Spasticity Management

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DISCLOSURE

- I have no conflicts of interest

Objectives

- Understand the clinical concept of spasticity
- Become familiar with spasticity assessment
- Find out options for spasticity management

GENERAL PRINCIPLES

- Spasticity has been defined as a velocity-dependent increase in tonic stretch reflexes or muscle tone with exaggerated tendon jerks resulting from increased excitability of the stretch reflex
- Disordered sensory-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles

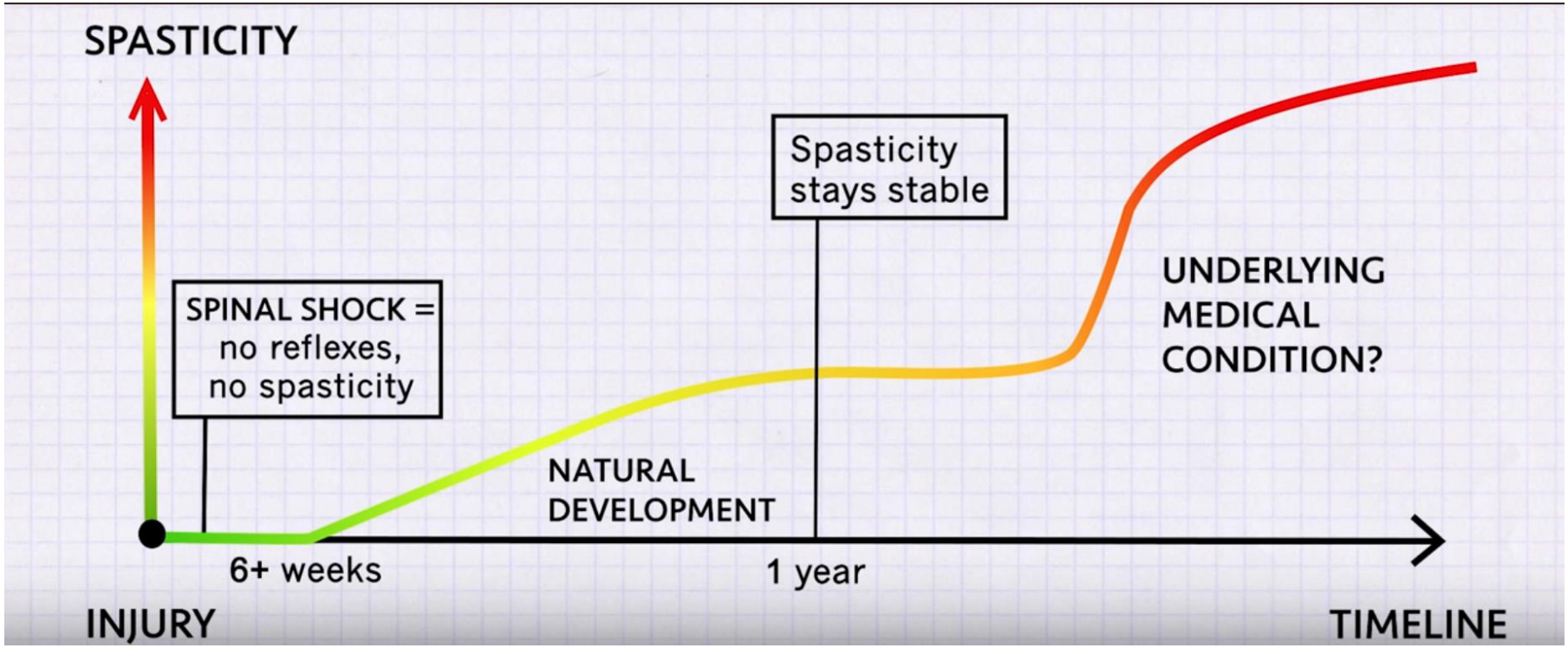
DEFINITION OF TERMS

- The term ***spasticity*** encompasses multiple features of the UMN syndrome including clonus, spasms, spastic co-contraction, and weakness, which can present individually or in combination. These phenomena are defined as follows:
  - **Clonus:** rhythmic pattern of muscle contraction provoked by a sudden stretch, which activates muscle spindles reflexes.
  - **Spasm:** sudden, involuntary muscle contraction, often causing pain and gross body movements.
  - **Spastic co-contraction:** the inappropriate co-activation of antagonistic muscle groups during voluntary activity resulting from loss of reciprocal inhibition (Yelnik et al 2010).
  - **Weakness:** results in decreased muscle force and early fatigue with voluntary muscle activation.

ETIOLOGY/PATHOPHYSIOLOGY

- Loss of descending inhibitory modulating signals as a result of SCI
- Loss of descending inhibition results in hyperactivity of segmental reflexes
- Denervation hypersensitivity that occurs at the receptor level over time, resulting in decreased threshold for MU activation and heightened response to stimulus

EVOLUTION OF SPASTICITY



https://www.youtube.com/user/SCIREWebVideo

SPASTICITY CAN BE …

DISABLING

- Physical Impairment
  - pain, fatigue, poor sleep, contractures, impaired sexual function
- Limits Activity and Participation Disabling
  - activities of daily living (ADLs), positioning, transfers, and mobility.
  - negative self-image, vocational disability, decreased quality of life.

