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Quantifying the velocity-dependent muscle response during gait of children with Cerebral Palsy

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ABSTRACT

A new method is introduced quantifying the velocity-dependent muscle response during gait in spastic muscles of children with Cerebral Palsy. The velocity-dependent muscle activation Index is calculated during a 3-dimensional gait analysis using segment angular velocity and the Instantaneous Mean Frequency calculated from surface electromyography. Typical developed children (n = 11) and children with hemiplegia (n = 11) aging from 8 to 19 years participated in the study. The rectus femoris and the medial gastrocnemius were assessed by calculating the velocity dependent muscle activation Index and the modified Ashworth Scale. Greater velocity-dependent muscle activation Index values for both medial gastrocnemius and rectus femoris muscles were associated with greater Ashworth Scale. Post hoc analysis revealed significant lower velocity-dependent muscle activation Index means in the Typical developed group compared with Ashworth Scale scores of 1, 2, 3, and 5. In addition, velocity-dependent muscle activation Index for Ashworth Scale 0, 1, and 2 were significantly lower than for Ashworth Scale 3 and 5. The velocity dependent muscle activation Index showed negative low correlation with walking speed and cadence. Findings show that spastic muscles can be quantified during dynamic functional task such as walking. Future studies should investigate the reliability of the velocity-dependent muscle activation Index that may be used for the assessment of spasticity management such as Botulinum toxin A interventions.

1. Introduction

Spasticity is the predominant type of Cerebral Palsy (CP), comprising around 80% of all CP (Access Economics, 2008), and its management (physiotherapy, orthotics, Botulinum toxin, dorsal rhizotomy and intrathecal baclofen) is the key focus of many health clinics across the world. The different management options vary dramatically in their cost to the families and the community, the potential complications for the child, and the involvement related to rehabilitation for the child and their family. A precise physical assessment of spasticity is therefore paramount to assist in clinical decision making and to evaluate the outcome of the various management options for children with spasticity. The relative nonexistence of treatment or therapy modalities available to reduce spasticity in the past may have explained the limited development of methods for its measurement. However, given the recent advances in treatments for spasticity, such as BoNT-A injections (Baker et al., 2002; Bjornson et al., 2007), there is now considerable incentive to develop advanced methods to measure spasticity to evaluate the outcomes of these interventions.

Clinical accepted, spasticity is characterised as 'velocity-dependent increase of tonic stretch reflexes with exaggerated tendon jerk' (Lance, 1980). This suggests that the velocity of muscle-tendon stretch and the muscle activation response to stretching are key components in the assessment of spasticity. A stretch response is a normal protective mechanism that occurs when a resting muscle is lengthened rapidly or forcefully. By definition patients with spastic CP have increased velocity-dependent resistance to passive stretch that may be focal (a single joint) or generalized (multiple joints) that interferes with active motion such as their gait with observed reduced gait velocity proportion to their level of neurological involvement (Abel and Damiano, 1996; Damiano and Abel, 1996), increased cadence, and reduced angular velocity at the hip, knee, and ankle (Granata et al., 2000). Levin and Feldman (Levin and Feldman, 1994) explained that children with CP are constrained in movement speed by spasticity preventing them from achieving faster velocities so as not to elicit a stretch response. In other words, in a 'spastic' person the threshold for stretch response is low and

