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Spasticity Assessment in Cerebral Palsy

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Contents

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Abstract

Spasticity is an important, but not the only, component contributing to the increased joint resistance experienced by children with spastic cerebral palsy. Conventional clinical spasticity scales, based on physical examination of the passive muscle, are easy to apply in pediatric populations. Unfortunately, these have low reliability and are unable to differentiate between the different components of joint hyper-resistance. To correctly differentiate spasticity from other neural and non-neural contributions, instrumented assessments that integrate electrophysiological and biomechanical measures are required. In the last 15 years, great advancements in clinically applicable, instrumented assessments were made. However, the translation from research to

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clinical setting is lagging behind. Simple, yet accurate, instrumented assessments are expected to greatly advance clinical practice in terms of treatment planning based on etiological classification and subsequent outcome evaluation. In addition, the transfer of the research findings to functional outcome would require to extend our research agenda to include assessments of hyperreflexia in the active muscle. Altogether these instrumented methods are not only needed to classify different aspects of joint hyper-resistance but will also provide further insight into its pathophysiology enabling the development of future treatment options for children with spastic cerebral palsy.

Keywords
Cerebral palsy · Spasticity · Electromyography · Hyper-resistance · Instrumented assessment

Introduction

Muscles in children with spastic cerebral palsy (CP) tend to have higher tone, hyperactive stretch reflexes, and an altered structure. These alterations manifest themselves as *muscle hyper-resistance* or "increased resistance perceived during passive muscle stretch" (van den Noort et al. 2017). Lower limb muscles that exhibit hyper-resistance due to spastic CP are the mm. gastrocnemius, m. soleus, m. tibialis posterior, mm. hamstrings, m. rectus femoris, mm. adductors, and the m. psoas. In the upper limb, spasticity is most frequently observed in the shoulder external rotators, the elbow, wrist and finger flexors, and the elbow pronators (Klingels et al. 2012).

Joint-Level Assessments to Infer About Muscle Function

Direct assessment of hyper-resistance of the distinct muscle during clinical examination is impossible. Therefore, routine physical examination includes assessments of the resistance during *passive joint rotation*. Examples of such assessments include the maximum passive range of motion (ROM) assessment, the (Modified) Ashworth Scale (Bohannon and Smith 1987), and the (Modified) Tardieu Scale (Tardieu et al. 1954). These examinations are clinically used to assess the degree and nature of muscle hyper-resistance. Moreover, in combination with other clinical assessments on impairment and functional levels, it is regarded an important factor to inform decisions on treatment options (Boyd and Graham 1999). However, much doubt has been raised regarding the reliability and validity of such joint-level assessments (Malhotra et al. 2009; Fleuren et al. 2010).

The difficulty of developing an assessment of muscle impairment in CP is related to the degree to which its findings represent the underlying pathology, i.e., what is actually being assessed? Firstly, a limited maximum passive joint ROM, assessed with goniometry, is often interpreted in terms of muscle contracture. However, any deduction of pathology in a single muscle based on this assessment is confounded by other muscular and nonmuscular soft tissue structures spanning the joint. Moreover, even when the other parallel structures are assumed not to contribute, passive joint ROM only allows to infer about the length of a muscular tendon complex, whereas the properties of the tendon, and not just the muscle belly, will affect joint rotation. Although tendon pathology has not extensively been studied in CP, its properties and interaction with the muscle, both during passive and active elongations, will affect muscle behavior (Kalsi et al. 2016).

Secondly, in CP, both *neural* and *non-neural* components may contribute to any resistance to muscle elongation during passive joint rotation. While these terms are very broad, neural components generally refer to features originating from the central nervous system resulting in increased muscular activation. Such increased muscle activation includes *spasticity*, defined by Lance as "a motor disorder characterized by a velocity dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflex, as one component of the upper motor neuron syndrome"

(Lance 1980). It is generally assumed that in spastic CP, spasticity is the primary neurological component contributing to muscle hyper-resistance. This neurological feature is related to the lack of inhibition of the stretch reflex and occurs as a direct result of the upper motor neuron lesion. The hyperactive stretch reflex, which is elicited during fast passive muscle elongation, such as in the (Modified) Tardieu Scale assessment, will result in involuntary muscle contraction. This pathological muscle contraction will resist the movement of the manipulated joint, being felt by the examiner. Next to spasticity also non-velocity-dependent neural components may increase muscle tone by involuntary muscle contraction during joint manipulation (see below and "Introduction"). The non-neural component of increased resistance during passive examination is a result of changes in the muscular tissue thought to be a result of tissue adaptations to the pathological neural regulation. Distinguishing between the neural and non-neural contributors to muscle hyper-resistance is imperative for understanding disease progression and for determining treatment options.

Definitions

The definition of spasticity by Lance acknowledges that spasticity is only one of the many features of the upper motor neuron syndrome. Many reflex circuits such as proprioceptive, cutaneous (Burke et al. 2013), and nociceptive (Kamper et al. 2001) can be affected by an upper motor neuron syndrome. These lead to a variety of pathophysiological mechanisms that give rise to involuntary muscle contractions. Therefore, in clinical settings, different features are commonly assessed in combination and spasticity is, wrongly, referred to in a broader sense (van den Noort et al. 2017).

In 2003, the North American Task Force for Childhood Motor Disorders redefined spasticity as: "a velocity dependent increase in hypertonia with a catch when a threshold is exceeded" (Sanger et al. 2003). This description is a good reflection of how spasticity can impede muscle elongation during the physical examination. However, as will be further discussed in the next section on existing clinical spasticity assessment scales, any resistance that relies on an examiners' subjective interpretation cannot truly isolate spasticity, and therefore, this is not a very useful definition. To avoid equating all resistance to muscle elongation during passive joint rotation with spasticity, it is important to clearly define all the expected neuromuscular responses to passive muscle stretch separately.