BENEFICIAL

- Facilitate function
- Improves circulation
- Protects against muscle atrophy
- Early warning device

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How to measure spasticity

ASHWORTH SCALE (AS)

0 No increased tone

1 Slight increase in tone, giving a "catch" when affected part is moved in flexion or extension

2 More marked increase in tone, but affected part easily flexed

3 Considerable increase in tone; passive movement difficult

4 Affected part rigid in flexion or extension

MODIFIED ASHWORTH SCALE (MAS)

0 No increased tone

1 Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the ROM when the affected part(s) is moved in flexion or extension

1 + Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM

2 More marked increase in tone, but affected part easily flexed

3 Considerable increase in tone; passive movement difficult

4 Affected part rigid in flexion or extension

TARDIEU SCALE

- Offers advantages over the AS and MAS in that it evaluates both the slow and fast components of spasticity, thereby taking into consideration soft tissues changes that may have occurred in the spastic muscle.
- The TS incorporates three components into the testing:
  - velocity of the stretch
  - quality of muscle reaction
  - tangle of muscle reaction.
- The TS incorporates the velocity-dependent component of spasticity into the assessment, rather than simply measuring resistance to passive movement

OTHER ASSESSMENT SCALES

The Penn Spasm Frequency Score (PSFS)

0 No spasms

1 Mild spasms induced by stimulation

2 Infrequent spasms occurring less than once per hour

3 Spasms occurring more than once per hour

4 Spasms occurring more than 10 times per hour

OTHER ASSESSMENT SCALES

The Spinal Cord Assessment Tool for Spastic Reflexes (SCATS)

SCATS: Clonus

Clonus quantified in response to rapid dorsiflexion of the ankle

0 No reaction
1 Mild, clonus maintained <3 seconds
2 Moderate, clonus persists 3–10 seconds
3 Severe, clonus persists >10 seconds

SCATS: Flexor spasms

Measurement of excursion of big toe into extension, ankle dorsiflexion, knee flexion, or hip flexion when pinprick stimulus applied to plantar surface of the foot

0 No reaction to stimulus
1 Mild, <10°
2 Moderate, 10°–30°
3 Severe, ≥30°

SCATS: Extensor Spasms

Starting position with hip and knee placed at 90 °–110° of flexion with contralateral limb extended. Hip and knee joints then simultaneously extended and duration of quadriceps muscle contraction is measured

0 No reaction
1 Mild, contraction maintained <3 seconds
2 Moderate, contraction persists 3–10 seconds
3 Severe, contraction persists >10 seconds

BIOMECHANICAL AND ELECTROPHYSIOLOGIC MEASURES

The Pendulum Test

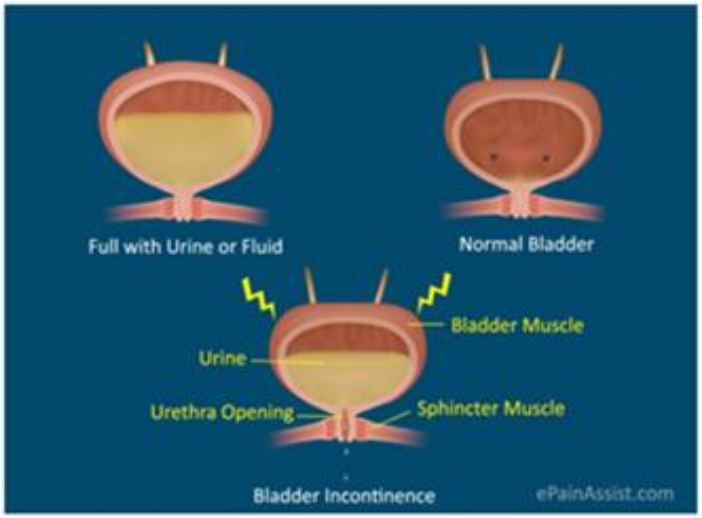
- Patient seated or supine with the exam table ending at the distal thigh. The leg is passively extended and then released, allowing it to fall into flexion.
- The rate of movement of the leg can be measured by tachometers, providing insight into the degree of spasticity in the knee extensors.

