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Spasticity: Classification, Diagnosis, and Management

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CLINICAL PEARLS

- Spasticity is a form of hypertonia that becomes more pronounced with the increasing speed of passive muscle stretch or passive motion beyond a critical angle. The disorder results from upper motor neuron lesions, such as cerebral palsy, stroke, or spinal cord injury.
- Physical and occupational therapies are central to the care of patients with spasticity. Tone reduction with botulinum toxin and oral antispasmodics such as baclofen are useful adjuncts.
- Baclofen can also be delivered directly to the cerebrospinal fluid by implantation of an intrathecal baclofen pump. This procedure may be appropriate for patients whose spasticity is refractory to oral medications.
- Selective dorsal rhizotomy is a surgical procedure that involves partial sectioning of sensory nerve roots of the cauda equina to interrupt the pathologic reflex arc causing spasticity. This procedure is most appropriate for individuals with spastic diplegia.

Introduction

Spasticity is defined as hypertonia occurring in response to passive muscle stretch that meets one of two criteria: the hypertonia increases with increasing speed of muscle stretch, or the hypertonia increases beyond a certain critical angle of joint motion.[1] Spasticity should be distinguished from rigidity, which is characterized by hypertonia that is present at very low movement speeds and does not worsen with rapid movement or movement beyond a threshold.[2] Spasticity can be classified based on the number of limbs involved: spastic quadriplegia affects all limbs, spastic diplegia affects the lower limbs, and spastic hemiplegia affects the limbs on only one side.

Several grading schemes for spasticity and hypertonia in general have been created. The modified Ashworth scale is shown in Table 54.1.[3] Although this scale is the most commonly used to grade the severity of spasticity, it does not distinguish whether the hypertonia is due to spasticity, rigidity, or dystonia. Thus rather than grading spasticity per se, it instead reflects the severity of the patient's hypertonia overall.[2] The Gross Motor Function Classification Scale (GMFCS), shown in Table 54.2, is used in children to describe the overall functional limitations caused by spasticity, but it also does not specifically describe the spasticity itself.[4]

Spasticity results from lesions of upper motor neurons that lead to hyperactivity of spinal motor neurons. This hyperactivity is associated with an involuntary, reflexive transition from relaxation to contraction as the stretch rate or degree of stretch reaches a threshold. In adults, it can occur as a consequence of spinal cord injury, multiple sclerosis, stroke, or other upper motor neuron lesions. In children, cerebral insults are far more common than are injuries to the spine. White matter lesions are particularly common causes among premature infants, possibly because of the sensitivity of oligodendrocyte precursors to hypoxic–ischemic insults during the third trimester.[5] Genetic and metabolic diseases such as X-linked adrenoleukodystrophy, metachromatic leukodystrophy, and Pelizaeus-Merzbacher disease can also cause this clinical picture.[6]

Cerebral palsy (CP) is a common cause of spasticity in children. This heterogeneous condition affects between 1.5 and 3 per 1000 live births and is characterized by abnormalities affecting movement and posture that interfere with normal activity.[9] It can be broadly categorized into spastic and dyskinetic forms based on the predominant motor findings, although no standardized classification scheme exists. Children with spastic CP experience increases in tone that interfere with normal activity and caretaking; dyskinetic CP is characterized by abnormal patterns of involuntary motor movements. Risk factors include prematurity, low birth weight, and a host of genetic and metabolic factors.[10]

Children presenting with signs and symptoms of CP should undergo a thorough evaluation, including assessment of

TABLE 54.1 Modified Ashworth Scale[3]

0—No increase in muscle tone
1—Slight increase in muscle tone, manifested by a catch and release or minimal resistance at the end of the range of motion when the affected part is moved in flexion or extension
1+—Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder of the range of motion
2—Notable increase in muscle tone through most of the range of motion, but the affected body part can still be easily moved
3—Considerable increase in muscle tone such that passive movement is difficult
4—Affected part is rigid in flexion or extension

TABLE 54.2 Gross Motor Functional Classification Scale[4]

