Childs Nerv Syst (2007) 23:981–990
DOI 10.1007/s00381-007-0379-5

SPECIAL ANNUAL ISSUE

Selective dorsal rhizotomy for spastic cerebral palsy: a review

Paul Steinbok

Received: 22 February 2007 / Published online: 6 June 2007

Abstract
Objective The purpose of this report is to review the historical development, current operative techniques, selection criteria, outcomes, and complications of selective dorsal rhizotomy (SDR) for treatment of spastic cerebral palsy (CP).
Materials and methods This review is based on a review of literature and personal observations.
Results SDR has evolved from the 1960s onwards into a standard neurosurgical procedure for spastic CP. There is much variation in the operative technique among surgeons with respect to the level of exposure, electrophysiological guidance, and extent of rhizotomies. Appropriate selection of patients for SDR requires determination that spasticity, not dystonia, is the major disabling hypertonia and that the lower limbs are maximally involved. Positive outcomes have been well demonstrated in the impairment, functional limitations, and disability dimensions, as per the National Center for Medical Rehabilitation Research Model of Dimensions of the Disabling Process. Complications have been relatively few.
Conclusions SDR is the procedure of choice for treatment of spasticity in spastic diplegic CP and in selected children with spastic quadriplegic CP. Optimal selection and outcomes are achieved using a multidisciplinary approach.

Keywords Spasticity · Cerebral palsy · Dorsal rhizotomy · Posterior rhizotomy · Selection · Outcome · Complication

P. Steinbok
Division of Pediatric Neurosurgery, Department of Surgery,
British Columbia's Children's Hospital and
University of British Columbia,
Vancouver, British Columbia, Canada

P. Steinbok (✉)
Division of Pediatric Neurosurgery,
4480 Oak St, #K3-159,
Vancouver, BC V6H 3V4, Canada
e-mail: psteinbok@cw.bc.ca

Introduction

A number of strategies, both medical and surgical, have been used to reduce tone in children with spastic cerebral palsy (CP). Therapeutic options include physiotherapy, occupational therapy, oral spasmolytics and anti-dystonic drugs, botulinum toxin injections, orthopedic procedures, and neurosurgical procedures. The most common neurosurgical procedures are continuous infusion of intrathecal baclofen (ITB) and selective dorsal rhizotomy (SDR). The purpose of this article is to review the current status of SDR in the treatment of children with spastic CP, based on a review of the literature and personal observations from an experience of more than 200 cases.

Mechanism

In spastic CP, damage to the cerebral hemispheres results in decreased descending input into the spinal interneuron pool, which leads to excessive alpha motor neuron activity and spasticity. The basis for dorsal rhizotomy is that the input into the spinal interneuron pool via the afferents in the dorsal roots has a net excitatory impact on the efferent output via the alpha motor neurons. Dorsal rhizotomy reduces the amount of excitation of the alpha motor neurons and thereby reduces spasticity.

Historical aspects

Lumbosacral dorsal rhizotomy for the treatment of spasticity in the lower limbs was first proposed by Foerster in the early 1900s [23]. The underlying basis for such a procedure was the ability of dorsal root section to relieve decerebrate rigidity created in a cat by a section of the brainstem. Foerster went on to report a series of patients in whom total sections of posterior nerve roots of L2 and L3, L5, and S1 with sparing of L4 were performed [24]. Included in his groups of patients were a number of children with CP and spastic diplegia. The results were generally good with marked improvement in spasticity and improvement in function in many patients. However, there were problems with the procedure: Firstly, there was some return of spasticity on long-term follow-up; secondly, there was some sensory loss, particularly with respect to proprioceptive sensation; and thirdly, the muscle tone in the limbs was sometimes so suppressed that patients who relied on their spasticity for support could be made worse functionally.

Foerster's technique was not reported by others until the 1960s [26], when Gros et al. [25] in Montpellier, France, modified the original procedure, as described by Foerster, and performed partial dorsal rhizotomies, with nonselective sectioning of 80% of each of the nerve roots from L1 to S1. Further refinements of the partial dorsal rhizotomy procedure followed in two different directions.

The Montpellier school advocated a selective partial rhizotomy procedure, wherein the extent of the rhizotomy was tailored to the clinical status of the individual patient [51]. Lower limb spasticity was categorized clinically into "disabling" versus "beneficial" spasticity, and the goal of the procedure was to relieve only "disabling" spasticity. Intraoperatively, electrophysiologic examination of the posterior rootlets was performed to identify the muscle groups primarily innervated by each individual posterior nerve rootlet. This allowed section of rootlets involved with muscle groups that were identified clinically to be involved with "disabling" spasticity while sparing those nerve rootlets innervating muscle groups that were identified as having "beneficial" spasticity. Initial reports on this procedure indicated improved results compared to the earlier results from simple partial rhizotomies [51], but long-term results have not been published.

The second approach, originally proposed in 1978 by Fasano et al. [20], involved the selection of the posterior rootlets to be cut on the basis of the functional electrophysiological results of intraoperative electrical stimulation and not on the basis of the clinical findings. Fasano et al. [22] stimulated lumbosacral posterior nerve rootlets in children with spastic CP at increasing frequencies and recorded responses from anterior roots and muscles. They identified distinct different patterns of responses to repetitive stimulation of the dorsal nerve rootlets at frequencies of 30–50 Hz. Stimulation of some rootlets produced a muscular contraction only with the first stimulus, and thereafter, there was relaxation during the remainder of the stimulation period. These responses were felt to be normal and indicated that these rootlets were inserted in spinal circuits having normal inhibitory activity. These rootlets were felt to be relatively uninvolved with the maintenance of spasticity. A second population of nerve rootlets could be identified where the response to repetitive stimulation was a sustained and synchronous activation of muscles. This was often associated with abnormal activation of other circuits, such that there was a spread of the muscle response to affect the contralateral lower limb, the upper limbs, or even the trunk and neck muscles. It was thought that these nerve rootlets were inserted into spinal circuits in which the normal inhibitory processes were lacking and were therefore involved in the maintenance of spasticity. This latter group of nerve rootlets would then be cut in an attempt to relieve the spasticity. The rootlets associated with exaggerated responses were divided, sparing the rootlets with more normal responses, and favorable results were noted [21].

