

# Spasticity

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*"One of the few things I will go to the doctor for is increased spasticity."*

*-- Woman with a spinal cord injury*

Spasticity is traditionally defined as a motor disorder characterized by velocity-dependent increase in toxic muscle stretch reflexes (muscle tone) with hyperactive tendon-jerks and occasional clonus. Real world cases of spasticity are often complex, making appropriate diagnosis and identification of treatment options challenging and sometimes frustrating for both patients and clinicians.

Spasticity (Greek *spastikos*, or to tug) is one feature of the upper motor neuron syndrome and manifests clinically as increased resistance to passive movements, taking the form of muscle stiffness or tightness. It may result in abnormal involuntary contractions of the voluntary muscles (spasm), which are often quite painful. Current understanding of the pathophysiology of spasticity is that it appears to be the result of an imbalance in excitatory and inhibitory synaptic activity at the motor neuron level of the spinal cord. Modulation of local spinal cord activity can be via a number of different descending and ascending neuronal pathways. With the exception of dantrium, medications used to treat spasticity (e.g., Valium, baclofen, zanaflex) aim to decrease excitation and/or increase inhibition by manipulating neurotransmitter activity in the spinal cord and or centrally.

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## THE BENEFITS OF SPASTICITY

Spasticity is a common symptom in myriad neuromuscular disorders including stroke (CVA), multiple sclerosis (MS), spinal cord injury (SCI) and tumors, cerebral palsy, and traumatic brain injury. Some degree of spasticity can be normal and even beneficial in certain situations. In cases of spasticity associated with weakness, a spasm may be useful and can be evoked to produce a desired limb movement (e.g., knee extension during a transfer or hip extension to help stand). It can be invaluable for maintenance of trunk posture and weight-bearing on weak arms or legs, and spasticity may help in preventing DVTs in paretic limbs and in maintaining muscle mass and bone density. It can also be useful in aiding bladder and bowel evacuation.

Treatment therefore should follow careful functional assessment of the patient with the goal of finding the right balance to meet the individual patient's needs and functional goals. Assessment tools include the Ashworth muscle tone scale, spasm scale, deep tendon reflexes, goniometry for range of motion, dynamometry for muscle strength, functional analysis (using scales such as the Functional Independent Measure, or FIM), and both pain and quality of life scales. Treatment goals include alleviating distressing symptoms (e.g., painful spasms), improving motor function, and preventing or reducing complications associated with excess spasticity.

## THE NEGATIVE EFFECTS OF SPASTICITY

Why treat spasticity? Spasticity can have a substantial detrimental impact on a person's physical and emotional well-being. Spasms can be painful and may be strong enough to throw a person out of bed or a wheelchair. Negative features of spasticity include weakness, fatigue, slowed initiation of movements and decreased dexterity. The secondary complications and consequences of untreated excessive spasticity include:

- **Sleep disturbances**, resulting from painful spasms or difficulty maintaining a comfortable position.
- **Contractions** can cause impaired positioning and lead to pressure sores, progressive scoliosis and bone deformities, and hip dislocations.
- **Chronic pain** can lead to depression, sleep problems, mood disturbances, poor nutrition and weight loss, and can increase social withdrawal.
- **Decreased functional mobility** can result as spasticity impairs transfers, ambulation, and an individual's ability to drive a car, wheelchair, or motorized scooter. Spasticity makes weight shifts difficult, which can lead to pressure sores, and increase the risk of disuse osteoporosis (and easier fractures).
- **Decreased self-care ability**, as spasticity can interfere with personal hygiene (e.g., leg adductor spasms), and bowel bladder functions.
- **Skin problems**, particularly in genital areas, can result from poor personal hygiene. Further, spasticity can cause increased maceration of skin at tight joints, and as noted above, can increase the risk for pressure sores.
- **Difficulty with care-giving tasks**, for those who are dependent on others for assistance with basic activities of daily living, such as personal hygiene, transfers, positioning. This can increase attendant care needs and costs.
- **Fatigue**, resulting from the increased energy cost for any movement.
- **Infections**, due to increases risk for UTI's and pneumonia (decreased lung secretions).
- **Self-esteem issues** can result from increased social isolation.

### *Spasticity as a Symptom*

It is important for the practitioner to recognize that an increase in spasticity may be an early symptom or warning sign of a myriad of problems, from relatively minor to potentially very serious (see below).

Spasticity may increase gradually over time in some situations. Immediately after a neurological event such as a CVA or traumatic SCI, the affected extremities may initially be flaccid, but typically become increasingly tight or spastic over subsequent months. Spasticity may also worsen over time in progressive diseases such as MS.

However, spasticity may be aggravated by many nociceptive factors, and particularly in women without normal sensation, may be the initial or even the only presenting symptom in conditions such as:

- urinary tract infection
- pyelonephritis
- constipation
- impaction
- decubitus ulcer
- burns
- ingrown toe nail
- vaginal infection
- pelvic inflammation disease
- fractures
- acute abdomen
- gall bladder disease

Atypical presentations of these conditions always represent a challenge. It is important to maintain a high index of suspicion when faced with any unexplained significant increase in spasticity.

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## PREVENTION AND TREATMENT OF SPASTICITY

Prevention management should include regular exercise program with stretching (both active and passive ROM) done at least once or twice a day, holding each joint at full range for 1-3 minutes. Proper positioning and seating is key, as they strongly impact the degree of spasticity. Physical and occupational therapy can be helpful in developing an effective exercise plan (including aquatic therapy when appropriate) and seating evaluation recommendations. Other physical medicine interventions include cryotherapy (icing), heat (ultrasound) treatments, orthotics, splinting, serial casting, tactile desensitization, weight bearing exercises (using parallel bars, standing frames, etc.), electrical stimulation (e.g., Transcutaneous Electrical Stimulation, or TENS, or Functional Electrical Nerve Stimulation, or FES), assistance devices (e.g., weighted utensils, Velcro shoes/zippers), relaxation techniques (guided imagery, meditation, music therapy) and seating/positioning aids (footsteps on wheelchair, knee blocks, adductor wedge, trunk/neck support).

