Demystifying spasticity in primary care

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Abstract

Objective To raise awareness of spasticity in primary care and clarify how to identify, diagnose, and manage it effectively and efficiently in patients with pre-existing neurologic conditions.

Sources of information PubMed was searched for articles published from 1970 to May 2018 using the terms *spasticity, spasticity in physical disability, spasticity in mobility impairment,* and *spasticity with family medicine or primary care*. Other relevant guidelines and resources were reviewed and used.

Main message Spasticity is a common secondary complication in conditions such as spinal cord injury, multiple sclerosis, stroke, cerebral palsy, and other neuromuscular physical disabilities and can have a negative effect on health and quality of life. Factors such as inconsistent definition, poorly understood mechanism, and relatively low prevalence make spasticity seem like a daunting condition to manage. Furthermore, its variable presentation and effect on a patient's quality of life, and its range of treatments with varying levels of evidence, can make treatment challenging in primary care and in other clinical settings. Family physicians play an important role in recognizing and inquiring about spasticity and its changes, triggers, and effects on function. Ruling out reversible causes is important. Many management strategies can be instituted by family physicians.

Conclusion Managing spasticity might be unfamiliar to many practitioners. It is important for physicians to understand spasticity and the potential treatment options available to improve quality of life. The current review provides concise information on the clinical relevance of spasticity in primary care and how to assess and manage it effectively and efficiently in those with chronic neurologic conditions.

Editor's key points

- Spasticity is a very common secondary complication of many neuromuscular physical disabilities, and symptoms vary from patient to patient. Primary care providers play an important role in recognizing, assessing, and managing spasticity. Understanding how patients describe and perceive spasticity is important in assessing and managing treatment.
- Spasticity can be beneficial for activities of daily living and quality of life (eg, by facilitating transfers, increasing muscle tone, increasing stability in sitting or standing); it is important to obtain a thorough clinical assessment to determine when the spasticity is troublesome, whether there is a reversible cause, potential triggers, and what strategies the patient is already using to relieve the symptoms.
- Management of spasticity should involve a comprehensive approach including the patient, his or her family members, and his or her caregivers; allied health practitioners; primary care providers; and specialists. Referral to specialists should be considered in cases of severe spasticity, complications, symptoms refractory to treatment, intolerance of treatment, or focal spasticity that might benefit from local treatment.

Secondary complications related to physical disabilities such as spinal cord injury (SCI), stroke, cerebral palsy, traumatic brain injury, and multiple sclerosis (MS) can lead to further functional impairment and emotional distress. For primary care providers (PCPs), knowledge of these secondary complications and possible strategies to circumvent further impairment are important to allow individuals living with physical disabilities to have comfortable and productive lives. One such complication, affecting 85% of children with cerebral palsy,[1] up to 78% of individuals with SCI,[2,3] more than 80% of individuals with MS,[4] and 30% of individuals after stroke,[5,6] is spasticity. Spasticity can vary in severity and is caused by various physiologic factors. Although it causes impairment in many cases, it is not always detrimental.[7-10]

Case description

Jeff is a 30-year-old man who suffered a complete SCI at T5 3 years ago, resulting in paraplegia. He is nonambulatory, uses a manual wheelchair, and has neurogenic bowel and bladder. He reports that he is bothered by his legs "jumping" on their own.

Jeff believes this has worsened during the past year and it is affecting his sleep and daily activities such as transferring and dressing. He reports

his bowel and bladder functions are managed well. He reports that his wheelchair and seat cushion are 5 years old and you note that the cushion is very worn. Jeff reports he was taking baclofen, but he forgot to renew the prescription over a year ago. On assessment, he lacks sensation from his mid chest distally, he has grade 4 and greater deep tendon reflexes throughout both lower extremities, and clonus is easily elicited by stretching his plantar flexors. You note these findings do not seem changed from previous assessments. He has mild knee flexion and ankle plantar flexion contractures. How would you manage his spasticity?