EMG

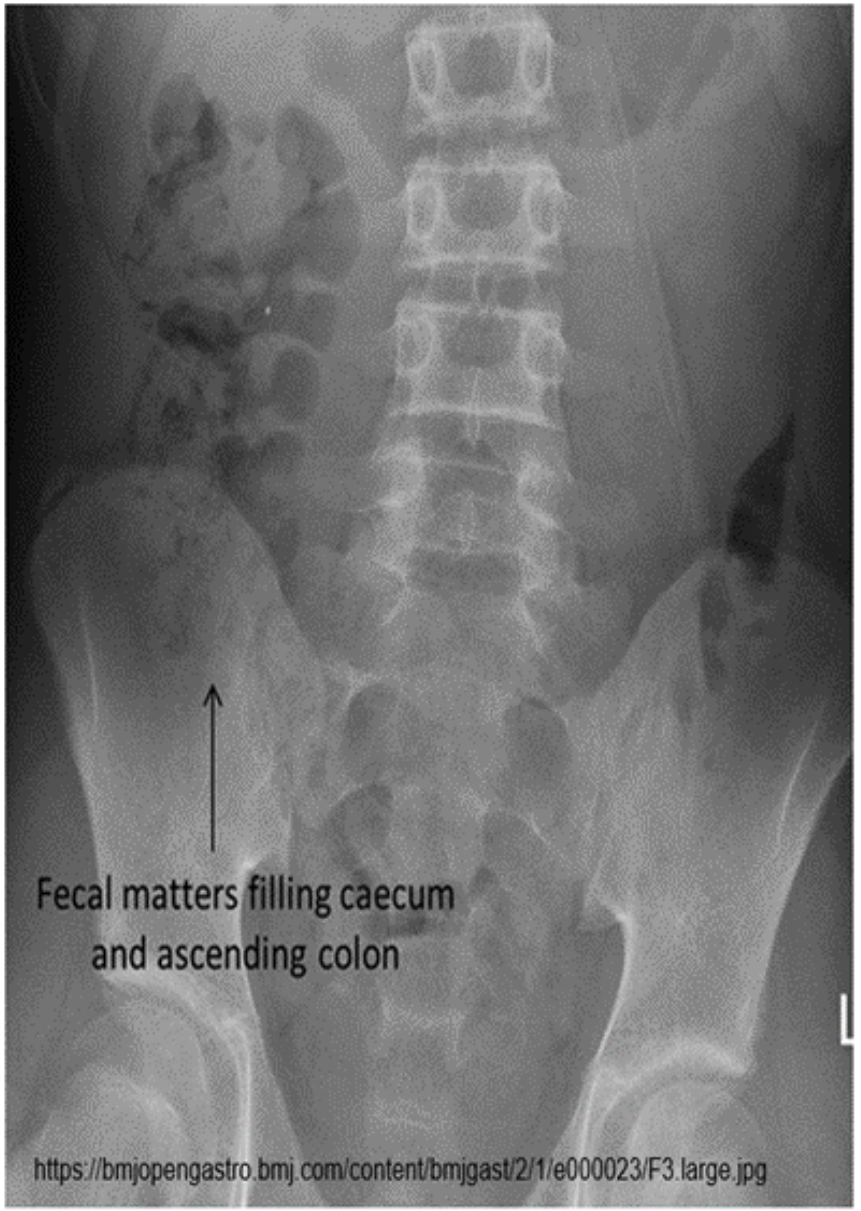
- Surface electromyography (EMG) represents a commonly used modality to evaluate the electrical activity of affected muscles.
- EMG additionally can measure electrical responses due to reflex activity. H-reflex amplitude, and F-wave amplitude may all be increased in spastic muscle, therefore monitoring of these measures may provide useful insight into the efficacy of treatment regimens

COMMON TRIGGERS OF SPASTICITY

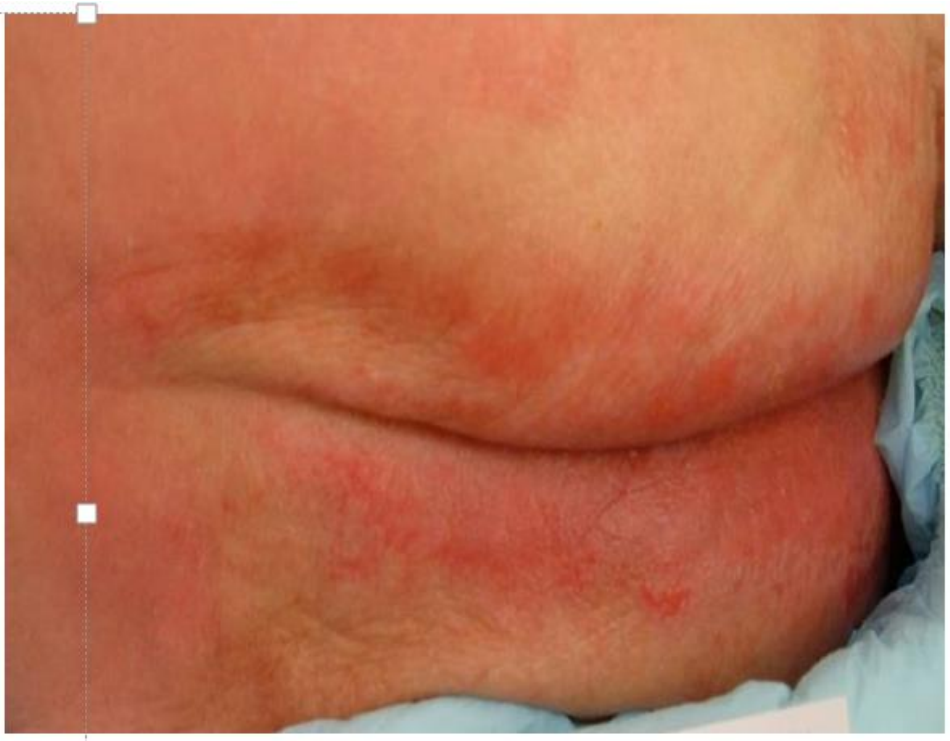
- Neurogenic Bladder:
  - UTI
  - Catheter
  - Stones



