Managing Movement Disorders: A Clinical Review

Recognizing crucial distinctions between muscle tightness, spasticity, and clonus.

ABSTRACT: Neuromuscular disorders are complex, difficult both to differentiate and to manage. Yet nurses, who encounter a symptomatically diverse neuromuscular patient population in various practice settings, are expected to be well versed in managing the variable associated symptoms of these disorders. Here the authors discuss how to assess such neuromuscular conditions as muscle tightness, spasticity, and clonus; the pathophysiology underlying each; and the available recommended treatments, an understanding of which is necessary for successful symptom management and clear provider–patient communication.

Keywords: clonus, movement disorders, muscle tightness, neuromuscular disorders, spasticity

Imagine a patient with a history of stroke that has affected her right side, particularly her right arm, hip, leg, and foot. She has both spasticity and clonus, which she manages by using an intrathecal baclofen pump—she finds the sedative effects of oral baclofen intolerable. She likes to stay active and performs regular stretches at home. (This case is a composite based on our experience.)

One day, the patient calls her clinic to report that she is experiencing muscle tightness in her left arm. The nurse relays the information to the physician, who orders an increase in the intrathecal baclofen dosage. A nurse visits the patient at home to increase her dosage in accordance with the revised order. Within an hour, the patient is confused and lethargic. She is admitted to the hospital. What went wrong?

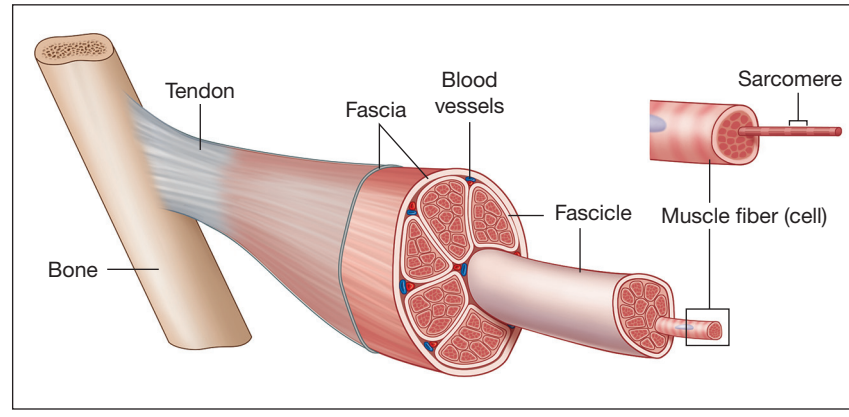
It's important for clinicians to bear in mind that spasticity, clonus, and muscle tightness are three distinct conditions. While spasticity and clonus are neurogenic in nature, muscle tightness is myogenic. The effective management of these movement disorders depends in large part on the clear description of the patient's signs and symptoms.

In this scenario, the nurse should not have assumed that, simply because the patient uses an intrathecal baclofen pump, a change in her symptoms was due to spasticity. A thorough triage would have revealed that, though the patient's spasticity affects her right side, she reported left-sided muscle tightness, which could have been managed conservatively with heat, ice, and additional short-term oral medication. Instead, she received an increased dosage of intrathecal baclofen, which resulted in an overdose, as evidenced by her confusion, drowsiness, and lethargy.

Neuromuscular signs and symptoms occur across a multitude of diseases and injuries. For example, muscle tightness may be experienced after a fall, motor vehicle accident, or sprain. Successful symptom management requires effective communication about symptoms, yet there are often discrepancies between patient and provider descriptions of neuromuscular symptoms and manifestations.[1] Further complicating matters, neuromuscular signs and symptoms can exist as single entities or in clusters. For example, a person with muscle tightness may or may not experience pain and muscle weakness; clonus may or may not coexist

with spasticity. Symptom differentiation and discrimination based on appropriate assessment and knowledge of neuromuscular pathophysiology is the first step in initiating a nursing care plan for a patient with a movement disorder. Since nurses working in diverse practice settings are likely to encounter such patients, this article discusses the pathophysiology and assessment of three different, common neuromuscular disorders—muscle tightness, spasticity, and clonus—as well as the treatment options for each.

Figure 1. Anatomy of a Muscle



With every muscle contraction sarcomeres shorten, causing the muscle fibers and ultimately the entire muscle itself to shorten. The forces generated by this contraction are transmitted to the connective tissue (fascia) that surrounds the contracting muscle fibers, resulting in movement. Illustrations by Sara Jarret.

