The Management of Childhood Spasticity

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SPASTICITY

Spasticity is a form of hypertonia characterized by a velocity-dependent increase in resistance to passive movement often associated with hyperactive tendon reflexes, clonus, and a decreased range of movement. Spasticity is but one feature of the upper motor neuron lesion that, in childhood, is encountered most frequently in association with cerebral palsy, a motor disorder caused by a static insult to the developing brain that occurs before, during, or after birth. Although there are many forms of cerebral palsy, such as spastic, hypotonic, dyskinetic, and mixed, the spastic type is most prevalent.[1] Features of cerebral palsy include the following:

1. Abnormal muscle tone (spasticity, rigidity, hypotonia, dystonia).
2. Increased tendon reflexes and clonus (in spastic patients).
3. Persistent early reflexes (e.g., asymmetrical tonic neck reflex, Moro).
4. Involuntary movements (athetosis).
5. A loss of fine and selective motor control.
6. Impairment of balance.
7. Decreased strength.
8. Delayed motor milestones.
9. Secondary contractures and deformities.[2, 3]

Although the exact cause of spasticity is not fully understood, it currently is believed to be a result of decreased inhibition from supraspinal centers (disinhibition), which leads to a decreased modulation of spinal level stretch reflexes.[4] Various types of inhibition may be diminished in spastic patients.

RECIPROCAL Ia INHIBITION

Within skeletal muscles, there are specialized intrafusal muscle fibers containing sensory organs that respond to stretch, the muscle spindles. The spindles respond to stretch by discharging types Ia and II afferent fibers, which are carried in the posterior roots and communicate directly or indirectly with the alpha motor neuron pools of the spinal cord inner-

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vating that muscle, its synergists, and its antagonists. The Ia afferent is integral to the monosynaptic stretch reflex, which directly causes excitation of the stretched muscle. Inhibition of the antagonistic muscle occurs via the Ia inhibitory interneuron. This interneuron also is affected by corticospinal tracts.[4] Impaired reciprocal inhibition may cause abnormal coactivation of opposing muscle groups or reciprocal "excitation."[5]

RECURRENT (RENSHAW) INHIBITION

Renshaw cells are activated by recurrent collaterals of the alpha motor neuron and inhibit the same alpha motor neuron. This mechanism is believed to be important in motor control, such as in graded voluntary contraction. Renshaw cell activity has been shown to be abnormal in spastic patients, but may be increased at rest rather than decreased.[6]

PRESYNAPTIC INHIBITION

Reduction in presynaptic inhibition may be involved in the hyperreflexia of spasticity. This depresses the monosynaptic stretch reflex by reducing transmission in the Ia fiber prior to its synapse with the alpha motor neuron. This occurs through an axoaxonic synapse of an interneuron onto the Ia terminal, which depolarizes the terminal, reducing the amount of available transmitter.[4]

ALTERNATE MECHANISMS

Other influences on the skeletal muscle include type II spindle afferents, which communicate indirectly with alpha motor neurons via interneurons and may excite or inhibit the homonymous muscle. Along with skin and joint receptors, type II afferents are involved in complex polysynaptic reflexes, such as the flexor withdrawal reflex, and often are termed collectively the flexor reflex afferents. Golgi tendon organs respond to muscle tension and inhibit the muscle of origin via interneurons. The muscle spindles themselves are adjusted by the gamma motor system. Hyperactivity of this system once was believed to be a major cause of spasticity, but this has been largely refuted.[4] Although the exact mechanisms involved in spasticity and the interplay between them may not be understood fully, intervention to reduce spasticity is aimed logically at either increasing inhibition or reducing the amount of facilitation of anterior horn cells, the major and final neurologic input to the voluntary effector muscles (Fig 1).

MEASUREMENT OF SPASTICITY

Spasticity is easy to recognize, but difficult to quantify. Quantitative evaluation has been attempted by many researchers over the years. This has included electrophysiologic measures such as the H-reflex,[7] as well as mechanical measurements of resistance to passive motion using spring sales and force transducers.[8–10] Unfortunately, electrophysiologic tests may not correlate well with the clinical picture,[11] and measurement of joint stiffness may reflect contracture as well as spasticity.[12] The Wartenberg pendulum test has been used to assess spasticity and rigidity of the knee

