The results from selective dorsal rhizotomy research suggest that therapists need to question some common clinical assumptions about movement dysfunction. The rationale for performing a selective dorsal rhizotomy is based on the clinical assumptions that spasticity is the underlying cause of disordered movement and that reducing or eliminating the spasticity will improve movement. This article reviews the literature related to movement dysfunction, the effects of selective dorsal rhizotomy, and the evidence for disordered motor control in children with spastic cerebral palsy. Selective dorsal rhizotomy appears to reduce spasticity and increase joint range of motion. Abnormal movement patterns, however, persist after the spasticity is reduced. Well-coordinated movement patterns are acquired slowly and appear to be related to an intense period of physical therapy. I argue that these results provide evidence that the presence of spasticity alone is an insufficient explanation for abnormal movement patterns. I propose that physical therapists redirect their efforts from developing methods for reducing spasticity to developing adequate assessment, treatment, and measurement techniques for assessing motor control in children with cerebral palsy. I believe we can maximize the functional potential of children with cerebral palsy by identifying problems related to motor control and applying sound principles of motor learning to treatment. [Giuliani CA. Dorsal rhizotomy for children with cerebral palsy: support for concepts of motor control. Phys Ther. 1991;71:248–259.]

Key Words: Cerebral palsy; Dorsal rhizotomy; Motor control; Pediatrics, treatment.

Improving movement function in children with cerebral palsy is a major goal of pediatric physical therapy. Physical therapists recently have witnessed the popularity of selective dorsal rhizotomy as a surgical approach for improving the care and function of children with spastic cerebral palsy. Several authors have reported improved function after dorsal rhizotomy; however, many of these studies were descriptive and presented anecdotal evidence for improvement. The results of these studies suggest, however, that selective dorsal rhizotomy does decrease spasticity and improve movement ability when augmented with an intensive physical therapy program after surgery. Reduced spasticity immediately after surgery is consistent among the reports published. Descriptions of continued abnormal movement patterns, however, suggest that we need to question some common clinical assumptions about the causes of movement dysfunction and the treatment of children with spastic cerebral palsy. The screening criteria for surgical candidates, the effects of surgery, and the program of physical therapy proposed before and after surgery provide some insights into the motor control problems of children with spastic cerebral palsy. The problems identified suggest applying concepts of motor control and learning for treatment. In this article, I will review the literature related to movement dysfunction, and I will discuss the effects of selective dorsal rhizotomy on movement ability in children with spastic cerebral palsy and the

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application of motor control concepts for improving movement in patients with spastic cerebral palsy.

**Movement Dysfunction in Cerebral Palsy**

Cerebral palsy encompasses a combination of motor disorders, sensory defects, and mental impairments varying from mild to severe and is usually caused by injury to the central nervous system (CNS) in the prenatal or perinatal period. The motor problems observed in children with cerebral palsy have been classified as dystonic, athetoid, ataxic, and spastic. Children with spastic cerebral palsy often have hypertonus, hyperreflexia, abnormal movement patterns, decreased movement speed, and poor coordination. Specific movement dysfunctions, however, may be expressed differently in each child, creating disparate clinical profiles among children.

The child with cerebral palsy often activates muscles in abnormal sequences and is frequently unable to produce compensatory postural movements. This lack of muscle control may result in abnormal muscle patterns when several muscle groups contract simultaneously and when co-contraction of the agonist and antagonist occur. These difficulties controlling muscle activation patterns are often attributed to problems of hypertonia and spasticity. When clinicians describe motor problems in children with cerebral palsy they frequently use the terms "tone" and "spasticity" interchangeably. At the very least, these terms are confusing, definitions often are vague, and the method of measurement does not always relate to the definition.

Landau defines tone as a state of muscle activation. Fasano et al suggest tone in terms of movement patterns. According to Peacock and Staudt, spasticity is characterized by an increased resistance to passive movement, hyperreflexia, and involuntary spasms of muscle contraction. Gans and Glenn define spasticity as a syndrome associated with increased involuntary muscle reflex activity in response to stretch. According to these authors, the clinical phenomena observed in spasticity are hypertonia, hyperreflexia, clonus, and spread of the reflex response to other muscles. They describe tone as the passive resistance to stretch and a subjective report of how the limb feels when being manipulated. Gans and Glenn emphasize the importance of defining these terms for discussing movement dysfunction. The definitions of these symptoms are important not only for discussing patient movement, but because each may have a different physiologic mechanism. In this article, I will use the definitions of spasticity and tone as described by Gans and Glenn.

**The Spasticity Problem**

Clinicians propose that spasticity prevents normal sensory experiences and contributes to abnormal movement patterns. Peacock and Staudt believe that, as a result of the spasticity, patients often develop abnormal posture and gait, characterized by reflex spasms in extensor, flexor, and adductor muscles that create toe-walking and scissoring. At times, clinicians find spasticity useful and may use extensor spasticity to increase function when considerable weakness is present. For example, extensor spasticity may produce stiffness in the trunk and about the joints of lower limbs that improves weight-supporting ability so that patients can stand. Although patients may depend on spasticity for some functional tasks, the relation of spasticity to function is unclear.

