A Primary Care Provider's Guide to Spasticity Management in Spinal Cord Injury

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Abstract: Background: Muscle spasticity is a common sequela of spinal cord injury (SCI) that may impact daily function. Spasticity dynamically varies and is an important physiologic response to illness or other stressors. The challenge for the general practitioner is in recognizing, treating, and developing an effective plan focused on the patient's individual goals. **Objective:** To provide the general practitioner with a basic contextual, diagnostic, and therapeutic approach to spasticity management for individuals with neurologic injury such as SCI. **Discussion:** Muscle spasticity can be disabling and can be managed effectively by using a comprehensive approach. We discuss a representative case and the assessment and planning for individuals with SCI and spasticity. Through an understanding of pathophysiology, careful history taking, and physical exam, a cause for increased spasticity can be identified, such as infection, constipation, or pregnancy. Symptomatology of these triggers is often quite different in the SCI population than in the general population. Management includes the treatment of this causative stressor as well as the thoughtful management of spasticity itself. **Conclusion:** Muscle spasticity is dynamic and requires a patient-centered approach. The general practitioner can play a key role in recognizing and treating spasticity in an individual with SCI. Comprehensive management to meet patient and caregiver goals involves primary care providers, specialists, and allied health practitioners. **Key words:** *myelopathy, neurorehabilitation, paralysis, primary care, spasticity, spinal cord injury*

Health Maintenance Checklist

1. Annual evaluation should review the adequacy of bladder and bowel programs, skin health, presence of neuropathic or nociceptive (e.g., musculoskeletal) pain, and any changes in spasticity, strength, sensation, or functional activities.
2. Annual review of efficacy of pharmacologic and nonpharmacologic interventions. Is the current regimen meeting the patient's treatment goals? Is the patient requiring increasing or decreasing doses of antispasmodic medication?
3. Monitor liver function tests if on tizanidine and dantrolene (at baseline, 1 month, and then every 3 months).

Episodic Care Key Points

1. Spasticity may change over time. A yearly assessment of spasticity (including its effect on patient's function, activities of daily living [ADLs], and overall quality of life), physical exam, efficacy of medications, and nonpharmacologic interventions is recommended.
2. Periodic reevaluation is needed to monitor the response to any changes in medication or treatment intervention.
3. In cases of new onset or worsening spasticity, identify potential triggers of spasticity. Bladder, bowel, and skin issues (e.g., UTIs, stones, constipation, wounds) are common causes.
4. Discuss spasticity and management with women of childbearing age preconception.

Case Report

Jane is a 34-year-old female motorcycle racer who sustained a spinal cord injury (SCI) 3 years ago resulting in T9 complete paraplegia. She is seeing you to establish primary care. Jane has neurogenic bowel and bladder and uses a manual wheelchair. Her only medications are oxybutynin (Ditropan) and baclofen (Lioresal). She reports her spasticity was well controlled on a low dose of baclofen until 3 weeks ago. Leg spasms are now increased, interfering with sleep, transfers, dressing, and positioning in her wheelchair and bike. She denies any preceding trauma or illness. Bladder and bowel programs remain stable, with no reported incontinence (urine or fecal) or constipation. Physical exam (including abdomen and skin) was normal. Sensory and motor exam

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was consistent with a T9 neurological level of injury. Pertinent findings include hyperreflexia, clonus, frequent extensor spasms of her legs, and slight swelling of the right leg.

Definition

Spasticity is defined as "a disorder of sensorimotor control resulting from an upper motor neuron (UMN) lesion, presenting as an intermittent or sustained involuntary activation of muscles."[1(p5)] This inclusive definition recognizes other symptoms of UMN syndrome that are part of the patient experience: spasms (flexor, extensor, adductor), hyperreflexia, hypertonia, clonus, dyssynergia (inappropriate co-contraction of antagonistic muscle groups), and abnormal reflexes (Babinski, Hoffman, crossed adductor). It acknowledges that spasticity is not due solely to stretch reflex hyperexcitability nor is it always velocity dependent.[2]

Muscle spasticity is a common sequela of SCI, with an estimated prevalence of 65% to 93% at 1-year post injury.[3,4] It can develop over weeks and months and persist for years. Spasticity is a dynamic entity. It changes over time and may be influenced by other conditions unrelated to the original damage to the cord. For the general practitioner, the challenge lies in identifying spasticity, knowing when to treat and when not to treat, and formulating a treatment plan that will meet patient goals.

