

Case-Based Learning Module: SPASTICITY

INTRODUCTION

Spasticity is a secondary complication affecting 65 – 78% of individuals with spinal cord injury (SCI),¹⁻³ and has been reported as a clinical impairment in 40 – 60% of these individuals.^{1,3} Spasticity may be caused by different physiologic factors, and should be managed to balance the problematic and beneficial effects that it may evoke. This module will focus on the pathophysiology, causes and treatment of spasticity in an individual affected by SCI in a primary care setting.

This module will enable clinicians to:

- Understand the pathophysiology of spasticity
- Identify potential causes of spasticity
- Effectively treat spasticity in the primary care setting
- Understand when to refer for assessment/management

CASE

Cindy, age 66

Cindy is a 66--year old woman who has been in your family medicine practice for the last 10 years. Two years ago, she had an MVA with fracture of C6, she underwent surgery and has an incomplete SCI. Since the injury, Cindy has experienced left sided hemiplegia, and difficulties with mobility. Cindy walks into your office with an obvious spastic gait, the aid of a quad cane, and is looking for your help with this “stiffness” she is experiencing on her left side, especially her leg.

What else would be important to know?

- *What is her understanding of “spasticity”?*
- *What are other words (besides stiffness) does she use to describe her spasticity?*
- *What effect does spasticity have on her daily activities such as walking, transfer, bathing, pain, and sleep?*
- *Has her spasticity changed since she was released from hospital?*
- *Is there an underlying infection/illness/stress such as a UTI or noxious stimulus such as constipation, fracture, or pressure sore?*
- *Does she find the spasticity is of any benefit to her ADL?*
- *If a noxious stimulus is present (constipation; fracture; pressure sore)?*
- *Is there a recent medication change and what does she take currently?*
- *What makes her spasticity worse? i.e.-when she moves, sitting, transferring*
- *Is there any time of the day that her spasticity worsens or gets better?*
- *What is she doing currently to treat her spasticity (stretching, icing, bracing etc)?*

You find her spasticity has not changed, there is no change in her function, but she is finding it interfering with sleep and gait, and has always wanted to avoid medications; however, Cindy feels she now might need medication.

How would you begin treatment of Cindy's spasticity?

- *Ensure there is no underlying condition/noxious stimuli that needed to be treated first*
- *Physiotherapy referral for passive stretching exercise*
- *Begin baclofen 5 mg PO tid*

INFORMATION SECTION

Introduction:

Spasticity is defined as, “a motor disorder characterized by velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflex, as one component of the upper motoneuron syndrome (UMN).”⁴ Spasticity can impact independence, secondary health conditions, emotional adaptation, and community integration, but can also be beneficial (transfers & mobility).⁵ The level of a patient’s injury may predict their likelihood of developing spasticity,¹ and may be treated by pharmacological, non-pharmacological, or surgical approaches. An injury above the cauda equina (usually above L1) has a higher likelihood of being complicated with spasticity. Injuries below this level affect the cauda equina resulting in flaccid paralysis not spastic paralysis.

Pathophysiology:

Spasticity is characterized by hyper-excitability of spinal reflexes leading to exaggeration of the stretch reflex, and is a manifestation of the UMN syndrome.⁶ After sustaining a SCI, one can undergo a period of flaccid muscle paralysis and loss of tendon reflexes below the level of the spinal cord lesion which leads to a gradual development of exaggerated tendon reflexes, increased muscle tone, and involuntary muscle spasms.⁷

A thorough clinical assessment of spasticity should always be undertaken as follows: 1) differentiate spasticity from other causes of increased tone, 2) identify potential triggers, 3) measure spasticity, 4) assess spasticity’s impact on function, and 5) gather input from patients, caregivers, therapists and other rehabilitation professionals.²⁵ Spasticity is not static and therefore, assessments should be done regularly and combined with establishing goals of treatment to make decisions regarding treatment strategy.⁹ There are many triggers, including inputs from the skin, subcutaneous tissue and joints for flexion spasms⁸ (see Table 1).

Clinical Pearl: Important Questions to Ask About Patient Spasticity Experience⁴¹

- 1) What is your understanding of the term “spasticity”? What are other words you use to describe your spasticity?
- 2) What is your experience of spasticity on a daily basis?
- 3) Has your spasticity changed? How has it changed?
 - a. May indicate progression of disease (CVA; MS exacerbation; syringomyelia; worsening of degenerative disc disease)
- 4) What effect does spasticity have on your daily activities such as walking, transfer, bathing, comfort, and sleep?

