Spasticity in Cerebral Palsy and the Selective Posterior Rhizotomy Procedure

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Spasticity in Cerebral Palsy and the Selective Posterior Rhizotomy Procedure

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Abstract

A review of the selective posterior rhizotomy procedure for reduction of spasticity in cerebral palsy is presented. The history of the procedure, selection of patients, operative technique, and results are described. The neurophysiologic basis for spasticity is considered, as well as the role of spasticity in the complex motor disorder of cerebral palsy. Cerebral palsy is a multifaceted disorder of which spasticity is only one aspect. Reduction of spasticity can be effectively achieved using the current technique of selective posterior rhizotomy, but careful patient selection and establishment of realistic goals are vital to successful outcome. Postoperative physical and occupational therapy are felt to be essential for regaining strength and improving motor function following the rhizotomy procedure. Further study in the areas of spasticity, cerebral palsy, and the effects of rhizotomy is expected to advance our treatment of spastic children. (*J Child Neurol* 1990;5:179–185).

Selective posterior rhizotomy is a neurosurgical procedure designed to reduce spasticity. Although the procedure was first used for this purpose almost 100 years ago,[1] it has recently become repopularized for patients with spastic cerebral palsy, due to refinements in technique.[2,3] The procedure today involves an L2 to L5 laminectomy, followed by the selective division of certain lumbosacral posterior spinal nerve rootlets, based on the electromyographic (EMG) responses to their electrical stimulation. It has been found to be beneficial in carefully selected patients.[3] In particular, spastic diplegic patients with pure spasticity, no underlying weakness or severe contractures, and the ability to walk have shown the most functional improvement from the procedure. Postoperative physical and occupational therapy were felt to be essential for successful surgical outcome.[3,4]

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History

Rhizotomy or "cutting roots" was originally performed on dorsal spinal nerve roots for relief of pain. Robert Abbe described his cases performed in New York as early as 1888 and credited C. L. Dana with the original concept.[5] Abbe reported his own four cases of upper extremity pain or pain plus spastic athetosis and also mentioned a slightly earlier case by Bennett in London who performed lumbosacral dorsal rhizotomy for lower extremity pain.

By 1908, Otfrid Foerster, a German neurosurgeon, had used lumbosacral posterior rhizotomy to relieve spasticity, although Munro is also credited with similar work as early as 1904.[1,5] This followed Sherrington's neurophysiologic studies in the late 1800s describing reduction of hypertonus following division of afferent posterior nerve roots in decerebrate cats.[6]

Foerster described 159 cases in 1913, including 88 cases of "congenital spastic paraplegia."[1] He divided the whole posterior nerve roots from L2 to S2, sparing either L4 or L5 to preserve knee extensor tone. Foerster used electrical stimulation during the procedure to identify the nerve root associated with knee extension and to distinguish between anterior and posterior roots. He emphasized many features of patient selection and treatment that continue to

be important today, such as identification of "real" spasticity and exclusion of cases with athetosis and underlying paralysis. He reported better results in those with lower extremity involvement and emphasized the benefit of motivation and intelligence to participate in a postoperative exercise program. Foerster's procedure fell from favor, presumably because of difficulties with sensory loss, particularly proprioception.[7]

In the 1960s, Gros et al revised the procedure in order to preserve sensation by cutting only a fraction of the posterior nerve rootlets that constitute the posterior root.[8] Gros had some difficulty with incomplete relief of spasticity, as well as weakness, and later adapted the procedure by attempting to spare rootlets innervating functionally useful muscle groups.[7] Following exposure of the posterior roots at the level of the conus, he used electrical stimulation of posterior nerve roots and EMG, working closely with a physiotherapist and neurophysiologist to map out the rootlets innervating "useful" muscles, such as the abdominal muscles, gluteus maximus, quadriceps femoris, and gastrocnemius. These rootlets were spared, while rootlets innervating muscles deemed to have "handicapping" spasticity, such as the adductors and hip flexors, were cut.[7,9]