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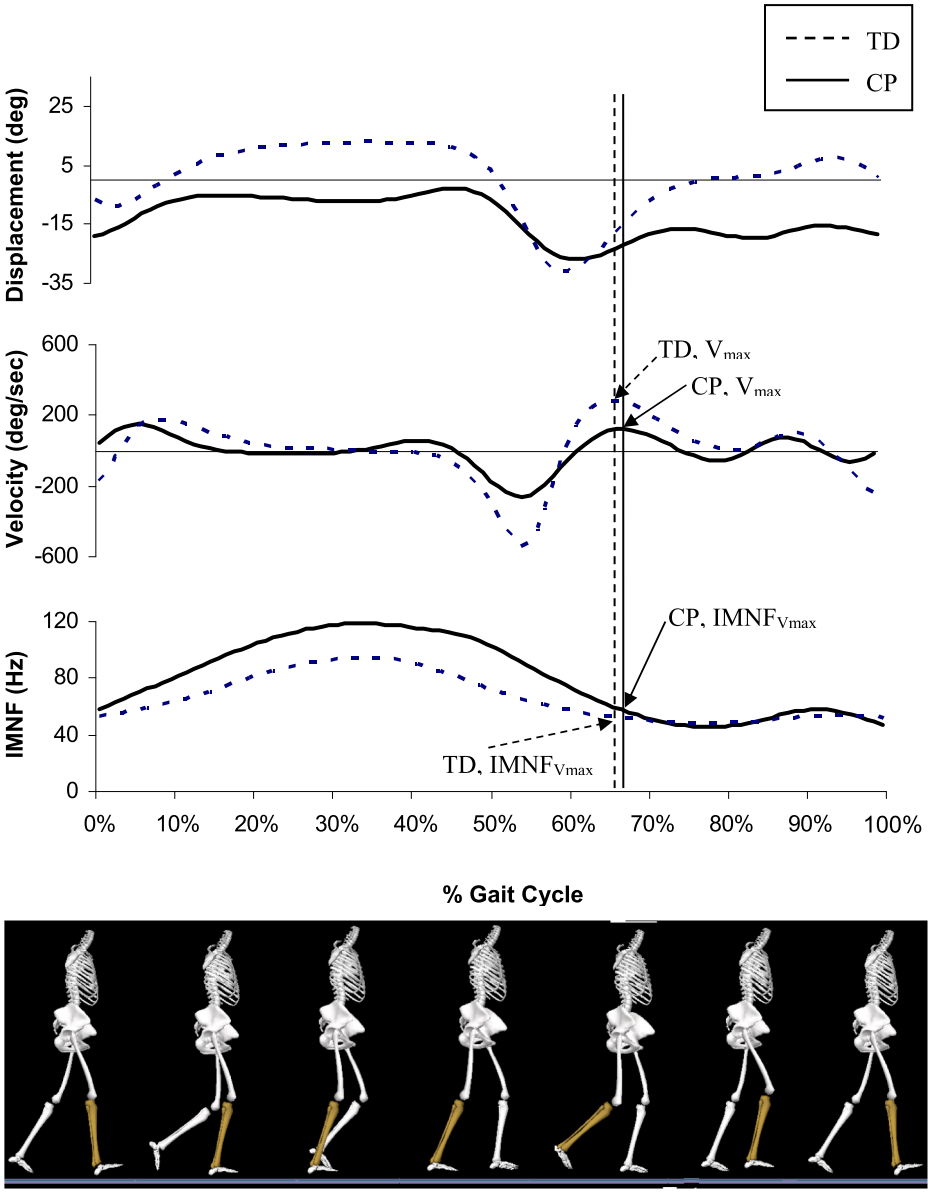

Fig. 1. Determination of the VDMA Index for the medial gastrocnemius using joint kinematics and muscle activation output in typical developing child (dashed) and CP (solid). VDMA Index was defined as the ratio of the peak joint angular velocity (V_{max}) measured from the initial swing phase and the instantaneous mean frequency ($IMNF_{Vmax}$) value calculated from the sEMG signal at the time where the peak joint angular velocity occurred.

can be elicited much more readily, even on passive examination. Assessing the presents and measuring the level of spasticity is, therefore, a primary clinical importance in the management of children with CP.

There has been variety of approaches to the measurement of spasticity. The most clinical common spasticity assessments are based on 'passive stretch' i.e. the stretch is performed by the clinician, including the modified Ashworth Scale (AS) and the Tardieu Scale (TS). The AS and TS are descriptive ordinal scales most commonly used across the pediatric disabled population (Ashworth, 1964; Bohannon and Smith, 1987; Tardieu et al., 1954). Although these measures provide broad indication on the level of spasticity, assessments of spasticity using these scales, however, should be interpreted with great caution for the following three reasons. First, the AS and TS are restricted to the application of passive movements in a resting position, which alone has been shown to be insufficient as an assessment method for spasticity (Fleuren et al., 2008), especially when exploring its effect on dynamic movement such as in gait. It is excepted that the evaluation of spastic locomotor disorders needs to incorporate voluntary movement and be performed during locomotion (Crenna, 1998; Fleuren et al., 2008; Fung and Barbeau, 1989). Secondly, AS and TS are 'subjective' assessments with test–retest results vary widely with an intraclass correlation coefficient ranging 0.21–0.72 for AS and 0.38–0.90 for TS in the calf muscle when assessed by 6 assessors (Fosang et al., 2003). Finally, the AS have been described as simply measuring resistance to passive movement which does not address the velocity-dependent aspect of the phenomenon as described earlier (Pandyan et al., 1999; Scholtes et al., 2006).

The need for 'objective' approach to the measurement of spasticity stemmed the methodology of measuring muscle excitability using surface electromyography (sEMG) in relation to the segment angular movement of the elbow (Calota et al., 2008) and the knee, (Tuzson et al., 2003). Calota et al. (2008) explored the Biceps brachii sEMG signals and elbow displacement during elbow stretches applied at different velocities. Velocity-dependent dynamic stretch reflex thresholds (angle where sEMG signal increased in the biceps for a given velocity of stretch) were recorded. These values were used to compute the tonic