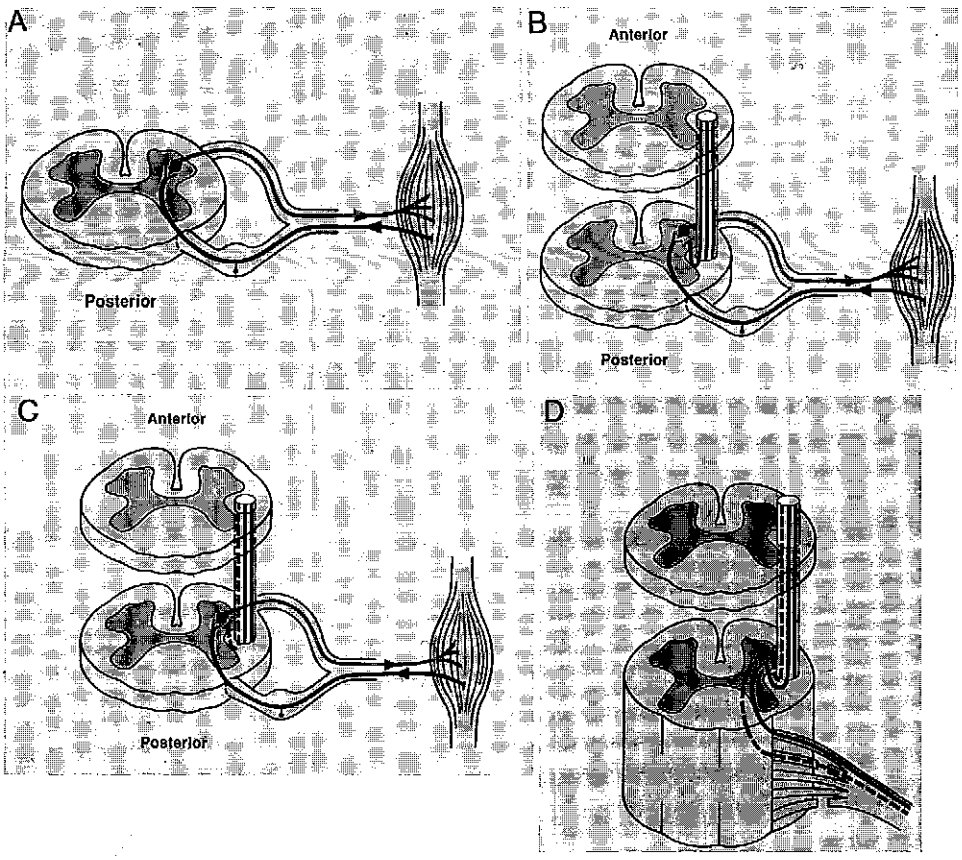

FIGURE 1.

A, an illustration of the stretch reflex. Ia afferents from the muscle spindle organs carry stretch-induced impulses through the posterior roots to the anterior horn cells, which then are activated to stimulate the muscle fibers via the anterior roots and thereby oppose the stretching influence. **B,** descending inhibitory tracts modulate the alpha motor neuron pool at the spinal cord level. **C,** because of an upper motor neuron lesion, the inhibitory effect at the cord level may be lost, producing a state of disinhibition. **D,** the effects of such disinhibition may be balanced by decreasing the afferent stimulatory effects on the alpha motor neuron by dividing the posterior rootlets.

in adults.[13–15] More sophisticated measures of reflex threshold obtained by monitoring electromyography (EMG) and torque at controlled speeds and angles have been proposed, but are difficult to apply clinically.[11, 16] Computerized systems involving measurement of torque and EMG during passive motion at different speeds of angular displacement of the ankle and knee,[17, 18] sinusoidal motion of the ankle,[19–21] and simultaneous hip and knee joint motion[22] have been described. Most of these methods are used frequently in research settings and are particularly difficult to apply to young children. Thus, clinicians largely still depend on examination of the patient's reflexes and resistance to passive limb movement. Many use the Ashworth scale or a modification of this scale to record clinical findings.[23] The Ashworth scale grades spasticity by resistance to passive movement (Table 1).

TABLE 1.
Scale for Grading Spasticity*

Score	Definition
0	Hypotonic: less than normal muscle tone, floppy
1	Normal: no increase in muscle tone
2	Mild: slight increase in tone, "catch" in limb movement or minimal resistance to movement through less than half of the range
3	Moderate: more marked increase in tone through most of the range of motion but affected part is moved easily
4	Severe: considerable increase in tone, passive movement difficult
5	Extreme: affected part rigid in flexion or extension

*Scale modified from those of Ashworth[26] and Bohannon and Smith.[23]

Although it is important to refine methods for quantifying spasticity, it is equally important to evaluate the role of spasticity in functional impairment. In adults with spastic upper extremities, Sahrmann and Norton found that poor agonist control was more important than spastic restraint in limiting voluntary movement.[24] Lee et al. concluded that stretch reflex enhancement was not responsible for stiffness of voluntarily activated elbow muscles of adult spastic patients.[12] Others have found that lower-extremity function is disturbed by hyperactive reflexes in adults and children.[13, 22] Corcos et al. concluded that interference by spasticity occurs when the patient has adequate strength and motor control to produce a voluntary contraction that is rapid enough for antagonistic stretch reflexes to be activated.[25] It is important to note that much research in spasticity has been performed on spastic upper extremities of adults and may not be applicable to the spastic lower extremities of a child with cerebral palsy. There is some evidence that spasticity is manifested differently in those with adult-onset injuries than in children who sustained an insult during a period of maturation. Myklebust et al. noted that reciprocal excitation or abnormal cocontraction was more common in children with cerebral palsy than in adults who acquired spasticity.[5] Others have noted abnormal coactivity of antagonistic muscles in spastic children during gait[27] and during balance perturbations while standing.[28] Dietz found that children with cerebral palsy as well as very young children without neuromotor deficits lacked normal polysynaptic reflexes during gait and exhibited Ia-mediated monosynaptic stretch reflexes, which normally are absent. This implied that children with cerebral palsy had failed to progress in motor development from a more primitive state.[27] Berbrayer and Ashby studied reciprocal inhibition in teenage and adult patients with cerebral palsy by stimulating the posterior tibial nerve and recording single motor unit responses in the tibialis anterior muscle. They found intact reciprocal inhibition; however, they described cocontraction in response to magnetic stimulation over the motor cortex in patients with cerebral palsy.[29] Thus, some of the abnormal cocontraction

observed in patients with cerebral palsy may be initiated or mediated centrally.

The most objective evaluation of function in the spastic child currently available to many clinicians is instrumented gait analysis. This may involve kinematic analysis, EMG, force plate studies, and footswitch studies. Kinematic analysis normally is performed by placing markers on bony landmarks and using videotape or film to assess motion at key joints throughout the gait cycle. This provides information about the pattern of motion and active range of movement during gait. Graphic analysis of kinematic data in the form of "angle-angle" diagrams is useful in analyzing selective motor control.[30–32] EMG allows examination of the timing of muscle action during gait. It also can help to identify overly active muscles, clonic activity, and abnormal cocontraction. Force plates record the method of limb loading and unloading, and aid in examining stability during unilateral stance. Integration of force plate data (kinetics) with angular velocity data (kinematics) allows the calculation of joint moments and power generation magnitudes. These parameters are valuable in assessing the quality and efficiency of gait. Footswitches are a rapid and useful way to obtain timing parameters and foot-floor contact patterns. Gait evaluation is valuable in documenting functional abilities of the patient, aiding patient management decisions (including the necessity for surgical intervention), and evaluating the results of surgery postoperatively.

