Management of Spasticity: A Review of Two Methods of Medical Intervention

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MANAGEMENT OF SPASTICITY:
A REVIEW OF TWO METHODS OF MEDICAL INTERVENTION

by

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An Independent Study
Submitted to the Graduate Faculty of the
Department of Physical Therapy
School of Medicine
University of North Dakota
in partial fulfillment of the requirements
for the degree of
Master of Physical Therapy

Grand Forks, North Dakota
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1995
This Independent Study, submitted by Andrea Grock in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Faculty Preceptor, Advisor, and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.

(Faculty Preceptor)

(Advisor)

(Chairperson, Physical Therapy)
PERMISSION

Title Management of Spasticity: A Review of Two Methods of Medical Intervention

Department Physical Therapy

Degree Master of Physical Therapy

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Signature Andrea L. Apvik

Date 12/8/94
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A special thank you to my family for their unending encouragement and support. With your help, I made it!

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ABSTRACT

Spasticity is an entity with which physical therapists are quite frequently faced. It is a common phenomenon among individuals who have sustained central nervous system damage. Spasticity is associated with a wide variety of neurological diagnoses and may have benefits as well as negative qualities. The negative effects can lead to multiple complications which interfere with functions of everyday living. Management of spasticity becomes necessary when it causes complications or interferes with function. There are several modes of intervention, including conservative physical therapy approaches and/or medical approaches.

The purpose of this independent study is to review two medical methods of intervention: pharmacological treatment and dorsal rhizotomy. Pharmacological treatments include medications or local injections of various agents. These treatments only provide temporary relief unless they are continued and/or repeated. Although pharmacological treatments have been found to be effective to reduce spasticity, they may have unwanted side effects. Dorsal rhizotomies have been successful for reducing spasticity in appropriate candidates. The effects of this treatment are permanent and irreversible. Side
effects are often temporary, such as sensory loss or muscle weakness, and there are risks related to surgery and anaesthesia.
CHAPTER 1

INTRODUCTION

Spasticity is a common phenomenon among individuals who have sustained some type of insult to the central nervous system (CNS). It is associated with a wide variety of neurological diagnoses. A few examples are: cerebrovascular accident (CVA), cerebral palsy (CP), traumatic brain injury (TBI), and spinal cord injury (SCI). Resulting from an upper motor neuron lesion,\(^1\) spasticity is defined as the hyperexcitability of the spinal stretch reflex resulting in a velocity-dependent increase in the tonic stretch reflex with exaggerated tendon jerks.\(^2\)

Spasticity has both desirable and undesirable effects. For example, it can be an asset to an individual as a means for movement. By the same token, it can become a problem if it is too severe and interferes with movement. The undesirable qualities of spasticity can lead to secondary physical complications. Consequently, it becomes necessary to control unwanted spasticity in an attempt to allow the individual his or her fullest potential for rehabilitation.

Intervention and management of spasticity can take a variety of forms. It can include conservative, medical, or surgical techniques. Which type is used will be affected by each individual's particular needs and problems. Treatment
is driven by symptoms because, in the majority of cases, the underlying cause cannot be treated or reversed (i.e., traumatic brain injury).  

The purpose of this independent study is to explore current literature and to discuss two medical approaches for the management of spasticity. This review will include an examination of spasticity, a discussion of pharmacological therapy, and a discussion of dorsal rhizotomy.
CHAPTER 2
SPASTICITY

Spasticity is often encountered in individuals who have sustained some type of insult to the CNS. More specifically, the lesion can be either to the brain or spinal cord.\(^3\) It is important to note that this spasticity stems from an upper motor neuron lesion,\(^4\) such as would result with injury or pathology to the brain or spinal cord.

