Norwegian University of Science and Technology Faculty of Social Sciences and Technology Management Department of Human Movement Science

Ankle muscle weakness, spasticity and gait function in children and adolescents with CP

BEV 3901 Master's thesis

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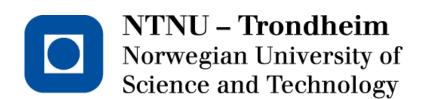


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Preface and acknowledgements

This study was originally planned to study ankle muscle weakness, spasticity and gait function in children and adolescents with CP, as compared to a control group with typically developing children and adolescents. However, a denial from the Regional Committees for Medical and Health Research Ethics hindered use of the control group due to a late change request.

This research project would not have been possible without the help and support of many people.

First and foremost I would like to offer special thanks to my main supervisor Karin Roeleveld for valuable and constructive suggestions and guidance throughout the research work, and for providing me with Matlab scripts used in the analyses.

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Abstract

Aim: The overall purpose of this study was to investigate whether ankle strength and spasticity measures evaluated by a dynamometer can detect the body function impairments important for gait function represented by gait speed, step length and phase of single support.

Methods: 12 children and adolescents with unilateral and bilateral spastic cerebral palsy were included in the study. All participants went through a 3D gait analysis to evaluate temporal spatial variables (gait speed, step length and phase of single support). To measure ankle strength and spasticity, the subjects performed plantarflexion and dorsiflexion in passive, concentric and isometric condition in a Biodex dynamometer. Both sessions included simultaneous surface electromyography recordings on mm. gastrocnemius medialis, soleus and tibialis anterior.

Results: Concentric and isometric weakness and impaired rate of torque development in the affected ankle was detected. Spasticity in the ankle was likely not detected in the dynamometer, as the muscle activity during passive stretch in the affected foot was not significantly different from the contralateral foot. The phase of single support was statistical significant lower in the affected foot than the contralateral foot, while step length was not different between the affected and the contralateral foot. There were some significant correlations between ankle strength and muscle activation during passive stretch, as well as between muscle activity during passive stretch and gait function. There were no significant correlations between ankle strength and gait function.

Conclusion: The dynamometer along with EMG recordings did likely provide a valid measurement for ankle strength, but not for ankle spasticity. Ankle weakness did not interfere with gait function in these subjects, while ankle muscle activity during passive stretch showed stronger relationships to gait function.

Keywords: Cerebral palsy, ankle, muscle weakness, spasticity, gait function, electromyography

List of abbreviations

BTX-A	Botulinum Toxin type A
СР	Cerebral palsy
DF	Dorsiflexion
FW	Fast walking speed
GM	Gastrocnemius medialis muscle
MVIC	Maximal voluntary isometric contraction
PF	Plantarflexion
PW	Preferred walking speed
RTD	Rate of torque development
RMS	Root mean square
SEMG	Surface electromyography
SO	Soleus muscle
ТА	Tibialis anterior muscle

1.0 Introduction

1.1 Definition and prevalence

Cerebral palsy (CP) is an umbrella term for a group of irreversible motor disorders that are attributed to a non-progressive lesion or abnormality in the developing central nerve system (CNS), occurring prior to birth, at birth or during the first two years of life (1, 2). The disorder is the most common cause of physical disability amongst children and adolescent, and the incidence worldwide of CP is considered to be around 2 to 2.5 per 1000 live births (3). This also matches the numbers for Norway (4). The most dominant type is spastic CP, which is characterized by persisting increased muscle tone in one or more limbs (1), with a proportion of 80-90% of all cases (5).

1.2 Changes in body function, structure and activity

The disorder is often accompanied by movement dysfunctions, but the causes of these are highly individual and multidimensional. Common for all are neuromuscular disturbances caused by brain damage, which can cause secondary progressive musculoskeletal changes (6). One such disturbance is muscle weakness, which can be defined as the inability to generate normal voluntary torque across a joint. "Normal" is based on values that are expected for healthy age- or size-matched children and adolescents or the contralateral unaffected limb of the same subject (7), although there has been found that the strength in the unaffected leg can also be impaired compared to the strength of healthy age-matched peers (8). Even children with mild CP demonstrate significant weakness compared to age-matched peers (2, 9), as well as a reduced ability to generate muscle force fast, i.e. reduced rate of force development (10). This can be attributed to neurological factors (e.g. incomplete muscle activation of the agonist and co-activation of antagonist muscles (2)), as well as structural factors (e.g. muscle atrophy (11), physiological cross-sectional area and muscle thickness (12)), which have been shown to be reduced in the affected limb(s) in children with CP (11, 13). There is good evidence to suggest that the distal muscles are more affected than the proximal muscles, i.e. the muscles around the ankle are more affected than the muscles around the knee (8, 14).

Another disturbance that may occur secondary to an upper motor neuron lesion is muscle spasticity, which is often defined as a velocity-dependent increase in muscle tone as a result of hyperexcitability of the stretch-reflexes compared to what is just a normal protective mechanism (15). Spasticity is velocity-dependent in the way that the faster the muscle is stretched, the more resistance and reflex activity will be present.

All or some of the above mentioned factors are likely to interact with gait function, as the disorder hampers the ability to initiate and control the motor output that is required for a task (15), for instance walking. Among European children, 90% of the children with unilateral spastic CP and 36% with bilateral spastic CP can walk unaided, while the remainder depend on walking aids, or are unable to walk (16). Furthermore, nearly all children with bilateral spastic CP start to walk later than typically developing children (17). A cross-sectional study on children with spastic CP revealed that compared to typically developing children, the children with CP had reduced gait speed and shorter stride length (18). Also, gait stability has been shown to be deteriorated with time and growth in a longitudinal study of children with bilateral spastic CP, and phase of single support is one of the gait parameters that drops with this deterioration (17). In contrary to this, the development of mature gait in typically developing subjects involve increased gait speed, step length and phase of single support along with increasing stability with growth (19).

Due to these changes in body function and structure, activity and participation in the society may be reduced (20). Hence, finding proper treatment options are essential to potentially increase autonomy and perhaps improve quality of life.

1.3 Treatment options

As spasticity is associated with tense and contracted muscles, which again are associated with bony deformations and contractures, the primary aim is often to treat spasticity in order to prevent developing contractures (21). One form of treatment is Botulinum Toxin (BTX-A), which temporarily inhibits the release of acetylcholine to local muscles that are treated. However, this treatment only reduces the hyperactivity of the current muscle, and has no positive effect on factors such as selective motor control, muscle weakness or poor balance (21). On the contrary, it is reported that injection of BTX-A has adverse effects, with local muscle weakness being one of them (22). It is suggested that these latter factors are more critical for gait function than spasticity is (21).

The idea that strengthening the muscles would exacerbate spasticity and trigger more muscle stiffness, which was dominating in clinical practice for a period (23, 24), has now been disproven (24-31). Some findings suggest that the relationship is reverse, i.e. lower voluntary

torque is associated with greater levels of spasticity (32), although another finding suggest that there is no relation between the two (14). It is incidentally shown that children participating in strengthening programs have demonstrated increased gait function in terms of increased gait speed, step length and phase of single support (33).

