Spasticity in children

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Abstract Spasticity is a wide spectrum disease that affects all age groups. Spasticity in children may have diverse etiologies; this article will focus on children with spasticity, most of whom have diagnoses of cerebral palsy; as approximately two thirds of all cerebral palsy patients suffer from spasticity. This review is meant to discuss various aspects of spasticity; nomenclature over successive decades, clinical manifestations; including clinical examination tips, etiology, pathogenesis and pathophysiology were also high lightened. We are discussing well established, as well as other recent measures of management spasticity in view of diagnostic, as well as therapeutic approaches; including two clinical vignettes.

Introduction

Spasticity may result from either diffuse or localized pathology of the cerebral cortex, brain stem, or spinal cord; Cerebral palsy is considered a main cause of spasticity in pediatric age group as 2/3 of all cerebral palsy patients suffer from spasticity. Management of spasticity is challenging as it is important to evaluate the advantages and disadvantages that the patients gain from their spasticity so that treatment strategies and goals can be identified. Prenatal care during pregnancy can avoid premature labor; prevent cerebral palsy in infants with its resulting spasticity. Despite major updates and innovations in the field of treatment of spasticity, there is no specific diagnostic test for spasticity.

Historical note and nomenclature

Treatment for spasticity was documented as early as the late 19th century, when surgeons Abbe and Bennet discussed the decreasing tone in a spastic limb through sensory rhizotomies. Later, in 1898, the scientist Sherrington published experiments in which the sensory roots of spastic cats were severed to relieve spasticity. The technique of sensory rhizotomies has been improved on and continues to be used today as a treatment for patients with spasticity as does neuromuscular blockade, a longstanding treatment, which has been used for over 30 years. The technique of sensory rhizotomies has been improved on and continues to be used today as a treatment for patients with spasticity as does neuromuscular blockade, a longstanding treatment, which has been used for over 30 years.

Clinical manifestations

Spasticity was defined by Lance as a “velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex”. Young further added characteristics of positive and negative symptoms. Positive symptoms consist of exaggerated
cutaneous reflexes, including nociceptive and flexor withdrawal reflexes, autonomic hyperreflexia, dystonia, and contractures. Negative symptoms include paresis, lack of dexterity, and fatigability.

Although there are many possible causes of spasticity, this article will focus on children with spasticity, most of whom have diagnoses of cerebral palsy; approximately two thirds of all cerebral palsy patients suffer from spasticity. A patient with spastic cerebral palsy presents with muscle imbalance, stands with bent knees and legs tightly together, and in severe cases, a scissors-type gait. The antigravity muscles are predominantly affected with arms in a flexed and pronated position and legs in an extended and adducted position. When the muscles are at rest they are flaccid to palpation and electromyographically silent.

Examination should begin with the patient in a relaxed, lying position with the head up and arms resting to the sides because it is easier to determine the extent of spasticity in this position. The examination should include tonic stretch reflexes by manual passive stretches, elicitation of tendon jerks and clonus in a relaxed position, and tonic and phasic stretch reflexes carried out in a sitting position.

The manual passive stretch maneuver is used to assess resistance at different rates. A joint is passively moved while the muscles corresponding to that joint are lengthened and shortened. In cases of mild spasticity, the muscles will only resist when stretched at a high rate, whereas in cases of moderate spasticity, resistance is noticed at a slower rate and the clasp-knife phenomenon may be exhibited. Movement of the muscle may be difficult to impossible in cases of severe spasticity. The muscle tone is graded according to the Ashworth scale, a scale ranging from 1 to 5, in which resistance to the passive muscle stretch is measured at various velocities.

Tendon jerks are easier to elicit in spastic patients than in patients with normal muscle tone, and reflex responses can be achieved in muscles without well-defined tendons. Percussion of the tendon reveals hyperactive tendon jerks, especially for the Achilles, patellar, biceps, and triceps tendons. On EMG, the jerks show greater amplitudes than are normal and are followed by after-discharge of the motor units that is often slightly longer lasting than normal. The size of tendon jerks can be measured by either EMG response or by recordings of mechanical events.