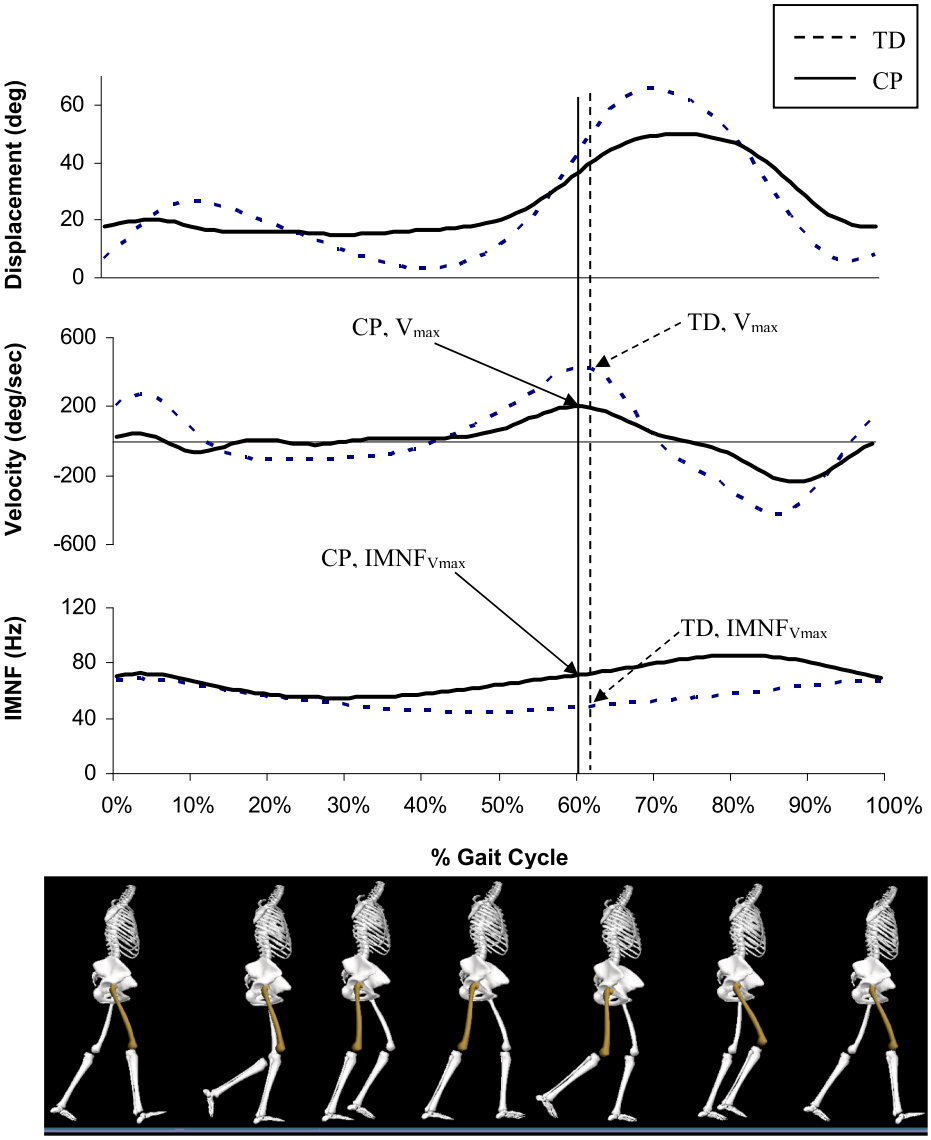

Fig. 2. Determination of the VDMA Index for the rectus femoris using joint kinemtics and muscle activation output in typical developing child (dashed) and CP (solid). VDMA Index was defined as the ratio of the peak joint angular velocity (V_{max}) measured from the initial swing phase and the instantaneous mean frequency ($IMNF_{Vmax}$) value calculated from the sEMG signal at the time where the peak joint angular velocity occurred.

stretch reflex threshold (TSRT) but they were found not to have significant correlation with AS scores. However, the authors argued that TSRT may be a more representative measure for subjects with moderate to high spasticity. Similar technique, the '*Spastic Velocity Threshold*', was suggested by Tuzson et al. (2003) when using sEMG signals at the knee extensors and flexors. The '*Spastic Velocity Threshold*' is quantified when subjects are seated and the knee passively moves at a range of angular velocities (30˚/s and then increased to 60˚, 90˚, 120˚, 150˚, 180˚, 210˚, 240˚, and finally 270˚/s) using isokinetic dynamometer. Spasticity is then identified by the lowest speed at which every trial show a consistent sEMG response indicated by clear, repeatable, sudden increase in the slope of the processed sEMG. A moderate correlation was found between '*spastic threshold velocity*' and Gross Motor Function Measure score ($r = 0.59$, $p < .047$), but the authors acknowledged that because the spastic response is elicited by muscle stretch, a more accurate method to compare the speed of muscle lengthening in the isokinetic dynamometer would be with matched speed of muscle lengthening during gait, which infer that a better technique will be in measuring spasticity while the person is walking.

The need to evaluate muscle spasticity during human locomotion in 'active stretch', as oppose to the above mentioned 'passive stretch' methods, was previously recognised in the 1990 (Fung and Barbeau, 1989; Crenna, 1999). Fung and Barbeau (1989) previously suggested, the use of the '*EMG profile index*' that is defined as the ratio of integrated envelope sEMG activity in the 'off' bin of the normalised gait cycle to that in the 'on' bin. This method was shown to be significantly lower in healthy compare to spastic muscle during walking. In addition, the '*EMG profile index*' was reported to be sensitive enough to show significant intervention effect of cyproheptadine to a spinal cord injury individual (Fung and Barbeau, 1989). Major shortcomings to the method, however, includes: (1) identification of the 'off' bins phase is 'subjective' and difficult in spastic muscle, (2) it does not provide a rating or continues scale of spasticity, and (3) it does not account for the velocity of the segment/muscle, which according to the definition (Lance, 1980) is a fundamental component when measuring spasticity.

