Child's Nerv Syst (1989) 5:337–346

Child's Nervous System CNS

Invited paper

Selective posterior rhizotomy for the treatment of spasticity: a review

Rick Abbott[1], Sandra L. Forem[2], and Marjorie Johann[3]

[1] Department of Neurosurgery, Division of Pediatric Neurosurgery, [2] Department of Neurology, Division of Pediatric Neurology, and [3] Rusk Institute of Rehabilitation Medicine, Department of Physical Therapy, New York University Medical Center, New York, NY 10016, USA

Abstract. The use of sensory rhizotomy has long been used for the treatment of spasticity. This review outlines the historical development of this treatment, the current surgical technique, and its physiologic rationale. Patient selection, postoperative treatment, and complications are also discussed.

Key words: Spasticity – Cerebral palsy – Surgery – Rhizotomy.

Over the past 2 years there has been an increasing interest by neurosurgeons in the use of selective posterior rhizotomy for the treatment of children with spasticity due to static encephalopathy (cerebral palsy). This interest has been generated by the work of Fasano and supported by the work of Peacock [18–20, 40, 41]. At present there is some confusion as to the origin and evolution of this procedure. To understand the rationale of this procedure fully, one must understand its evolution as well as the pathophysiology of the spasticity, and to perform this procedure one must not only understand the surgical technique but also how to select the patients and how to care for them postoperatively.

Physiology

In considering muscle tone and contraction it is best to start at the segmental level of the spinal cord. This is supported both phylogenically and ontogenically, for it is only as the CNS develops that the descending fiber tracts start to modulate the reflex circuitry. With further development, the descending fibers establish monosynaptic connections with alpha motoneurons to drive the fine motor movements of the distal musculature [52]. Conversely, it is spinal reflexive activity and its modulation that principally drives the larger, proximal musculature of the limbs [5].

At the segmental level, Ia sensory fibers make direct connections with alpha motoneurons and interneurons [5]. The interneuronal pool is also innervated by flexor reflex afferents (FRA) and high-threshold afferents (secondary muscle afferents, joint receptor afferents and cutaneous receptor afferents) [36, 46]. It is the interneuron pool's function to modulate the pattern and degree of responsiveness of the spinal cord's reflex circuitry. This pool, in turn, is modulated by the brain via descending fiber tracts, spinal segmental afferents and spinal propriospinal fibers (fibers that arise as collaterals of afferent neurons and course just outside of the spinal gray matter to travel to adjacent and distant segments) [29, 30, 35–38, 45].

Spasticity is an increase in muscle tone [14, 15]. Its hallmark is an increasing resistance to stretch as the speed of the stretching increases. When a spastic muscle is stretched, afferent signals from the muscle's spindle fibers travel up Ia fibers to the cord. Within the cord of a spastic individual, there is a variable loss of modulation of the interneuronal pool by the descending fiber tracts and, as a result, the incoming Ia signals and associated collateral propriospinal signals do not see the normal pattern of facilitory and inhibitory influences from the interneurons [4]. Similarly, to the degree that an alpha motoneuron is receiving facilitatory or inhibitory tone from a descending fiber, its responsiveness will be altered with the loss of that descending fiber. Finally, there have been studies to suggest that sprouting of neurons to fill synaptic sites vacated with death of other neurons may occur in an injured cord [34]. It is conceivable that with the death of a modulating descending fiber its synaptic site could be filled with either a modulating fiber of reverse polarity or by a primary afferent with stimulatory tone. This possibility of sprouting is particularly strong in the still-developing nervous system of the neonate.

Regardless of the type of alteration in the circuitry, what results is a spreading activation of muscle contraction in response to afferent stimuli. This spreading is reinforced via afferent gamma loop impulses from muscle spindles in antagonistic muscles that are undergoing pogressive stretching [15]. The end result is the hypertonic limb, with the pattern and degree of hypertonicity in the limb being a function of the location and extent of the CNS lesion. The expression of a lesion due to static encephalopathy (cerebral palsy) will differ from that due to postinfectious encephalopathy which arose after birth, and these will differ from the symptom

Offprint requests to: R. Abbott

complex seen with poststroke or posttraumatic encephalopathy that arose in adulthood. This variation undoubtedly contributes to the variation in the efficacy of treatments.

Historical development

The use of dorsal rhizotomies for the treatment of spasticity came into being as a result of the experience gained with its use in the treatment of unremitting limb pain. In September 1888, a neurologist, Dr. Charles Dana, first suggested dorsal rhizotomy for the treatment of limb pain in a letter to Dr. Robert Abbe [1]. In December of that same year, Dr. Abbe performed his first rhizotomy to treat an individual suffering from neuralgia of the arm. At about the same time, a British surgeon, William H. Bennett, reported using dorsal rhizotomies to treat painful spasms in an individual and reported success in alleviating the pain [7].

It was also during the late nineteenth century that Sherrington was developing his decerebrate cat model to study spasticity. As a corollary to his study, Sherrington performed dorsal rhizotomies, deafferenting a limb that had previously been rendered spastic by a midbrain lesion [47]. What he found was that this subgroup no longer exhibited spasticity in the deafferented limb. Instead, their deafferented limbs became flaccid.

In 1905, in an address to the New York Neurological Society, Spiller stated:

> Where athetosis exists the muscles are in a state of greatly increased tonicity and often are abnormally well developed and abnormally strong. Theoretically, the proper procedure might be to cut the posterior roots of the affected limbs, the number to be cut depending on the condition in each case; but this is always a serious operation and the results have at times been unexpectedly grave [51].

Shortly thereafter, Foerster published a series of papers considering the theoretical merits of posterior rhizotomies for the treatment of spasticity as well as the technique to be used. Additionally, in these papers he reported on five cases treated by Professor Teitz.

In short order, several papers appeared outlining individual experiences with this operation for the treatment of spasticity [2, 12, 23]. Described in these papers were individuals with pure spasticity or individuals with spasticity and athetoid movement disorders. The etiology of these symptoms varied: birth injuries, meningoencephilitis, trauma with cord transection, stroke. Not surprisingly, the treatment results varied from complete success to absolute failure. The overall impression was, however, favorable.