The procedure done by Fasano was via surgical exposure of the region of the conus at the T12–L2 level. This surgical approach was modified by Peacock and Arens [47], who exposed the lumbar and sacral nerve roots at their exit foramina via laminectomies extending from L2 to the sacrum. This made it easier to identify very specifically the exact roots that were being stimulated and divided and helped to avoid one of the complications noted by Fasano namely, bladder dysfunction. The procedure was still electrophysiologically guided, using criteria, similar to those proposed by Fasano. The initial criteria, which were thought to indicate involvement in the spastic process, included a low threshold to a single stimulus, a sustained response to a 50-Hz tetanic stimulation at threshold, and spread of the response outside the spinal cord segment being stimulated. This type of SDR, in which the posterior rootlets are cut on the basis of the functional electrophysiological results of intraoperative electrical stimulation, became the predominant type of SDR, particularly in North America. However, the validity of the electrophysiologic concept underlying the Fasano/Peacock SDR and the importance of the electrophysiologic guidance for a good outcome have been questioned [13, 33, 35, 50, 58, 69]. Each posterior rootlet contains hundreds or thousands of nerve fibers, and except for the dorsal root entry zone, where there is spatial segregation of sensory axons according to size [56], there is no anatomical or physiological evidence to indicate that axons with a particular

distribution of central connections are segregated together in the posterior root more peripherally, which is where the rootlets generally are sectioned. Furthermore, if an 'abnormal' electrophysiologic response is observed with tetanic stimulation of a rootlet at threshold, it is not known whether this reflects the functional status of the majority of nerve fibers in that rootlet or merely the status of the axons with the lowest thresholds.

Skepticism about the importance of the electrophysiologic guidance in SDR was also stimulated, when it became clear that some of the original electrophysiologic criteria introduced by Fasano and adopted by Peacock were invalid, despite good clinical results. The original electrophysiological criteria, which were said to indicate involvement of the posterior rootlet in the spastic process, included (1) a low threshold for response to the single electrical stimulus, (2) a "sustained" response to the 50-Hz tetanic stimulus at threshold level, where "sustained" was defined as a response that persisted throughout 1 s of 50-Hz stimulation, and (3) spread of the response to the 50-Hz stimulation beyond the segmental level being stimulated. However, it has been shown that a low threshold to single stimulus and a "sustained" response to the 50-Hz stimulation occur in posterior rootlets of nonspastic children and therefore do not indicate involvement in the spastic process [13, 58].

As it was recognized that the original electrophysiologic criteria were invalid, different approaches to new electrophysiological criteria were developed in different centers. In some centers, reliance is placed on the pattern of the response to the 50-Hz stimulation, with an incremental or clonic pattern indicating involvement in the spastic process, while flat (square wave) or decremental responses, although "sustained" according to the original criteria of Fasano and Peacock, are considered to be normal [68]. In other centers, reliance is placed on the nature of the H reflex recovery curve, with an increased H2-to-H1 ratio indicating involvement in the spastic process [65]. Others place reliance on the extent of the spread of the response to the 50-Hz stimulation beyond the segmental level stimulated, particularly to the contralateral side or suprasegmentally into the upper limbs or the face [44, 58], and in some centers, combinations of these electrophysiological criteria are used.

Current status of SDR

Despite the skepticism about the importance of the electrophysiologic criteria, SDR has become accepted as a standard neurosurgical procedure for the treatment of spasticity associated with CP. There are significant variations between centers in the way the procedure is done, as demonstrated in a North American survey done in 1995 [60] and a more recent international survey done in 2004 (Steinbok, unpublished data from the International Society For Pediatric Neurosurgery survey). The operation is done either at the level of the conus or at the level of the exit foramina of the lumbosacral roots. Most commonly, SDR is done using variations of the surgical approach described by Peacock, via laminectomies or osteoplastic laminotomies from L1 or L2 to S1 [12]. With this technique, the root level can be easily determined, the dorsal root can be usually be separated readily from the ventral root at each level, the amount of each dorsal root cut can be easily tailored for the individual clinical situation if this is felt to be important, the spinal cord is not at risk of damage, and the procedure is readily and safely accomplished with loupes or no magnification. The disadvantages are that the skin incision is long, the muscle dissection is extensive, the laminae are cut at multiple levels, the ventral roots may be damaged during separation from the dorsal roots, and there is significant postoperative pain. On the other hand, a smaller number of surgeons prefer to do the procedure at the level of the conus, via one- or two-level laminectomies [44]. Localization of the tip of the conus can be achieved in most children with percutaneous ultrasound or after removal of one spinous process and before laminectomy, thus minimizing the extent of the skin incision and laminectomies. The procedure is technically more demanding, magnification with an operating microscope is required for safety, the root levels are not as easy to determine, it is more difficult to tailor the operation to the individual clinical situation, and the conus is at risk. Compared to the multilevel operation, the advantages are the small incision, small amount of muscle dissection, decreased number of laminae cut, less postoperative pain, and avoidance of the ventral roots, which are totally separate from the dorsal roots at the level of the conus.

Irrespective of the level at which the SDR is done, from 20 to 70% of each posterior root may be cut from L2 to S1, with most surgeons cutting between 50 and 70%. Often, relatively less of L4 is cut to preserve some quadriceps tone, which may be useful in allowing the child to ambulate rapidly. If there is marked spasticity of the ankle plantar flexors, partial rhizotomy of S2 is often done, and to try and avoid bladder dysfunction, usually less than 35% of the S2 posterior root is cut. Stimulation of the clitoris or penis and perianal area with monitoring of nerve action potentials in the S2 rootlets may be used to assist in determining which S2 rootlets to preserve.