Spasticity continues to be identified as the primary factor associated with movement dysfunction in patients with neurological disorders. Fasano et al suggest that normal muscle tone is maintained by the balance of inhibition provided by descending motor tracts and facilitation produced by the afferent stimulation carried in the posterior roots, which act on motoneurons in the spinal cord. A common belief in cerebral palsy is that descending tracts are damaged, thereby reducing central inhibition to the motoneurons. An imbalance is created in the stimulation and inhibition to the motoneurons, which results in increased muscle tone. Proponents of this hypothesis believe that increased muscle tone produces an antagonist co-contraction. This co-contraction creates resistance to passive movement and limits active movement, resulting in abnormal movement patterns.

Physical therapy has been directed at inhibiting spasticity, with the expectation that this inhibition would allow more normal sensorimotor experiences, and, consequently, that normal movement patterns would emerge. Once spasticity is reduced, improved movement could occur by facilitating normal movement patterns, which produce relatively normal sensory feedback. Clinical observations suggest, however, that without continual inhibition of spasticity, the normal movement patterns acquired in therapy sessions often are temporary and seldom carry over to functional tasks.

**Spasticity Related to Function**

Campbell suggests that pathological, neuroanatomical, and musculoskeletal factors all contribute to the problem of movement dysfunction. Motor problems in children with cerebral palsy cannot be explained simply by the pathologic symptoms of spasticity and tone. Decreased strength (weakness), range of motion (ROM), posture control, and coordination are characteristic of children with cerebral palsy. I believe these deficits are important and should be addressed in assessing and treating children with cerebral palsy, instead of spasticity and tone. According to Young and Wiegner, "Spasticity is often a dramatic symptom, but even if there were a treatment to eliminate spasticity . . . one would not expect it to restore function in most patients." In a recent editorial, Landau and Hunt discuss the murky waters of the relationship between spasticity and function. They present compelling evidence that, even when spasticity is reduced, there is no evidence of improved function.
I believe we would have better success at improving movement by applying concepts of motor control for identifying movement problems. Therapists should emphasize exercise training principles to improve strength, ROM, posture, and coordination, instead of emphasizing reduced spasticity and tone. What evidence do we have that children with cerebral palsy have reduced strength, ROM, posture, and coordination, and what evidence do we have that these variables can be changed?

**Weakness.** Strength is the capacity of a muscle to produce and grade tension appropriately for maintaining posture and producing coordinated movement. There is a large amount of evidence for neuromuscular changes contributing to weakness in adult patients with spastic hemiparesis secondary to cerebrovascular accident (see article by Bourbonnais and Vanden Noven for a recent review). Less information has been reported about changes in children with spastic cerebral palsy. The information that is available does reveal neuromuscular changes similar to those of adults with spasticity; however, comparisons of adult onset of hemiparesis with that of cerebral-palsied children should be interpreted with caution.

Weakness of the agonist muscle in children with spasticity has been attributed to spasticity or hyperactivity of the antagonist muscle. This hypothesis suggests that the constant or prolonged activity of the antagonist inhibits the agonist. This prolonged inhibition prohibits the agonist from increasing strength, producing a type of disuse syndrome. Disuse alone does not account for the changes observed in muscles of patients with spasticity.

Castle et al. and Milner-Brown and Penn reported atrophy and myopathy of type I and II muscle fibers in children with cerebral palsy. Although there appears to be a predominance of type I fibers affected, there are reports of a reduced number and size of both types of fibers in muscles of children with cerebral palsy. In addition to these morphological changes in spastic muscles, Berger et al. found that electromyographic (EMG) values were reduced for children with cerebral palsy. The reduced number, atrophy, and reduced activity level of muscle fibers support the concept of muscle weakness in children with cerebral palsy.

In patients with adult onset of hemiplegia, there are multiple reports of muscle atrophy, reduced number of motor units (MUs), a selective loss of fast-twitch fibers, decreased firing rate of available MUs, and impaired MU recruitment. Scelsi et al. investigated the tibialis anterior (TA) muscle in 16 patients with hemiplegia and reported selective atrophy of fast-twitch fibers 3 to 17 months post-stroke. McComas et al. studied 46 patients with lesions of cerebrovascular origin. These authors reported muscle atrophy, decreased timing, and that half the number of MUs in the extensor digitorum brevis muscles were functioning between 2 to 6 months after a stroke. Twenty months after stroke, the surviving MUs tended to have slow contraction times and appeared to increase in size.

Deficits in MU recruitment are related to impaired force production and abnormal firing patterns in patients with hemiplegia. Rosenfalck and Andreasson examined the TA muscles in 10 patients with spasticity from various CNS lesions and reported changes in the firing pattern and recruitment order of single MUs. These patients had difficulty maintaining a constant firing rate and force output. Tang and Rymer compared elbow flexors on the normal and affected side in patients with hemiplegia and noted abnormal MU activity and force production of the muscles in the affected limbs. Comparing force-EMG curves for both limbs, the authors suggested that an increased number of MUs were recruited to meet force requirements in the affected limb. Lehmann studied length-tension curves in patients with diseases of the CNS and reported decreased maximal contractions and a reduced ability to produce adequate force.