In the 1970s, Fasano et al discovered that the response to electrical stimulation was variable among the posterior nerve rootlets in patients with spasticity.[2] Some rootlets responded to a train of electrical stimuli with a localized brief muscular contraction, while others showed a continuous or prolonged response that diffused to other muscle groups, including the upper limb and trunk musculature. The rootlets associated with the brief localized responses were spared, and the others were cut. This procedure was found to relieve spasticity without significant recurrences or sensory disturbances. It was found to be most useful in spastic cerebral palsy, and improvement in function frequently accompanied reduction in spasticity. Children without dystonia who had good underlying strength were identified as appropriate surgical candidates. Cooperation between the orthopedic surgeon and the neurosurgeon was emphasized. Laitinen et al applied the approach of Fasano et al to a small group of adults with spinal cord injury and multiple sclerosis, with positive results.[10]

Peacock et al adapted the procedure of Fasano et al to allow for positive identification of the exact neuroanatomical level.[3] By working at the level of the cauda equina rather than the conus, the lower sacral nerve roots that supply the bladder and bowel sphincters could be positively identified and spared. Thus, an L2-L5 laminectomy was used, and posterior nerve rootlets between L2 and S2 were each electrically stimulated and selectively divided, based on the EMG and visually observed muscular responses.

We now use 10 channels of EMG to simultaneously monitor activity in the hip adductors, quadriceps femoris, anterior tibialis, hamstrings, and gastrocnemius muscles bilaterally. The clinical picture of the child is taken into account in cases where the EMG responses are equivocal. Follow-up for as long as 7 years has shown maintenance of reduction in muscle tone and functional improvements in patients with spastic cerebral palsy.[4] Careful patient selection and postoperative therapy are felt to be vital to successful surgical outcome.[3]

Cerebral Palsy

Cerebral palsy is a motor disorder resulting from damage to the immature nervous system. Spastic cerebral palsy is the most frequent type.[11,12] This is often associated with the sequelae of premature birth. The dyskinetic types of cerebral palsy have become less common presumably due to the reduced incidence of kernicterus from successful treatment of neonatal jaundice.[13] Despite the marked reduction in the incidence of dyskinesia, the overall incidence of cerebral palsy has remained at about 2 per 1000 live births.[14] This is probably due to improved survival of low-birth-weight infants who sustain hypoxic brain injury or the damaging effects of intraventricular hemorrhage.

Cerebral palsy is a multifaceted disorder with a range of types and severity. Individuals may present with one or more tonal abnormalities, including spasticity, rigidity, dystonia, and hypotonia. Persistent primitive reflexes and impairments of balance, strength, selective motor control, and coordination are usually present in varying degrees. Secondary joint contractures and deformities, including hip dislocation and scoliosis, often occur as a result of abnormal tone and postures. Disorders of vision, speech, hearing, sensory processing, cognition, and learning may be associated with cerebral palsy. These children may fail to thrive and may have seizure disorders, hydrocephalus, or other medical problems.

The selective posterior rhizotomy procedure is aimed at improving function or care in children who have spasticity as their primary handicapping factor, although it must be realized that various other fea-

tures of the disorder will continue to be present and interfere with function.

Spasticity

Spasticity has been defined as a velocity-dependent increase in resistance to passive stretch associated with hyperactivity of deep-tendon reflexes.[15] It is a consequence of an upper motor neuron lesion at the spinal or cerebral level, along with other features such as weakness and loss of motor control and dexterity.

The neurophysiologic mechanism behind spasticity has long been debated. Among the theories have been fusimotor hyperactivity and deficient inhibition of several types, including recurrent (Renshaw) inhibition, presynaptic inhibition, reciprocal Ia inhibition, Golgi tendon organ inhibition, and group II afferent inhibition.[16,17] Although the hyperexcitability of the gamma drive was once a popular theory, it is no longer felt to be a tenable explanation.[16] The loss of inhibition to the motor neuron pool at the spinal segmental level is an accepted view; however, the type of inhibition that is lacking is not clearly defined and may vary in different patient populations.

The Renshaw cells are inhibitory interneurons in the spinal cord that are activated by recurrent collaterals of the axon of the alpha motor neuron. They inhibit the motor neuron pool from which they originally received stimulation and tend to reduce inhibition of antagonistic motor neuron pools. Katz and Pierrot-Deseilligny studied recurrent inhibition in spastic patients and found evidence for increased recurrent inhibition at rest, although Renshaw cell activity was reduced or absent in spastic adults during activity.[18]

Decreased presynaptic inhibition of Ia afferents has also been suggested as a possible contributing mechanism in spasticity. Presynaptic inhibition of the Ia terminals in the spinal cord is mediated by γ-aminobutyric acid (GABA).[17] Morin et al have described a method to study presynaptic inhibition in humans that may lead to further knowledge about its role in spastic patients.[19]

Reciprocal Ia inhibition refers to the disynaptic inhibition of antagonistic muscles via the Ia fiber of the agonist and the Ia inhibitory interneuron. Tanaka et al studied this mechanism in adult patients with spastic hemiplegia, revealing that reciprocal inhibition from triceps surae to pretibial muscles was intact, but the reverse was absent.[20] Thus, the increased extensor tone and imbalance of motor power between these muscle groups in spastic patients is exaggerated further by intact inhibition of flexors and lack of reciprocal inhibition to extensors.

