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TREATMENT OF SPASTICITY IN MS

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Spastic” comes from the Greek “spastikos” which means “drawing or pulling up”. The word dates back to the late 1700s, and it was first used in the 1800s to describe people with muscle problems or spasticity.

Spasticity is estimated to affect at least half of people with MS at some point in their illness. It is estimated that up to one-third of people with multiple sclerosis need to alter their daily activities because of spasticity.

What causes spasticity?

Spasticity is a common residual of MS relapse. The pathophysiology of spasticity relates to an imbalance occurring between excitatory and inhibitory signals coming from the brain and spinal cord. As neural pathways are disrupted, related to demyelination or axonal damage in MS, there is an interruption of the inhibitory signals which results in an exaggerated excitation.

The degree of spasticity is often variable among individuals and within the same individual at different times. When weakness is severe, spasticity can be helpful to the patient, as it can provide strength to the weak muscle, resulting in better ambulation or transferring. However, spasticity is often painful and typically impairs these abilities.

Accompanying signs on examination include clonus, particularly at the ankles, exaggerated deep tendon reflexes, extensor or flexor muscle spasms, and when most severe, scissoring (adductor spasms with involuntary crossing of the legs), and contractures.

Spasticity can:

- Result in alteration in mobility and inability to exercise or perform activities of daily living.
- Reduce endurance and increase energy expenditure
- Result in pain, spasms and interfere with sleep
- Decrease a patient’s ability to be independent

- Interfere with ability of caregivers to assist with hygiene and perform safe transfers
- Decrease a patient’s quality of life

Triggers for spasticity include:

- Quick or sudden movements
- Stress/anxiety
- Infections, particularly UTI
- Skin breakdown or decubiti
- A rise in internal temperature due to fever, exercise, excitement or anger, comorbid medical conditions, or the external environment or weather
- A full bladder or constipation

Treatments

When spasticity is acute, the first step is to identify the cause or trigger for the change in function, specifically looking for underlying, easily treatable conditions. If spasticity is more chronic, specific treatment may be warranted with the objective of improving function and comfort without compromising strength.

A comprehensive, multi-disciplinary treatment plan is most helpful. Participants often include neurologists, nurses, physiatrists, and occupational and physical therapists, and both pharmacologic and non-pharmacologic therapies have important roles. Physical therapy or tailored exercise programs aimed at muscle stretching, range of motion, and strength training can prevent shortening of muscles and can help reduce the severity of symptoms. In addition, positioning techniques or use of orthotics, including taping, splinting, and bracing can be enormously helpful. In some cases, the use of a wheelchair or stander can be of benefit. Rating scales such as the Modified Ashworth Scale, are available to help make assessments and measure the effectiveness of treatment.

Pharmacologic therapies: Oral medications are the first line of pharmacologic therapy. **Baclofen** is considered the gold standard for management of spasticity. It is a GABA-b receptor agonist that results in a decrease in excitatory neurotransmitter release. Initial starting dose is 5-10 mg orally one to three times a day, with a maximum recommended dose of 80mg/day. Dosing is tailored to the individual patient and situation. Abrupt withdrawal should be avoided as this may cause seizures or hallucinations.

Tizanidine also acts centrally and is an alpha-2 adrenergic agonist, preventing the release of excitatory amino acids by suppressing polysynaptic excitation of spinal cord interneurons. Initial starting dose is 2-4mg at bedtime or TID. Maximum dose is 36mg/day. Common side effects are drowsiness, hypotension, weakness and dry mouth. Tizanidine is metabolized mainly by the liver and serial monitoring of liver function is recommended.

Benzodiazepines act centrally to facilitate the release of inhibitory GABA, resulting in some degree of muscle relaxation. These agents are often used for treatment of anxiety, and they may be sedating. Because of their abuse potential and weak anti-spasticity actions, they are used less frequently than the other medications. Withdrawal can occur after only 4-6 weeks of regular use. **Clonidine** is an alpha-2 agonist, similar to tizanidine. It is typically used as an antihypertensive. Initial dose is 0.1mg BID; maximum dose is 1.2mg BID. Important adverse events include hypotension, CHF and conduction block, as well as drowsiness, dizziness and constipation. **Dantrolene** acts peripherally by inhibiting the release of calcium from the sarcoplasmic reticulum. This interferes directly with the muscle's ability to contract. It can result in muscular weakness, which limits benefit. Dantrolene is not first line therapy, especially in ambulatory patients. Initial dose is 25mg/day with slow titration to 100mg TID.

Non oral medications: **Botulinum Toxin (Botox)** is a neurotoxin that can be injected directly into the spastic muscle. It prevents acetylcholine release pre-synaptically at the neuromuscular junction. The benefit is temporary, typically lasting 12 weeks, when it must be repeated. Excessive dosing can result in generalized weakness, and it is common practice to start with a low dose. A small number of patients (3-10%) develop neutralizing antibodies to botulinum toxin and no longer benefit from treatment. **Phenol** nerve blocks (intramuscular) are administered via an injection and are occasionally useful in spasticity management, particularly in the case of neurolytic blockade of the obturator nerve for severe adductor spasticity. Disadvantages of phenol include its permanent effect and the possibility of numbness.

Intrathecal baclofen pump (ITBP)

Oral baclofen has poor lipid solubility with low CSF levels, and cerebral side effects (fatigue, sedation) often occur before the full therapeutic effect can be realized. Administering baclofen directly to the spinal fluid reduces spasticity 100-fold better than the oral preparation. A test dose is typically given first (particularly in ambulatory patients) via lumbar puncture. Responders are then considered for ITBP implantation. During implantation, a small catheter is inserted through a needle into the spinal fluid and then threaded caudally. The other end of the catheter is tunneled under the skin of the abdomen and connected to the reservoir and pump. The reservoir for the pump is a battery-powered disc measuring one inch thick and 3 inches in diameter.

Infusion rate is adjustable using a transponder held over the skin. The pump is refilled percutaneously in an outpatient setting. ITB pumps can be used during MRI testing and during pregnancy. Adverse events include infection (5%) and technical malfunctions (pump failure, catheter kinking or breakage, or dislodgement of the catheter, all quite uncommon).

Alternative therapies

There is little data on the use of **acupuncture** as treatment for spasticity in MS, but there are many anecdotal accounts of benefit. The same is true for **marijuana**. Many patients seem to derive benefit from yoga, pilates, and other "physical" therapies.

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