MUSCLE TIGHTNESS

Muscle tightness may occur after any acute injury that strains or tears the soft tissue. The patient often experiences swelling, bruising, weakness, and the inability to fully stretch the muscle. Common acute injury sites are the back, hamstring, quadriceps, and gastrocnemius. Muscle tightness may also result from sustained postures. For example, "text neck" refers to a tightness of neck muscles from prolonged texting on a cell phone.[2,3] If not properly assessed and treated, muscle strain may lead to chronic muscle tightness. Currently, there is no consensus definition of muscle tightness, though the National Library of Medicine categorizes it as a form of muscle tonus.[4]

Pathophysiology. Skeletal muscles consist of long multinucleated cells called muscle fibers, each of which can extend the entire length of the muscle. Fascicles are bundles of muscle fibers wrapped in connective tissue. Skeletal muscles are composed of several fascicles wrapped in a connective tissue sheath called fascia. The connective tissue surrounding the entire muscle is continuous with the tendons that anchor the muscle to the skeleton (see Figure 1). The tendon attachment that remains fixed in place is called the origin; the tendon attachment that moves with muscle contraction is called the insertion.

On a cellular level, skeletal muscle fibers are composed of the filamentous proteins actin and myosin, which are interspersed in orderly, repeated units called sarcomeres. With a muscle contraction, the shortening of many sarcomeres in a series allows the muscle fibers and ultimately the entire muscle within its layers of connective tissue to shorten. The forces generated by the sarcomeres during muscle contraction are transmitted to the connective tissue around the contracting fibers by protein complexes known as costameres.

Isotonic contractions change the length of skeletal muscles, enabling people to move objects. Isotonic contractions that shorten the muscle are called concentric; those that lengthen the muscle are called eccentric. Concentric contractions allow people to lift objects and eccentric contractions allow people to set them down.

Eccentric contractions commonly lead to skeletal muscle injuries. Such injuries, commonly called strains, can lead to muscle tightness. First, a hematoma forms at the site of the tear. Immune cells then infiltrate to begin the repair process, causing swelling and inflammation but also removing the cellular debris so the injury can be repaired. Satellite cells, which are muscle-specific stem cells, proliferate, fuse into myotubes, and fill in the tear. Whether healing is successful depends on the severity of the strain. If many fibers and supporting connective tissues are damaged, the injury may result in prolonged pain, atrophy, muscle weakness, loss of flexibility, and disorganized replacement of the connective tissue.

Isometric contractions produce tension without causing the muscle to change in length, as when a person tries to move an item that's too heavy to lift. A movement may have components of both isometric and isotonic contractions, depending on what the movement requires of the muscles involved.

Assessment. There is no standardized way to assess muscle tightness. Evidence of muscle tightness is both objective and subjective. The most common objective means of assessment is to measure the patient's active and passive range of motion with a goniometer, a protractor-like instrument that clinicians

can use to quantify, in degrees, joint angle and range of motion in a patient's limbs. However, to assess muscle tightness in other parts of the body, such as the abdomen, clinicians must rely on palpation, patient history, and patient reports of pain. A patient's subjective report is crucial in assessing muscle tightness. If a patient reports that muscle tightness is increasing, that self-report should be accepted and the tightness treated accordingly. Numeric rating scales, which are commonly used to assess many conditions, may also be used to assess muscle tightness.

Treatment. Muscle tightness is often initially treated with ice or heat. After an acute injury, it's generally recommended that ice be used for the first 24 hours and heat later. The frequency of ice application varies with patient tolerance but applying ice in 15-to-20-minute increments is often helpful. Heat applied in 10-to-15-minute increments often relieves chronic muscle tightness, though no specific frequency is recommended. When applying heat, it's important to ensure that patients' sensation is intact and that the heat source is not too hot.

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Muscle tightness can also be managed with a centrally acting muscle relaxant, such as cyclobenzaprine (Amrix) or methocarbamol (Robaxin), though these drugs are not recommended for older adults, given their anticholinergic activity and cyclobenzaprine's long half-life.[5] Since muscle tightness can entrap local nerve endings, it may produce pain and stinging sensations. If muscle tightness becomes chronic, it may require physical or occupational therapy, and severe chronic cases may produce contractures, requiring tendon-lengthening surgery.

