'Spasticity after spinal cord injury. Treatment alternatives.'

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Definition

'a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motoneuron syndrome'


- intrinsic tonic spasticity: exaggeration of the tonic component of the stretch reflex (manifesting as increased tone),
- intrinsic phasic spasticity: exaggeration of the phasic component of the stretch reflex (manifesting as tendon hyper-reflexia and clonus),
- extrinsic spasticity: exaggeration of extrinsic flexion or extension spinal reflexes (manifesting as spasms triggered by an afferent input)

*Decq P. Pathophysiology of spasticity. Neurochirurgie 2003;*
Maestro = Brain  
Musical Instruments = Muscles
Epidemiology

- 65–80% of SCI pt have symptoms of spasticity in acute or chronic phase.


- Cervical SCI: 93% of ASIA A / 78% of ASIA B-D
- Thoracic SCI: 72% of ASIA A / 73% of ASIA B-D

- Problematic spasticity was significantly correlated with cervical incomplete (ASIA B-D) injury

‘Useful spasticity’

- Prevention of osteopenia (increase muscle bulk and strength)
- Prevention of deep vein thrombosis (increase venous return)
- Increase stability in sitting and standing
- Facilitate the performance of transfers
- Facilitate gait performance
- Facilitate the performance of ADL
‘Problematic spasticity’

Spasticity has a negative impact on QOL through restricting activities of daily living (ADL), in more than 60% of spinal cord injured patients


- inhibiting effective walking,
- inhibiting self-care,
- causing pain and fatigue,
- disturbing sleep,
- increasing the risk of falls,
- contributing to the development of contractures, pressure ulcers and infections,
- complicating the role of the caretaker,
- impeding rehabilitation efforts.
Treatment algorithm
Physical techniques

- Positioning in bed and during sitting
  (maintenance of muscle length)
- Range of motion/stretching
  (prevents contractures)
- Weight-bearing (standing frame)
  (prolonged stretch of ankle plantar flexor muscles)
- Splinting/orthoses (enables long-term stretch)
- Hydrotherapy
- Cold/heat application

Albert T, Yelnik A. Physiotherapy for spasticity. Neurochirurgie 2003;
Bohannon RW. Tilt table standing for reducing spasticity after spinal cord injury.
Arch Phys Med Rehabil 1993;
Kesiktas N et al. The use of hydrotherapy for the management of spasticity.
Neurorehabil Neural Repair 2004;
Recent research


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- Ness LL, Field-Fote EC. Effect of whole-body vibration on quadriceps spasticity in individuals with spastic hypertonia due to spinal cord injury. Restor Neurol Neurosci 2009;

Pharmacologic management

- ‘GABAergic’ – (baclofen and diazepam),
  act at interneurons that use the neurotransmitter gamma-aminobutyric acid (GABA) in the CNS

- alpha-2-adrenergic – (tizanidine and clonidine),
  act at alpha-2 receptors in the CNS

- peripheral acting – (dantrolene)
  act at the neuromuscular level

Kita M, Goodkin DE. Drugs used to treat spasticity. Drugs 2000;
Diazepam

- GABA\textsubscript{A} agonist: increase presynaptic inhibition by opening chloride ion channels

- Most effective in the treatment of hyperactive reflexes and painful spasms in individuals with SCI

- Clonazepam, causes less sedation and has slightly lower risk for dependence.

- Functional measures have been shown not to improve.

Schlosser W. Action of diazepam on the spinal cord. Arch Int Pharmacodyn Ther. 1971;
Baclofen

- A structural analogue of GABA and an agonist of GABA\textsubscript{B} receptors
- Acts in part presynaptically by reducing neurotransmitter release through the reduction of Ca\textsuperscript{2+} influx and in part postsynaptically allowing the flow of potassium out of the Ia afferent terminal, resulting in membrane hyperpolarization and, hence, interruption of action potential transmission.

- Baclofen may have no positive effect on walking ability or the performance of ADL.

Clonidine

- Commonly used to treat hypertension.
- Binds to alpha-2 receptors, preventing the normal action of norepinephrine to act as a neurotransmitter.
- It reduces spasticity by enhancing alpha-2-mediated presynaptic inhibition of sensory afferents, thereby suppressing spinal polysynaptic reflexes.