H-reflex (Hoffmann's reflex) studies are electrically elicited tendon jerks and are restricted mostly to the soleus and flexor carpi radialis muscles in normal adults. In cases of upper motor neuron lesions, the H-reflex may be elicited in muscles where it is not normally seen, such as the intrinsic hand muscles, tibialis anterior, or peroneal muscles.

Measurement of resistance to passive stretch, reduction in the tonic vibration reflex, and reduction of the plantar withdrawal reflex should also be evaluated. Motoneuronal overactivity should also be evaluated because any input to motoneurons produces excessive and prolonged activity that can be observed in the contractions of many limb muscles.

The amount of function the patient derives from spasticity can be evaluated by having the patient obtain and maintain standing and seated positions. To determine the degree to which the hamstring tone is affecting the alignment of the pelvis and knees, have the child sit with feet straight in front. The patient can sit in a chair to allow the examiner to assess trunk control. The side sit position exhibits a patient's ability to maintain control in an asymmetric position. Video cameras are often helpful during evaluation as the patient's movements can be recorded and compared against movements during and after treatment.

It is important to evaluate the advantages and disadvantages that the patient gains from their spasticity so that treatment strategies and goals can be identified. Disadvantages may include interference with activities of daily living, inhibition of good sleep, contractures, dislocations, skin breakdown, bowel and bladder dysfunction, impairment of respiratory function, pain with stretching, and the masking of the return of voluntary movement. However, patients may rely on a certain amount of spasticity to function and the advantages they may receive include maintaining muscle tone, supporting circulatory function, assisting in activities of daily living, and preventing the formation of deep vein thrombosis.

Clinical vignette

Patient A was a 5-year-old African–American boy with a history of developmental delay and a diagnosis of cerebral palsy of the spastic-diplegic type. His first presentation at 18 months with severe spasticity in both lower extremities. The patient walked on tip toes and had hip and knee flexion. There was some scissoring of the legs. On examination, exaggerated deep tendon reflexes were elicited, as were sustained clonus and bilateral Babinski sign. MRI of the brain showed findings that may be secondary to previous hypoxic injury, compatible with cerebral palsy. Prior treatments included physical therapy, bilateral ankle-foot orthosis, serial casting, and oral baclofen. Before treatment with botulinum toxin injections, the patient was a tip-toe walker. With treatment the patient's gait has improved; he is flat-footed and presently wears bilateral ankle-foot orthosis. His hygiene and positioning have also improved and he returns every 6 months to 9 months for reinjection.

Patient B was a 7-year-old African–American boy with a history of cerebral palsy of the spastic-diplegic type. On primary examination he presented with tightness of both hamstrings and heel cords with the right more involved than the left. The patient had good toe standing, especially on the right side and good sitting balance with a kyphotic sacral-type sitting due to the tight hamstring. He uses a walker to ambulate and walks on tip toes. The EEG was abnormal, indicating the presence of epileptiform activity from the left central parietal head region and diffuse background disorganization, which indicates underlying neuronal dysfunction. Treatments before intrathecal baclofen pump implantation included bilateral ankle-foot orthoses, tendon releases, alcohol block, and botulinum toxin injections. Before treatment with intrathecal baclofen the patient was dependent on a care giver and used a walker to ambulate. With the intrathecal baclofen pump the patient has gained function, does not use a walker to ambulate, and performs activities of daily living independently.