SPASTICITY

Spasticity, which manifests as neurogenic muscle tightness, may occur as a sequela of such neuromuscular disorders as spinal cord injury, multiple sclerosis, stroke, or traumatic brain injury, with reported prevalence and annual incidence varying widely. According to a systematic review by Martin and colleagues, the prevalence of lower limb spasticity per 100,000 is 2 to 350 in multiple sclerosis, 240 to 360 in cerebral palsy, 22 to 90 in spinal cord injury, and 40 to 600 in stroke; the annual incidence of lower limb spasticity per 100,000 is 100 to 235 in traumatic brain injury, 30 to 485 in stroke, and 0.2 to 8 in spinal cord injury.[6]

Although researchers agree that spasticity is an upper motor neuron syndrome, there is no consensus on its definition. In 1980, Lance defined spasticity as a component of the upper motor neuron syndrome that results from "hyperexcitability of the stretch reflex" and is characterized by a "velocity-dependent increase in tonic stretch reflexes."[7] Subsequent researchers have described spasticity similarly.[8-11]

However, in 2005, the Support Programme for Assembly of a Database for Spasticity Measurement (SPASM) proposed the following as a new definition for spasticity: "disordered sensory-motor control, resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary activation of muscles."[12] By identifying the roles played by both the nervous and musculoskeletal systems in producing spasticity, this newer definition is broader in that it recognizes the role of sensory involvement in spasticity.

Upper motor neuron pathway disorders may produce pain, insomnia, and pressure injuries, reducing quality of life and increasing caregiver burden.[13] In addition, they can prevent the transmission of information essential for such vital functions as walking, breathing, and swallowing. Some degree of spasticity, however, may benefit certain patients, providing tone for weak muscles and thereby aiding in ambulation and transfer.

Pathophysiology. Spasticity stems from a disruption of upper motor neuronal pathways that normally send messages from the brain to the spinal cord to execute muscle functions. Impairment of the descending inhibitory pathways of the corticospinal tract results in increased excitability of the α motor neurons, which innervate skeletal muscles (see Figure 2). The increased α motor neuronal excitability, in turn, causes muscle tightness and contracture, particularly in the flexor muscles.[14]

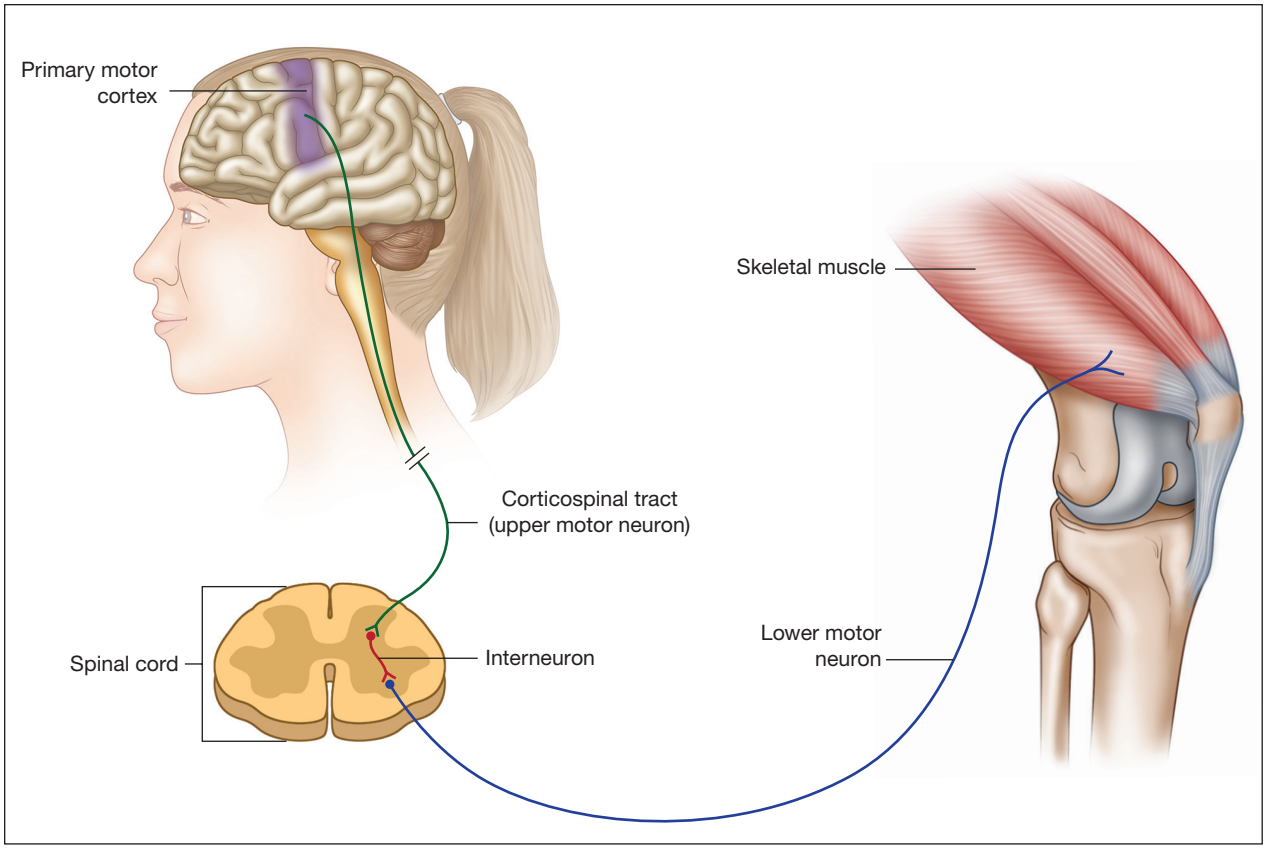
Assessment. Symptoms of spasticity may be intermittent or sustained. Urinary tract or other infections, renal calculi, pressure injuries, constipation, poorly fitting orthotics, and other maladies may act as noxious

