Spasticity is a neuromuscular dysfunction characterized by tight or stiff muscles. Spasticity occurs across the spectrum of upper motor neuron disease and complicates the course and quality of life of those affected. Accurate and precise assessment of spasticity is the first step in providing safe and effective treatments to patients for management of spasticity. Examiner evaluations (Ashworth Scale, Modified Ashworth, and Visual Analog Scale) and patient self-reports (Visual Analog Scale and Numeric Rating Scale) are used to assess spasticity in clinical practice. We reviewed the biology of spasticity and summarized research that assessed properties of scores obtained from clinical scales when used in a variety of upper motor neuron diseases. The definition of spasticity was inconsistent. Rater reliability or agreement on clinical scales varied widely. Correspondence with electromyogram results was mixed. There was dissimilarity in patient reports and examiner assessments. Scores from clinical scales are responsive (decrease after initiation of treatment with known effectiveness), but the utility of scores for indexing individual change associated with the natural history of upper motor neuron disease is unknown. Future research incorporating patient reports and examiner findings over time will help to clarify the definition and capture the essence of spasticity.

Upper motor neuron (UMN) lesions are central nervous system impairments that diminish motor control and create emergence of pathological signs such as spasticity, rigidity, clonus, and hyperreflexia (Leonard, Gardipee, Knootz, Anderson, & Wilkins, 2006). Spasticity, characterized by tight or stiff muscles, occurs in UMN diseases such as spinal cord injury, multiple sclerosis, stroke, and traumatic brain injury (Pettibone, 1988). Spasticity contributes to pain, insomnia, and fatigue and can interfere with mobility, transfers, self-care, activities of daily living, and social functioning (Bhimani, 2008). Spasticity increases caregivers’ burden. A person experiencing spasticity requires passive range of motion to the limbs upon awakening and at bedtime so that personal care activities such as toileting and dressing can be carried out with some ease. Untreated spasticity can lead to permanent muscle contractures (Ashworth, Satkunam, & Deforge, 2006).

Clinical factors such as urinary tract infections and decubitus ulcers sometimes increase spasticity (Nuyens et al., 1994; Sköld, 2000). Spasticity varies in dynamic (moving) and static (sustained contraction) states (Leonard et al., 2006; Pandyan et al., 2005). Repeated stretching and posture changes may also affect spasticity (Bakheit, Maynard, Curnow, Hudson, & Kodapala, 2003; Sköld, Levi, & Seiger, 1999; Wood et al., 2005).

Measurement of spasticity is an important part of patient care. Members of the clinical team quantify spasticity with a variety of standardized approaches when they assess patient status, select interventions, and evaluate intervention effectiveness over time. Obtaining accurate, precise measurements is a challenge because the nature of spasticity is elusive and assessment is subjective even when evaluated using established clinical protocols.

This article reviews the biology of spasticity and evaluates approaches to measurement of spasticity used in every day clinical assessment. Specific aims are (a) to review anatomy and pathophysiology of spasticity and link them to electromyogram (EMG) findings as a foundation for evaluating clinical measurement issues, (b) to identify scales used to measure spasticity in clinical practice and research, (c) to summarize information about measurement properties of scores obtained from examiner and self-report scales in clinical populations, and (d) to judge the state of the science of spasticity measurement and comment on implications for practice and future research.
The Biology of Spasticity
Definitions
The definition of spasticity is evolving. There is agreement that it is characterized by increased stiffness in skeletal muscles. In a narrow sense, spasticity is observed as an increased velocity-dependent response in the tonic stretch reflex (Lance, 1980). The SPASM project (Burridge et al., 2005) proposed a broader definition. This new definition adds effects of abnormal sensory input to motor control that result in hyperreflexivity, increased tone over extended periods of rest, or disordered motion occurring during activities such as walking. Other abnormalities of neuromuscular functioning (e.g., clonus, ataxia, akathisia, athetosis, hypertonia, rigidity, restless leg syndrome) may occur with spasticity but are distinct from it (see Table 1 for definitions and Web links for video demonstration; Larsen & Stensaas, 2009; We Move, 2009).