Pathophysiology

The spinal cord becomes areflexic immediately after SCI. This period is called spinal shock. It is characterized by loss of tendon reflexes, flaccid paralysis, decreased muscle tone, and loss of sensation below the lesion level. If the SCI is due to an UMN lesion, spasticity emerges over the next 4 weeks and generally stabilizes after the first 12 months.[5] Spasticity progression and severity vary, even amongst individuals with similar injury levels. Hyperreflexia, increased muscle tone, and involuntary muscle spasms occur.[5,6] Multiple complex spinal mechanisms contribute to spasticity. The loss of descending inhibitory signals results in hyperactivity of segmental reflexes. Loss of monoaminergic input leads to enhanced excitability of motoneurons, interneurons, and denervation hypersensitivity. Axonal sprouting of neurons involved in segmental reflexes also plays a role. This all culminates in a decreased threshold for motor unit activation and increased response to stimuli.[7] Mechanical changes to muscle after SCI is thought to contribute to the development of increased tone.[8]

Epidemiology

Incidence of spasticity is higher in individuals with cervical and upper thoracic injuries and incomplete SCI. Spasticity is most prevalent in individuals with incomplete tetraplegia.[9] Those with injuries at or below the conus medullaris typically present with areflexia and flaccid paralysis. Problematic spasticity (i.e., spasticity requiring treatment or causing functional limitation) is highest in those with motor incomplete SCI. Patients with an American Spinal Injury Association Impairment Scale (AIS) C have a higher incidence of problematic spasticity compared to other grades. They are at the most risk for having spasticity refractory to treatment.[4]

Impact of Spasticity

Spasticity can be disabling. It can interfere with the ability to perform activities of daily living (ADLs), such as catheterization, bowel care, dressing, positioning, transfers, mobility, and participation in rehabilitation therapies. It can increase caregiver burden. Severe hip flexor and adductor spasms can interfere with performing catheterization or perineal hygiene, while extensor spasms can interfere with the bowel regimen. Spasticity may be associated with pain, fatigue, poor sleep, impaired sexual function, negative self-image, and decreased quality of life.[2,3] Spasticity can cause pain or be the result of something painful, like a tight strap or a sunburn.

Spasticity can be beneficial. It can facilitate function (e.g., transfers, sitting, standing, walking), improve venous return (decreases edema and the risk of deep venous thrombosis),[10] reduce fracture risk, and protect against muscle atrophy.[11,12] Lipid profile and glucose metabolism may be improved.[13] Spasticity can also be a warning sign of pathology or injury below the SCI.

Clinical Assessment

History and physical examination are crucial in the diagnosis and management of spasticity. As spasticity changes over time, a yearly assessment is recommended. A more frequent assessment may be warranted to monitor the response to any interventions instituted.

History

When obtaining a patient's medical history, PCPs should:

- Establish the patient's perceived experience with spasticity. Clarify symptoms, severity, and localization. How does spasticity affect the individual's function, daily activities, comfort, sleep, and overall quality of life? Does the spasticity assist in trunk stability, transfers, standing, walking, edema control, weight management, or alerting the individual that something is wrong?
- Assess for positive and negative signs of UMN syndrome (**Table 1**).[14] Patients may present with muscle spasms, muscle stiffness, hyperreflexia, or involuntary movement of trunk and limbs. A recent study found that muscle stiffness was more problematic than spasms or clonus. It had the greatest negative impact on ADLs and psychological agitation.[15]
- Identify whether the spasticity is focal, regional, or generalized. Individuals with SCI tend to have generalized spasticity below their lesion level.

Table 1. Features of upper motor neuron syndrome[14]

Positive	Negative
Hyperreflexia	Paralysis/paresis
Spasticity	Lack of coordination
Clonus	Loss of dexterity
Flexor / extensor spasms	Fatigue
Dyssynergic co-contraction during movement	
Dystonia[a]	
Athetosis[a]	

[a]Seen in basal ganglia and movement disorders.