Level I—Able to walk in the community and climb stairs without the use of a railing. Running and jumping are possible, but speed, balance, and coordination are limited.
Level II—Generally able to walk without assistance, although patients may require assistive devices when walking over uneven terrain or over long distances. Running and jumping are severely limited. A railing is required to climb stairs.
Level III—Handheld mobility device is required for walking. Wheeled devices are required for travel over long distances.
Level IV—Physical assistance from others or powered mobility devices are required in most settings. Walking short distances with assistance or a body support walker may be possible.
Level V—Limited ability to maintain head and trunk postures and to control limb movements. Transport by wheelchair is required.

developmental milestones, a general medical history, and screening for associated neurologic impairments such as epilepsy or sensory disturbances. The physical examination should focus on muscle bulk, muscle tone, posture, and, if the child is old enough, gait. Magnetic resonance imaging of the brain should be obtained to evaluate for structural abnormalities. Children with CP do not typically regress in terms of function, so loss of acquired milestones should prompt further workup and evaluation. Notably, the severity can vary with the patient's alertness and emotional state, which can complicate evaluations in young children.

Associated neurologic impairments are common. One-third of children with CP experience seizures, which are frequently challenging to treat and often require treatment with multiple medications.[11] Developmental delay is frequently present as well, and the domains affected can differ substantially from child to child. Some children with limited verbal IQ may have normal nonverbal intelligence, and others with normal verbal IQ can have profound disability in other types of reasoning. Detailed neuropsychiatric testing should therefore be performed in all children with CP.[12]

Signs and symptoms of CP are typically present by 1 year of age, and in more than half of cases the symptoms actually improve over time.[13] Nonetheless, other neurologic disability, such as developmental delay and seizures, may linger.

As with other diseases caused by upper motor neuron lesions, spasticity is almost always associated with hyperreflexia, clonus, weakness, and poor motor control. Although hypertonia can cause functional issues, the associated weakness and lack of control are more likely to be the primary factors underlying disability.[6,7] In fact, spasticity is to some extent an adaptive response to a decrease in control. Complete treatment of spasticity when associated with weakness can actually lead to a decline in function, such as deterioration in head or trunk control or worsened ambulation.[6,8]

Not all muscle groups are affected equally. Generally speaking, flexors are more affected than extensors, adductors are more affected than abductors, and muscles of internal rotation are more affected than muscles of external rotation.[8] The cumulative effects of hypertonia can cause problems with time. Fixed contractures can develop in muscles and tendons because of prolonged, involuntary muscle contraction, leading to shortening of the tissue and reduced range of motion. Increased stiffness due to these changes can superimpose on the rigidity due to increased muscle tone and cloud the clinical picture. In severe cases, bone and joint deformities such as hip dislocation can develop and cause significant discomfort. These soft tissue and bony changes can significantly impair mobility and complicate daily care.

Nonsurgical Management

Physical and occupational therapy are central to the care of patients with spasticity. The goal is to teach parents and caretakers skills to minimize the effects of hypertonia on daily life. Exercise can help address muscle atrophy, and stretching is used to prevent contractures, although in cases of severe hypertonia stretching may not be feasible. Adjunctive therapies such as treadmill training can help improve gait, and there is experimental evidence that transcranial stimulation can improve the response to this intervention in children via modulation of cortical activity.[14,15] Direct electrical stimulation of the muscles has also been found to help improve range of motion and gait.[16–18]

In patients whose spasticity interferes with function, cosmesis, or daily care, tone reduction should be considered. Botulinum toxin is a neurotoxic protein produced by *Clostridium botulinum* that is used for local control of spasticity. The agent is injected intramuscularly, where it is internalized by

presynaptic neurons at the neuromuscular junction to prevent the release of acetylcholine. Of the seven types of botulinum toxins, types A and B have biologic activity in humans and are available commercially in the United States; of these two, type A has been studied more extensively.[8] Botulinum toxin is highly effective. It has been shown to substantially improve spasticity in the targeted muscles, and its use also helps to alleviate pain. Several randomized trials have also demonstrated significant improvements in gait and functional status, although improved functional outcomes have not been seen universally.[19–24] Functional gains appear to be more robust when injections are combined with occupational therapy.[1,25] Serial casting is an effective adjunct to botulinum toxin for children with ankle equinus.[26]