Another 'objective' method to quantify spasticity during locomotion was introduced with the attempt to combine kinematics and muscle activation output in assessing the spasticity of the hamstring muscles during walking (Crenna, 1999). The '*Lengthening Velocity Threshold*' (LVT) was proposed and defined as the lengthening velocity of the

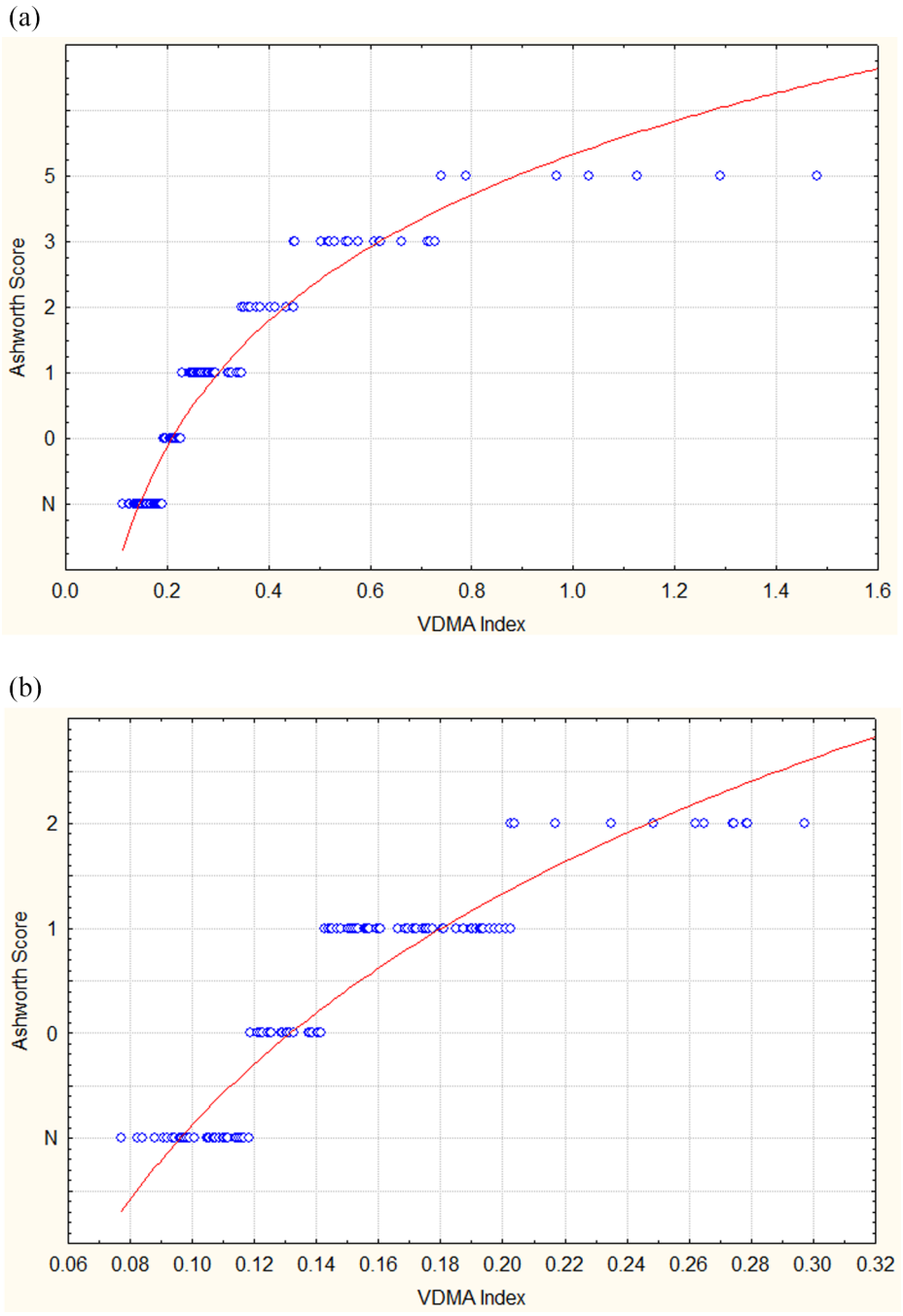

Fig. 3. Quantile-quantile plot for both MG (a) and RF (b) comparing the two probability distributions of VDMA Index and Ashworth Scores. Ashworth N represent scores for the typical develop children.

muscle at which the lengthening contraction burst, measured by sEMG, commence. Greater LVT values of knee flexors were found in healthy participant when compared to spastic diaplegia individuals. Although this method advantage is the coupling of velocity and muscle activation components, major limitations in the proposed method include: (1) the degree/level of spasticity cannot be acknowledged by the LVT values, and (2) the identification of the 'on' time of muscle activation in the lengthening period, indicated by sEMG, is 'subjective' and much problematic in spastic muscles that are activated during most of the gait cycle.