The role of other afferents, such as the Ib Golgi tendon organ fibers and type II fibers, in spasticity is not known.[16] In addition to the disinhibition or release phenomenon associated with spasticity, it has also been proposed that sprouting of Ia afferents occurs in response to damage to descending motor tracts, thus increasing the hyperexcitability of the motor neuron pools.[21]

Although both adult-onset injuries and cerebral palsy may result in spasticity, there is evidence suggesting that the neurophysiologic mechanisms and resultant functional disorders are different in individuals with cerebral palsy in whom the insult occurred at an early stage of nervous system development.

Myklebust et al studied the EMG recordings in response to ankle rotation in individuals with spastic cerebral palsy, patients with adult-onset spasticity, and nonspastic subjects.[22] On rapid dorsiflexion or stretching of the triceps surae, they found a pattern of "reciprocal excitation" or cocontraction in the patients with cerebral palsy, whereas the nonspastic subjects and adult-onset spastic patients had a response consistent with reciprocal inhibition. This led to the conclusion that the disorder in cerebral palsy is a developmental one that affects spinal cord circuitry in addition to brain function.

Further work by Myklebust et al revealed that tendon jerk reflexes in normal neonates differed from those of adults, with simultaneous activity of antagonistic muscles as a characteristic feature.[23] Thus, it was hypothesized that the reciprocal excitation in response to quick stretch seen in the child with cerebral palsy may be a persistence of an early pattern or reflect lack of development of the mature pattern of reciprocal inhibition.

Several authors have attempted to examine the relationship between spasticity and the functional motor disorder in individuals with upper motor neuron lesions.[24–28] Sahrmann and Norton examined the upper extremities of individuals with adult-onset upper motor neuron lesions and found that impairment of movement was primarily related to poor agonist control, rather than limitation of movement due to antagonistic stretch reflexes.[24] Lee et al similarly concluded that stretch reflex enhancement was not responsible for stiffness of voluntarily activated elbow muscles of adult spastic hemiplegic patients.[25] Although impairment of voluntary motor power and

control is certainly a feature of cerebral palsy, it is doubtful that these findings in adult upper extremities can be applied to lower extremity spasticity in children with cerebral palsy.

Knutsson and Martensson used isokinetic dynamometry and EMG to look at spastic restraint during active and passive lower extremity movements in adults with spastic paresis.[26] Antagonistic restraint was most commonly found during active motion at the fastest speeds. Holt demonstrated the complex nature of neuromuscular function in patients with cerebral palsy using EMG.[27] He noted phenomena such as cocontraction in response to stretch, and variation between responses elicited during clinical examination and those seen during functional movement, such as gait.

Nashner et al studied posture and equilibrium in children with spastic cerebral palsy, using a movable platform to perturb their balance under various conditions of visual feedback.[28] Deficits in muscle coordination were primarily seen in three spastic hemiplegic children, while problems related to sensory organization were found in three ataxic children. Three children with spastic diplegia had various combinations of abnormal muscular coordination and/or sensory organization. The sequence of muscle firing in spastic limbs was in a proximal to distal direction, which was reversed from the normal response. Cocontraction of antagonistic muscles was noted for spastic children and in young normal children in response to backward sway.[28,29]

Dietz investigated spinal reflexes during normal and impaired gait of children and adults, revealing that the presence of Ia mediated monosynaptic stretch reflexes and the absence of polysynaptic reflexes is characteristic of very young children and of older children with cerebral palsy.[30] The reverse is true for unaffected children over 5 years of age and adults. Cocontraction of antagonistic muscle groups during the stance phase of gait and reduced amplitude of EMG were found in both spastic children and those with Duchenne muscular dystrophy. The coactivation of muscles during stance phase of gait was seen as a persistence of an immature pattern in individuals with motor dysfunction of early onset.