These reports provide evidence of muscle pathology in children with spastic cerebral palsy and adults with hemiplegia. This evidence supports the concept that their muscles are weak and they are probably using as much muscle activity as they have available. The relationship of decreased muscle force to abnormal movement patterns has not been elucidated. It is easy to understand, however, how changes in muscle force could disturb the balance required within and among joints to produce smooth, coordinated movement.

**Passive restraint.** Children with cerebral palsy often have limited joint ROM. Berger et al. reported that EMG amplitudes during gait in children with cerebral palsy were lower than in healthy children. When EMG values during gait were compared with maximum voluntary contraction values, however, the TA muscle was more active in the children with cerebral palsy than in the healthy children, whereas the gastrocnemius muscle EMG values were similar in both groups. These findings suggest that neither an overactive gastrocnemius muscle nor a silent TA muscle was the cause of reduced ankle dorsiflexion in children with cerebral palsy. Measurement of Achilles tendon tension and EMG recording confirmed that, in children with cerebral palsy, there was increased tension during plantar flexion, whereas the gastrocnemius muscle EMG level was reduced. The authors concluded that mechanical changes in muscle fibers created increased extensor muscle stiffness (passive tension), which required greater TA muscle activation to produce dorsiflexion. From these results, it appears the limited dorsiflexion was related to the passive restraint of the extensors rather than to active restraint produced by extensor spasticity. Other researchers provide evidence of mechanical changes in muscle properties that may contribute to abnormal movement patterns.

Hufschmidt and Mauritz provided further evidence of changes in passive muscle properties in patients with...
spastic cerebral palsy. Comparing 20 healthy subjects with 21 individuals with spasticity, these authors concluded that spastic contracture was a consequence of a complex process involving degenerative atrophy and fibrosis of muscle tissue, as well as an alteration in passive muscle properties. Any approach to improve patient function that is directed only at the nervous system may result in a patient who still has ineffective motor control attributable to mechanical changes in muscle properties. Both systems must be considered when assessing patients with motor dysfunction and when prescribing treatment.

**Abnormal spinal circuitry.** Besides weakness and changes in passive muscle properties, there is some suggestion of abnormal spinal circuitry in children with cerebral palsy. Myklebust et al.\(^1\) define cerebral palsy as damage to immature supraspinal structures that imposes a secondary disorder on a developing spinal cord. Their conclusions are based on observations of abnormal muscle responses during reflex testing as well as during voluntary movement. Abnormal patterns of reciprocal excitation, reciprocal innervation, and changes in agonist control have been reported in children with cerebral palsy.\(^2\)\(^-\)\(^6\) Myklebust et al.\(^4\) report reciprocal excitation of the TA muscle in response to a soleus muscle stretch, which suggests a functionally disordered spinal circuitry. Kundi et al.\(^7\) reported that children with cerebral palsy had abnormal somatosensory evoked potentials (SEPs) before posterior rhizotomy. After posterior selective rhizotomy, H-reflexes and dorsal cord potentials from SEPs were depressed, but the SEPs recorded over the cortex did not change. These investigators concluded that the somatosensory disorder is in the spinal cord below the cervical level. These observations and a recent report by Brouwer and Ashby\(^26\) provide additional evidence for the hypothesis that cerebral palsy may be a disorder of spinal circuitry as well as a disorder of the brain. There appears to be some evidence that cerebral damage alone cannot explain the movement disorders observed in children with cerebral palsy. Secondary changes in spinal circuitry should be considered. Surely, the belief that problems of movement control in children with cerebral palsy are the result of a release from inhibitory supraspinal control is at best an oversimplification of the complexities of this disorder.

**Muscle coordination.** Patients with cerebral palsy often activate muscles in abnormal sequences and are unable to produce compensatory postural movements.\(^9\)\(^-\)\(^12\) This lack of muscle control may result in abnormal muscle patterns in which several muscle groups contract simultaneously and co-contraction of the agonist and antagonist occurs.\(^9\) These difficulties controlling muscle activation patterns are often attributed to problems of hypertonia and spasticity.

Abnormalities in muscle activation patterns, reciprocal movement, and stretch reflexes may affect voluntary movement in patients with hemiplegia.\(^5\)\(^7\)\(^-\)\(^40\) Sahrmann and Norton\(^40\) investigated isometric, isometric, and passive ROM activity of the upper extremity in 14 patients with hemiplegia. They reported that primary impairment of movement is due to limited and prolonged recruitment of agonist muscles and the delayed cessation of agonist contraction at the end of movement. During reciprocal movement, there was an inappropriate overflow and maintenance of contraction of the agonist muscle. Several authors\(^7\)\(^8\)\(^,\)\(^13\)\(^-\)\(^14\)\(^,\)\(^27\)\(^,\)\(^29\) have reported abnormalities in muscle activation during hemiplegic gait. The most striking characteristic of muscle activity in hemiplegic subjects is the variability of muscle patterns. Although there are differences among subjects, some general characteristics consistently reported were (1) less muscle activity in the paretic limb than in the nonparetic limb, (2) prolonged muscle burst durations, (3) tonic rather than phasic activity at transitions in gait, and (4) periods of peak muscle activity that do not coincide with requirements for a normal gait pattern.\(^5\)\(^8\)\(^-\)\(^44\) For example, Peat et al.\(^41\) reported that activation of the TA muscle was absent at toe-off and heel-strike and that gastrocnemius muscle activity did not peak at push-off as expected.