Etiology

Spasticity may result from either diffuse or localized pathology of the cerebral cortex, brain stem, or spinal cord. Possible causes of such injuries include cerebral palsy, traumatic brain injury, stroke, multiple sclerosis, spinal cord trauma, or disease and anoxic insults. The neurologic localization of the lesion causing spasticity may result in different clinical manifesta-
tions. Thus, it is important to consider whether the spasticity results from cerebral pathology, whether it is diffuse or localized, or whether it is a result of spinal cord injury. Diffuse cerebral injury or diseases would include anoxia, toxic, or metabolic encephalopathies, whereas localized cerebral injury would include tumor, abscess, cyst, arteriovenous malformations, hemorrhage, or trauma. Spinal cord injury or disease may result as an insult to descending pathways by trauma, inflammatory or demyelinating disease, degenerative disorders, or compression such as is caused by a tumor or cyst.19,26,3 Common causes of cerebral palsy in children that may result in spasticity are prolonged second stage labor, fetal distress, cystic degeneration of the brain, prematurity, periventricular encephalomalacia, cortical abnormalities such as porencephaly, or congenital malformations of gyri such as micropolygyria. The annual incidence of spinal cord injuries in the United States is estimated to be 30–40 new cases per million individuals. About 3–5% of cases each year occur in children younger than 15 years of age.45 The male-to-female ratio of patients is 4:1 in the general population, but in younger age groups, the ratio is approximately 1.5:1.59

Pathogenesis and pathophysiology

There are many different types of spasticity. Because of this, more than one mechanism may be responsible for the disturbance in muscle tone and the mechanisms may vary between patients. The neuropathophysiologic processes involved in spasticity are complex and not fully understood, but there is a widely accepted hypothesis that spasticity depends on hyper-excitability of spinal alpha motor neurons, which is due to the interruption of descending modulatory influences carried by the corticospinal, vestibulospinal, and reticulospinal tracts and other possible tracts.24 Ia afferent fibers provide segmental input from muscle spindles to alpha motor neuron pools. They synapse on segmental inhibitory interneurons that then inhibit alpha motor neurons innervating antagonist muscles in the Ia reciprocal inhibition pathway. Ib afferents inhibit alpha motor neurons by way of the Golgi tendon organs via the Ib inhibitory interneuron in another pathway known as nonreciprocal inhibition.24,57 Increased excitation of these afferents does not seem to be the cause of spasticity. Instead, evidence supports that reduced reciprocal inhibition of antagonist motor neuron pools by Ia afferents, decreased presynaptic inhibition of Ia afferents, and decreased nonreciprocal inhibition by Ib terminals are all possible pathophysiologic mechanisms of spasticity.57 The pathophysiology of traumatic brain injury involves a complex combination of forces that has been a subject of substantial debate.21 On occasion, autonomic dysreflexia may occur after an intramuscular injection, although this is relatively rare.49 In some patients, autonomic dysreflexia may occur even if the level of spinal injury is below T6.7,37 The use of antihypertensive pharmacologic agents in treating spasticity is unclear because randomized trials have not been performed. Nifedipine has been used in a bit-and-swallow technique; more recently, captopril also has been found to be of benefit.22

Epidemiology

As stated before, spasticity is present in about two thirds of cerebral palsy patients, and cerebral palsy affects anywhere from 1.5 to 2.5 per 1000 live births in the United States.3 The number of spastic patients continues to increase due to an increased survival rate of premature births. Males and females are equally affected.

Prevention

To prevent cerebral palsy; due to preterm labour; and, thus, the resulting spasticity, it is important that mothers receive prenatal care during pregnancy, that measures are taken to avoid premature labor, and that special consideration is given to pregnancies involving multiple gestations. Early detection and treatment of other causes of cerebral palsy, e.g. neurodegenerative diseases may prevent the development of spasticity as well as detect the underlying diseases that could result in brain injury. If children have conditions that make them susceptible to brain or spinal cord injury or both, safety measures should be taken (i.e., helmets for patients who have frequent seizures).