Overall the studies have revealed the need for an 'objective' with 'active stretch' method for scaling the level of spasticity during human locomotion. Today there are more advanced techniques of sEMG processing methods exploring the differences in signal amplitude and frequency rather than 'on-off' temporal measurements. Time–frequency characteristics of muscle activation quantified by Instantaneous Mean Frequency (IMNF) have been suggested to be more reflective of the resultant changes in gait kinematics (Lauer et al., 2010, Lauer et al., 2007b), and more sensitive to change in muscle function after hamstring surgery in children with CP (Lauer et al., 2007a). In the proposed study we were interested exploring the inclusion of the IMNF processing method in the analysis of muscle spasticity.

Here we propose a new approach to the measurement of velocity-dependent muscle response during walking for identifying the level of spasticity based on the rational that spasticity is quantified as the relationship between muscle activation and segmental velocity when the muscle is stretched during functional voluntary movement i.e 'active stretch'. The aim of the study was to evaluate a new method that couple

Table 1
Means, Standard deviation (in round brackets), and number of gait trials (in square brackets) of the calculated VDMA Index for the different modified Ashworth Scale levels (AS) and for the typical development (TD) participants. Results are presented for both the for the Medial gastrocnemius (MG) and Rectus femoris (RF) muscles.

	VDMA index	
	MG	RF
TD	0.18 (0.05) [44]†	0.13 (0.01) [44]†
AS-0	0.22 (0.04) [16]‡	0.16 (0.01) [24]†
AS-1	0.29 (0.09) [35]†	0.15 (0.01) [50]‡
AS-2	0.29 (0.10) [12]*	0.21 (0.01) [12]†,‡
AS-3	0.59 (0.15) [16]†,‡,*	
AS-5	1.00 (0.32) [7]†,‡,*	

Abbreviations: AS, modified Ashworth Score; MG, medial gastrocnemius; RF, Rectus Femoris.
† $p < .01$.
‡ $p < .01$.
* $p < .01$.

Table 2
Correlation coefficients and Significance of VDMA Index, modified Ashworth Scale (AS), walking velocity, and cadence for the Medium Gastrocnemius (MG) muscle.

Variables (MG)	AS	Walking velocity (% height/sec)	Cadence (steps/min)
VDMA index	r = 0.721 p < .01	−0.240 p < .01	NS
AS		NS	NS
Walking velocity			r = 0.493 p < .001

Table 3
Correlation coefficients and Significance of VDMA Index, modified Ashworth Scale (AS), walking velocity, and cadence for the Rectus Femoris (RF) muscle.

Variables (RF)	AS	Walking velocity (% height/sec)	Cadence (steps/min)
VDMA index	r = 0.334 p < .001	−0.483 p < .001	−0.543 p < .001
AS		−0.201 p < .05	NS
Walking velocity			r = 0.493 P < .001

gait kinematics and muscle activation quantified by IMNF to measure the velocity-dependent muscle activation patterns in both the rectus femoris and medial gastrocnemius during walking, and to demonstrate the relationship between the new method and AS.

2. Methods

2.1. Subjects

A total of 22 children aged between 8 and 19 years participated in this study. Eleven children (age = 14.1 ± 3.7 years; height = 159.1 ± 15.4 cm; weight = 57.3 ± 17.6 kg) with a diagnosis of hemiplegic CP of Gross Motor Function Classification System (GMFCS) levels I and II, and 11 typically developing (TD) children (age = 12.5 ± 3.1 years; height = 149.8 ± 15.0 cm; weight = 42.1 ± 12.0 kg) who have not experienced any significant musculoskeletal disorders or walking difficulties. Participants gave written consent for the use of their data for research purposes, in accordance with the local ethical committee requirements.

2.2. Setup

Testing was carried out in a clinical gait analysis laboratory with a 10 m level walkway. Three-dimensional gait analysis of the lower limb was recorded using 6 VICON 460 motion system cameras (Oxford Metrics Ltd., UK) sampled at 120 Hz. The Helen Hayes Marker set (Kadaba et al., 1990) was used for marker placement and a knee alignment device was used to define the knee joint axes. Surface electromyography from the rectus femoris (RF) and medial gastrocnemius (MG) were collected on both sides, using 8 channel system (Zebris, Tubingen, Germany; amplifiers from Biovision, Wehrheim, Germany). Electromyography signals were pre-amplified, band-pass filtered (10–700 Hz) at a sampling rate of 2520 Hz. After shaving and cleaning the skin, bipolar Ag/AgCl surface electrode pairs with a diameter of 10 mm and an inter-electrode spacing of 22 mm were placed. The SENIAM (Hermens and Freriks, 1999) recommendations for surface EMG (sEMG) were followed for electrode placement. The ground electrode was placed overlying the tibial tuberosity.