Knutsson\(^44\) observed that activation of muscles occurred in the wrong phase of the gait cycle and that misdirected coactivation of muscles caused muscles to cancel the action of each other. Knutsson and Richards\(^38\) examined the EMG patterns in 26 hemiplegic subjects. They reported that an individual demonstrated one of three characteristic EMG patterns during gait. Pattern I was characterized by premature activation of the triceps surae muscle during early stance; pattern II was characterized by low levels of muscle activity, with a normal temporal pattern; and pattern III was characterized by coactivation of several muscle groups during the gait cycle. Knutsson and Richards concluded that each hemiplegic subject has a unique motor control problem during locomotion, which is reflected in the EMG pattern.

Timing and force production appear to be important factors necessary for well-coordinated movement. There is evidence that controlling both the onset, termination, and level of muscle activity may be difficult for patients with cerebral palsy and patients with adult onset of hemiplegia.

**Gait.** According to Sutherland et al.\(^45\) both maturation of the CNS and learning are necessary for the development of normal gait. During early stages of motor development, children have a high cadence, a slow walking velocity, and a short single-limb support time. Duration of single-limb support and velocity increase and cadence decreases with increasing age.\(^45\) Gait characteristics of children with spastic diplegic cerebral palsy are different from normal gait characteristics because of the lack of motor control and the slow rate of development.\(^8\) Because the control mechanisms are at fault in cerebral palsy, rather than the motor system alone, analyzing the deficits in ambulation of children with cerebral palsy is difficult.\(^8\)
According to Strotzky, healthy children walk with an average cycle duration of 0.89 second and a velocity ranging from 1.01 to 1.25 m/s, whereas children with cerebral palsy have a slower average cycle duration of 1.01 seconds and a walking velocity between 0.71 and 0.86 m/s. The slower gait was attributed to restricted stride length rather than to decreased cadence. Generally, there was a decreased amplitude of movement in all children with cerebral palsy as compared with healthy children.

Norlin and Per Odenrick studied the gait of 50 children with spastic cerebral palsy between 3 and 16 years of age and compared them with a group of age-matched healthy children. They reported that, in children with cerebral palsy, the maximum cadence decreased as age increased. Increasing age, more time was required for each stride and the entire gait cycle slowed. In contrast, the cadence increased with age in the control group. This discrepancy is understandable because of the higher cadence in the children with cerebral palsy than in the age-matched controls. The children with cerebral palsy also showed a prolonged stance phase and shortened swing phase compared with the controls. Similarly, Strotzky observed that five of the six children with cerebral palsy had step asymmetries when compared with the consistent step symmetry of healthy children. Strotzky and Norlin and Per Odenrick considered these asymmetries a compensatory adjustment for poor posture control and balance during single-limb support. These authors reported that the typical child with cerebral palsy had an increased double-limb support time when compared with healthy children, which also suggests problems with posture and balance.

Patients with adult onset of hemiplegia also have diminished amplitudes and velocities of joint movement. Several authors attribute the small step lengths and joint amplitude to a limited ability to produce selective joint movement and to poor balance. This selectivity problem is evident when patients attempt simultaneous hip flexion with knee extension at terminal swing. Walking velocity for healthy subjects is strongly correlated to stance but not to swing time, whereas walking velocity for hemiplegic subjects correlated with both stance and swing time of the hemiplegic limb. Hemiplegic subjects' inability to move their hemiplegic limb quickly through the swing phase may be an important factor limiting walking velocity.

Brunnström suggested that gait abnormalities result from the slowness of the movement itself in addition to the inability to control selected movements. This concept predicts that walking faster will improve gait in patients with hemiplegia and that walking slowly will increase abnormalities in healthy subjects as well as in subjects with hemiplegia. It is possible that healthy subjects walking at the same velocity as patients with hemiplegia would have similar gait abnormalities.

Studies by Borkowski et al and by Lehmann et al characterized the gait of healthy individuals walking at slow speeds. These authors reported an increased variability of spatial-temporal characteristics and of limb movement patterns at slow walking speeds compared with self-selected or fast walking speeds. Some abnormalities of gait could be explained by walking speed alone, whereas others could not. Lehmann et al examined the gait of subjects with hemiplegia secondary to stroke and of healthy subjects matched for gender, age, height, and weight. Hemiplegic subjects walked at a self-selected speed only, and healthy subjects walked at a self-selected speed and at the same speed as the hemiplegic subjects. Abnormalities in step length, stance, swing, and double-support duration in subjects with hemiplegia were attributed to walking speed alone. Gait asymmetries, which included a shortened step length, a prolonged stance and shortened swing duration of the unaffected limb, and a shortened stance duration of the affected limb, were unique to hemiplegic gait and could not be explained by walking speed alone.