Oral medications can also be used to treat generalized spasticity. Baclofen is a gamma-amino butyric acid ($GABA_B$) receptor agonist that has been shown in some studies to reduce spasticity and improve both passive and active range of motion, although high-quality evidence of improved functional outcomes is wanting.[24,27–29] Side effects, including sedation and confusion, can be significant. Withdrawal syndromes are a risk with sudden cessation. Tizanidine is a centrally acting α_2-agonist that has also been used, and limited evidence suggests that it may be more effective and better tolerated than baclofen.[30,31] Diazepam may also be considered.[24]

Orthopedic Surgery

Children with severe spasticity frequently require orthopedic procedures to help address deformities. Contractures are treated with tendon-lengthening procedures to improve range of motion. Osteotomies may also be required to correct bony malalignment and restore muscle action to the desired plane.[32] Arthrodesis is performed for progressive scoliosis to help with sitting and may be required to definitively treat hallux valgus (great toe bunion). Although these deformities may appear sequentially as the child ages, many providers now prefer to wait until the child is approximately 7 to 9 years of age before intervening. Delaying surgery allows the provider to better understand the relative contributions of dystonia and spasticity to the overall clinical picture, and it also facilitates gait analysis, which is important in operative planning.[32,33] It also enables the child to mature and understand his or her condition and the reasons for surgery.[32] These procedures should be conducted simultaneously in a single setting, an approach termed *single-event multilevel surgery* (SEMLS). Performing these operations together prevents the phenomenon of "birthday surgeries," where the patient undergoes regular, nearly annual operations during development as deformities arise; this practice leads to a near constant state of recovery and rehabilitation, significantly impairing the child's ability to live a normal life. SEMLS has been shown to provide good outcomes, both in the short term and on long-term follow-up.[34,35] Hip dislocations are an important exception that should be treated sooner, if they occur.[32]

Intrathecal Baclofen

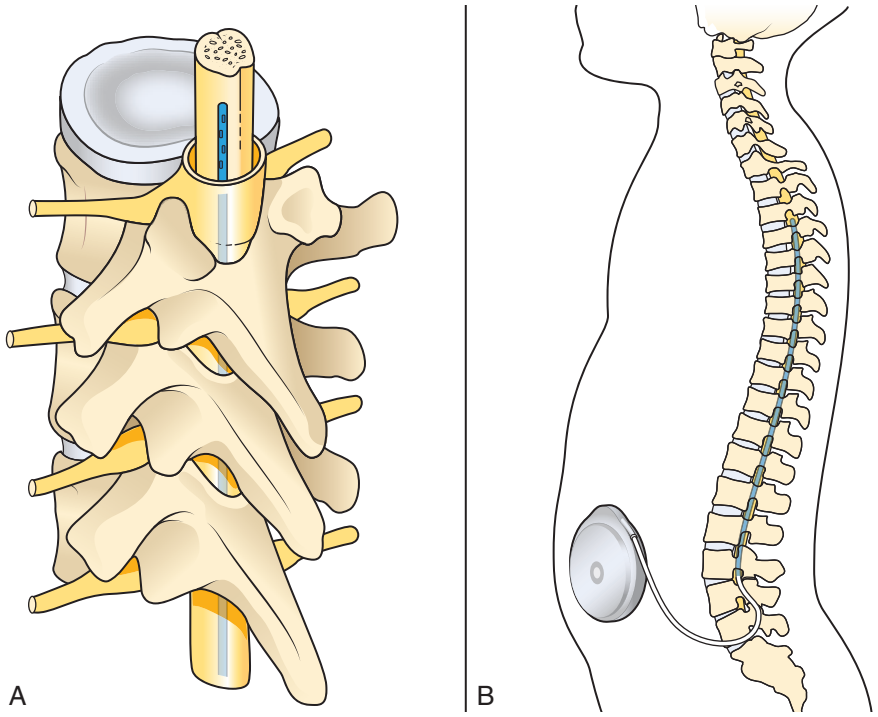
Patient Selection

Candidates for intrathecal (IT) baclofen infusion are patients with spasticity who have intractable spasticity not controlled by drug therapy or those who experience intolerable side effects to oral baclofen.[36] Overall, IT baclofen appears to be an effective treatment for spasticity in several small trials and larger meta-analyses. A small randomized controlled trial was the first to show a sustained, long-term benefit of IT baclofen in patients with spasticity due to spinal cord injury or multiple sclerosis,[37] a finding that has been replicated in this population in meta-analyses.[38,39] Evidence favoring its use in children with spastic CP soon followed, and IT baclofen is now routinely employed in children with severe spasticity.[40–43] Although the individual response to therapy varies, on average patients experience an approximately two-point improvement on the Ashworth scale.[39,43]