In 2005, a European Thematic Network to Develop Standardized Measures of Spasticity (the SPASM consortium) suggested the opposite and broadened the representation of spasticity by defining it as: "disordered sensory-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles" (Pandyan et al. 2005). Both the previously mentioned narrow definitions and this broader approach have shortcomings. When translating research findings to the clinic, the narrow definition results in a compromise on internal validity due the inability to isolate spasticity. On the other hand, a broad definition hinders the development of etiologically targeted treatments. Rather than compromising and broadening the definition, efforts should be directed at effectively isolating and measuring the underlying pathophysiological components in a clinical setting.

The lack of consensus on the definition has especially led to the use of the terms hypertonia and spasticity interchangeably, which has resulted in further confusion. A review by Malhotra et al. in 2009 highlighted the inconsistent use of the term spasticity in research articles. They found that 31% of articles referred to the definition of Lance, 35% equated spasticity with the term hypertonia, 31% provided no definition, and 2% included their own definition (Malhotra et al. 2009). Due to the variations in definitions, whether in clinical or scientific reporting, it is important either to clearly specify what is meant by the different terms or to avoid them all together.

In the latest attempt to clear up the controversy regarding spasticity and its definition, from 2014 to 2016, a European consensus study was performed. Rather than redefining spasticity, the consensus aimed to summarize the neuromuscular

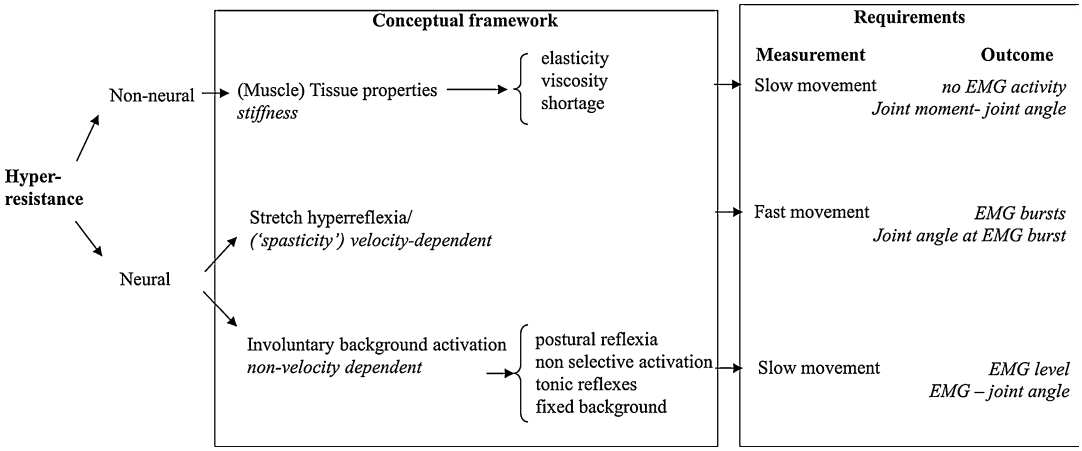

Fig. 1 Conceptual framework of pathophysiological neuromuscular responses to passive muscle stretch. (With permission from van den Noort et al. 2017)

responses to passive muscle stretch in subjects with an upper motor neuron syndrome. Thirty European clinicians and researchers participated. Using the Delphi approach for reaching consensus (Hasson et al. 2000), a conceptual framework was defined as a guideline for understanding the neuromuscular responses to passive muscle stretch in subjects with an upper motor neuron syndrome and for designing the requirements of an assessment method to quantify the different contributing components (van den Noort et al. 2017).

The term muscle hyper-resistance was conceived in the meeting and defined as "increased resistance perceived during passive muscle stretch." It was agreed that the neural and non-neural contributions to hyper-resistance have to be distinguished. Furthermore, it was unanimously agreed that spasticity cannot be equated with hyper-resistance but, given the definition offered by Lance, was a contributor. Therefore, it was recommended that the term spasticity should only be used next to the term "stretch hyperreflexia," thus helping to avoid its laden historical context.

In conclusion, three subgroups of components of hyper-resistance were defined in the consensus (Fig. 1). The neural contributions were subdivided into stretch hyperreflexia (velocity dependent, e.g., spasticity) and involuntary background activation (non-velocity dependent, e.g., postural reflexes, non-selective activation, tonic reflexes, and fixed background tone). The non-neural components involve muscle tissue changes including increased elasticity, viscosity, and shortening. Such adaptations predominantly occur due to reduced muscle growth (in terms of length and volume) and stiffening of the muscle tissue due to increased amounts of extracellular matrix (Mathewson et al. 2014). The consensus recommended being aware that muscle stiffness cannot directly be equated to joint-level stiffness (the relation between joint angle and joint moment) as it is not necessarily a reflection of the muscles intrinsic stiffness.

As we will show, having a well-defined framework of unambiguous terminology of the neuromuscular response to passive muscle stretch has many advantages for the management of spasticity in CP. Firstly, it facilitates communication between patients and caregivers, clinicians, and researchers. Secondly, it creates the requirements for developing valid, standardized, and objective clinical assessments of muscle hyper-resistance and its components. Thirdly, it promotes the development of feature-targeted individualized treatment.

Measurement Methods

How to assess a phenomenon whose definition is not agreed upon has been a matter of debate for several decades. A thorough set of reviews on the topic were published in 2005 by the SPASM

consortium (Burridge et al. 2005; Voerman et al. 2005; Wood et al. 2005). Approaches to assess the neuromuscular responses to passive muscle stretch when the subject is at rest can be divided into clinical qualitative approaches and instrumented quantitative approaches. Quantitative approaches can further be divided into those methods that assess a neurophysiological response to passive muscle elongation and those that assess the biomechanical response. Furthermore, a distinction can be made between robotic designs where the passive limb is manipulated by a motor-driven system and manual designs, where an examiner applies the muscle stretches. The following section will briefly discuss the different methods. The literature regarding instrumented, manually controlled spasticity assessments is summarized in a systematic review (Bar-On et al. 2014b).