In summary, patients with spasticity demonstrate neurophysiologic and biomechanic problems associated with stretch reflexes, muscle atrophy, MU recruitment, firing rate, force production, timing, and muscle activity patterns. All of these deficits contribute to dysfunctional movement patterns and abnormal postures in children with cerebral palsy. There is ample evidence that the movement dysfunction is related to parameters of motor control that involve initiation, execution, and control of movement trajectory, speed, and accuracy.

Neither the mechanisms of abnormal movement in children with spastic cerebral palsy nor how abnormalities occur during development is clear. Research is needed in this area to clearly identify mechanical, neuromuscular, and neural changes occurring during development that contribute to movement dysfunction. The challenges to pediatric physical therapy are identifying specific characteristics of motor control that contribute to movement dysfunction and developing treatment to improve motor control. As we develop treatments, it is important that we determine the most effective treatment for improving functional movement.

Where Do We Go from Here?

Spasticity continues to be associated with movement dysfunction, although the relation of spasticity to function is unclear. It is possible that spasticity is not the cause of movement dysfunction, but that the mechanisms associated with spasticity and voluntary movement control are interactive. There is a continued belief that reducing spasticity will improve motor control. Some texts are devoted to the treatment and management of spasticity. These treatments include various physical therapy techniques that are designed to teach the patient new postures and movement patterns; pharmacological agents that affect the peripheral nervous system or the CNS; and surgical intervention for tendon division, release of contractions, and osteopathies. Each of these methods may decrease spasticity and...
increase function with varying degrees of success. Several authors suggest, however, that these techniques produce only a temporary increase in function because they do not address the source of the problem, which is a fundamental imbalance in the CNS. The proponents of selective dorsal rhizotomy believe that the selective division of abnormal rootlets addresses the problem of neural imbalance and produces a permanent change in the nervous system. My review of the literature and my own studies demonstrate that the effects of selective dorsal rhizotomy and the accompanying physical therapy provide some interesting insights into motor control problems in children with spastic cerebral palsy.

**Selective Dorsal Rhizotomy**

For years neurosurgeons attempted to decrease spasticity by reducing input to the anterior horn cells, although only recently dorsal rhizotomy has become a popular treatment for children with cerebral palsy. Dorsal rhizotomy was first documented by Sherrington in 1898. He discovered that extensor rigidity in some decerebrate cats was eliminated by sectioning posterior rootlets. Fifteen years later, Foerster used this technique to reduce tone in patients with congenital spastic paraplegia. Because of the negative side effects associated with sensory loss, however, Foerster did not recommend this procedure for improving function. Fasano et al. described a modified method for identifying and sectioning aberrant posterior rootlets that reduced the sensory loss associated with rhizotomy. This method involves electrically stimulating rootlets to identify the aberrant rootlets. In response to the stimulation, aberrant rootlets show an abnormal tonic contraction in the muscles innervated by the stimulated root as well as in distant muscles. These rootlets also revealed a higher excitability threshold and a greater variability of response. Through selective interruption of these abnormal circuits, Fasano et al. claimed to reduce spasticity and preserve sensation by sectioning only the aberrant rootlets. Peacock and colleagues further modified this surgical procedure by changing the surgical site from the conus medullaris to the cauda equina, thereby preserving the sacral nerve roots innervating the bowel and bladder. Generally, reports of the efficacy of rhizotomy for decreasing spasticity and improving function have been favorable.

Although the emphasis of this article is on improving functional capacity for children with cerebral palsy, rhizotomy is frequently performed on children with severe spasticity to improve daily care and comfort. Families report that patients are easier to bathe and position after rhizotomy, and some patients report increased comfort when the spasticity is "released."

Peacock et al. performed selective posterior rhizotomy on 22 children who had increased muscle tone of cerebral origin. Cinematographic records taken before and after surgery revealed decreased spasticity and increased motor function for sitting, standing, and walking. Those patients who had walked independently prior to surgery walked with what was described as an "improved" pattern after surgery. This study provided no objective measurement of spasticity and function. Using a quantitative, although descriptive, analysis of behavior, Irwin-Carruthers et al. noted that, before surgery, one child ambulated with a toe-heel pattern at the ankle, and with knee flexion during initial contact. After surgery, this child had a heel-toe pattern at initial contact, and very minor gait abnormalities. A recent report by Vaughan et al. provides quantitative evidence of improved gait kinematics after dorsal rhizotomy. Kinematic gait analysis of 14 children was performed 1 to 2 days before surgery and between 5 and 14 months after surgery. Significant increases were reported for stride length and for thigh and knee angular displacement, whereas walking speed and cadence were unchanged.

Many clinicians believe that physical therapy is necessary for maximal functional improvement in patients who have a posterior dorsal rhizotomy. According to Laitinen et al., dorsal rhizotomy deprives patients of previously learned motor patterns and forces them to "relearn" how to use their limbs. These authors suggested that, even though the selective sectioning of dorsal rootlets decreases tone and spasticity, it is necessary for patients to learn new and more efficient movement patterns.