In 1913, Foerster published a review of his group's experience with this procedure as applied in the treatment of pain and spasticity [21]. In this paper the results of 159 cases were discussed; 88 cases of congenital spastic paraplegia were reviewed. It was Foerster's opinion that the latter subgroup responded particularly favorably to posterior rhizotomy, and he felt that this was due to a greater number of pyramidal fibers being preserved in the cases of congenital spastic static encephalopathy compared to spinal spastic paraplegia. In his studies, Foerster believed that it was important to preserve quadriceps spasticity, viewing this as useful spasticity since it supported knee extension while standing. Initially, he simply preserved the L4 dorsal root in attempting to preserve quadriceps tone. Later, he began to use intraoperative electrical stimulation of the dorsal roots while monitoring the evoked responses in the legs, seeking to determine which roots were triggering the quadricep's hypertonicity. This appears to be the first report of intraoperative stimulation being used to direct the surgical lesioning.

While spasticity due to supraspinal injury seemed very amenable to posterior rhizotomy, complete sensory rhizotomies frequently resulted in the side effects of sensory ataxia and hypesthesia with resultant stasis ulceration. This, coupled with the variation in results experienced by various authors, resulted in the procedure falling into disfavor after World War I [42]. In 1948, Freeman and Heimburger published a series of papers outlining a treatment scheme for patients plagued by spasticity due to spinal cord injury. For patients with dense or complete cord injury and resultant spastic paraplegia, anterior rhizotomy was advocated but in "... a somewhat larger group of patients ... irrevocable procedures do not seem necessary or justified" [24]. For this group they applied peripheral nerve sectioning and, in two cases, dorsal rhizotomy, cutting the T10 through S1 dorsal roots. Both experienced relief of adductor spasticity, but flexor spasticity persisted, with the authors stating that overall: "the results were disappointing." They conjectured that there had been insufficient deafferentation despite the breadth of their lesioning.

In 1970, Kottke presented a small series of spastic athetoid patients who were experiencing troublesome spasms throughout their limbs in response to tonic neck reflexes. For these patients he had performed dorsal rhizotomies of the first three cervical roots, resulting in improvement in hand function due to a lessening in spasms elicited by the tonic neck reflexes [32]. Heimburger, aware of this presentation, applied the technique to a group consisting of eight spastic quadriparetics and seven athetoid quadriparetics with associated spasms and spasticity [26]. He found "... some encouraging improvements" in tone, with only two failing to respond (one a spastic quadriparetic and one an athetoid). Seven of 15 patients experienced improvement in their paraspinal hypertonicity with 14/15 stabilizing their progressive scoliosis. Of the spastic quadriparetics 5/8 experienced improvement in hypertonic muscles outside the cervical segmental levels while in the athetoid group, only one experienced improvement. However, for whatever reason, there has been no further discussion of this in the literature.

Meanwhile the technique of sensory rhizotomy had been evolving in Europe. In Montpellier, France, Professor Gros and his students worked with modifications of the surgery in an attempt to limit the side effects. In 1973 at the International Congress of Neurological Surgery in Tokyo, Gros presented his experience in treating 50 individuals (18 infantile spastic diplegics, 14 post-traumatic spastic paraplegics, and 18 spastics with a degenerative myelopathy), using a modification of "Foerster's operation" [25]. In his modification, one of five rootlets of a given root were not cut. The

results were described as effective in reducing or suppressing hypertonicity.

In a later presentation, his group discussed a refinement that allowed them to decide which rootlets would be selected for cutting [43]. The group divided muscle hypertonicity into two varieties: "useful" and "harmful." In the useful category was abdominal, quadriceps, gluteus maximus and gastrocnemius muscle spasticity while in the harmful category was hip flexor and adductor and soleus muscle spasticity. During surgery, the dorsal rootlets near their root entry zone in the caudal cord were stimulated, and a map of evoked motor activity in the legs was made. The rootlets were cut that caused activity in muscles with harmful spasticity. They found that this not only resulted in a decrease in the tone of the muscles innervated by motoneurons from the rootlet's segment, but also in a decrease in tone in muscles outside the lesioned segment: e.g., upper extremity effects. It was found, however, that this technique was not completely successful in preserving proprioception [22].

One direction taken by Sindou, a student of Gros, was to move the lesioning to the dorsal root entry zone [48, 50]. Work done by Sindou in the late 1960s confirmed that in humans, as in animals, there was a segregation of fiber types in the dorsal rootlet as they approached the root entry zone [49]. This segregation was in preparation for the divergence of the various fiber types toward their respective targets within the spinal cord. Sindou initially advocated lesioning the spinal cord just ventral to the dorsal root entry zone, with the incision thus severing the smaller myelinated and unmyelinated fibers while preserving the larger myelinated fibers destined for the dorsal columns. It should be noted that Ia fibers involved in reflex circuits course between the proprioceptive and pain fibers into the dorsal horn toward their synapses with interneurons and alpha motor neurons in the intermediate and ventral gray zones. As Sindou gained experience lesioning patients with primary complaints of pain, he also gained experience with patients with concomitant painful spasms. In short, he found his technique to be not only effective in the treatment of pain but also of spasticity. This information, both anatomical and clinical, was later useful to investigators seeking to refine the lesioning process at the level of the posterior roots.

Fraioli in 1977 published an article that took note of Sindou's work, stating "... it is not necessary to cut the posterior rootlets completely to interrupt the Ia fibers" [22]. He felt that "... the aim of any operation for spasticity on the posterior roots, that is, the interruption of the afferent arcs of the stretch reflex, can be achieved by partially cutting the posterior rootlets." In his operation, one-half to two-thirds of each rootlet was cut about 1 mm from the dorsal root entry zone, with the direction of the cut being dorsomedial to ventrolateral. In those with "tonic spasticity" or spasticity unaffected by limb movement, 9/13 were moderately to markedly improved, while of those with "phasic spasticity" or spasticity aggravated by limb movement, only 2/12 experienced moderate improvement. There was no improvement in the one dystonic patient operated upon.

Ouakine in 1980 wrote of a further refinement where he combined the experience of Fraioli with that of his former professor, Gros, and advocated partial sectioning of the rootlets deemed abnormal by peroperative stimulation, i.e., the rootlets that triggered contraction of muscles deemed harmfully spastic [39]. In 75 cases operated on over a 5-year period, 70% maintained the improvement seen in their muscle hypertonicity, with 22/75 cases experiencing suprasegmental improvement in their arms and 8/75 improvement in their speech.