Measurement Errors

With any assessment method, it is important to quantify and consider associated measurement errors. Common sources of measurement error include inaccuracies in the measurement instrument, variability in the phenomenon being measured, and, in case of manually performed assessments, the individual taking the measurements. Most measurement errors can be quantified using well-designed reliability studies. Such studies help establish standard error of measurement (SEM) and minimally detectable difference (MDD) values that are imperative when interpreting measurements results (de Vet et al. 2006). For example, for a treatment effect to be considered successful or unsuccessful, any post--treatment changes assessed with the instrument should at least be larger than the MDD.

Qualitative Assessment Methods

The most common clinical spasticity assessment scales, the Modified Ashworth's Scale (MAS) (Bohannon and Smith 1987) and the Modified Tardieu Scale (MTS) (Boyd and Graham 1999), are an established part of a standardized clinical assessment for children with CP (Boyd and Graham 1999; Graham et al. 2000). Other clinical tools have also been suggested, but their sensitivity to treatment effect is less investigated in CP (Jamshidi and Smith 1996; van den Noort et al. 2010; Jethwa et al. 2010; Morris and Williams 2018). The MAS is a qualitative 6-point ordinal scale to subjectively classify the resistance felt during passive stretch (Bohannon and Smith 1987). It has been developed for lower and upper limb muscles and is performed by moving a joint passively through its ROM at one velocity. The MTS is often performed on muscles that score 1 or above on the MAS. During the MTS, the angle (R1) at which a spastic catch is felt during a quick passive stretch is defined relative to the maximum available ROM (R2) defined when the joint is moved at slow velocity (Boyd and Graham 1999). We prefer the MTS over the MAS as it assesses the muscle reaction at two very distinct velocities, thus incorporating the velocity-dependent characteristic of spasticity. However, both tests have been criticized for their low inter-rater reliability (Morris and Williams 2018). For example, it has been shown that reliability of the MTS catch angle is compromised by the difficulty of repositioning the distal segment after a catch in order to read the angle using a goniometer (van den Noort et al. 2010). SEM values for passive ROM and for catch angles in lower limb muscles as assessed with the MTS are provided by Fosang et al. (2003). Among six raters, the SEM values in spastic hamstrings ranged from 6 to 10° for assessing the knee catch angle during fast passive stretch with the MTS (Fosang et al. 2003). This translates to an MDD value of 20° (de Vet et al. 2006). To correctly infer the effect of treatments, any post-treatment changes in the catch angle will need to exceed this value. However, in a group of 40 medial hamstring muscles treated with botulinum toxin A and casting in children with spastic CP, the average change in the MTS catch angle post-treatment was only 5° (±15°) (Bar-On et al. 2014f).

Importantly, since no specific physiological phenomena are being assessed using these clinical scales, the scales cannot convincingly

differentiate the neural from the non-neural components of muscle hyper-resistance (Fosang et al. 2003; Biering-Sørensen et al. 2006; Haugh et al. 2006; Fleuren et al. 2010) thus limiting their construct validity (Platz et al. 2005). The definition of spasticity as provided by Lance refers to the activation of stretch reflexes. However, multiple studies have reported poor correlations between exaggerated stretch reflexes measured by electromyography and the clinical assessment scales (Pandyan et al. 2001; Fleuren et al. 2010). In the highly cited article entitled "Stop using the Ashworth Scale," Fleuren et al. were the first to unmask the construct validity of the Ashworth Scale using surface electromyography (sEMG) and dynamometry. They showed low associations of the Ashworth Scale with these simultaneously assessed electrophysiological and biomechanical measurements (Fleuren et al. 2010). Similarly, the MTS catch angle was found to have little association with increased work assessed at the joint (Gholami et al. 2017). Also disconcerting is the finding that some subjects diagnosed as having spasticity by the clinical scales showed no signs of increased reflex activation as measured with EMG (Sinkjaer and Magnussen 1994; Galiana et al. 2005). Clinical scales can already be markedly improved by simultaneously measuring sEMG from the assessed muscle, which at least confirms the presence of hyperactive stretch reflexes.

Given the important limitations of clinical scales, it is commonly agreed upon that more robust spasticity assessments that are valid, objective, and provide quantitative data are needed. Only through proper assessment can the mechanisms underlying the pathology be efficiently addressed through treatment.

Quantitative Assessment Methods

Passive Muscle Assessments

Quantitative passive assessments can be divided into those methods that assess the neurophysiological response and those that assess the biomechanical response. Neurophysiological assessments help quantify elevated reflex responses. A commonly assessed example is the Hoffman reflex (H-reflex), elicited by low-threshold electrical stimulation of a mixed peripheral nerve. Alternatively, a tendon tap will elicit the tendon reflex (T-reflex), which follows a similar pathway to that of the H-reflex, but may also include the stretch reflex. Higher stimulation intensity of the mixed peripheral nerve results in the production of an M-wave and the eventual disappearance of the H-reflex. Lower H- and T-reflex latencies and higher reflex amplitudes are indicative of increased α-motor neuron excitability. The ratio of M-wave and reflex amplitudes (H_{max}/M_{max} and T_{max}/M_{max}) has been used as a measure of spasticity. However, there is much overlap in the values of these ratios between healthy and spastic muscles, reducing their diagnostic ability (Voerman et al. 2005). Eliciting M_{max} also requires a supramaximal stimulation, which is uncomfortable and therefore rarely used in children.

Neurophysiological responses can be combined with measures of the biomechanical behavior of muscles, joints, and limb segments. The most common way of doing this is recording sEMG synchronized with recordings of angular velocity and joint moments during various, well-defined conditions (such as electrical stimulation, passive oscillations and pendulum tests, ramp-and-hold stretches, or various types of active movements) (Voerman et al. 2005; Wood et al. 2005).

