

Current Challenges to Clinical Assessment of Spasticity

Chetan P Phadke, Farooq Ismail, Chris Boulias

Chetan P Phadke, Farooq Ismail, Chris Boulias, Spasticity Research Program, West Park Healthcare Centre, Toronto, Canada

Chetan P Phadke, Farooq Ismail, Chris Boulias, Faculty of Medicine, University of Toronto, Toronto, Canada

Chetan P Phadke, Faculty of Health, York University, Toronto, Canada

Correspondence to: Chetan P Phadke, Spasticity Research Program, 82 Buttonwood Ave., Toronto, ON, Canada.

Email: chetan.phadke@westpark.org

Telephone: +1-416-243-3600 x 2716 Fax: +1-416-243-3747

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INTRODUCTION

Spasticity is a positive sign of upper motor neuron lesions characterized by an increase in resistance to movement perceived when a clinician quickly moves the limb through the available passive range of motion. Spasticity has been classically defined by Lance (1980) as “a velocity-dependent increase in resistance” as a result of “hyperexcitable stretch reflexes”^[1]. More recently, an updated and more comprehensive definition of spasticity described as “disordered sensori-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles” has been proposed^[2]. For the purposes of this paper, we discuss assessments of spasticity based on the classic definition of Lance. Spasticity requiring treatment is most commonly seen in conditions such as stroke, multiple sclerosis, and cerebral palsy^[3]. Spasticity and the resulting tightening of muscles has a multi-dimensional impact in areas such as reduced range of motion in arms and legs, potential for contractures if left untreated, hygiene issues specifically in areas of hip and shoulder adductor and finger flexor spasticity, poor body image, mobility, pain, balance, and walking variability^[4,5]. Treatments are available to help manage spasticity which can present an ongoing and long-term problem^[4].

Choice of treatment options is dependent on the underlying mechanisms that result in an increased resistance to movement. There are two mechanisms to explain the increased resistance: (1) hyperexcitable spinal reflexes (neural)^[1] and (2) secondary tightening of soft tissue structures around the joints (musculotendinous)^[6]. The neural or reflex component can be teased apart by assessing the muscle response to manipulation of movement speed, whereas, the musculotendinous or non-reflex component can be isolated by measuring the end range or resistance to slow passive movement of the joint^[6]. The neural component can be managed using physical means such as rhythmic repetitive motion exercises^[7-9] or using pharmaceutical agents delivered either orally or using targeted

ABSTRACT

A high prevalence of spasticity in patients with a variety of upper motor neuron lesions has been reported and spasticity assessments and treatments have been the focus of several recent research articles. It is important for researchers based in non-clinical settings, who are designing scales to measure spasticity, to understand the nuances of the challenges facing clinicians involved in spasticity management. Likewise, it is also important for clinicians to understand the need for objectivity and systematic approach to measuring spasticity that is at once valid and reliable. This paper summarizes the challenges clinicians face when using clinical tools for spasticity assessments, presents variety of lab-based measures available to clinicians, and describes the pros and cons of each approach. Finally, information on devices to measure spasticity that can satisfy both clinical as well as research demands are presented with areas for future work.

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Key words: Muscle spasticity; Botulinum toxin; Spinal reflexes

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delivery such as in the case of Baclofen pumps^[4] or intramuscular interventions^[10]. Botulinum toxin (BoNTA) intramuscular injections chemically denervate parts of the muscle and are also used to target the neural component of spasticity^[10]. The musculotendinous component can also be managed by active as well as passive exercise involving systematic stretching and joint positioning using orthosis^[4].

Irrespective of the choice of treatment, accurate assessment of spasticity level is critical to the success of the treatment. Spasticity is commonly seen in several muscles in arms and legs either unilaterally such as in stroke or bilaterally as in the case of MS^[3]. Muscle groups typically requiring treatment for spasticity are elbow, wrist, and finger flexors, hip adductors, knee flexors, ankle plantar flexors, and toe flexors^[3]. Thus, clinicians face the pressure of completing and recording spasticity assessment on multiple limb muscle groups (or individual muscles) in an accurate and a timely manner. There are several tests available to assess spasticity. Based on the feasibility of the tests in a clinical environment, spasticity assessment techniques can be divided into two groups: (1) clinic-based and (2) lab-based. There are other tests that measure impact of spasticity on function and quality of life in patients with spasticity^[4]; however, this review purely focuses on tools specifically used to assess level of spasticity only.

There are number of clinic based tests for spasticity assessments such as Modified Ashworth Scale (MAS), Modified Tardieu Scale (MTS), Wartenberg Knee Pendulum Test (WKPT), Numerical Rating Scale (NRS), Penn Spasm Frequency Scale (PSFS), time taken for muscle to relax, and Range of Motion (ROM; resting, passive, and active available ROM). These assessments have been described in detail elsewhere^[4]. Common elements in all spasticity assessments are passive movement of the limb and perception and rating of the increased resistance to movement sometimes described as a catch. Among these scales, clinicians perceive MAS to be the most feasible test in terms of time requirements and ease of testing in multiple muscles. There are indications from research studies in patients with spinal cord injury that MAS is correlated with reflex hyperexcitability^[11] as well as in patients with stroke where changes in MAS are correlated with changes in muscle spindle sensitivity^[10]. Unfortunately, MAS has limited reliability and poor validity leading to appeals for clinicians to stop using it in its current form^[12,13]. There have also been efforts to further modify the MAS and have shown promising improvements in intrarater reliability^[13].