Before implantation of the IT pump, a test dose of IT baclofen must be administered. Practice varies from center to center, but test doses range from 50 to 100 μg, and some centers use a temporary catheter to evaluate different doses. Several parameters such as pulse, respiratory rate, blood pressure, and hypertonia based on the Ashworth score are assessed at specific time points after administration. If the patient does not respond to 100 μg given intrathecally, the patient is considered to have an inadequate response and should not undergo pump implantation.[36] Most providers offer a pump to patients if there is a two-point reduction in the Ashworth score for >4 hours after a bolus of drug.[36] The starting daily dose is typically twice the test dose, approximately 20 μg/day.

Surgical Technique

The system consists of a catheter and a pump, which are implanted in the operating room while the patient is under general anesthesia (Fig. 54.1). Once the patient has been anesthetized, he or she is placed in the lateral decubitus position. If the patient has a gastrostomy tube in place, the system should be placed on the right side. An approximately 8-cm oblique incision is made through the skin of the right lower quadrant, ending laterally above the anterior superior iliac spine. Dissection is continued down through the fascia of the external oblique muscle, where a pocket between the fascia and muscle is created. Placing the pump deep to the fascia helps to reduce tension on the incision and can help prevent wound healing complications, particularly in small children and in those who are underweight.[44,45] With the pump in place, a 1- to 2-cm incision is made over the lumbar spine, and dissection is continued down to fascia. A Tuohy needle is then passed down to the thecal sac, and the intrathecal catheter is advanced rostrally. For patients with spastic diplegia, the catheter tip is placed at T10 to T12, and the positioning is confirmed with intraoperative fluoroscopy.[45] Spastic tetraplegia requires more rostral placement at C5 to T2. The Tuohy needle is then withdrawn, and the catheter is secured to the fascia. The tip of the



• **Figure 54.1** Baclofen pump placement. (A) The intrathecal catheter is placed in the thecal sac and advanced to the appropriate level for the therapeutic indication. For spastic diplegia, the target level is T10 to T12; for spastic tetraplegia, C5 to T2 are targeted. (B) The catheter is then tunneled around the flank to the pump, which is placed in a subfascial pocket in the anterior abdominal wall.

catheter is checked for spontaneous return of cerebrospinal fluid. Once this is confirmed, the catheter is then tunneled around the flank to the pump.

Using an external programmer, adjustments can be made to the rate of administration, titrating to therapeutic effect. The dose required to achieve the desired effect generally increases over time as patients become habituated to the effect of the drug.[39] Higher baclofen doses deplete the pump's reservoir more quickly, requiring more frequent refills. Fortunately, this can be accomplished percutaneously and is rarely required more than once every 2 to 3 months. With time, however, the device's battery will wane, requiring replacement of the pump in the operating room every 5 to 7 years.

Complications

Some complications associated with implantation include local infection (4%), overdose (2%), and catheter malfunction necessitating surgical exploration (17%).[36] Pump failure can precipitate a dangerous withdrawal syndrome, characterized by hypertonia, fever, seizures, and even cardiac arrest, coma, and death.[46,47] Prompt replacement of the IT baclofen with oral drug is required, and, in severe cases, benzodiazepines and muscle relaxants such as dantrolene may also be needed.[46]

Intraventricular Baclofen

Intraventricular (IV) delivery of baclofen is a novel approach that has been studied less than IT baclofen. In one retrospective study of 22 patients who underwent IV baclofen therapy because of complications related to IT baclofen, the investigators found that IV dosing could achieve similar rates of therapeutic relief with lower rates of surgical complications.[48] Equivalent efficacy was seen in another study comparing children who received IT baclofen with those who received IV baclofen; however, although they found a trend toward a lower risk of catheter or leak-related complications with IV baclofen, the trend was not statistically significant.[49]

