Spasticity

Spasticity is a condition in which certain muscles are continuously contracted. This contraction causes stiffness or tightness of the muscles and can interfere with normal movement, speech, and gait. Spasticity is usually caused by damage to the portion of the brain or spinal cord that controls voluntary movement. The damage causes a change in the balance of signals between the nervous system and the muscles. This imbalance leads to increased activity in the muscles. Spasticity negatively affects muscles and joints of the extremities, and is particularly harmful to growing children.

Prevalence and Incidence

- Spasticity affects more than an estimated 12 million people worldwide.
- About 80 percent of people with cerebral palsy (CP) have varying degrees of spasticity. With an estimated 500,000 people in the United States with some form of CP, this equates to about 400,000 people with some degree of CP-related spasticity.
- About 80 percent of people with multiple sclerosis (MS) have varying degrees of spasticity. With an estimated 400,000 people in the United States with MS, this equates to about 320,000 people with some degree MS-related spasticity.

Other conditions that may cause spasticity include:

- Traumatic brain injury (TBI)
- Spinal cord injury (SCI)
- Brain damage due to a lack of oxygen
- Stroke
- Encephalitis
- Meningitis
- Adrenoleukodystrophy
- Amyotrophic lateral sclerosis (Lou Gehrig's disease)
- Phenylketonuria

Spasticity in Cerebral Palsy (CP)

In a person with CP, brain damage has occurred. For unknown reasons, the damage tends to be in the area of the brain that controls muscle tone and movement of the arms and legs. Therefore, the brains of people with CP are unable to influence the amount of flexibility muscles should have. The command from the muscle itself dominates the spinal cord and results in muscles that are too tense or spastic. People born with CP do not have deformities of the extremities present at birth, but develop them over time. Spasticity of muscles, along with the limitations on stretching and use of muscles in daily activities, is a major cause of these deformities.

Spasticity in Multiple Sclerosis (MS)

Spasticity is a very common symptom of MS. There are two types of MS-related spasms: flexor and extensor. Flexor spasticity is an involuntary bending of the hips or knees (primarily involving the hamstring muscles on the back of the upper leg). The hips and knees bend up toward the chest. Extensor spasticity is an involuntary straightening of the legs. Extensor spasticity involves the quadriceps (muscles on the front of the upper leg) and the adductors (inner thigh muscles). The hips and knees remain straight with the legs very close together or crossed over at the ankles. Spasticity may also occur in the arms, but is less common in people with MS.

Spasticity may worsen due to sudden movements or position changes, muscle tightness, temperature extremes, humidity, or infections, and may even be triggered by tight clothing.

Spasticity in Traumatic Brain Injury (TBI)

Spastic hypertonia often occurs after a TBI as a result of damage to the brain stem, cerebellum or mid-brain. This damage affects the reflex centers in the brain, interrupting message flow along different nerve pathways. This disruption can cause changes in muscle tone, movement, sensation and reflex. The location of the TBI may determine which areas of the body are affected and what motor deficits occur. The reflex centers in the brain are more complex than those in the spinal cord. This can make the treatment of spastic hypertonia in individuals with TBI more difficult to treat than in persons with spinal cord injuries or neurological disorders.

Shortly after a brain injury, many people experience a period of increased muscle tone in which their body posture becomes very rigid. A common position is elbows held rigidly at the sides, wrists and fingers bent, and fists clenched. The legs are usually extended at the hips and knees with ankles and toes flexed. As a TBI patient recovers, nerve signals that control motor functions may change. Some signals may not reach the reflex centers of the brain, or the brain may send too many signals, causing the muscles to not respond properly.

Symptoms

Spasticity may be as mild as the feeling of tightness in muscles or may be severe enough to produce painful, uncontrollable spasms of the extremities; most commonly the legs and arms. Spasticity may also create feelings of pain or tightness in and around joints, and can cause low back pain.