2.3. Protocol

Participants were initially assessed for spasticity level at the RF and MG using AS. The AS was recorded from the knee extensors and calf dorsiflexors, and the complete GMFCS assessment was performed by trained physiotherapists. The AS (Bohannon and Smith, 1987) is a 6-point ordinal scale, ranging from 0 (no increase in tonus) to 5 (rigid limb), based on the subjective impression of the examiner of the resistance felt to passive stretch. Following the spasticity assessment, participants were asked to walk barefoot along the 10 m pathway at their self-selected speed. After the 4 familiarization walking trials, 4 testing walking trials were recorded for further analysis and checked for consistency. During the 4 testing walking trials kinematic and sEMG data were collected simultaneously and were later processed and expressed as percentage (0–100%) of the gait cycle.

2.4. Data processing

For each walking trial the knee and ankle joints angular velocity with the RF and MG muscle IMNF activation patterns were calculated and expressed as percentage (0–100%) of the gait cycle. Using the joints angular velocity and muscle IMNF patterns the velocity-dependent muscle activation (VDMA) Index for the RF and MG muscle groups of both left and right side for the CP subjects and only the right side for the TD subjects were calculated. Following are the steps used to calculate the VDMA Index:

2.4.1. Identifying the peak joint angular velocity

Angular changes of the ankle and knee joints during walking were estimated by the motion analysis system. The angular velocity was calculated as the first derivative with respect to time and was normalised in time (100%) over a single gait cycle. Using the toe marker, the foot-off event was identified. Short after foot-off event, in which the muscle is lengthening (Jonkers et al., 2006; Van der Krogt et al., 2008), the peak joint angular velocity for both MG and RF muscles was measured (see Figs. 1 and 2, respectively). This peak joint angular velocity was used to calculate the VDMA Index.

2.4.2. Calculation of the instantaneous mean frequency (IMNF)

Using Matlab software (The MathWorks Inc. Natrick, MA, USA) the sEMG were first bandpass filtered (20–500 Hz). The IMNF was then calculated as describe earlier (Lauer et al., 2007b; Tirosh et al., 2013). In brief, the recorded sEMG signal was normalised to 100% of each gait cycle. The sEMG signal was then analysed with the continues wavelet transform (CWT) with the Morlet wavelet as the mother function, using the Time-Frequency Toolbox (Auger et al., 1996). The CWT technique preserve both time and frequency characteristics of the sEMG signal

which is a scalogram representation in a three-dimensional graph where time (% gait cycle) is along the x-axis, frequency (scale) is along the y-axis, and power (magnitude) is on the z-axis. By calculating the mean frequency at each time interval the IMNF curve across the gait cycle is generated which has been used in the past as a representation of time–frequency information to assess the degree of motor impairment in CP (Lauer et al., 2005). The IMNF at the time of the identified peak joint angular velocity was used to calculate the VDMA Index (see Figs. 1 and 2).

2.4.3. Calculating the velocity-dependent muscle activation (VDMA) index

Velocity-dependent muscle activation Index was defined as the ratio of the peak joint angular velocity measured from the initial swing phase and the IMNF value calculated from the sEMG signal at the time where the peak joint angular velocity occurred, Eq. (1).

$$VDMA_{indx} = IMNF_{Vmax}/V_{max} \quad (1)$$

where V_{max} is the peak joint angular velocity after foot-off, $IMNF_{Vmax}$ is the instantaneous mean frequency at time of V_{max}, and $VDMA_{indx}$ is the calculated VDMA Index. The ankle and the knee joints sagittal angular velocity was used to calculate the $VDMA_{indx}$ of the MG and RF, respectively. Example on how to calculate the $VDMA_{indx}$ is illustrated in Figs. 1 and 2.

The rational was having the segment to freely move without ground reaction forces, as during AS test, when muscle is in lengthening contraction reaching its maximum angular velocity. Angular velocity greater then 30 deg/s was reported to demonstrate sufficient stretch response in CP individuals (Damiano et al., 2006). The MG muscle in TD and CP children reported to have stretch phase from initial swing throughout swing (Van der Krogt et al., 2008). The RF muscle continues to lengthen during initial swing until reaching its maximal length, after which the muscle shortens during the remainder of swing phase (Jonkers et al., 2006).

2.5. Data analysis

Data were analysed to identify: (a) differences between the VDMA Index values obtained at different AS, (b) the relationship between AS and VDMA Index, cadence, and normalised walking speed (walking speed/participant height), (c) inter-trial reliability of the calculated VDMA Index values between the 4 walking trials for each participant, and d) the relationship between VDMA Index, cadence, and normalised walking speed. In total 132 VDMA Index values were calculated from $11 \times 4 \times 1$ (TD participants × walking trials × leg side) and $11 \times 4 \times 2$ (CP participants × walking trials × leg side i.e. left and right). Six (6) AS categories were used including; 0, 1, 2, 3, 5, and N. The N score was given to all TD participants and AS 4 was not present in the CP participants. All statistical tests were conducted at a 0.05 alpha level.