Selective Dorsal Rhizotomy

Selective dorsal rhizotomy (SDR) is a neurosurgical procedure that alleviates the spasticity associated with CP. The concept of transection of lumbar and sacral sensory nerve roots for the treatment of lower limb spasticity dates to the early 1900s, when Foerster described a series of patients in whom the total sectioning of the posterior nerves of L2, L3, L5, and S1 was performed.[50] The method was later adapted by Gros and colleagues in Montpellier, France, such that 80% of the dorsal nerve roots from L1 to S1 were sectioned.[51] Further refinements to the technique resulted in a "selective" rhizotomy procedure that tailored the degree of nerve root sectioning to the functional status of the patient. Rootlets innervating spastic segments deemed "disabling" were sectioned, whereas segments thought to have "beneficial" spasticity were spared. Yet another modification advanced by Fasano and associates[52] and Peacock and Arens[53] determined the degree of rootlet sectioning by the electrophysiologic results of intraoperative electrical

stimulation of the posterior rootlets. Those rootlets associated with a sustained or diffuse muscular contraction were divided until stimulation produced only brief localized contraction.[54] The underlying tenet of this procedure is that abnormal peripheral afferents from spastic segments are identifiable by electrophysiologic responses to rootlet stimulation. Modern techniques tend to utilize both physiologic and electrophysiologic information to determine the extent of rootlet sectioning.

The method of exposure of the nerve roots also has evolved over the years. The technique described by Peacock and coworkers[54] involved wide lumbar laminectomies with exposure and stimulation of the dorsal nerve rootlets from L2 to S1 bilaterally. Adults who underwent the procedure did not evidence spinal instability as a late complication. Multilevel laminectomies in young children, however, were associated with progressive kyphosis, anterior subluxation, and spinal deformity with age. One study found that after multilevel laminectomy for conditions that do not usually cause spinal deformity in children, spinal deformity developed in 46% of patients under 15 years of age and 6% in patients from 15 to 24 years of age.[55] Another study found the risk of developing a structural spinal deformity after wide laminectomy without laminoplasty for SDR was 36%, with 6% of patients requiring stabilization at an average of 4.9 years after SDR.[56]

In response to the concerns about progressive spinal instability after SDR, Raimondi and colleagues[57] described a technique in 1976 that utilized laminotomy and reconstruction of the posterior spinal elements after the rhizotomy. An outcome study of 79 patients with no preexisting spinal deformity who underwent SDR with laminoplasty found that scoliosis developed in 16% and spondylolisthesis in 12%.[58] One prospective study directly comparing laminectomy and laminoplasty found a higher incidence of spinal deformity, including lumbar hyperlordosis, spondylolisthesis, and scoliosis, after SDR than in patients without spasticity and in a historical control population, but no significant difference in spinal deformity rates was observed between patients who underwent laminoplasty and those who underwent laminectomy.[59]

In 1993 Park and colleagues[60] popularized a technique for SDR that further minimized the extent of spinal disruption, utilizing laminectomy only at L1–L2, with intraoperative ultrasound identification of the conus. This was followed by a description in 2006 of a series of 1500 patients who had undergone SDR via single-level laminectomy at the level of the conus.[61] The advantages of the single-level laminectomy over the multilevel procedure include decreased operating time, decreased postoperative pain, and less risk of future lumbar instability.[61]