Anatomy and Physiology
Knowledge about the relevant anatomy and physiology provides the foundation for theory that guides measurement of spasticity in research and clinical practice (Levin, 2005). The stiff, tight, overstimulated muscles and disordered sensory-motor control in patients with spasticity result from disinhibition of the tonic stretch reflex by impaired function of UMN s (Burridge et al., 2005, p. 72; Lance, 1980, p. 485). The interconnected structures and dynamic functioning of the nerves and muscles are all involved in production and maintenance of spasticity. A discussion of muscle anatomy in normal sensory-motor physiology is essential.

| Table 1. Terms Describing Neuromuscular Anatomy, Physiology, and Dysfunction |
|-----------------------------|-------------------------------|
| **Term**                      | **Definition**                                                                 |
| Action potential             | A large depolarization conducted along the membrane of muscle fiber or nerve axon<sup>a</sup> |
| Akathisia                    | Subjective feeling of unease and inability to remain still; compulsion to move with little relief in the absence of anxiety; objectively seen as restlessness and inability to sit<sup>b</sup> |
| Ataxia                       | Gross incoordination of muscle movement when standing and walking<sup>c</sup> |
| Athetosis                    | Complex worm-like irregular nonpurposeful movements that involve limbs and face<sup>c</sup> |
| Clonus                       | One form of phasic stretch where muscles rapidly contract and relax<sup>c</sup> |
| Contractures                 | Tendon and soft tissue of limbs in a fixed position due to increased muscle tone leading to muscle shortening<sup>c</sup> |
| Extrafusal fiber             | Part of muscle fiber that is innervated by alpha motor neurons; they aid in muscle contraction<sup>a</sup> |
| Hypertonia                   | Simultaneous co-contraction of agonists and antagonists muscles where non-velocity-dependent resistance to passive movement experienced by the examiner as increased muscle tone and rigidity<sup>c</sup> |
| Intrafusal fiber             | Type of muscle fiber that is innervated by gamma motor neurons and sensory Ia neurons; they aid in sensory proprioception<sup>a</sup> |
| Load                         | An applied force or effort to move a resistance<sup>a</sup> |
| Muscle spindle               | Encapsulated receptor in a skeletal muscle cell that is sensitive to muscle stretch<sup>a</sup> |
| Muscle stiffness             | Condition where muscles feel tight and are not in relaxed or normal supple state<sup>c</sup> |
| Proprioceptors               | Receptors that provide information about posture, muscle tone and movement usually found in muscles, joints, and tendon<sup>a</sup> |
| Restless leg syndrome        | A sensation to move limbs to reduce restlessness sensation when at rest<sup>b</sup> |
| Rigidity                     | Abnormal muscle stiffness and resistance to movement<sup>c</sup> |
| Spasm                        | Hyperexcitability of muscles induced by stimulation<sup>c</sup> |
| Stretch reflex               | Predictable, rapid motor response initiated by a muscle spindle when muscles are stretched |
| Stretch velocity             | Direction and speed of the muscle stretch when an external stimulus is applied<sup>a</sup> |

<sup>Note</sup>.  <sup>a</sup>Widmaier et al. (2007).  <sup>b</sup>Allen and Earley (2001).  <sup>c</sup>We Move (2009).
**Motor System**

A motor system consists of higher centers (motor cortex), middle centers (brain stem), and lower centers (spinal cord). Components of a motor system activate muscles; they also receive sensory information about muscle length, muscle tension, and joint position to activate muscles with the intensity, sequence, and timing needed for smooth execution of a particular movement.