To explore significant differences between the VDMA Index values obtained (dependent variable) at different AS (independent variable) a one way Analysis of Variance (ANOVA, 132×6) was performed for each muscle (RF and MG) with subsequent post hoc multiple comparisons with the Bonferroni procedure used.

To identify the relationship between AS and VDMA Index, cadence, and normalised walking speed the Spearman rank order correlation was performed for each muscle (RF and MG).

To explore inter-trial reliability a repeated measure ANOVA with walking trials as the repeated measure (33 legs × 4 walking trial) for each muscle (RF and MG) was performed with subsequent post hoc multiple comparisons with the Bonferroni procedure used.

To explore the relationship between VDMA Index, cadence, and normalised walking speed the Pearson correlation coefficients were calculated for each muscle (RF and MG).

3. Results

In the TD group 44 VDMA Index values were calculated for MG (11 subjects × 4 right side walking trials), and 44 VDMA Index values for RF. In the CP group only one participant had 3 trials due to malfunction of the EMG system. Thus, for the 11 CP participants in all walking trials, 86 (43 for each side, right and left) VDMA Index values were calculated for MG and 86 VDMA Index values for RF. Modified Ashworth test score of 4 for MG and scores of 3, 4, and 5 for RF muscles were not recorded for any of the CP participants.

Fig. 3 represents the quantile-quantile plot for both MG and RF comparing the two distributions of VDMA Index and AS. For the MG muscle, the VDMA Index ranges found were 0.15–0.21 (n = 44), 0.16–0.27 (n = 16), 0.25–0.32 (n = 35), 0.22–0.34 (n = 12), 0.54–0.64 (n = 16), and 0.92–1.08 (n = 7) for AS scores of N, 0, 1, 2, 3, and 5, respectively. For the RF muscle, the VDMA Index ranges found were 0.11–0.13 (n = 44), 0.14–0.17 (n = 24), 0.13–0.16 (n = 50), and 0.16–0.25 (n = 12) for AS scores of N, 0, 1, and 2, respectively.

Table 1 summarises the VDMA Index means and standard deviation for both MG and RF muscle groups across the recorded AS. Generally, greater VDMA Index values for both MG and RF muscles were associated with greater AS ($F(5,124) = 92.11$ and $F(3,126) = 11.56$, $p < .05$ for MG and RF muscles, respectively). Post hoc analysis for the MG muscle revealed significant lower VDMA Index means in the TD group compared with AS-1, AS-3, and AS-5 (0.18, 0.29, 0.59, 1.00, respectively, $p < .05$). Furthermore, AS-0, AS-1, and AS-2 were significantly lower than AS-3 and AS-5 (0.22, 0.29, 0.29, 0.59, 1.00, respectively, $p < .05$), and AS-3 was significantly lower than AS-5 (0.59 and 1.00, respectively, $p < .05$). Similarly for the RF muscle, VDMA Index means in the TD group were significantly lower compared to AS-0 and AS-2 (0.13, 0.16, 0.21, respectively, $p < .05$). The inter-trial reliability repeated measures ANOVA analysis did not found significant differences between individual 4 trials for both MG ($F(3,96) = 1.25$, $p = 0.29$) and RF ($F(3,96) = 1.24$, $p = 0.30$) muscles.

Tables 2 and 3 present the relationship between AS, VDMA Index, cadence, and normalised walking speed. Spearman correlation analysis showed significant correlation between VDMA Index and AS for both MG and RF ($r = 0.72$ and $r = 0.33$, $p < .01$, respectively). Pearson correlation showed negative low correlation between VDMA Index and normalised walking speed (-0.240 and -0.483 for MG and RF muscles, respectively).

4. Discussion

In this study a new method was introduced to facilitate the measurement of the degree of velocity-dependent muscle response in children with CP during a routinely clinical 3-dimensional gait analysis. The proposed VDMA Index overcomes limitations of other previous attempts by integrating both segment velocity and muscle excitation, which are the key factors defining “Spasticity” (Lance, 1980). Furthermore, the VDMA Index is advantageous as it is quantified from a dynamic and functional movement, such as walking, and provides a continuous numerical scale indicating the degree of impairment.