The postoperative management varies between centers. In our institution, the child is returned to the neurosurgical ward from the operating room and is maintained for 48 h on an intravenous infusion of morphine, combined with oral or rectal acetominophen and diazepam. The child is main-

tained in a head-down position for 24–36 h and then is mobilized with the assistance of a physiotherapist. The child is discharged on the fourth or fifth postoperative day with temporary splints that have been made by an occupational therapist or orthotist. Thereafter, the child receives outpatient physiotherapy in their home community three times weekly for 3 months, then twice weekly for 6 months, before reverting back to the usual preoperative schedule, which is typically once per week. The physiotherapy program stresses strengthening exercises in addition to the usual stretching. Immediately after surgery, the children typically are hypotonic and appear weak. The tone and strength rapidly increase over the first 2 months postoperatively, and the need for orthoses changes during this time. Tone typically increases gradually over 6–9 months and then stabilizes at a level less than preoperatively. In some centers, children are mobilized more slowly and are discharged later, and sometimes, many weeks of in-hospital physiotherapy are provided. No restrictions are placed on the child's activities postoperatively.

Selection of patients for SDR

In selecting a treatment modality for a patient with spastic CP, it is critical for all parties involved, including the caregivers, to agree on the goals of treatment. The goals are improved motor function, increased mobility, increased independence, and, for the severely affected quadriplegics, increased ease of care. Relief of hypertonia or improved range of movement per se are not the goals but are the means to achieve the goals. To make the most appropriate recommendations, the management team must be familiar with the various options available, both neurosurgical and non-neurosurgical.

In the assessment of the child with spastic CP, one first has to determine the nature of the hypertonia. Children with "spastic" CP are typically referred to a neurosurgeon or specialized clinic for management of their spasticity, but it is important to recognize that the label of "spasticity" applied to these patients may or may not be correct. Clarification of the definition of the various disorders that might be demonstrated by children with "spastic" CP is important and has been published by the Task Force on Childhood Motor Disorders [54]. These children all have "hypertonia," which has been defined by the task force simply as "abnormally increased resistance to externally imposed movement about a joint" [54]. The hypertonia may be caused by spasticity, dystonia, or rigidity, individually or in combination. In children with CP, rigidity as seen typically in Parkinsonian patients occurs rarely, if ever, so that in practice, one has to determine whether the hypertonia is due to spasticity, dystonia, or a combination of both.

Spasticity is defined [54] as: "hypertonia in which one or both of the following signs are present:

1. Resistance to externally imposed movement increases with increasing speed and varies with the direction of joint movement
2. There is a rapid rise in resistance to externally imposed movement above a threshold speed or joint angle."

The second criterion defines one aspect of the spastic catch, which may be felt on examination. After the rise in resistance, there may be a rapid decrease in resistance if the externally imposed force is maintained, the so-called clasp-knife response.

Spasticity is part of the upper motor neuron syndrome and usually coexists with other motor symptoms and signs. These include hyperreflexia, clonus, reflex overflow, a Babinski response, weakness, and incoordination.

Dystonia is defined [54] as a "movement disorder in which sustained muscle contractions cause twisting and repetitive movements, abnormal postures, or both." Dystonia may cause hypertonia, but dystonia may be present in the absence of hypertonia.

Having determined the nature of the hypertonia, the next question is whether it is the hypertonia or some other problem, such as contractures, incoordination, or weakness, that is inhibiting the child's function or the ability of the caregivers to look after the child. The motor disorder in children with hypertonic CP is complex and is the result of a number of factors in addition to the effects of spasticity and/or dystonia. Weakness and incoordination are common. There usually is cocontraction of agonists and antagonists, excessive reflex spread to other muscle groups, and reduced selective motor control, defined as the impaired ability to isolate the activation of muscles in a selected pattern in response to demands of a voluntary posture or movement [55]. As the child gets older, contractures and deformities of the extremities often develop, particularly knee flexion and ankle plantar flexion contractures. There may be hip dislocations, rocker bottom feet, and spinal deformity, especially scoliosis, which may add to the motor dysfunction and discomfort and the difficulty in providing care. There is probably an element of impaired sensory perception that adds to the impairment of motor function.

It is important to find out from the parents or other caregivers what they think is holding the child back or making care difficult. It is instructive to hear how they think the child would benefit if the hypertonia but nothing else in the lower or upper limbs was reduced. If it is not felt that reducing hypertonia will improve function, the child is not a candidate for a hypertonia-relieving procedure.

If it is determined that treatment of the hypertonia is indicated, one then needs to decide what intervention would be most appropriate. In children with spasticity, in whom

the functionally disabling spasticity is predominantly in one muscle group, for example, the ankle plantar flexors or hamstrings, consideration is given to an orthopedic tendon-lengthening procedure, neurectomy, or botulinum toxin. In general, even when the functionally important spasticity affects one muscle group, there is usually spasticity throughout the lower limbs, and one can argue for doing a SDR or using ITB to decrease the lower limb spasticity more diffusely in this patient population. However, we opt for a simpler procedure if the functional results might reasonably be expected to be similar, and in this regard, it is useful for the neurosurgeon and orthopedic surgeon to have a cooperative, rather than competitive, working relationship.

If the child has hypertonia and might benefit functionally from relieving the hypertonia diffusely in the lower limbs, one considers options such as ITB or SDR. In choosing between these two options, one takes into account a number of basic facts. Firstly, SDR improves spasticity but not dystonia, whereas ITB can improve both spasticity and dystonia. Secondly, SDR primarily impacts spasticity of the lower limbs and has a minor impact on the upper limb tone, whereas ITB can reduce hypertonia significantly in both lower and upper limbs. In general, SDR is the procedure of choice for children with spastic diplegia and little upper limb involvement, between 3 and 8 years of age. This is a group of children, in whom dystonia is usually minimal. ITB is recommended for children with spastic quadriplegia, especially if improvement in upper limb function is a major goal, because it is possible to direct the subarachnoid catheter higher in the spinal canal, such that upper limb spasticity is reduced [6]. Furthermore, in the children with spastic quadriplegia, there is a high probability that a significant component of the hypertonia will be dystonia, which responds to ITB but not to SDR.