- Identify with the patient the potential triggers of spasticity (**Table 2**). These may emerge upon reviewing different organ systems affected by paralysis. Bladder, bowel, and skin problems (e.g., urinary tract infections [UTIs], constipation, wounds) are common culprits. For example, UTIs in this population typically present as changes in urine smell or color or an increase in spasticity. Limb or joint swelling may herald the presence of fracture, heterotopic ossification (HO), or deep venous thrombosis, which can trigger spasticity.
- Expand the review of systems as necessary to assess for systemic illnesses and other conditions that can present as increased spasticity. Ask if the patient has experienced any increase in weakness, numbness, pain, or loss of nociceptive and thermal sensation. This may indicate the presence of syringomyelia or a tethered cord, which are other potential complications of SCI.
- Review medications for side effects that can exacerbate spasticity. Constipation is a well-known side effect of opioids. Constipation can lead to bowel distention that in turn can trigger spasticity. Constipation is a common side effect of oxybutynin and other anticholinergics used for treatment of neurogenic bladder. Selective serotonin reuptake inhibitors can increase spasticity.[16]
- Inquire about the condition of the patient's durable medical equipment (DME), such as a wheelchair or orthotic devices (e.g., braces). Ill-fitting or inadequate DMEs can predispose the patient to abnormal posture/positioning and pain and increase the risk of contracture and skin breakdown, which in turn can trigger spasticity.
- Ascertain if the patient considers spasticity as beneficial or problematic. Work with the patient in identifying goals of treatment.

Physical examination

PCPs should conduct a concise physical examination to confirm the history and look for common triggers of spasticity. The exam should include an evaluation for bladder or abdominal distention and skin breakdown at pressure-

Table 2. Triggers of spasticity

Triggers	Examples	Recommendations
Bladder and bowel	UTI Urinary tract calculi Gallstones Diverticulitis Appendicitis Abscess Other infection or inflammation Constipation	Check urinalysis, C&S Renal US to evaluate for hydronephrosis or stones Abdominal x-ray to evaluate stool burden and rule out obstruction Typical sensory signs of abdominal pathology may be absent or nonspecific in SCI; if initial exams (US or x-ray) are inconclusive or you have a high index of suspicion, obtain CT scan of abdomen.
Skin	Pressure injury Maceration or skin irritation Ingrown toenails Tight clothing Osteomyelitis	Patient education regarding skin checks, pressure-relieving techniques, hygiene Observe transfer technique Podiatry referral MRI if osteomyelitis is suspected
Pain	Neuropathic pain Nociceptive (musculoskeletal, visceral pain)	Check for organic causes (i.e., other triggers) Medication adjustment
Pregnancy	Physical and hormonal changes (enlarging uterus, joint laxity) Postpartum	Preconception counselling Education regarding medications during pregnancy Referral to therapy for anticipated needs as pregnancy progresses (e.g., wheelchair, transfers, etc.)
Positioning	Change in position Ill-fitting orthoses Wheelchair issues	Orthotic evaluation Wheelchair/seating evaluation
Temperature	Cold Heat	Appropriate clothing; dress in layers to adjust to changes in temperature and temperature dysregulation
Mental stress	Anxiety Mental stress	Consider psychology/psychiatry consult for coping and behavioral strategies
Physical or neurological change	Syringomyelia Tethered cord Fracture Heterotopic ossification Compression neuropathy	MRI of spine with and without contrast to evaluate for syringomyelia or tethered cord X-rays if warranted EMG/NCS
Systemic illness	Deep venous thrombosis Systemic infection	Obtain doppler ultrasound Check CBC, CMP

Note: CBC = complete blood count; CMP = complete metabolic profile ; C&S = culture and sensitivity; EMG/NCS = electromyography/nerve conduction study; US = ultrasound; UTI = urinary tract infection.

sensitive areas including the buttocks and heels. The patient's feet should be examined for inflammation or breakdown at the toe nails. Limb swelling may be the only sign of a deep venous thrombosis, undiagnosed fracture, or HO.