1.4 Role of the ankle

The working muscles across the ankle joint have important roles during a normal gait cycle. A major plantarflexion (PF) takes place at toe off and opposite initial contact (34). Hence, the plantarflexor muscles, among them mm. gastrocnemius medialis (GM) and soleus (SO) are important for a powerful toe off in order to generate force needed for acceleration of the limb, and thereby an adequate step length (35). Excessive PF during both swing phase and stance phase, which results in an equinus gait pattern, has been shown to be a pathological gait characteristic among children and young adults with unilateral spastic CP (36).

Dorsiflexion (DF), on the other hand, occurs before initial contact for the pre-positioning of the foot, in the loading response and in the swing phase for foot clearance (37). M. tibialis anterior (TA) is an important muscle to investigate because many of the children with CP experience difficulties in DF during gait and because of the greater relative strength of the plantarflexors compared to the dorsiflexors. Inadequate DF during the swing phase can cause the toes to be dragged on the floor during swing phase, which leads to a decreased step length and gait speed (38). This can also be reinforced by spastic plantarflexors, which can contribute to restricted ankle DF (39).

1.5 Measuring gait, strength and spasticity

Measuring gait function, as well as strength and spasticity in the affected limb(s), can be essential for treatment and rehabilitation decision making. A premise for this is that the measurements are valid, reliable and sensitive enough to identify outcome measures that reflect actual function.

In gait analysis, the ultimate goal is usually to identify factors that contribute to impaired gait function. One requirement for efficient gait function is balance, and there has been found a strong relationship of muscle strength in the lower extremity to balance and gait (40). Good balance can ensure stability in for instance the phase of single support during gait. The ability to balance on one leg may further affect the step length of the opposite foot, and thereby also possibly effecting gait speed (which is the product of step length and cadence). Both gait

speed and phase of single support have proved to be good indicators of gait function, as they are shown to correlate well with GMFCS functional level (41).

Ankle strength measurements can be executed in various ways which each have their strengths and weaknesses. Manual muscle testing (MMT) is a relatively accessible method as it does not require any equipment, but the method relies much upon the training of the tester and standardized positions which can be challenging in a neuromuscular disease like CP, considering pathologies like spasticity and contractures (21). The same applies to hand-held dynamometers, although these have been shown to be a reliable measurement for the strength of children with CP (42). Furthermore, the tester has to be stronger than the patient to maintain the dynamometer stationary (43). More advanced isokinetic torque systems evade all these problems to a great extent, and a stationary dynamometer has for instance been found to be reliable for testing the knee flexors and extensors in children with CP (44). However, such dynamometers have the disadvantage of being expensive. Also, there is a lack of standardized procedures for testing children with impairments (45), and testing of the calf muscles poses additional challenges. Nor have any studies been found to have tested the reliability of ankle measurements in an isokinetic dynamometer.

Experience from earlier in-house testing with the dynamometer have also found that many children with CP is unable to generate enough torque to overcome the gravity and the weight of the foot pad in an upright position, as the foot pad is too heavy. Furthermore, subjects with spastic CP are prone to contractures (i.e. shortened tendons and muscles reducing the flexibility of a joint), which stresses the need for a testing method that renders distinction between the effects of gravity and the effects of passive forces across the joint. A position where movements take place in the horizontal plane has the potential of eliminating these problems. However, the dynamometer is not directly designed for this position, and there does not exist any known standardization protocols for this testing method by our knowledge.

The dynamometer may also have the potential to measure spasticity, and it has even been shown to be more reliable than the more traditional Ashworth and Tardieu scales (46). The dynamometer has the advantage of testing muscle strength through the entire range of motion (RoM) of a joint, and can objectively measure possible spasticity at different velocities. Spasticity can be measured as resistance to passive stretch, although such resistance may occur due to both passive and active components in the spastic muscle (47) which complicates the measurement of what is actually spasticity and what is structural changes. In this case, EMG may be a helpful tool, allowing us to see when the muscle is (over)activated (48).

Strength and spasticity measures, along with an analysis of gait function, enables a groundwork for treatment options, as one can identify factors that interfere with function.

1.6 Aim and hypotheses

The overall purpose of this study was to investigate whether ankle strength and spasticity measures evaluated by a dynamometer can detect the body function impairments important for gait function represented by gait speed, step length and phase of single support.

Therefore, the following specific aims were defined:

- I. Investigate whether isometric and isokinetic strength, rate of torque development (RTD) and spasticity in the ankle joint can be measured in a Biodex dynamometer with the ankle placed in a position where movements take place in the horizontal plane for children and adolescents with spastic CP.
- II. Investigate whether the measurements are shown to distinguish between (the most) affected and contralateral side.
- III. Investigate whether there is a difference between the affected and the contralateral foot in step length and single support during gait.
- IV. Investigate whether muscle strength and spasticity are related to gait speed, step length and phase of single support in this study group, with focus on the affected foot.

The main hypotheses were as follows:

- I. The dynamometer provides a feasible measurement method for strength, RTD and spasticity.
- II. The affected foot shows more muscle activity during passive stretch with increasing velocity in the plantarflexors, lower strength, and lower RTD compared to the contralateral side.
- III. The affected leg has shorter step length and less phase of single support than the contralateral leg.
- IV. Weakness and spasticity are strongly correlated with each other, and also strongly positively correlated with impaired gait function.

2.0 Methods

2.1 Participants

12 children and adolescents with CP (7 boys and 5 girls) in the age range of 7-18 years (mean age 12.5), referred to gait analysis as part of their follow up at St. Olavs Hospital in Trondheim, were included. Informed consent was obtained from each participant or the participant's parent or guardian when aged below 18.

To be included, the participants had to (1) be diagnosed with CP, affecting the lower extremities, (2) be under the age of 19, (3) be able to walk independently and (4) be able to receive verbal instructions. Exclusion criteria were (1) Botulinum Toxin treatment (BTX-A) in the lower extremities within the previous 4 months and (2) surgery in the lower extremity during the last 2 years.

On the GMFCS (Gross Motor Function Classification System) scale, which includes five levels of gross motor function, where level V is the least functional, the participants in this study were all on level I and II. This reflects the ability to respectively walk without restrictions indoors and experiencing some limitation walking outdoors and in the community (49).

2.2 Equipment and procedures

The data collection lasted from September 2012 to February 2013. Each participant went through a test session that lasted approximately three hours. Prior to testing, anthropometric measures (body weight, height and leg length) were collected and surface electromyography (SEMG) sensors were placed on the selected calf muscles. The test session comprised two parts: (1) gait analysis and (2) spasticity test and strength test in a dynamometer. SEMG data was collected during both parts, but for this thesis only the SEMG during the dynamometer testing were used.

2.2.1 SEMG

Muscle activity was measured using SEMG (Myon Prophysics). The electrodes were placed parallel to the muscle fibers on GM, SO and TA on both legs. Procedures and placements were executed according to SENIAM recommendations (50). Recordings were made wireless and the signal was amplified and digitized before transmitted. The electrodes that were

placed on the skin were bipolar with 1 cm spacing. The sampling frequency was 2000 Hz. The dynamometer and SEMG signals were synchronized and collected by the same system (ProEMG Stand-Alone Application, version 1.3.0.6).