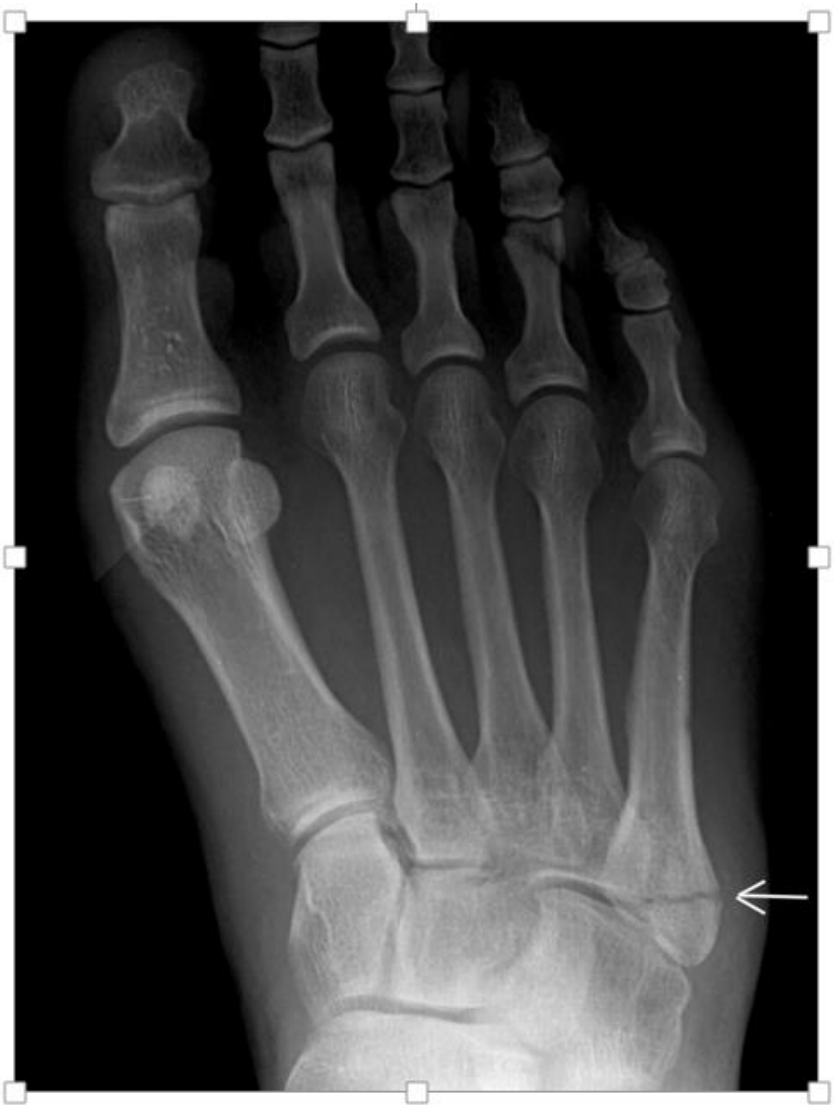
- Neurogenic Bowel
  - Constipation
  - Hemorrhoids
  - Fecal impaction

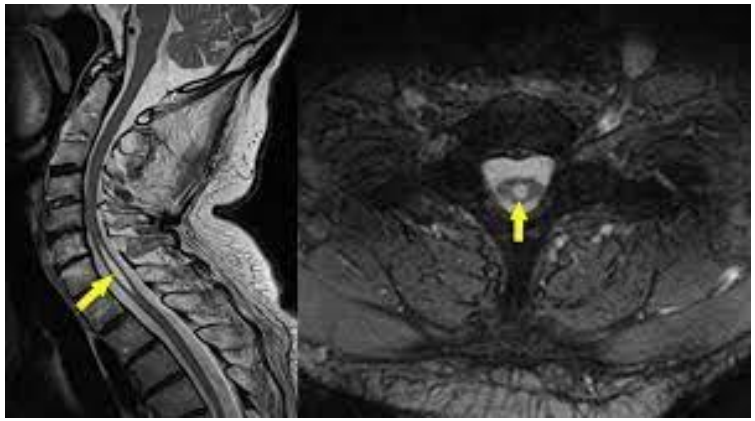


- Skin
  - Pressure injury
  - Ingrown toenails



- Other
  - Fracture
  - Tight clothing
  - Poor positioning
  - Seating
  - Menstrual periods





- Pregnancy
- Mental Stress
- Temperature
- Physiologic or neurologic change
  - Syringomyelia
- Systemic Illness
  - DVT, systemic infection

PHARMACOLOGICAL TREATMENTS

- As spasticity is thought to be due to a loss of baseline inhibition, common medication options predominately potentiate GABA or function as GABA receptor agonists, alpha-2 presynaptic agonists, or peripherally (69).
- Based on unique side effect profiles, each medication should be trialed individually and only the minimal amount necessary used.