The neuromuscular exam consists of evaluation of tone, reflexes, motor strength, sensation, range of motion (ROM), and posture. The Modified Ashworth Scale (MAS) and Penn Spasm Frequency Scale (PSFS) are some of the commonly used measures to assess spasticity.[15] The MAS assesses tone, or resistance to passive movement, which is only one aspect of spasticity. The PSFS is a self-reported measure of severity and frequency of spasms. The combined use of the MAS with PSFS can enhance the characterization

Table 3. Spasticity scales[15]

Modified Ashworth Scale		Penn Spasm Frequency Scale	
0	No increase in muscle tone	0	No spasms
1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion (ROM) when the affected part(s) is moved in flexion or extension	1	Mild spasms with movement or stimulation
1+	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM	2	Spasms <1 per hour
2	More marked increase in muscle tone through most of the ROM, but affected part(s) easily moved	3	Spasms >1 per hour
3	Considerable increase in muscle tone; passive movement difficult	4	Spasms >10 per hour
4	Affected part(s) rigid in flexion or extension		

of the patient's spasticity.[17] The MAS and PSFS assess specific components of spasticity and do not address the impact of spasticity on function. ROM limitations should be determined. Contractures can interfere with positioning in the wheelchair or brace, leading to increased pain, wounds, pressure injury, and spasms. A decline in strength and sensation (e.g., increase in numbness or sensory loss) should alert the PCP to assess for other pathologies (e.g., syringomyelia or peripheral nerve compression).

The PCP should evaluate the patient's posture in the wheelchair, checking for abnormal positioning and pressure points that cause pain or skin breakdown. The ischial tuberosity should be in contact with the wheelchair seat. If possible, the patient should be observed during functional tasks such as transfers, changing position (e.g., lying down, sit to stand) or walking (if applicable).

Management

Spasticity treatment necessitates a patient-centered approach. The severity and extent of symptoms need to be considered in relation to their impact on the individual's function, risk of developing secondary complications (e.g., pain, wounds, contractures), and quality of life. It is important to remember that not all spasticity is disabling or needs treatment. Once spasticity is identified as problematic, treatment goals are established together with the patient.

Defining the extent of spasticity will guide the PCP in deciding options for treatment. Focal spasticity involving a limb or a specific muscle group (e.g., shoulder adductors, ankle plantar flexors) may benefit from oral antispasmodics, injections, or physical modalities. Some individuals with SCI present with generalized spasticity, involving multiple muscle groups that may not be adequately managed by oral medications, injections (trigger point or Botox), or therapy. Intrathecal delivery of medications may be needed, solely or in combination with other treatment.[18]

A combination of pharmacologic and nonpharmacologic interventions may be needed to optimize management. A stepwise and multimodal approach is customary; however a more aggressive approach will be warranted in certain instances. The response to treatment needs to be reevaluated periodically.

Referral to a physiatrist or neurologist is indicated in the following cases: severe spasticity, spasticity refractory to initial management, and if focal chemodenervation injections (e.g., Botox, phenol, ethanol) or intrathecal delivery of baclofen is being considered. Intrathecal baclofen may benefit individuals with generalized spasticity that is unresponsive to oral medications or who cannot tolerate the side effects of oral medication.

Other neurosurgical interventions such as rhizotomy, cordotomy, cordectomy, and myelotomy will require referral to a tertiary center. An orthopedic consultation may be needed for surgery (e.g., tendon lengthening, tenotomy) to correct or improve limb deformities and positioning that have not responded to conservative measures.

Nonpharmacologic management

Fundamental to management is the identification of treatable conditions that trigger, exacerbate, or prevent optimum control of spasticity. UTIs, constipation or bowel impaction, wounds, and other pathologies can trigger new onset or worsening spasticity in a patient with chronic SCI with controlled spasticity. Addressing these conditions typically results in improvement of spasticity.

Physical and occupational therapy utilizes physical interventions such as stretching and positioning, range of motion, orthotic management, wheelchair seating, cryotherapy, heating modalities, and electric stimulation. An individualized exercise program can be designed to incorporate these modalities.