Prior to electrode placement the skin was cleaned and scrubbed with cotton and alcohol to remove dead skin cells and impurities. SEMG sensors were attached on the skin with double sided tape, and cables were fixated with silk tape. The SEMG recordings were monitored live as the participant performed the different tasks. The raw baseline quality was also inspected when the participant was asked to relax as much as possible to ensure that all sensors worked properly.

2.2.2 Gait analysis

Gait analysis was conducted using an eight camera Vicon MX-13 motion capture system (Vicon Motion Systems, Ltd, Oxford, UK) with a capture frequency of 200 Hz. Three AMTI (Watertown, USA) force plates were embedded in the walkway which measured ground reaction force at 1000 Hz. Marker placement was according to a conventional gait model for lower extremities (51).

All participants underwent a barefoot clinical gait protocol on a walkway of 11.5 meters, where the mid 4-5 meters were the active area for camera sampling.

The subjects walked at two different speeds; preferred speed by the instruction "walk as you usually walk", and thereafter at fast speed by the instruction "walk as fast as you can safely walk".

A minimum of six trials were completed at each speed. Participants were allowed to rest between the trials if they felt fatigued. Instructions were given prior to each speed.

2.2.3 Spasticity and strength testing

Resistance against passive stretch and muscle strength was measured using a stationary dynamometer (Biodex System 3 Pro, Biodex Medical Systems, Shirley NY, USA), which controlled and measured torque, angle and angular velocity throughout the range of motion of each participant.

The participants were placed laying sideways in the stationary dynamometer in order to perform PF and DF in a gravity neutral position. The medial malleolus of the ankle was

aligned with the center of the axis of rotation, and the foot was fixed to the footpad with Velcro straps. The knee joint was set to 40° of flexion from full extension. Prior to the tests, RoM in the ankle joint was established for each participant. For the unilaterals the affected foot was tested first, followed by the contralateral foot. For the bilaterals, the right foot was tested first, independent of which foot was dominant. All subjects got verbal feedback and encouragement during all tests.

Prior to data collection, the participants were familiarized with the motions (i.e. PF and DF) in the dynamometer. The testing consisted of three main sequences with a given order: (1) passive movement (2) concentric contraction, and (3) isometric contraction. Instructions were given prior to each sequence and 2-3 trials were conducted prior to each type of contraction to ensure the participant was familiarized with the task. Between each sequence there was a break 2-5 minutes to give the muscles some recovery time.

The passive test was used to evaluate resistance to passive stretch through the entire RoM in the ankle joint, which can indicate presence of spasticity. The subjects were instructed to relax as much as possible, as the foot plate moved automatically. Two repetitions were performed at 10° /sec and four repetitions at 90° /sec and 180° /sec.

Concentric contractions were performed with the dynamometer set at an angular velocity of 10°/sec. The participants completed two maximal PF and two maximal DF in total, with one PF and one DF in one set. A 30 second break was given between the two sets to let the muscles recover. The participants were instructed to give maximal effort in each direction, during the whole movement.

Isometric contractions were performed with the foot pad set to 15° PF from a perpendicular position, enabling static testing at zero velocity. The participants were asked to contract as hard and as fast as possible when given the start signal, in order to measure peak torque and maximal RTD. The contraction window was set to 10 seconds, but the participants were asked to stop the contraction when the instructor registered visually on the screen that the torque slope declined and hence that peak torque was reached.

2.3 Data analysis and outcome measures

Digital processing of spasticity, strength and SEMG data was carried out using Matlab (The Mathworks Inc.), version 8.0.0.783 and Microsoft Excel 2010.

2.3.1 SEMG

Initially, the EMG frequency distribution of each signal was inspected to check the baseline quality and to identify possible external noise sources and other artifact sources. A Butterworth Low-Pass was set to 500 Hz and High-Pass was set to 20 Hz to reduce noise and artifacts. Root Mean Square (RMS) was used to smooth the signal before further analyses. The smoothing window size was set to 200 ms in both the strength and spasticity data and gait data.

2.3.2 Gait analysis

The 2D data captured by each camera was processed to obtain the marker trajectories in 3D space, using Nexus (Vicon, UK). A Woltring low-pass filter was employed to the kinematic data to filter out the high frequency marker movements (52). Ground reaction force data was used to define gait cycle events (initial contact and toe-off), which allowed normalization of kinematic data to 0-100% cycles.

In order to investigate gait function, the following gait parameters were included in the analyses; gait speed (m/s), step length (m) and phase of single support (% of full gait cycle).

Averaged spatiotemporal gait parameters of all gait cycles, within the camera target area, of each six trials, were used for further analysis. All gait parameters comprise both preferred and fast gait. Step length and phase of single support were analyzed separately for the affected and contralateral (or least affected) leg, and gait speed was averaged because of minor differences between the two legs.

Gait speed and step length were normalized to leg length to eliminate the effect of differences in height. Phase of single support was not normalized, as it is already normalized to 100% of the gait cycle.

2.3.3 Muscle spasticity

In the passive test at three different velocities, the outcome measure was average EMG RMS during the isokinetic phase in order to reflect muscle tone which reflects one aspect of spasticity (53). In order to investigate the possible pathological stretch response, the plantarflexors (GA and SO) were the outcome muscles during DF, and the dorsiflexor (TA) was the outcome muscle during PF.

2.3.4 Muscle strength

To ensure that the peak torque reflected *active* torque, and not passive torque due to body structures, we chose to analyze baseline-to-peak torque. The baseline torque is subtracted as this likely reflects passive torque and possible low frequency baseline drift. Prior to analyses, all torque signals were low-pass filtered with a cut-off frequency of 10 Hz.

Concentric strength was measured as peak torque in the isokinetic phase of PF and DF. Muscle activity was measured as EMG RMS with an interval of +/-100 ms around peak torque.

Isometric strength was measured as peak torque during the MVC. Muscle activity was measured as EMG RMS with an interval of +/-100 ms around peak torque.

RTD was calculated to measure the ability to generate force fast. This was done by differentiating the torque-time-curve, and then selecting the peak value of Nm/s. This is illustrated in figure 1.

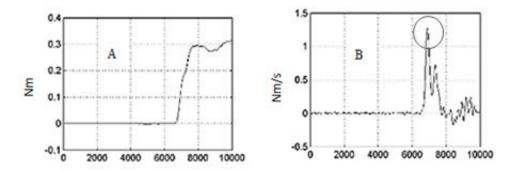


Figure 1. A: torque-curve (to peak) during isometric plantarflexion. *B*: derivative of the torque-curve for calculation of peak rate of torque development (circled).

In the isometric and concentric tests the highest peak torque value in PF and DF was chosen to represent maximal contraction and used for further analyses. This gave a total of one maximal PF and one maximal DF for each foot in each contraction type. In the passive test, the average EMG activity for each muscle and for each velocity was used in further analyses.

Similar to a number of studies, torque was normalized to body weight to eliminate the effect of size (54-57).

An overview along with brief explanations of all variables is presented in table 1.