While the Gros school of thought was seeking to limit the side effects of the procedure by using anatomical criteria (targeting rootlets within the segment of muscles labeled as harmfully spastic), Fasano and his pupils were taking a different approach [16, 18]. They noted, as had others, that the reflex circuits within a rootlet not only had segmental effects but also triggered muscle activity outside the rootlet's spinal segment in spastic individuals. Therefore, they set out to develop a technique for identifying the rootlets that cause this abnormal, multisegmental reflexive activity thought to be responsible for spasticity and mass spasms. They stimulated the dorsal rootlets about 1 cm caudal to the level of their entry into the cord while noting the patterns of EMG responses. A 1:1 pairing of muscle twitch to stimulus pulse regardless of the frequency of stimulation, with diffusion of muscle activity outside the stimulated rootlet's segment, and the after-discharge of muscles after cessation of stimulation were deemed abnormal [19]. Two mechanisms were thought to be at play in this group [6]. The 1:1 responsiveness of the muscle to the dorsal rootlet stimulation was felt to be due to a loss in the normal refractory period at the synapse of the monosynaptic reflex circuit (caused by a loss of presynaptic inhibition by descending fibers, facilitation by long loop reflexes, or increased excitability of the alpha motor neurons). The diffusion of muscle activity was felt to be due to activation of a polysynaptic reflex circuit by the afferent volley. Interestingly, they also found that activation of these polysynaptic circuits seemed to occur only at a current intensity above the threshold and that as the current was increased there was increasing activation of the polysynaptic circuits [6]. Clinically, the patients were reported to be doing well at a 2–7 year follow-up with no return of the spasticity [20].

Concern for the preservation of sphincter control led Peacock to move the site of this operation into the lumbosacral canal, as reported in 1982 [40]. This approach allowed reliable identification of the roots being operated upon. He subsequently used his modification on 60 children to treat their spasticity with good results, as discussed later in this paper. It was his reporting these results during the mid 1980s that regenerated interest in using rhizotomies to treat spasticity surgically.

Patient selection

Selective posterior rhizotomy is "selective" in more ways than one. Although the term refers to the intraoperative

selection of rootlets responsible for spasticity, it also alludes to the fact that not all individuals with spasticity should undergo this procedure. There are two broad categories of candidates [40]. The first consists of patients who are functionally limited by their spasticity, but have sufficient underlying voluntary power to maintain and eventually improve their functional abilities once the spasticity has been alleviated. Individuals in this first group are usually normal or near normal in intelligence and can actively participate in their daily therapy. The second category consists of nonambulatory patients in whom spasticity interferes with sitting, bathing, positioning, and generally looking after oneself. Frequently this degree of spasticity can cause a great deal of discomfort. Many individuals in this second group are quite handicapped intellectually. The goal for this group is to ease all aspects of daily living and to make the individual more comfortable.

Although spasticity or increased tone can be surgically diminished in any individual, this is clearly not always clinically beneficial. Examples of situations in which a selective posterior rhizotomy might be to the detriment of the patient include: a functionally ambulatory individual with significant weakness in the antigravity muscles of the legs who depends upon hypertonicity in the knee extensors to bear weight, or an individual with a progressive rather than static encephalopathy (such as seen in patients with leukodystrophy) in whom the underlying disease will not relent. There should also be concern when an individual exhibits signs of choreoathetoid dystonia since both Fasano and Peacock have reported a less favorable response to the surgery in individuals exhibiting this type of dystonia [20, 40, 41]. Our policy has been only to consider individuals with this type of mixed picture of spasticity and dystonia if the hypertonicity is of such a degree as to cause severe, painful hip dislocations and bony deformities that are beyond orthopedic repair owing to the hypertonicity. Finally, there are individuals whose spasticity is of such a limited degree with regard to its distribution that a less extensive procedure associated with a lesser degree of morbidity could accomplish the same goal. An example of this would be individuals exhibiting "toe walking" in whom an Achilles tendonotomy might suffice.

When evaluating patients for selective posterior rhizotomy, it is helpful to use a team approach so that all questions regarding the etiology of the spasticity, the functional impediments due to the spasticity, the individual's ability to participate in a therapy program, and the appropriateness of applying the rhizotomy are addressed. At New York University Medical Center (NYUMC), the team consists of a pediatric neurologist, a pediatric neurosurgeon, an orthopedic surgeon, a pediatric physiatrist, a pediatric physical therapist, and occupational therapists. Each potential candidate is examined by every member of the team, and then the appropriateness of applying the procedure to the patient is discussed at a group meeting. Valuable adjuncts to this evaluation are preoperative X-ray films of the hips and spine, urologic and/or urodynamic assessments, kinematic motion analysis and, occasionally, other testing such as neuropsychological testing. Investigational interests may dictate other types of testing such as somatosensory-evoked potentials or MR imaging of the brain. Of course, each patient's general health and other associated medical conditions must be thoroughly evaluated prior to the completion of the selection process.

In most instances the patient will be a child, necessitating that a comprehensive history be supplied by the parents or guardian and that the previous medical records are reviewed in detail. Careful attention is given to the patient's birth, developmental and family histories, looking for a history confirming an injury to the CNS that occurred at birth and resulted in retarded physical development. It is also important to ascertain what types of treatments (e.g., surgery) have been performed in the past and that there has been a previous commitment to physical therapy, as the postoperative therapy needs are generally extensive. Care must be taken in educating the patient and family as to what can realistically be accomplished with the surgery and at what price with regards to transient loss(es) in function. Postoperative therapy needs are also discussed.

The patient's examination can be divided into two broad parts, the physical examination and the functional examination. In the physical examination the patient's hypertonicity is described as well as its consequences (restricted range at the joints, bone deformities, joint dislocations and spinal curvature). The functional part of the examination is aimed at the effect of the spasticity on trunk and limb function. This is done by evaluating the patient's ability to move through the motor developmental sequence. The examination in essence mimics the normal chronological sequence observed in growth and development of children who are not afflicted by physical, motor or psychological handicaps. The examiner must be cognizant of the influence of spasticity during all physical movement, specifically with regard to whether or not spasticity is aiding or hindering the movement being assessed. Equally important is the patient's functional strength or voluntary power under the influence of the spasticity. One of the most opportune evaluations of functional strength occurs during the transition from one position to another. The most important transitional maneuver to assess is the patient's ability to move repetitively from a deep knee squat to a standing position and back using graded muscle contraction and relaxation [17]. The ability to accomplish this task repetitively predicts adequate underlying voluntary power to stand without the splinting action of spasticity. Other predictors of ambulation such as protective reactions in the sitting and standing positions and persisting primitive reflexes can also be assessed [8].