There are a variety of neurological diagnoses in which spasticity manifests itself. These include traumatic brain and spinal cord injury, multiple sclerosis, cerebrovascular accident, cerebral palsy, and some rare degenerative processes of the CNS.\(^1,3,5\)

Clinically, there are a variety of other phenomena which are related to spasticity. These phenomena vary somewhat depending upon whether it is a cerebral lesion or an insult to the spinal cord.\(^6\) Spinal cord lesions present with flexor patterns in the lower extremities, early flexor spasms, clasp-knife phenomenon, clonus, spinal shock for 1-16 weeks, exaggerated exteroceptive reflexes, and autonomic dysreflexia.\(^6\) Cerebral lesions present with tone predominant in antigravity muscles, rare flexor spasms, less clasp-knife phenomenon, less clonus, cerebral shock for two to three weeks, absence of
superficial abdominal and cremasteric reflexes, absent autonomic phenomena.\textsuperscript{6}

Samkoff\textsuperscript{7} states that when damage to the CNS occurs, it leads to a disconnection of the lower motor neuron from the control of upper motor neurons of the brain, brainstem, and spinal cord. Spasticity is only one manifestation of an upper motor neuron lesion, and there are associated positive signs such as the Babinski response, hyperactive nociceptor reflexes, flexor and extensor spasms, and dystonia. Weakness and loss of dexterity are negative signs.

Considering the various neurological diagnoses and their site of insult, it becomes evident that the clinical picture of spasticity will vary from patient to patient. For example, consider the differences between an individual with multiple sclerosis and an individual who has sustained a CVA. Multiple sclerosis is a progressive degenerative disease in which spasticity may stem from the involvement of multiple sites\textsuperscript{8}; thus, spasticity could present in more than one area of the body simultaneously. An individual who has sustained a CVA typically has had an insult of cerebral origin, with deficits occurring on the side of the body opposite to the site of the lesion.\textsuperscript{1} This example demonstrates how the physical manifestation of spasticity varies with diagnosis and the specific location of the lesion.

A closer examination of the definition of spasticity becomes necessary to truly gain a better knowledge of this complex phenomenon. Lance\textsuperscript{2} describes spasticity as the hyperexcitability of the spinal stretch reflex resulting in a
velocity-dependent increase in the tonic stretch reflex with exaggerated tendon jerks. Lance proposed this definition in 1980, and it is now widely accepted as the definition of spasticity by most clinicians and physiologists.9

Young and Wiegner10 stated that "spastic patients may be more handicapped by reduction in reciprocal inhibition than by hyperexcitability of the stretch reflex." Pierrot-Deseilligny11 suggests that motor disability comes from the triggering of a stretch reflex in antagonistic muscles during active movement. An exaggeration of the stretch reflex or a malfunction of reciprocal la inhibition are both means by which this could happen.

Although there are different opinions about the underlying cause of spasticity, a similarity does exist: involvement of the stretch reflex and reciprocal inhibition.

The stretch reflex begins when extrafusal (skeletal) muscle fibers are stretched, resulting in increased activity in the alpha motor neurons of the agonist muscle, causing it to contract. The afferent limb of the reflex consists of the type Ia and II afferents which run from the muscle spindle, enter the spinal cord, and then synapse with the alpha motor neurons of the agonist muscle. The Ia afferents also synapse with inhibitory interneurons to the antagonist preventing contraction (reciprocal inhibition). The stretch reflex can be evoked by a dynamic stretch, such as tapping the tendon, or by a static stretch. In this pathway is another interneuron called the Renshaw cell. It is stimulated each time the alpha motor neuron is activated. This activation
causes inhibition of the same motor neuron which activated it. It is believed that the Renshaw cell diminishes the activity of the alpha motor neuron to prevent oscillation or, in other words, continuous reflex action.\textsuperscript{12}

As physical therapists work with neurological patients, the role of spasticity becomes an important issue. Spasticity can be beneficial or problematic. Spasticity may be welcomed for the following reasons: bowel training, hemiplegic gait, increased muscle bulk, reduced risk of osteopenia.\textsuperscript{13} Spasticity of sphincter muscles may provide a means by which a patient can develop a bowel and bladder control program. The hemiplegic patient may have enough muscle tone as a result of spasticity which will aid in learning to walk again with a leg that could have potentially been flaccid or weak. An increase in muscle tone due to spasticity may be a favorable source of stress on bones in preventing breakdown of bone which would result if muscular stresses are removed.