Independent variables	Spasticity	Average EMG RMS (µV)
	Strength	Isometric peak torque (Nm)
		Concentric peak torque (Nm)
		Isometric RTD (Nm/s)
		Agonist EMG RMS \pm 100 ms around peak torque ($\mu V)$
Dependent variables	Gait	Speed (m/s)
		Step length (m)
		Single support (%)

Table 1. Overview of the outcome variables.

EMG RMS = *electromyography root mean square. RTD* = *rate of torque development.*

2.4 Statistical analyses

Statistical analyses were carried out in IBM SPSS Statistics Version 19.

As most strength and spasticity variables were not normally distributed, descriptive statistics are based on non-parametric analyzes. These data are presented in boxplots which display the median value at the center of the plot and a box including the middle 50 % of the observation (the interquartile range). Two whiskers on the top and bottom of the box extend to the top and bottom extreme values respectively. Outliers are displayed above or below the whiskers, and are excluded from the calculation of the median and interquartile range.

In the descriptive analyses, nine subjects with unilateral CP and one child with bilateral CP were included. There were originally three subjects with bilateral CP, but two were excluded due to inconsistency in which foot was the weakest between isometric and concentric contraction. The third bilateral child was pooled with the unilaterals as one foot was consistently weaker, and hence probably more affected than the other. Data are presented both absolute and normalized.

The Related-Samples Wilcoxon Signed Rank Test was conducted to investigate possible side differences between affected and contralateral side. A p-value of <0.05 was considered statistical significant.

Correlation analysis was used to investigate the relation between normalized strength, spasticity and gait data. Due to some extreme values, Spearman was chosen in preference to Pearson because of less sensitivity to outliers. All 12 subjects were included to provide more powerful tests. Analysis was carried out on all the variables we expected to see associations between, with solely focus on the affected foot. Based on Portney and Watkins' guidelines, a correlation of 0.00-0.25 was considered little or no relationship, 0.25-0.50 was considered fair degree of relationship, 0.50-0.75 was considered a moderate to good relationship and >0.75 was considered a good to excellent relationship (58).

For the variables where there were missing data on some subjects, cases were excluded variable by variable.

For the remainder of this thesis, the two feet will be termed *affected* and *contralateral*. In the case of the bilaterals, the *affected* foot represents the *most affected* foot.

3.0 Results

All 12 subjects completed the gait analysis. In the spasticity and strength test, two participants tested only the affected foot, and not the contralateral foot due to fatigue after the test session.

Anthropometric measures are reported in Table 2 with mean \pm SD, along with other participant characteristics split in unilateral and bilateral CP.

Variable	Unilateral CP $(n = 9)$	Bilateral CP ($n = 3$)
Age (years)	12.9 ± 3.9	11.3 ± 3.1
Body weight (kg)	49.6 ± 22.5	36.7 ± 15.4
Sex (male/female)	4/5	3/0
Height (cm)	152.8 ± 18.7	143.8 ± 21.2
Leg length right (cm)	79.1 ± 10.2	75.7 ± 12.1
Leg length left (cm)	79.2 ± 10.8	75.7 ± 12.1
GMFCS (I/II)	8/1	0/3
Affected limb (right/left)	2/7	n/a

 Table 2. Participant characteristics

GMFCS = *Gross Motor Function Classification System. Age, body weight, height and leg length are presented as mean* ± *SD.*

3.1 Gait

The results for gait speed during PW and FW are presented in figure 2 A (absolute data) and B (normalized to leg length). The gait speed during PW was significantly lower compared to FW (p = 0.005) in both the absolute data, and the data normalized to leg length.

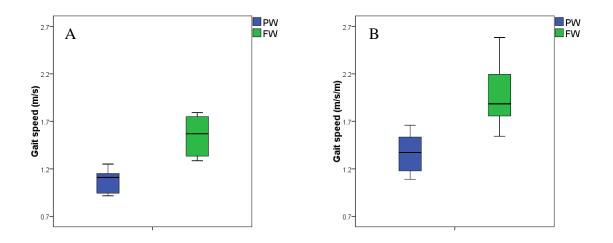


Figure 2. Boxplots illustrating differences in gait speed (m/s) during preferred gait speed (PW) and fast gait speed (FW). Speed is averaged for the two feet. A: absolute data. B: normalized to leg length.

The results for step length during PW and FW for the affected and contralateral foot are presented in figure 3 A (absolute data) and B (normalized to leg length). The step length increased with the increase in speed from PW to FW in both the affected (p = 0.005) and contralateral (p = 0.005) foot in both absolute and normalized data. The step length of the affected foot was not significantly different from the contralateral foot in both the absolute data and normalized to leg length, neither in PW (p = 0.889), nor in FW (p = 0.258 and 0.214).

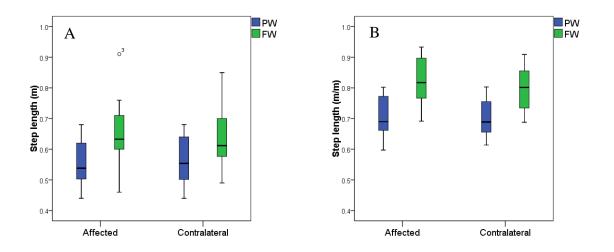


Figure 3. Boxplots illustrating differences in step length during preferred gait speed (*PW*) and fast gait speed (*FW*) for the affected and contralateral foot. *A*: absolute data. *B*: normalized to leg length.

The results for phase of single support during PW and FW for the affected and contralateral foot are presented in figure 4.

The phase of single support increased with the increase in speed from PW to FW (p = 0.005) and contralateral (p = 0.017). The phase of single support of the affected foot was significantly shorter compared to the contralateral foot in both PW (p = 0.005) and FW (p = 0.013).

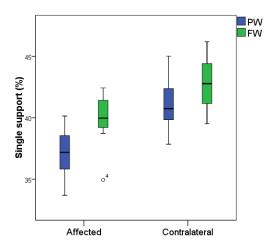


Figure 4. Boxplot illustrating differences in single support (%) during preferred gait speed (PW) and fast gait speed (FW) for the affected and contralateral foot.

3.2 Muscle strength

The results for isometric and concentric peak torque are presented for the affected and the contralateral foot in figure 5 A (absolute data) and B (normalized to body weight).

The strength of the affected foot was significantly lower than the contralateral foot in all tasks both in absolute and normalized data (p = 0.008 - 0.012).

In DF, the peak torque in isomeric contraction was statistical significant higher than in concentric contraction in both the affected and contralateral foot, as well as in both absolute and normalized data (p = 0.012 - 0.028). In PF, there were no statistical significant differences between the isometric and concentric contractions in neither the affected nor the contralateral foot, due to more variation between subjects (p = 0.401 - 0.859).

Although statistical significant differences, all peak torque median values were approximately the same across all tasks in the affected foot. In the contralateral foot, the median peak torque was somewhat higher in PF than DF, especially in the concentric PF.