Differential diagnosis

Spasticity can be confused with rigidity when a patient is being evaluated. Stretching can distinguish rigidity from spasticity. Rigidity will relax through repeated stretching of a muscle, whereas a spastic muscle will continue to increase in resistance as the velocity of the stretch is increased.19,57

Diagnostic workup

There is no specific diagnostic test for spasticity. Testing can be done to establish the presence of any lesions or brain or spinal cord injury. MRI of the brain can be performed to rule out periventricular leukomalacia. A baseline EEG to establish underlying seizure activity can also be done as well as basic lab studies. Neurophysiological studies, such as the H-reflex study, may be performed in patients with neurodegenerative disease; an enzymatic assay should also be performed.

Prognosis and complications

Spasticity results in limited functional capacity and increased inactivity. The sequelae of this inactivity may include decubitis, cardiovascular problems, thrombophlebitis, respiratory infections, fixed contractures, osteoporosis,33 bladder and bowel problems, and social isolation. Ultimately, these consequences of inactivity may lead to a further decrease in strength and function.25 The patient’s quality of life may be compromised as spasticity has negative impacts on mobility, hygiene, self care, sleeping patterns, self esteem, mood, and sexual function.

Management

Traditional treatments for spasticity include physical and occupational therapy, in which the patient is stretched anywhere from once daily to several times per day, but this has only a limited effect on the patient’s spasticity. Rehabilitation treatment options include casting, orthotics or splints, strengthening, electrical stimulation, practice of functional tasks, sensory integration; muscle stretching, and targeted muscle training.23 Within the scope of pediatric neurorehabilitation, distinct
diseases can produce specific complications. These complications; however, can also occur in association with many disorders. For example, spasticity from injury to the upper motor neuron unit can develop in many neurologic disorders in children. Several of these complications, such as autonomic dysreflexia, deep vein thrombosis, and heterotropia ossification, can be severe and potentially life-threatening. 20,18

Oral medications can be used to decrease spasticity; however, many have unwanted side effects such as drowsiness, sedation, confusion, and fatigue. Benzodiazepines, such as diazepam, are rarely used because of their strong sedating effects. They result in enhanced presynaptic inhibition, but because they are presumed to enhance the postsynaptic effects of GABA, they can only work if the GABA-mediated process functions. Benzodiazepines have a long half-life and an active metabolite. Benzodiazepine therapy is indicated in spinal cord injury and multiple sclerosis with possible application in traumatic brain injury, cerebral palsy, and cerebrovascular accident. Clinical effects include sedation and reduced anxiety, decreased resistance to passive range of motion, decreased hyperreflexia, and reduction in painful spasms. Side effects of all benzodiazepines include sedation, weakness, hypotension, gastrointestinal symptoms, memory impairment, incoordination, confusion, depression, and ataxia. Also, benzodiazepines are controlled substances with the potential for dependency. Diazepam is the most widely used benzodiazepine for spasticity management. The recommended initial dose is 250 mcg/kg three times daily with a maximum dose of 60 mg daily (20 mg three times daily). If nocturnal spasticity is the presenting problem the patient should be started with a single dose at night.

Another group of oral medications used in spasticity management includes clonidine and tizanidine, which are alpha2, nonadrenergic receptor agonists that release excitatory neurotransmitters and inhibit supraspinal facilitatory pathways. 57,25 Tizanidine is an oral antispasticity agent that is selective in decreasing tone and spasm frequency in only spastic muscles, eliminating the unwanted side effect of generalized muscle weakness. Tizanidine is reported to have reduced symptoms of spasticity in patients with multiple sclerosis or spinal cord injury and is well tolerated in most patients. It is an imidazoline derivative similar to clonidine but without the cardiovascular effects when appropriately titrated. Tizanidine results in a direct reduction of excitatory amino acid release from spinal interneurons and inhibits facilitatory caeruleospinal pathways. Its peak effect occurs 1–2 h following administration and its half-life is 2.5 h. The clinical effects of tizanidine include reduced muscle tone, spasm frequency, and hyperreflexia. Animal studies with tizanidine demonstrate antinociceptive activity under specific conditions with increased dose titration. 17,14,53,39 As with other antispasticity medications, the potential side effects of tizanidine are dose related and may be mitigated by dosage titration. The potential side effects include drowsiness, dry mouth, and dizziness. Literature suggests that tizanidine may be better tolerated than other antispasticity agents as measured by the global tolerance rating scale. 38 In placebo-controlled studies, tizanidine has been shown to be effective in multiple sclerosis and spinal cord injury. It is also useful for spasticity of spinal pathology when weakness is of concern. Tizanidine may also prove effective in managing spasticity of cerebral origin. 40