Main message

Spasticity is a poorly understood condition; it is commonly referred to by patients as "stiffness" or "tightness." It can result from an upper motor neuron (UMN) lesion[7-10]; these lesions are characterized by positive (exaggerated) and negative (reduced) features (**Box 1**). Positive features result from an increase in involuntary movement, and negative features result from a reduction in voluntary motor activity.[10] *Spasticity* is often used interchangeably with *clonus* and *hyperreflexia,* but these are separate features of UMN lesions. The current accepted definition of *spasticity* is "disordered sensori-motor control, resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary activation of muscles."[11] The new definition is broader to reflect various sensorimotor signs and symptoms of UMN lesions (eg, clonus, dyssynergic patterns during movement)[11] as opposed to the classic definition, "velocity-dependent exaggeration of stretch reflexes, resulting from abnormal intraspinal processing of primary afferent input. Clinically this implies increased muscle tone."[10] Spasticity can be further examined from a functional point of view, ie, whether it is perceived by the individual or his or her caregivers as hindering body function, activities, or participation.[12,13]

Owing to the various defining criteria of spasticity, as well as it having both adaptive and maladaptive properties, extensive knowledge of the individual's experience of spasticity and a thorough clinical assessment are required for treatment.

Box 1. Motor features of upper motor neuron lesions

Positive (exaggerated) features
- Increased tendon reflexes
- Clonus
- Spasticity
- Flexor or extensor spasms
- Dyssynergic patterns of co-contraction during movement
- Babinski sign present

Negative (reduced) features
- Muscle weakness
- Loss of dexterity
- Fatigability

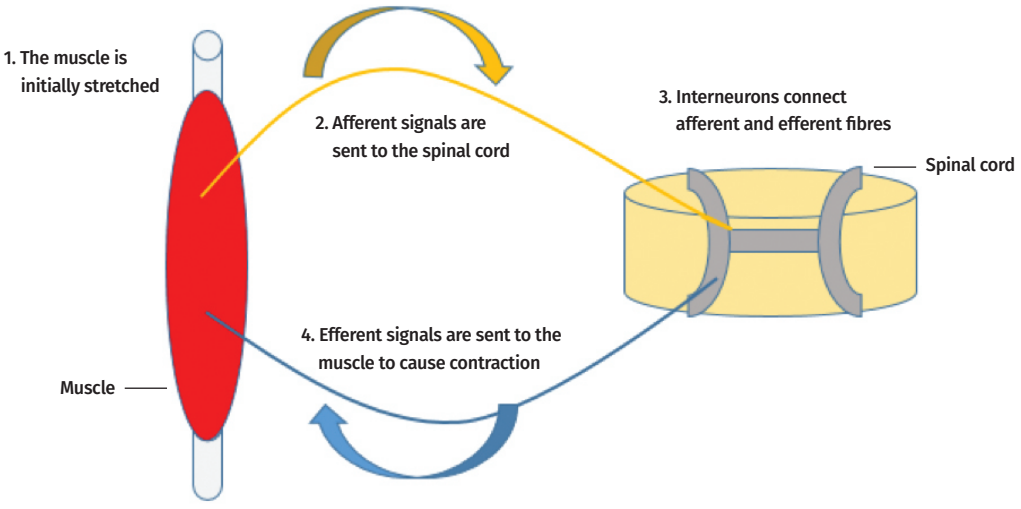
Pathophysiology. The natural stretch reflex is a reflex pathway that originates in the muscle spindles and travels via sensory neurons to the spinal cord and synapses, with motor neurons innervating the muscle from which the stimulus originated (**Figure 1**).[14,15] To prevent injury caused by excessive or sustained stretching of the muscle, sensory neurons synapse with interneurons in the spinal cord to facilitate muscle contraction. Increased muscle tone following passive stretching of a muscle is caused by the hyperexcitability of this tonic stretch reflex owing to SCI, stroke, MS, or another similar neurologic condition.[14,15] The classic stretch reflex, whereby a tendon is externally tapped and the muscle reacts by lengthening (eg, patellar tendon tap results in lengthening of the quadriceps muscle), might be exaggerated with neurologic disease or damage. Reduced inhibition results in an exaggeration of the muscle response, leading to hyperreflexia and clonus.[14,15]