For children in whom SDR is being considered, the ambulatory potential must be assessed. An attempt is made to determine if reduction in lower limb spasticity might be detrimental to the child's ambulatory function, because the child is using the spasticity to support weight in the upright position. In some children, 3D gait analysis may be helpful because assessment of joint moments and joint powers may give some objective evidence of a potential benefit/risk ratio. ITB is sometimes proposed in preference to rhizotomy in children with spastic diplegic CP, when there is concern that spasticity is needed for standing or walking support [5]. The argument is that the dose of baclofen is titratable, and in the worse case scenario, if the result is unsatisfactory, the treatment, unlike SDR, is reversible. This concern about the possible loss of ability to stand or walk may be more theoretical than real. In our experience, children who are walking independently without a walker or crutches have enough underlying strength in the lower limbs, and no such child has lost the ability to walk or stand after SDR. Hence, we recommend SDR as the procedure of choice in this group of children. In younger children, the ability to crawl on knees and arms (four-point crawling) predicts a positive outcome after SDR. For more involved patients, it may be more difficult to determine how much underlying voluntary strength is present. The child's ability to rise in a slow graded fashion from the squatting position is assessed. If the child is able to do this with the examiner supporting the arms or upper body minimally, the probability is high that there is adequate underlying strength to support the child's weight if the spasticity is reduced. One clearly needs to be concerned about this issue of maintaining function not only in children with ambulatory potential but even in the more severely involved children with spastic diplegia or quadriplegia, in whom ambulation is not a realistic goal. Many of these children, who move around in a wheelchair, use their spasticity to assist in standing transfers. They may be worse functionally if that spasticity is reduced and they are no longer able to support their weight. This may not appear to be a major issue in the young child, who is easily lifted, but it attains increasing importance as the child gets older, heavier, and more difficult to lift.

In children with spastic quadriplegia, in whom the lower limb spasticity is disabling or making care difficult and in whom relief of upper limb hypertonia is not an important goal, either ITB or SDR may be considered. It is important to recognize that many of the quadriplegic children, who seem to have "spasticity" in the first 5 years of life, develop progressively more dystonia as they get older. If significant and disabling dystonia develops, ITB clearly is the appropriate treatment, and SDR is contraindicated. We therefore do not recommend SDR in spastic quadriplegic children until they are at least 8 years old, by which time, dystonia will generally have declared itself. There are no studies that compare SDR and ITB in children with spastic quadriplegic CP, but of all the patients in our experience who have had SDR, it is the spastic quadriplegic children who have had the poorest outcome [32]. In the spastic quadriplegic children, in whom either ITB or SDR might be considered as reasonable options, our practice had been to recommend SDR. However, recognizing that this is the population in whom we have had our only poor outcomes [32], we currently recommend ITB as the preferred option, all other things being equal. In these children with spastic quadriplegia, there are some disadvantages to ITB compared to SDR, which may result in SDR being chosen by the team as the preferred option: ITB requires continuing long-term management, requires that the child live close to a medical center with some expertise in dealing with problems associated with ITB, requires reoperation for battery failure even if everything goes well, and has more complications, which may require additional operations.

Another consideration, which is not important in our decision making but may be important in some jurisdictions, is that SDR is less costly than ITB [59].

At the completion of the assessment, it is critical that one ascertains what the expectations and hopes of the parents or other primary caregivers are and ensures that they are realistic expectations. This should be documented because almost all parents are hoping for a 'cure' for their child's motor problems or, at least, that their child will walk. Many have unrealistic expectations from the treatment that is proposed. These parents may not be happy even if the child improves functionally because the child does not achieve their expectations. In most cases, one can wait many months before treating the hypertonia, and this allows time for the parents to think about the recommendations that are suggested, especially if an operation is advised. Occasionally, when the hips of the child are dislocating because of excessive hip abductor spasticity, a more urgent intervention is recommended.

Outcomes

Outcomes after SDR have been well characterized and have been reviewed in 2001 according to the schema developed by the National Center for Medical Rehabilitation Research (NCMRR) for assessing treatment outcome in CP [63] (Table 1). According to this schema, outcomes are considered across five dimensions namely, pathophysiology, impairment, functional limitations, disability, and societal limitations (Table 1). The impairment, functional limitations, and disability dimensions in this schema correlate closely with the World Health Organization (WHO) Classification of Functioning, Disability, and Health [52] (Fig. 1). In the WHO classification, the negative terms "impairment," "functional limitations," and "disability" have been replaced by the neutral and more positive terms "body structure and functions," "activity," and "participation." In the review by Steinbok [63], each reported study was graded according to both the level of evidence as recommended by Sackett [53] (Table 2) and the classification system used by the Brain Trauma Foundation and the American Association of Neurological Surgeons in the development of guidelines for the management of severe head injury [10] (Table 3).

Table 1 NCMRR model of dimensions of the disabling process

Dimension	Description
Pathophysiology	The cellular and molecular processes of injury or disease pertinent to a particular condition
Impairment	Involves dysfunction of the organ system level resulting from a disease process or injury. Examples of impairment level outcomes include tone, spasms or strength
Functional limitation	Defines limitation to the set of skills required to perform specific activities, either of the whole body or body segments. Examples include limitations in gait, sitting, or manipulating objects
Disability	Defines difficulties in carrying out the role or function expected for an individual such as attending school, returning to work, partaking in age-appropriate recreation, or performing activities of daily living
Societal limitations	Represents the barriers placed by society, which limit full participation by people with disabilities. These barriers may be physical, economic or attitudinal. Examples include wheelchair unfriendly architecture, or inability to afford a power wheelchair