Patient Selection

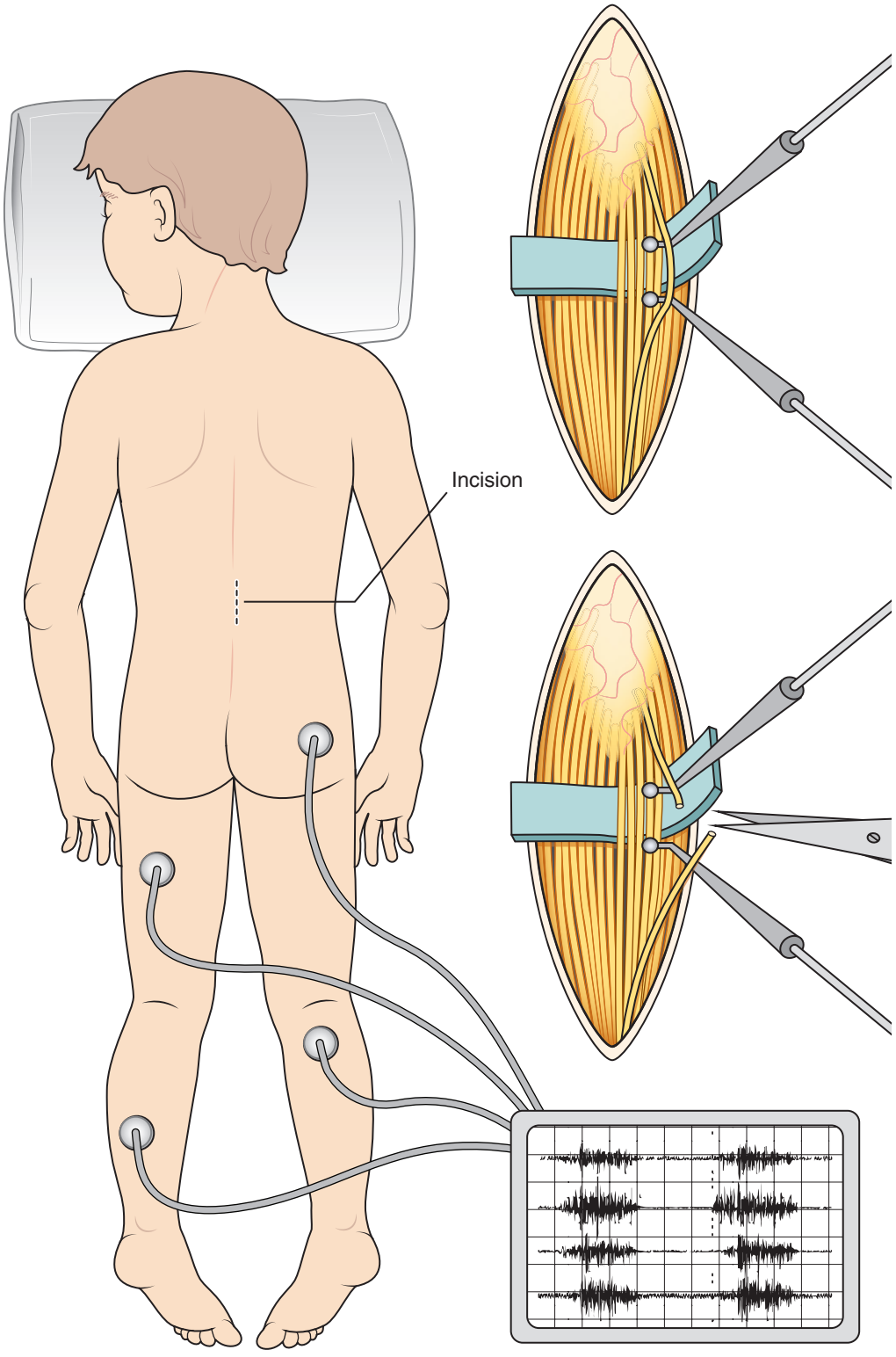
Children who are possible candidates for SDR should undergo an interdisciplinary evaluation by specialists that include physical and occupational therapists, a rehabilitation medicine physician, an orthopedic surgeon, and a neurosurgeon. A full physical examination and assessment of tone, strength, range of motion, motor control, gait, and movement as well as family and other social factors are taken into account.[62] Anteroposterior and lateral spine radiographs as well as magnetic resonance imaging scans of the brain and spine are acquired to evaluate the surgical anatomy. Gait is assessed using either observational videos or three-dimensional gait analysis. Target muscle groups and functional goals are identified based on these preoperative assessments.