In addition to the above examinations done on all patients, ambulators undergo observational gait analysis and, when possible, a complete motion analysis to include dynamic EMG recording and kinematic analysis.

Surgical technique

The evolution of the technique of dorsal rhizotomies to treat spasticity has been to save some of the afferent fibers and

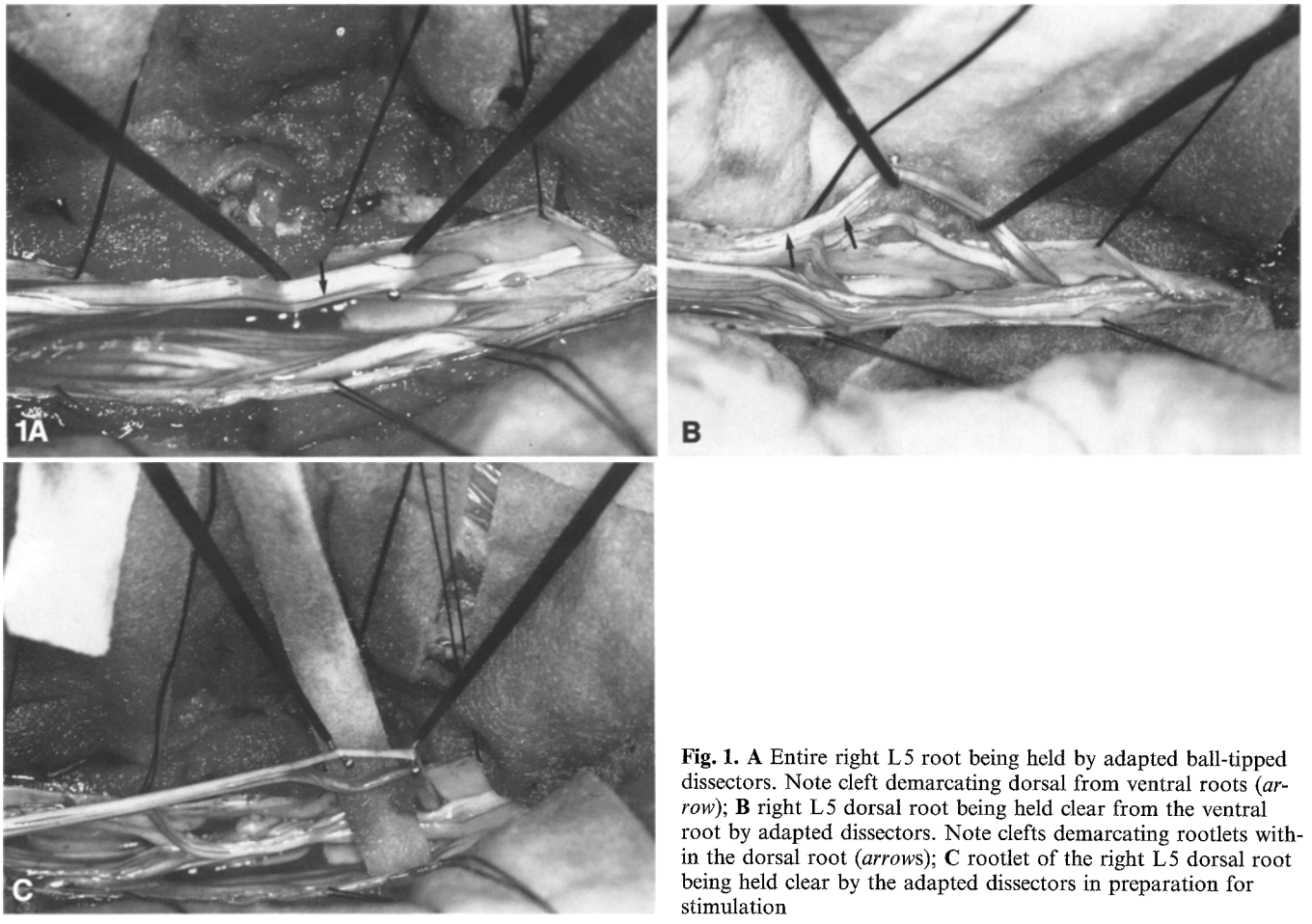

Fig. 1. A Entire right L5 root being held by adapted ball-tipped dissectors. Note cleft demarcating dorsal from ventral roots (*arrow*); **B** right L5 dorsal root being held clear from the ventral root by adapted dissectors. Note clefts demarcating rootlets within the dorsal root (*arrows*); **C** rootlet of the right L5 dorsal root being held clear by the adapted dissectors in preparation for stimulation

thus avoid the difficulties of sensory ataxia and stasis ulceration. After Gros demonstrated that these side effects could be avoided with partial sectioning of the roots, several authors published reports of technical modifications that sought to differentiate normally processed afferent input from abnormally processed afferent input [22, 25, 39, 48]. It was Fasano who presented the concept that, by monitoring the pattern of muscle response to the stimulation of afferent rootlets, one could differentiate rootlets with normally modulated synapses from those with abnormal modulation [18]. His observations on the types of response patterns seen form the bases for the surgery performed at most centers today [19].

Anesthesia must be given in a manner that preserves completely a muscle's ability to contract in response to motoneuron action potentials during the portion of the operation when afferent stimulation is occurring. Thus, at most, an ultrashort-acting muscle relaxant may be used at the beginning of the case to aid in intubation. After that, no relaxants should be used until all stimulation and lesioning has been completed.

The preferred site for the operation is currently the cauda equina, as it affords a secure identification of the roots at the point of exit from the dural sleeve. An L1 or L2–S1 laminectomy [44] or laminotomy is performed, and the dura is opened at midline to expose the L2–S2 roots at their point of exit from the dural sleeve. The dorsal roots are separated from the ventral roots using specially adapted microdissectors, which have been insulated except for their distal, right angled tips (Fig. 1). After the dorsal roots have been separated from their ventral partners, a current is delivered via these modified dissectors to the root (Fig. 2). The cathode is held 1 cm rostral to the anode. The initial round of stimulations seeks to determine the threshold of activity for the various roots. Typically, reflex circuits abnormally modulated in the cord have a lower threshold of activation [6, 19]. Once the threshold values have been obtained for a 1 Hz stimulus pulse, a 50 Hz stimulation train is then delivered while both the clinical and EMG pattern of muscle response is observed. It is important that all muscles in the legs be monitored either using palpation or EMG to enable the surgeon to appreciate those circuits which, when activated, respond in an abnormal manner [3]. Clinical palpation is used to determine abnormal muscle contraction, with the EMG being used as a "safety net." This is because we frequently see false-positive EMG activity due to volume conduction of electrical potentials from adjacent, contracting muscle groups.