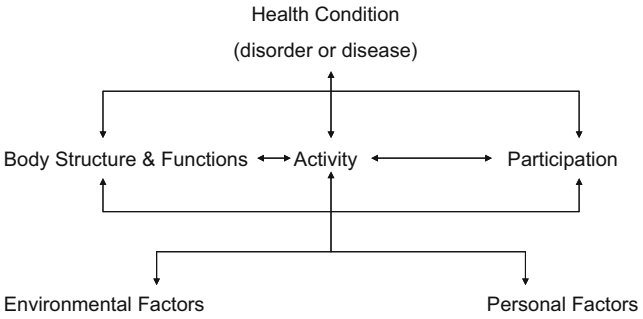

Fig. 1 World Health Organization 2001 International Classification of Functioning, Disability, and Health as per Rosenbaum and Stewart [52]

Based on this review, there is very strong evidence from three randomized controlled trials and other nonrandomized prospective studies that SDR results in improvements in lower limb spasticity (impairment dimension) [18, 19, 40, 63]. This is reflected in the electrophysiologic parameters of decreased spasticity namely, decreased lower limb H reflex-to-M response ratio and improved electromyography responses (pathophysiology dimension), for which there is also strong evidence of improvement in the short term. There is also strong evidence (Sackett Grade A) that there is an increase in the range of movement at the lower limb joints (impairment dimension) and either no change or improvement in lower limb strength (impairment dimension) after SDR [18, 19, 63]. There is a moderate degree of

Table 2 Categorization of levels of evidence according to Sackett [53]

Level	Description
Level I	Large randomized trails with clear-cut results (and low risk of error)
Level II	Small randomized trials with uncertain results (and moderate to high risk or error
Level III	Nonrandomized, contemporaneous controls
Level IV	Nonrandomized, historical controls
Level V	No controls, case-series only

Table 3 Classification of evidence according to the Brain Trauma Foundation and the American Association of Neurological Surgeons [10]

Class	Description
Class I	Prospective randomized trails
Class II	Studies in which data was collected prospectively, and retrospective analyses were based on clearly reliable data. These include observational studies, cohort studies, prevalence studies, and case control studies
Class III	Retrospective studies

certainty that these improvements in the impairment dimension are maintained up to 5 years after SDR and some weaker evidence that the improvements are maintained in the even longer term [63]. In ambulatory patients, there is strong evidence from studies using instrumented gait analysis for improved range of movement in the lower limbs during walking and increased stride length after SDR and moderate evidence for increased gait velocity [17, 63].

The effects of SDR on parameters in the dimension of functional limitations have been assessed by examining sitting ability, level of ambulation, changes on various nonvalidated motor function scales, and the validated Gross Motor Function Measure (GMFM). There is a moderate degree of certainty that SDR results in improved sitting ability in more than 70% of patients and improved level of ambulation in more than 50% of patients, who are not already independent ambulators before SDR [63]. One of the drawbacks of the studies on sitting ability and ambulation is that for almost all the studies, the outcome assessment tool has not been validated, thus detracting from the certainty of any conclusions about the outcome being studied. The most widely used validated tool to assess motor function has been the GMFM. Multiple studies, including three randomized controlled trials, have shown an improvement in GMFM after SDR, ranging from 3.2 to 12.1% in different studies [19, 30, 38, 39, 61, 67, 70]. Thus, there is strong evidence that there is an increased GMFM after SDR. However, in only two of the three randomized controlled trials was the improvement in GMFM statistically more than in the control group receiving physiotherapy only [61, 70]. In the other randomized controlled trial, there was no difference between the SDR group and the control group with respect to GMFM improvement alone [39]. In a meta-analysis of these three randomized controlled trials, there was a small but statistically significant improvement in the patients undergoing SDR plus physiotherapy compared to those having physiotherapy only [37].

In the disability dimension, there is good evidence from prospective case series, using validated assessment tools, either the Functional Independence Measure for Children or the Pediatric Evaluation of Disability Inventory, that there are improvements in self-care and performance of activities of daily living after SDR [9, 16, 34, 41, 43, 67]. These findings are supported by other prospective case series using less-validated outcome tools [63]. These outcomes have not been assessed in a randomized controlled trial.

There have been no studies assessing outcomes in the NCMRR dimension of societal limitations.

In addition to the impact of SDR on the lower limbs, there have been well-documented suprasegmental improvements, as initially reported by Fasano et al. [22], and there have been multiple more-recent reports of these suprasegmental phenomena. Improvements have been demonstrated most convincingly for upper limb function using a validated tool to measure upper limb function namely, the Quality of Upper Extremities Skills test [34] and the fine motor skills section of the Peabody Developmental Motor Scales test [42]. There is a single small study that suggests improvement in cognitive function [14].

One of the options for treatment of children with spastic CP is one or more orthopedic procedures. Thus, a reduction in the rate of orthopedic procedures after SDR can reasonably be considered a positive outcome. The few studies of this question indicate with moderate certainty that the rate of orthopedic procedures after SDR is approximately 65% [7, 11, 36]. It is probable that all patients who underwent SDR would have had orthopedic procedures had the SDR not been done. Hence, it can be argued that SDR led to avoidance of orthopedic surgery in at least 35% of patients. Furthermore, our experience suggests that in some children, improved function after SDR allowed consideration of an orthopedic procedure that would not have been indicated before the SDR. Thus, the 35% figure for avoidance of orthopedic surgery may be an underestimate if one is looking at equivalent goals for the SDR versus orthopedic surgery. There is weak evidence, from one retrospective study only, that patients in whom SDR was done between 2 and 4 years of age had a lower postoperative rate of orthopedic procedures than patients in whom the SDR was done at more than 4 years of age. One of the difficulties in interpreting these data is the variation in indications for orthopedic surgery among different orthopedic surgeons.

Complications

The complications of selective posterior rhizotomy have been few in most series [3, 62]. In at least one report, intraoperative bronchospasm was frequent, with an incidence of 8.3% [3]. This was attributed to increased airway reactivity in ex-premature children and was most common in children with spastic quadriplegia. With special precau-

tions, such as premedication with an H2 blocker, the incidence of this complication was reduced dramatically. In our center, the incidence of bronchospasm was less than 1%, even without special precautions.