Clinical signs and symptoms. As described, *spasticity* refers to a component of the UMN syndrome and reflects abnormal increased muscle tone.[13] Spasticity might present clinically with increased muscle tone (muscle stiffness), enhanced tendon reflexes, muscle spasms, clonus, abnormal gait, and inability to bend limbs. Muscle spasms are characterized by sudden, involuntary muscle contractions, and are particularly common in knee extension and hip flexion.[14-16] Clinically, increased muscle tone in spasticity typically affects "antigravity" muscles. In the upper extremities, there is increased tone in the shoulder adductors; elbow, wrist, and finger flexors; and forearm pronators.[13] In the lower extremities, there is increased tone in the hip adductors, knee flexors, and ankle plantar flexors and invertors.[13] Spasticity is highly variable within and among patients.[9] Severity of spasticity can vary throughout the day and in some cases the presence of spasticity can be beneficial for movement (**Box 2**).[9,10,15,16]

The severity and presentation of clinical symptoms varies with neurologic disease. For example, 93% of individuals with cervical SCI and complete quadriplegia reported spasticity, whereas 72% of individuals diagnosed with complete thoracic SCI reported spasticity.[17] Although spasticity is still very common among lower-level lesions, it is highly prevalent in cervical SCI. Research has suggested that the presence of spasticity has been associated with poorer mobility in MS, although this might not be the case in stroke and cerebral palsy.[4] These inconsistencies are most likely a result of incongruent definitions and measurements of spasticity. Spasticity can be exacerbated by a number of factors such as pregnancy, mental stress, and temperature change (**Box 3**).[7-11,13,15,16]

Office management and diagnosis. This clinical review discusses pre-existing neuromuscular conditions and not new-onset spasticity. Primary care providers often have

Figure 1. Monosynaptic stretch reflex



Box 2. Potential positive and negative effects of spasticity

Potential benefits[9,10,15,16]
- Assists with activities of daily living (eg, facilitates grip for holding objects)
- Improves peripheral blood circulation
- Facilitates movement transfers
- Reduced muscle atrophy; improved bone strength
- Reduced incidence of fractures
- Improved ambulation due to stiffening of muscles

Negative effects[9,10,15,16]
- Impairs activities of daily living (eg, feeding, dressing, hygiene)
- Pain or contractures
- Interferes with mobility
- Increased incidence of pressure ulcers
- Loss of dexterity
- Reduced quality of life or high care needs (social isolation, mood disturbance, relationship problems, vocational disruption)

Box 3. Spasticity triggers

Bladder and bowel dysfunction[7,13]
- Urinary tract infection
- Bladder retention
- Constipation

Pregnancy[8-10]
- Postpartum
- Hormonal changes, fatigue, body change

Positioning[7,8,11,13,15]
- Standing
- Transferring
- Improper wheelchair or seating

