB therapy because a global reduction in tone can lead to an improvement in comfort, positioning,

and the daily administration of care.²⁹ The second group includes those children with a spastic diparesis that use their increased tone for ambulation and functional mobility. If an SDR is performed in these children, the concern is that the significant reduction in tone might impair function. ITB therapy can be effective because the intrathecal dosing can be titrated to balance tone reduction with functional improvement.

All potential candidates for ITB therapy are first evaluated with a trial injection of intrathecal baclofen. After the patient receives a thorough baseline evaluation by the spasticity team, a bolus of 50 micrograms of baclofen is given into the spinal fluid through a lumbar puncture. Anti-spastic effects of intrathecal baclofen can be seen within 30 minutes, peak between 2 and 4 hours, and wear off after 6 to 8 hours. Serial examinations are then performed approximately every two hours to determine its efficacy. A reduction by one point on the

The objective of surgical neurotomy is to expose and isolate the nerve branches that supply the spastic muscle. A complete or partial division is then performed.

Ashworth scale is considered a positive response and is seen in approximately 90% of patients. Escalating doses of 75 or 100 micrograms of intrathecal baclofen can be attempted if the first dose is unsuccessful.

Implantation of a pump to administer baclofen continuously can be undertaken after a positive trial. The pump itself, which is roughly the size of a thin hockey puck, is implanted into the anterior abdominal wall in either the subcutaneous

space or in a subfacial location. Flexible tubing runs subcutaneously around the flank to the lumbar spine and into the subarachnoid space. The tip of the intrathecal tubing is positioned under fluoroscopic guidance at a spinal cord level as determined by the pattern of spasticity preoperatively (ie, higher placement of the catheter if severe upper extremity spasticity is present). The pump is programmed via a telemetry magnet on the skin directly over the pump, with the initial setting typically delivering around 50 micrograms per day. The dose can be increased as the child recovers from surgery in the hospital, and then in the outpatient setting until the desired goal is achieved.

The child should be monitored closely after an ITB trial or pump placement, as side effects including sedation, hypotonia, and respiratory depression can result in rare circumstances. Refilling of the pump is required every 2 to 6 months, depending on the dose administered, and is done via a percutaneous injection. The battery in the pump expires after seven years. The minimum weight requirement for a child to have an ITB pump is approximately 10 kg.

Multiple clinical studies have demonstrated that the majority of patients with ITB therapy have a significant decrease in spasticity and some functional improvement.³⁰⁻³³ The multicenter trial that resulted in FDA approval was reported in 2000 by Gilmartin et al.¹⁴ This study assessed the effectiveness of intrathecal baclofen in reducing spasticity in cerebral palsy through an open-label trial of intrathecal baclofen administered through a chronic implanted pump. Candidates were first screened with randomized, double-blind, intrathecal injections of baclofen and placebo. Responders were defined as those who experienced an average reduction of one point in the lower extremities on the Ashworth Scale. Ultimately, 44 patients received chronic therapy and were observed for up to 43