Fasano described three types of influence on the reflex circuits at the spinal level in cerebral palsy patients [19].

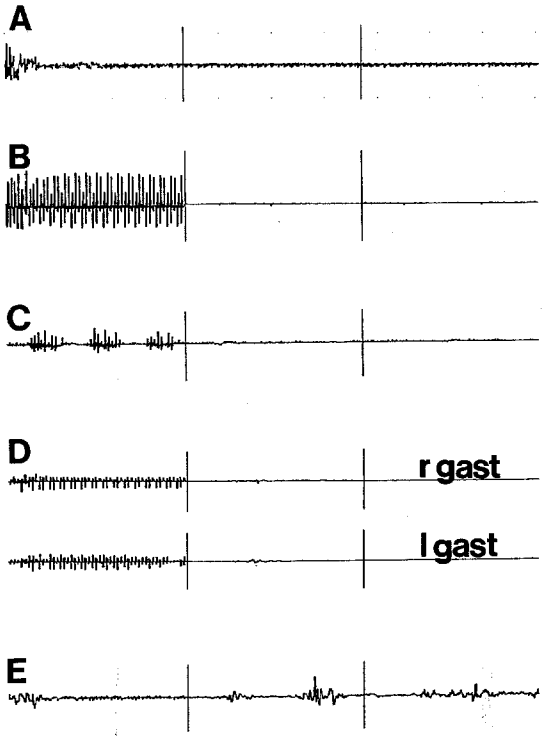

Fig. 2A–E. Patterns of EMG response to dorsal root and rootlet stimulation. **A** Normal response as described by Fasano et al. [18] with diminution in the muscle response after the first several stimulus pulses of the stimulation train. **B** Nonfatiguing of muscle response to stimulation train. **C** Waxing and waning in the amplitude of muscle response to the stimulation train. **D** Spreading of muscle activity to outside of the myotome of the nerve being stimulated – here to the contralateral gastrocnemius. **E** Sustained muscle contraction after cessation of stimulation

First, a "normal inhibitory activity" where a progressive decrease in the duration of muscle response to a 1-s stimulation train occurs as the frequency of the train increases above 5 Hz. At frequencies between 30 and 50 Hz, there is muscle contraction in response to only the first several pulses of the train, then no further muscle contraction. With this type of activity there is no muscle activity outside of the myotome of the root being stimulated. Second, there can be a loss of the normal inhibitory activity with a resultant one for one response in the muscle to the incoming stimulus pulses of the train regardless of the frequency (Fig. 2B). With this type of dysinhibition, one can see activity outside of the myotome of the segment being stimulated (so called diffusion), as well as sustained muscle contraction after cessation of the stimulus train (Fig. 2D). Also, one can see a fluctuation in the amplitude of the EMG recording during the stimulation (Fig. 2C). Finally, there can be an abnormal amount of inhibition of the reflex circuitry, with a resultant lengthening of the refractory period in the circuit poststimulation. Consequently, one sees a diminution in response to secondary stimuli in a train even at frequencies as low as 2 Hz. Sectioning of adjacent, abnormal rootlets frequently rectifies this abnormal inhibition. Most centers use motor activity outside the myotome of the spinal segment being stimulated and sustained motor activity after cessation of the stimulation as markers of abnormal roots and rootlets. The stimulus train typically used is 50 Hz for a 1 s duration. The current intensity will vary, based on the threshold values obtained at the outset, but typically are between 6 and 10 mA.

It is not unusual for the majority of the rootlets to respond abnormally. The question that then arises is what the safe number to cut is and how dense a lesion can be without creating a sensory deficit. Foerster noted that he could safely perform complete rhizotomies at two adjacent levels without creating a sensory deficit [19]. We have used this as a guide whereby we will only do a partial rhizotomy ($\leq 50\%$) at a level adjacent to two levels, which have received complete sensory rhizotomy. With this as a guide, we have witnessed a demonstrable sensory loss (S1 dermatome unilaterally) in only 1 of our initial 120 patients.

Treatment

Standard physical therapy for children with spastic cerebral palsy consists of first inhibiting spasticity by relaxation techniques and then facilitating normal movement to reeducate and strengthen motor performance. After an individual has undergone a selective posterior rhizotomy, muscle tone is significantly reduced throughout the extremities. Because of this decrease in tone, the majority of treatment time can be devoted to muscle reeducation and strengthening.

There are three components to the therapy plan for the post rhizotomy patient: muscle stretching to gain joint mobility and range, muscle strengthening to increase endurance, and reeducation to impart a better pattern of muscle use [17, 28]. Our 2 years of experience in treating these patients has left us with the impression that this treatment must be intense and consistent. Also, the individual's active movements must be limited outside of the therapy setting during the initial recovery period to avoid the old patterns of limb use that the therapist is working to alter.

The treatment philosophy at NYUMC is an eclectic approach, incorporating the principles of motor control, neurodevelopmental treatment, proprioceptive neuromuscular facilitation and the Rood approach to therapy [9, 11, 31, 53]. In the observation of the patient's movement skills, the therapist must consider the patient's motor development or lack thereof. Also, the individual's ability to control the timing of muscle contractions and the ability to balance the contractions of agonist and antagonist muscles must be evaluated. Further, the therapist must consider how best to facilitate or reeducate movement of the limb to incorporate the missing components of movement, and whether muscle contracture or biomechanical malalignment of the skeleton is affecting limb movement. This is particularly the case for patients whose treatment goals include improvement in ambulatory skills.

Patients who have moderate-to-severe muscle contractures preoperatively require a program of active, daily stretching in traditional stretch positions administered by a physical therapist. Trying to obtain increases in range by

Table 1. Summary of results at follow-up examination of patients of authors listed. Tone: 1, hypotonic; 2, normalized tone; 3, improved tone; 4, little or no change in tone. Functions of sitting and movement: A, loss in functional ability; B, little or no change in ability; C, improved function. A 1–5 year follow-up for Peacock's patients, 6–26 months for Laitinen's and 2–7 years for Fasano's

Author	Tone				Sitting			Recip/walk			Total patients
	1	2	3	4	A	B	C	A	B	C	
Peacock et al. [41]	18	27	8			2	54	1[a]	14	45	56
Laitinen et al. [33]		4	4						4	4	8
Fasano et al. [20]		11	61	8		1	79		24	56	80

[a] No difficulty with standing preoperatively

having the patient perform functional activities that passively stretch the contracted muscles (such as bending only at the waist to tie the shoes is inadequate to achieve the desired range increases at the involved joint. In an aggressive stretching program, full range should be accomplished by 6–8 months. Children under 6 can be given full range of movement with this type of stretching, while with the older children and adults this task becomes increasingly difficult. If, after 6–8 months, full range has not been achieved, then thought should be given to tendonotomies. Once the desired range is achieved, the stretching program must continue to avoid losses in range that can occur during growth or due to impaired limb function. To reinforce the stretching program, the parents should do daily ranging of the limbs after bathing so as to maintain the range obtained in therapy. We do not, however, expect the parents to be aggressively stretching any muscle.