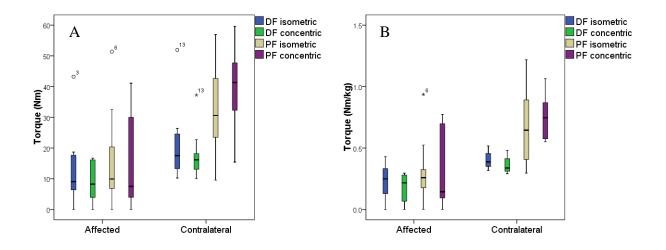


Figure 5. Boxplots illustrating isometric and concentric strength during dorsiflexion (*DF*) and plantarflexion (*PF*) of the affected and contralateral foot. *A*: absolute data. *B*: normalized to body weight.

Figure 6 illustrates the agonist muscle activation during isometric and concentric contraction for both affected and contralateral GM (A), SO (B) and TA (C), i.e. absolute values of EMG amplitude (RMS) around peak torque.

All the affected muscles showed statistical significant lower muscle activity than the muscles in the contralateral foot during both isometric and concentric contraction (p = 0.008 - 0.036).

In the affected foot, the agonist muscle activation was statistical significant higher in concentric than in isometric contraction in SO and TA (p = 0.011 - 0.025), but not in GM (p = 0.110). In the contralateral foot the concentric contraction was statistical significant higher in TA (p = 0.012), while not statistical significant different in GA and SO (p = 0.123 - 0.263).

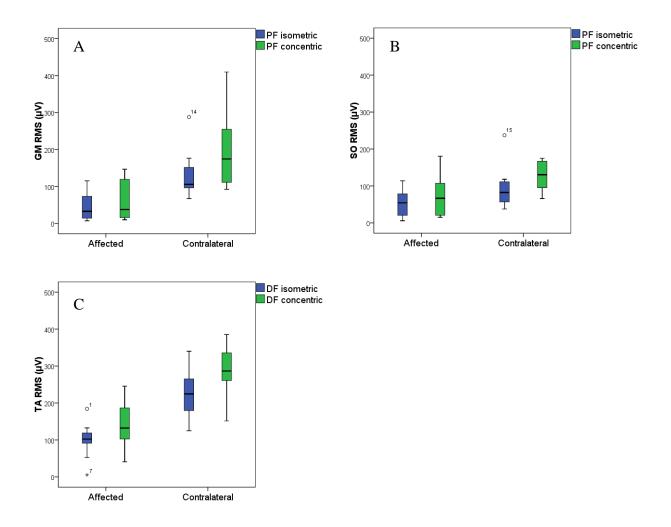


Figure 6. Boxplots illustrating agonist muscle activity (EMG RMS (μV) of ± 100 ms around peak torque) during isometric and concentric contraction for gastrocnemius (GM) during plantarflexion (PF) (**A**), soleus (SO) during plantarflexion (PF) (**B**) and tibialis anterior (TA) during dorsiflexion (DF) (**C**).

The results for RTD during DF and PF are presented for the affected and the contralateral side in figure 7 A (absolute data) and B (normalized to body weight).

In DF, the affected foot showed slightly lower RTD in the absolute data compared to the contralateral foot, but this difference was only close to statistical significance (p = 0.069). When normalized to body weight, the affected side showed statistical significant lower values compared to the contralateral side (p = 0.036).

In PF, the affected foot had statistical significantly lower RTD compared to the contralateral foot in both the absolute data and normalized to body weight (p = 0.012).

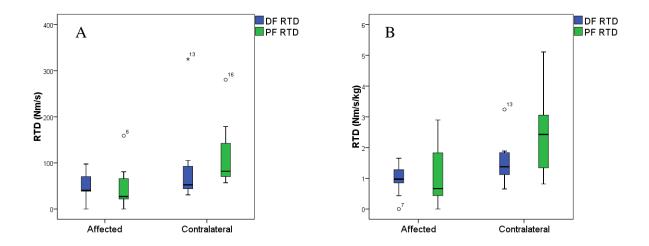


Figure 7. Boxplot illustrating rate of torque development (*RTD*) in dorsiflexion (*DF*) and plantarflexion (*PF*) of the affected and contralateral foot. **A**: absolute data. **B**: normalized to body weight (kg).

3.3 Muscle activation during the passive stretch test

Figure 8 illustrates the absolute and normalized (% of max EMG during MVC) muscle activity with increasing velocity during the passive stretch test for GM (A and D), SO (B and E) and TA (C and F).

Both the absolute data and normalized data showed a clear tendency to gradual increase in muscle activity with increasing velocity in GM and SO. In TA there was a more unclear response to the increase in velocity, but there was clearly higher muscle activity with high degree of variation during 180°/s compared to the lower velocities. As TA gave such unclear responses, and is furthermore not expected to be spastic, only the results of GM and SO are presented in detail.

In both GM and SO there were overall higher and more spanned muscle activity during passive stretch in the affected foot than in the contralateral foot. However, there were no statistical significant differences between the affected and the contralateral foot.

In GM, the difference between the affected and contralateral foot was close to statistical significance during 180°/s (p = 0.066) in the absolute data. Remaining p – values for GM ranged from 0.441 to 0.859. In the normalized data, the differences were overall closer to significance, with p-values ranging from 0.075 to 0.116

Neither in SO, the difference between the two feet was significant, with p-values ranging from 0.173 to 0.374 in the absolute data, and from 0.345 to 0.600 in the normalized data.

Regarding the increase in muscle activity with increasing velocity in the affected foot, the increases from 10°/s to 90°/s, and from 90°/s to 180°/s were statistical significant in both GM and SO in the absolute data (p = 0.008 - 0.013). In the normalized data there were also significant increases in both GM and SO (p = 0.018 - 0.043, except for GM from 10°/s to 90°/s (p = 0.128).

Increases in muscle activity with increasing velocity in the contralateral foot were also statistical significant (p = 0.021 - 0.028), except in GM from 90°/s to 180°/s (p = 0.314) in the absolute data. In the normalized data there were also significant increases in both GM and SO (p = 0.025 - 0.036), but not in GM from 90°/s to 180°/s (p = 0.263), while the increase in SO from 10°/s to 90°/s was only close to significant (p = 0.069).

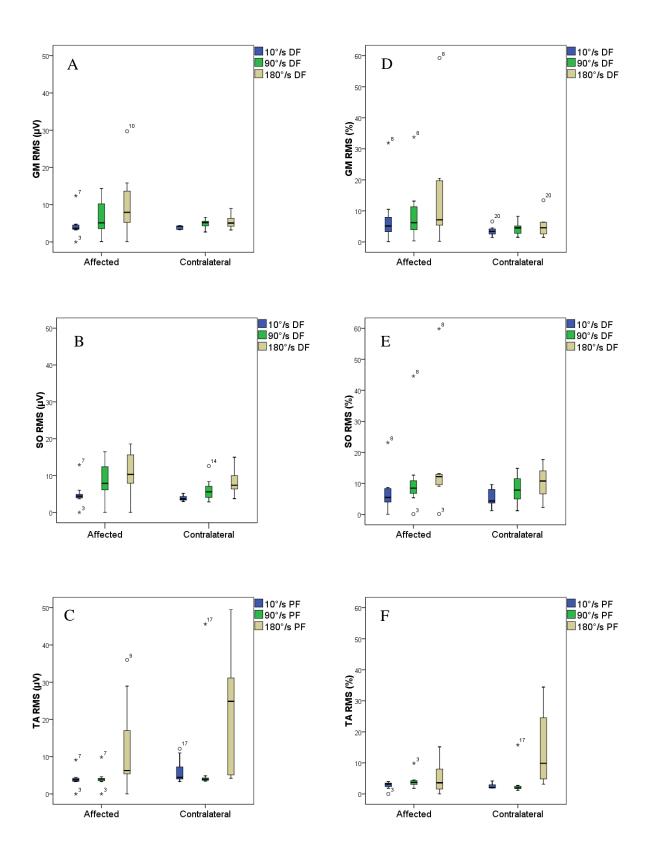


Figure 8. Boxplots illustrating absolute (A-C) and normalized (D-F) RMS (μ V) of average EMG with increasing velocity during the passive test for gastrocnemius (GM) and soleus (SO) during dorsiflexion (DF), and tibialis anterior (TA) during plantarflexion (PF).