For most children receiving a dorsal rhizotomy, an intensive period of physical therapy is prescribed after surgery and sometimes before surgery. Although several authors emphasize that physical therapy is an important determinant for successful postsurgical outcome, the duration and intensity of preoperative and postoperative care prescribed varies greatly among physicians. Physicians and therapists both have reported that, for a 2- to 6-month period after the rhizotomy, the children were weak. Therapists often described the patient's limbs and trunk as hypotonic and somewhat limp, although no specific strength evaluation was conducted. This apparent postoperative hypotonia was attributed to an underlying weakness that is revealed when spasticity and tone are reduced. These children appear to have underlying weakness more often than they have underlying voluntary control. The spasticity does not appear to be masking underlying control, as Bobath suggested. Removing spasticity in patients whose function depends on the spasticity has concerned the surgeons performing this procedure. Several surgeons agree that the underlying weakness and resultant hypotonia seen after surgery are related to poor functional outcome. Physicists and therapists both report the importance of developing a careful screening protocol that can identify those patients who have underlying strength or weakness. Other than the anecdotal reports, the reliability and validity of these screening evaluations has not been reported.

How does one evaluate strength in children with spastic cerebral palsy? The value of strength testing using
standard muscle testing procedures has been considered inappropriate for patients with spasticity. The definition of strength as the control of graded muscle force is not questioned. It appears that the problem is with the standard testing procedures used to assess muscle strength. What is questioned is the validity of the assumption that forces produced by a spastic muscle during muscle testing are an indication of the subject's ability to control those muscle forces for functional movement. Patients may be able to produce considerable force in their muscle, but are not able to control and grade forces for fine movement control.

Evaluating the strength of spastic muscles has been troublesome for therapists. Several therapists have suggested that testing for voluntary isolated joint movement and controlling the speed and trajectory of limb or trunk movement were valid methods for assessing functional strength and muscle control in children with spastic cerebral palsy. Controlling movement initiation, speed, timing, and direction changes are important factors in motor control theory. The concept that children with cerebral palsy are weak and that hypertonic muscles are weak is probably unsettling to many therapists. Once the spasticity and tone are reduced as a result of dorsal rhizotomy, the weakness is revealed and the importance of parameters such as strength and motor control becomes apparent. What is the explanation for profound weakness and abnormal movement patterns in the absence of spasticity? With better insight into the basis of the movement dysfunction in cerebral palsy, we will be able to direct treatment more efficiently to improve movement control.

The rationale for performing a selective dorsal rhizotomy is based on the clinical assumption that spasticity is the underlying cause of disordered movement and that reducing or eliminating the spasticity will improve motor control and will increase the capacity for improved function. This assumption has produced considerable controversy among clinicians and neuroscientists. The relationship of spasticity to movement is unclear and mostly depends on the investigator's definition of spasticity. The purpose of performing dorsal rhizotomy is to reduce spasticity and allow the development of normal movement patterns. The emphasis on reducing spasticity is evident in studies of the effects of selected dorsal rhizotomy. Cahan et al tested F-waves and H-reflexes before and after selective dorsal rhizotomy in 20 children with spastic cerebral palsy who were between 25 and 9.8 years of age. The authors concluded that the reduced values in these reflexes after surgery confirmed the clinical observation of reduced spasticity. Kundu et al reported that, after posterior selective rhizotomy, H-reflexes and dorsal cord potentials from SEPs were depressed. Fasano et al conducted a 2- to 7-year follow-up of 80 children and reported that spasticity returned in only 5% of the cases. Peacock and Arens reported an increased maximum plantar-flexion torque in four patients with multiple sclerosis 4 weeks after selective dorsal rhizotomy. Several authors have observed that, after dorsal rhizotomy, lower limb strength decreased initially, then gradually increased, resulting in improved function. Landau and Hunt challenge the evidence for improved function. They point out the lack of quantitative measurement and of controls for development and therapy. The surgeons performing rhizotomy, however, acknowledge the weaknesses in their studies and stress the importance of therapy following surgery.

Peacock and colleagues suggest that the children with cerebral palsy who appear to have the most improved functional mobility after selective dorsal rhizotomy are those children with spasticity as the major clinical symptom, who are of normal intelligence, and who are motivated. Those benefiting the least in the area of improved functional mobility are patients with ataxia, athetosis, severe joint contracture, and marked underlying weakness. The result of dorsal rhizotomy is reduced peripheral sensory input to the spinal cord. A release of supraspinal inhibition and increased gamma or fusimotor drive was thought to be the cause of spasticity. Spasticity attributed to excessive gamma drive should be reduced if feedback from the muscle spindle is diminished. The role of gamma drive as a source of spasticity, however, has been challenged, and sample evidence exists to suggest that excessive spindle bias is not the culprit. Peacock and colleagues believe that some of the peripheral reflex circuits are abnormal and that the abnormal circuits can be identified by electrically stimulating the roots. Using a procedure that interrupts only the abnormal circuits protects the normal circuits and preserves valuable sensory feedback from the limb. The effects of selective dorsal rhizotomy on parameters of motor control have not been elucidated in the literature. We need to identify specific characteristics of motor control that contribute to dysfunctional motor patterns and then evaluate the effects of treatment on these control parameters.

