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Biometric Characterization of the Stretch Response in Children with Cerebral Palsy and its Relation with the Tardieu Scale

BEV 3901 Master's thesis

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Abstract

Objective The main purpose of this study was to determine to what extent resistance to passive stretch, obtained as in the Tardieu test (TS), can evaluate spasticity in the elbow flexors of children with cerebral palsy (CP). Secondary objectives were to evaluate if the TS reflects spasticity severity and if there were any differences in stretch response between the CP group and controls.

Methods Fifteen children and adolescents with CP and 15 healthy non-spastic controls were included in the study. All CP participants were affected in the upper extremities, however with different severity of motor impairments. Both groups performed passive elbow extensions at slow and fast velocities, with simultaneous recordings of the Tardieu Scale and biometric (velocity, acceleration and electromyographic activity) measurements. A maximum voluntary isometric contraction was also completed.

Results There was a good agreement (about 65 %) between detected catch with TS (sudden increase in felt resistance) and biometric (EMG burst followed by acceleration change) recordings during the passive elbow extension. However, the passive extension resulted in variable maximum extension velocities in CP participants and at very high velocities a catch was also detected in controls. When high velocity catch was defined as a normal stretch response, the TS overestimated CP participants with abnormal catch in relation to biometrically defined abnormal response. The TS reflected resistance to passive movement in the CP group, but did not separate participants with high muscle activity amplitude from participants with low muscle activity amplitude. Compared to controls, the CP group generally developed higher peak muscle activity in combination with lower maximum extension velocity. However, without knowledge of extension velocity, there was similar muscle activity response in the two groups.

Conclusion In all, resistance to passive stretch, obtained as in the TS, seems to provide a useful evaluation of spasticity in CP children. The results underlines that the TS test protocol should include a maximum extension velocity limit in which a normal stretch reflex does not occur. Further, TS reflect spasticity severity to a limited degree, but resulted in a difference in stretch response in CP and control groups.

Keywords Spasticity, cerebral palsy, Tardieu Scale, biometric measures, electromyography

Contents

1.0 Introduction

Cerebral palsy (CP) is a collection of non- progressive syndromes of posture and motor impairment that results from damage to the developing central nervous system prepartum, intrapartum or up to two years postpartum. With prevalence from 1.5 to 3 children per 1000 live births (Cans et al., 2000), CP is the most common cause of severe physical disability in childhood (Hutton et al., 1994). Spastic CP is one of the main subtypes of CP and is characterized by at least two of the following characteristics; abnormal posture and movement pattern, increased muscle tone and/or pathological/increased reflexes (Cans et al., 2000).

Spasticity is a disordered sensory-motor control, part of the upper motor neuron syndrome (UMNS), characterized by intermittent or sustained involuntary muscle activation (Pandyan et al., 2005). Spasticity is also often defined as a velocity-dependent increase in the tonic stretch reflex. So when a stretch is done slowly the tone feels close to normal, but the resistance increases with increasing velocity of the stretch (Sheean, 2002). Moreover, the velocity threshold for evoking a stretch reflex is lower in spastic compared to normal muscles (Levin & Feldman, 1994).

During development, the functional abilities of a child with spastic paresis commonly deteriorate and spasticity is claimed to be related to this functional decline (Scholtes et al., 2006). Spasticity is thought to be one of the main causes of impaired hand function in children with CP (Law et al., 2008) and may also be related to musculoskeletal complications like pain, weakness and contractures. Therefore accurate identification of spasticity is important to give the right individual treatment and to reduce the negative effects of spasticity (Voerman et al., 2005). Several methods have been used to evaluate spasticity (Biering-Sørensen et al., 2006) and these can be divided into clinical and biometric methods.

There are two common clinical methods, the Ashworth scales and the Tardieu scales. The Ashworth scales (AS) are subjective scales where the clinician states the felt resistance to movement on a five (AS) or six (Modified AS) point scale while extending a joint passively (Scholtes et al., 2006). Both validity (Spearman Correlation: 0.05- 0.73) (Fleuren et al. 2010) and reliability (ICC: 0.31- 0.82, Cohen's Kappa: 0.14- 0.62) for this scale are though variable (Fleuren et al. 2010; Mehrholz et al., 2005; Mutlu et al., 2008) and it is not consistent with all

aspects of spasticity (Scholtes et al., 2006). The Tardieu Scales (TS) has been suggested to be more suitable than AS for assessing spasticity (Alhusaini et al., 2010; Patrick & Ada, 2006; Scholtes et al., 2006). During the TS, the joints are passively stretched at fast and slow velocities and the intensity and duration of during the fast stretch is subjectively rated on a five point scale together with the joint angle where this reaction is first felt (Gracies et al., 2010). TS is stated to be related to the velocity dependency of spasticity, due to the use of both slow and fast velocities (Scholtes et al., 2006). The TS has shown variable reliability (Cohen's Kappa: 0.29- 0.87) (Mehrholz et al., 2005). However, for the elbow joint of children with CP, the TS has shown a good intra-rater (Cohen's Kappa: 0.75-0.78, Agreement: 80-97%) and moderate to good inter-rater reliability (Cohen's Kappa: 0.48- 0.51, Agreement: 79-90%) (Gracies et al., 2010; Mehrholz et al., 2005). The ability of TS to identify the severity of spasticity in children with CP has been stated to be inadequate (Alhusaini et al., 2010). However, there are still too few studies examining the validity of TS (Haugh et al., 2006).

Because the quality of the subjective methods has been questioned, there has been a growth of biometric methods, including both neurophysiologic and biomechanical instruments (Johnson, 2002). Biomechanical methods use force transducers to quantify resistance to passive movement and goniometers to register angular displacement (Pandyan et al., 2001) or a dynamometer to control velocity and measure velocity, angle and torque during a passive stretch (Pierce et al., 2006), hence it gives an objective indirect measurement of spasticity. Neurophysiologic methods, on the other hand, study the muscle response to an external passive stretch of the joint using electromyography (EMG) and are therefore the most direct quantification of spasticity (Biering-Sørensen et al., 2006). Although, the resistance to passive movement is used as a measure of spasticity in clinical practice, there are studies questioning the relationship between a sudden increase in resistance and a sudden increase in muscle activity in fast passive stretch (Malhotra et al., 2008; Pandyan et al., 2005).

Further, there have been recommendations of combining the biomechanical methods with EMG for a better assessment of spasticity (Malhotra et al., 2008; Voerman et al. 2005; Wood et al., 2005). By using EMG together with biomechanical instruments it would be possible to know if the resistance to passive movement is due to muscle activity or passive neuromuscular changes, since one would have the necessary equipment for measuring if the resistance actually is due to velocity-dependent muscle activity (Nielsen et al., 2007). This

combination has been suggested to be essential in the definition of a spasticity measurement 'gold standard' (Biering-Sørensen et al., 2006).

The increased use of biometric measurements of spasticity and the lack of validation studies examining TS, forms the basis of the current study. The aim of this study was to determine to what extent resistance to passive stretch, performed as in the Tardieu test, can evaluate spasticity in the elbow flexors of children with CP. The Tardieu Scores were compared with the presence of a burst of muscle activity during a fast passive stretch. Secondary objectives were to inspect the tests ability to reflect spasticity severity and evaluate the difference in muscle reaction to a passive elbow extension in children with CP and healthy non-spastic controls.

2.0 Materials and methods

2.1 Study participants

Participants were recruited through a physiotherapist at the neuro-orthopedic team for children with cerebral palsy at St. Olavs Hospital in Trondheim. To be included in this study, one would have to meet the following criteria: Diagnosed with CP, affected upper extremity, age 8 to 18 and the ability to take verbal instructions. Participants were excluded if treated with Botulinum Toxin type A in the affected arm in the last 6 months and/or surgery in the affected arm in the last 2 years.