Potential Triggers¹⁰:

Spasticity can be exacerbated by a number of factors (see Table 1), significant worsening of spasticity along with other symptoms might give a clue to a potentially reversible condition (eg. UTI) or perhaps serious complication (i.e. Syringomyelia, pressure ulcer). Be sure to treat the reversible causes of spasticity prior to starting pharmacological spasticity treatment.

<u>Triggers</u>	<u>Cause/Timing</u>
Bladder & Bowel	Urinary Tract Infection ^{12,23} Infection/inflammation ¹³ Urinary tract calculi ²³ Constipation ²³
Pregnancy ¹⁸	Postpartum ¹⁴ First 30 weeks; significantly increased by 32 weeks & so severe by 35 weeks it was unable to be controlled pharmacologically ¹⁵ Pregnancy is not a single stressor - can be viewed as multiple stressors (i.e. hormonal changes, increased intra-abdominal pressure, muscle fatigue, and joint hypermobility) ¹¹
Positioning	Greater spasticity seen in supine vs. sitting position ^{17,18} Improper wheelchair seating ²³ Ill-fitting orthotics ²³ Making transfers (in & out of car) ^{13,19,20} Change in posture (sitting to supine, supine to sitting, changing arm position in wheelchair, changing position in bed) ^{13,19,20} Extended periods of staying in one position (sleep or traveling long distances)
Temperature	Cold (& heat) may affect spasticity ¹³
Circadian Rhythm	Impact depends on type of injury <ul style="list-style-type: none"> • More prevalent in “complete” injuries²¹
Mental Stress	Emotions, anxiety & mental stress may increase spasticity ^{13,20}
Skin	Pressure ulcers and/or ingrown toenails ^{20,23}
Tight Clothing	Tight clothing may increase perceived spasticity ^{13,20}
Pain	
Neurological Change	Syringomyelia ⁵ , spinal infections

Table 1: Potential triggers of spasticity

Clinical Pearl: Syringomyelia- formation of an intramedullary cyst within the spinal cord after traumatic SCI. Although rare (2%), it can cause worsening neurological function. Signs and symptoms may include: reduction in sensation, worsening spasticity, gait ataxia, autonomic dysreflexia (see AD CBLM) and pain.⁵

Potential Benefits & Negative Impact:

Spasticity may be beneficial (transfers, mobility, prevent atrophy and fracture) and reducing it may worsen function, so a comprehensive assessment including patient function and goals is important.⁵

Potential Benefits	Negative Impact
Improving ambulation ²²	Reduced quality of life ^{26,27,31}
Assists with activities of daily living (ADL) ²²	Impairs activities of daily living (ADL) ^{1,13,19,29,30}
Improves Peripheral blood circulation ^{22,25}	Interferes with mobility ²¹
Facilitating movement transfers ^{24,25}	Pain ^{1,5,23,28}
Diminishes loss of muscle mass ²⁵	Contractures ^{1,13,23,29,30}
Clinical indicator for noxious stimuli below the level of the lesion ²⁰	Pressure ulcers ¹
Reducing incidence of fracture ⁵	High care needs ²³
	Loss of dexterity ^{13,23,29,30}

Table 2: Benefits and negative impacts of spasticity

Treatment:

Spasticity treatment will vary from individual to individual and it is important to strike a balance that adequately manages positive and negative impacts. **Effective management should begin with more a more conservative approach initially** (stretching, active exercise, posture/seating assessment), but will often require a combinational approach with the understanding that there is not a universally successful approach for all individuals. ³⁰ If an individual's spasticity is refractory to medication and non-pharmacological approaches, referral to a specialist (i.e. – physiatrist, neurologist) may be warranted. **Conservative approaches such as stretching, exercise and physiotherapy should be initial management** if individual has access to it.⁵ Involve occupational therapy/physiotherapy for splinting/stretching.

Non-Pharmacological⁵	Pharmacological⁵
Passive stretching ^{8,23,32,34}	Oral Medications (see below)
Active exercise ^{8,32}	Intrathecal baclofen ^{10,23,35,37}
Posture/Seating assessment	Local injections (Botulinum toxin, phenol) ^{10,23,36,37}
Electrical stimulation ^{23,32} (<i>not commonly done anymore</i>)	

Table 3: Treatment options for spasticity

Clinical Pearl: Spasticity can affect not only physical aspects, but psychological ones as well. When managing spasticity, it is important to consider patient reports, sensory experiences, and psychological evaluation as part of the complete management of spasticity.⁴¹

Medications⁵:

Baclofen is the most common medication used for spasticity, sometimes it may be necessary to switch or add other medications. It is important to be careful of the risks and side effects of

individual medications in relation to patient co-morbidities and monitor appropriately.
Medications can be combined to manage spasticity.