In addition to physical medicine approaches, three other important spasticity management approaches include medications, regional and local pharmacologic injection options, and surgical management.

### *Medications*

The most common oral drugs used to treat spasticity are Baclofen, Dantrium, and the newer agent Tizanadine (Zanaflex). The benzodiazepines, particularly Valium and Klonopin, have also been used. More recently, Gabapentin (Neurontin) has been shown to be effective in off-label use, particularly in multiple sclerosis. It is important to note that the group of medications commonly referred to as "skeletal muscle relaxants," such as Flexeril, Soma, Robaxin or Skelaxin, may be indicated for relief of short term muscle

spasm related to an acute injury, but are NOT considered appropriate treatment for spasticity of spinal cord or central origin. Each of the medications used for spasticity has associated potential adverse side effects, and therefore careful review for each individual should be considered. Because the agents work at different sites, they may safely be combined in low doses, to minimize side effects and maximize function. Medication administration should always start at the lowest possible dosage and be titrated upward until an effective dose is determined.

**Table 1: Medications for Spasticity**

Medication	Dose	Adverse Effects	Concerns for Women
Lioresal (Baclofen)	5mg po bid-tid Titrate slowly up to max of 20mg qid. Intrathecal: usual dose range 50mcg - 900mcg/day	Sedation, dizziness, weakness, fatigue, dry mouth, urinary retention. Acute withdrawal syndrome, risk of seizures esp. with intrathecal	Pregnancy Class C., probably safe orally. Excreted in breast milk.
Tizanadine (Zanaflex)	2mg hs, titrate slowly up to: 2mg bid and 4mg hs. Max 12mg tid	Dizziness, somnolence, dry mouth. Hepatotoxicity: monitor LFT's.	Pregnancy Class C., possible increased risk of fetal loss. Probably excreted in breast milk. Potentiated by oral contraceptives.
Gabapentin (Neurontin)	100mg tid Titrate slowly up to 300mg tid Max: 900mg tid	Somnolence, impaired cognition. Generally well tolerated; check LFT's, WBC with higher doses.	Pregnancy Class C.
Dantrolene (Dantrium)	25mg qid Max 100mg qid	Excessive muscle weakness; Hepatotoxicity: monitor LFT's.	Pregnancy Class C. Estrogens may increase risk of hepatotoxicity.
Benzodiazepines: Diazepam (Valium) Clonazepam (Klonopin)	2mg - 4mg bid, titrate up to 60mg/day 0.5mg bid Max 3mg/day	Sedation, memory impairment, withdrawal syndrome	Pregnancy Class D (Valium), Class C (Klonopin). May cause withdrawal syndrome in infants if given late in pregnancy.

### *Regional and Local Injections*

Regional and local injections, such as motor point blocks or nerve blocks using alcohol and phenol, mostly have been used to treat spasticity of an individual extremity muscle group. These injections, however, can be quite painful and technically difficult, can produce scarring and painful dysesthesias, and have variable duration of effect. Local injections of botulinum toxin A is an alternative treatment that is increasing in popularity and often produces good functional outcomes and has little pain associated. "Botox" injections cause a neuromuscular blockade, whose effect is temporary (approximately 3 months usually), making repeat dosing often necessary. There is a possibility of antibodies developing, so that subsequent injections may not be as effective as the initial treatments. This is believed to be less likely with the newer Type B formulation. Botox may be a good treatment of choice for spasticity that is isolated in one or two extremities. As with oral medications, combining botox injection with physical therapy is beneficial and synergistic in effect, and a combination therapy approach is strongly recommended.

### *Surgical Management*

Surgical management of severe spasticity is rarely used today except in extreme situations when more conservative treatment approaches have failed. A new and exciting surgical option, however, is placement of a pump for administration of intrathecal baclofen (ITB). This allows for a continuous infusion of very low doses of baclofen (usually 100-1000 times less than oral baclofen dose) directly into the CSF to achieve effective antispasmodic management without the side effects encountered with higher oral dosages. ITB can be useful in treating spasticity of both cerebral and spinal cord origin and is an option for those who have failed oral medication trials or physical modalities intervention. After ITB placement, follow-up with physical and occupational therapy is paramount to maximize functional outcome effect. This treatment option also requires substantial maintenance, with outpatient visits every 2-3 months for medication refills.

### *Other Surgical Options*

More traditional surgical options include rhizotomy, and orthopedic procedures such as tenotomy, tendon lengthening or tendon training. Rhizotomy, which involves sectioning of selected dorsal segmental roots to modify different sensory input may still play a role in the treatment of young children with CP, but is rarely performed on adults now. Orthopedic surgery such as tendon lengthening or tendon transfer procedures may sometimes be recommended to increase function, correct deformities, improve range of motion to facilitate self-care, or for cosmetic reasons. These procedures should not be considered until at least 12 to 24 months following any traumatic injury, to ensure that no further spontaneous recovery will occur. These procedures are generally more successful if the underlying spasticity is treated first, for example with intrathecal Baclofen, to prevent recurrence of contractures.

The treatment of spasticity is an ongoing lifelong process and preventive options are available. The key is to start treatment early and maintain a regular appropriate exercise stretching or range of motion program. A combination therapy approach is usually the most beneficial with the goal of finding the right balance of spasticity that is most useful and functional for each individual's needs and clinical situation.

## REFERENCES

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