Highly sophisticated, motor-driven devices are the most accurate in standardizing and controlling joint trajectory and movement velocity. Modeling the behavior of muscles to such systems provides insight into the different components contributing to the measured hyper-resistance (de Vlugt et al. 2010; Gäverth et al. 2014; Sloot et al. 2015b). However, these methods are deemed impractical for clinical use, especially in pediatric populations. They may also be less representative of functional joint motion (Sloot et al. 2016). Alternatively, several manually controlled methods that integrate signals have also been developed (Pandyan et al. 2006; Fleuren et al. 2010; van den Noort et al. 2010; Wu et al. 2010; Bar-On et al. 2014b). These methods resemble the clinical assessment scales but additionally collect

quantitative data using synchronized instruments. Collecting instrumented data during clinical assessments provides the means to regulate performance, creating a measure of standardization, as well as a method to decompose the sources of measured hyper-resistance. Lately, the use of manually applied instrumented spasticity assessments in clinical trials involving children with spastic CP is increasing (Flamand et al. 2013; Bar-On et al. 2014b; Pennati et al. 2016). However, there is a paucity in reports on the associated measurement errors of outcome parameters hindering the transfer of instrumented manual methods to the clinic.

We developed a manual instrumented clinical assessment that combines neurophysiological and biomechanical measurements for the lower limb muscles of children with CP (Bar-On et al. 2012a, b, 2014a, c, d, f). The method involves simultaneous collection of sEMG, angular velocity, and net joint moment during ramp-and-hold passive joint manipulations through the full ROM. Several parameters, extracted from signals collected during the joint manipulations and compared between movement velocities, have proven sensitive to distinguish between muscles with differing levels of hyper-resistance and to the effects of treatment. Figure 2 shows examples of such signals collected during fast passive ankle rotations in three different children with spastic CP. Despite being manipulated at similar angular velocities, the EMG reactions and consequent joint moment are markedly different between subjects. In the first example, the EMG signal continues for a longer duration than in example 2 where only a short burst was recorded. In example 3, a clear clonus is triggered by the manipulation. Examples of parameters that can be extracted from such data include the amount of reflex hyper-activation (average root mean square-EMG) (Bar-On et al. 2012b, 2014a); the degree of hypersensitivity to reflex activation (the spastic threshold) (Bar-On et al. 2014c); the presence, location, and severity of a spastic catch (Bar-On et al. 2012a); the type of muscle reflex activation pattern (phasic or tonic) (Bar-On et al. 2014c); joint moment at a particular joint angle; and work (the integral of joint moment versus position) (Bar-On et al. 2014a). A similar application for the elbow flexors has been validated for children with CP (Wu et al. 2010).

The combination of EMG with biomechanical signals (e.g., joint angle, angular velocity, angular acceleration, net joint moment, or power) allows quantification and exploration of the biomechanical triggers and effects of reflex activity. For example, by expressing the timing of EMG onset in terms of joint angle, angular velocity, or angular acceleration, a particular parameter of the neuromuscular system like the "spastic threshold" can be quantified (Calota and Levin 2009). In the gastrocnemius and medial hamstring muscles, it was found that a reduced stretch-reflex threshold constrains peak muscle lengthening velocity during gait in children with CP (Bar-On et al. 2014e). Also, in preliminary work, lower spastic thresholds were associated with a poorer response to treatment with botulinum toxin A (Bar-On et al. 2015).

Study of the biomechanical signals following EMG onset allows quantification of the effect of the reflex on the joint. An example is the catch angle, which can be quantified in several ways (van den Noort et al. 2010; Wu et al. 2010; Bar-On et al. 2012a). The quantified catch was found to be the most related to reflex activation when defined as the angular position corresponding to the first local minimum of power after a local maximum of power (with power defined as the product of angular velocity and joint moment) (Bar-On et al. 2012a) (Fig. 2). However, it should be realized that the relationship between evoked EMG and force production is not straightforward. Moment-related biomechanical parameters collected during passive stretch have proved to be less sensitive to the construct of spasticity than the simultaneously collected EMG-related parameters (Voerman et al. 2005; Pandyan et al. 2006; Bar-On et al. 2012a). In the medial hamstring and gastroc-soleus, studies show less response to botulinum toxin A in the moment compared to the EMG-related parameters (Bar-On et al. 2014a, d, f). These findings suggest that these moment-related parameters may not adequately capture the contribution of hyperactive reflexes to hyper-resistance. To do this, a more sophisticated decomposition of the moment signal is required.