Adverse effects of spasticity include:

- Muscle stiffness, causing movements to be less precise and making certain tasks difficult to perform
- Muscle spasms, causing uncontrollable and often painful muscle contractions
- Involuntary crossing of the legs
- Muscle and joint deformities
- Muscle fatigue
- Inhibition of longitudinal muscle growth
- Inhibition of protein synthesis in muscle cells

Additional complications

- Urinary tract infections
- Chronic constipation
- Fever or other systemic illnesses
- Pressure sores

Treatment Options

There are several types of treatment available which must be evaluated on a case-by-case basis, depending on the underlying cause, age of the patient, and severity of the spasticity. Different treatments share the common goals of:

- Relieving the signs and symptoms of spasticity
- Reducing the pain and frequency of muscle contractions
- Improving gait, hygiene, activities of daily living, and ease of care
- Reducing caregiver challenges such as dressing, feeding, transport, and bathing
- Improving voluntary motor functions involving objects such as reaching for, grasping, moving, and releasing
- Enabling more normal muscle growth in children

Physical and Occupational Therapy

Physical and occupational therapy for spasticity is designed to reduce muscle tone, maintain or improve range of motion and mobility, increase strength and coordination, and improve comfort. Therapy may include stretching and strengthening exercises, temporary braces or casts, limb positioning, application of cold packs, electrical stimulation, and biofeedback.

Oral Medications

The use of oral medications to treat spasticity may be indicated when symptoms interfere with daily functioning or with sleep. Effective medication management may require the use of two or more drugs or a combination of oral medications with another type of treatment. It is very important to work closely with a doctor to devise an individualized treatment plan. Side effects vary greatly by class of medication and patient.

Medications include:

- Baclofen
- Benzodiazepines
- Dantrolene sodium
- Imidazolines
- Gabapentin

Botulinum Toxin (BTA) Injections

BTA, also known as Botox injections have proven effective when used in tiny amounts, by paralyzing spastic muscles. Injection sites are carefully determined based on the pattern of spasticity.

When Botox is injected into the muscle(s), the release of acetylcholine is blocked, resulting in a relaxation of overactive muscles. The injection(s) generally take effect within a few days and last about 12-16 weeks, until new nerve endings grow back and the affected muscle(s) recover. Functional benefits may last longer than this. There are limitations in the number of injections that can be administered.

Surgery

The primary neurosurgical procedures to treat spasticity are intrathecal baclofen (ITB) pumps and selective dorsal rhizotomy (SDR).

Intrathecal Baclofen (ITB)

In severe cases of spasticity, baclofen can be administered through a pump that has been surgically implanted in the patient's abdomen. By delivering baclofen directly to the spinal fluid, a much more powerful reduction in spasticity and pain can be achieved, with fewer side effects. ITB has been found to be extremely effective in treating spasticity in the lower and upper extremities.

Selective Dorsal Rhizotomy (SDR)

In SDR, the neurosurgeon cuts selective nerve roots (rhizotomy), the nerve fibers located just outside the back bone (spinal column) that send sensory messages from the muscles to the spinal cord. SDR is used to treat severe spasticity of the legs that interferes with movement or positioning. By cutting only the sensory nerve rootlets causing the spasticity, muscle stiffness is decreased, while other functions remain intact. Decreasing spasticity can improve mobility and function, and help prevent severe muscle scarring (contractures), as well as joint and bone deformities. It is utilized most effectively in patients with CP who meet specific criteria. Individuals with the following criteria are **not** candidates for SDR:

- Patients who have experienced meningitis, congenital brain infection, congenital hydrocephalus unrelated to premature birth, head trauma, or familial disease
- Patients who have mixed CP with predominant rigidity or dystonia, significant athetosis, or ataxia
- Patients with severe scoliosis
- Patients who will not make functional gains after surgery

The benefits of surgery should always be weighed carefully against its risks. Randomized, controlled clinical trials have demonstrated that a large percentage of CP patients report significant reduction in spasticity and improved function after surgery. However, surgery is not an option for all cases of spasticity.