Problems can arise when spasticity becomes too severe. Some of the negative effects which result may include: pain, impaired hygiene, joint contracture, hydronephrosis and renal damage, pressure ulcer formation and skin breakdown, and impaired activities of daily living and functional activities.\textsuperscript{13,14} Pain may result from severe and violent spasms. Hygiene of the perineal area will be limited if adductors become tight and prevent proper washing of that area. Joint contracture will be a result of prolonged contracture which leads to insufficient movement throughout the range of motion and
results in contracture formation. When excessive spasticity of the urinary sphincter exists, it may cause reflux of urine and result in problems with the renal system. Pressure sores and skin breakdown occur when there is an inability to sufficiently relieve pressure. Impaired activities of daily living and functional activities result when there is absence of voluntary control and movement.

Physical therapists treating neurological patients will primarily be interested in how motor function has been affected. After all, rehabilitation of motor function is the area in which the physical therapist spends all of his or her time. Haley and Inacio contend that spasticity in upper motor neuron syndromes influences motor dysfunction in three ways. First, it affects passive movement and static postural alignments. Second, it affects voluntary movement. Third, it may coexist with other upper motor neuron deficits and may play an insignificant role in regard to motor dysfunction. Earlier, we examined specific examples of problems and benefits of spasticity. These are clearly related to the influence that spasticity has on motor dysfunction. These three factors involve the very areas addressed by physical therapy.

Assessment becomes necessary to evaluate whether spasticity is interfering with movement and, if so, to what degree. When spasticity is too severe and interrupts motor function, it becomes necessary to implement management techniques in order to decrease the severity of the negative effects. Ideally, intervention aims at making positive gains in motor function.
By decreasing or negating spasticity and its effect upon motor function, it is thought that motor movement will become less difficult.

Homberg\textsuperscript{15} suggests that the rehabilitation program should be a multidisciplinary process involving goal setting and continual reassessments. He suggests a rehabilitation process in spastic syndromes. The process involves four steps: 1) Identify actual functional deficits; 2) Establish a plan to alleviate deficits and exploit residual function; 3) Before considering drugs, exploit residual abilities and/or physical therapy; 4) Do not use drugs without defining clear goals for activities of daily living.

Although the author intended this system to evaluate the need for drug therapy, it clearly is a useful system when considering any type of intervention. It should be kept in mind that treatment should be planned based upon goals and desired outcome. Treatment should not simply aim at eliminating spasticity, but look at spasticity in terms of how it is affecting the individual's level of function. The overall goal should be to maximize the individual's ability to function in daily living.

There are a variety of approaches for the management of spasticity. The physical therapist may be directly involved by using conservative physical therapy techniques. These may include such things as range or motion, therapeutic exercise, functional electrical stimulation, heat and cold modalities, vibration, and orthoses.\textsuperscript{13} Other techniques include seating, splinting, and casting.\textsuperscript{16} However, the use of conservative management alone may not be
enough. It may be necessary to implement medical methods of managing spasticity. Prescribing medical intervention is not within the scope of physical therapy practice. However, the physical therapist and consequently the physical therapy program will be directly affected by the medical treatment of spasticity. Furthermore, conservative techniques and medical management may be combined to create a spasticity management program customized to the individual's needs. Barnes\textsuperscript{16} states that treatment should be based upon establishment of specific functional goals which are based upon the improvement of function, the prevention of complications, and the alleviation of pain.
CHAPTER 3
PHARMACOLOGICAL TREATMENT

Pharmacological agents are frequently used for reducing spasticity. In the literature\textsuperscript{7,16-18} pharmacological agents are listed as a very relevant and commonly used method of intervention for spasticity. Pharmacological agents include oral and intrathecal administration of medications and local injections of various agents.

Medications

Several articles\textsuperscript{7,16,18} list the three most common antispastic medications as diazepam, dantrolene sodium, and baclofen. Though often used singly, they may be used in various combinations.\textsuperscript{16} This allows a combination of effects from several drugs in attempt to gain maximal benefits.