Secondary oral and systemic agents include tiagabine, cyproheptadine, clonidine, lamotrigine, gabapentin and carbidopa-levodopa. 27 Multiple medications have been recommended, of which the most recent addition is gabapentin. 59

Other treatments include chemical neurolysis, in which the nerve conduction is impaired through the use of chemical agents and therapeutic nerve block using phenol or alcohol. The goals of these treatments are to prevent muscle contractions and improve the patient’s function. A common side effect is that after the nerve is injected, alcohol levels measure above the legal limit in children. Other side effects include damage to sensory and motor nerves, pain at injection site, scarring, and dysesthesias. To ensure the correct site, injection must be made using an electrical stimulator. 27,25

Another treatment used alleviate spasticity in children with cerebral palsy is rhizotomy. Studies have shown that performing selective dorsal rhizotomy at a young age can reduce the need for orthopedic surgery. 14 Goals of rhizotomy are decreased tone, increased mobility, and the facilitation of care for the patient, however, the reduction in spasticity cannot be predicted and sometimes results in excessive hypotonia. 29 The procedure is very meticulous, requiring general anesthesia and a neurophysiologist who must be present to identify which nerve is to be severed.

Other neurosurgical approaches include peripheral neuroectomy, myelotomy, and dorsal column electrical stimulation.

Orthopedic procedures are the most frequently performed operations for spasticity. The targets of these operations are muscles, tendons, or bones. Muscles may be denervated and tendons and muscles may be released, lengthened, or transferred. The goals of surgery may include reducing spasticity, increasing range of motion, improving access for hygiene, improving the ability to tolerate braces, or reducing pain. Orthopedic problems that may result from a spastic limb include cubital or carpal tunnel syndrome, spontaneous fracture, gait difficulties, dislocation of the hip or knee, and heterotopic ossification.

The most common orthopedic procedure for the treatment of spasticity is a contracture release. In this procedure, the tendon of a muscle that has a contracture is partially cut, completely cut or lengthened. The joint is then positioned at
Alternative medicine in spasticity management

Physical modalities include electrical stimulation and cold temperature, acupuncture and homeopathic approaches, herbs and hyperbaric oxygen, constraint induced training, the Adeli suit, conductive education, craniosacral, and manipulation and patterning.

Context therapy is a new intervention approach that focuses on changing the task and the environment rather than children’s impairments. It can be a viable treatment to achieve parent-identified functional goals for children with cerebral palsy.

New treatments for spasticity, such as botulinum toxin type A, have proved easier, more effective, and less painful for patients. First clinically introduced in the United States in the early 1980s, botulinum toxin is a potent neurotoxin derived from the anaerobic bacteria Clostridium botulinum, but when used in treatment, no serious systemic toxin effects have been reported. The medication is more costly than alcohol or phenol but the cost is offset by less physician time and the lack of anesthesia. The formation of antibodies has been a concern, but this can be prevented by allowing 2–3 months between injections. Botulinum toxin works by acting in the neuromuscular junction, preventing the release of acetylcholine, which results in functional denervation. It can be given without EMG and anesthesia, does not cause dysesthesias, and is no more painful than an injection of saline solution. Effects are local and last 3–4 months, or longer. It is contraindicated during pregnancy, lactation, in individuals with neuromuscular disorders (such as myasthenia gravis), in patients taking aminoglycosides, or in those who have a known allergy to the drug. Adverse effects are not common and are usually associated with the site of injection, such as bleeding, bruising, and soreness or redness at the injection site, or diffusion to nearby muscle groups. In patients that do not respond to botulinum toxin, possible reasons should be considered before labeling the patient as unresponsive. Reasons could be related to injection technique, improper toxin storage, or the patient’s individual characteristics. Overall, botulinum toxin has proven clinically to be effective, safe, and less painful than other invasive therapies. Botulinum toxin is available in serotypes A and B, which have different unit potencies, side-effect profiles, and dilution schedules. Both have been used in children with cerebral palsy, although serotype A has been used more extensively. Dosing guidelines have been suggested for botulinum toxin A for adult and pediatric patients. Adult recommendations are available for botulinum toxin B, but studies are ongoing for pediatric patients.