Perry has attributed gait disorders in patients with upper motor neuron lesions, including cerebral palsy, to "spasticity, primitive locomotor patterns, impaired selective control and contracture formation."[31] It is clear that spasticity alone is not the causative factor in motor dysfunction in cerebral palsy but persistent cocontraction or antagonistic muscle activity appears to be a dominant feature. Successful outcome in surgical reduction of spasticity is therefore dependent on the degree to which spasticity interferes with care and function, as well as the other features of the motor disability that remain, such as weakness, synergy patterns, abnormal timing of muscle activity, lack of balance, and contractures.

Patient Selection

Selection of patients for rhizotomy is performed by a team including the pediatric neurosurgeon, physical therapist, and orthopedic surgeon. Neurologists, occupational therapists, physiatrists, nurses, psychologists, and social workers also participate in the selection process and refer patients for surgical consideration. The patient's history is taken, and the diagnosis of cerebral palsy is confirmed. We have noted that children with a history of premature birth seem more likely to be purely spastic, rather than having a mixed tonal picture. Veelken et al found that spastic diplegic children who had a preterm birth had fewer associated handicaps than those who were born at term.[32] Neurologic examination for evidence of spasticity, including velocity-dependent increase in resistance to passive movement, hyperactive stretch reflexes, limitation in range of motion, and frequently, clonus, is performed. Other forms of abnormal tone and involuntary movement, such as athetosis, ataxia, dystonia, and rigidity, must be ruled out.

The goal of surgery is either to improve current functional performance or to ease the daily care and handling of the child. It is also expected that reduction of spasticity will help to prevent the continued formation of contractures and deformities. If functional improvement is anticipated, the child must have adequate strength, motor control, balance, and freedom from fixed contractures to achieve this in the absence of spasticity. If weakness is a factor, upright motor function will be at risk.

Children with spastic diplegia who can walk independently without a device frequently have the strength, motor control, and balance to benefit maximally from the procedure. Children with hemiplegia are not usually considered, due to their excellent functional status and the tendency toward weakness and hypoplasia on their involved side. Quadriplegic patients who are nonambulatory may be considered for the procedure if spasticity is interfering with positioning, sitting ability, or care. Children with moderate to severe involvement with some functional

ability must be carefully evaluated to consider the risks and benefits of surgical reduction of spasticity. For example, a child who can walk for short distances with a walker may depend on spasticity for antigravity support, although his or her freedom of movement for sitting, dressing, and other activities may be improved through rhizotomy. The goals of the procedure should be clearly defined based on the child's current motor ability.

Operative Technique

The surgical procedure is performed under general endotracheal anesthesia without long-acting muscle relaxants. The patient is placed prone on the operating table with bolsters under the chest and pelvis to allow the abdominal wall to move freely. This prevents epidural vein distension and reduces bleeding. The lumbosacral spinous processes are mapped out, using the posterior superior iliac spines as reference, to the level of the L4 spine. A midline laminectomy or laminotomy is performed from L2 to L5. If a laminectomy is performed, a subperiosteal dissection is used to allow for continued bone formation from intact periosteum after closure. In cases where a laminotomy is used, the bone is sutured back in place.

After exposure of the cauda equina, the nerve root levels are confirmed using electrical stimulation of the anterior roots of S1 (the largest root) and S2 and observing the muscular responses. The posterior roots can be distinguished from anterior roots by position, size, shape, color, and threshold for muscular response on electrical stimulation. The posterior root is significantly larger and flatter than the anterior root, which is rounder and has a slightly darker color. The threshold for the anterior roots is significantly lower than that of the posterior roots.

The posterior nerve root of L2 is identified and electrically stimulated using specially insulated microneurosurgical blunt hook electrodes (Aesculap Surgical Instruments, Burlingame, CA) to find a threshold voltage. Individual nerve rootlets constituting that root are individually stimulated with single pulses to find the threshold and then with trains of stimuli at a frequency of 50 Hz for 1 s. The rootlet is either divided or spared, depending on the muscular response. Characteristic EMG patterns that are associated with spared rootlets include decremental and squared type responses. Rootlets associated with incremental, clonic, multiphasic, or sustained responses are generally divided. Responses that spread proximally, distally, or contralaterally are also considered to be abnormal. In equivocal cases, visual inspection and/or palpation of the muscular contraction are helpful. The pattern of rootlets previously divided and the child's clinical picture are also taken into account. The use of muscle relaxants will disturb the monitoring procedure and the level of anesthesia may need to be adjusted if EMG responses are generally excessive or hypoactive.