Fifteen children and adolescents with CP participated in the study, including six females and nine males. The participants had different levels of spasticity in the upper extremities. In addition, 15 healthy controls (five females and ten males) were recruited through workers at NTNU, Trondheim. Written informed consent from each child and the child's parent/guardian was obtained before participation. The study was approved by the Norwegian Regional Committee of Medical and Health Research Ethics (REK).

2.2 Equipment

A Noraxon® TeleMyo ™ 2400T Direct Transmission System (Noraxon, Scottsdale, Arizona, USA Inc) was used for the collection of all biometric data. Prior to sampling, all data were low pass filtered with a cut off frequency of 1500Hz. At sensor location, the data were sampled with 3000Hz, and resampled with 1500 Hz prior to telemetric transmission.

Surface electromyography (EMG) was collected using disposable Dual Noraxon bipolar electrodes with 1 cm conductive area and 2 cm inter-electrode distance (Noraxon, Scottsdale, Arizona, USA Inc). Three electrodes were placed after recommendations of Surface Electromyography Non-Invasive Assessment of Muscles (SENIAM); proximal on the biceps brachii, lateral and medial on the triceps brachii. To ensure a good connection, the skin was cleaned with isopropyl alcohol before placing the electrodes.

To measure the elbow joint angle, a flexible 2D goniometer (Noraxon, Scottsdale, Arizona, USA Inc) was used during the passive elbow extension. The goniometer was placed over the elbow joint, with the arm at full possible stretch, with the axis of the goniometer over the axis of the elbow. The placement was verified by a handhold goniometer at 0 and 90 degrees. For measuring acceleration (g m/s^2), a lightweight 3D accelerometer (Noraxon, Scottsdale, Arizona, USA Inc) was placed horizontal on the proximal phalanx of the first finger with the hand in neutral position when performing the passive elbow extension.

A stationary dynamometer Biodex System 3 Pro (Biodex Medical Systems, Shirley NY, USA) was used to assess maximal voluntary contraction (MVC). Biodex controls position and velocity and records velocity, angle and torque. The Biodex used a 500 Hz low pass filter and the analogue signal was digitized by the Noraxon system.

2.3 Procedures

This study was a part of a more extensive study. Below only the aspects relevant for this study were included. All the passive elbow extensions were done before the flexion and extension isometric MVC to avoid warming up interference.

2.3.1 Anthropometric measures

Prior to the test procedure, anthropometric data were obtained. Information about age (years and months), affected/non-dominant arm was collected. Skinfold thickness (mm) was measured using caliper on the bulk of m. biceps brachii and triceps brachii. Height (cm) and weight (kg) was measured without shoes.

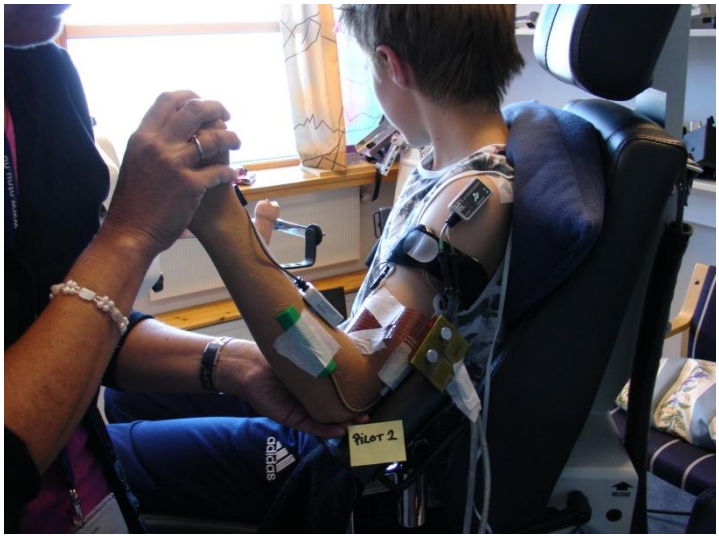

Figure 1. Experimental setup with EMG sensors, goniometer and accelerometer placed on the arm

2.3.2 Passive elbow extension

The goniometer, accelerometer and EMG sensors were attached to the participant's affected (CP) or non-dominant (controls) arm before starting the passive elbow extension (figure 1). The quality of the EMG signals was visually inspected before continuing the protocol. The passive elbow extension was done with the child/adolescent sitting in a chair, with upper-arm and back support and legs relaxed (see figure 1). The examiner held one hand under the elbow and the other one in the participant's hand. The participants were explained the test procedure, and asked to relax as much as possible in the affected/non-dominant arm.

When carrying out the passive elbow extension, three passive elbow stretches were done as fast as possible followed by one passive elbow stretch at a slow velocity. The Tardieu spasticity score and angle of catch were written down immediately after each trial for the CP group. The control group did not get a score, even though a resistance was felt in some participants. The velocity of a limb falling under gravity was not used in the current study, as this is not practical for most muscle groups.

Due to practical reasons, the examiner was a specialized ergonomist for the CP children and a specialized physiotherapist for the controls. To ensure that the two testers did not affect the results of the two groups of participants, an inter-tester reliability test for maximum velocity and ROM was done on seven healthy students (figure 2). The paired samples t-test showed no significant difference between the two testers (ROM: P = 0.200, Maximum velocity: P = 0.788).

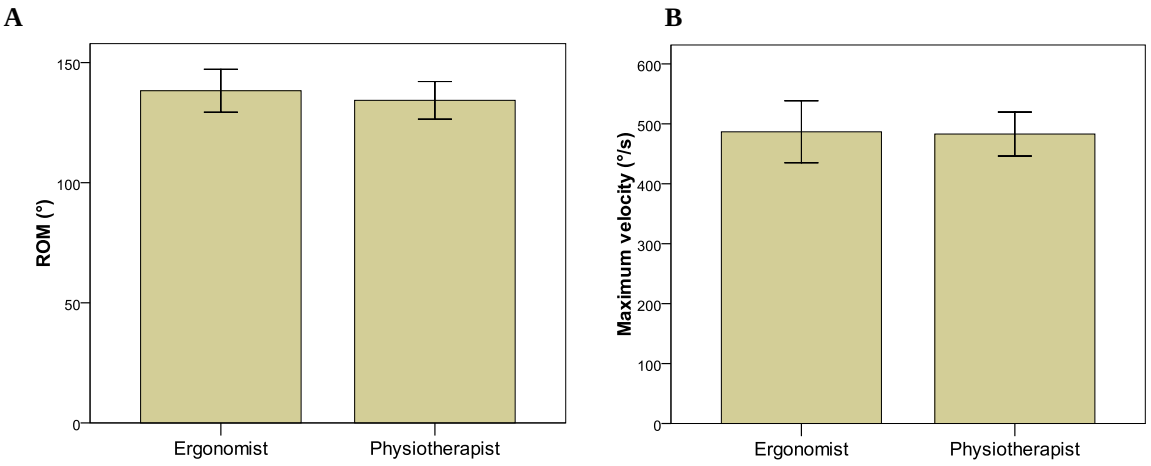

Figure 2. Inter-tester reliability test for the two testers. **Figure A** illustrates ROM (°) presented with mean values and 95 % confident interval (CI). **Figure B** illustrates maximum obtained velocity (°/s) during the passive stretch presented with mean values and 95 % CI.

2.3.3 Maximal Voluntary Contraction

A measure of the elbow flexion and extension strength was obtained through maximal voluntary isometric contractions (MVC). Each participant was placed in the Biodex chair, with the upper body fixed to the chair and the arm fixed to the upper arm support. The shoulder position was approximately 45 degrees flexion and the anatomical elbow angle was 60 degrees (from full extension). A hand flexion-glove was used by all the participants to avoid the grip strength to affect the measurements. A wrist orthosis was also used by all participants to counteract the possibility of bent wrists during the MVC. Three MVC's were obtained with a contraction time of five seconds with a break of 30 seconds between each trial. The participants were instructed to push with maximal force on the lever arm.