Several studies have reported the successful use of botulinum toxin A for the treatment of drooling in children with cerebral palsy, using injection into the submandibular or parotid glands alone or in combination with other agents. In some studies, the beneficial effects have lasted for up to 4 months without serious side effects or disturbances of oral function.

It has been established that oral baclofen does not cross the blood–brain barrier effectively and that higher doses of the medication result in serious side effects. Intrathecal baclofen results in a greater decrease in spasticity by allowing higher concentrations of baclofen in the cerebrospinal fluid at about 1% the daily oral dosage. To be considered for intrathecal baclofen pump placement, the patient must have severe lower limb spasticity that does not respond to other less-invasive treatments. The patient must first be given a trial of 50 μg baclofen through a lumbar puncture or spinal catheter. If unresponsive, 75 μg can be tried after 24 h and a third trial of 100 μg can be tried 24 h after that, after which if the patient is still unresponsive he or she must be excluded from the treatment. Implantation lasts 1–2 h and the pump is easy to refill subcutaneously. It is programmed by a computer-controlled radiotelemetry programmer that is linked to the pump’s internal computer and that selects the rate and pattern of baclofen.
Complications to intrathecal baclofen include hypersensitivity to baclofen, intolerance to the side effects of baclofen including drug tolerance, cerebrospinal fluid leakage, pump pocket seroma, hematoma, infection, and soft tissue erosion. The objective of intrathecal baclofen is to individualize the patient’s dose and infusion so that the lowest dose that yields the greatest response can be achieved. In comparison, intrathecal baclofen has less complications and side effects than other treatments and more generalized results in both cerebral and spinal spasticity, making intrathecal baclofen the most effective current tool for the treatment of spasticity in non-ambulant individuals. A recent systematic review showed that there was no evidence to support the clinical use of intrathecal baclofen in ambulant individuals with hypertonicity without further rigorous longitudinal studies.

As a precaution, families are prescribed diazepam or diazepam rectal as well as oral baclofen to have at home. If there is evidence of withdrawal, one of these medications is administered, and the patient is instructed to go immediately to the emergency department. Although aggressive use of benzodiazepines and oral baclofen may be helpful, recognition and return to appropriate intrathecal baclofen dosage is essential for rapid recovery.

The goals of and benefits to the patient are important when considering the path of treatment. In some cases, function will not return, but treatment can result in pain reduction and allow easier management of patient care. Common goals are to decrease pain, prevent or decrease contractures, improve ambulation, facilitate activities of daily living, facilitate rehabilitation participation, save caregiver’s time, improve the ease

### Table 1: Useful tests for diagnosis.

<table>
<thead>
<tr>
<th>Test</th>
<th>Use</th>
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<tbody>
<tr>
<td>Basic lab studies (electrolytes, bone profile, etc.)</td>
<td>Metabolic derangement</td>
</tr>
<tr>
<td>Enzymatic assay (Arylsulfatase, Neuraminidase, etc.)</td>
<td>Neurodegenerative disease</td>
</tr>
<tr>
<td>EEG</td>
<td>Underlying seizure activity</td>
</tr>
<tr>
<td>NCS</td>
<td>Neurodegenerative disease (Leukodystrophies)</td>
</tr>
<tr>
<td>MRI of the brain</td>
<td>Periventricular leukomalacia</td>
</tr>
</tbody>
</table>