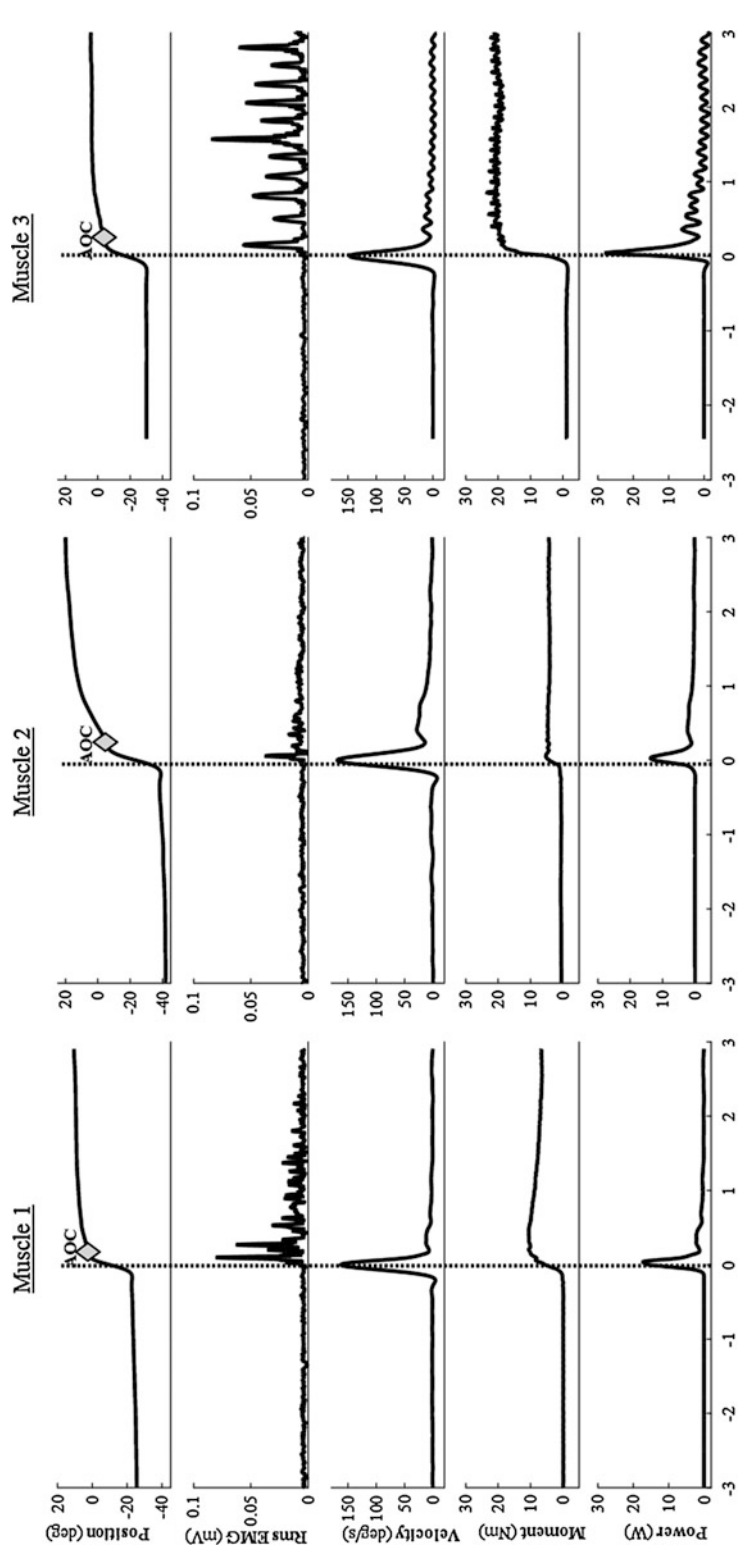

Fig. 2 Electrophysiological and biomechanical signals collected during fast passive stretches to the medial gastrocnemius from three different children with spastic cerebral palsy. Time at EMG onset is indicated by the dotted vertical line. The angle of catch (AOC) is defined as the first local minimum of power after a local maximum of power. Despite being stretched at a similar angular velocity, it can be appreciated that there is a large variability between the signals collected from each muscle

Several models to decompose the net joint moment have been developed to better understand hyper-resistance (Sinkjaer and Magnussen 1994; Galiana et al. 2005; Chung et al. 2008; de Vlugt et al. 2010; Lindberg et al. 2011), although only few have been applied in CP (de Gooijer-van de Groep et al. 2013; Willerslev-Olsen et al. 2013). The straightest forward of these models describe only the behavior of the non-neural components of hyper-resistance, such as passive stiffness (Harlaar et al. 2000) and viscosity (Meyer and Lieber 2011). These non-neural components have been extensively studied in both healthy and hemiplegic subjects and have been previously described by polynomial or exponential mathematical models (Harlaar et al. 2000; de Vlugt et al. 2010). More sophisticated mathematical algorithms have additionally modeled the neural contribution to the resistance measured during passive stretch (Chung et al. 2008; de Vlugt et al. 2010; de Gooijer-van de Groep et al. 2013). De Gooijer-van de Groep et al. reported that reflex-related joint moment in the plantarflexors of children with CP was almost six times higher and tissue stiffness twice as high than that of controls (de Gooijer-van de Groep et al. 2013). Contradictory findings were reported by Willerslev-Olsen et al. where the majority of assessed soleus muscles exhibited abnormal non-neural-related stiffness, and only a minority showed a reflex-related joint moment that was higher than that of controls (Willerslev-Olsen et al. 2013). These contradictions may be a reflection of different perturbation methods (small (6°) movements in de Gooijer-van de Groep et al. (2013) vs. ramp-and-hold rotations over the entire range of motion in Willerslev-Olsen et al. (2013)) and different joint moment decomposition models. Additionally, while de Gooijer-van de Groep et al. (2013) included all three plantarflexors, Willerslev-Olsen et al. (2013) analyzed only the soleus. Another example by (Sloot et al. 2015b) applied a modified model (de Vlugt et al. 2010) with ten neuromuscular parameters to fit the relation between the ankle angle and the ankle net joint moment measured during controlled slow and fast full range ankle rotations in typically developing subjects and subjects with spastic CP. The authors reported good reliability of the extracted parameters that were sensitive to the effects of botulinum toxin A and selective dorsal rhizotomy (Sloot et al. 2015b). All three mentioned studies reported a large variability between patients and muscles in the contribution of stiffness and neural components to ankle joint resistance. Variability could not be explained by measurement error or age differences and may therefore reflect different clinical phenotypes. This emphasizes the need to individually define the components of joint hyper-resistance in order to better tailor treatment.

Using a simplified version of the same model as Sloot et al., Bar-On et al. (2014d) extracted the neural component based on measurements from a manually controlled instrumented assessment (Bar-On et al. 2014d). The model, which included only stiffness and viscosity, was fitted to the joint moment-position data collected at the ankle during low velocity full range manipulations. This model was then fitted to stretches in which a stretch reflex was evoked (high velocity manipulations). The amount of deviation between the modeled and measured moment during these latter stretches represented the pathological neural component. This "deviation parameter" was found to be repeatable between assessments and to distinguish between healthy and spastic muscle. Additionally, unlike the previously described net joint moment-related parameters containing both neural and non-neural components, the deviation parameter was found to decrease post-treatment with botulinum toxin A (Bar-On et al. 2014d). These methods help break down the measured net moment into a clinically relevant representation of the contribution of stretch reflexes to joint hyper-resistance. Unfortunately, model assumptions on the relation between muscle lengthening and joint rotation prevent accurate and realistic estimates of the amount of muscle tissue stiffness that contributes to joint hyper-resistance. To achieve this, a combination of muscle imaging to measure the actual muscle belly lengthening from the different muscles acting on the joint is required and is scope for further study (Zhao et al. 2011; Haberfehlner et al. 2016).