CLINICAL EFFECTS OF SPASTICITY

Spasticity interferes with movement in any patient, but has particularly profound effects in the growing child. Severe spasticity can interfere with daily care such as hygiene, skin care, positioning, feeding, and toileting. Respiratory and oral motor function, including speech, can be affected. It is particularly difficult to meet the hydration and nutrition needs of spastic children with severe total body involvement. Spasms and clonus may interfere with comfort, sleep, safety, and function. Lack of mobility can prevent appropriate relief from pressure over bony prominences. Major effects on bone, joint, and muscle may be particularly troublesome in the acquisition of motor skills in a developing child. Spinal deformities and hip dislocations are common in this population. Even those with relatively mild involvement are at risk for the development of contractures and gait abnormalities. Energy expenditure during movement is much higher for spastic individuals in comparison to their nonspastic peers.[33]

CLINICAL ASSESSMENT OF THE SPASTIC CHILD—THE TEAM APPROACH

Because spasticity is due to a neurologic disorder that affects the musculoskeletal system, an effective approach requires a multidisciplinary assessment and treatment plan. One way to achieve this is through a team consisting of a pediatric orthopedic surgeon, a pediatric neurosurgeon, and a pediatric physical therapist. The patient and family are seen simul-

taneously by all team members to prevent repetitive examinations and to stimulate interaction between the team members.

First, a careful developmental and medical history is obtained to confirm the diagnosis of cerebral palsy, along with an assessment of the child's current functional level. Of prime importance is a history of the involved pregnancy, delivery, and perinatal events such as development of jaundice, intraventricular hemorrhage, and posthemorrhagic hydrocephalus. Any history of seizures, mental retardation, and motor delay is noted. The child then is examined in turn by each of the three specialists.

The neurologic examination emphasizes evaluation of muscle tone and tendon reflexes. It is important at this stage to look for the presence of spasticity and to exclude the presence of other forms of hypertonia. Spasticity is a velocity-dependent form of hypertonia that is characterized by the "clasp-knife" phenomenon. There usually is an initial "grab," followed by a release during passive movement of the limb. It is associated with brisk tendon reflexes, clonus, and a reduced range of motion. In contrast, rigidity is not velocity-dependent and presents with constant resistance throughout the range of motion in both directions. Dystonic children have fluctuating muscle tone, and some have mixtures of spasticity with rigidity or dystonia. Abnormal involuntary movements that are writhing in nature and rotate about the axis of the limb are classified as athetosis. Ataxia is a disturbance in equilibrium characterized by intention tremor, past pointing, and staggering gait that rarely is seen in patients with cerebral palsy. Strength, motor control, balance, and gait are especially important to assess in patients for whom selective posterior rhizotomy will be considered. Other features are assessed also, such as persistent primitive reflexes, head circumference, cranial nerve function, sensory function, and cognitive status, depending on the individual patient.

The orthopedic portion of the examination emphasizes an evaluation of range of motion, contractures and deformities, strength, balance, posture, and gait. Clinical and radiographic assessment of the spine and hips are performed routinely. The range of motion of the hips, knees, and ankles is noted using quick stretch and persistent stretch techniques. Any contracture of hip adductors and flexors is evaluated as well as internal/external rotation range ratios for the assessment of femoral anteversion. Ryder's test for anteversion also is used. With the patient in the prone position and the knee flexed to 90 degrees, the trochanter is rotated to be as lateral as possible. The degree of anteversion is estimated to be the angular deviation of the leg from its presumed neutral position perpendicular to the examining table. The popliteal angle is utilized as a measure of hamstring tightness, and inability to straighten the knee to within 30 degrees of full extension is considered to be significant. Katz et al. found that the range of the popliteal angle in normal children over the age of 5 years was 0 to 50 degrees, with a mean of 26 degrees.[34] In addition, it is helpful to look at hip abduction with the knees flexed and extended to determine if the medial hamstrings are involved. Ankle dorsiflexion often is limited and either varus or valgus may accompany equinus. In spastic diplegic patients, valgus frequently is seen with a tendency

toward a rocker-bottom foot. The equinovarus deformity is noted more often in the spastic hemiplegic child. The thigh foot axes and bimalleolar axes are employed as measures of tibial torsion. Many children have difficulty with intoeing, which may be caused by a variety of factors, including internal hip rotation due to spastic or overactive muscles, especially the medial hamstrings. Alternative explanations include medial femoral torsion (anteversion), internal tibial torsion, or adduction of the forefoot, which accompanies a varus foot and ankle posture. There may be a combination of factors present in any particular child's limb. Some children have femoral anteversion with compensatory external tibial torsion. Correction of only one aspect may cause further malalignment by altering the compensation that already has occurred. Thus, the postural and biomechanical features are analyzed and possible causes are considered.