SDR is best indicated in children with spastic diplegia due to CP, but it can also provide benefit in children with spastic quadriplegic CP and some adults younger than 40 years who have mild spastic diplegia and can walk independently.[61] The procedure is not indicated for hemiplegic CP, because spasticity is not a major contributor to motor impairment in this condition.[61] CP cannot be definitively diagnosed in children under the age of 2 years; therefore SDR cannot be offered prior to this age. Coincident dystonia does not worsen with SDR and is not a contraindication. Conversely, rigidity does not improve with SDR, and severe damage to the basal ganglia on magnetic resonance imaging, which indicates possible rigidity, is a contraindication to SDR. Other contraindications include multiple orthopedic operations because of fixed deformities and severe muscle weakness that limit the potential to improve and increased muscle tone due to conditions other than CP such as hydrocephalus, intrauterine and neonatal infections, neural migration disorders, and head trauma, as these do not respond to SDR. The exception to this last contraindication is spastic diplegia due to schizencephaly.[61]

Surgical Technique

At the authors' institution, the surgical technique uses an extension of the single-level laminectomy of Park and colleagues,[61] which additionally compares electromyography stimulus responses to separate dorsal and ventral nerve roots, as described by Bales and coworkers (Fig. 54.2).[62] In brief, general anesthesia is induced and the patient is positioned prone. The anesthesiologist should use short-acting neuromuscular blocking agents so that electromyography recordings can be obtained soon after intubation. Anesthesia during stimulation is a combination of intravenous propofol and up to 0.5 minimum alveolar concentration of sevoflurane. Recording electrodes are applied bilaterally to muscles innervated by the L2 to S2 nerve roots. The muscles recorded are the bilateral gluteal, iliopsoas, hip adductors, biceps femoris, rectus femoris, vastus medialis, vastus lateralis, tibialis anterior, gastrocnemius, adductor hallucis, extensor hallucis longus, and anal sphincter muscle groups.[62]

The vertebral level below the conus, as identified by preoperative magnetic resonance imaging, is selected for laminectomy. Intraoperative fluoroscopy is used to identify the desired level, and an incision is planned on the overlying skin. After exposure of the appropriate laminae, a partial laminectomy is performed with Kerrison rongeurs. Intraoperative ultrasound confirms that the level of exposure is below the level of the conus, and then the laminectomy is completed. The dura is opened in the midline, and a Silastic sheet is inserted ventral to all roots of the cauda equina.



• **Figure 54.2** Selective dorsal rhizotomy. The patient is positioned prone with neurophysiologic monitoring leads attached. A small incision is made just below the level of the conus, and a laminectomy is performed. The dura is opened, and the nerve rootlets are identified. Each rootlet is stimulated separately, and those with pathologic neurophysiologic responses are sectioned.

Stimulation of the nerve roots is performed with bipolar stimulation with a 0.1-ms square-wave pulse at 3.11 Hz. The nerve root must be held free of tension, clear of cerebrospinal fluid, and with tip separation of the stimulation electrodes between 5 and 10 mm.[63] The amplitude of the stimulation voltage is gradually increased from 0 to 5 mA until a response is noted. Ventral (motor) rootlets tend to exhibit thresholds less than 0.4 to 0.5 mA, whereas dorsal (sensory) rootlets have thresholds greater than 0.5 mA and generally above 1.0 mA.

Once a stimulation threshold is established, all rootlets are stimulated sequentially. Motor rootlets are excluded behind the Silastic sheet, as are any rootlets associated with anal sphincter responses at any sacral level. Sphincter responses from lumbar rootlet stimulation are considered pathologic reflex activity and do not preclude sectioning of those rootlets.[63] The sensory rootlets are then divided into smaller subsets, with a total of 60 to 80 nerve rootlets tested in most patients. The sensory rootlets are exposed to a 1-second train of 50-Hz tetanic stimulation with electromyography responses graded by an electrophysiologist as normal, slightly abnormal, or markedly abnormal based on the following criteria: a persistent response; a waxing or waning response; an increasing, decreasing, or burst response; and a spread of tetanic response to other muscle groups. Rootlets that elicit a response in one of the target muscle groups are incised with microscissors. The degree of abnormality by these criteria determines the amount of rootlet sectioned. If the response is markedly abnormal, 75% to 90% of the rootlet is cut. For slightly abnormal responses, 50% of the rootlet is cut. If the response to tetanic stimulation is normal but the rootlet serves only target muscle groups, then 50% of the rootlet is sectioned. Normally responding rootlets in nontarget muscle groups are spared.

After completion of the sensory nerve rootlet sectioning, the intrathecal space is irrigated, closed primarily, and coated with an autologous blood patch. The patients are maintained on strict flat bedrest for 3 days postoperatively. As activity is increased, patients are transferred to an inpatient rehabilitation unit to complete a course of intensive physical therapy for 3 weeks, followed by an intensive outpatient program of 3 to 5 days of therapy per week.