The muscles are contractile in nature and are joined together by connective tissue known as fascicle to provide shape. These muscle structures are made up of muscle fibers that contract whole muscles. The muscle fibers are innervated via nerve receptors such as muscle spindles, which control proprioceptors (body position sensors; see Figure 1). As the muscle is stretched (e.g., by a tendon tap or in the course of daily activities), the muscle spindle is also stretched, generating a faster frequency of action potentials in the sensory neuron (see Figure 2a). As the muscle contracts, the stretch on the spindle are relieved, decreasing the frequency of action potentials in the sensory neuron (see Figure 2b). Muscle spindles not only participate in reflexes but also provide essential dynamic sensory information about the length and tension of muscles during as voluntary movement occurs (see Figure 3). For example, as a person performs a motor task like reaching for an elevator button, the motor system requires information about the starting condition of the muscles before a trajectory of movement can be planned and executed. In this way, information about body position (proprioception) is supplied by muscle spindles (Nielsen, Crone, & Hultborn, 2007; Widmaier, Raff, & Strang, 2007).

**Muscle Fibers**

A skeletal muscle consists of two kinds of muscle fibers. First, the bulk of the muscle is made of extrafusal fibers (synonymous with skeletal muscle fiber), which are innervated by alpha motor neurons; when extrafusal fibers contract, the muscle shortens. The second type of muscle fiber, intrafusal fibers, functions within muscle spindles and is innervated by gamma motor neurons and sensory Ia neurons (see Figure 1). When intrafusal fibers contract, there is no direct contribution to muscle tension or muscle shortening. Instead, activation of gamma motor neurons and contraction of intrafusal fibers exert stretch on muscle spindles making them more sensitive to length changes in the muscle. This unique receptor system permits the brain to control the length information received from the muscle spindle (see Figure 2; Widmaier et al., 2007).

**Motor Neurons**

On the basis of the location of motor neurons in the body, a neuron is either a UMN or a lower motor neuron. A UMN arises in the motor cortex of the brain and extends to the spinal cord. An LMN extends from the spinal cord to the muscle that it innervates. The

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**FIGURE 1 Overview of Stretch Reflex Pathways and the Influence of Higher Motor Centers**

![Image](http://thebrain.mcgill.ca/flash/i/i_06/i_06_cl/i_06_cl_mou/i_06_cl_mou.htm)

Note. Figure modified from freely available content (copyleft) at: http://thebrain.mcgill.ca/flash/i/i_06/i_06_cl/i_06_cl_mou/i_06_cl_mou.html.
UMNs project to and synapse on lower motor neurons in the spinal cord.

An LMN also receives sensory information about muscle position and stretch from the muscle spindles. Thus, the lower motor neurons are the “final common pathway” to coordinate voluntary movement because the lower motor neurons receive information from both UMNs and sensory neurons from the periphery. The lower motor neurons integrate sensory and motor information for the activation of muscles during both reflexive and voluntary movement (Widmaier et al., 2007).

The Stretch Reflex

The physiologic role of the stretch reflex is to help a muscle maintain a steady state. If a person picks up a weight with his or her hand, the load pulls on the biceps muscle. The stretch depolarizes muscle spindles which send action potentials to the spinal cord via sensory neurons. The sensory neurons synapse on the alpha motor neurons within the spinal cord that innervate the muscle that was stretched. The biceps contracts and maintains the load. This is the monosynaptic stretch reflex. Concurrently, the triceps, an antagonist muscle to the biceps, is inhibited by interneurons activated by the same reflex and the triceps muscle relaxes. Inhibition of the triceps eliminates opposition to contraction of the biceps and the load is maintained (Widmaier et al., 2007).

UMN Lesion Effects

An UMN lesion is a disease or injury that disrupts the anatomical integrity and/or physiological functioning of the UMNs. Multiple sclerosis, spinal cord injury, and stroke are examples of UMN lesions. UMN disease, that produces spasticity, affects both reflexive and voluntary motor function.

Reflexive Function and Spasticity

UMN lesions alter the activation of the LMN, producing a state of net disinhibition of spinal reflexes. In spasticity, the negative feedback system between muscle spindles and alpha motor neurons is disrupted because of the UMN lesion, and the abnormal response of tight muscles is obtained. For example, the UMN lesions decrease the inhibitory drive in the corticospinal tract, affecting alpha-motor neuron excitability and causing increased muscle contraction. Also, disruption of inhibition of the antagonist muscle or increased action potentials in the sensory neurons from the muscle spindle can lead to muscle tightness (Nielsen et al., 2007).