Last, the child is examined from a functional and motor developmental standpoint by the physical therapist. The entire team is interested in the degree of spasticity, strength, volitional motor control, balance, trunk control, and functional skill of the child. Balance generally is evaluated by perturbing the child in a sitting position. More functional children also may be examined standing. Voluntary strength is assessed by looking at the child's ability to maintain and move through antigravity postures, usually standing and unilateral standing. Graded control of motions such as sitting to standing can help to distinguish between reflexive antigravity movements and voluntary control of such movements. The Rancho Los Amigos evaluation of upright motor control used for hemiplegic adults can be applied to children in order to estimate antigravity control in standing even when the child is dependent on synergistic patterns of movement.[35] This involves systematic assessment of the flexion and extension ability of the hip, knee, and ankle in a unilateral standing position, and is a good indication of both coordination and strength. Further ability to isolate independent joint movements is assessed by asking the child to combine various motions, such as knee extension with ankle dorsiflexion. Many children can perform ankle dorsiflexion only while using a synergistic pattern of hip and knee flexion ("the confusion test"). Observation of the child's ability to sit, stand, crawl, and walk probably is most important. The use of a device in walking should be noted and also the degree to which the child is dependent on the device. It sometimes is useful to support the child manually in order to gauge the amount of support required. Typical abnormal gait features include forward flexion at the hips or a backward lean (usually signifying hip extensor weakness), lateral trunk lean and/or pelvic drop on the weight-bearing side (Trendelenburg sign indicating hip abductor insufficiency), scissoring, lack of hip and knee motion (stiffness or spasticity) or exaggerated patterns of flexion and extension in a pistonlike motion (synergy dependence), crouch with equinus (compensation) or the foot flat (weak calf or overlengthened Achilles tendon), equinovalgus (pronation may make the foot appear plantigrade in spite of a heelcord contracture), equinovarus, toe-in, toe-out, and toe-drag (lack of knee flexion in initial swing or lack of dorsiflexion in late swing).

The observations of the entire team are recorded and videotapes are

made of the assessment and the child's gait during the clinic visit. Each team member articulates his or her findings to the family and the other team members. The major problems are summarized and appropriate intervention strategies are discussed. Surgical options including reduction of spasticity using selective posterior rhizotomy and orthopedic surgical procedures are proposed when appropriate. Those children for whom rhizotomy is recommended generally fall into two groups: severely spastic children with total body involvement and more functional children with spastic diplegia who have achieved some level of independent ambulation. Important factors to consider for rhizotomy candidates in whom functional improvement is the goal are the presence of pure spasticity without significant dystonia, the lack of significant fixed contractures, the presence of adequate lower extremity strength and motor control, and the presence of reasonable balance and sitting ability. Contraindications to rhizotomy include rigidity, significant athetosis or dystonia, weakness of the trunk or lower extremities, and lack of spasticity. Children with spastic hemiplegia generally are not considered to be candidates for rhizotomy. Also generally excluded are spastic children who have severe contractures at several joints and those who have overlengthened heelcords or multiple orthopedic surgeries in whom spastic postures have not recurred. Many patients for whom rhizotomy is recommended also require orthopedic surgical intervention. Nonsurgical interventions such as serial casting, orthotics, and physical therapy are recommended also. Many children undergo instrumented gait analysis to assist in surgical decision-making and program planning.

MANAGEMENT OF CHILDHOOD SPASTICITY

Physicians are taught to treat the causes of disease rather than the symptoms. Because of the complexity of the central nervous system, this rarely is possible in spastic children. Instead, we treat various peripheral manifestations that arise as a result of the central nervous system lesion. A prime consideration in treatment is that spasticity rarely exists independently, but usually in association with motor incoordination, underlying weakness, and problems with perception, balance, and selective muscle control. Frequently, there may be difficulty with behavior, learning, speech, swallowing, etc. Thus, the comprehensive management of spastic children calls for many special considerations in addition to the management of spasticity. In many cases, reduction of spasticity will not lead to a significant improvement in function, but will only unmask and highlight associated underlying problems. In some patients, spasticity is depended upon for antigravity activities, and its elimination as a result of treatment could lead to worsening of the clinical situation.

In practice, a major consideration for the child with cerebral palsy is the acquisition of mobility. This can be achieved by pushcarts, specially fitted bicycles, or wheelchairs, or by the patient's own ambulation, aided as necessary by orthotics, therapy, and surgery to facilitate the best situation possible. Wisdom in managing cerebral palsy is largely a matter of selecting the right patient for the proper treatment at an ideal time in his or her development and preventing or treating untoward events such as

a subluxing hip and scoliosis. For parents and the lay public, expectations must be tempered by realistic goals, a caring and flexible medical establishment, and the ability always to keep the overall picture in perspective. Treating physicians must be able to recognize which patients are likely to show significant benefit from the various therapeutic manipulations available. Although a complete treatise on the management of cerebral palsy is beyond the scope of this discussion, we will review some frequent considerations, as well as a few evolving treatment approaches now available.

MEDICAL TREATMENT OF SPASTICITY

At present, there is no completely satisfactory form of drug therapy for the treatment of spasticity.[36] There are a variety of agents that have been used clinically to ameliorate the effects of spasticity and to investigate further the specific nature of spasticity in any one patient. Most agents attempt to reduce the excitability of the spinal reflex arc within the spinal cord to some extent. The goal is to reduce spasticity without accentuating muscular weakness. Many other methods of reducing spasticity are directed more peripherally, such as motor point blocks or neurectomies. The disadvantage of these procedures is that the motor nerve is compromised and weakness is an unavoidable consequence. We will consider briefly a few medical approaches in further detail.

GAMMA AMINOBUTYRIC ACIDERGIC INHIBITION

Within the spinal cord, the terminals of the afferent neurons and interneurons that synapse with the motoneurons release the amino acid L-glutamate as a fast excitatory transmitter, along with a slower transmitter, substance P.[37–39] Both of these substances are inhibited by gamma aminobutyric acid (GABA). GABA is an endogenous inhibitory neurotransmitter that reduces monosynaptic and polysynaptic spinal cord reflexes in response to supraspinal stimuli conducted to axoaxonic synapses on the terminals of the primary afferent fibers. The GABA is taken up by receptors on these primary afferent terminals, which also can be occupied by GABA agonists such as baclofen. The major mechanism, therefore, is one of presynaptic inhibition of primary afferent terminals. The presynaptic inhibitory effects of GABA likewise can be potentiated by a class of agents known as the benzodiazepines, which includes diazepam (Valium). These agents may operate additionally at the motoneuron level by enhancing inhibitory postsynaptic potentiation, or directly at higher levels such as the brain stem reticular formation. Most studies of such effects are performed, by necessity, in experimental animals.