Urinary incontinence is probably the most concerning problem. Transient urinary retention is frequent, with an incidence of between 1.25 and 24% [15, 20, 47, 62], but permanent urinary retention or incontinence is rare. In an attempt to avoid such complications, pudendal monitoring has been instituted in our center and many others [2], and the amount of the dorsal root of S2 cut is limited to less than 35%. It was to prevent urinary complications that Peacock and Arens [47] modified the original procedure described by Fasano et al. [20]. However, SDR is still done at the level of the conus in many centers, and urinary complications may be avoided by meticulous microsurgical techniques and section of no more than one third of the S2 dorsal root.

Transient dysesthesias, lasting up to a few weeks, have been reported in 2.5 to 40% of patients [3, 38, 62], but the reported incidence of permanent hypesthesia after SDR is very low, generally varying from 0 to 6% in different series, and usually being of no functional significance [1, 7, 21, 62]. The incidence of hypesthesia is probably underestimated because mild changes may not be reported. Furthermore, in younger children or those who are intellectually challenged, it may be impossible to determine mild sensory loss.

One of the concerns in children with significant spasticity affecting the hips is the complication of hip subluxation. With respect to hip subluxation, less than 20% deteriorated, and the vast majority of patients were stable (43–80%) or actually showed improvement (9–38%) in the amount of hip subluxation after SDR [28–30, 45].

The surgical procedure for SDR is done either at the level of the conus, via 1- or 2-level laminectomies or at the level of the root exit foramina, via multilevel laminectomies or laminotomies with replacement of the laminar flap. A concern with laminectomies or laminotomies in young children is the later development of back pain or spinal deformity [71]. Back pain, occurring weeks to years after SDR, has been reported in 4–7% of patients [4, 48, 62], but it is unusual for this to be severe enough to interfere with activities of daily living or to lead to hospitalization.

The issue of spinal deformity is more contentious. It has been recognized for a long time that children with spastic CP are at a higher risk of spinal deformity, particularly scoliosis, than the normal population [8], and the population at highest risk is the nonambulatory spastic quadriplegic patients. Patients with spastic CP have a fourfold higher incidence of spondylolysis of the fifth lumbar vertebra and an increased lumbar lordosis compared to the normal population [27]. A number of recent reports have suggested that SDR done via multilevel laminoplasties or laminectomies may increase the incidence of thoraco-lumbar scoliosis, kyphosis, and hyperlordosis and lumbosacral spondylolisthesis in these at-risk children with CP [31, 49, 57, 64, 66]. The possible increased risk of spinal deformities after SDR is noted primarily in the nonambulatory spastic quadriplegic population [57, 64], who currently tend to be treated with ITB rather than rhizotomies. It has been suggested that these potential complication can be reduced by doing the SDR via one- or two-level laminectomies at the level of the conus [46], but there is no evidence at present to support this hypothesis.

Conclusions

The management of the motor impairment in the child with hypertonic CP should be multidisciplinary and may involve a physiotherapist, occupational therapist, orthotist, developmental pediatrician, orthopedic surgeon, pediatric neurologist, and physiatrist in addition to the neurosurgeon. The neurosurgeon is usually asked for an opinion about the management of the hypertonia. It is important that he/she appreciates the various neurosurgical and non-neurosurgical interventions available for the relief of hypertonia. It is critical to recognize that the goal of treatment is not the relief of hypertonia per se but the improvement of function. The neurosurgeon should work as part of a team to identify the best management option to optimize the functional outcome. Furthermore, if a neurosurgical intervention directed at the relief of hypertonia is felt to be indicated, the best outcome is achieved with input from physiotherapists, occupational therapists, orthotists, and orthopedic surgeons in the postoperative period.

References

1. Abbott R, Forem SL, Johann M (1989) Selective posterior rhizotomy for the treatment of spasticity: a review. Child's Nerv Syst 5:337–346
2. Abbott R (1992) Complications with selective posterior rhizotomy. Pediatr Neurosurg 18:43–47
3. Abbott R, Johann-Murphy M, Shiminski-Maher T, Quartermain D, Forem SL, Gold JT, Epstein FJ (1993) Selective dorsal rhizotomy: outcome and complications in treating spastic cerebral palsy. Neurosurgery 33:851–857
4. Abbott R (1996) Sensory rhizotomy for the treatment of childhood spasticity. J Child Neurol 11:S36–S42
5. Albright AL (1996) Intrathecal baclofen in cerebral palsy movement disorders. J Child Neurol 11:S29–S35
6. Albright AL, Turner M, Pattisapu JV (2006) Best-practice surgical techniques for intrathecal baclofen therapy. J Neurosurg 104:233–239