When compared to AS that is considered to be the most common spasticity clinical evaluation method, the VDMA Index was found to have significant relationship showing greater values in muscles with greater spasticity. This relationship was found to be significant for both MG and RF with high correlation with ankle plantarflexors ($r = 0.72$) but low with knee extensors ($r = 0.33$). Our results, however, differ from previous findings (Tuzson et al., 2003; Jobin and Levin, 2000; Engsberg et al., 1996). Tuzson et al. (2003), for example reported non-significant correlation between their ‘*spastic threshold velocity*’ score and AS, while Engsberg et al. (1996) reporting only low correlation ($r = 0.28$). Today, the AS is the most commonly clinical used measurement to assess spasticity despite its being a semi quantitative subjective test with limited validity when assessing the velocity-dependent

components of spastic function (Damiano et al., 2002; Pandyan et al., 2001). It is thus questionable whether the AS can truly represent muscle spasticity and can represent the "Gold Standard". The use of 3-dimentional analysis and sEMG data in planning complex orthopaedic surgery for children with cerebral palsy is now well established and greatly used (DeLuca et al., 1998; Graham et al., 2005; Ounpuu et al., 2002; Rodda et al., 2006). Surgical planning without gait analysis leads to different recommendations to (Cook et al., 2003; Kay et al., 2000) and such surgery has better outcomes (Chang et al., 2006; Lee et al., 1992). Usage of kinematic data with sEMG to assess spasticity is now, therefore, more accessible and may be a better option in assessing the level of spasticity than AS.

In less spastic muscles indicated by low AS values (between 0 and 2), differences between the associated VDMA Index were less apparent. There may be two main reasons for this. The first may be related to the fact that the AS is a subjective rating measurement and the intervals between the ranks are subjectively not equal. Furthermore, the AS rating was previously reported to vary in one degree between assessors (Bohannon and Smith, 1987). Thus, low VDMA Index value may have been associated with AS of 2 instead of 1 due to assessor error. The second reason may be related to the small number of cases of AS 0, 1, and 2. For the MG muscle there were 4 individuals with AS score of 0 (4 participants × 4 trials = 16 cases) and 3 individuals with AS score of 2 (3 participants × 4 trials = 12 cases). The small sample size in these AS levels increase the probability of type II error and was notable (ß = 0.46 and 0.62 for 16 and 12 cases, respectively), indicating that a larger sample size is necessary to draw conclusions of the differences between VDMA Index values at the low AS scores. A limitation that needs further exploration in future studies.

The effect of walking speed on muscle activation has been previously documented. Using linear envelope curves of the amplitude and time measurements it was shown that faster walking increased amplitude but the shape remained essentially unchanged. Tirosh et al. (2013) also reported that the linear envelope waveforms were affected by speed with greater amplitudes at increased speed, but in the time-frequency domain the IMNF waveforms did not support significant speed effects on the frequency component. Tirosh et al. (2013) finding encouraged the use of the time-frequency IMNF processing in the proposed VDMA Index method to reduce the effect of walking speed on VDMA calculation. The IMNF method have been previously suggested to be more reflective of the resultant changes in gait kinematics (Lauer et al., 2007a). Spectral content of the sEMG can be related to recruitment of additional motor units that include fast twitch muscle fibers to generate increased force at higher mean firing frequency, and synchronizing the firing rate of the motor units currently in use reducing the frequency spectrum of the sEMG signal (Ricard et al., 2005; Wakeling, 2009). Thus the IMNF might represent motor unit recruitment and/or motor unit firing frequency modulation, whereas the increase in mean frequency of the power spectrum might represent the additional recruitment of superficial high threshold motor units (Moritani and Muro, 1987). In this study correlation between walking speed, cadence and VDMA Index values for the MG muscle were found to be small ($r = -0.24$). Crenna (1999) found significant but high correlation of their '*velocity threshold*' method with walking speed ($r = 0.71$). Difference in findings between this study and Crenna (1999) study may relate to the different sEMG processing methods used i.e. time domain (linear envelope) and time-frequency domain (IMNF). This trend, however, was not presented in the RF muscle. The RF showed significant medium correlation with walking speed ($r = -0.48$). This may suggest that the VDMA Index is muscle specific and/or that spastic muscles respond differently to changes in walking speed. The former may be supported by the findings of lower VDMA Index values for the RF compare to MG muscle at the same AS level. Further investigation, however, is needed to confirm this.

The present study proposed a new methodological approach which is relevant for the assessment of spastic muscle behaviour during functional task such as walking. It does in fact takes in account both the velocity of the segment and muscle activation pattern that constitute the definition of "Spasticity" as defined by Lance (1980). The proposed VDMA method was found to be consistent between walking trials showing its inter-trial reliability, but some limitations exist. Its major limitation relates to the fact that velocity was calculated using segment kinematics and not muscle-tendon kinematics, which may have affected the results primarily with the RF muscle. While ankle and knee angular velocities were reported to relate to spasticity (Damiano et al., 2006; Granata et al., 2000), not using muscle-tendon velocity might had an impact on the factors discussed earlier, especially when the examined muscle is a double jointed muscle. It may be more appropriate, in the future, to investigate the use of the lengthening velocity of the muscle-tendon to calculate the VDMA Index values. It is clear that more studies are needed to explore the use of VDMA index in clinical setting. Overall, the proposed VDMA Index showed encouraging results necessitate further investigation.

Declaration of Competing Interest

There were no conflicts of interest in the preparation of the attached manuscript submitted for publication in *Journal of Electromyography and Kinesiology* by Tirosh et al. "Quantifying the velocity-dependent muscle response during gait of children with Cerebral Palsy."; including financial and personal relationships with other people or organisation that would have influenced or biased the work.

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