BACLOFEN

Baclofen binds to GABA and, through an activation of calcium conductance, initiates transmitter release in the primary afferent terminal. This means that there is less excitatory transmitter to be released as a result of normal afferent action potentials. Because baclofen is equally effective in complete and incomplete spinal cord lesions, it is thought that the spi-

nal cord is the major area of action. The oral administration of baclofen achieves only mildly therapeutic levels in the spinal cord.[40–43] Hence, clinical trials have focused on delivering the drug directly to the cord via intrathecal catheters and pumps. An initial pilot study of six children showed reduction in spasticity without side effects.[44] An additional randomized, double-blind trial of intrathecal baclofen vs. placebo was performed in 17 patients with cerebral palsy and 6 patients with other forms of spasticity. Lower extremity tone was decreased in the patients who received baclofen, but upper extremity tone and function were not changed. Confusion and drowsiness occurred transiently in two young patients.[45] Others have reported minor complications such as catheter disconnection and displacement,[46] as well as more serious problems such as impaired consciousness.[47] Repeated surgeries aimed at maintaining the catheters and pumps incur the risk of infection, including meningitis. Further clinical studies should clarify the relative risks, benefits, and indications for this method of treatment.

OTHERS

Progabide is another pharmacologic agent that may be metabolized to a more active substance that binds to GABA receptors and has been reported to reduce muscle tone, hyperactive reflexes, and flexor spasms.[48] Other substances that have been explored pharmacologically include the catecholamines, L-glutamate, and L-aspartate. Of particular note clinically is glycine, the major spinal cord inhibitory neurotransmitter released both by inhibitory interneurons responsible for reciprocal inhibition and by Renshaw cells, which mediate recurrent inhibition.[36, 49, 50] Dantrolene is used clinically for its effect on skeletal muscle. It inhibits the release of calcium from the sarcoplasmic reticulum, preventing activation of the contractile apparatus. It also decreases the discharge produced by stimulation of the fusimotor fibers. Whyte and Robinson recently have reviewed the pharmacologic management of spasticity in some detail.[51] They concluded that only dantrolene has been shown to be effective thus far in improving spasticity in cerebral palsy. There is concern regarding its use in patients with significant weakness, and the side effects of drowsiness, lethargy, dizziness, nausea, diarrhea, and paresthesias remain problematic.

For the most part, pharmacologic agents have not become the mainstay of treatment for childhood spasticity. Rigorous double-blind studies often have failed to demonstrate clear improvement, and there are serious side effects associated with many agents.

SURGICAL TREATMENT OF SPASTICITY

GENERAL COMMENTS

Until recently, the treatment of children with cerebral palsy was considered the realm of the pediatrician, physical therapist, neurologist, physiatrist, and orthopedist. Neurosurgical treatment was regarded as largely anecdotal and empirical, limited to gross attempts at facilitation of voluntary efforts with cerebellar stimulators[52] or various ablation procedures directed toward controlling unwanted muscular activities centered in the

brain or spinal column.[53] The neurosurgical focus now has shifted to the control of spasticity by the more selective procedure of dividing only certain posterior nerve rootlets on the basis of intraoperative nerve stimulation and peripheral EMG recordings, as recently espoused by Peacock.[54, 55] For completeness, we will approach the subject of rhizotomy by considering alternative neurosurgical procedures as well.

NEUROSURGICAL PROCEDURES

Interruption of Efferent Components

The efferent limb of the stretch reflex is composed of the anterior horn cell and motor nerve, which runs in the anterior spinal nerve root. Neurosurgically, it can be interrupted by peripheral neurectomy, anterior rhizotomy, or motor point block. Motor point blocks are covered later in this chapter; a variety of agents have been employed, from phenol to alcohol and, more recently, botulinum toxin. Anterior rhizotomy no longer is used for spasticity, as the complications included flaccid paralysis and loss of muscle bulk. It may have a limited role in the treatment of choreoathetosis, however.[56] Peripheral neurectomy is exemplified by such procedures as anterior branch obturator neurectomy. It is used to weaken an overactive muscle group. In the upper extremity, excessive external rotation of the shoulder and flexion of the elbow have been approached with suprascapular and musculocutaneous neurectomies, respectively. It is important to remember that peripheral nerves contain both motor and sensory fibers. Undesirable side effects include weakness, muscle atrophy, and sensory loss. Neurectomies as initial treatment are avoided, since they are not aimed at spasticity per se, but rather are a direct attack on muscle strength.

Interruption of Afferent Components

Selective posterior rhizotomy is a neurosurgical procedure designed to reduce spasticity during which certain posterior spinal nerve rootlets are divided. Although the indications and goals for orthopedic surgery and selective posterior rhizotomy may appear similar at first glance, they are not equivalent procedures. The goal of releasing muscle and tendons is eliminating *contractures*, not spasticity. Abbe described posterior rhizotomy in which whole posterior nerve roots were cut as early as 1889 for neuralgic pain relief.[57] The use of rhizotomy for the treatment of spasticity was introduced by Foerster in 1908.[58] He later described a larger series in 1913, including many children with spastic cerebral palsy.[59] He divided the whole posterior nerve roots from L2 to S2, but preserved L4 to help preserve extensor tone for standing. Foerster emphasized identification of "real" spasticity, exclusion of individuals who displayed athetosis or paralysis, and the benefits of the procedure for the spastic diplegic child. These continue to be important considerations today. The procedure fell out of favor, but was revived and modified by Gros in the 1960s.[60] Section of rootlets rather than whole roots was used to prevent sensory complications. Fasano introduced the use of electrical stimulation of rootlets in the 1970s and found that certain rootlets were associated with hyperactive EMG responses.[61, 62] Peacock revised Fasano's procedure by changing the surgical site from the level of the conus to the

level of the cauda equina to ensure accurate identification of spinal root levels and to prevent bowel and bladder complications.[54, 55, 63–65] The procedure has been reviewed by Ouaknine,[66] Laitinen et al.,[67] and others.[30, 68–70] The modern basis of selective posterior rhizotomy is the detection and division of posterior rootlets involved in disinhibited spinal reflex circuits. Intraoperative electrical stimulation of nerve rootlets and EMG recording of lower extremity musculature are used to help determine which rootlets should be selectively cut. Those rootlets associated with hyperactive responses to 1-second-long trains of electrical stimulation are divided and the remainder are spared. In sacrificing only a portion of a nerve root and avoiding total deafferentation, excessive weakness and sensory losses are minimized.

