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Identification of the neural component of torque during manually-applied spasticity assessments in children with cerebral palsy

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ABSTRACT

Clinical assessment of spasticity is compromised by the difficulty to distinguish neural from non-neural components of increased joint torque. Quantifying the contributions of each of these components is crucial to optimize the selection of anti-spasticity treatments such as botulinum toxin (BTX). The aim of this study was to compare different biomechanical parameters that quantify the neural contribution to ankle joint torque measured during manually-applied passive stretches to the gastrocsoleus in children with spastic cerebral palsy (CP). The gastrocsoleus of 53 children with CP (10.9 ± 3.7 y; females $n = 14$; bilateral/unilateral involvement $n = 28/25$; Gross Motor Functional Classification Score I–IV) and 10 age-matched typically developing (TD) children were assessed using a manually-applied, instrumented spasticity assessment. Joint angle characteristics, root mean square electromyography and joint torque were simultaneously recorded during passive stretches at increasing velocities. From the CP cohort, 10 muscles were re-assessed for between-session reliability and 19 muscles were re-assessed 6 weeks post-BTX. A parameter related to mechanical work, containing both neural and non-neural components, was compared to newly developed parameters that were based on the modeling of passive stiffness and viscosity. The difference between modeled and measured response provided a quantification of the neural component. Both types of parameters were reliable (ICC > 0.95) and distinguished TD from spastic muscles ($p < 0.001$). However, only the newly developed parameters significantly decreased post-BTX ($p = 0.012$). Identifying the neural and non-neural contributions to increased joint torque allows for the development of individually tailored tone management.

1. Introduction

Common clinical assessment of spasticity in children with cerebral palsy (CP) is based on manipulation of the joint to feel the resistance in a passively stretched muscle. In 1954, Tardieu and colleagues emphasized the importance of differentiating between different causes of this increased resistance, or hypertonia [1]. According to the currently prevailing definition of spasticity, an increase in resistance during passive muscle stretch is termed spasticity when there is an accompanying velocity-dependent pathological stretch reflex activation resulting in muscle activity that resists or stops the motion [2]. In the absence of muscle activation, all other excessive increase in resistance during stretch is thought to be caused by passive stiffness and viscosity due to alterations of intra- and extracellular muscle, soft-tissue, and joint structures. Therefore, different components contribute to the feeling of increased resistance in passively stretched muscle: neural and non-neural components. Direct quantification of the different components in a clinical setting is highly relevant allowing for comprehensive tone assessment.

Tone-reducing medication, such as botulinum toxin-A (BTX) targets the neural component by blocking the release of acetylcholine at the cholinergic nerve terminals, which prevents the muscle from contracting. This treatment does not work when the increased resistance is of non-neural origin [3]. Methods to treat passive stiffness include casting, orthotic management, or when fixed contractures arise, orthopedic surgery. Therefore, to

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provide the appropriate treatment to children with CP, it is imperative to differentiate and quantify the components.

The clinical test proposed by Tardieu, and its modifications [4] attempt to differentiate components by comparing the range of motion (ROM) during a slow muscle stretch (R1 angle) to the catch angle during a fast stretch (R2). However, muscle activation during slow stretch has been reported in some spastic muscles [5,6]. Without verifying whether the muscle is inactive, the validity of R1 is uncertain. Additionally, inaccuracies when manually determining R2 have been reported [5] and its reliability is further compromised since the velocity of the fast stretch is not measured. Only an assessment that simultaneously collects electro physiological and biomechanical signals during passive muscle stretch has the potential to differentiate the influence of different components in a valid and reliable way. This approach is used in research settings using motor-driven devices [7–9] that permit standardization of position displacements [7] or of the applied force [9]. For example, using a torque-motor that applied sinusoidal movements at a constant force, Lakie et al. [9] used the peak resonance frequency as a measure of linear stiffness and viscosity in the wrist. On the other hand, by controlling displacement position, De Vlugt et al. [7] modeled the contribution of different non-linear components to measured ankle torque. However, to what extent 'clinically' assessed spasticity can be replicated by a motor-driven device is questionable. Rabita et al. [10] have shown that fewer stretch-reflexes are elicited when spastic muscles are stretched by a robot, than by an examiner.

A different group of evaluation techniques make use of the straight forward clinical application combined with a quantitative approach, and are referred to as instrumented manual techniques [11,12]. These methods replicate a clinical spasticity test by having an examiner apply muscle stretches while simultaneously collecting synchronized electromyography (EMG), kinematics, and/or joint torque. By examining the change in signals with increasing muscle lengthening velocity, parameters that quantify spasticity have been developed and have been shown to be applicable and valid in clinical settings [6,11]. However, in these studies, the velocity-dependent effects from both neural and non-neural components are reflected in the parameters that quantify joint torque. To differentiate between the components, further muscle modeling is required.