When quantifying muscular responses to passive joint perturbations, it is important to consider that the stretch-reflex response may differ between muscles possessing different morphology (e.g., mono- vs. biarticular, short vs. long tendon, pennate vs. parallel). In a study carried out with a manual instrumented assessment on several lower limb muscles in children with CP, we identified lower stretch-reflex thresholds and less velocity-dependent activation in the hamstrings and adductor muscles when compared to the gastrocnemius and rectus femoris muscles (Bar-On et al. 2014c). Similarly, Kamper et al. found earlier reflex thresholds and greater reflex responses in the finger flexors than in the elbow flexors (Kamper et al. 2001). Activation differences between muscles may be caused by differences in central and peripheral stretch-reflex modulation and/or by the different morphology. For example, muscle force generation is influenced by muscle-specific properties such as moment arm, cross-sectional area, and pennation angle. Differences may also reflect the dependence of a reflex response on joint position prior to stretch (Musampa et al. 2007). The rectus femoris and gastrocnemius have been found to be less sensitive when stretched from initially longer lengths (Meinders et al. 1996), while in the hamstrings, the opposite has been reported (Sheean 2008). In biarticular muscles, the position of both joints is important when considering length dependency (Musampa et al. 2007). Therefore, subject positioning and measurement setup are important confounders when assessing spasticity and must be standardized for each specific muscle being assessed.

Active Muscle Assessments

The ultimate goal of spasticity management in CP is to improve function, such as gait. Since joint hyper-resistance in CP has mainly been assessed in response to imposed stretches on relaxed muscle, little is known about the role of hyper-resistance during purposeful movements involving voluntary muscle contraction. Consequently, the level of hyper-resistance assessed in relaxed muscles cannot fully explain the variability in gait pathology among children with CP (Van Campenhout et al. 2014).

The difference between passive and active muscle assessment lies in the role of the healthy stretch reflex. When a healthy relaxed muscle is suddenly stretched, stretch reflexes are inhibited by the central nervous system (CNS). In muscles affected by CP, due to lack of CNS inhibition, the same stretch results in a hyperactive reflex response. In active muscle, these mechanisms are different. When a healthy active muscle is suddenly stretched, CNS inhibition is depressed such that stretch reflexes are activated. In active muscles affected by CP, the already depressed CNS inhibition cannot be depressed any further, and thus stretch reflexes are also active. This makes the distinction in stretch-reflex contribution to overall muscle resistance between healthy and affected muscles when activated less obvious (Nielsen et al. 2005). The phenomenon is further complicated by task dependency of stretch-reflex inhibition. For example, stretch reflexes play an active contribution to joint stability during healthy stance phase of gait, but during swing, they are inhibited (Jansen et al. 2014). Furthermore, unlike in the passive muscle, reflex regulation during gait in pediatric populations is age-dependent, probably due to maturation of the central control of gait (Willerslev-Olsen et al. 2014).

Several research groups have sought to relate neurophysiological measures (muscle activation) to biomechanical measures (muscle lengthening) using instrumented 3D gait analysis. 3D gait analysis involves the simultaneous capture of joint kinematics using a motion analysis system. Joint kinematics entered into musculoskeletal models can be used to derive muscle lengths and muscle lengthening velocities during gait (Delp et al. 2007). Capturing ground reaction forces with force plates embedded into the walkway enables the computation of net moment around each joint, computed using inverse dynamics. Lastly, muscle activity during gait can be simultaneously recorded using sEMG.

As spasticity is velocity dependent, it is presumed that a faster muscle lengthening velocity during gait triggers stretch reflexes in a spastic muscle. Therefore, one approach to evaluate the effect to spasticity on gait has been to record the changes in muscle length and sEMG data with

increasing walking velocity (van der Krogt et al. 2009; Van Campenhout et al. 2014). However, it has been found that children with CP apply similar mechanisms to increase walking speed as typically developing children (Schwartz et al. 2008; Van Campenhout et al. 2014). Therefore, isolating the effects of spasticity from those changes required to increase walking velocity has proven challenging.

A more direct approach to assess spasticity during gait is to examine the motor output (EMG) over the lengthening phases of the involved muscle groups (Crenna 1998; Lamontagne et al. 2001; van der Krogt et al. 2009, 2010). Studies that have followed such an approach have shown that during particular phases of the gait cycle, the relationship between EMG and muscle lengthening velocity differs between typically developing children and children with CP (Crenna 1998; van der Krogt et al. 2010). Sloot et al. (2015) investigated whether belt accelerations and decelerations of five different intensities applied during the stance phase of treadmill walking evoked reflexes in the gastrocnemius, soleus, and tibialis anterior in healthy subjects (Sloot et al. 2015a). They found clear changes in muscle length and stretch velocity relative to unperturbed walking and that stretched muscles showed a surplus in muscle activity, i.e., EMG bursts on top of the reference activity following perturbation, exposing a clear stimulus response relation (Sloot et al. 2015a). This method of evoking stretch reflexes during gait now needs to be investigated in children with spastic CP. In a more complex setup in children with CP and controls, Willerslev-Olsen et al. (2014) used an orthotic device during treadmill walking to directly apply an ankle perturbation to the soleus muscle, either lengthening or shortening it during crucial phases of the gait cycle (Willerslev-Olsen et al. 2014). They reported that short-latency reflexes were enhanced in children with CP, while long-latency reflexes were depressed. Given the aforementioned role of healthy stretch reflexes during the gait cycle, this resulted in the same amount of m. soleus muscle activation prior to heel strike in children with CP and controls. The authors therefore argue that spasticity is unlikely to contribute to foot drop and toe walking in children with CP. Rather, they proposed that altered central drive to the ankle muscles and increased passive muscle stiffness are the main causes of foot drop and toe walking (Willerslev-Olsen et al. 2014).