Voluntary Movement and Spasticity

For voluntary movement, impulses are generated in the brain and relayed from UMN to lower motor neuron. This impulse coactivates gamma and alpha motor units. Contraction of muscle relieves the stretch on the muscle spindle such that the unloaded muscle spindle
and its sensory neuron stop sending action potentials (information indicating muscle length) to the central nervous system. By coactivating the alpha and the gamma motor neurons, the muscle spindles continue to provide information about length to the central nervous system throughout the entire range of motion (see Figure 3).

Gamma motor neurons can also be activated before alpha motor neurons. Gamma activation exerts a stretch on the muscle spindle. Sensory neurons from the muscle spindle synapse on alpha motor neurons in the spinal cord and activate them. Thus, gamma activation and contraction of intrafusal fibers can drive alpha activation and the contraction of entire muscle (Nielsen et al., 2007).

As in reflex responses, UMN lesions decrease the inhibitory drive in the corticospinal tract to produce spasticity during voluntary movement and create abnormalities of posture and tone associated with spasticity. The consequences of spasticity during voluntary movement depend on the severity and source of the UMN syndrome. Patients may experience muscle weakness and lose dexterity; weakness of an agonist muscle and excessive coactivation of an antagonist muscle may reduce range of motion or slow the speed at which a patient can complete a task (Bhakta, Cozens, Chamberlain, & Bamford, 2001; Leonard et al., 2006). Spasticity during voluntary movement is generated through local activation of muscle spindles, but the propagation and manifestation of spasticity requires involvement of the central nervous system (Nielsen et al., 2007; Pandyan et al., 2005).

**The EMG**

Electromyography is an electrical summary of neuromuscular functioning. Spasticity generates characteristic EMG tracings, so it can serve as a criterion for assessing decisions about spasticity made on the basis of clinical examination or self-report.

EMG is a common neurophysiologic measure that uses surface electrodes to monitor electrical activity generated from skeletal muscle. Surface measuring electrodes are placed on the skin over the muscles of interest. EMG activity can then be recorded in
response to mechanical or electrical stimuli. For example, reflex EMG activity can be measured in response to a tendon tap. The time from stimulus to EMG response is the latency, and the size of the EMG response is the amplitude. Patients with spasticity exhibit shorter latency periods and greater amplitudes as compared to healthy subjects (see Figure 4).

A passive stretch can also elicit contraction and EMG activity, albeit the velocity of the stretch stimulus is critical for detection of spasticity (Voerman, Gregoric, & Hermens, 2005). Surface electrodes monitor the EMG response as a clinician or researcher quickly stretches the patient’s muscle. Healthy control subjects demonstrate little or no EMG response regardless of the stretch stimulus velocity, whereas patients with spasticity do exhibit EMG activity. The amplitude of the EMG responses increases in amplitude with the increased velocity of the stretch (Sorionola, White, Rushton, & Newham, 2009).

The Hoffman reflex (H-reflex) uses electrical stimulation of a peripheral nerve to elicit EMG activity. A low-intensity current from the stimulating electrode to the mixed peripheral nerve brings sensory neurons to threshold, such that action potentials are propagated toward the spinal cord. As described earlier in the stretch reflex, these sensory neurons synapse on and activate alpha motor neurons that innervate skeletal muscle. Action potentials in alpha motor neurons activate the muscle and excitation of the muscle can be detected in the form of an H-reflex wave on the EMG tracing. A high-intensity current stimulus directly brings alpha motor neurons to threshold, and action potential is propagated toward the muscle; muscle activation subsequent to alpha motor neuron stimulation is detected as an M-wave. As with a tendon tap or a stretch stimulus, the latency and the amplitude H and M waves are recorded. The M-wave typically has a larger amplitude and shorter latency than does the H-reflex wave (Voerman et al., 2005).