Diazepam, also known as Valium, has an affect both on the brain and on the spinal cord.\textsuperscript{7} It belongs to the family of benzodiazepines.\textsuperscript{19} Taken orally, diazepam acts to increase inhibitory activity of lower motor neurons by facilitating transmission of GABA at GABA\textsubscript{A}-receptor sites. Common side effects include drowsiness, dizziness, weakness, hypotension, fatigue, vertigo, ataxia, and withdrawal.\textsuperscript{7,16,18}
A study of the effects of a low dose benzodiazepine (Clonazepam) revealed that treatment with a low dose benzodiazepine was effective in reducing spasticity in children with cerebral palsy.17 Young and Delwaide18 state that Diazepam is a useful treatment approach in individuals with spinal cord damage and sometimes for individuals with cerebral palsy. Penn20 feels that it is the second most effective drug for treating spasticity next to baclofen and that the side effects limit its use, often before clinical relief of symptoms are seen.

Dantrolene sodium is administered orally and has a direct effect on the muscle tissue itself.7,18 It interrupts muscle contraction by suppressing the release of calcium from the sarcoplasmic reticulum,7,20 thus interfering with the sequence of steps involved in a muscle contraction.

Common side effects include drowsiness, dizziness, weakness, fatigue, and diarrhea.16 Barnes16 notes that there have been reports of hepatotoxicity and that liver function should be closely monitored in those individuals receiving dantrolene. Additionally, dantrolene sodium and its action on the muscle cause muscle weakness.7,20 Several authors7,18,20 suggest that Dantrolene sodium may only be desirable for individuals who are not expected to make functional gains due to the effect of muscle weakness.

Young and Delwaide18 state that dantrolene may be helpful in reducing spasticity regardless of where the lesion is located. This includes individuals
with multiple sclerosis, spinal cord injury, cerebral palsy, and cerebrovascular accident.

Baclofen may be administered orally\textsuperscript{7,16,20} or intrathecally\textsuperscript{21-23}. Baclofen acts as an agonist to \textit{GABA}{\textsubscript{B}}-receptors\textsuperscript{7}. Its action is to inhibit the release of excitatory neurotransmitters and it primarily targets polysynaptic spinal reflexes\textsuperscript{16}. The main side effects include drowsiness, fatigue, and muscle weakness which are usually dose-dependent effects. It has been suggested by several authors\textsuperscript{16,20} that baclofen may be most effective for spasticity of spinal origin.

Intrathecal baclofen is administered via an infusion pump which delivers baclofen into the subarachnoid space\textsuperscript{21}. The pump is implanted under the skin on the lateral aspect of the abdomen. It is then hooked to a catheter in the intrathecal space. Penn\textsuperscript{20} explains the benefit of administering baclofen intrathecally over administering it orally. He states that the dose of oral baclofen required to relieve symptoms in individuals with severe spasticity will often cause unwanted side effects. However, if delivered directly into the subarachnoid space, the concentration is higher around the spinal cord while remaining low around the brain. Thus, unwanted side effects can be avoided while delivering adequate levels of baclofen to the spinal cord promoting its effect on spasticity.

Several studies\textsuperscript{24,25} have shown the effectiveness of intrathecal baclofen for treating spasticity in cerebral palsy and other causes of cerebral origin.
These studies showed significant decreases in muscle tone after intrathecal administration of baclofen. Confusion and drowsiness occurred in two of the subjects of one study, but cleared two hours following the dose. Intrathecal baclofen may also be useful for long term treatment of multiple sclerosis or spinal cord injury.

Side effects from intrathecal baclofen are due to the side effects of the drug itself. An overdose may lead to seizures, coma, respiratory problems, and flaccidity. There may be technical difficulties with kinks, dislodging, disconnections, or tears in the catheter. Additionally, infection at the site of the pump or pump failure may occur.

There are many other medications which have been used to decrease spasticity; however, they are not as common. Two recent examples include tizanidine and divalproex sodium. Tizanidine, an alpha2-receptor agonist, is thought to reduce the release of the excitatory neurotransmitter, aspartate. However, it is currently not available in the United States. Divalproex sodium, an antiepileptic agent, was shown to be an effective oral agent for improving pain and spasticity in three out of four patients in a recent study.