The debate about the contribution of each component to joint hyper-resistance during movement continues because it remains hard to experimentally assess in vivo muscle function. Therefore, more comprehensive datasets are required (e.g., dynamic ultrasound imaging combined with EMG in a variety of different passive and active muscle conditions) complemented by neuromusculoskeletal modeling. The assessment of hyperreflexia in the active muscle is needed such that future research findings can be used to optimize the functional outcome of children with spastic CP. This requirement necessitates broadening our research agenda.

Clinical Interpretation of Instrumented Assessments

In contrast to the clinical scales whose outcome is limited to ordinal subjective scoring, instrumented assessment, capturing both the biomechanical and electrophysiological reactions to passive muscle stretch, potentially yields relevant information to identify etiology of joint hyper-resistance. As an example, in this section, we will present data collected from two clinical cases concerning spastic cerebral palsy where clinical and instrumented data were collected during passive ankle joint manipulation. Case 1 is a 5-year-old girl with spastic right hemiplegia, and case 2 is an 8-year-old girl with spastic left hemiplegia. Both children received intramuscular injections of botulinum toxin A to the medial gastrocnemius as part of a multilevel treatment. Injections were followed by 2 weeks of lower leg casting and intensive physical therapy. The children were assessed with the MAS, with the MTS, and with an instrumented assessment (Bar-On et al. 2012b), before and about 8 weeks after treatment.

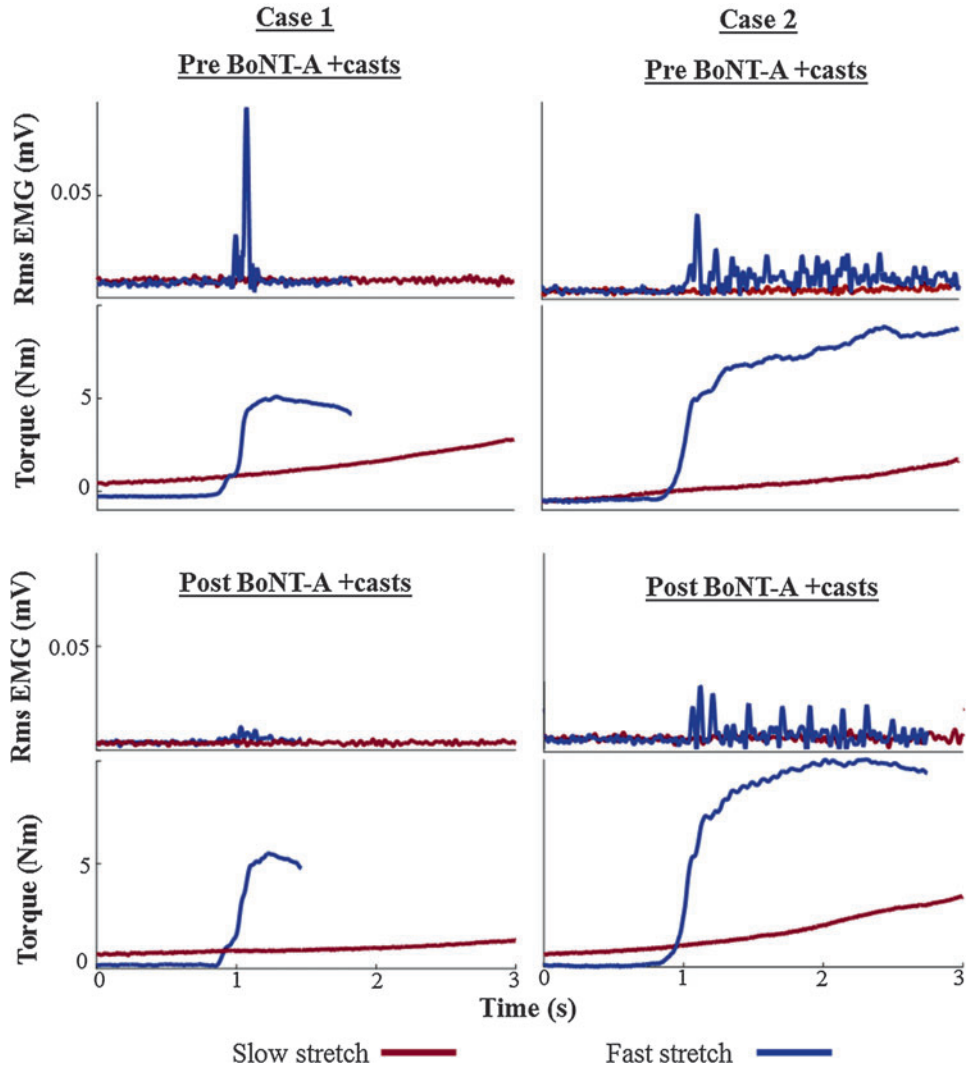

Fig. 3 Medial gastrocnemius rms-EMG and net ankle joint moment collected during slow (gray) and fast (black) velocity ankle angle rotations pre- and post-treatment with Botulinum toxin A (BoNT-A).

Results from the clinical and instrumented assessments can be found in Fig. 3 and Table 1. Pre-treatment assessment with the clinical scales resulted in the same values being assigned to the muscles. The parameters calculated from the instrumented assessments for the two clinical examples clearly reflect the effect of increasing stretch velocity on acquired root means square of the EMG (rms-EMG) signal and on the resulting ankle joint moment. Unlike the clinical sores, the values established for the two clinical examples are markedly different, despite being stretched at the same angular velocity. Furthermore, changes post-treatment were detected with the instrumented tests, but not with the clinical scales.