The H-reflex is a variable measure because the limb and head position of the patient, any other sensory input experience by the patient (e.g., visual stimulation) and stimulus duration, and frequency can all influence EMG activity. However, the H-reflex is a helpful tool for detecting increases in neuron excitability and decreases in higher center inhibition in patients with spasticity (Burridge et al., 2005; Voerman et al., 2005). For example, the increased excitability in patients with spasticity is shown as decreased latency and increased amplitude in H-reflex waves as compared with those elicited in healthy control subjects. Another EMG measure of motor neuron excitability is the $H_{\text{max}}/M_{\text{max}}$ ratio. This ratio also increases in patients with spasticity and has the advantage of less variability and greater reproducibility over time in people with UMN disease.

**Clinical Scales for Spasticity Measurement**

Table 2 lists clinical scales for measurement of spasticity that are easily administered by an examiner at the bedside or by patients themselves. Examiners use a prescribed maneuver to elicit limb spasticity and then grade severity based on response to the examination stimulus. Self-reports of spasticity require that patients discriminate among various manifestations of their neurological dysfunction, identify spasticity, and rate severity using a scale with standardized anchors.

Detailed assessment technique, rater, and scoring criteria are described for three examiner scales and two self-report scales used with adults.

The Ashworth Scale (AS) was developed more than 45 years ago to evaluate the clinical efficacy of the antispasticity medication carisoprodol in multiple sclerosis patients (Ashworth, 1964). The AS is a clinician’s subjective interpretation of the resistance or catch given by limbs when a quick stretch is applied during passive range of motion. During the examination, patients are asked not to assist with any voluntary movement of the limbs. A grade (score) is assigned by the examiner on the basis of felt resistance to passive movement. The AS is an ordinal scale with five levels, ranging from 0 (no increase in tone) to 4 (affected part rigid in flexion or extension). It is assumed that resistance to passive movement is exclusively due to spasticity (Pandyan et al., 1999).

The Modified Ashworth Scale (MAS) includes an additional grade termed 1+ (Bohannon & Smith, 1987), intended as a mid-classification between a slight increase in tone and a marked increase in tone. The purpose of this addition was to enhance precision in the clinical measurement of spasticity at lower
levels. The assessment technique is the same as the AS, but the additional scoring requires that examiners possess greater awareness of nuances in patient response to quick stretch during passive movement.

The Modified Modified Ashworth Scale (MMAS) deleted the additional grade of 1+ between 1 and 2 from MAS and changed evaluation criteria (Ansari et al., 2006) to enhance the validity and reliability of the MAS. Like the original AS, the MMAS has five categories scored 0–4. The scoring criteria for categories 1 and 2 reflect slight or marked increase in tone in the AS and MMAS, but the MMAS includes additional stipulations about catch and resistance, which is similar to the MAS.

A Visual Analog Scale (VAS) is used by patients for self-report of perceived spasticity. A 100-mm straight line marked at 0 (no spasticity) and 100 (worst spasticity imaginable) is used. Patients are asked to indicate their level of spasticity by identifying a point on that straight line that corresponds to their momentary current experience of spasticity.

The Numeric Rating Scale (NRS) has also been used to report patient perception of spasticity (Farrar, Troxel, Stott, Duncombe, & Jensen, 2008). Commonly, an NRS of 0 (no spasticity) and 10 (worst possible spasticity) is used, and patients are asked to indicate their level of spasticity by identifying a whole number between those two anchors.
Reliability, Validity, and Responsiveness to Change

Measurement in clinical practice is the systematic process of assigning numbers to represent characteristics of people related to their health–illness status. Numbers are arrived using carefully prescribed procedures, such as the techniques described for the clinical and self-report scales for measurement of spasticity (see Table 2). Reliability (repeatability), validity (accuracy), and responsiveness (to change over time and because of intervention) are all desired qualities (Platz et al., 2005).