Prolonged stretch can decrease muscle spindle sensitivity and IA and group II afferent input thus decreasing excitation of motoneurons. This can be facilitated through physical stretching, weight bearing, use of tone-reducing orthotics (e.g., ankle-foot orthoses), or serial casting.[19] Bivalve casts allow for skin inspection. Focal injections prior to casting are utilized to improve stretch. Proper posture and positioning can reduce triggering of symptoms.[20] Cold decreases spasms and clonus by decreasing muscle spindle sensitivity to stretch. Heat decreases muscle spasms and tone and increases the pain threshold. The effects of heat and cold are short lived (30-60 minutes). They are best combined with other modalities. Monitoring of skin is advised, as heat and cold application can lead to skin damage.[19] Electric stimulation over spastic muscles exerts immediate but short-term effects. Other adjunctive treatments include aquatic therapy, vibration therapy, and robotic-assisted gait training.[20] Other emerging therapies include neuromodulation via epidural or transcutaneous spinal cord stimulation (TSCS). Application of TSCS at higher frequencies (50 Hz) has shown to temporarily improve spasticity in SCI.[21,22] Epidural spinal cord stimulation at frequencies of 50 Hz or higher can improve spasticity, however precise placement of the leads is important.[23] Complementary alternative treatments such as acupuncture, yoga, and massage may be considered, however there are no current studies on their efficacy in treating spasticity in SCI.[19]

Pharmacologic management

Oral antispasticity agents include those that act on the gamma aminobutyric acid (GABA) system (baclofen, gabapentin, benzodiazepines), α-2 adrenergic system (tizanidine, clonidine), or peripherally (dantrolene). Common side effects include sedation (specifically with GABA and α-2 agonists) and weakness. Weakness is thought to be related to the unmasking of underlying UMN weakness and is not necessarily a direct drug effect.

A "start low and go slow" approach is advised when initiating treatment, as inappropriate dose escalation can lead to the development of side effects. Before the medication is deemed ineffective, the PCP should titrate to the maximum dose tolerated by the patient. If the patient can tolerate only low doses or does not respond to one agent, a combination of drugs can be tried. Gradual tapering of medication is advised as sudden discontinuation can lead to rebound spasticity or withdrawal symptoms. A summary of common oral antispasticity agents used in SCI is listed in **Table 4**.[24-26]

PCPs should consider the individual's age, comorbidities, and cognitive status when choosing a medication. Drug efficacy and patient tolerance need to be routinely assessed. PCPs can explore with the patient how spasticity affects daily activities, as this can help identify optimum timing and dosing of medications. A dose may be required first thing in the morning if spasticity interferes with bed mobility or ADLs such as toileting and dressing. A lesser dose may be required during the day in a person who uses spasticity to transfer, stand, or walk.

Patients should be monitored for any unexpected side effects or drug interactions. Ciprofloxacin, commonly used for UTI treatment, can increase plasma tizanidine levels resulting in hypotension and increased sedation. Medication choice depends on the individual's response, tolerance, or in some cases, insurance.

Current medications, their safety during pregnancy and lactation, and alternative treatment options should be discussed with females of reproductive age who are considering pregnancy in the future.[27]

Table 4. Oral antispasticity medications[24-26]