Given the variable response to treatment between the clinical cases, it is worthwhile to investigate whether instrumented assessments can help us understand this. The 3D graphs in Fig. 4 illustrate the degree of length dependency versus velocity dependency of muscle activation in the medial gastrocnemius during passive ankle dorsal flexion of the two clinical cases pre--treatment. On the y-axis, three different angular

Table 1 Details of clinical and instrumented assessments in two clinical examples

	Clinical case 1	**Clinical case 2**
Age	5 years old	8 years old
Diagnosis	Spastic CP, right hemiplegia	Spastic CP, left hemiplegia
Amount of injected botulinum toxin A in medial gastrocnemius as part of multilevel treatment	3 U/kg	3 U/kg
Gastrocnemius Modified Ashworth's Scale score		
Pre-treatment	1+	1+
Post-treatment	1+	1+
Gastrocnemius catch angle (Modified Tardieu Scale)		
Pre-treatment	−15°	−15°
Post-treatment	−15°	−10°
Average rms-EMG from the medial gastrocnemius during fast passive stretch minus average rms-EMG during slow passive stretch		
Pre-treatment	6.65 μV	4.42 μV
Post-treatment	1.96 μV	4.10 μV
Net ankle joint moment assessed at 0° during fast passive stretch minus value at 0° during slow passive stretch		
Pre-treatment	3.40 Nm	1.98 Nm
Post-treatment	1.03 Nm	3.25 Nm

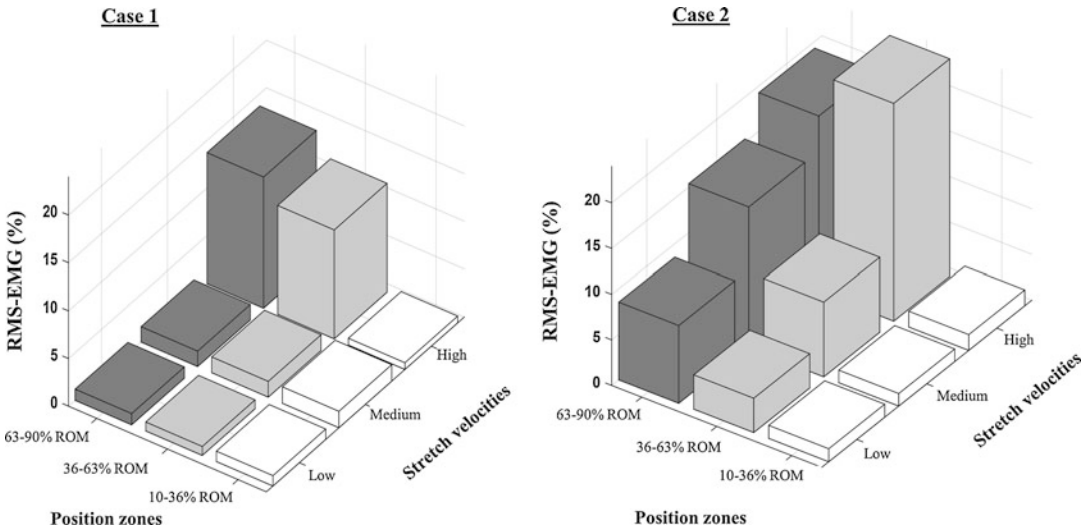

Fig. 4 Average normalized rms-EMG measured during joint motions applied passively to the ankle at three joint angular velocities (low, medium, and high) across three equally spaced position zones spanning 10–90% of the joint range of motion (ROM)

velocities are represented. On the x-axis, the ankle ROM has been divided into three equal zones between 10% and 90% of the joint's ROM from plantar to dorsal flexion. The zones are defined as the time windows corresponding to 10–36.6% ROM, 36.6–63.3% ROM, and 63.3–90% ROM. Average rms-EMG per position zone was calculated. These values were normalized by expressing them as a percentage of the peak rms-EMG value of three maximum voluntary contractions (Bar-On et al. 2014c). Inspecting the 3D graphs, it can be seen that in case 1, the muscle reacts only when lengthened at very high velocity. On the other hand, in case 2, activation

occurs already during a slow stretch, with the amount of activation increasing with increased joint ROM. Interestingly, pilot studies have shown that muscles with larger amounts of length-dependent activation also tended to be poorer responders to treatment with botulinum toxin A, both in terms of the reduction in rms-EMG post-treatment as assessed during passive stretch and on gait kinematics (Bar-On et al. 2015). In the presented clinical examples, case 1, with a more velocity-dependent activation pattern, reacted better to treatment than case 2, whose pre-treatment activation pattern showed more length than velocity dependency.

Conclusion

In summary, it important to realize that each of the different components of muscle hyper-resistance needs to be objectively quantified in order to be meaningful to inform clinical decision-making. In children with spastic CP, it has been demonstrated that different contributions to muscle hyper-resistance can be assessed using instrumented methods that collect synchronized electrophysiological and biomechanical signals during passive muscle stretch. This approach offers a objective, reliable and valid alternative to the simple clinical scales. The richness of information that can be collected using instrumented assessments can help create individualized muscle profiles, acknowledging the variability in response to stretch between muscles. Analysis of this variability suggests that it can be used to identify responders, i.e., to fine-tune treatment to those muscles that would benefit most. Furthermore, instrumented assessments can be used to more accurately understand the effects of different kinds of treatments and may encourage the development of new treatments.

Cross-References

- ▶ Cerebral Palsy Gait Pathology
- ▶ Diagnostic Gait Analysis Technique for Cerebral Palsy Gait
- ▶ Focal Spasticity Management in Cerebral Palsy- (Botulinum, Phenol, Neurectomy)
- ▶ Gait Analysis Interpretation in Cerebral Palsy Gait: Developing a Treatment Plan
- ▶ Gross Anatomic Muscle Changes in Cerebral Palsy
- ▶ Measuring Outcomes in Children with Cerebral Palsy
- ▶ Muscle Changes at the Cellular-Fiber Level in Cerebral Palsy

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