7. Arens LJ, Peacock WJ, Peter J (1989) Selective posterior rhizotomy: a long-term follow-up study. Child's Nerv Syst 5:148–152
8. Balmer GA, MacEwen GD (1970) The incidence and treatment of scoliosis in cerebral palsy. J Bone Jt Surg Br Vol 52:134–137
9. Bloom KK, Nazar GB (1994) Functional assessment following selective posterior rhizotomy in spastic cerebral palsy. Child's Nerv Syst 10:84–86
10. Bullock R, Chestnut RM, Clifton G, Ghajar J, Marion DW, Narayan RK, Newell DW, Pitts LH, Rosner MJ, Wilberger JE (1996) Guidelines for the management of severe head injury. J Neurotrauma 13:643–645
11. Carroll KL, Moore KR, Stevens PM (1998) Orthopedic procedures after rhizotomy. J Pediatr Orthop 18:69–74
12. Cochrane DD, Steinbok P (1992) Laminotomy: a technical note. Child's Nerv Syst 8:226–228
13. Cohen AR, Webster HC (1991) How selective is selective posterior rhizotomy? Surg Neurol 35:267–272
14. Craft S, Park TS, White DA, Schatz J, Noetzel M, Arnold S (1995) Changes in cognitive performance in children with spastic diplegic cerebral palsy following selective dorsal rhizotomy. Pediatr Neurosurg 23:68–74
15. Deletis V, Vodusek DB, Abbott R, Epstein FJ, Turndorf H (1992) Intraoperative monitoring of the dorsal sacral roots: minimizing the risk of iatrogenic micturition disorders. Neurosurgery 30:72–75
16. Dudgeon BJ, Libby AK, McLaughlin JF, Hays RM, Bjornson KF, Roberts TS (1994) Prospective measurement of functional changes after selective dorsal rhizotomy. Arch Phys Med Rehabil 75:46–53
17. Engsberg JR, Olree KS, Ross SA, Park TS (1998) Spasticity and strength changes as a function of selective dorsal rhizotomy. J Neurosurg 88:1020–1026
18. Engsberg JR, Ross SA, Wagner JM, Park TS (2002) Changes in hip spasticity and strength following selective dorsal rhizotomy and physical therapy for spastic cerebral palsy. Dev Med Child Neurol 44:220–226
19. Engsberg JR, Ross SA, Collins DR, Park TS (2006) Effect of selective dorsal rhizotomy in the treatment of children with cerebral palsy. J Neurosurg 105:8–15
20. Fasano VA, Broggi G, Barolat-Romana G, Sguazzi A (1978) Surgical treatment of spasticity in cerebral palsy. Child Brain 4:289–305
21. Fasano VA, Broggi G, Zeme S, Lo Russo G, Sguazzi A (1980) Long-term results of posterior functional rhizotomy. Acta Neurochir Suppl 30:435–439
22. Fasano VA, Broggi G, Zeme S (1988) Intraoperative electrical stimulation for functional posterior rhizotomy. Scand J Rehabil Med Suppl 17:149–154
23. Foerster O (1908) Uber neue operative Methode der Behandlung spastiseher Lahinungen mittels Resektion hinterer Ruckenmarkswurzein. Z Orthop Chir 22:203
24. Foerster O (1913) On the indications and results of the excision of posterior spinal nerve roots in man. Surg Gynecol Obstet 16:463–474
25. Gros C, Ooaknine G, Vlahhovitch B, Frerebeau P (1967) La radicotomie selective posterioeure dans le traitment neurochirurgical de l'hypertonie pyramidale. Neurochirurgie 13:505–518
26. Gros C (1979) Spasticity: clinical classification and surgical treatment. In: Krayenbujl H (ed) Advances and technical standards in neurosurgery. Springer, Vienna, pp 55–97
27. Harada T, Ebara S, Anwar MM, Kajiura I, Oshita S, Hiroshima K, Ono K (1993) The lumbar spine in spastic diplegia. A radiographic study. J Bone Jt Surg Br Vol 75:534–537
28. Heim RC, Park TS, Vogler GP, Kaufman BA, Noetzel MJ, Ortman MR (1995) Changes in hip migration after selective dorsal rhizotomy for spastic quadriplegia in cerebral palsy. J Neurosurg 82:567–571
29. Hicdonmez T, Steinbok P, Beauchamp R, Sawatzky B (2005) Hip joint subluxation after selective dorsal rhizotomy for spastic cerebral palsy. J Neurosurg 103:10–16
30. Hodgkinson I, Berard C, Jindrich ML, Sindou M, Mertens P, Berard J (1997) Selective dorsal rhizotomy in children with cerebral palsy. Results in 18 cases at one year postoperatively. Stereotact Funct Neurosurg 69:259–267
31. Johnson MB, Goldstein L, Thomas SS, Piatt J, Aiona M, Sussman M (2004) Spinal deformity after selective dorsal rhizotomy in ambulatory patients with cerebral palsy. J Pediatr Orthop 24:529–536
32. Kim HS, Steinbok P, Wickenheiser D (2006) Predictors of poor outcome after selective dorsal rhizotomy in treatment of spastic cerebral palsy. Child's Nerv Syst 22:60–66
33. Landau WM (1990) Dorsal rhizotomy, a treatment of unproven efficacy. J Child Neurol 5:174–178
34. Loewen P, Steinbok P, Holsti L, MacKay M (1998) Upper extremity performance and self-care skill changes with spastic cerebral palsy following selective posterior rhizotomy. Pediatr Neurosurg 29:191–198
35. Logigian EL, Wolinsky JS, Soriano SG, Madsen JR, Scott RM (1994) H reflex studies in cerebral palsy patients undergoing partial dorsal rhizotomy. Muscle Nerve 17:539–549
36. Marty GR, Dias LS, Gaebler-Spira D (1995) Selective posterior rhizotomy and soft-tissue procedures for the treatment of cerebral diplegia. J Bone Jt Surg Am Vol 77:713–718
37. McLaughlin J, Bjornson K, Temkin N, Steinbok P, Wright V, Reiner A, Roberts T, Drake J, O'Donnell M, Rosenbaum P, Barber J, Ferrel A (2002) "Selective" dorsal rhizotomy: meta-analysis of three randomized controlled trials. Dev Med Child Neurol 44:17–25
38. McLaughlin JF, Bjornson KF, Astley SJ, Hays RM, Hoffinger SA, Armantrout EA, Roberts TS (1994) The role of selective dorsal rhizotomy in cerebral palsy: critical evaluation of a prospective clinical series. Dev Med Child Neurol 36:755–769
39. McLaughlin JF, Bjornson KF, Astley SJ, Graubert C, Hays RM, Roberts TS, Price R, Temkin N (1998) Selective dorsal rhizotomy: efficacy and safety in an investigator-masked randomized clinical trial. Dev Med Child Neurol 40:220–232
40. Mittal S, Farmer JP, Al-Atassi B, Gibis J, Kennedy E, Galli C, Courchesnes G, Poulin C, Cantin MA, Benaroch TE (2002) Long-term functional outcome after selective posterior rhizotomy. J Neurosurg 97:315–325
41. Mittal S, Farmer JP, Al-Atassi B, Montpetit K, Gervais N, Poulin C, Benaroch TE, Cantin MA (2002) Functional performance following selective posterior rhizotomy: long-term results determined using a validated evaluative measure. J Neurosurg 97:510–518
42. Mittal S, Farmer JP, Al-Atassi B, Montpetit K, Gervais N, Poulin C, Cantin MA, Benaroch TE (2002) Impact of selective posterior rhizotomy on fine motor skills. Long-term results using a validated evaluative measure. Pediatr Neurosurg 36:133–141
43. Nishida T, Thatcher SW, Marty GR (1995) Selective posterior rhizotomy for children with cerebral palsy: a 7-year experience. Child's Nerv Syst 11:374–380
44. Park TS, Gaffney PE, Kaufman BA, Molleston MC (1993) Selective lumbosacral dorsal rhizotomy immediately caudal to the conus medullaris for cerebral palsy spasticity. Neurosurgery 33:929–933
45. Park TS, Vogler GP, Phillips LH II, Kaufman BA, Ortman MR, McClure SM, Gaffney PE (1994) Effects of selective dorsal rhizotomy for spastic diplegia on hip migration in cerebral palsy. Pediatr Neurosurg 20:43–49
46. Park TS, Johnston JM (2006) Surgical techniques of selective dorsal rhizotomy for spastic cerebral palsy. Technical note. Neurosurg Focus 21:e7
47. Peacock WJ, Arens LJ (1982) Selective posterior rhizotomy for the relief of spasticity in cerebral palsy. S Afr Med J 62:119–124