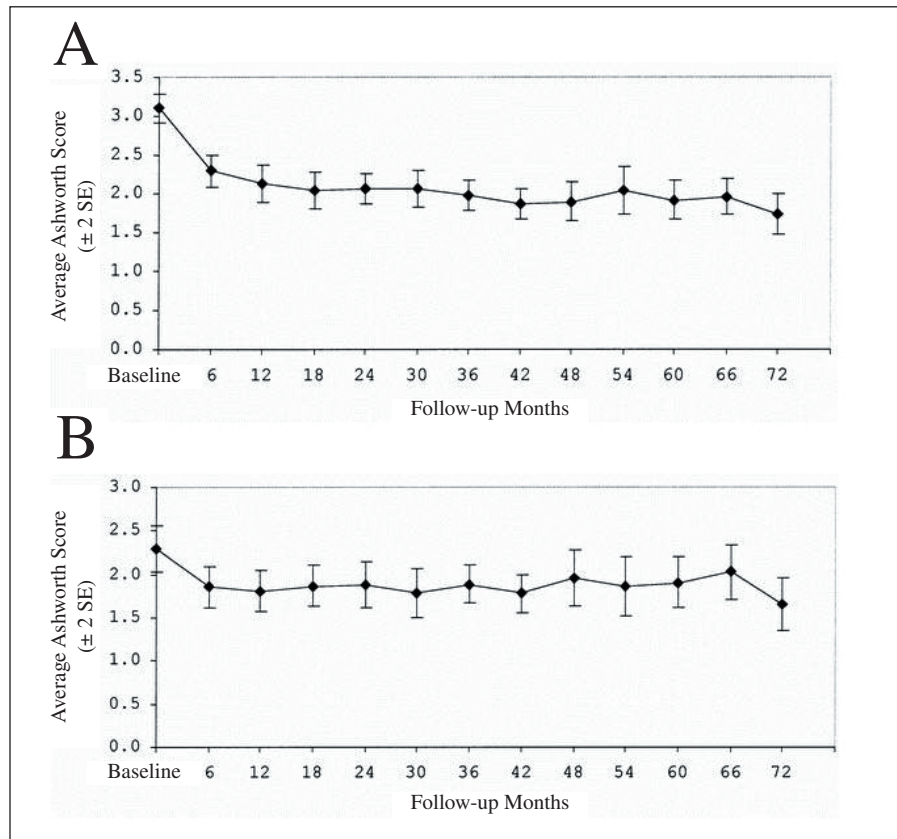


Figure 4. (A) Graph demonstrating the mean Ashworth scores in the lower extremities at 6-month intervals after pump implantation. The scores were significantly decreased at 1 and 2 years post-implantation ($P < .005$). (B) Graph demonstrating the mean Ashworth scores in the upper extremities at 6-month intervals after pump implantation. The scores were decreased significantly at 1 and 2 years post-implantation ($P < .005$). (SE = standard error.)³⁰

months. Lower-extremity and upper-extremity spasticity decreased significantly in all patients. The side effects observed were mostly drug-related and included temporary hypotonia, seizures, somnolence, and nausea or vomiting.

In 2003, Albright³⁰ reported a prospective, multicenter study of 68 patients with chronic intrathecal baclofen therapy who were followed closely for an average of 70 months. The majority (76%) of these patients were younger than 16 and willing to participate in long-term surveillance. Spasticity in both upper and lower extremities decreased significantly and remained decreased throughout the study period (Figure 4). The dosage of baclofen doubled on average during the initial 2 years and then remained stable; there were no significant differences with dosage in children of different ages.

Awaad³¹ also reported on 29 patients with cerebral palsy with short-term follow-up (48 months or less) after intrathecal baclofen therapy. The outcome measures of spasticity as rated by the Ashworth scale and the caregiver assistance scales of the Pediatric Evaluation of Disability Inventory (PEDI) were improved in all patients.

The significant advantage of ITB therapy is the adjustable and nondestructive nature of the therapy. The amount of drug delivery can be adjusted to meet the needs of each specific child. Because no nervous tissue is destroyed, the effect of the therapy is reversible. The disadvantage, however, is that complications arise in some patients. Patients can be overdosed with baclofen with subsequent hypotonia and lethargy, which usually are managed with supportive care and

adjustments of the pump rate. Catheter migration, disconnection or fractures can occur, as well as other surgical problems such as seromas, cerebrospinal fluid leaks, and infections.³⁴

Gooch et al. provided a retrospective analysis of complications seen with ITB therapy in the pediatric population. At 1 year, 24 patients (24%) within a group of 100 patients experienced 48 total complications. The most common complications were catheter disconnection (9%), catheter dislodgement (8%), pump site infection (4%), and cerebrospinal fluid infection (1%).³⁵ Also, if ITB therapy is discontinued abruptly, baclofen withdrawal can occur, leading to rebound spasticity, high fevers, and mental status changes.

SUMMARY

The management of childhood spasticity requires a multidisciplinary effort. With input from pediatricians, physical and occupational therapists, neurologists, orthotists, orthopedic surgeons, neurological surgeons, and other health-care personnel, effective treatment for spasticity can be initiated and maintained that can lead to meaningful improvements in quality of life for vast numbers of children. Neurosurgical treatment of spasticity will continue to evolve and be refined as procedures and techniques are appropriately evaluated with reliable and validated outcome measures.

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