The procedure is performed under general endotracheal anesthesia without the use of long-acting muscle relaxants. The patient is positioned prone and is draped to allow access to the lower extremities by the EMG team. A midline laminectomy or laminotomy is performed from L2 to L5 and the first sacral spine is reflected to allow exposure of the nerve roots from L2 to S2. After opening the dura, the posterior roots are identified and separated from the anterior roots by observing their position, vascularity, shape, and threshold for electrical stimulation. Anterior nerve roots of S1 and S2 are stimulated to confirm the spinal levels. Stimulation of S1 causes knee flexion and ankle plantarflexion, and stimulation of S2 causes ankle plantarflexion and toe flexion. The posterior nerve root of L2 then is identified by counting upward. Each rootlet of each posterior root from L2 to S2 bilaterally is stimulated using two insulated electrodes and the responses are analyzed using EMG recordings to be described. Approximately 50 to 75 rootlets are tested and about one fourth to one half of these subsequently are divided. Pairs of needle recording electrodes are placed in five muscle groups of each lower extremity (adductors, quadriceps, tibialis anterior, hamstrings, and gastrocnemius muscles). Additionally, EMG recordings are obtained simultaneously from the external anal sphincter to prevent division of rootlets associated with reflex arcs involving the bowel and bladder sphincters, both of which are supplied by the pudendal nerve. "Normal" responses to the steady train of stimuli are considered to be those that either have a decreasing amplitude or remain steady over the 1-second stimulation period. These responses also should be confined to the muscle groups innervated by that spinal cord segmental level. Responses that are considered to be "abnormal" include incremental, sustained, clonic, and very irregular patterns. Additionally, those responses to subthreshold level stimuli that spread to muscle groups not normally innervated by the root tested are considered to be abnormal (Fig 2). The EMG information is used in conjunction with clinical information obtained during careful preoperative assessment, such as distribution of spasticity, functional level, and areas of weakness. Clinical judgment plays an important role in intraoperative decision-making. EMG responses that are equivocal in nature may be spared or divided depending on this clinical information as well as on the number and distribution of rootlets that already have been divided. The goal is to reduce unwanted spasticity while preserving as many nerve rootlets as possible.

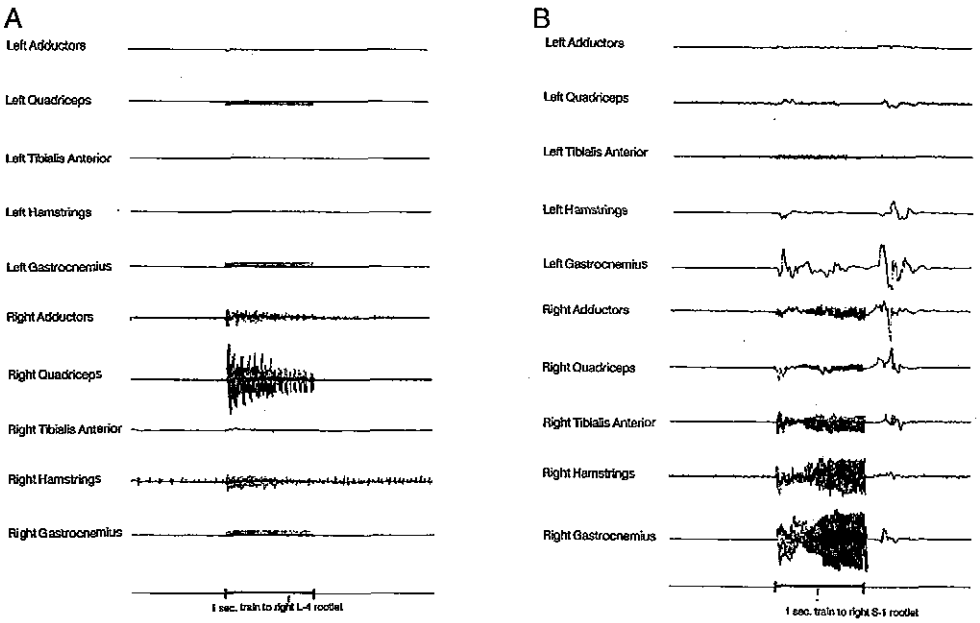

FIGURE 2.

A, a typical decremental response frequently observed when a posterior rootlet is stimulated by a 1-second train of impulses during the rhizotomy operation. **B,** an example of an abnormal incremental response to a similar stimulation applied to the right S1 rootlet. In addition to the incremental nature of the primary response, there also is spread to other muscle groups. Such a response would be considered abnormal, and the associated rootlet likely divided. Intraoperative EMG responses form the basis for selective posterior rhizotomy.

Following rhizotomy, spasticity is reduced markedly, but weakness often is a feature of the early postoperative period. Equipment such as ankle-foot orthoses and walkers are used to help protect weak muscles. Strengthening exercises and activities become dominant in the therapy program rather than repeated efforts to help reduce muscle tone and improve range of motion. In the early days to weeks following the procedure, some children experience hypersensitivity of the feet and legs or flexor spasms that resolve spontaneously. Some children have an initial period of hypotonia that also resolves after a few weeks. Crouched posture may be seen, especially if the previously spastic triceps surae muscles are weak. Exaggeration of the pronated posture of the foot may occur once the spastic calf muscles are relaxed. These problems often are helped by the use of orthotics.