<u>Medication</u>	<u>Dosage (maximum)</u>	<u>Side Effects</u>
Baclofen (oral) ^{5,8,10,23,32,33,34,37} (Level 1a) GABA-B Agonist	Initial: 5mg TID <ul style="list-style-type: none"> • May increase by 5mg q 3d • Usual maximum: 20 mg QID 	Lower seizure threshold Sedation Weakness Baclofen withdrawal (seizures & hallucinations) Appears in breast milk ²³
Tizanidine ^{5,8,10,23,32,37} (Level 1b)	Initial: 2mg daily <ul style="list-style-type: none"> • Increase 2-4mg over 2-4 weeks Maintenance: 8mg tid-qid Discontinue: taper 2-4 mg/day (max: 36mg/d)	Sedation Dizziness Dry mouth Hypotension COST NOT COVERED Avoid use in liver dysfunction *monitor liver function (ALT/AST)
Dantrolene ^{5,8,10,23,37} (Level 1b) Peripheral Calcium Inhibitor	Initial: 25mg daily for 7d <ul style="list-style-type: none"> • Then 25mg tid for 7d • Then 50mg tid for 7d • Then 100mg tid/qid Use lowest effective dose (100mg qid)	Weakness Lightheadedness Nausea, diarrhea Constipation Hepatotoxicity (monitor AST/ALT q 3-6 months) COVERED COST *monitor liver function
Clonidine ^{5,8,10,32,34,37} (Level 1b) Alpha₂-adrenergic Agonist (rarely used as a single treatment) ¹⁰	0.1mg tid (1.2 mg bid)	Sedation Dizziness Bradycardia Hypotension Respiratory depression CNS depression Dry mouth Constipation
Diazepam ^{5,8,10,23,32,34,37} GABA-A Agonist	2-10mg tid-qid	Sedation Withdrawal Confusion Risk with opioids: sedation, respiratory depression, coma, death
Gabapentin ⁵ (Level 1b) GABA Analog	100-400mg capsules 600-800mg in tablets <ul style="list-style-type: none"> • Initial dose of 400mg three times a day and increase up to 600mg three times a day. • Daily max is 2400mg (800 three times a day) 	Risk of respiratory depression (worsened with opioid) CNS depression
Nabiximols ³⁸	4 to 12 sprays daily	Increase in pain

<p>(THC and CBD combination - mucosal spray)</p> <p>Nabilone³⁸</p>	<p>2 to 6mg daily</p>	<p>Psychological side effects Avoid smoked, oils and edibles due to limited evidence in the literature May consider nabilone due to lower cost</p>
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Table 4: Pharmacologic treatment of spasticity

Spasticity may change throughout the day and over time, so medication dosage may need to be adjusted

*Possibility of elevated liver enzymes; hepatotoxicity

- Note: some patients will need combination of medications

There is limited clinical information for individuals with SCI regarding the use of cannabinoids to treat symptoms such as pain, spasticity, muscle spasms, urinary incontinence, and difficulties. The current Health Canada Marijuana for Medical Purposes Regulations (MMPR) allow the use of dried cannabis in the context of severe pain and persistent muscle spasms associated with SCI or spinal cord disease in patients who have either not benefited from, or would not be considered to benefit from conventional treatments.^{44,38}

Clinical Pearl: If prescribing cannabis, the prescription must include:

- Licence info; patient name and DOB; 30 day limit; quantity in grams

CFPC authorizing cannabis for chronic pain

Prescription is not appropriate if:

- Under 25
- Have personal history of psychosis or strong family history
- Current of past cannabis use disorder
- Active substance abuse disorder
- CVD
- Respiratory disease
- Pregnant or breastfeeding

Dosing^{45, 38}

- As with all psychoactive medications, cannabis can cause mood altering, euphoria, speech changes, dizziness and sedative effects even at low doses.
- Start low and go slow (level 3)
- Vaporized cannabis are preferred over smoked cannabis