Medication	Dose[a]	Side Effects
Baclofen GABA-B agonist (Level 1a) 1st line in SCI	Initial: 5 mg tid Max daily dose: 80 mg divided into 3 to 4 doses Increase: 5 mg/dose every 3 days	Somnolence, dizziness, asthenia, constipation, nausea, poor muscle tone Precaution: Dose adjustments in renal disease Avoid abrupt discontinuation
Tizanidine[b] Alpha-2 adrenergic agonist (Level 1a)	Initial dose: 2 mg daily Max daily dose: 36 mg in three divided doses Increase: 2-4 mg over 2 to 4 weeks Discontinuation: decrease dose by 2-4 mg per day	Somnolence, dry mouth, asthenia, dizziness, hypotension Precaution: Hepatotoxicity; monitor LFTs
Gabapentin GABA analog (Level 1b)	Initial dose: 300 mg tid Max daily dose: 3600 mg in three divided doses	Dizziness, somnolence, dry mouth, edema, constipation Precaution: Dose adjustment in renal impairment Avoid abrupt discontinuation
Dantrolene[b] Peripheral calcium inhibitor (Level 1b) 1st line if SCI + cognitive dysfunction, otherwise 2nd line	Initial: 25 mg daily x 7 days Max dose: 100 mg qid Increase: 25 mg tid x 7 days 50 mg tid x 7 days 100 mg tid x 7 days	Muscle weakness (peripheral and respiratory) Sedation Nausea, diarrhea Precaution: Blackbox warning: Hepatotoxicity, symptomatic hepatitis; monitor LFTs May cause more weakness in patients with marginal strength
Clonidine Alpha-2 adrenergic agonist (Level 1b)	Initial: 0.05 mg bid to 0.1 mg bid Max daily dose: 0.4 mg in two divided doses	Orthostasis, bradycardia, dry mouth, constipation, drowsiness Precaution: Abrupt discontinuation may lead to withdrawal symptoms (e.g., rebound hypertension, headache, agitation) Dose adjustment in renal impairment
Diazepam GABA-A agonist	Initial dose: 2 mg tid Max daily dose: 10 mg qid	Drowsiness, impaired memory, decreased attention Precaution: Tolerance, dependency; risk for withdrawal syndrome Caution in elderly Avoid in alcohol

Note: bid = twice daily; qid = four times a day; tid = three times a day.
[a]Upon discontinuation, gradual dose reduction for all medications is recommended. Risk of withdrawal is seen with abrupt discontinuation of baclofen, diazepam, clonidine, and gabapentin. For gabapentin, do gradual dose reduction, discontinuation, or substitution over a minimum of 1 week.
[b]Monitor liver function tests (LFTs) periodically with dantrolene and tizanidine at baseline, 1 month, 3 months, and 6 months.

Concerns have been raised regarding antispasticity medications interfering with neurologic recovery or improvement. It is important to stress that use of antispasticity agents does not necessarily preclude neurologic recovery and in fact may be needed to allow an individual to participate in therapy or perform ADLs. A longitudinal study of individuals who received baclofen within 4 weeks of acute injury showed no evidence that baclofen limits neurologic recovery during the transition from acute to chronic injury.[28]

Limited evidence exists regarding the beneficial effect of medical cannabinoids (e.g., nabiximols, dronabinol, nabilone) in treating spasticity in SCI.[29-31] They may be considered in spasticity refractory to standard therapies. Medical marijuana in other forms (smoked, oils, or edibles) are inadequately studied and not recommended as first line. Only dronabinol and nabilone are available in the United States; they are indicated for chemotherapy-induced nausea and vomiting. Nabiximols are available elsewhere (e.g., Canada, the UK, New Zealand, etc.) for treatment of moderate to severe spasticity in multiple sclerosis unresponsive to standard treatment.[31]

Case Resolution

In approaching Jane's case, first rule out any treatable noxious or triggering stimuli. Initial labs were unremarkable including urinalysis, complete blood count (CBC), basic metabolic panel (BMP),

and a negative pregnancy test and stool guaiac. It is unlikely that she has a UTI, any intra-abdominal process, or pregnancy. You suspect a fracture or deep vein thrombosis due to right leg swelling. X-ray showed a small nondisplaced fracture of the distal femur and was negative for HO. You order a hinged knee brace and refer Jane to orthopedics. You discuss treatment options with Jane to decrease her spasticity. You recommend doubling the baclofen dose at night to allow for sleep. Jane is counseled on the potential effects of increased baclofen on cognition and driving. You have her return in 6 to 8 weeks for reevaluation of spasticity, with the plan to reduce oral baclofen to its prefracture dose once her spasticity returns to baseline.

Conclusion

Muscle spasticity is a common condition following SCI. Utilizing a patient-centered approach in defining the patient's experience with spasticity and its impact will aid the PCP in instituting a treatment plan that will be in concordance with the individual's needs and optimize function. Comprehensive management will involve the patient, family, caregivers, PCPs, allied health providers, and specialists.

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