2.4 Outcome measures

2.4.1 Subjective TS score

There were two subjective measures obtained during the passive elbow extension. The Tardieu Spasticity Score (TSS) is a scale from 0 to 2 on the upper extremities (0 to 4 elsewhere), which the tester used to evaluate the felt resistance (muscle response) during the fast passive stretch. The more resistance felt, the higher the reported score. If no resistance was felt, the participant was defined with value 0. If some resistance was felt, but not a catch, the participant was defined with value 1. A cut-off value of 2 indicated a catch (spasticity).

The Tardieu Angle Score (TAS) was determined as the angle where the resistance (catch) was felt subtracted from the passive range of motion (slow velocity angle). Thus, a catch early in the extension resulted in a large TAS-score and indicated more spasticity.

2.4.2 Biometric data

Biometric data analyses was carried out using Matlab (The Mathworks Inc.), version 7.10 (R2010a).

The EMG on the biceps brachii was used to measure stretch induced muscle activity during the passive elbow stretch. If a clear stretch induced burst of muscle activity (minimum duration of 50 ms) was present on visual examination, a catch was registered. An accelerometer was used to ensure that the burst of muscle activity altered resistance. If a burst was detected in at least two of the three trials, the participant was determined to have a catch. One person completed only one trial correct, hence the results of this one trial was used in the analysis.

Pre-stretch Root Mean Square (RMS) was calculated 1 second prior to the extension (window width of 0.05 sec). The RMS was also calculated during the whole extension to give information of the average EMG amplitude during the stretch. Further, the pre-stretch amplitude was subtracted from the average stretch induced muscle activity to determine the average increase/decrease of muscle activity during the test in relation to pre-stretch. Additionally, the peak RMS amplitude (window width of 0.05 sec) during the stretch was obtained. Peak RMS amplitude was also seen in relation to pre-stretch to give an indication of change in muscle activity from pre-stretch.

The goniometer measured the angle of the arm in degrees during the passive elbow extension. The smallest elbow angle was set to start and the largest angle was used to determine the end of each trial. The position was derivated to get information about maximum angular stretch velocity.

There was no systematic difference in the three fast stretches in velocity or EMG amplitudes; hence a 'fast mean' was calculated for velocity and all RMS variables. Therefore, further statistical analysis only evaluates the difference between groups of participants and difference in fast and slow trials within each group. However, in the analysis of the relation between biometric measures and TS catch, all three fast trials were included.

Peak torque (Nm) and maximum EMG amplitude developed during the elbow flexion and extension isometric MVC was also measured to describe the different groups of participants.

2.5 Statistical analysis

All statistics was carried out using statistical package for the social science version 17 (SPSS Inc. Chicago, USA). A P-value of < 0.05 was considered statistically significant throughout the analysis. A multivariate Analysis of Variance (ANOVA) was used to assess group and gender effects on the descriptive characteristics. A Mann-Whitney U test was used to compare differences between groups in the MVC data as it violated the distribution of normality (Shapiro-Wilk).

The maximum velocity and RMS amplitude (pre-stretch, average and peak) variables violated the test for normal distribution (Shapiro- Wilk) also, hence non-parametric statistics was performed. To examine the group differences in these variables a Mann-Whitney U test was performed. Further, a Wilcoxon signed rank test was used to see if there were any within group difference with trials.

To evaluate the agreement between the TSS and the laboratory measurements, the percentage agreement was calculated. To evaluate the severity of spasticity, the difference in RMS amplitude, velocity and torque for each TSS value was tested with Mann-Whitney U test. The

biometric responses (RMS amplitude, velocity and torque) and TAS values were presented in scatter plots, but were not tested statistically as there were no clear relations.

3.0 Results

All 30 participants completed the MVC task. All controls (n = 15) completed the passive elbow stretch. However in the CP group, one person did not complete trial 2 and 3 and two other persons did not complete the slow velocity trial in the passive stretch.

The anthropometric characteristics of the female and male CP and control (CO) participants are presented in table 1. There was no significant difference between the groups in age, height, weight, BMI or skinfold thickness (table 1). However, the weight and BMI of the two genders were differently distributed in the two groups; the female CP were, on average, lighter and had lower BMI than the female CO, while average body weight and BMI of the male CP were higher than male CO. From the CP group, also the passive range of motion (ROM) was obtained. On average, the ROM was 174.5 degrees for CP. This was significantly different from the expected 180 degrees full extension in healthy subjects (P = 0.002) when tested with the Mann-Whitney U test.

Table 1. Anthropometric participant characteristics with p-values resulting from a MANOVA test with group and gender as factors.

		N	Age (years)	Height (cm)	Weight (kg)	BB skinfold (mm)	TB skinfold (mm)
CP	Female	6	12.39 ± 3.11	149.40 ± 12.73	38.30 ± 10.13	8.00 ± 3.26	12.40 ± 3.29
	Male	9	12.55 ± 2.45	156.09 ± 16.72	50.04 ± 17.91	7.78 ± 3.36	11.00 ± 4.35
CO	Female	5	12.05 ± 3.48	151.20 ± 20.68	49.80 ± 17.68	6.80 ± 2.20	9.40 ± 2.75
	Male	10	12.22 ± 2.73	157.44 ± 17.25	42.78 ± 11.33	6.60 ± 2.94	11.30 ± 4.47
P-value	Group		0.860	0.763	0.788	0.325	0.399
	Gender		0.983	0.372	0.755	0.860	0.875
	Group*Gender		0.899	0.978	0.124	0.993	0.304

Values are mean ± SD. P- value < 0.05 is considered as significant difference. CP; Cerebral Palsy group, CO; Control group, BB; m. biceps brachii, TB; m. triceps brachii.

The peak torque and EMG amplitudes produced during MVC flexion and extension are presented in table 2. During the MVC tasks, the peak torque developed was, generally, higher for the CO group than the CP group (table 2). This difference between groups was significant in elbow flexion (P = 0.029), but not for elbow extension (P = 0.071). Gender seemed to have an effect, because when split for gender, the differences in peak flexion and extension torque between the CP and CO were significant for the female groups (Flexion: P = 0.018, Extension: P = 0.018), but not for the male groups (Flexion: P = 0.369, Extension: P = 0.624).

Overall, the peak RMS amplitudes developed during MVC were lower for the CP than CO group (table 2). The differences were statistically significant for m. biceps brachii (P = 0.002) and medial triceps brachii (P = 0.000), but not for the lateral triceps brachii (P = 0.110). The same pattern was present when the difference is seen for each gender separately (table 2).

Table 2. Peak torque and RMS amplitude during MVC with group difference p-values resulting from Mann-Whitney U test

		N	Torque EF (Nm)	Torque EE (Nm)	RMS BB EF (µV)	RMS LTB EE (µV)	RMS MTB EE (µV)
CP	Female	6	9.71 ± 1.96	12.21 ± 4.75	141.69 ± 75.25	180.56 ± 134.32	143.61 ± 46.95
	Male	9	19.58 ± 16.63	27.23 ± 16.04	423.94 ± 383.56	451.59 ± 243.67	212.96 ± 163.53
	Total	15	12.41 ± 14.96	19.17 ± 15.08	202.43 ± 343.00	253.82 ± 232.89	168.52 ± 140.30
CO	Female	5	23.06 ± 9.23	25.70 ± 10.54	477.93 ± 352.06	358.05 ± 277.98	322.99 ± 152.22
	Male	10	25.23 ± 15.67	29.14 ± 15.52	755.64 ± 476.81	448.58 ± 433.71	591.52 ± 298.01
	Total	15	23.06 ± 13.97	25.70 ± 13.87	718.78 ± 464.53	417.36 ± 391.93	516.08 ± 289.62
P-value	Female	11	0.018	0.018	0.006	0.201	0.018
	Males	19	0.369	0.624	0.041	0.414	0.003
	Total	30	0.029	0.071	0.002	0.110	0.000

Values are median ± SD. P- value< 0.05 is considered as significant difference. EF; elbow flexion, EE; elbow extension, BB biceps brachii, LTB; lateral triceps brachii, MTD: medial triceps brachii, CP; Cerebral Palsy group, CO; Control group.