Reliability

Reliability is concerned with repeatability of scores or, conversely, with error of measurement. Spasticity assessment using the AS, MAS, and MMAS involves subjective classification into distinct, ordered categories by individual examiners, so agreement indices are used to evaluate repeatability. Obtaining a high level of agreement in spasticity assessment requires that prescribed technique be followed under the same conditions for the same patients; for example, the same level of stretch applied at the same speed is needed to obtain the same scores on examiner assessments. The sense of catch or resistance must be the same. Further, examiner differences such as experience or tendency to rate high or low must be minimal to avoid systematic distortion of scores and to obtain high interrater agreement. Cohen’s kappa is the proportion of agreement corrected for chance, which has values ranging from −1 (complete disagreement) to 0 (chance agreement) to 1 (perfect agreement) (Viera & Garrett, 2005), and is appropriately used to assess agreement in spasticity scores by different examiners.

Table 3 shows values for kappa for the AS, MAS, and MMAS when used in spinal cord injury, stroke, and traumatic brain injury populations. In these studies, spasticity was evaluated by two to three raters in the upper and/or lower extremities (including digits) of 15–50 patients. Raters were PTs, PT students, or MDs. (No nurse raters were included in any of the studies.) More information was available for the MAS than that for the AS or the MMAS. In general, agreement was low to moderate. On the basis of available findings, it was not possible to determine whether agreement was higher or lower when tests were used in different patient populations.

Validity

Validity, or accuracy, refers to confidence in making inferences from test scores. Concurrent validity reflects relationships between spasticity scores obtained using two or more approaches or between spasticity scores and some theoretically associated variable obtained on the same occasion. Correlations, group differences, and regression techniques are used to evaluate concurrent validity (Brink & Wood, 1997). EMG and patient self-reports are both used to assess concurrent validity of examiner scores obtained using the AS, MAS, and MMAS.

Correspondence With EMG

EMG results are often selected as the criterion in validity assessments of clinical scales because the latency and the amplitude of the H-reflex provide direct information about spasticity under controlled conditions. In addition, a stretch-induced reflex response can be measured simultaneously as a clinical scale such as the MAS is administered. In general, associations between EMG and clinical examination are mixed.

AS scores were associated with EMG findings in five lower extremity muscle groups in patients with spinal cord injury (Sherwood, Graves, & Priche, 2000). Among patients with stroke, AS scores and EMG were significantly associated when upper extremities were tested (Patrick & Ada, 2006; Starsky, Sangani, McGuire, Logan, & Schmitt, 2005) but not in the lower extremities (Patrick & Ada, 2006). The pattern was opposite when MAS scores were used with stroke patients, where correlations with scores from lower extremity examinations were statistically significant (Cooper, Musa, van Deursen, & Wiles, 2005; Kim et al., 2005; Lamontagne, Malouin, & Richards, 2001), but those in the upper extremities were not (Leonard et al., 2006; Naghdi et al., 2008). Sample sizes were small (range = 13–31), except for Sherwood et al. (2000) (n = 97). The EMG response was stimulated.

### TABLE 3. Rater Agreement Using Cohen’s Kappa

<table>
<thead>
<tr>
<th>Condition</th>
<th>Test</th>
<th>AS</th>
<th>MAS</th>
<th>MMAS</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCI</td>
<td></td>
<td>.21–.61&lt;sup&gt;a&lt;/sup&gt;</td>
<td>.14–.35&lt;sup&gt;b&lt;/sup&gt;</td>
<td>.20–.62&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>CVA</td>
<td></td>
<td>.17&lt;sup&gt;d&lt;/sup&gt;</td>
<td>.21&lt;sup&gt;d&lt;/sup&gt;</td>
<td>.63&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>TBI</td>
<td></td>
<td>.16–.42&lt;sup&gt;e&lt;/sup&gt;</td>
<td>.49–.54&lt;sup&gt;f&lt;/sup&gt;</td>
<td>.81&lt;sup&gt;f&lt;/sup&gt;</td>
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</table>

electrically at the nerve rather than with passive stretch; this procedure is inconsistent with the definition of spasticity as a movement-related, velocity-dependent response.