In an ongoing longitudinal study of dorsal rhizotomy, my colleagues and I are measuring the changes in ROM, reaching, sit-to-stand movement patterns, sitting and standing posture, and temporal gait parameters. The children with cerebral palsy are assessed before and after surgery. Subjects are tested twice preoperatively, again at 6 weeks after surgery, and then at 4- to 6-week intervals for a period of 6 months. Children are videotaped during quiet standing, long sitting, and bench sitting to record posture alignment. Movement patterns are recorded during sit-to-stand, reaching, creeping, and walking tasks. Last, we record passive ROM of
the hips, knees, and ankles. Preliminary data have been reported on a small number of children. Data collection and analysis are ongoing as subjects become available.

**Sitting and Standing Posture**

Before surgery, all children had difficulty maintaining a long-sitting posture. They sat with a posteriorly tilted pelvis and with maximum trunk flexion in an effort to keep from falling backward. By the first session after surgery (4-6 weeks), they were able to maintain a long-sitting posture easily, with increased trunk extension and less posterior pelvic tilt. No change was observed in posture during bench sitting. During standing, ankle dorsiflexion and knee extension increased after dorsal rhizotomy. In some children, ankle dorsiflexion was excessive the first 2 months after surgery, then gradually improved to a neutral position. An example of the changes for ankle and knee ROM in one subject is shown in Figure 1A. After surgery, all children were fitted with ankle-foot orthoses that they wore during weight-supported activities.

Prior to surgery, trunk trajectory plots during the sit-to-stand task revealed segmental patterns within and among subjects. Movement ratios (linear distance/total distance) for all children with cerebral palsy were below the normal values of age-matched subjects. Average velocity was less than normal for all subjects and as much as eight times less than normal in one subject. Movement times for subjects with cerebral palsy were less than normal and showed more variability. During the 6 months following dorsal rhizotomy, trunk trajectories became smoother and more curvilinear and had a greater horizontal component than prior to surgery (Fig. 2). Visual observations suggest that patients became increasingly independent and required less assistance from the therapist to achieve standing. Although patients improved in functional ability, there were periods when they showed a decrement in kinematic values. Movement trajectories had increased irregularities, and movement time increased rather than decreased. Review of the video records during these periods indicated that the apparent regression in behavior was associated with the child's improved level of independence. The trajectories and velocity curves from early successful attempts at the sit-to-stand task without therapist assistance looked worse than trials recorded with therapist assistance. As children became more skilled in the independent mode, the trajectories gradually became smoother and more curvilinear and movement time decreased. These periods of regression or change in behavior marked an increased level of independence in the sit-to-stand task.

As functional ability increased, movement patterns improved slowly. Reviewing the videotape records, we observed that, even though spasticity decreased (resistance to quick strength and clonus), children contin-

| Figure 1. Ankle and knee angles of one subject during quiet standing (A) and at initial foot contact during gait (B). Two evaluations were conducted before surgery (PRE-OP), and five evaluations were conducted at monthly intervals after surgery (POST-OP). |
Figure 2. Trunk trajectories of a 4-year-old before (PRE-OP) and after (POST-OP) selective dorsal rhizotomy. Vertical and horizontal coordinates of a marker on C-7 represent the path of the trunk in two-dimensional space. Before surgery, rocking movements are shown as intersecting loops during horizontal movement. After surgery, the trajectory gradually became smoother.

Gait

Overall, all subjects showed improvements in gait characteristics following surgery. Each subject, however, progressed at a different rate and made varying degrees of improvement, emphasizing the need for within-subject comparisons. Cycle duration, stance time, and double-limb support time increased, whereas velocity and cadence decreased, 6 weeks postsurgery. Over the next 5 months, however, cycle duration, stance time, and double-limb support time decreased and walking velocity increased (Fig. 3). After surgery, knee and ankle changes during walking were variable and there was a tendency toward decreased knee flexion and ankle plantar flexion at foot contact and toe dragging during the swing phase (Fig. 1B). Generally, the children showed increased knee and ankle flexion during stance for the first 2 months after surgery. Gradually, the pattern changed to knee extension and a neutral ankle position. Interestingly, even though stride characteristics and passive ROM improved fairly quickly after surgery, the same abnormal movement patterns of limbs were observed before and after surgery. For example, all children had adequate passive ROM at the ankle; however, during gait they continued to have a forefoot strike pattern, which was exaggerated when they walked faster or were observed at play. The children, however, were able to place their foot flat during gait when they were instructed, attended to the task, and walked slowly. It appears that selective dorsal rhizotomy initially improves static movement patterns more than it does dynamic movement patterns.