Muscle strengthening is tailored to encourage graded movements, which require the individual alternatively to contract the muscles in a concentric and eccentric fashion. Activities such as using a tricycle adapted for the patient's riding are used to increase endurance as well as strengthening.

Reeducation of the pattern of limb use is an important phase of therapy postoperatively. The key to minimizing the time spent in reeducation is allowing only normal patterns of limb use. We frequently control movement postoperatively by placing the individual in either a wheelchair or tricycle when not in therapy until the individual shows good control over his or her limb movements. Often individuals' balance skills will improve faster than their control in ambulation. This is especially the case for individuals who were ambulatory preoperatively. For these patients it is desirable to slow the speed of ambulation to allow the individual to concentrate on the gait pattern, and to accomplish this we frequently use assisting devices such as walkers or canes. As the individual's control over the limbs improves, therapy is advanced to walking with less assistance from devices.

In addition to "hands on" therapy, orthotic management is extremely important for the patients engaged in ambulatory training. All of our patients have required orthotics postoperatively to provide stability and control of the lower extremities. We have been impressed with the fact that control over the anterior tibialis during gait has been most difficult to develop. This is not surprising if one considers the history of disuse in this muscle owing to spasticity of the gastrocnemius. The lack of control over the anterior tibialis is most evident at the time of initial heel contact, and what results is a foot slap. As the patient gains in strength and in the ability to control the movement at the hip and knee joints, mobility can be introduced at the ankle using a hinged ankle-foot orthotic (AFO) as opposed a solid AFO. This allows the patient to work on dorsi flexing the foot while plantar flexion is blocked, thus avoiding the foot slap.

Results

Little is available in print reviewing the results of treating spasticity with selective posterior rhizotomies. Three papers published in the 1980s report on the results at three centers (Torino, Italy; Cape Town, South Africa; and Umea, Sweden) (Table 1) [20, 33, 41]. In an article published in 1980, Fasano et al. reported on the 2–7 year follow-up examinations of 80 patients with cerebral palsy who had undergone selective posterior rhizotomy to treat their spasticity [20]. In patients with hypertonicity but no muscle contracture, there was complete normalization in the muscle tone, which persisted at the follow-up examination. In patients who had contracture of the musculature associated with the hypertonicity, 91% had normal or near-normal muscle tone at reexamination. The patients who had dystonia in addition to hypertonicity did not improve to the degree that those without dystonia did.

In 1987, Peacock published the results of the 1–5 year follow-up examinations on the 60 children with spastic cerebral palsy on whom he had performed selective posterior rhizotomy [41]. Forty of his patients were spastic diplegics and all had high tone preoperatively. Postoperatively, 22 had normal tone and 18 were hypotonic at rest. Sixteen of his patients were spastic quadriplegics who were operated upon to ease the task of their caretakers. With these patients, he found that 9 were left with normal tone while 5 had a reduced but persisting hypertonicity. There was no mention of postoperative tone in 4 individuals with hypertonicity and associated choreoathetoid movement disorder, but the author in a personal communication has not advocated operating upon this subgroup. Finally, Laitinen published the results of using selective posterior rhizotomy to treat spasticity due to multiple sclerosis, nonspecific myelopathy, spinal injury and cerebral hemorrhage [33]. Four of nine experienced normalization in their tone while 5/9 regarded

their hypertonicity as "much diminished" in the follow-up examinations at 6 to 26 months postoperatively. Unfortunately, all of these reports suffer from the disadvantage of subjective grading of muscle tone, which reflects the current lack of a standardized tone assessment scale.

Sitting and walking are the most easily assessed functional abilities in a spastic individual. Thirty-one of 65 of Fasano's patients had a greater than 30% improvement in their sitting posture and in 8/65 patients, the sitting posture became "normal" (70%–100% improvement) [20]. Peacock reported that 39/40 diplegic patients experience an improvement in stability while in the side-sitting position (the other being normal preoperatively) and that 15/16 of the quadriplegics he operated upon improved in their side-sitting abilities postoperatively [41]. With regard to walking, 15/65 of Fasano's patients demonstrated >30% improvement while 7/65 had >70% improvement [20]. Of the 14 independent ambulators (not requiring assisting devices such as canes, crutches, or walkers) whom Peacock operated upon, 12 experienced an increase in stride length due to an increased ability to extend at the knee [41]. Five of eight of the dependent ambulators became independent, while one required long leg bracing postoperatively due to a loss of functionally important spasticity. Nine of thirteen who reciprocated while held preoperatively gained the ability to ambulate to some degree, as did all of the diplegics who failed to demonstrate an ability to move their limbs in a reciprocal pattern or to bear weight unsupported before surgery.

At NYUMC we have seen an 80% success in decreasing muscle tone to a point where it no longer limits voluntary movement in the limb based upon 6-month follow-up examinations of our initial 45 patients. We, too, have been impressed by the improved stability in the sitting position, and temporal distance gait analysis [27] performed on several ambulators showed an increase step and stride length with a maintenance of velocity, implying improved efficiency in walking [16].

Side effects

There has been little discussion of complications or side effects associated with selective posterior rhizotomies. Fasano noted a 5% incidence in sensory disorder, with half being temporary and half permanent (Table 2) [20]. He described this disorder as being a hypesthesia in a restricted area in one leg. One of his patients had transient difficulty with sphincter control, and 5% of his patients had transient, diffuse hypotonia of a profound degree. Laitinen reported that 2/9 patients undergoing the procedure experienced a decrease in pin-prick sensation of a slight but permanent nature, while 1/9 had a transient difficulty with bladder dysfunction lasting 3 months [33]. Peacock's patients experienced no early surgical complications, but one female experienced a progression of a lumbar pars interarticularis defect to frank spondylolisthesis [41]. None of his patients experienced post operative hypesthesia, but he did find that an "... occasional patient would complain of increased cutaneous sensitivity during the first few postoperative weeks."