When patients are selected properly, reduction of spasticity helps to improve function and facilitates acquisition of daily living skills. Although much of the postoperative assessment has been subjective rating of muscle tone and function, more objective evaluations have been performed recently. Peacock et al. followed a group of 60 rhizotomy patients and found the best results in those children who were purely spastic and had more involvement in the lower extremities than in the upper extremities. Independent side sitting and locomotion also were favorable factors. Improved function accompanied decrease in tone. About one half of

children who had preoperative difficulty with upper extremity function benefited in this area. Some children progressed in breath control and articulation, which improved their speech.[55] Follow-up to 7 years revealed that muscle tone changes and functional improvements were maintained; however, most patients had residual weakness of lower extremity muscle groups.[71] Kinematic analysis of gait was performed in 16 of these patients, revealing gains in joint range of motion, stride length, and speed of walking.[65] Further gait studies by Gage, Cahan et al., and Perry et al. have confirmed these findings in postrhizotomy patients from different centers.[72–74] EMG obtained during quick stretch maneuvers revealed diminished or absent stretch reflexes and clonus following rhizotomy. Muscle firing patterns during gait, however, were not altered dramatically.[72] Peacock and Staudt[64] evaluated 25 rhizotomy patients using clinical scales, a hand-held force transducer, goniometric measures, and motion analysis. Repeated preoperative measures were obtained over time on a subset of patients. Reduction of muscle tone and increased passive and active range of motion were seen postoperatively. There were no significant changes during the preoperative testing period.

CEREBELLAR AND SPINAL STIMULATORS

Cerebellar and spinal stimulators attempted to augment or simulate the inhibition emanating from higher centers. Both underwent a wave of popularity, but follow-up reports indicated that the initial enthusiasm was premature. Subsequent studies of functional and physiologic parameters failed to demonstrate improvement with either method, and they no longer are utilized for spasticity.[52, 75–79]

ORTHOPEDIC MANAGEMENT OF SPASTICITY

Orthopedic procedures are designed to address the peripheral effects of spasticity rather than the spasticity itself. Since the musculoskeletal problems in cerebral palsy are acquired, rather than congenital, the orthopedic goals are to prevent deformity from developing and to reduce or correct established deformities.[80]

SOFT-TISSUE PROCEDURES

Too often in the past, soft-tissue assessment and surgery has been a matter of identifying tight structures and simply releasing or lengthening them. It was standard practice to release the hip adductors for inability to abduct greater than 30 degrees, and to release the psoas or rectus for a flexed hip posture.[81] Although such procedures continue to be of value in the treatment of cerebral palsy, we now recognize a difference between truly contracted (shortened) musculotendinous units and those spastic muscles that give the clinical impression of contracture, but in fact are only dynamically shortened. Due to the dynamic nature of spasticity, the clinician may be surprised to learn on examining a patient under anesthesia that a structure he intended to lengthen, such as the Achilles complex, seems relatively loose. This laxity could not be appreciated in the clinic, where the patient walked on his or her toes and the ankle could

not be brought from plantarflexion to a neutral position. The question then becomes one of whether the risk of overlengthening should be accepted in order to compensate for the clinical appearance. As expected, EMG studies preoperatively and postoperatively do not show any changes in a spastic muscle pattern, even when the associated tendon has been lengthened. In lengthening a tendon, the relative position of the joint may be changed, but the length of the muscle fibers themselves is not altered.[82] The arc of force generation merely is shifted. The lengthening of a tendon does not render a muscle less spastic, but may result in functional weakness. This is an undesirable side effect of such treatment. For example, overlengthened heel cords result in crouching, rather than simply the elimination of toe-walking. Lengthened hamstrings may accentuate an unrecognized hip flexion posture, or reinforce a stiff knee gait as a result of rectus femoris spasticity.[83, 84] If the patient's crouch is due to poor balance or weak calf muscles, the ability to ambulate may be compromised rather than enhanced. Neurectomies, chemical blocks at a motor point, or inhibiting substances placed directly into a muscle belly all relieve spasticity, but at the price of weakness. Thus, such procedures may result in adverse consequences. When these procedures are contemplated, it is particularly important to have an idea of the volitional strength underlying the contractures or spasticity, so that enough strength and control will remain to make such releases, transfers, and antispasticity attempts worthwhile. This is one of the greatest challenges facing the clinician: not simply identifying what is tight, but determining what the child really will look like if the tightness is removed.

Should any musculotendinous unit be lengthened at all and, if so, how should the lengthening be effected? In contrast to surgical lengthening, muscles can be altered in terms of the number of sarcomeres by chronic stretching forces usually applied by casts. In fact, there is some suggestion that the tendon itself can be elongated in growing animals through casting.[85, 86] Conversely, constant contractile forces secondary to spasticity result in a chronic loss of sarcomeres. From a theoretical standpoint, the ideal treatment might involve relieving the spasticity, and then restoring muscle length by stretching. In practice, milder forms of true tissue contractures can be stretched out, but for more severe contractures, this form of treatment may not be feasible. Sometimes, the musculotendinous unit can be stretched fairly readily, but in the presence of continued spasticity, recurrence is seen quickly when the cast is discontinued. Bracing can potentiate the effect of serial casting in some cases, but there still will be patients for whom casting seems futile in retrospect. Though stretching and inhibitory casts still are utilized frequently,[87–89] the use of inhibitory casting to relieve spasticity per se seems to have lost some of its initial luster.[90, 91] Similarly, orthoses are useful to maintain corrections once they are obtained by stretching casts or surgery, or to provide a more stable platform for ambulation when the associated joint can be positioned properly passively, but they offer no real effect on underlying spasticity.