Temperature[8,9]
- Unusual cold or heat

Emotion[9,10,13]
- Anxiety and mental stress

Skin[7,8,13]
- Pressure ulcers
- Ingrown toenails
- Tight clothing

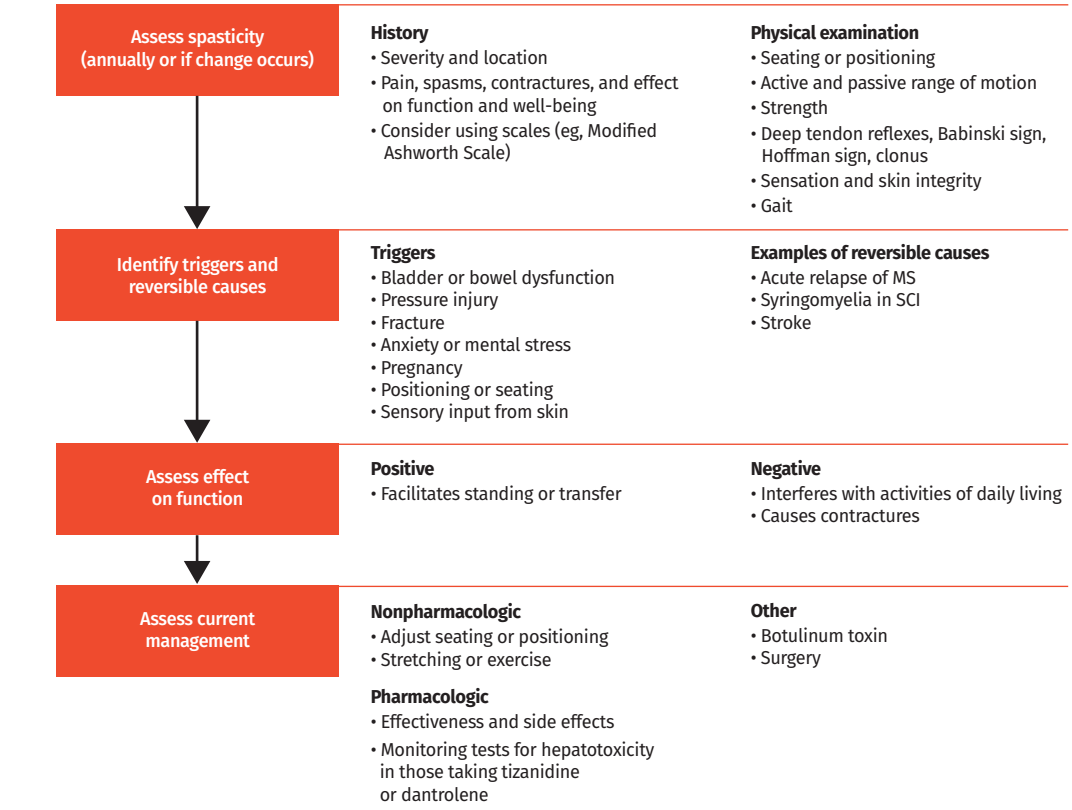
Physical or neurologic change[9-11,13]
- Worsening of underlying condition (eg, multiple sclerosis exacerbation, recurrent stroke)
- Syringomyelia (fluid-filled cavity or cyst within spinal cord)
- Fractures

Other[10,11,13,16]
- Discontinued medications
- Deep vein thrombosis
- Generalized infection

long-standing relationships with patients and therefore have an important role in determining if worsening spasticity is a result of triggers (**Box 3**),[7-11,13,15,16] disease progression, or a new condition that requires investigation or referral.[13] To do this, clinicians must perform a comprehensive assessment that involves a patient history and physical examination (**Figure 2**).[12] It is important that PCPs complete and document a baseline assessment.[12]

Unfortunately, there is no single test that captures all the aspects of spasticity and how it might present or affect an individual.[12] It is important to inquire about pain, muscle and joint stiffness, and spasms, and how they affect an individual's health (eg, fractures, mobility), function (sleep, falls, activities of daily living), and emotional well-being (eg, mood, relationships).[7,12] Active and passive range of motion of potentially affected limbs should be examined. Assessing active movement allows clinicians to determine the degree of movement along with quality (eg, presence of abnormal movement synergies).[12]

Figure 2. Strategies for assessing spasticity



MS—multiple sclerosis, SCI—spinal cord injury.

Assessing passive movement allows clinicians to determine the degree of resistance to rapid motion and the presence of soft-tissue shortening (contractures).[12] In the Ashworth Scale and the Modified Ashworth Scale, a muscle is taken passively from its shortened position to its maximal length over 1 second and the degree of resistance is rated (eg, to test the biceps, flex the elbow fully and extend it in 1 second).[12] Weakness might be detected using manual muscle testing; scores should be documented each time for reference. Appropriate neurologic examination should be performed to exclude any worsening or new disease.[7] The patient should be examined for exaggerated or abnormal deep tendon reflexes, clonus, and Babinski and Hoffman signs. Gait (when possible) should be examined for dyssynergic patterns, especially for changes from baseline.[7] Skin should be assessed for breakdown or evidence of excessive pressure.[7] As spasticity is not static, inquiry about spasticity (affected areas, severity, effect on function and changes) should be done regularly (at least yearly or as needed owing to change).

It is important to note that a substantial change or increase in spasticity should prompt an evaluation to rule out new or reversible causes; spasticity is often worsened in common complications like urinary tract infection, constipation, and pressure injury (**Box 3**).[7-11,13,15,16] In traumatic SCI, syringomyelia (development of a fluid-filled cavity or cyst in the spinal cord) can occur, resulting in new or worse spasticity and decline in neurologic function (change in sensory level, motor function, pain, gait, overall function), and requires magnetic resonance imaging or referral.[3,12] In MS, a change in spasticity might indicate an acute relapse and warrant investigation, referral, or other treatment (eg, steroids). Treating an underlying cause of worsened spasticity, when identified, is preferable to reflexively increasing medications, which might worsen the underlying condition (eg, constipation) and spasticity.