3.1 Passive stretch characteristics

Maximal velocity obtained during the passive elbow extension (fast and slow) are presented for the two groups of participants in figure 3. Within both groups, fast velocities were significantly higher than slow velocities (CP: P = 0.002, CO: P = 0.001). The fast velocity

was also significantly higher for the controls compared to CP group (P = 0.002), while slow velocity was not significantly different between the two groups (P = 0.489).

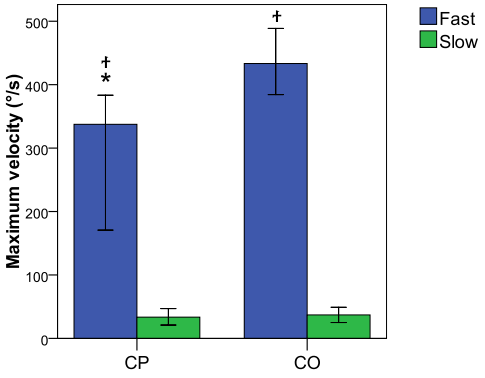

Figure 3. Maximal velocity (°/s) presented with median values and a 95 % CI during the fast and slow trials for both groups of participants. CP; Cerebral Palsy group, CO; Control group, *; significant different from CO, †; significant different from slow.

Pre-stretch, average and peak RMS amplitudes during passive elbow extension are presented in figure 4. Pre-stretch RMS (1 second prior to stretch) was not significantly different between the two groups of participants (fast: P = 0.254 and slow: P = 0.395). There was also no difference between the two trials in each group of participants in pre-stretch amplitude (CP: P = 0.570, CO: P = 0.256). The change in pre-stretch muscle activity from slow to fast trial was not different in the two groups of subjects (P = 0.604).

Average RMS during passive elbow extension was close to significantly different between CP and CO groups in fast trials (P = 0.065), but not different in slow trials (P = 0.575). Differences in average RMS in fast and slow trials within the CP group was borderline significant (P = 0.053), but not significant for the CO group (P = 0.532). The change in average muscle activity from slow to fast trials was not significantly different between CP and CO group (P = 0.178).

Peak RMS amplitude during the passive stretches were not significantly different between the two groups (fast: P = 0.097 and slow: P = 0.381). Furthermore, there was no systematic change in peak RMS amplitude with fast and slow trial for either of the groups of participants (CP: P = 0.099, CO: P = 0.551). There is not a significant difference between the two groups of participants in change in peak muscle activity from slow to fast stretch (P = 0.097). In relation to peak RMS during MVC, the peak RMS during the fast passive extensions was very

low in the CO group (about 2 %) and quite low in the CP group (about 10 %). However, there was a large variation in stretch induced muscle activity in the CP group because the participants were affected to different extent.

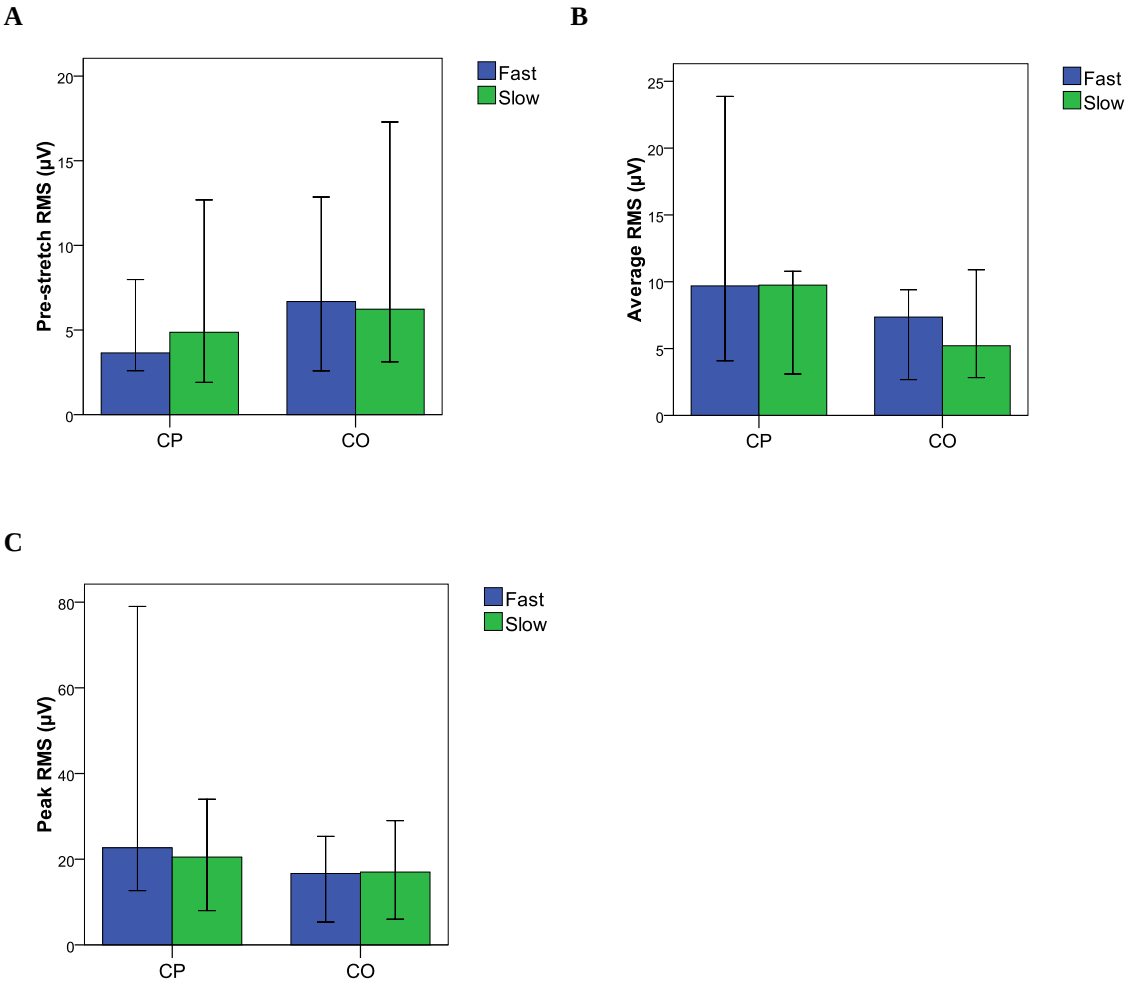

Figure 4. Biceps brachii EMG (µV) during different phases of fast and slow passive elbow extension for both groups of participants, presented with median values and a 95 % CI. **Figure A** illustrates pre-stretch RMS 1 sec prior to extension. **Figure B** illustrates average RMS during the whole extension. **Figure C** illustrates peak RMS during the extension. CP; Cerebral Palsy group, CO; Control group.

During the stretch, controls had a tendency to decrease their average muscle activity from pre-stretch while the CP group increased their average muscle activity from pre-stretch (figure 5A). Change in RMS from pre-stretch to average muscle activity during the passive elbow stretch was significantly higher for the CP group compared with the CO group in the fast trials (P = 0.001), but not in the slow trials (P = 0.081). When analyzing within-group effects, there was a borderline significant difference in slow and fast trials for the CP group (P = 0.055), but not significant for the CO group (P = 0.582). The change in increased average

muscle activity from slow to fast trials was not significantly different between the two groups of participants (P = 0.149).

Both CP and CO group increased their peak stretch induced muscle activity from pre-stretch, however the CP group increased their peak muscle activity considerably more than the CO group in the fast trials (figure 5B). Pre-stretch to peak RMS amplitude was statistically higher for the CP group in the fast trials (P = 0.016), but not in the slow trials (P = 0.170). There were no significant differences between fast and slow trials for neither the CP (P = 0.117) nor CO group (P = 0.955). The change in increased peak muscle activity from slow to fast trials was not significantly different between the two groups of participants (P = 0.107).