stimuli and exacerbate spasticity.[15] This may be because the same changes that cause an enhanced response to the stretch reflex in patients with upper motor neuron damage also increase sensitivity of the spinal cord to sensory input from painful stimuli. Although patients' experience of spasticity is subjective, symptoms can be assessed using the Ashworth or Modified Ashworth scale, which relies on examiner observation.[16] The use of these scales requires assessment expertise and close attention paid to changes in patient responsiveness over time and with intervention.

The Ashworth scale is a five-level (0 to 4) ordinal scoring scale developed in 1964 by Bryan Ashworth to evaluate the clinical efficacy of the antispasmodic medication carisoprodol in patients with multiple sclerosis.[17] Scores are assigned based on the resistance or "catch" of patients' limbs as clinicians guide the patients through a passive range of motion exercises. In 1987, to enhance the scale's sensitivity, Bohannon and Smith added a level (1+) to the scale between the scores 1 and 2, creating the Modified Ashworth scale.[18] In addition to these scales, clinicians consider patients' rating of their spasticity on a 0-to-10 numeric rating scale, in which 0 signifies no spasticity and 10 the worst possible spasticity.[18] See Table 1[17-20] for a description of scales used in evaluating spasticity, clonus, and muscle tightness.

Treatment. Spasticity is managed through various treatments based on specific patient needs. Centrally acting antispasmodic medications, such as baclofen and tizanidine (Zanaflex), are commonly used. Adverse effects of these medications include sedation, dizziness, gastrointestinal upset, hypotension, headache, convulsions, agitation, leukocytosis, chills, urinary retention or frequency, hypotonia, and dry mouth. Patients who cannot tolerate adverse effects of large doses may require an intrathecal baclofen pump, which enables therapeutic management at lower doses with fewer adverse effects. Good candidates for this treatment are patients who have lower limb spasticity and are unable to tolerate oral baclofen or require higher doses to manage spasticity.[21] Patients

Figure 2. The Upper Motor Neuronal Pathway



An upper motor neuron descends through the corticospinal tract and synapses in the gray matter of the spinal cord to a lower motor neuron, which innervates skeletal muscle. If present, the interneuron at the synapse can be excitatory or inhibitory. If the upper motor neuron is damaged, it can be either difficult to inhibit the muscle, resulting in spasticity, or difficult to excite the muscle, resulting in muscle weakness.