3.4 Correlations

Table 3 shows correlations between muscle activity during passive stretch and muscle strength. With the exception of PF in concentric contraction there were mainly negative correlations.

The degree of the relationships was relatively spanned, ranging from little or no relationship to good relationships. Four of the correlations reached statistical significance (p < 0.05), and these were all found between isometric contraction (mainly in DF) and different velocities of the passive stretch test. There was no clear consistency in which velocity of the passive stretch test was most related to the strength variables.

the affected foot.							
		DF			PF		
		Isometric	Concentric	RTD	Isometric	Concentric	RTD
		(Nm/kg)	(Nm/kg)	(Nm/s/kg)	(Nm/kg)	(Nm/kg)	(Nm/s/kg)
		(n = 12)	(n = 12)	(n = 12)	(n = 12)	(n = 12)	(n = 12)
GM DF	10°/s	-0.68*	-0.13	-0.42	-0.13	0.17	-0.42
(% of max	90°/s	-0.05	0.283	-0.32	-0.65	0.40	0.32
RMS at MVC)	180°/s	-0.65	-0.02	-0.28	0.05	0.25	-0.28
(n = 9)							
SO DF	10°/s	-0.72*	-0.28	-0.35	-0.17	0.03	-0.05
(% of max	90°/s	-0.25	0.17	0.33	-0.80*	0.18	-0.35
RMS at MVC)	180°/s	-0.83*	-0.17	-0.33	-0.23	0.02	-0.10
(n = 9)							

Table 3. Correlations between muscle activity during passive stretch and muscle strength in the affected foot.

GM = gastrocnemius. SO = soleus. DF = dorsiflexion. PF = plantarflexion. RTD = rate of torque development.* = statistical significant (p = <0.05).

The correlations between the gait and strength variables for the affected foot are presented in table 4. There was a predominance of positive correlations, but mainly weak relationships, and none strong enough to reach statistical significance.

		DF			PF		
		Isometric	Concentric	RTD	Isometric	Concentric	RTD
		(Nm/kg)	(Nm/kg)	(Nm/s/kg)	(Nm/kg)	(Nm/kg)	(Nm/s/kg)
		(n = 12)	(n = 12)	(n = 12)	(n = 12)	(n = 12)	(n = 12)
PW	Gait speed (m/s/m)	0.01	0.40	0.20	-0.01	0.33	0.33
(n = 12)	Step length (m/m)	0.18	0.12	0.20	-0.13	-0.07	0.15
	Single support (%)	-0.01	0.31	-0.12	0.39	0.44	0.50
FW	Gait speed (m/s/m)	-0.03	0.27	0.07	0.15	0.23	0.36
(n = 12)	Step length (m/m)	0.23	-0.04	0.11	0.20	-0.18	0.18
	Single support (%)	0.03	0.16	-0.38	0.45	0.24	0.33

Table 4. Correlations between gait, and muscle strength variables in the affected foot.

PW = *preferred* walking speed. *FW* = *fast* walking speed. *DF* = *dorsiflexion*. *PF* =

plantarflexion. RTD = rate of torque development.

The correlations between gait and muscle activity during passive stretch in the affected foot are presented in table 5.

For gait speed, there were mainly little or no relationships to muscle activity during passive stretch, both during PW and FW.

Step length showed solely negative relationships to muscle activity during the passive stretch test, and there was a predominance of fair to moderate and good relationships. Two correlations reached statistical significance, both between GM and different velocities.

Phase of single support did on the other side show mainly positive correlations with a predominance of fair to moderate and good relationships. Two correlations reached statistical significance, both found during PW.

GM DF SO DF (% of max RMS at MVC) (% of max RMS at MVC) (n = 9)(n = 9)10°/s 90°/s 180°/s 10°/s 90°/s 180°/s PW Gait speed (m/s/m) 0.17 0.10 0.10 0.00 0.18 0.37 (n = 12)Step length (m/m) -0.52 -0.30 -0.75* -0.43 -0.05 -0.27 Single support (%) 0.77*0.05 0.65 0.65 -0.03 0.67* FW Gait speed (m/s/m)0.13 -0.27 0.03 0.13 -0.10 0.35 (n = 12)Step length (m/m) -0.55 -0.80* -0.65 -0.38 -0.57 -0.33 Single support (%) 0.53 -0.10 0.50 0.37 -0.18 0.45

Table 5. Correlations between gait variables and muscle activity during the passive stretch test in the affected foot.

PW = preferred walking speed. FW = fast walking speed. GM = gastrocnemius. SO = soleus. DF = dorsiflexion. PF = plantar flexion. * = statistical significance (p = <0.05).

4.0 Discussion

The main findings did confirm that the affected foot was significantly more impaired than the contralateral foot in terms of isometric and concentric strength, RTD and agonist muscle activation. The affected foot did also show tendencies to more muscle activation in the plantarflexors during the passive stretch test compared to the contralateral foot, although these differences were not statistical significant. Contrary to what was expected, the affected foot did not have shorter step length than the contralateral foot, but in line with the expectation, phase of single support was reduced in the affected foot. The results did to a great extend disprove the expectations of strong positive correlations between the strength and gait variables, and strong negative correlations between muscle activity during passive stretch and gait variables.

In the following, the results from the testing and the testing methods will be discussed, with main focus on evaluation of the measurements of function in these subjects. The testing of strength and spasticity in the dynamometer will be discussed at first, followed by the gait test. Finally, the relation between muscle weakness, muscle activation during the passive stretch and gait will be discussed with main focus on the affected foot.

4.1 Gait

The aim of the gait analysis was to get a picture of gait function and to identify possible impairments in the affected foot versus the contralateral foot. The question here is whether the variables gait speed, step length and phase of single support reflects function in this group.

As the phase of single support was the only variable that was statistical significant lower in the affected foot compared to the contralateral foot, this was perhaps a more sensitive outcome variable than step length. As phase of single support reflects the ability to balance on one foot, the balance may have been impaired in the affected foot. This is also in line with findings stating that when one foot is affected, a person tends to spend shorter time on this foot, and longer time on the contralateral foot (34).