- The most commonly used medications include:

  - Baclofen (Lioresal®)
  - Tizanidine (Zanaflex®)
  - Clonidine (Catapres®)
  - Benzodiazepines
  - Dantrolene (Dantrium®)

PHARMACOLOGICAL TREATMENTS

Baclofen (Lioresal®)

- Analog of gamma amino butyric acid (GABA) which is an inhibitory neurotransmitter that binds presynaptically at the GABA-B receptors.
- GABA-B receptor agonist, particularly effective for flexor spasms (68,71–73).
- Excreted in the urine (74)

Dosage:

- Daily doses of 5 mg three times a day and 80 mg/day, respectively (74).
- Some practitioners will titrate baclofen to up to 240 mg a day,
- Maximum dose for a patient essentially limited by side effects including sedation, fatigue, nausea, constipation, weakness, and confusion (75).

Side effects:

- As baclofen can lower the seizure threshold, caution must be used in patients who have a history of seizures (74).
- It must be titrated down prior to cessation to avoid withdrawal symptoms including seizures, worsening spasticity, fever, mental status changes, and organ failure (74).

PHARMACOLOGICAL TREATMENTS

Tizanidine (Zanaflex®)

- An alpha-2 presynaptic agonist that increases presynaptic inhibition on interneurons in the dorsal horn of spinal cord and are believed to depress polysynaptic reflexes by decreasing the release of excitatory amino acids such as glutamate and aspartate and facilitating the actions of glycine an inhibitory amino acid neurotransmitter.

Dosage:

- Typically started at 2 to 4 mg at bedtime time (as it can cause sedation) then increased (as tolerated) to 8 mg 3 to 4 times/day
- Maximum FDA approved dosage: 36 mg/day (82).
- Available as a tablet and capsule.
    - On an empty stomach, the time to peak concentration is equal for both the tablet and capsule; and are *however*, after a meal, the tablet is absorbed 80 times faster than the capsule (86).

Side effects:

- Sedation, dry mouth, dizziness, hypotension, bradycardia, constipation, and urinary frequency (82).
- Can cause elevated liver enzymes in 5% of cases (82), as it is metabolized in the liver necessitating monitoring of liver function (82). Hallucinations have been reported in small percentage in the initial weeks of starting tretament.

Contraindications:

in patients taking ciprofloxacin (Cipro®) and fluvoxamine (Luvox®) because the combination of tizanidine with CYP1A1 inhibitors, such as fluoroquinolones, fluvoxamine, and oral contraceptives can result in increased serum concentration of tizanidine, causing severe hypotension (82).

PHARMACOLOGICAL TREATMENTS

Clonidine (Catapres®)

- Centrally acting alpha-2 presynaptic agonist, is an antihypertensive medication with both oral and transdermal formulations and has been shown to be effective in treating spasticity in patients with SCI (68,73,78,87– 91).

Dosage:

- Typical dosage of the oral formulation starts at 0.05 mg twice daily and can be increased to 0.1 mg/ day after 3 days (88),
- Titrating up to 0.4 mg/day with dose increases of 0.1 mg/week (88).
- Available in 0.1 to 0.3 mg doses (an advantage of the clonidine transdermal formulation has fewer side effects than the oral (Catapres- TTS).
- Delivers a steady dose over the course of 7 days (91).

Side effects include:

- Drowsiness, dry mouth, dizziness, sedation, fatigue, headache, hypotension, and constipation (79).
- Metabolized by the liver and partially excreted in the urine (79).
- Should be weaned gradually as rebound hypertension can occur.

PHARMACOLOGICAL TREATMENTS

Gabapentin (Neurontin®)

- An anticonvulsant structurally similar to GABA, is active at the neocortex and hippocampus and is excreted in the urine (73).
- Not FDA approved for the treatment of spasticity in patients with SCI, but studies have shown improvement of spasticity via surface EMG, and AS when compared with placebo (97–100).

Dosage:

- Initiated at 300 mg three times a day, which can be titrated up to 3,600 mg/day (96,97).

Side effects include:

- Somnolence, dizziness, ataxia, tremor, dyspepsia, and constipation (101).
- Must be tapered gradually to cessation to decrease the risk of withdrawal symptoms including acute seizures, anxiety, insomnia, nausea, pain, and sweating (96).

PHARMACOLOGICAL TREATMENTS

Pregabalin (Lyrica®)

- An anticonvulsant, is thought to increase GABA levels in the brain and is renally cleared (102,103).
- FDA approved for treatment of neuropathic pain (102).
- Not FDA approved for the treatment of spasticity, retrospective case studies have shown improvement of spasticity in patients with multiple sclerosis (104).

Dosage:

- Typically initiated at doses of 75 mg twice daily, with titration as necessary to a maximal dose of 600 mg/day.

Side effects include:

- Peripheral edema, angioedema, ataxia, visual impairment, weight gain, and confusion (102).
- To discontinue pregabalin, it must be tapered off to reduce the risk for withdrawal seizures (102).