Local Injections

Inclusive in this category are nerve blocks and other local injections. Nerve blocks involve the injection of a nerve with phenol, alcohol, or a local anaesthetic agent using a needle. Nerve blocks decrease spasticity by blocking nerve conduction through chemical neurolysis of nerve tissue.
Barnes\textsuperscript{16} states that it is preferable to block motor nerves rather than mixed sensorimotor nerves. However, it is possible to inject either motor nerves or mixed sensorimotor nerves.\textsuperscript{27} Nerves which are commonly blocked include: obturator, sciatic, tibial, femoral, paravertebral, thoracodorsal, musculocutaneous, median, and ulnar.\textsuperscript{16,27}

Gunduz, et al\textsuperscript{28} conducted a study in which peripheral nerve blocks were used to treat spasticity in spinal cord injured patients. The results showed that peripheral nerve blocks were effective in relieving spasticity, but only on a temporary basis.

Keenan, et al\textsuperscript{29} performed a study with percutaneous block of the musculocutaneous nerve to control elbow flexor spasticity. The results revealed that this method provides a reliable and temporary means for reducing spasticity. The authors felt it would be especially helpful in those individuals who have potential to improve neurologically.

Side effects of peripheral nerve blocks are minimal. Patients may become anxious and need to undergo general anaesthesia or local anaesthetic may be applied, however, will not be effective if there is a need to penetrate more deeply.\textsuperscript{16}

Botulinum-A toxin, an exotoxin produced by the bacterium Clostridium Botulinum, is a new agent used for local intramuscular injections.\textsuperscript{30} It is injected directly into the muscle.\textsuperscript{7} The botulinum toxin blocks the presynaptic release of acetylcholine causing a neuromuscular block.\textsuperscript{30}
Snow et al,\textsuperscript{31} conducted a study with nine chronic multiple sclerosis patients who were confined to a bed or wheelchair and were residents of a long-term care facility. The study involved injecting the botulinum toxin into spastic adductors of the subjects to see if it would allow for easier nursing care. The results of the study revealed that there were significant improvements in muscle tone, frequency of spasm, and hygiene. No side effects were noted.

In a double-blind study testing the effectiveness of botulinum-A toxin to manage dynamic equinus deformity associated with cerebral palsy, Koman and associates\textsuperscript{32} reported improvement in the gait pattern of five out of six patients. Although no significant side effects were noted, soreness occurred at the site of injection in three of six subjects. However, this side effect also occurred with the placebo.
DORSAL RHIZOTOMY

Dorsal rhizotomy, more commonly referred to as selective posterior rhizotomy (SPR) or selective dorsal rhizotomy (SDR), is a neurosurgical procedure that reduces spasticity and hyperactive stretch reflexes by cutting through the sensory portion of selected posterior nerve rootlets in the lumbosacral area. They are most commonly performed on individuals with cerebral palsy; however, they have sometimes been used for spasticity in patients with myelomeningocele, multiple sclerosis, head injury and, spinal cord injury.

Surgery is performed with the patient under general anaesthesia and begins with a laminectomy or laminotomy medial to the facet joint of L2-L5 vertebrae. The posterior nerve roots and rootlets from L2 through S2 are identified and then stimulated electrically. Motor responses are monitored visually, by palpation, and via electromyography (EMG). Abnormal responses are considered to be: a duration sustained beyond the one second stimulus, spread of contraction to other muscles not innervated by the rootlet which was tested, and other irregular patterns of contraction. Those rootlets which present...
with abnormal responses are severed. All other rootlets are left intact.\textsuperscript{34,35} Generally, 25% to 60% of the sensory rootlets are clipped.\textsuperscript{35}