Table 1. Assessment Scales for Spasticity, Clonus, and Muscle Tightness

Scale	Technique	Rater(s)	Scoring	
Ashworth[17]	Resistance or catch when "quick" PROM is performed	Expert assessors	0	No increase
			1	Slight increase in tone, giving a "catch" when limb is moved in flexion or extension
			2	More marked increase in tone but limb is easily flexed
			3	Considerable increase in tone—passive movement is difficult
			4	Limb rigid in flexion or extension
Modified Ashworth[18]	Resistance or catch when "quick" PROM is performed	Expert assessors	0	No increase
			1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the ROM when limb 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 limb is easily moved
			3	Considerable increase in muscle tone, passive movement is difficult
			4	Limb is rigid in flexion or extension
Numeric Rating Scale[19,a]	Patient self-report of spasticity	Patient(s)	0 10	No spasticity Worst possible spasticity
Spasm Frequency Score[20]	Frequency of clonus elicited with PROM or activity	Any clinician	0	No spasms
			1	Mild spasms induced by stimulation
			2	Infrequent full spasms occurring less than once per hour
			3	Spasms occurring more than once per hour
			4	Spasms occurring more than 10 times per hour

PROM = passive range of motion; ROM = range of motion.
[a] Numeric rating scales can also be used to assess muscle tightness.

who experience focal spasticity, however, can often manage symptoms through botulinum toxin injections in upper or lower extremities. Botulinum toxin is a neuromuscular blocker and a muscle relaxant. Another treatment option is the injection of the chemical neurolytic phenol directly into the peripheral nerves to block the stimulus that activates overactive muscles in spasticity. In some severe cases, selective dorsal rhizotomy surgery, in which spasticity-causing nerve roots are isolated and destroyed, is performed.

Physical and occupational therapies are management strategies commonly used by patients with spasticity, who often struggle with mobility and activities of daily living. Nursing modalities for patients with spasticity include heat, stretching, massage, and aromatherapy. Some research indicates that ice may be helpful as well.[22] Ice, however, is not commonly used in clinical practice. Some patients have reported that alternative therapies, such as acupuncture, are beneficial in treating spasticity.[23, 24]

CLONUS

Clonus is a neurologic symptom that manifests as an abnormal contracting and relaxing of the affected body part. Patients often experience clonus, spasticity, and neuropathic pain concurrently. Discrimination among these various symptoms may be difficult.[25, 26] Clonus, however, has a distinct pattern in which the first oscillation (contraction and relaxation cycle) is longer than subsequent oscillations.[27]

Pathophysiology. Clonus is an involuntary, rhythmic response to stretch reflex stimulus. Though clonus is more often induced in the ankles and feet, it can occur at the patella, triceps surface, wrist, jaw, and biceps brachii. For example, when the ankle is dorsiflexed, muscle spindles are stretched in the gastrocnemius and soleus muscles. Depolarization of the spindles

generates action potentials, which are transmitted to the spinal cord by sensory neurons called Ia afferents. Ia afferents synapse on the α motor neurons to the calf, leading to increased activation of the gastrocnemius and soleus.[25] The foot vigorously points downward (plantar flexion) and then releases back into dorsiflexion, such that the gastrocnemius and soleus are stretched repeatedly as oscillating contractions move the foot from dorsiflexion to plantar flexion (see Figure 3). The first oscillation is always the longest, with subsequent oscillations becoming shorter and eventually stabilizing into a rhythmic pattern by the fourth or fifth repetition until the oscillations stop.[27] As with spasticity, clonus results from increased excitability of the α motor neurons, brought on by impaired descending inhibitory pathways. Highly activated γ motor neurons increase the sensitivity of muscle spindles, making the oscillating circuit possible.[28]

Assessment. Clonic oscillations are episodic. The frequency of oscillations can be visually assessed using the Spasm Frequency Score proposed by Penn and colleagues in 1989 as a means of reporting the effects of intrathecal baclofen for spinal spasticity.[20] The Spasm Frequency Score can be used by both clinicians and patients to report the frequency (the number per hour) of clonic oscillations, which can increase if coexisting spasticity worsens.