PHARMACOLOGICAL TREATMENTS

Dantrolene (Dantrium®)

- Acts directly on the muscle by preventing the sarcoplasmic reticulum from releasing calcium, thus inhibiting muscle contraction (68,78,87).
- The drug is non-selective and may act on spastic and non-spastic tissues to cause weakness which may be a concern for those with marginal strength (78).

Dosage:

- Initiated at 25 mg a day and then titrated up slowly to 400 mg/day divided into dosing three or four times a day (92).

Side effects include:

- Weakness, headache, dizziness, somnolence, fatigue, and visual disturbances (92).
- Metabolized by the liver and can cause elevation of liver enzymes, it is essential to monitor liver function (92).
- It may cause hepatotoxicity and rarely, fulminant hepatic failure so baseline and periodic LFT testing is indicated.

PHARMACOLOGICAL TREATMENTS

Benzodiazepines

- Includes: Diazepam (Valium®) and Clonazepam (Klonopin®)
- Binds at a site near the GABA-A receptors on presynaptic neuron and enhances GABA-mediated chloride conductance into nerve terminals and thereby increases inhibitory activity of the neurons. It also facilitates post-synaptic effects of GABA.
- commonly used to treat significant spasms rather than tightness aspects of spasticity (78).
- Long-acting
- Metabolized by the liver (68,79)

Dosage:

- Diazepam (most commonly used) (80): typically dosed 2 to 10 mg two to three times a day (79).
- Clonazepam: typically initiated at a dose of 0.25 mg to 1 mg at bedtime with gradual titration to a total dose of 3 mg (81).

Side effects:

- weakness, sedation, drowsiness, hypotension, ataxia, and fatigue (79).
- The dose must be titrated down prior to benzodiazepine cessation to avoid potential withdrawal symptoms including seizures, hyperthermia, and mental status changes (79).

PHARMACOLOGICAL TREATMENTS

Cyproheptadine (Periactin®)

- A serotonin and histamine antagonist that is hepatically metabolized.
- not FDA approved for the treatment of spasticity (105).
- Has been shown to decrease spasms and clonus in patients with multiple sclerosis and SCI (106). Studies have shown increase walking speed in patients with spasticity affecting their gait (107,108).

Dosage:

- Initiated at 4 mg at bedtime with the maximum recommended dose of 36 mg per day in divided doses (68).
- Can be increased by 4 mg every 3 to 4 days.

Side effects include:

- Sedation, dry mouth, appetite stimulation, and weight gain (as it is an appetite stimulant) (105).
- The use of cyproheptadine with a selective serotonin reuptake inhibitor (SSRI) can reverse the serotonergic effect of the SSRI (105).

PHARMACOLOGICAL TREATMENTS

Cannabinoids (Marinol®, Cesamet®)

- Not FDA approved for the treatment of spasticity (109), but marijuana (cannabis), which is hepatically cleared, can prevent or relieve spasticity in patients with SCI and spasticity (110).
- Usage of D9-tetrahydrocannabinol (THC), the active component of marijuana, has shown spasticity improvement, measured by decreased EMG activity and decreased tone measured by the MAS compared with placebo (111).

Dosage:

- Cannabinoids in patients with SCI have been found to be safe up to 15 to 20 mg/day (111).

Side effects include:

- tachycardia, orthostatic hypotension, hot flashes, sweating, rhinorrhea, loose stools, hiccups, and anorexia (109).

PHARMACOLOGICAL TREATMENTS

4-Aminopyridine (Fampridine®)

- Potassium channel blocker typically used to improve ambulation in individuals with multiple sclerosis (105).
- Not FDA approved as treatment for spasticity, but one study has shown reduction of spasticity reflected by Ashworth score (112).

Side effects include:

- Headaches, paresthesias, insomnia, and nausea with doses less than 80 mg/day (105,113)
- Seizures in the case of an overdose (105).

CHEMODENERVATION

- Chemodenervation with botulinum toxin injections is an option for individuals with focal or multifocal spasticity.
- Introduced for the treatment of strabismus in the 1980s (115).
- Its use has extended to treatment of multiple disorders including:
  - Cervical dystonia
  - Overactive bladder
  - Migraine
  - Blepharospasm
  - Cosmetic indications
  - Treatment of upper and lower limb spasticity.

ONABOTULINUMTOXINA (BOTOX)

OnabotulinumtoxinA (Botox)

- FDA approved for the treatment of upper and lower limb spasticity in adult patients.
- OnabotulinumtoxinA is available in 100- and 200-unit vials and must be stored refrigerated (2°C to 8°C).

Dosage and Instructions:

- Recommended maximum dosing is 400 units per treatment session; however, there are reports of dosing as high as 600 to 1,200 units per session (120).
- The toxin is reconstituted with preservative-free normal saline.
- Following injection of the saline into the vial, the vial is swirled gently for reconstitution.
- The onabotulinumtoxinA must be used within 24 hours of reconstitution (64).

ABOBOTULINUMTOXINA (DYSPORT)

AbobotulinumtoxinA (Dysport)

- Currently FDA approved for the treatment of adult upper and lower limb spasticity as well as pediatric lower limb spasticity.