In addition to considering the length of the musculotendinous units, consideration must be given to their function, in terms of both strength and timing. If the muscle is not active at the proper time, either abnor-

mal cocontraction results or the patient appears weak and unable to accomplish a given task volitionally. Many children move in synergistic patterns of mass flexion and extension. In order to augment or balance muscle forces, it frequently is possible to transfer a muscle contracting in the appropriate phase to reinforce the activities of a compromised agonist. In patients with equinovarus, the transfer of a posterior tibialis muscle that is active in swing to the dorsum of the foot is an example of this principle, as is the transfer of the flexor carpi ulnaris to the dorsal wrist extensors. For optimal planning of such transfers, dynamic EMGs are particularly useful.[92–95] Generally, if muscles can be shown to be abnormally active in an alternate phase of gait (i.e., the muscle exhibits a phase shift), they can be transferred to a site at which that timing can be put to best advantage as an agonist of joint motion. When the muscle is continuously active, it can be split to provide balanced pull throughout the gait cycle. Split anterior and posterior tibial tendon transfers are examples of this approach.[96, 97] At the knee, EMG analysis has been helpful in defining the role of transfer of the rectus femoris posterior to the joint line. With this method, as opposed to simple release, knee flexion and limb clearance in the swing phase of gait can be enhanced.[84] Although modern technology has provided us with sophisticated gait analysis tools, with experience and logic, most decisions still can be made clinically, without the need for expensive kinetic or kinematic analyses.

BONY PROCEDURES

In contrast to the controversies associated with soft-tissue techniques, the use of bony procedures in addressing structural deformity is on a firmer foundation. These procedures are performed at a later age, and generally hold up better against unbalanced muscle forces than do simple temporizing soft-tissue procedures. Although there remain some questions of efficacy with respect to individuals with severe spastic quadriplegia,[98] the use of spinal fusion techniques for progressive scoliosis is well established for patients with lesser degrees of involvement. Untreated, such a deformity can lead to loss of an erect posture and balance in ambulatory patients, loss of sitting balance and endurance in nonambulatory individuals, and, in severe cases, a compromise of cardiopulmonary function.[99] Segmental spinal fixation appears to offer some advantages in terms of achieving correction and managing patients postoperatively.[100, 101] Fusion to the sacrum often is performed in nonambulatory patients when pelvic obliquity is present.

Hip subluxation also is a frequent indication for treatment in spastic children. Many authors believe that, with early proper soft-tissue release, progressive hip subluxation may be preventable.[102–107] For the young adult, traditional methods of femoral varus derotation osteotomies,[108] acetabuloplasties, or a combination remain favored treatments.[81, 109, 110] For the more mature child with an unrecoverable painful dislocation, resection arthroplasty offers some hope for pain relief.[111, 112] Occasionally, a patient's overall situation will justify joint replacement arthroplasty.[113]

In patients in whom the hips are stable and balanced, but for whom an internal rotation gait appears to be caused by excessive femoral anteversion of at least 45 degrees, supracondylar derotational osteotomy is of

help. In one series, the criteria for this procedure included a lack of knee contracture and the ability to achieve straight leg raising to at least 70 degrees.[114] Contracted medial hamstrings are an alternate cause of internal rotation gait, and should be sought and treated as necessary.[115]

For recalcitrant foot and ankle deformities, aside from the soft-tissue balancing approaches, a Grice or other subtalar fusion procedure for instances of marked equinovalgus is very popular,[116] but perhaps not as efficacious as originally believed.[117, 118] Calcaneal osteotomies for residual fixed varus or valgus deformities of the ankle, and triple arthrodesis can be of value in properly selected patients. Frequently, soft-tissue and bony procedures are combined for optimal treatment. Although underlying spasticity may go unabated after these types of procedures, the fact that they generally are performed in older children when the rate of growth is lessened means that they have a better chance of enduring over time. Undoubtedly, if the various methods of managing the underlying spasticity itself prove fruitful, these procedures will be even more successful when indicated and carried out in conjunction with a spasticity reduction program.

RELATION OF SELECTIVE POSTERIOR RHIZOTOMY TO TRADITIONAL ORTHOPEDIC SURGERY

In our experience, even when an alternative such as selective posterior rhizotomy proves highly efficacious, traditional procedures such as muscle transfer and osteotomy are not eliminated from consideration. Approximately one half to three fourths of patients still require some form of orthopedic intervention. The final result is a better one than could be obtained with either approach alone. Orthopedic treatment plans often are modified following the reduction of spasticity. Casting may suffice for residual deformity as opposed to surgical release. When spasticity is diminished, the orthopedist is in a better position to assess residual true tissue contracture and thus deal with it surgically or nonsurgically. It is difficult to imagine how hip dysplasia or scoliosis, once established, can benefit from rhizotomy, but the effect of rhizotomy in preventing such problems in the first place eventually will have to be resolved. At the University of California, Los Angeles, when other considerations are put aside, we prefer to perform rhizotomy first and then deal with residual contractures 6 to 12 months later. If scoliosis fusion is contemplated, consideration of course is given first to rhizotomy. Selective posterior rhizotomy, however, rarely is performed in the presence of severe spinal deformity. Clearly, until the indications are defined better for both the neurosurgical and orthopedic approaches, close cooperation between the two specialties is mandated.

SUMMARY

The overall care of the cerebral palsied child continues to challenge a variety of disciplines in medicine. The management of spasticity per se is integral to, but distinct from, the management of complications of spasticity. The orthopedic surgeon is concerned primarily with the latter. The

evolution of treatments designed specifically to counteract spasticity, such as the various pharmacologic agents and procedures exemplified by selective posterior rhizotomy, of necessity will cause orthopedists to re-evaluate their own approaches. As our understanding of the basic lesion increases, and as the tools of management become ever more sophisticated, these children can look forward to less time wasted on ineffectual treatments and more resources directed toward techniques that can lead to truly enhanced overall function.

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