It is important that PCPs are aware of spasticity and routinely ask about it. There are a number of clinical and functional measures for spasticity[12]; however, most were not developed specifically for primary care and have variable levels of reliability and validity.[12] The Ashworth Scale and Modified Ashworth Scale, Modified Tardieu Scale, Penn Spasm Frequency Scale, clonus score, and Spinal Cord Assessment Tool for Spastic

Reflexes have been recommended to assess the presence and extent of spasticity in SCI.[12] Primary care providers should choose assessment measures that are conducive to their clinical practice and be sure to document findings to measure changes.[12,13] Self-reports from the patient are also highly valuable (**Figure 2**).[18]

Nonpharmacologic treatment. The treatment and management of spasticity will vary from patient to patient and must be addressed at the individual level. As spasticity can have a positive effect on activities of daily living and quality of life (increased muscle tone, increased stability in sitting and standing, facilitates transfers, etc), the potential beneficial effects of spasticity have to be weighed against the negatives to create a balanced treatment, especially when many pharmacologic treatments have negative side effects.[7-11] Treatment should begin conservatively, and might include exercise, stretches, physiotherapy, and massage.[19,20] Specific stretches and exercises are aimed at spastic and synergistic muscles, reducing muscle tone, and improving joint mobility and range of motion.[20] Various forms of electrical stimulation (usually provided by a physiotherapist) have shown promise in reducing spasticity; however, their use in clinical practice is still limited and not accessible to all (eg, expertise, cost, location).[19] Improved posture and positioning, especially in those using wheelchairs, is a very important factor; if there are concerns with patient positioning and seating, referral to an occupational therapist, a seating clinic, or a specialist in physical medicine and rehabilitation should be pursued.[7] If emotional or mental health stressors are thought to trigger spasticity, referral for counseling or pharmacologic treatment would be appropriate. Primary care providers play an important role in facilitating services in their communities, given the challenges of accessing experts (occupational or physiotherapists, seating clinics, or other specialists).

Pharmacotherapy. A combination of nonpharmacologic and pharmacologic treatments is often used to manage spasticity. The most commonly used pharmacologic treatments are presented in **Table 1**,[10,17,19-22] along with their actions, common dosages, side effects, and monitoring details. Although antispasmodic medications have been used for some time, it should be noted that research on their effectiveness is lacking. Baclofen is commonly the first-line medication for spasticity; however, it might be necessary to add or switch to the other medications listed depending on response, tolerance, coverage,

Table 1. Pharmacologic treatment of spasticity

DRUG	DESCRIPTION	DOSAGE	SIDE EFFECTS AND CAUTIONS
Baclofen ($GABA_B$ agonist)	Often first-line medication[10,19]	• Initial dosage: 5 mg 3 times/d • Can increase by 5 mg every 3-5 d • Maximum dosage: 20 mg 4 times/d (up to 80 mg/d)	• Sedation • Lower seizure threshold • Weakness • Dizziness • Ataxia
Tizanidine (α_2-adrenergic agonist)	Most widely used next to baclofen[17] Inhibits excitatory neurotransmitters, demonstrating potent muscle-relaxing properties. Has been shown to reduce muscle tone and frequency of muscle spasms	• Initial dosage: 4 mg/d • Increase by 2-4 mg at a time over 2-4 wk • Maximum dosage: 36 mg/d divided 3-4 times/d	• Sedation • Dizziness • Dry mouth • Hypotension • Hepatotoxicity • Monitor liver function* (ALT and AST levels)[21]
Gabapentin (GABA analogue)	Limited evidence[20] Does not undergo metabolism and is excreted in the urine	• Initial dosage: 400 mg 3 times/d • Maximum dosage: 3600 mg/d	• Risk of respiratory depression • CNS depression • Dizziness
Dantrolene (peripheral calcium inhibitor)	Limited evidence Reduces contraction of skeletal muscle. Most often used in treatment of upper motor neuron lesions, and has been shown to improve functional spasticity	• Initial dosage: 25 mg/d for 7 d • 25 mg 3 times/d for 7 d • 50 mg 3 times/d for 7 d • Maximum dosage: 100 mg 3 or 4 times/d	• Sedation • Weakness • Nausea, diarrhea • Hepatotoxicity • Monitor liver function* (ALT and AST levels)[22]
Diazepam ($GABA_A$ agonist)	Limited evidence Not as effective as baclofen at relieving flexor spasms	• 2-10 mg 3 times/d to 4 times/d	• Most sedation • Withdrawal • Avoid taking with alcohol • Confusion