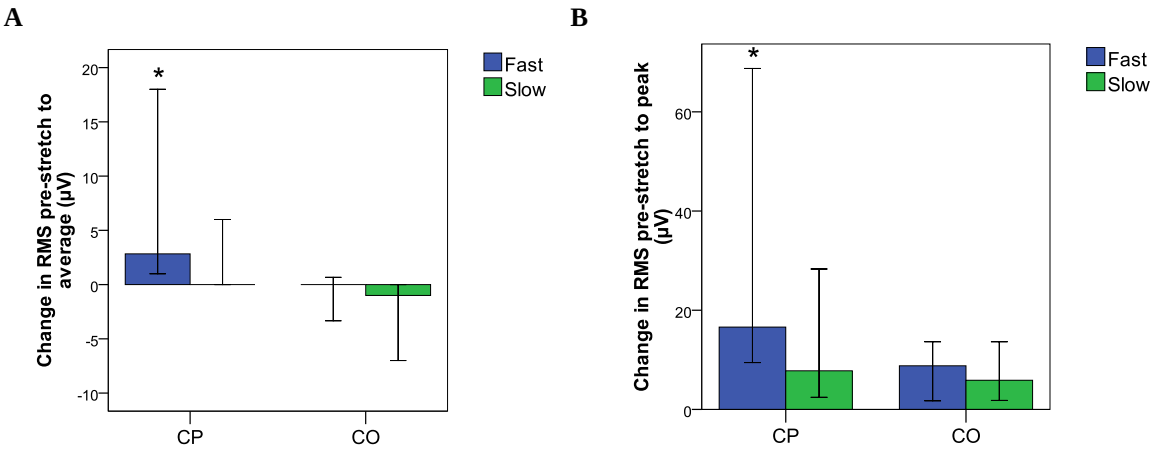

Figure 5. Stretch induced m. biceps brachii muscle activity (µV) in relation to pre-stretch for the two groups of participants in fast and slow velocities, presented with median values and a 95 % CI. **Figure A** illustrates average RMS amplitude during passive elbow extension in relation to pre-stretch RMS. **Figure B** illustrates peak RMS amplitude during passive elbow extension in relation to pre-stretch RMS. CP; Cerebral Palsy group, CO; Control group. *; significant different from CO.

Earlier, a difference in velocity for the two groups of participants was presented (figure 3). This large difference in velocity suggested velocity to be an important part of stretch response development. The relation between peak stretch induced muscle activity and maximum velocity was therefore investigated for the two groups of participants (figure 6). During slow passive extension, the muscle responses in CP and CO were similarly distributed in relation to maximum extension velocity. Conversely during fast passive extension, the amount of muscle response was differently distributed for CP and CO. Controls generally reached a higher maximum extension velocity (above 350°/s) in combination with lower (below 40 µV) peak stretch induced muscle activity compared to CP participants. The CP group generally obtained

lower maximum extension velocity (below 350 °/s) in combination with higher peak stretch induced muscle activity (above 40 μV). Nevertheless, some control participants were high in peak muscle activity, but this was in combination with very high maximum velocities (about 500 °/s). Further, there were also some CP participants with similar response to the controls', with low muscle activity and high stretch velocity.

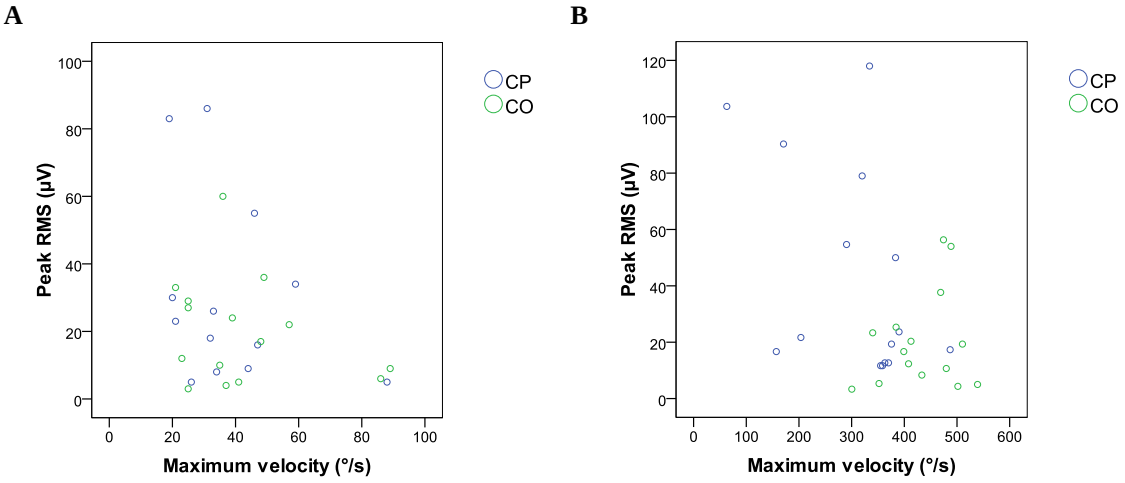

Figure 6. Relation between peak m. biceps brachii muscle activity (μV) and maximum extension velocity (°/s). **Figure A** illustrates slow passive elbow extension. **Figure B** illustrates fast passive elbow extension.

During the passive elbow extension, the TSS value was registered for all 15 CP participants and altogether in 43 trials (table 3). A total of 9 participants (26 trials) were registered with a TSS value of 2, indicating spasticity, of whom 6 participants were females and 3 participants were males. There were 1 male participant (5 trials) registered with a TSS value of 1, indicating some resistance. The remaining 5 participants (12 trials) were also males and registered with a TSS value of 0, indicating no resistance. There was a gender difference in the distribution of TSS during the passive stretch. All CP females were defined as spastic, while CP males were more distributed over the three TSS values. As mentioned, for the CO group, no score was obtained although in some participants some resistance or even a catch was felt.

Table 3. Number of participants (trials) on each Tardieu Spasticity Score (TSS)

TSS	No spasticity (0)	Some resistance (1)	Spasticity/Catch (2)	Total
Female	0 (0)	0 (2)	6 (16)	6 (18)
Male	5 (12)	1 (3)	3 (10)	9 (25)
Total	5 (12)	1 (5)	9 (26)	15 (43)

3.2 The relation between subjective and biometric measures

The biometric measurements defined spasticity in more (32) trials than the TS (26) (table 4). The biometric catch and the TS agreed in 10 of the 15 participants in trial 1, in 8 of the 14 participants in trial 2 and in 9 of the 14 participants in trial 3. In total, the two measurement methods agreed on 27 out of the 43 (63%) trials done in this test procedure and on 10 out of 15 (67%) participants (table 4), this results in a percentage agreement of about 65%.

Table 4. Agreement of subjective and biometric catch detection

	Trials			Participants		
	TS +	TS -	Total	TS +	TS -	Total
BIO +	21	11	32	8	4	12
BIO –	5	6	11	1	2	3
Total	26	17	43	9	6	15

TS+; Subjectively defined with catch, TS-; Subjectively defined without catch, BIO+; biometric defined with catch, BIO-; Biometric defined without catch.

3.3 Spasticity severity

The biometric and subjective measurements were used to evaluate the agreement of spasticity severity. Peak RMS during the passive elbow stretch was slightly decreasing for each TSS value (Figure 7A), however, the difference was not significant (0 vs. 1: P = 0.527, 0 vs. 2: P = 0.637, 1 vs. 2: P = 0.307). There was a similar pattern between average stretch induced RMS and TSS values (figure 7B). The difference in average stretch induced RMS was lower in TSS value 2 compared with 0 and 1, but was not significantly different between any of the TSS values (0 vs. 1: P = 0.833, 0 vs. 2: P = 0.510, 1 vs. 2: P = 0.361). Additionally, change in muscle activity from pre-stretch to average (figure 7C) was also not significantly different between the three TSS values (0 vs. 1: P = 0.831, 0 vs. 2: P = 0.987, 1 vs. 2: P = 0.893).