- **Clonidine has been found to be associated with improved walking ability in individuals with incomplete SCI**

  (longer cycles, increased treadmill speed, upright posture)

*Stewart JE, Barbeau H, Gauthier S. Modulation of locomotor patterns and spasticity with clonidine in spinal cord injured patients.*

*Can J Neurol Sci 1991;*
Tizanidine

- A centrally acting (spinally and supraspinally) alpha-2-adrenergic agonist.
- It acts by inhibiting the release of excitatory amino acids from the presynaptic terminals of excitatory spinal neurons.
- It may also facilitate the inhibitory neurotransmitter glycine.
- It has been shown to reduce muscle tone and frequency of muscle spasms in individuals with SCI
- **No increase in functional measures has been noted.**

Dantrolene

- Dantrolene sodium acts peripherally at the muscle tissue, rather than at the spinal cord level.
- It inhibits muscle action potential-induced release of calcium.
- It appears to have a greater effect on phasic than on tonic stretch reflexes and on fast twitch rather than slow twitch muscle fibers.

- **Individuals with SCI are rarely treated with dantrolene, likely because its peripheral site of action results in muscle weakness.**

Cyproheptadine

- An histamine and a serotonin antagonist

- It is proposed to reduce spasticity via inhibition of motor neurons by neutralizing the spinal and supraspinal serotoninergic excitatory inputs.

- It has been associated with an improvement in the walking pattern of individuals with SCI

  (reduced need for manual assistance, increased treadmill speed, and reduced ankle clonus)

Tetrahydrocannabinol

- The main active ingredient in cannabis.

- Some literature supports the hypothesis that the relaxing effect of marijuana on muscles in patients with SCI-related spasticity is due to inhibition of polysynaptic reflexes, rather than simply due to a general relaxation response.

- THC is an effective and safe drug in the treatment of spasticity. At least 15-20 mg per day were needed to achieve a therapeutic effect.

Intrathecal baclofen

- Combines the pharmacologic administration with a surgical technique.
- Bypassing the blood–brain barrier allows as much as four times the concentration of baclofen to be delivered to the spinal cord with only 1% of the oral dose.
- The long-term effects of treatment with intrathecal baclofen are not yet known.

- Recent reviews have described the effects of intrathecal baclofen on reducing spasm frequency, reflex intensity and/or spasticity-related discomfort, as well as improving QOL.

Injection techniques

- Treatment of focal spasticity
  (local chemodenervation)

- The chemodenervation agents used include phenol, ethanol, and, more recently, botulinum toxin

- Administration of phenol or ethanol to the nerve trunk causes short-term effects similar to a local anesthetic: blocking of sodium channels reduces nerve depolarization.
  (not commonly used in SCI individuals)

Botulinum toxin

- First choice pharmacological treatment for focal spasticity.

- Acts on the neuromuscular junction to inhibit the release of acetylcholine. The toxin causes chemical denervation of intrafusal and extrafusal muscle fibers, and its effect is reversible.

- Always in combination with other treatments.

- The literature indicates that botulinum toxin injections potentially can be useful in the treatment of spasticity after SCI (improvements in pain, nursing care, hygiene, comfort and functional activities)

Surgical techniques

- **Tenotomy**
  (release of a tendon from a severely spastic muscle, in individuals without voluntary movement)

- **Tendon lengthening**
  (to reduce the pull on spastic muscles, positioning the joints at a more natural and useful angle)

- **Tendon transfer**
  (moving the tendon attachment to the bone closer to the muscle, in muscles that have at least partial voluntary function)

- **Selective rhizotomy**
  (cutting of posterior roots to interrupt the peripheral reflex arc)

- **Intrathecal baclofen administration**
  (it is considered to be the most successful of the surgical treatments)

*Chambers HG. The surgical treatment of spasticity. Muscle Nerve Suppl 1997;*
Electrical stimulation

- **Epidural**
  - Epidurally placed electrodes on the dorsal columns.
  - The clinical effect probably results from activation of inhibitory networks within the dorsal column–brainstem–spinal loop.

- **Intraspinal microstimulation**
  - Fine microwires implanted in the spinal cord.
  - Aim to suppress the intrinsic excitability of spinal motoneurons through the application of extracellular current pulses.

Assessment

- There are generally poor correlations among clinical scales and reductions in spasticity are not necessarily correlated with improvement in function.

- There is some question about the validity of **Ashworth and modified Ashworth scales** in the lower limbs of persons with SCI.

  Haas BM, Bergstrom E, Jamous A, Bennie A. The interrater reliability of the original and of the modified Ashworth scale for the assessment of spasticity in patients with spinal cord injury. Spinal Cord 1996;

- The ideal scale should not only quantify the degree and nature of the spasticity, but also patient satisfaction, global function, and technological assessment.

Conclusions

✓ Decisions regarding the management of spasticity must be based on the goal of achieving balance between the useful and detrimental effects on patient’s quality of life.

✓ Given the multifactorial origin of spasticity, a successful strategy would most likely require a combination of interventions to achieve the best clinical outcome.
Thank you

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