48. Peter JC, Arens LJ (1993) Selective posterior lumbosacral rhizotomy for the management of cerebral palsy spasticity. A 10-year experience. S Afr Med J 83:745–747
49. Peter JC, Hoffman EB, Arens LJ (1993) Spondylolysis and spondylolisthesis after five-level lumbosacral laminectomy for selective posterior rhizotomy in cerebral palsy. Child's Nerv Syst 9:285–287
50. Pollack MA (1994) Limited benefit of electrophysiological studies during dorsal rhizotomy. Muscle Nerve 17:553–556
51. 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
52. Rosenbaum P, Stewart D (2004) The World Health Organization International Classification of Functioning, Disability, and Health: a model to guide clinical thinking, practice and research in the field of cerebral palsy. Semin Pediatr Neurol 11:5–10
53. Sackett DL (1986) Rules of Evidence and Clinical recommendations on the use of Antithrombotic agents. Chest 39:25–35
54. Sanger TD, Delgado MR, Gaebler-Spira D, Hallett M, Mink JW (2003) Classification and definition of disorders causing hypertonia in childhood. Pediatrics 111:e89–e97
55. Sanger TD, Chen D, Delgado MR, Gaebler-Spira D, Hallett M, Mink JW (2006) Definition and classification of negative motor signs in childhood. Pediatrics 118:2159–2167
56. Sindou M, Fischer G, Goutelle A, Schott B, Mansuy L (1974) Selective posterior rhizotomy in the treatment of spasticity. Rev Neurol 130:201–216
57. Spiegel DA, Loder RT, Alley KA, Rowley S, Gutknecht S, Smith-Wright DL, Dunn ME (2004) Spinal deformity following selective dorsal rhizotomy. J Pediatr Orthop 24:30–36
58. Steinbok P, Keyes R, Langill L, Cochrane DD (1994) The validity of electrophysiological criteria used in selective functional posterior rhizotomy for treatment of spastic cerebral palsy. J Neurosurg 81:354–361
59. Steinbok P, Daneshvar H, Evans D, Kestle JR (1995) Cost analysis of continuous intrathecal baclofen versus selective functional posterior rhizotomy in the treatment of spastic quadriplegia associated with cerebral palsy. Pediatr Neurosurg 22:255–264
60. Steinbok P, Kestle JR (1996) Variation between centers in electrophysiologic techniques used in lumbosacral selective dorsal rhizotomy for spastic cerebral palsy. Pediatr Neurosurg 25:233–239
61. Steinbok P, Reiner AM, Beauchamp R, Armstrong RW, Cochrane DD (1997) A randomized clinical trial to compare selective posterior rhizotomy plus physiotherapy with physiotherapy alone in children with spastic diplegic cerebral palsy. Dev Med Child Neurol 39:178–184
62. Steinbok P, Schrag C (1998) Complications after selective posterior rhizotomy for spasticity in children with cerebral palsy. Pediatr Neurosurg 28:300–313
63. Steinbok P (2001) Outcomes after selective dorsal rhizotomy for spastic cerebral palsy. Child's Nerv Syst 17:1–18
64. Steinbok P, Hicdonmez T, Sawatzky B, Beauchamp R, Wickenheiser D (2005) Spinal deformities after selective dorsal rhizotomy for spastic cerebral palsy. J Neurosurg 102:363–373
65. Storrs BB, Nishida T (1988) Use of the 'H' reflex recovery curve in selective posterior rhizotomy. Pediatr Neurosci 14:120–123
66. Turi M, Kalen V (2000) The risk of spinal deformity after selective dorsal rhizotomy. J Pediatr Orthop 20:104–107
67. van Schie PE, Vermeulen RJ, van Ouwerkerk WJ, Kwakkel G, Becher JG (2005) Selective dorsal rhizotomy in cerebral palsy to improve functional abilities: evaluation of criteria for selection. Child's Nerv Syst 21:451–457
68. Vaughan CL, Berman B, Peacock WJ (1991) Cerebral palsy and rhizotomy. A 3-year follow-up evaluation with gait analysis. J Neurosurg 74:178–184
69. Weiss IP, Schiff SJ (1993) Reflex variability in selective dorsal rhizotomy. J Neurosurg 79:346–353
70. Wright FV, Sheil EM, Drake JM, Wedge JH, Naumann S (1998) Evaluation of selective dorsal rhizotomy for the reduction of spasticity in cerebral palsy: a randomized controlled trial. Dev Med Child Neurol 40:239–247
71. Yasuoka S, Peterson HA, MacCarty CS (1982) Incidence of spinal column deformity after multilevel laminectomy in children and adults. J Neurosurg 57:441–445