Long-Term Changes

We have followed one 2-year-old female subject for 20 months, who before surgery was ambulating with assistance in a rear-wheeled walker, had severe ankle extensor spasticity, walked in equinus, sat on a bench independently, required maximum assistance for the sit-to-stand task, and had difficulty maintaining a long-sitting posture. The subject received physical therapy for approximately 1 year prior to surgery. The first examination conducted 6 weeks after surgery revealed that the subject's passive ankle dorsiflexion, knee flexion, and hip extension and abduction increased greater than 50%. For 2 to 3 months after surgery, her gait was slow, she dragged her feet, and she had difficulty standing, because her knees buckled. By 6 months postsurgery, she was sitting, with her trunk erect in a long-sitting posture; standing with foot flat, ankles in neutral, and knees extended; and ambulating independently in a walker using a forefoot pattern with a high-steppage gait. One year after selective dorsal rhizotomy, she stood independently for 1 minute, was beginning to walk with Lofstrand crutches, and stood from a bench-sitting position without support with her feet flat. She still had an equinus gait pattern when playing or moving quickly, but corrected to a foot-flat pattern for a brief period when instructed. By 20 months postsurgery, she ambulated independently with Lofstrand crutches and most of the time walked with good foot placement and erect posture. She was able to maintain independent quiet standing balance for 3 or more minutes and took a few steps without any assistance or assistive devices before losing her balance.

What Does It Mean?

The decreased walking velocity and knee buckling during standing observed in most subjects after surgery suggest lower limb weakness. The mechanism for the observed weak-
gradually returned to preoperative levels during the 6 months after surgery. The subjects who showed a steady improvement following surgery may be the result of decreased resistance to passive stretch (spasticity). As stated previously, all subjects had more normal knee and ankle ROM in sitting, in standing, and during initial contact in gait; however, these changes were not consistent, and abnormal movement patterns persisted. This persistence of abnormal patterns in the presence of reduced spasticity and increased ROM suggests the influence of learned abnormal movement patterns. It is feasible that a period of learning, exercise, and practice is necessary for the continued development of normal movement patterns. This possibility is consistent with the reports from several authors that an intensive period of physical therapy after surgery is required for maximum functional improvement.

There are no studies to date that have included a control group to account for developmental changes with age or for therapy. A three-group research design would be the best design for an efficacy study. One group would have rhizotomy and therapy, the second group rhizotomy alone, and the third group therapy alone. I believe the results would be interesting, but the design would be fraught with difficulty. Consider the problems in matching groups for severity of disorder, functional ability, amount and frequency of physical therapy, family support, motivation, the number of rootlets severed, and the type of therapy received. Despite those potential problems, however, such research is the only way we will be able to identify the effect of the rhizotomy and the therapy.

**Conclusions**

Observations of reduced tone, resistance to stretch, and increased passive joint ROM within 48 hours of selec-

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**Figure 3.** Cycle period, velocity, and cadence of one subject before (PRE-OP) and after (POST-OP) selective dorsal rhizotomy. Test sessions were conducted at monthly intervals before and after surgery. Cadence decreased and cycle period increased after surgery, then gradually returned to preoperative levels during the 6 months after surgery.
tive dorsal rhizotomy in humans is consistent with reports of reduced stretch responses and rigidity after complete rhizotomy in controlled animal studies. Although there is evidence of more normal knee and ankle angles, the subjects frequently used previously learned patterns of abnormal movement.

The effect of dorsal rhizotomy documented in the literature provides evidence that, immediately after surgery, selective dorsal rhizotomy reduces spasticity and increases joint range of movement, apparently limited by abnormal tone. Improvements in strength and in the ability to control and coordinate movement patterns, however, were gained slowly over a prolonged period of time with therapy. Recent evidence suggests that passive and active properties of the antagonist are more related to movement dysfunction than those of the agonist (see article by Bourbonnais and Vandcn Noven for a review). I hypothesize that decreasing spasticity alone is insufficient for producing normal movement patterns and that a period of learning is required for patients to produce improved movement patterns and to use the expanded joint ROM available to them. I believe that the functional potential of patients receiving selective dorsal rhizotomy can be maximized through exercise programs that include strengthening, practice, and feedback for learning new patterns of movement in the presence of more normalized tone.

Why is this information important for physical therapists or scientists studying developmental motor control and motor learning? Therapists should be aware that treatment aimed at reducing tone and spasticity will not necessarily improve movement coordination. Reducing the tone may increase ROM, but may unmask underlying weakness rather than underlying control, as many therapists believe. If there is weakness in children with cerebral palsy, then therapists should place more emphasis on strengthening exercises. Many therapists are reluctant to use strengthening exercises for fear that they will increase spasticity and produce abnormal movement patterns. Hall and Light recently reported that adults with head trauma and severe spasticity are not adversely affected by strengthening exercises. They reported that, after strengthening exercises, performance on a side-to-side tapping task improved significantly. Kolobe recently reported that upper extremity strengthening exercises (eg, push-ups) for children with spastic cerebral palsy did not increase spasticity and function appeared improved.

It is time to test the clinical assumptions that are the basis for many therapeutic techniques and explore the concepts of motor learning and control. With the knowledge that patients with spasticity are weak and have difficulty initiating and controlling movement and that normal movement patterns are not necessarily underlying spasticity, we can forge ahead. Applying theoretical concepts of control and learning will expand our knowledge of movement and provide therapists with a direction for evaluating and treating patients with movement dysfunction. I propose that, as clinical therapists, we accept the role of clinical scientists who are willing to explore, discover, test hypotheses, and develop new hypotheses for treating patients with movement dysfunction.

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