Maximum extension velocity decreased for each TSS value (figure 7D), however the difference was not significant between scores 0 and 1 (P = 0.193) or scores 1 and 2 (P = 0.519), but significantly different between 0 and 2 (P = 0.023). Since spasticity often is defined as a velocity-dependent increase in the tonic stretch reflex (Sheean, 2002), the relation between TSS and stretch induced muscle activity (average and peak) normalized to muscle activity during slow extension was also investigated. However, the relation was not enhanced by doing so and the results were therefore not included.

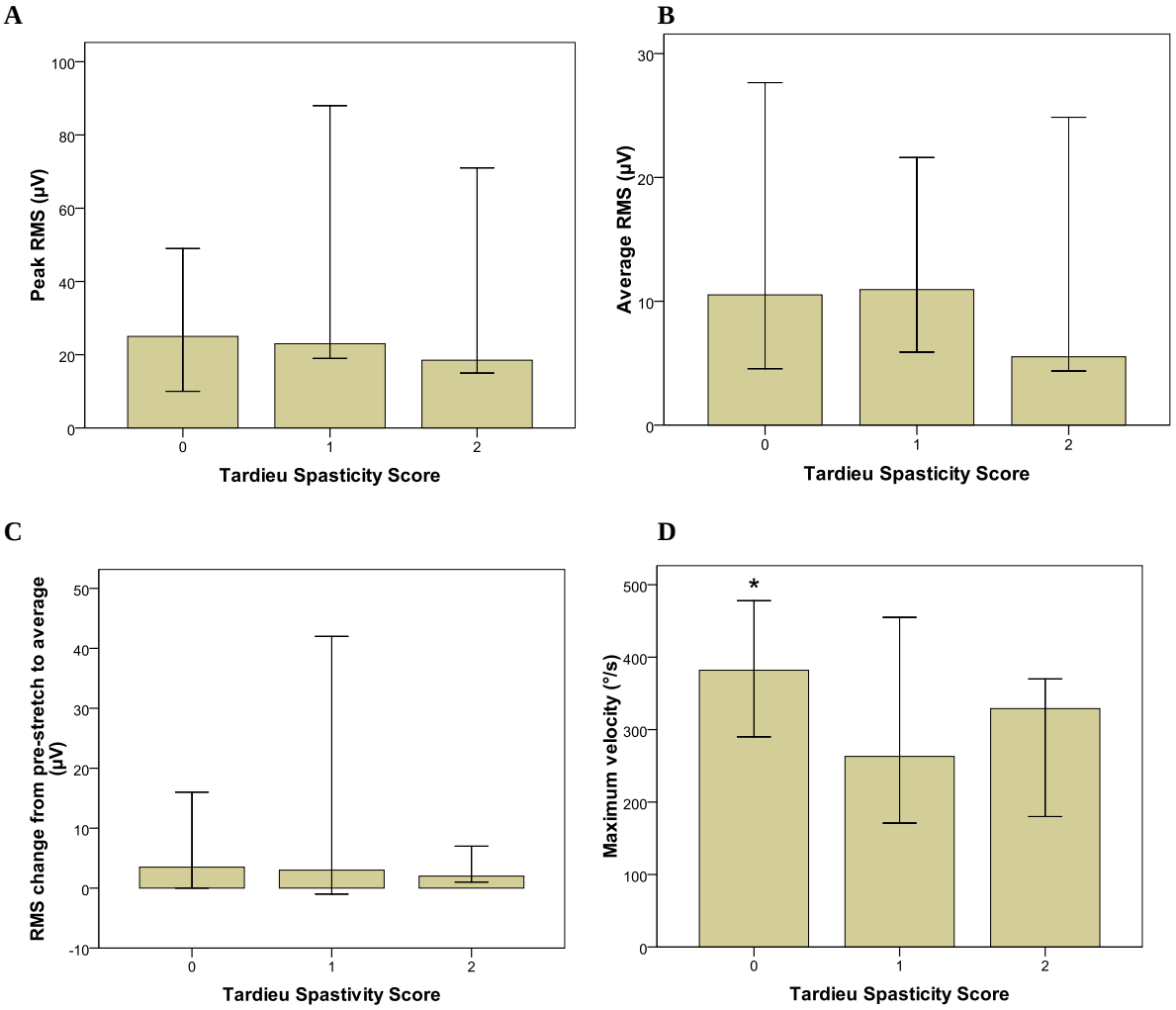

Figure 7. Stretch induced m. biceps brachii muscle activity (µV) and velocity for each TSS value, presented with median values and a 95 % CI. **Figure A** illustrates peak stretch induced RMS for each TSS value. **Figure B** illustrates average stretch induced RMS for each TSS value. **Figure C** illustrates change in muscle activity from pre-stretch to average for each TSS value. **Figure D** shows maximum extension velocity for each TSS value. *; Significant different from value 2.

Figure 8 illustrates the relation between the biometric measures and the TAS values. The figures show that there is no relation between the TAS value and the biometric measurements. Maximum extension velocity had a tendency to decrease with increasing TAS value (figure 8D), especially when seen for the spastic group only. However, no clear relation was present.

The relation between strength and subjective spasticity severity (TSS and TAS) is presented in figure 9. Peak torque (Nm) was significantly different between TSS values 0 and 2 (P = 0.000) and 1 and 2 (P = 0.032), but not between scores 0 and 1 (P = 0.667). There were no clear relation between peak torque and TAS value.