### Table 2: Therapeutic interventions for spasticity.

<table>
<thead>
<tr>
<th>Therapeutic intervention</th>
<th>Mechanisms</th>
<th>Major points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-pharmacologic treatments</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical and occupational therapy</td>
<td>Stretching exercises anywhere from once daily to several times per day</td>
<td>Only a limited effect on the patient’s spasticity</td>
</tr>
<tr>
<td>Rehabilitation program</td>
<td>Splints, strengthening, electrical stimulation, practice of functional tasks, sensory integration, muscle stretching, and targeted muscle training</td>
<td>Mainstays and cornerstones in spasticity management; complications, such as autonomic dysreflexia, deep vein thrombosis, and heterotropic ossification, can be severe and potentially life-threatening Temporary effect</td>
</tr>
<tr>
<td>Casting and orthosis</td>
<td>Extend joint range diminished by hypertonicity; reduce an abnormal pattern by positioning</td>
<td>Permanent effect; sometimes results in excessive hypotonia In moderate to severe spasticity; permanent effect</td>
</tr>
<tr>
<td>Selective posterior rhizotomy</td>
<td>Balancing spinal cord-mediated facilitatory and inhibitory control</td>
<td></td>
</tr>
<tr>
<td>Orthopedic surgery</td>
<td>Corrects deformity induced by muscle overactivity involving muscles, tendons, or bones</td>
<td></td>
</tr>
<tr>
<td>Pharmacological treatments, oral medications</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Benzodiazepine</td>
<td>Increases the affinity of GABA for GABA-A receptors; inhibitory effect at both the spinal cord and supraspinal levels</td>
<td>Short-term treatment; strong sedating effects</td>
</tr>
<tr>
<td>Dantrolene sodium</td>
<td>Inhibits release of calcium from sarcoplasmic reticulum in muscle; works peripherally at the muscle fibers</td>
<td>Serious side effects; hepatotoxicity in 1% patients; respiratory muscle weakness</td>
</tr>
<tr>
<td>Baclofen</td>
<td>GABA agonist; binds at the GABA-B receptor; restricts calcium influx into presynaptic nerve terminals in the spinal cord</td>
<td>Rapidly absorbed after oral administration; levels in the CSF are low because of low lipid solubility</td>
</tr>
<tr>
<td>Tizanidine</td>
<td>Centrally acting alpha-2 noradrenergic agonist; inhibits release of excitatory neurotransmitters in the spinal cord and supraspinally</td>
<td>Drowsiness, dry mouth, and dizziness; monitor liver function</td>
</tr>
<tr>
<td>Pharmacological treatments, chemodenervation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol/phenol block</td>
<td>Nonselective proteolytic agents; selective denervation when injecting into motor nerves or muscles</td>
<td>Damage to sensory and motor nerves; painful dysesthesias</td>
</tr>
<tr>
<td>Botulinum toxin injection</td>
<td>High affinity and specificity to the presynaptic membranes of cholinergic motor neurons</td>
<td>Recommended as effective treatment; no sensory disturbance</td>
</tr>
<tr>
<td>Pharmacological treatments, other</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intrathecal baclofen pump</td>
<td>Using a programmable implanted pump, baclofen can be delivered intrathecally</td>
<td>Severe, generalized spasticity; less complications and side effects</td>
</tr>
</tbody>
</table>
of care, and increase safety. Appropriate management choices are based on therapeutic objectives. Physical and occupational therapists can play a key role in identifying these objectives. Treatments with the fewest side effects are usually given prior to other options. Both the patient’s and the caregiver’s goals must be considered.

A summary of therapeutic interventions is provided in Tables 1 and 2.

16. Pregnancy

The patient with spasticity may expect to have a difficult pregnancy and delivery as well as difficulty managing and caring for an infant. Close medical follow up throughout pregnancy is recommended, hospitalization, immediate post natal care, etc.

Anesthesia

Not applicable.

References