ALT—alanine aminotransferase, AST—aspartate aminotransferase, CNS—central nervous system, GABA—γ-aminobutyric acid.
*Cases of hepatotoxicity have been reported; avoid prescribing in patients with known liver dysfunction.[21,22] Experts recommend monitoring liver function test results at baseline, at 3 mo, and at dose changes.

or other individual patient characteristics. It is important to be careful of the risks and side effects of individual medications in relation to patient comorbidities and to monitor the patient appropriately. In particular, patients taking tizanidine and dantrolene should be monitored for potential hepatotoxicity.[20] Ongoing reevaluation of effectiveness and tolerance should include physical reexamination and asking patients to report symptoms.

If pharmacologic and nonpharmacologic treatments are unsuccessful at treating an individual's spasticity, referral to a specialist such as a neurologist or physiatrist should be considered. Referral should be considered when spasticity is due to another cause that cannot be resolved (eg, syringomyelia), the patient does not tolerate initial treatments, the patient's symptoms do not respond to initial treatments, or spasticity is severe or causes substantial disability (eg, fracture).

Other treatments. There is little research with regard to cannabinoids in treating spasticity; most studies only recommend trying it after conventional treatments have failed.[19] Botulinum toxin has good evidence for relieving focal spasticity in many conditions; advantages include local effects (although they wear off in 3 to 6 months) and a lack of systemic side effects.[7,11] Botulinum toxin treatment typically requires a referral to a specialist (eg, physiatrist, neurologist). Intrathecal baclofen can be administered by surgically implanted pumps and has been used in many conditions. Indications for intrathecal baclofen include intolerance to oral agents, and refractory or severe spasticity.[8,11,19,20] Many tertiary care centres have specialists who offer this service. Surgical treatments are available for specific cases, including rhizotomy (severing the nerve roots), cordotomy (disabling selected nerve tracts), cordectomy (removing part of the spinal cord), and myelotomy (severing nerve fibres in the spinal cord).[19,20,23]

Case resolution

Jeff has bothersome spasticity that appears to be worsened by triggers (positioning and seating, stopping medication). Through regional home-care services he is referred to an occupational therapist who is knowledgeable about wheelchair seating for a new cushion and wheelchair assessment. He has some insurance coverage for physiotherapy and is able to see a physiotherapist to learn range of motion exercises, which can be continued with his care attendant. As he is interested in taking medication, you prescribe 5 mg of oral baclofen to be taken 3 times daily and schedule follow-up in 2 to 4 weeks for re-assessment of the extent and severity of, and impairment caused by, spasticity, and to assess his tolerance of the medication.

Conclusion

Spasticity is a very common secondary complication of many neuromuscular physical disabilities, and symptoms vary from patient to patient. Primary care providers play an important role in recognizing, assessing, and managing spasticity. Understanding how patients describe and perceive spasticity is important in assessing and managing treatment. Spasticity can be beneficial for activities of daily living; it is important to obtain a thorough clinical assessment to determine when the spasticity is troublesome, whether there is a reversible cause, potential triggers, and what strategies the patient is already using to relieve the symptoms. Management of spasticity should involve a comprehensive approach including the patient, his or her family members, and his or her caregivers; allied health practitioners; PCPs; and specialists. Referral to specialists should be considered in cases of severe spasticity, complications, symptoms refractory to treatment, intolerance of treatment, or focal spasticity that might benefit from local treatment.

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Contributors

All authors contributed to the literature review and interpretation, and to preparing the manuscript for submission.

Competing interests

None declared

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