Dosage and Instructions:

- Recommended dose range is **500 to 1,000 units.**
- guidelines are based upon a recent clinical trial that evaluated the safety and efficacy of abobotulinumtoxinA use in upper limb spasticity secondary to stroke or brain injury (119).
- however, many in clinical practice dose up to 1,500 units per session (120).
- AbobotulinumtoxinA is available in 300- and 500-unit vials and must be stored in refrigeration between 2°C and 8°C.
- Following injection of normal saline into the vial, the vial is gently swirled. The reconstituted abobotulinumtoxinA should be used with 4 hours (63).

INCOBOTULINUMTOXINA (XEOMIN)

IncobotulinumtoxinA (Xeomin)

- Currently FDA approved for the treatment of upper limb spasticity in adults.
- The toxins are FDA approved for specific upper extremity muscles.

Dosage and Instructions:

- Based upon a recent clinical trial evaluating the safety and efficacy of incobotulinum toxin injections in individuals with spasticity secondary to stroke, some authors have expressed concern about exceeding 400 units in the upper extremity (121), while others have reported using doses of 800 units for the treatment of upper limb spasticity (122).
- It should be noted that each of these studies was completed in populations with spasticity of cerebral origin.
- IncobotulinumtoxinA is available in **50-, 100-, and 200-unit vials** and can be stored at room temperature (20°C t 25°C) for up to 36 months.
- It is also stable refrigerated (2°C to 8°C) or frozen (–20°C to –10°C).
- The product should be reconstituted using preservative-free normal saline injected into the vial and then swirled
- The reconstitution for incobotulinumtoxinA differs from the other toxins slightly, as this product requires inversio two to four times after swirling to reconstitute any product remaining on the rubber stopper.
- The incobotulinumtoxinA must be used within 24 hours of reconstitution (65).

RIMABOTULINUMTOXINB (MYOBLOC)

RimabotulinumtoxinB (Myobloc)

- Injectable solution that does not require reconstitution.
- Not FDA approved for use in spasticity, but it has been used off-label for the treatment of spasticity.

Dosage and instructions:

- Available in **2,500-, 5,000-, and 10,000-unit vials**.
- Maximum recommended dose per treatment session is 10,000 units (66).

NERVE BLOCKS

FOCAL SPASTICITY THAT IS LIMITING FUNCTION OR CAUSING PAIN

Diagnostic nerve blocks performed with anesthetic agents, such as **lidocaine or marcaine**, temporarily block the axonal sodium channels (134).

- ES is used to locate the nerve by producing contraction of the muscles innervated by the target nerve.
- The stimulus is gradually decreased with the goal of producing maximal muscular contraction with delivery of current at or below 1.0 mA.
- Neurolysis performed with phenol or alcohol denatures nerve protein and destroys the peripheral nerve; however, spasticity eventually returns with peripheral nerve remyelination and axonal regrowth (135–138).

Effects:

- Can be seen immediately following the injection
- Last from 1 month to 3 years with an average duration of 3 to 9 months following phenol injection (87,135,136).
- Both phenol and alcohol can affect motor, sensory, and mixed nerves.
- Common nerves targeted include:
  - musculocutaneous, obturator, and tibial nerves.

Adverse events include:

- Injection site pain, phlebitis, permanent nerve damage, dysesthesia, tissue necrosis, pain, and muscle weakness (68,133,134).
- Dysesthesias can develop following neurolysis, motor nerves or the motor branch of a mixed nerve is selectively targeted (135,139,140).
  - The treatment for dysesthesia includes repeat block at the same site or administration of oral anticonvulsants (139).

Motor Point Blocks

MOTOR POINT BLOCK INJECTIONS

The technique is similar to that described earlier for peripheral nerve blocks, with the goal being to elicit a muscular contraction with the delivery of current at or below 1.0 mA.

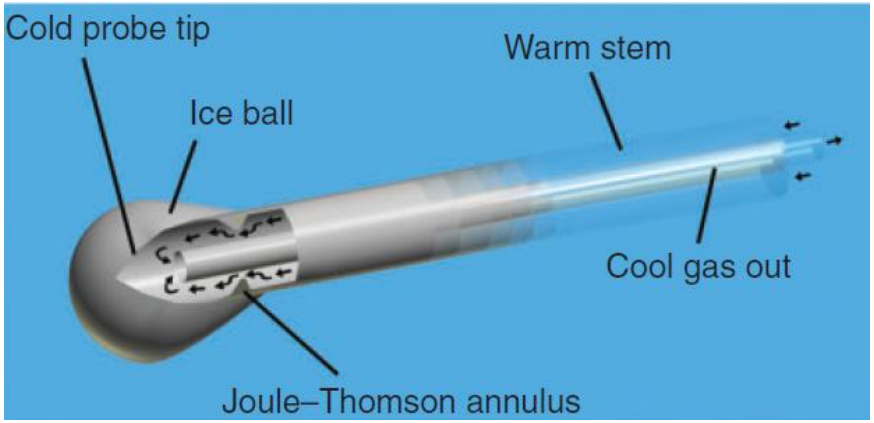
- The motor point has the greatest concentration of motor end plates.
- Phenol or alcohol are used to improve functional activities for the upper extremity (78).
- Repeat nerve block has been shown to be effective (141).
- Surgical neurolysis maybe be considered in severe cases that persist despite conservative treatment (142).