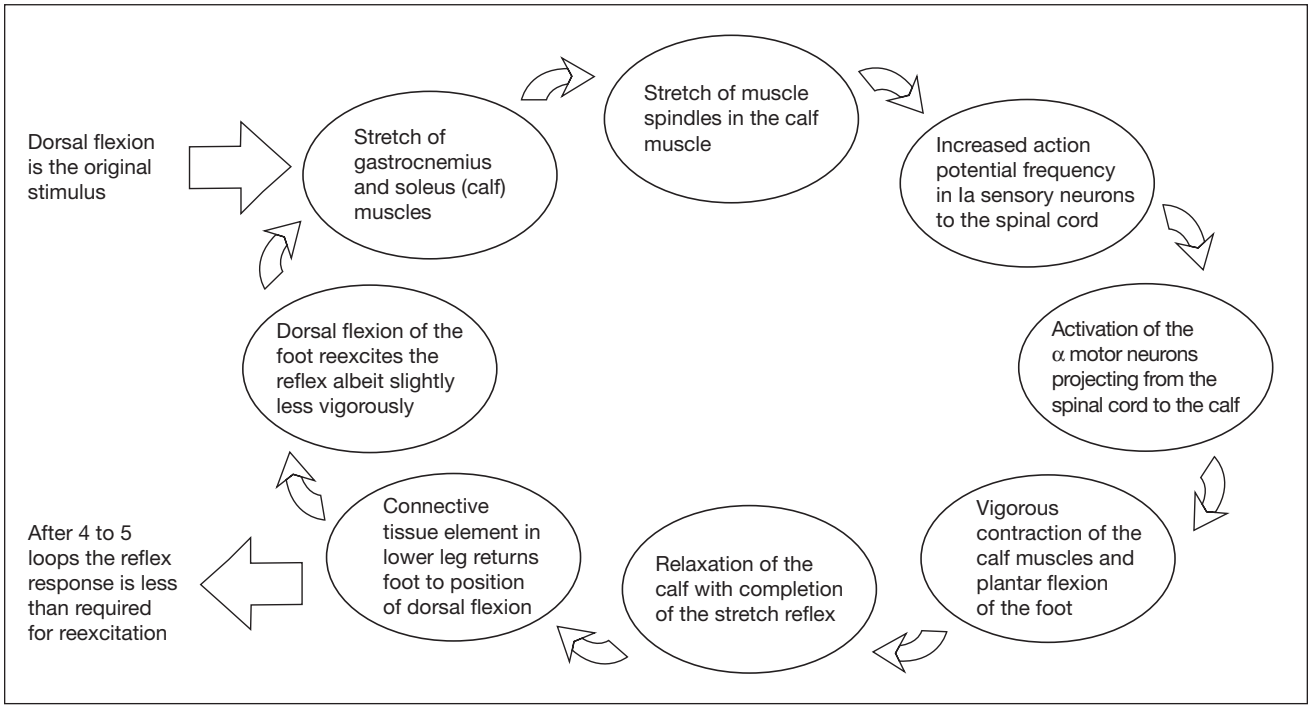
Treatment. Clonus is managed medically with such oral medications as tizanidine, baclofen, diazepam (Valium), clonazepam (Klonopin), and dantrolene (Dantrium). Diazepam and clonazepam are schedule IV anxiolytics, which can depress the central nervous system. Some researchers have reported that botulinum toxin and phenol injections may be beneficial in treating clonus.[29, 30] The application of cold packs[27, 31] and manual pressure[32, 33] are common nursing treatments for clonus.

DIFFERENTIATING MOVEMENT DISORDERS

Spasticity, clonus, and muscle tightness may appear to be similar, but there are important differences between them. Spasticity and clonus are neurogenic conditions resulting from a disruption in upper motor neuronal pathways, while muscle tightness is an orthopedic condition that results from traumatic injury. While spasticity and clonus arise with neurologic injury, they are not progressive in nature. They may, however, be exacerbated by noxious stimuli or comorbid conditions. Muscle tightness, on the other hand, can progress if not managed appropriately in the initial phase of injury.[34]

There are differences and commonalities in the management of these conditions. All three can be managed through oral medications such as baclofen and tizanidine; however, use of botulinum toxin or the intrathecal baclofen pump is reserved for spasticity and in some cases clonus. Ice packs can be used to manage clonus and muscle tightness but is not preferred for spasticity. Similarly, heat may help reduce

Figure 3. The Feedback Loop Demonstrated in Clonus of the Foot



spasticity and muscle tightness but not clonus. Manual pressure may help temporarily stop clonus, while stretching, repositioning, and massage may alleviate spasticity and muscle tightness.

Nurses working in a variety of settings are likely to encounter patients with symptoms of spasticity, clonus, and muscle tightness. To effectively manage these frequently overlapping disorders, nurses must understand the underlying pathophysiology of each. Future research should focus on identifying continuing education strategies that allow nurses to learn about these movement disorders and translate their knowledge into bedside evidence-based practice. ▼

To see a short video on spasticity, go to www.youtube.com/watch?v=TFmSM7SoXDg. To see examples of clonus and how to test for it, go to www.youtube.com/watch?v=MUOwuxiI5Gw and www.youtube.com/watch?v=UX75k8s5QUE.

For six additional continuing nursing activities related to movement disorders, go to www.nursingcenter.com/ce.

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