Drug	Dosage
Nabilone	2 to 6mg daily
Nabiximols	4 to 12 sprays daily
Dried Marijuana	-Daily inhaled dose of 100-700 mg of up to 9% THC content in dried cannabis -Can increase the dose approaching or exceeding 3g/day up to 5g/day

Other Treatments:

Botulinum Toxin^{5,43}

- Indicated for focal muscle spasticity (level 1)⁵
- Benefits include focal effect and no sedation as per oral medications; may last months
- Side effects can include weakness and local injection reaction
- Requires referral to specialist

Intrathecal Baclofen (ITB)^{5,43}

- A pump is implanted
- Referral to tertiary centre specializing in ITB
- Side effects can include: spinal fluid leaks, hemorrhage, infection, catheter dislodgement, pump failure.
 - intrathecal baclofen withdrawal (spasticity, itching, fever, confusion, rhabdomyolysis, seizures, death)
- Indications for ITB therapy referral include patients with intractable spasticity uncontrolled by drug therapy or intolerable side effects from oral baclofen⁴⁴
- Used as a focal treatment of spasticity
- Can be combined with morphine & other medications for pain control

Surgery^{5,43}

- Surgical destructive processes for refractory cases (rarely done):
 - Rhizotomy- sever nerve roots
 - Cordotomy- disable selected nerve tracts
 - Cordectomy- removal of part of spinal cord
 - Myelotomy- surgical incision in spinal cord

Measuring Spasticity:

There is not a universally accepted measure of spasticity. Spasticity is a multi-dimensional secondary complication of SCI and there is not a single outcome measure that can capture all the issues present with spasticity.⁵ One must choose an effective outcome measures that monitors the range of medical outcomes,

“1) technical outcome (i.e. reduction of spasm frequency, 2) functional outcome, 3) patient satisfaction, and 4) cost effectiveness.”³⁹

In clinic, the Modified Ashworth Scale can be used to measure spasticity at each joint⁴⁰ (see Appendix).

CASE CONTINUED

Cindy, age 66

Part 2

Cindy returns 4 months later to follow up, and despite treatment, she reports her mobility and coordination are worsening. She tells you that she is still experiencing problematic “stiffness” and pain. On physical exam, you note her gait is worse, that she has painful contractures in her shoulder, elbow and hand, and there is a spasm when she transfers onto the examination table. She is hoping you are able to prescribe something to alleviate her symptoms.

What questions will you ask Cindy that may provide answers about why her spasticity is continuing?

- *Is she completing her exercises and taking her baclofen?*
- *Did the baclofen initially alleviate her symptoms/did it work at all?*
- *Is there a change in her sensation, motor function, bowel, bladder, sexual function?*

She reports she is taking her medications and working on range of motion exercises. She has noticed she lacks sensation further up her body, coordination is worse, she is noticing numbness in other arm and is having trouble with starting urination.

What would you do next?

- *Complete physical to determine if there is any change in neurologic status (sensory or motor)*
- *Order MRI for syringomyelia investigation*

If Cindy’s spasticity continues to worsen, what will you do and what other treatment options are available?

- *Refractory spasticity may warrant addition of other agents (maximize baclofen)*
- *Referral to physiatry*
- *Potential for intrathecal baclofen for intractable spasticity*

SUMMARY

- Spasticity is a secondary complication affecting 65 – 78% of individuals with spinal cord injury (SCI)
- Spasticity can be exacerbated by a number of factors (see Table 1)
- Spasticity treatment will vary from individual to individual and it is important to strike a balance that adequately manages positive and negative impacts
- Effective management should begin with more a more conservative approach initially
- Conservative approaches such as stretching, exercise and physiotherapy should be initial management
- See Medications table for pharmacological treatment

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APPENDIX

Modified Ashworth Scale

(derived Bohannon and Smith, 1987):

Instructions

Place the patient in a supine position

If testing a muscle that primarily flexes a joint, place the joint in a maximally flexed position and move to a position of maximal extension over one second (count "one thousand one")

If testing a muscle that primarily extends a joint, place the joint in a maximally extended position and move to a position of maximal flexion over one second (count "one thousand one")

Score based on the classification below.

Scoring

- 0** No increase in muscle tone
- 1** Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion 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 muscle tone through most of the ROM, but affected part(s) easily moved
- 3** Considerable increase in muscle tone, passive movement difficult
- 4** Affected part(s) rigid in flexion or extension

Patient Instructions:

The patient should be instructed to relax.

Modified Ashworth Scale Testing Form

Name: _____

Date: _____

Muscle Tested

Score

_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____