Patient Outcomes

A comparative analysis and meta-analysis of the 9- and 12-month outcomes of randomized clinical trials confirmed a reduction of spasticity and greater functional improvement with SDR plus physiotherapy compared to physiotherapy alone.[64] Multivariate analysis showed a relationship between the percentage of dorsal root tissue transected and the amount of functional improvement; however, the sectioning rate was not randomized, and the sectioning technique varied across studies. The review also concluded that SDR plus physiotherapy had a small but significant positive effect on gross motor function.[64]

A review of 63 articles[65] describing the results of SDR cataloged a wide array of outcome measures, including degree of impairment, instrumented gait analysis, sitting, ambulation, Gross Motor Function Measure, Pediatric Evaluation of Disability Inventory (PEDI), Quality of Upper Extremities Skills Test (QUEST), incidence of orthopedic procedures after SDR, and incidence of hip subluxation after SDR. The review determined that there is conclusive evidence that SDR decreases lower limb spasticity and increases lower limb range of motion, strong but not conclusive evidence that SDR improves motor function, moderate evidence that SDR improves disability and results in positive suprasegmental effects, and weak evidence that SDR may reduce the need for orthopedic procedures.[65] The maximal duration of follow-up was 12 years, although most studies were shorter.

SDR has also been found to have a beneficial effect on dysfunctional bladder voiding symptoms. In one study, there was significant improvement in both silent bladder dysfunction and overt bladder symptoms. On urodynamic testing, total bladder capacity and pressure specific volumes showed statistically significant improvement after SDR. All children had neurologic improvement postoperatively, 71% of those who were incontinent preoperatively became continent postoperatively, and none had deterioration on urodynamic testing.[66] There has also been some suggestion that children with CP who receive SDR may have improvements in specific attentional and cognitive functions, with suggested possible mechanisms relating to improved mood, reduced physical discomfort, increased therapeutic intervention, or possible cortical effects of SDR.[67]

More recently, as the cohort of patients who have undergone the procedure has aged, studies of long-term outcomes have been possible (Table 54.3).

Complications

Complications reported from various series on outcomes of SDR include bronchospasm (5.5%), aspiration pneumonia (3.5%), urinary retention (7%), and sensory loss (2%).[68] Progressive spinal deformity, as discussed above, can include hyperlordosis, hyperkyphosis, spondylolisthesis, and scoliosis.

Conclusion

Spasticity is an important contributor to disability in children and adults with neurologic disorders. Although nonsurgical management with physical and occupational therapies, tone-reducing drugs, and botulinum injections are the mainstays of therapy, many patients still require surgical treatment. The cumulative effect of spasticity can produce skeletal and soft tissue changes requiring correction by an orthopedist. Severe cases of spasticity not responsive to oral medications can be treated with IT baclofen delivery. Finally, SDR is an important technique that can significantly improve gait in children and young adults with spastic diplegia.

TABLE 54.3 Long-Term Outcomes After Selective Dorsal Rhizotomy

Study	N	Length of Follow-Up	Gross Motor Function Classification Scale					Adverse Outcomes
			I	II	III	IV	V	
Josenby et al.[69]	24	5 to 10 years	Improvements in functional skills, mobility, caregiver assistance self-care, caregiver assistance mobility during the first 5 years postop with small (not statistically significant) changes between 5 and 10 years postop			Improvements noted in the first 5 years postop but not after		Not reported
Bolster et al.[70]	29	5 and 10 years	GMFM-66 mean score showed significant increase ($p < 0.001$) between baseline and 5-year follow-up. The difference between 5- and 10-year follow-up was not statistically significant.			None included		Scoliosis in 1, spondylolysis and listhesis in 1, subluxation in 3
Tedroff et al.[71]	18	15 to 20 years	The effect of normalized muscle tone in lower extremities after SDR was sustained for a median of 17 years. The best function was seen at 3 years, after which a gradual decline followed. SDR does not improve long-term functioning nor prevent contractures but can reduce pain.					Not reported

GMFM, Gross Motor Function Classification Scale; *SDR,* selective dorsal rhizotomy.

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