Contrary to what was expected, step length was not lower in the affected foot than the contralateral foot, and the ability to take longer steps from PW to FW was present in both feet. This may indicate that possible muscle weakness and/or spasticity in the affected ankle

does not interfere with step length, alternatively that the step length in the contralateral foot is also impaired because of the affected foot, as a tactic to avoid an asymmetric gait pattern.

When comparing the absolute gait speed at PW in this group to that of healthy children, we see that gait speed is slightly lower in this group of subjects, as we can see that 12-year olds had a mean speed of 1.24, while the subjects in this study with a mean age of 12.9 had a mean speed of 1.08 (34).

4.2 Muscle weakness

The main question regarding the strength testing was whether muscle weakness could be identified in the dynamometer. There are both pros and cons regarding the experimental testing procedures in this study.

The horizontal position in the dynamometer seemed to cope well with the problems of passive forces which may otherwise have hampered testing of calf muscles in these subjects.

We found that strength evaluated by all types of contraction, i.e. isometric and concentric in DF and PF, was significantly lower in the affected foot compared to the contralateral foot. This indicates that strength testing in a dynamometer offers a good method for identifying ankle weakness in this group. Correspondingly, the EMG data confirmed that there was more agonist activation in the contralateral foot compared to the affected foot in all muscles.

There were, however, some limitations accompanying the strength testing in the dynamometer. First, there were problems linked to the fact that the dynamometer was not directly designed for the horizontal position. The main challenge was to manage proper fixation and standardization of position across participants. The position of the knee at 40° was the only body part that was objectively standardized with an angle gauge. The rest of the body position was judged by the eye, and no joint were fixated except for the foot tested that was attached to the foot pad.

Second, there were also some restrictions during the isokinetic strength testing, as two of the subjects did not manage to generate enough concentric force in DF in the affected foot for the dynamometer to initiate and maintain an isokinetic phase of 10%s, as this demands a certain torque threshold. This can be associated to findings showing that dorsiflexors can be difficult to activate in isolation (31).

Third, the present study initially included eccentric contraction, but due to problems both in the dynamometer settings and the participant's understanding of the task, the eccentric contraction was cut from the protocol.

Comparing the results to normative data for isometric torque values in healthy children, both DF and PF were impaired (59) in the affected, as well as the contralateral foot. The PF strength was the relative most impaired compared to healthy children, especially in the affected foot. Results also pointed towards isometric strength being more impaired than concentric strength. Isometric strength is normally expected to be higher than concentric strength as one can normally generate less torque with increasing velocity. In this group however, there were indistinguishable differences between the torque generated in isometric and concentric contraction both in the affected and contralateral foot. This indicates that the concentric contraction may have been easier to perform. This consideration is reinforced by the finding that EMG data showed overall higher agonist activity during the concentric contractions compared to the isometric contractions. However, it may also be due to an order effect in the testing protocol, which is discussed below.

The fact that the affected foot was found to be impaired compared to the contralateral foot, as well as to normative data, is most likely mainly explained by factors linked to the neuromuscular disorders in these subjects. In line with what we expected, the results indicate an impaired ability to voluntarily activate the affected agonist muscles, which is likely due to poor selective muscle control (60). The findings that the contralateral foot was also impaired compared to normative data may for instance be explained by a lower activity level, which is one of the questions raised by Wiley and Damiano (8).

Regarding data interpretation, one remark is worth mentioning. Passive muscle properties may interfere with the measure of *active* torque that is generated, especially in subjects with spastic CP, as they tend to have tense muscles, and may even have contractures. To try to avoid the effect of passive muscle properties, different methods were used for the isometric and concentric contraction. In the isometric contractions, the baseline value registered during the inactive period before the MVC was subtracted from the peak torque obtained during the MVC. In the concentric contractions, where the muscle length changes through the range of motion, this was done by subtracting the torque measured at the same angle during the passive stretch test at 10°/s from the peak torque obtained during the concentric test at 10°/s.

However, one issue with the latter was the finding of that the muscles were not fully relaxed during the passive stretch test.

Overall, there were perhaps more drawbacks than benefits using the dynamometer for this group of subjects, especially considering the misfit for the smallest and weakest subjects. A simpler ankle dynamometer like that of Moraux et al. (59) may perhaps be a better alternative for testing weak subject.

4.3 Muscle activation during the passive stretch test

The main question regarding the passive stretch test was whether we actually measured spasticity or not. In line with an aspect of the definition itself, it was expected that the muscle activation in the plantarflexors being stretched would increase with increasing velocity in the dynamometer. The results did to a large extend confirm this anticipation, which at first sight indicates that spasticity is identified. However, the muscle activity in the affected foot was not significantly different from the contralateral foot. As the contralateral foot should not show spastic reactions, this questions the validity of the measurement. Moreover, unpublished data showed that even the muscles that were not in stretch, i.e. in flexion, showed the same pattern with increased muscle activity with increasing velocity in both the affected and the contralateral foot.

One possible explanation to all this may be that it is difficult to relax the muscle completely in this type of task, and that this is even more difficult with increasing velocity. This may also explain why TA, which is not expected to be spastic, showed a major increase in muscle activity at the highest velocity in both the affected and the contralateral foot.

Another factor is that we cannot really know whether EMG activity during the passive test is a result of spasticity or voluntary activation. It has also been suggested that speed-dependent effects that are found cannot necessarily be attributed to spasticity, but may be a result of limited selective control. This has been shown to be associated with CP, especially at higher velocities (61).

There was, however, a large variation in the muscle activation, indicating that some may have and some may not have spasticity. This underlines the importance of individual follow-up to identify who will benefit from treatment like BTX-A, and who will not. Regarding data interpretation, there were originally two outcomes of the passive stretch test; torque, i.e. muscle resistance against passive stretch, and EMG, i.e. muscle activation during passive stretch. As for the torque data, the results of the highest speed were too difficult to analyze due to missing isokinetic phases, so that the torque values were to a large extend affected by the rapid accelerations. Therefore, only the EMG results were included in further analyses. The fact that the torque output could not be analyzed weigh against the usefulness of the dynamometer for measuring spasticity.

In conclusion, the measurements in the isokinetic dynamometer were probably not valid for identifying spasticity, as the results were perhaps due to a general response of inability to relax the muscles. It may however be that this group of subjects had only mild or no spasticity, and that a clearer difference between the affected and the contralateral foot would be present with more severe spasticity.

4.4 The relation between muscle strength and muscle activity during passive stretch

There was a predominance of negative correlations between strength and muscle activity during the passive stretch test, which indicates that weak subjects have more muscle activity during the passive stretch test. One exception was concentric contraction during PF, which solely showed (weak) non-significant positive correlations.

As isometric contractions involve unchanged muscle-tendon length, stretch responses should not occur. This differs from concentric contractions, where the agonist is shortening and the antagonist is stretching. Hence, a stretch response can occur in the antagonist muscle during a concentric contraction, potentially causing impaired muscle force production in the agonist muscle (24). In divergence with this, isometric contraction did correlate negatively with antagonist activity during different velocities in the passive test, while the concentric contraction did not.

The finding that the muscle activity during passive stretch did not show any good negative relation to the concentric contraction may indicate that the passive stretch test does not reflect spasticity, or just that other neurological factors associated with CP contributed more strongly to the impaired strength in the affected foot.