Table 2. Summary of side effects experienced by authors/centers listed

	Peacock et al. [40]	Fasano et al. [18]	NYUMC
No. patients	60	80	120
Hypesthesia	Occasional	2.5%	<1%
Dysesthesis (transient)		2.5%	25%
Hypotonia	30%	5% (transient)	1% (transient)
Hypertonia			5% (transient)
Bladder dysfunction	11%	1.25% (transient)	10% (transient)
Ileus			10% (transient)
Aspiration pneumonia			4%
Bronchospasm (intraoperative)			3%

Cahan et al. investigated somatosensory-evoked potentials (SSEP) in 20 children with cerebral palsy who underwent selective posterior rhizotomies [10]. Nine of twenty patients had abnormal SSEP studies preoperatively in response to posterior tibial nerve stimulation, with 6 of these normalizing after rhizotomy, 3/11 with normal preoperative SSEPs showed abnormal patterns postoperatively. He failed to demonstrate sensory loss in any of these patients clinically.

This is a patient population that is at higher risk of having perioperative complications. Of the 120 patients who have undergone this surgery at NYUMC, 4 have experienced intraoperative bronchospasm, with 3 of these incidences being of sufficient severity to cause termination of the procedure. Common to these children was a history of reactive airway disease or a recent upper respiratory tract infection. Of the 120 patients, 5 had intraoperative aspiration, resulting in pneumonia; 2 of these cases required artificial ventilation with PEEP for at least 18 h after surgery. Frequently, these children had markedly enlarged gastric bubbles on abdominal X-ray films and were among the most spastic cases. In addition, several had had a prior history of hospitalization for pneumonia. Postoperatively, 25% of our patients experienced a transient dysesthesia while one individual reported a loss of pain sensation in the right S1 dermatome, which has persisted. Only 1 patient has experienced a transient hypotonia while 6 have experienced a transient hypertonia associated with severe incisional pain and spasms lasting for 48 h postoperatively. We have seen a 10% incidence of transient urinary retention requiring intermittent catheterization for up to 3 weeks and have also seen a 10% incidence of transient ileus, requiring NG decompression.

Of theoretical concern is late-onset scoliosis due to the extensive laminectomy/laminotomy being performed. The site of this surgery seems to be the key to there being no reports of kyphoscoliosis in children who have undergone

selective posterior rhizotomy. As reported by Yasuoka, the tendency to develop postlaminectomy scoliosis seems to be a function of the level at which the laminectomies are performed, and it was his finding that postoperative scoliosis after lumbar laminectomy did not occur in the three patients whose postoperative X-rays he reviewed [54].

Conclusion

There is much support in the literature for a judicious evaluation of this modification of sensory rhizotomy for the treatment of spasticity. It is important for those using this technique to remember that no procedure in the final analysis turns out to be an absolute cure [13]. Therefore, it is incumbent upon those of us performing this procedure that we do it in a protocol setting allowing for a uniform, rigorous evaluation of the patients outcome so that we can better identify candidates in the future and accurately predict their outcome. We at NYUMC feel that this type of study will ultimately identify a subgroup of children with static encephalopathy who will be aided by this procedure.