Sorionola et al. (2009) studied EMG response of wrist flexors in the hemiplegic wrist of 10 persons with stroke who met the inclusion criterion of a screening MAS score of 1 or more. They used a procedure consistent with definitions of spasticity and the protocol for clinical exams. Experimenters completed the MAS. They also applied passive manual stretches that were measured objectively using a rig applied to the forearm and hand. Target velocities ranged from slow (60 degrees/second) to quick (360 degrees/second). The EMG outcome was the percentage of maximal voluntary contraction. They found that experimenters had difficulty reaching absolute target stretch velocities but were able to successfully apply “slower” (50 degrees/second) or “faster” (240 degrees/second) stretch. Average EMG response and variability in response showed accelerating increase as velocity increased. The correlation of EMG response and MAS score was significant \( r = .72, p < .05 \) only at the lowest measured velocity. The authors urged caution because of the small sample size but suggested that the MAS measure the mechanical compliance of the muscle rather than the velocity-dependent response of spasticity. The study is important because it goes beyond asking simple questions about agreement between raters or correlation between standard EMG and clinical ratings to explore biological mechanisms that can explain some of the disparate results in other research.

**Examiner and Patient Correspondence**

Another line of evidence important to validity assessment is correspondence between patient self-report of spasticity and examiner assessment. Among patients participating in the Stockholm Spinal Cord Injury Study, “yes” or “no” answers to a query about whether they experienced spasticity was associated with examiner identification of any spasticity using the MAS; this association held for most joints and movements tested (Sköld et al., 1999). The correlation between self-ratings using the VAS with MAS scores ranged from .44 to .62 \( p < .001 \) when measured in 45 people with spinal cord injury who were taking part in an intervention study for spasticity (Sköld et al., 2000). The cross-sectional correlation between VAS and AS scores was .70 among 47 patients with spinal cord injury but was much lower \( r = .36 \) when a question about general level of spasticity was used for self-report (Lechner, Frotzler, & Eser, 2006). In a subset of 8 of the 47 patients, correlations between VA and AS over time were weak but significant for three patients and low for the remaining five patients. Pain and other symptoms seemed to interfere with self-report of spasticity specifically. Taken together, findings about correspondence between self-report and examiner ratings are mixed. The longitudinal studies suggest intra-individual variations in the experience of spasticity as well as between-individual differences in the ability to pinpoint spasticity as a specific unpleasant symptom.

**Responsiveness**

Responsiveness, a change in observed score that occurs with true change over time as disease progresses or after intervention, has long been a key concern in spasticity measurement (Dekker, Dallmeijer, & Lankhorst, 2005). The AS (Ashworth, 1964) was originally developed to assess responsiveness to treatment of spasticity to treatment with carisoprodol in multiple sclerosis. More recent studies have demonstrated that AS and MAS scores decrease in response to treatment with intrathecal baclofen (Delhaas, Beersen, Redekop, & Klazinga, 2008; Pohl et al., 2003) and botulinum neurotoxin NT 201 (Caty, Detrembleur, Bleyenheuft, Deltombe, & Lejeune, 2008; Kanovsky et al., 2009). Neuromuscular function changes after insults such as stroke (Mirbagheri, Tsao, & Rymer, 2009) and spinal cord injury (Sköld et al., 1999), but we were not able to locate any studies that formally measured spasticity over time in conjunction with progression of UMN disease.

**State of the Science**

Spasticity is a neuromuscular alteration that occurs concurrently with other manifestations of dysfunction in the presence of UMN disease. The unpleasant experience of spasticity is associated with complications such as pain, insomnia, difficulties in performance of activities of daily living, and persistent spasticity contributes to development of permanent contractures. Thus, accurate and precise measurement of spasticity over time is critical to effective clinical practice but continues to be challenging. The evolving definition of spasticity (Burridge et al., 2005; Lance, 1980) makes the conceptual basis for measurement a moving target and validity difficult to achieve with both clinical examinations and patient self-reports.