4.5 The relation between gait and muscle strength

As the plantarflexors are the main muscles for propulsion in gait, we expected a strong positive relation between PF and step length, and thereby also gait speed. Strength in DF could also be positively correlated to step length and gait speed, as sufficient dorsiflexion will make sure that the toes do not drop against the floor which would hamper the step length. In contrast to what we expected, we found a variety of non-significant positive and negative correlations, indicating that neither isometric nor concentric strength, as well as RTD in the ankle do not interfere with step length and gait speed.

These results are also in contrast to studies that have found strong positive relationships between strength in the lower extremity and gait speed and stride length (35, 62, 63). One possible explanation is different testing procedures and/or different subjects.

As phase of single support may reflect balance, and balance depends much on strength in the lower extremities (40) it may be expected that increased phase of single support correlates positively with increased strength in the calf muscles. However, the findings showed no consistent relations between these variables, as there was a variety of positive and negative correlations between phase of single support and the different contraction types. As we found that phase of single support was actually impaired in the affected foot compared to the contralateral foot, this may mean that other factors are more determining for the phase of single support.

Regarding RTD, it is a fact that gait demands relatively rapid limb movements, and thus, the ability to contract the muscles fast enough is a critical aspect of gait function. We expected that this could be even more determining for gait function than the maximal force generation. However, there was not found stronger correlations between RTD and gait parameters than between maximal strength and the gait parameters in this study. These results are in agreement with the results of Moreau et.al (10) who found that rate of force development was not predictive for temporal-spatial gait measures.

It may be that the difficulty of the walking task was too easy for the impairment to have any effect on gait function. This would be in accordance with the conclusion of Abel et.al (64) who studied the relationship between musculoskeletal impairments and functional health status among ambulatory children and adolescents with spastic CP.

To sum up, no relation was found between the gait and strength variables, contrary to what was expected. This may indicate that ankle weakness is not a major determinant for gait function, or alternatively that the measuring methods were not good enough to detect any possible relation between them.

4.6 The relation between gait and muscle activation during passive stretch

Since we are not sure whether the test that aimed at measuring spasticity actually *did* measure spasticity, it is difficult to draw any conclusions on a possible relation between spasticity and gait based on these results. Hence, the discussion below is probably not directly based on the relation between gait and *spasticity*, which originally was one of the aims of this study.

The results were relatively far from what was expected beforehand. Regarding gait speed, there was a predominance of very weak positive correlations to gait speed. In other words: the degree of ankle muscle activity during passive stretch did not seem to interfere with gait speed.

Regarding step length, the results were more in line with the hypothesis, i.e. fair to good negative correlations. This may indicate that there is an aspect of the muscle activity during passive stretch that interferes with the ability of taking long steps.

The relation between single support and the passive stretch test was also interestingly not what we expected. If the muscle activity during passive stretch is expected to be a negative matter, then the phase of single support would be expected to decrease with increasing passive stretch response. In contrary to this, there were almost solely moderate to excellent positive correlations between these variables with the exception of 90°/s (which seem to be an irregularity). This indicates that there is an aspect of the muscle activity during the stretch test that is actually beneficial for the phase of single support and hence for balance. It may be that the stretch response contributes to stabilization of the foot, and function as a coping mechanism during gait.

Regarding the difference between PW and FW, the muscle activation during the passive test was expected to correlate stronger with gait variables during FW as spastic reactions are expected to increase with increasing velocity. As it did not, this may indicate that the results are in harmony with the thought that the passive test did not actually reflect spasticity.

The uncertainty of whether it was spasticity that was measured makes it difficult to compare the results to other literature. It is for instance found that there are minor or no relationships between spasticity in the lower extremities and gait speed and step length (63), while in this study we found strong relations between step length and muscle activation during passive stretch. However, one should not without consideration take for granted that other studies have succeeded in identifying spasticity.

4.7 Limitations

This study has some limitations worth mentioning. For one thing, the group of subjects is relatively small and heterogenic, which did not render possible stratified analyses where one could control for factors like age, sex and GMFCS level. This also compelled the use of correlation analyses, mostly due to non-normally distributed data, instead of regression analyses which would be preferable to get a clearer picture of how the variables are related to each other. Also, it is difficult to attain statistical significant results with this sample size.

The lack of a control group did not provide us with the possibility of validating the test protocol in the dynamometer on healthy subjects. This may have given us a better foundation for interpreting the equipment's ability to detect possible spasticity.

Moreover, intertest reliability has not been performed, and hence we do not know if the results would have been similar if we performed re-tests. Even though the time of day was about the same for all subjects, there may have been factors affecting the results that may vary from day to day.

There may also be some limitations associated with the testing protocol. For one thing, there may be an order effect in the strength testing in the dynamometer. As the concentric contraction test was performed before the isometric MVC test with a maximum of a few minutes in-between, the peak torque and RTD values may be underestimated if fatigue was an issue for some subjects. It is also thinkable that the generation of MVC is dependent on which foot was tested first. If there is an issue of general fatigue here, it will underestimate the MVC of the contralateral foot, as the affected foot was tested first. It is also questionable how well the subjects could generate MVC after approximately two hours in the lab. However, this will only lead to a greater difference than we were able to detect, and hence the relations we have found may only be stronger.

Furthermore, normalization to EMG at MVC may be problematic in subjects with CP, due to the possible reduced capacity to fully activate the agonist muscle. This was evident in at least two subjects, as we could see that they showed more activation in the affected muscles during the passive test, where the task was to *relax* the muscles as much as possible. Consequently, the normalized EMG RMS during the passive stretch test may be overestimated in the affected foot if the agonist muscle was not fully activated during the MVC test.

4.8 Clinical implications

Regarding the ankle strength measurements, results from this study indicate that ankle weakness does not interfere with gait function, and hence that possible weakness in the ankle is perhaps not what one should emphasize in the search of factors that impair gait function.

When it comes to the testing of spasticity, it is difficult to draw conclusions of whether there is an association with function or not as it is uncertain whether spasticity *was* measured. However, this study underlines the importance of making sure that the measurements are valid, as well as reliable, for their purposes so that the tests can form a basis for the best possible treatment for each patient.

5.0 Conclusion

The concentric and isometric dynamometer tests, along with EMG recordings, did likely provide a valid measurement for ankle strength as the affected foot proved to be weaker, as well as showing lower agonist muscle activity than the contralateral foot. Whether the isokinetic testing in the dynamometer offered a valid measure of spasticity in the affected ankle is more uncertain, as the muscle activity during passive stretch was not significantly different from the contralateral foot. Regardless, as the horizontal positioning in the dynamometer posed certain issues regarding fixation and standardizations of the joints, this should be improved if similar testing in the dynamometer is to be used in future testing of the ankle.

Step length was not significantly different in the affected and the contralateral foot, while the phase of single support was significantly shorter in the affected foot. This indicates that balance may be impaired in the affected foot.

Ankle weakness did not relate to the gait variables in this group of subjects, suggesting that other factors are more determining for gait function. Ankle muscle activity during passive stretch was, on the other hand, negatively related to step length, and positively related to phase of single support. There was no relation between muscle activity during the passive stretch and gait speed.

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