One of the criticisms of the MAS is that it does not provide spasticity rating in response to different movement speeds, which has been shown to be a sensitive way of assessing the reflex hyperexcitability (neural component) underlying spasticity^[14]. On the other hand, MTS is performed both at slow and fast velocities and inherently has better validity than MAS considering Lance's definition of spasticity. In the MTS, slow movement velocity is used to determine if there is a contracture or loss of range of motion and the fast movement speed is used to trigger the stretch reflex. In addition to the qualitative rating of spasticity similar to the MAS (examiner's perception of resistance), MTS also incorporates measurement of the spasticity angle. Spasticity angle is the angle of catch at fast speed and as spasticity decreases, it is conceivable that the spasticity angle would also decrease. Thus, MTS is more promising scale because it is able to discriminate the neural component better by using two movement speeds^[15]; however, MTS requires use of a goniometer which is difficult to align with the joint during limb movements and may be a factor affecting reliability of the MTS^[16-18].

Among other tests to assess spasticity, WKPT is only useful for spasticity in knee muscles and cannot be readily used for other

joints in the arm and leg because it relies heavily upon the long lever and gravity to provide consistent repeatable limb movement to produce muscle stretch and elicit spasticity. Specifically ankle plantar flexors spasticity which is commonly treated with BoNTA^[3] cannot be tested using WKPT because of a very small lever arm (foot). NRS and PSFS are useful measures to assess patient perception of spasticity^[4,19]; however, they cannot be solely relied upon because patient perception of spasticity only partially represents the neural component of spasticity^[20]. Time taken by the muscle to relax in response to a stretch may be an important and readily testable measure that reflects muscle spindle sensitivity and reflex hyperexcitability underlying spasticity and clonus^[21], but no research has been done on this measure. ROM measurements are useful in assessing the end range, but do not discriminate between neural or musculotendinous changes. Additionally, goniometry method to assess ROM has validity and repeatability concerns due to variability in placement, force application, and alignment of the goniometer arms^[22] and is thus, non-sensitive to small changes.

In contrast to the clinic-based measures, in the lab-based spasticity assessments limb movement and perception of increased movement resistance are both automated and objectified; thus, requiring specialized equipment. A motor drives the passive limb movement and force transducers quantify speed of movement and increased resistance or surface electromyography (EMG) sensors quantify output from the muscle being stretched^[23]. A good working knowledge of biomechanical principles is necessary to conduct lab-based tests of spasticity. Force entities such as torque (measured in Newton-N), stiffness (damping), or elasticity (viscosity) are estimated using equations^[24] and measured using length of the lever arm and joint angle^[23]. For example, tonic stretch reflex threshold measurement (TSRT-a lab-based measure) more accurately reflects spasticity as defined by Lance^[1] than other clinical tests of spasticity^[23]. TSRT is measured using a combination of joint movement speeds and is velocity-dependent in evoking a muscle response^[23].

Although TSRT incorporates velocity-dependence, a hallmark of Lance's definition, it is worth noting that both neural as well as non-neural structures such as the connective tissues surrounding the joint contribute towards the velocity-dependent increase in resistance^[2,24]. Thus, a velocity-dependent increased resistance to movement could be attributed to reflex (neural) or viscoelastic properties of joints and muscles (non-neural)^[2]. To counter this velocity-dependent behavior of both neural as well as non-neural structures, Gaverth *et al* (2013) have used an elegant biomechanical model to isolate the neural from the non-neural components of increased resistance to movement^[25]. Their method involves isokinetic wrist extension using a NeuroFlexor device that moves the joint at different velocities and is thus limited only to the wrist and finger joints^[25]. They define the neural component (after removing the elastic and viscous components) as an estimate of true spasticity which has been shown to be a sensitive method to assess changes in spasticity after botulinum toxin injections^[26].

Among the spasticity measurements available, the lab-based measures appear to be the most robust assessments. These approaches have been routinely used in animal^[27] as well as human^[14,26] spasticity research. Although these motor-driven assessments are robust and provide a consistent and accurate assessment of spasticity, they require specialized equipment run by a computer software, which is also required for data analysis^[28]. This process can be time intensive requiring a special facility and personnel to collect and process the data and does not provide point-of-care (POC) spasticity assessment.

POC assessments are valuable for timely and efficient spasticity management. To address this issue, efforts have been made to design clinic friendly POC equipment that balances the need for ease of testing and scoring as well as a need for accurate and valid measurements^[14,25]. These devices are able to discriminate the neural from the musculotendinous components in a relatively short period of time. However, this technology is in its early stages of development and is limited to testing a single joint (elbow or wrist/fingers) and thus, may be cost-prohibitive.

In the absence of a better outcome measures that match the ease of testing and scoring of MAS, clinicians continue to use MAS to assess spasticity. There is also some evidence to support using MAS^[10] and hence researchers continue to make efforts to refine this scale to improve its validity and reliability^[13]. In addition to the scoring method, another concern with MAS that needs to be addressed in future work is the need to test increased movement resistance at various movement speeds. A recent paper used a simple yet promising approach to moving limbs at consistent and repeatable speeds across sessions using a metronome^[6]. Reasons why this approach is promising are that it uses very little if any specialized equipment, assesses the neural component using different limb movement speeds, and allows a consistent way to replicate movement speeds before and after spasticity treatment^[6].

If spasticity assessments are time-consuming, require multiple steps, and specialized equipment and personnel, then it will be harder for clinicians to adopt and implement such assessments in their clinical practice. Researchers should keep this in mind as future studies are designed to investigate the best way to clinically assess spasticity. It is clear that MAS in its current form is not the perfect answer and future work is being directed towards improving the validity and reliability of MAS.

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CONFLICT OF INTERESTS

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Peer reviewer: Fang Li, MD, PhD, Department of Rehabilitation Medicine,, Huashan Hospital, Fudan University, Shanghai, 200040, China; Arzu Yagiz On, Professor, Ege University Medical Faculty, Department of Physical Medicine and Rehabilitation, 35100, Izmir, Turkey.