Success of SPR relies heavily upon appropriate patient selection and a commitment by the family and patient to participate in an aggressive postoperative physical therapy program.\textsuperscript{36} Patient selection should be conducted by a team and may include the following disciplines: physical therapist, occupational therapist, social worker, pediatrician, neurophysiologist, orthopaedic surgeon, and surgical neurologist.\textsuperscript{36} Candidate selection is very crucial because it attempts to rule out those who have motor impairments due to dystonia, athetosis, ataxia, and abnormal reflex posturing.\textsuperscript{5} Peacock and Staudt\textsuperscript{35} list the following six factors for the selection process: 1) Confirm diagnosis of CP; 2) Identify spasticity as a predominant interfering factor; 3) Rule out other forms of hypertonicity, such as rigidity and dystonia; 4) Evaluate range of motion (ROM) - osteotendinous deformities will not be helped by rhizotomy; 5) Evaluate underlying strength and selective control; 6) Goals should be carefully considered and discussed with patient and family prior to surgery.

The overall goal of patient selection is to select those individuals who have the potential to make some improvements from their present functional level.\textsuperscript{34-36} For example, a child who adducts severely during gait may have eased gait after the spasticity in the adductors has decreased. However, a child who does not have the potential to ambulate will not become ambulatory
simply from undergoing the surgery. In contrast, however, a child who relies on spasticity for movement may experience a decrease in function when that is taken away. Surgery can help to improve the quality and performance of skills that the child can already do and will heavily depend upon strength, motor control, absence of osteotendinous deformities, and other factors which exist prior to surgery.\textsuperscript{35} Selective posterior rhizotomy may be favorable to reduce severe spasticity in individuals to ease positioning and to inhibit formation of deformities.\textsuperscript{37} Several authors\textsuperscript{35,36} list characteristics desirable for SPR candidates (Table 1).

Contraindications for SPR include weakness of antigravity muscles, weakness or hypotonia of the trunk, major non-spastic motor disorders (athetosis, ataxia, rigidity, dystonia), severe spinal deformity or scoliosis, multiple fixed contractures, multiple prior extremity surgery (with exception), and mentally retarded (with exception).\textsuperscript{35,36} Children who have severe spastic quadriplegia may be unrealistic candidates because all rootlets which are stimulated react abnormally and a partial rhizotomy of 60\% to 70\% of all rootlets may only provide minimal long term relief.\textsuperscript{36}

Boscarino and associates\textsuperscript{38} examined the effects of selective dorsal rhizotomy on the gait pattern of children who had cerebral palsy. Nineteen children who were ambulatory (both independent and dependent upon a walking aid) underwent preoperative and postoperative (one year) gait analyses that included assessment of range of motion (ROM), muscle tone, motion
Table 1. Desirable characteristics of selective posterior rhizotomy candidates.

<table>
<thead>
<tr>
<th>Characteristic</th>
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<tr>
<td>Born prematurely</td>
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<td>Spastic diplegic</td>
</tr>
<tr>
<td>Age 2-10 years</td>
</tr>
<tr>
<td>Progressive motor development history</td>
</tr>
<tr>
<td>Evidence of selective motor control</td>
</tr>
<tr>
<td>Good trunk control</td>
</tr>
<tr>
<td>Good underlying strength</td>
</tr>
<tr>
<td>Potential ambulator (preferably independent ambulator)</td>
</tr>
<tr>
<td>Absence of fixed contractures</td>
</tr>
<tr>
<td>Minimal or no previous lower extremity surgery</td>
</tr>
<tr>
<td>Minimal or no osteotendinous changes</td>
</tr>
<tr>
<td>Absence of postural hypotonia</td>
</tr>
<tr>
<td>Motivation/commitment to postoperative physical therapy</td>
</tr>
<tr>
<td>Intelligence (IQ &gt; 80) for effective therapy participation</td>
</tr>
<tr>
<td>Family support</td>
</tr>
<tr>
<td>Possibility of eased nursing care</td>
</tr>
<tr>
<td>Prevent/stop severe joint contractures and subluxations</td>
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analysis in three dimensions, and electromyography during gait. Examiners noted improvements in passive ROM, lower extremity spasticity, and sagittal plane hip, knee, and ankle motion. Additionally, on the average there was a greater amount of plantargrade foot position during stance phase. No changes were noted in coronal plane motion of the hip and pelvis. An increase in anterior pelvic tilt position in the independent ambulators was noted as the only negative change.