References

1. Abbe R (1896) Intradural section of the spinal nerves for neuralgia. Boston Med Surg J 135:329–344
2. Abbe R (1911) Resection of posterior roots of spinal nerves to relieve pain, pain reflex, athetosis and spastic paralysis – Dana's operation. Med Rec 79:377–381
3. Abbott R, Johann M, Spielholz N, Epstein F (1989) Selective posterior rhizotomy and spasticity; relationship between intraoperative abnormal muscle contractions and the tone examination. In: Park TS (ed) Management of spasticity. Hanley & Belfus, Philadelphia (in press)
4. Ashby P, Verrier M, Lightfoot E (1974) Segmental reflex pathways in spinal shock and spinal spasticity in man. J Neurol Neurosurg Psychiatry 37:1352–1360
5. Baldissera PW, Hultborn H, Illert M (1981) Integration in spinal neuronal systems. In: Brookhart JM, Mountcastle VB (eds) Handbook physiology, sec 1: The nervous system, vol II: Motor control. American Physiology Society, Bethesda, pp 509–595
6. Barolat-Romana G, Davis R (1980) Neurophysiological mechanisms in abnormal reflex activities in cerebral palsy and spinal spasticity. J Neurol Neurosurg Psychiatry 43:333–342
7. Bennett WH (1889) Acute spasmotic pain in the left lower extremity was completely relieved by subdural division of the posterior roots of certain spinal nerves, all other treatment having proved useless. Med Chir Trans 79:329–348
8. Bleck EE (1987) Orthopaedic management in cerebral palsy. (Clin Dev Med, no 99/100) Blackwell, Oxford; Lippincott, Philadelphia
9. Bobath K (1980) A neurophysiological basis for the treatment of cerebral palsy. (Clin Dev Med, no 75) Lippincott, Philadelphia; S.I.M.P. with Heinemann, London
10. Cahan LD, Kundi SM, McPherson D (1987) Electrophysiologic studies in selective posterior rhizotomy for spasticity in children with cerebral palsy. Appl Neurophysiol 50:459–460
11. Carr JH, Shepherd RB, Gordon J (1987) Movement science: foundations for physical therapy in rehabilitation. Aspen Systems, London
12. Clark LP, Taylor AS (1910) The new treatment of spastic paralysis by resection of posterior spinal nerve roots. NY Med J 91:215–220
13. Cooper IS, Riklan M, Amin I (1976) Chronic stimulation in cerebral palsy. Neurology 26:744–753
14. Dimitrijevic MR (1985) Spasticity. In: Swash M, Kennard C (eds) Scientific basis of clinical neurology. Churchill Livingstone, Edinburgh, pp 108–115
15. Dimitrijevic MR, Nathan PW (1967) Studies in spasticity in man. Brain 90:1–31
16. Dzialga MJ (1988) Early ambulation patterns in cerebral palsy children who have undergone selective posterior rhizotomies. Master's thesis, New York University, NY
17. Elk B (1980) Preoperative assessment and postoperative surgical occupational therapy for children who have undergone a selective posterior rhizotomy. S Afr J Occup Ther 14:49–50
18. Fasano VA, Broggi UG, Barolat-Romana G (1977) New aspects in the surgical treatment of cerebral palsy. Acta Neurochir [Suppl] 24:53–57
19. Fasano VA, Barolat-Romana G, Zeme S, Sguazzi A (1979) Electrophysiological assessment of spinal circuits in spasticity by direct dorsal root stimulation. Neurosurgery 4:146–151
20. Fasano VA, Broggi G, Zeme S, Lo Russo G, Sguazzi A (1980) Long-term results of posterior rhizotomy. Acta Neurochir [Suppl] 30:435–439
21. Foerster O (1913) On the indications and results of the excision of posterior spinal nerve roots in men. Surg Gynecol Obstet 16:463–475
22. Fraioli B, Guidetti B (1977) Posterior partial rootlet section in the treatment of spasticity. J Neurosurg 46:618–625
23. Frazier CH (1910) The treatment of spasticity and athetosis by resection of the posterior roots of the spinal cord. Surg Gynecol Obstet 11:251–263
24. Freeman LW, Heimburger RF (1948) The surgical relief of spasticity in paraplegic patients. II. Peripheral nerve section, posterior rhizotomy and other procedures. J Neurosurg 5: 556–561
25. Gros C, Frerebeau PH, Kuhner A, Perez-Dominguez E, (1973) Technical modification in the Foerster operation. Selective posterior lumbar root section. The results of 18 years of practice. 5th International Congress of Neurosurgery, Tokyo 1973
26. Heimburger RF, Slominske A, Griswold P (1973) Cervical posterior rhizotomy for reducing spasticity in cerebral palsy. J Neurol Sci 39:30–34
27. Holden M, Gill K, Magiozzi M (1984) Clinical gait assessment in the neurologically impaired. Phys Ther 64:35–40
28. Irwin-Carruthers SH, Davids LM, VanRensburg CK, Magasiner J, Scott D (1985) Early physiotherapy in selective posterior rhizotomy. Fisiotherapie 41:44–49
29. Jankowski E, Lundberg A, Stuart D (1973) Propriospinal control of last order interneurons of spinal reflex pathways in the cat. Brain Res 53:227–231
30. Jankowski E, Lundberg A, Roberts WJ, Stuart D (1974) A long propriospinal system with direct effects on motoneurons and on interneurons in the cat lumbosacral cord. Exp Brain Res 21:169–194
31. Knott M, Voss DE (1969) Proprioceptive neuromuscular facilitation. Patterns and techniques. Hoeber Medical Division, Harper & Row, New York
32. Kottke FJ (1970) Modification of athetosis by denervation of the tonic neck reflex. Dev Med Child Neurol 12:236–237
33. Laitinen LV, Nilsson S, Fugl-Meyer AR (1983) Selective posterior rhizotomy for treatment of spasticity. J Neurol Sci 58:895–899
34. Liu CN, Chambers NW (1958) Intraspinal sprouting of dorsal root axons. Arch Neurol Psychiatry 79:46–61
35. Lloyd DP (1942) Mediation of descending long spinal reflex activity. J Neurophysiol 5:435–458
36. Lundberg A (1964) Supraspinal control of transmission in reflex paths to motoneurons and primary afferents. In: Eccles JC,

Schade JP (eds) Physiology of spinal neurons. Elsevier, Amsterdam, pp 197–221
37. Lundberg A (1969) Convergence of excitatory and inhibitory action on interneurons in the spinal cord. In: Brazier MAB (ed) The interneuron. University of California Press, Los Angeles, pp 231–265
38. Nathan PW, Smith MC (1959) Fasciculi proprii of the spinal cord in man: review of present knowledge. Brain 82:610–688
39. Ouakine GE (1980) Le traitement chirurgical de la spasticitite. Union Med Can 109:1424–1443
40. Peacock WJ, Arens LJ (1982) Selective posterior rhizotomy for the treatment of spasticity in cerebral palsy. S Afr Med J 62:119–124
41. Peacock WJ, Arens LJ, Berman B (1987) Cerebral palsy spasticity. Selective posterior rhizotomy. Pediatr Neurosci 13:61–66
42. Pederson E (1969) Spasticity. Mechanisms, measurement, management. Thomas, Springfield, Ill, pp 91–98
43. Privat JM, Benezech J, Frerebeau P, Gros C (1976) Sectorial posterior rhizotomy, a new technique of surgical treatment for spasticity. Acta Neurochir 35:181–195
44. Raimondi AJ, Gutierrez FA, DiRocco C (1976) Laminotomy and total reconstruction of the posterior arch for spinal canal surgery in childhood. J Neurosurg 45:555–560
45. Rymer WZ (1984) Spinal mechanisms for control of muscle length and tension. In: Davidoff RA (ed) Handbook of the spinal cord, vols 2, 3. Dekker, New York Basel, pp 609–646
46. Scheibel AB (1984) Organization of the spinal cord. In: Davidoff RA (ed) Handbook of the spinal cord, vols 2, 3. Dekker, New York Basel, pp 47–77
47. Sherrington CS (1898) Decerebrate rigidity and reflex coordination of movements. J Physiol (Lond) 22:319–337
48. Sindou M (1972) Etude de la jonction radiculo-medullaire posterieure. La radicellotomie posterieure selective dans la chirurgie de la douleur. MD thesis, University of Lyon
49. Sindou M, Qudex CC, Baleydier C (1974) Fiber organization at the posterior spinal cord-rootlet junction in man. J Comp Neurol 153:15–26
50. Sindou M, Jeanmonod (1989) Microsurgical DREZ-otomy for the treatment of spasticity and pain in the lower limbs. Neurosurgery 24:655–670
51. Spiller WG, Frazier CH (1905) The treatment of cerebral palsies and athetosis by nerve anastomosis and transplantation. J Nerv Ment Dis 39:310–317
52. Stelzner DJ (1982) The role of the descending systems in maintaining intrinsic spinal function: a developmental approach. In: Sjolund B, Bjorklund A (eds) Brainstem control of spinal mechanisms. Elsevier Biomedical Press, Amsterdam, pp 297–321
53. Stockmeyer SA (1967) An interpretation of the approach of Rood in the treatment of neuromuscular dysfunction. Am J Physiol Med 46:900–926
54. Yasuoka S, Peterson HA, Laws ER, MacCarty M (1981) Pathogenesis and prophylaxis of postlaminectomy deformity of the spine after multiple level laminectomy: difference between children and adults. Neurosurgery 9:145–152

Received February 28, 1989