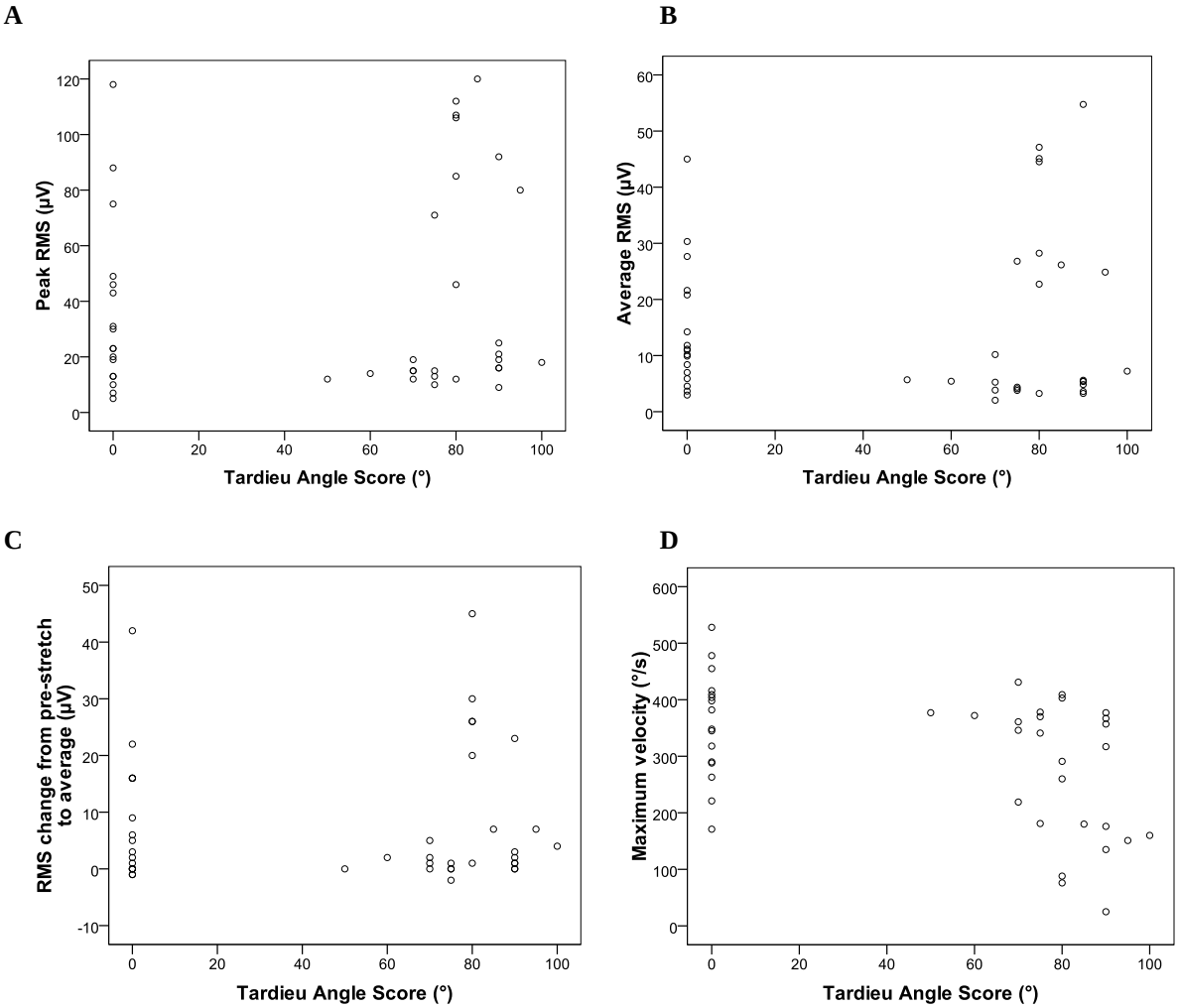

Figure 8. Stretch induced m. biceps brachii muscle activity (µV) and velocity (°/s) in relation with TAS value. **Figure A** illustrates peak stretch induced RMS and TAS value. **Figure B** illustrates average stretch induced muscle activity and TAS value. **Figure C** illustrates change in muscle activity from pre-stretch to average in relation to the TAS value. **Figure D** illustrates maximum velocity of passive stretch in relation to TAS value.

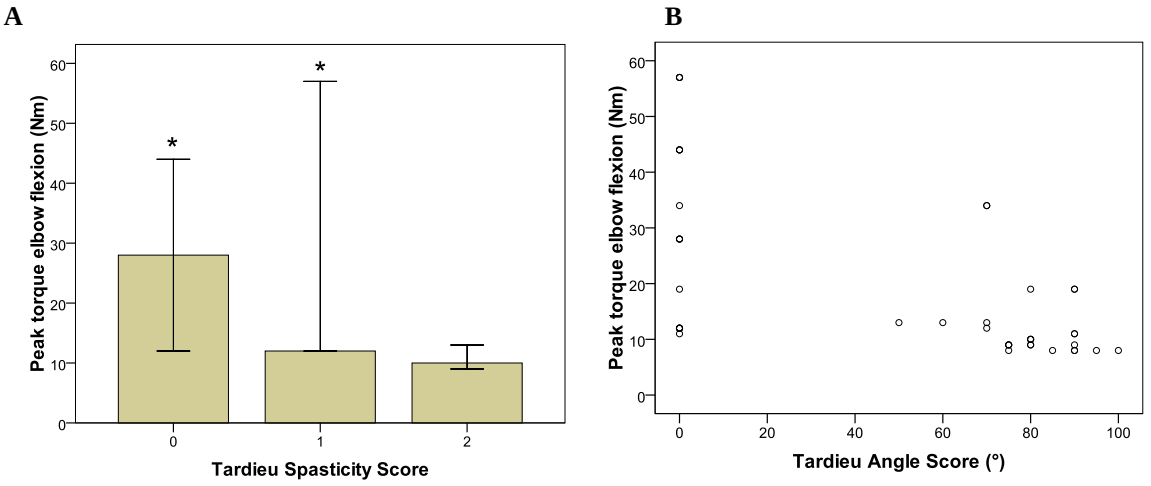

Figure 9. The relation between strength and subjective spasticity severity. **Figure A** illustrates peak torque elbow flexion (Nm) for each TSS value, presented with median values and 95 % CI. **Figure B** illustrates peak torque (Nm) development in relation to TAS value. ***** = Significant different from value 2.

4.0 Discussion

The aim of this study was to determine to what extent resistance to passive stretch, obtained as in the Tardieu test, can evaluate spasticity in the elbow flexors of children with CP. Secondary objectives were to evaluate if the TS reflect spasticity severity and if there were any differences in stretch response between the CP and control group. Catch detection with biometric measures and TS will be discussed first and thereafter the tests ability to reflect spasticity severity in CP participants. Following, the biometric responses to the passive elbow extension will be evaluated in the CP group compared to controls. At last, strengths and limitations of the current study will be mentioned.

4.1 Catch detection with biometric measures and TS

The results show a good agreement (about 65 %) between detected catch with TS (sudden increase in felt resistance) and biometric (EMG burst followed by acceleration change) recordings during the passive elbow extension. The agreement between the TS and the biometric measures in the current study is lower than what was previously found (88.9% (Alhusaini et al., 2010) and 100% (Patrick & Ada, 2006) respectively) using EMG burst to define a biometric catch. However, Patrick & Ada (2006) compared the TS and biometric measures in post-stroke patients. Spasticity caused by a different pathological disorder (i.e. stroke) in adults cannot be generalized to children with CP, as the muscles and passive tissue alters differently (Van der Noort et al., 2010). Alhusaini et al. (2010) evaluated spasticity in the lower extremities of CP participants only. This may indicate that the TS is a better tool when determining spasticity in the lower extremities compared to the upper extremities in children with CP. The greater relation between TS and EMG burst may also be due to higher number of study participants, because a larger selection of participants decreases the consequence of each single participant.

Furthermore, a sudden burst of muscle activity was also present in some control participants at velocities above 340 °/s. This reflects that at high enough velocities, a burst of muscle activity is normal. To avoid defining CP persons with a normal stretch response as having an abnormal catch, the velocity of where controls develop a catch should be implemented as a maximum extension velocity for the TS test. If a muscle burst at velocities above 340 °/s was considered a normal response, only six CP persons were defined as having abnormal catch

biometrically. When maximum velocity of 340°/s was taken into account, the agreement between the subjectively and biometrically defined abnormal catch decreased (53%).

The number of participants with an abnormal catch was overestimated by TS because the testers do not have any limitations of maximum extension velocity. The tester is supposed to do the passive elbow extensions as fast as possible when performing the TS (Gracies et al., 2010), and does not take into account that normal stretch reflexes occur at very high velocities. In consensus with the current results, previous studies showed that a velocity of 300 °/s was not enough to develop a catch in normally developing children (Jobin & Levin, 2000; Levin & Feldman, 1994). However, Cooper et al. (2005) detected a catch in controls at velocities as low as 180 °/s. Further research is needed to evaluate at what extension velocities a normal stretch reflex occurs, to include a maximum extension velocity in the TS protocol. The results accordingly indicate that a catch alone is not enough to define spasticity.

In resemblance with previous results (Van der Noort et al. 2010), a sudden burst of muscle activity was also present in some CP participants during the fast stretch while no catch was registered subjectively by the tester. A possible explanation may be that the muscle activity was not detected by the tester because of short duration of the muscle burst. Even though all registered bursts were over 50 ms, this is a very short amount of time to feel, or even develop, a resistance. Another explanation may be that the RMS amplitude was very low in some individuals. Although the amplitude increased considerably from pre-stretch, the amplitude of some muscle bursts was as low as 20 µV. Pandyan et al. (2005) found that even though a small increase in muscle activity was present, a resistance to the passive movement was not developed until after an additional increase in muscle activity.

4.2 Spasticity severity

The CP participants included in the study were affected in the upper extremities, however with different severity of motor impairments. The TS differentiate CP participants with no felt resistance and participants with a subjectively defined catch in maximum extension velocity. The maximum extension velocity is significantly lower for participants with subjectively defined catch (TSS = 2) compared to participants with no felt resistance (TSS = 0), which reflects an increase in resistance to passive movement. Resistance to passive movement has previously been suggested to be caused by alterations in passive structures as an alternative to

spasticity (Booth et al., 2001; Pandyan et al., 2005; Van der Noort et al., 2010). Yet, the difference in maximum passive stretch velocity was not present in the slow velocity trials, which suggests that the resistance is velocity-dependent. This strengthens the indication of spasticity being the cause of resistance to passive movement.