The underlying biology is complex. There is an expansive body of research on spasticity but much of the literature is technical and can be difficult for nonspecialists to interpret. Spasticity results from the injury and dysfunction of UMs. Loss of inhibitory UMNs results in unchecked synaptic transmission in the spinal cord and exaggerated stretch reflexes. With the loss of UMN control, new synaptic connections appear in the spinal cord, but the nature of these new
synaptic connections is poorly understood (Nielsen et al., 2007). In other words, although UMN dysfunction is essential to spasticity, the effects may occur at many points within the nervous system, creating a myriad of effects. The exact pathophysiology of spasticity in traumatic brain injury may be different than spasticity in multiple sclerosis, for example, making it difficult to generalize study conclusions from one population to another. Spasticity in upper limbs may be different from spasticity in lower limbs, also depending on the type of UMN injury. Variation in clinical examinations (AS, MAS, and MMAS) combined with variation in the way muscles were stimulated for EMGs (PROM, tendon tap, voluntary contraction, and electrical current) creates a complex literature. Simons and Mense (1998) emphasized the importance of using electrical stimulation to distinguish the stiff muscles of spasticity from the rigidity characteristic of other motor diseases like Parkinson’s disease, thus indicating the unique role of the H-reflex in assessing the validity of scores obtained from clinical assessment of spasticity.

Spasticity changes in response to different stimuli (e.g., electrical, mechanical tendon tap, velocity of passive range of motion, voluntary movement) and conditions (e.g., posture, fatigue, stress, disease progression), making measurement context dependent (Woolacott & Burne, 2006). Thus, true intrindividual variation rather than stability should be anticipated when spasticity is measured. The intrindividual variation may explain some reported instances of low levels of interrater reliability in clinical scores such as the AS, MAS, or MMAS, even when well-trained, experienced examiners are used.

Spasticity management is an important patient care issue as it can impact quality of life. Spasticity increases the tone of the muscles, and some spasticity in a UMN syndrome may be beneficial if it provides the muscle tone needed to maintain body posture, mobility, and ADLs in otherwise weak patients. When problematic spasticity outweighs any beneficial effects of spasticity, treatments such as use of oral medications, Botox injections, and intrathecal baclofen pumps are indicated. To manage spasticity and to evaluate effectiveness of a treatment plan, clinicians need valid and reliable scales that can be easily administered and interpreted and are sensitive to change in spasticity. Using the AS or its variations is a start, but information can be augmented by systematic, concurrent utilization of patient self-reports.

Both the VAS and the NRS allow incorporation of patient perceptions of their own spasticity to complement examiner findings, but little is known about patient ability to sort spasticity from related dysfunctions (such as clonus or hypertonia) to produce an accurate (valid) report. At this time, protocols for VAS and NRS self-report scales for spasticity do not include education about exactly what is meant by spasticity. Because patients with spasticity are prone to a wide array of unpleasant disturbing symptoms, this step is essential and should be included to optimize accuracy whenever self-report is used. Concordance between self-report and examiner scores may improve when this step is taken, contributing to the believability of patient reports (NIH PROMIS, 2007). Inclusion of patient report and self-assessment scales in research is of paramount importance. New scales or modification of existing scales must account for patients’ lived experiences of spasticity.

Summary

Spasticity is a movement-related velocity-dependent neuromuscular dysfunction experienced as tight or stiff muscles that affects quality of life for people who experience this phenomenon. Spasticity is manifested as increased amplitude or shortened latency of EMG activity in response to stretch or electrical stimulation. Systematic examiner assessment (AS, MAS, and MMAS) and patient-reported approaches are used clinically to measure spasticity. Discrepancy between self-and examiner evaluation of spasticity complicates decision making about treatment and evaluation of treatment effectiveness. Understanding of the pathophysiology, definition, and patient conceptions of spasticity is the starting point to translate research into better clinical practice for spasticity.

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