During closure, the dura is filled with saline, and a Valsalva maneuver is used to ensure watertightness. Following the procedure the patient is taken to the pediatric intensive care unit for 1 to 2 days, where medication, regular turning, and chest care are routinely given. Patients remain flat in bed for 5 days to prevent any potential dural leak. Physical therapy begins on the third postoperative day for bed mobility, range of motion, and strengthening. After the fifth day, sitting, transfers, and early weight bearing may begin. Children are discharged from the hospital on the eighth day and resume outpatient physical therapy on an intensive basis to improve strength and functional motor skill.

Results

Reduction of spasticity and improvement in function following rhizotomy have been noted by several authors over the years.[1–3] The original series of 60 patients reported by Peacock et al showed improvement in functional skills, such as sitting, standing, crawling, walking, upper extremity use, and speech.[3] Maximal benefit was seen in intelligent, spastic, diplegic children with good trunk control (the ability to side sit), no significant weakness, and the ability to locomote. As no significant postoperative improvement was found in four children with spastic athetosis, the procedure is no longer used for patients with this diagnosis.

Long term follow-up for up to 7 years on 55 of these patients has revealed that reduction of spasticity and functional improvement have been maintained.[4] Weakness continued to be present in 46 children, most notably in trunk flexors and extensors, hip extensors and abductors, and calf muscles. Several children required orthopedic procedures to address contractures and deformities that were not affected by rhizotomy, although some children who appeared to have contractures were able to improve their range of motion without orthopedic surgery. Two-dimensional gait analysis of 14 of these children revealed significant improvements in range of motion and stride length after rhizotomy.[33]

Recent studies using instrumented gait analysis of patients undergoing rhizotomy have shown similar improvements in range of motion, stride length, and speed of walking.[34] In addition, EMG analysis during gait has revealed the persistence of primitive locomotor synergy patterns following rhizotomy. Evaluation of foot-floor contact patterns revealed a transition from primarily forefoot only at initial contact to a heel or flatfoot position.[35] Excessive knee flexion related to calf weakness was noted in some patients, for which orthotic management was recommended.

Neurophysiologic studies of individuals with cerebral palsy undergoing selective posterior rhizotomy have confirmed reduction of spasticity, using the H reflex.[36] Evaluation of somatosensory-evoked potentials has revealed spinal cord abnormalities that were improved in some patients following rhizotomy.[36] Comparison of growth curves before and after rhizotomy have been performed, revealing improvement in 51% of 39 patients who had preoperative weight below or equal to the 5th percentile for their ages.[37] Twenty-five percent of these children improved to greater than the 5th percentile for their ages. The authors speculated that spasticity may diminish growth by increasing the energy requirements of these children.

Continued investigation of various aspects of cerebral palsy, spasticity, and the outcomes of the selective posterior rhizotomy procedure are expected to improve our ability to manage this disorder. Treatment of spastic children has long been focused on the need to reduce muscle tone in order to improve motor function. With the selective posterior rhizotomy procedure, we can now direct attention to the remaining motor disorders associated with cerebral palsy and develop programs to more effectively evaluate and treat weakness, improper timing and coordination of muscle action, inadequate balance, and other motor problems. Further exploration of the neurophysiologic dysfunction in cerebral palsy should lead to new approaches for the comprehensive management of these children, including further refinement and development of surgical techniques, optimal use of modalities such as electrical stimulation and EMG biofeedback, and development of new orthotic and technologic aides. The selective posterior rhizotomy procedure has given us a new management tool for selected children with cerebral palsy and has provided the opportunity to more directly evaluate the effects of spasticity in children with cerebral palsy.

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Vignette

A Psycic Analysis of the American Neurological Association by Milt Gross

This is a series of 16 cartoons depicting prominent early members of the American Neurological Association. The original is in the archives of the American Neurological Association, housed in the Coy C. Carpenter Library of the Bowman Gray School of Medicine of Wake Forest University, and is reproduced with the kind assistance of Ms Sarah-Patsy Knight, Archivist, and Dr James F. Toole, Historian of the American Neurological Association.