In resemblance with the conclusion by Alhusaini et al. (2010), the lack of relation between muscle activity amplitude and TS values can be due to TS' limited ability to determine spasticity severity. However, the relation between resistance to passive movement and increased muscle activity has previously proven not to be proportional (Pandyan et al., 2005) which can be a reason for the lack of any significant difference in stretch induced muscle activity in the different severity grades of spasticity determined by the TS.

Furthermore, there was a relation between strength and TSS value. CP participants with subjectively defined catch, was significantly weaker than participants subjectively rated with no or some resistance. Strength has previously shown to have a relation with function in spastic participants (Ross & Engsberg, 2007; Sharp & Brouwer, 1997) which further is suggested to have a relationship with spasticity (Damiano et al., 2001; Scholtes et al., 2006). Of the CP participants subjectively defined with a catch, the majority had very low strength. Strength in the CP participants without a subjectively felt catch generally increased with age in the current study, explaining the large variance in this group of CP participants (figure 9B). An explanation may be that a spasticity-affected arm can lead to reduced usage of that arm, thus reduce strength.

The TAS is also supposed to reflect spasticity severity, where larger TAS value indicates more spasticity (Gracies et al., 2010). The maximum extension velocity had a tendency to be lower in participants with larger TAS value (figure 8F). However, there was no clear relation between TAS and any of the biometric measures. Nevertheless, a lack of agreement between TAS and biometric measurements does not prove TAS to be a poor measurement of catch angle. Initially, the relation between TAS and biometric catch angle (goniometer) was supposed to be evaluated. However because of differences in time-delay between the Noraxon sensors, this approach was not followed through.

4.3 Biometric response to passive stretch in CP and controls

Passive elbow extensions were performed at two velocities, as slow and fast as possible. During the slow passive elbow extension, both CP and control groups obtained a maximum extension velocity of about 30 °/s with little variations between participants. The fast passive elbow extension resulted in a significantly lower maximum extension velocity for the CP group compared to the control group and less variation between control participants. The tester intended to perform the passive extension at a velocity as fast as possible in both groups and within each subject the maximum velocity was very reliable. Therefore, the difference in fast passive extension velocity reflects a velocity dependent increase in resistant to passive stretch in the CP group compared to the control group.

A velocity-dependent increase in resistance to passive stretch is often described to be due to muscle overactivity (Sheean, 2002). In the current study, there were no significant differences in EMG amplitude (average and peak) between CP participants and controls during fast and slow passive elbow extension. This indicates that muscle activity alone does not separate between the stretch response in CP and control participants. Still, the variation of muscle activity amplitude was larger in the CP group compared to controls. This implies that the control group is more homogenous when it comes to muscle response to passive stretch compared to the CP group. This may also reflect that the participants in the CP group are affected to different extent in the upper extremities.

Nevertheless, the CP group had significantly higher increased muscle activity (average and peak) from pre-stretch compared to the control group in the fast trials. There was though no significant difference in increased muscle activity from pre-stretch during slow passive extension. This indicates a velocity-dependent increase in muscle activity (from pre-stretch) in the CP group which is not present in the control group.

Differences in the two groups in velocity-dependent muscle response could also be detected by normalizing peak fast stretch-induced muscle activity to peak slow stretch-induced muscle activity. No difference in peak muscle activity normalized to slow was present, hence it did not differentiate CP and control group better than absolute muscle activity. This may be because even though a catch was detected biometrically, the amplitude in some of the participants was very low. This can affect the amplitudes during slow and fast extension to be

more similar in the two groups of participants. A lot of control and CP participants had a similar muscle response to passive stretch when differences in maximum extension velocity were not taken into account. This may be the reason for the lack of difference in velocity-dependent muscle response.

Peak stretch induced muscle activity in the CP group generally developed at a lower maximum extension velocity compared to controls in the fast trials (figure 6B). However, there was some control participants developing high amplitude muscle activity during the passive elbow extension, but at a higher maximum extension velocity compared to CP participants. There were also some CP participants with a response similar to controls' with high maximum extension velocity and low EMG amplitude, which reflects a normal stretch response. In addition, during slow passive extension, there was a similar relation between maximum extension velocity and peak stretch induced muscle activity in the CP and control group (figure 6A). This difference in distribution of peak stretch induced muscle activity over maximum extension velocity in fast and slow extensions, reflect that a stretch reflex is normal at very high velocities. Therefore the maximum extension velocity should be taken into account when describing an abnormal catch, because at very high velocities a catch is normal. It also revealed that some CP participants had an abnormal stretch response with high muscle activity at low maximum extension velocities.

In addition, muscle activity amplitude is affected by differences in distance from the muscle to the electrode (Biering-Sørensen et al., 2006). To eliminate the effect of differences in distance to the electrodes, stretch induced muscle activity can be normalized. However, the skinfold thickness was not significantly different between the two groups of participants (table 1) and the maximum voluntary activation was significantly lower in the CP group compared to controls (table 2). Therefore, normalized muscle activity would only reflect the decreased ability to voluntarily activate muscles in the CP group. To correctly normalize muscle activity, there should be done recordings of maximum muscle activation.

4.4 Strengths and Limitations

This study tested the stretch response in both CP children and controls. This, together with a measure of velocity, made it possible to detect CP participants with a normal stretch response which otherwise might have been defined as spastic. There would though be possible to do

more analysis with the inclusion of TS values of control participants as well, which is suggested to be included in the next study.

Children and adolescents were studied in this project, which makes it clinically relevant. The degree of spasticity should be investigated early, since spasticity may lead to further complications. So when spasticity is detected and treated in children and adolescents with CP, one may increase function earlier and avoid secondary problems.

Initially, a selection of 20 CP and 20 control participants was supposed to be included in the study. Because of difficulties with recruitment of test participants, 15 CP and 15 controls were included in the selection. Although, this is an acceptable participant selection, among the CP participants there was an uneven distribution of spastic and non-spastic participants. More participants with each TS score could strengthen the power of the results, since there now is more participants with subjectively defined catch (TSS value of 2), than no catch (0 and 1). Hence the next study should include more participants with a broader distribution of TS scores.

The fact that two testers performed the passive elbow extension for the different groups could have affected the results. However, this was taken into account and an inter-tester reliability test was performed, and no significant difference in maximum velocity and ROM was present (figure 2).

The simultaneous registration of both TS and biometric measures during the passive elbow stretch avoided a difference in time between the different recordings. This eliminates the chance of any differences in response to be due to change in time, surroundings or order of measurements. Nevertheless, the relation of angle of catch determined subjectively by the tester and biometric determined catch angle (goniometer) was supposed to be investigated. However, the uncertain sensor time-delay made it very difficult to interpret the timing of muscle burst, which lead to an elimination of this approach in the current study.

In a few cases, it was difficult to determine the start of the movement, both objectively and visually, in the biometric data. This made it difficult to define a catch in some individuals because the muscle burst broke out early after or before the passive movement started. It was

therefore difficult to see if the movement or muscle activity started first. This may be caused by a difference in time-delay between the Noraxon sensors.

5.0 Conclusion

In all, resistance to passive stretch, obtained as in the TS, seems to provide a useful evaluation of spasticity in children with CP. However, when a biometric response similar to controls' is defined as normal, TS overestimates CP participants with abnormal catch. This result underlines that the TS test protocol should include a maximum extension velocity in which a normal stretch reflex does not occur, to decrease the possibility of falsely defining a normal stretch response as a spastic catch.

Furthermore, TS reflects spasticity severity to a limited degree, considering muscle activity. On the other hand, the test seems to detect CP participants with increased resistance to passive movement. In addition, the CP group responds to fast passive stretch with higher muscle activity amplitude at lower maximum velocity compared to controls. Nevertheless, a catch is present in some controls at very high velocities.

6.0 References

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