The non-linear behavior of passive stiffness and viscosity have been well described in healthy and hemiplegic subjects [7,13]. However, to the best of our knowledge, these models have rarely been applied to data from an instrumented manual spasticity assessment [14]. The aim of this study is to quantify the amount of neural contribution to the joint torque measured during stretches of the gastrocsoleus in children with spastic CP. To achieve this, we model the non-neural components of passive stiffness and viscosity on data collected during stretches at increasing velocities. We then assume that the difference between modeled and measured response represents the neural component. Specifically, we hypothesized that: (1) the neural component will have good between-session reliability; (2) all components will be higher in children with CP than in typically developing (TD) children; and (3) in comparison to a previously-described parameter [11], the neural component will be more sensitive to the effect of BTX treatment.

2. Method

2.1. Participants

Children with spastic CP aged 5–18 years were recruited from the University Hospital Leuven. Exclusion criteria were: presence of ataxia or dystonia; ankles with fixed varus or valgus deformities hindering pure sagittal plane passive ankle motion; cognitive problems that impeded assessment; BTX injections within 6 months prior to first testing; previous lower-limb orthopedic or neuro-surgery. Age-matched TD children acted as a control group. The hospitals' ethical committee approved the protocol (B32220072814) and all children's parents signed an informed consent.

To assess between-session reliability, a subgroup of children with CP underwent a repeated assessment (including replacement of all sensors) after a two hour rest interval in which no treatment was administered. As part of an individually-defined, multilevel treatment, a second subgroup of children with CP were additionally measured 4–8 weeks after intramuscular BTX injections (Allergan, UK) in the gastrocnemius under short anesthesia. After injections, the children underwent lower-leg casting for ±10 days, intensive rehabilitation, and orthotic management (day and night).

In children with unilateral CP, only the affected side was tested. In children with bilateral involvement, if time allowed, both sides were tested. If not, the most involved side was tested as defined by the clinical spasticity scales [4,15].

2.2. Experimental procedure

All assessments were performed by the same trained assessor as detailed in [11]. Joint motion was tracked using two inertial measurement units, joint forces and torques were measured using a 6 dof load-cell, and surface EMG (sEMG) was collected from the lateral gastrocnemius and tibialis anterior (Fig. 1). The subjects were asked to remain relaxed throughout the measurement. The ankle joint was moved through the full ROM, at low velocity during 5 s, at a medium velocity (1 s), and finally at high velocity, performed as fast as possible. At each velocity, four repetitions were carried out with an interval of 7 s.

2.3. Data analysis

2.3.1. Data processing

Data visualization and analyses were performed in MATLAB©. To estimate joint angles, a Kalman smoother [16] was applied on the inertial measurement unit data. Average maximum angular velocity (V_{max}) was calculated per velocity trial. Using measured segment-lengths and moment-arms, the net internal ankle joint torque was calculated from the measured external forces and moments as outlined in [11]. The EMG onset in the lateral

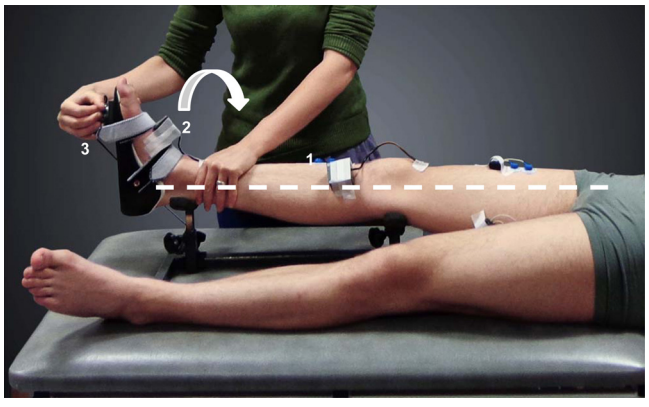

Fig. 1. Test starting position and direction of stretch (white arrow). Muscle activity was measured with surface electromyography (1); joint-angle characteristics with inertial measurement units (2); and torque using a force-sensor (3) attached to a foot orthosis. The mass of the foot plus orthosis is considered negligible. The resulting ankle joint torque was calculated after compensation for movement in non-perpendicular directions and the moments exerted on the handle (see [11] for more detail).