Adverse events include:

- Phlebitis, permanent nerve palsy, muscle necrosis, and systemic effects (68).
- Intramuscular injection can be painful and may require sedation or general anesthesia (78).
    - The burning sensation following motor point block can persist up to 24 hours after injection, while the effect of the motor point block can last 3 to 8 months (78).
    - Treatment includes desensitization techniques, compressive garments, antidepressants, or anticonvulsants (140).

Cryoneurolysis

- Percutaneous minimally invasive therapy
- Application of cold: -60°C to -88°C to nerves of spastic muscles
  - Joule-Thomson Effect
- Ice ball generated, measuring 3.5-18 mm at cryoprobe tip
- Rapid generation of ice ball → axonotmesis of peripheral nerve



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Cryoneurolysis

Side Effects

- Infection
- Bleeding
- Sensory dysesthesias
- Unwanted numbness
- CRPS II or neuritis

Contraindications

- Cryoglobulinemia
- Paroxysmal cold hemoglobinuria
- Cold urticaria,
- Raynaud’s disease
- Open infected wounds at/near treatment site

Intrathecal Pharmacologic Treatments

ITB

For patients with severe generalized spasticity unable to be sufficiently managed with oral medications or for whom medication side effects are intolerable.

Baclofen

- Commonly used intrathecally in patients with significant spasticity due to SCI (143).
- The GABA-B receptors in the spinal cord can be directly accessed by the ITB and, as such, significantly lower amounts of ITB can be used compared to the oral formulation, decreasing cognitive and sedative side effects (144).
- ITB has been shown to improve function and quality of life and decrease spasticity and pain (146–151). Given that ITB concentration is higher caudally compared with cranially (152), lower limb spasticity is generally better controlled compared with upper limb spasticity (143,146,147).
- Goals of ITB include a decrease in spasticity, improvement in function, and decrease in pain.

Dosage:

- The therapeutic dose of ITB is 1% of the oral dose (145).

Concerns:

- Individuals with a history of poor social support and medical compliance may be determined to be poor candidates for this approach.
- In the case of device malfunction, failure, or a late/missed pump refill, withdrawal symptoms including seizures, dizziness, nausea, hypotension, headache, and urinary retention can develop if a medication bridge with oral baclofen is not provided (74).

Intrathecal Pharmacologic Treatments

Dosage:

- The initial dose is usually considered to be twice the trial dose, but if the effects of the trial last more than 8 hours, then the initial dose is the same as the trial dose (24,74,148) and then titrated to achieve goals.
- After the first 24 hours, the rate can be increased by 10% to 30% as frequently as every 24 hours until optimal control of spasticity is obtained (74).
- The average maintenance dose is 400 to 600 mcg/day (range 50–1,000 mcg/day; 144).

Side effects:

- dizziness, nausea, hypotension, headache, weakness, urinary retention, seizure, erectile and ejaculatory dysfunction (144,156).

Baclofen overdose:

  - As ITB overdose may be caused by dosing error (157), or mechanical pump issues
- Symptoms of an overdose:
  - respiratory depression, lethargy, dizziness, nausea, hypotension, and weakness.
  - In the case of an overdose, the pump should be decreased to the minimum rate or emptied, and preparations should be made for a potential intubation due to respiratory depression

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Surgical Management

FOR SEVERE SPASTICITY

SURGICAL OPTIONS:

A surgical referral may be necessary to decrease spasticity and spasms while minimizing detrimental effects on motor, sensory, bowel, and bladder function.

- Patients with focal spasticity may benefit from an orthopedic tendon procedure to decrease tension of spasticity muscles, improve function, or correct a deformity (133,161).
- Orthopedic procedures include:
  - Tendon lengthening (to decrease pull on spastic muscles)
  - Tenotomy (release of a tendon from spastic muscle)
  - Tendon transfer (movement of tendon attachment to a different bone location; 133). One commonly performed tendon transfer is the split tibialis anterior tendon transfer (SPLATT), which is utilized to correct equinovarus deformity of the foot due to a spastic tibialis anterior muscle.

NEUROSURGICAL OPTIONS:

- Neurotomy and selective rhizotomy (78).
  - Instead of the temporary effects of neurolysis of peripheral nerves, neurotomy involves exposing and transecting the selected peripheral nerve to decrease spasticity and spasms (162).
  - **Obturator neurotomy** to decrease adductor spasticity, **tibial neurotomy** to decrease foot spasticity and **sciatic nerve neurotomy** to decrease knee flexor spasticity (162).
- Selective dorsal rhizotomy
  - Involves selective cutting of the dorsal rootlets to disrupt the reflex arc, is rarely used in patients with SCI compared with patients with cerebral palsy (78,161).

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