Park and colleagues\(^\text{39}\) examined how dorsal rhizotomy affects lateral hip migration in children with spastic diplegia. A migration percentage was calculated through preoperative and postoperative radiographs of the hips of 67 children. All children were between the ages of two and eleven at the time of surgery. Follow-up periods ranged from six to 46 months. The migration percentages remained unchanged in 75%, decreased in 17%, and increased in 7% of all patients. The examiners felt that results suggested that lateral hip migration is halted by selective dorsal rhizotomy in the great majority of cases of children with spastic diplegia.

McLaughlin and associates\(^\text{40}\) conducted an observational study of 34 consecutive children who received SDR at their facility. Twenty-four of the children had spastic diplegia and ten had spastic quadriplegia. All children received one month of postoperative physical therapy and received physical therapy prescriptions in their own communities for one year. They were assessed preoperatively and one year after surgery. The following tools were used for assessment: Ashworth scale, which is a scale for grading spasticity; range of motion; deep tendon reflex responses; and the Gross Motor Function Measure,
which measures gross motor skills and changes in children with cerebral palsy. Results revealed a great deal of variability with each individual; however, often spasticity in the lower extremities was reduced and functional capabilities improved with SDR. The authors felt it necessary to conduct further studies to determine the effectiveness of SDR.

None of the studies\textsuperscript{38-40} listed any major complications as a result of surgery. McLaughlin, et al\textsuperscript{40} report some mild complications in a small number of children postsurgically, such as mild transient paresthesias lasting two weeks or less, dysesthesias for more than two months, postoperative bladder incontinence as a result of urethrities which resolved with antibiotic treatment. Additionally, some had reported back or hip pain for two weeks to six to nine months, intermittent mild back pain for approximately six months. Peacock and Staudt\textsuperscript{35} list the possibility of the following postoperative problems: hypersensitivity of the feet and legs and flexor spasms early following surgery; crouched posture; poor tibial control; ankle valgus; weakness of trunk, hip, quadriceps, calves and dorsiflexors. Yet other authors\textsuperscript{41,42} list these possible temporary complications: kyphosis, wound infection or meningitis, cerebral spinal fluid leak, motor paralysis, transient or permanent sensory loss, bladder or bowel dysfunction, valgus or varus foot position, transient muscle weakness and hypotonia, risk from anaesthesia.
Spasticity is a very complex and intriguing phenomenon. It is an entity with which physical therapists are quite frequently faced. Although it may be beneficial in some cases, it is more commonly a major source of disability to those who have sustained an insult to the central nervous system. When it becomes too severe, it can cause multiple complications which interfere with activities of daily living. It is for this reason that management of spasticity becomes necessary.

Physical therapists will frequently be directly involved in the treatment of individuals with spasticity, including approaches to decrease unwanted spasticity. However, often this will not suffice and medical management will become necessary.

Medical management may include the use of pharmacological agents or neurosurgical techniques, such as selective dorsal rhizotomy. Pharmacological treatment may include medications or local injections with various agents. These methods are temporary and the effects only last for short periods unless use is continued and/or repeated. The commonly used oral medications are diazepam, dantrolene sodium, and baclofen. Baclofen may be administered orally as well as
intrathecally. Local treatment includes injections with phenol, alcohol, a local anaesthetic agents, or the botulinum-A toxin.

Dorsal rhizotomy may be an option for spasticity management if other methods have proven ineffective. Its effects are permanent and irreversible because nerve rootlets are actually severed.

The particular method of management will depend upon the severity of spasticity and the goals to be achieved with reduction. The pros and cons of each method will need to be considered carefully in order to select the most appropriate treatment. Physical therapists may not prescribe pharmacological or surgical treatments; however, they may be a member of a multidisciplinary team which assesses and sets goals for patients. The physical therapist serves a vital role by contributing information as to whether or not an individual could improve functionally if spasticity were reduced.
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