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Signs of spasticity during walking in children with spastic cerebral palsy

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Abstract

Introduction: Spasticity is one of the most common neurological impairment that can lead to gait problems in cerebral palsy (CP). It limits function by reducing normal muscle lengthening and voluntary activation during gait. Assessments of spasticity is usually done in a clinical setting by passively stretching the muscle and classifying the resistance felt, but its relation to spasticity during gait is not clear. During the mid-stance phase in the gait cycle the calf muscles are stretched, possibly resulting in a spastic contraction.

Aim: The aim of the study is to see if there is a relation between muscle activity during walking and a passive test for spasticity, in children with spastic cerebral palsy.

Method: Twelve children with CP, 7-16 years and 7 typically developing (TD) children 6-15 years conducted a 3D gait analysis with surface EMG from m. soleus (SOL) and m. gastrocnemius medialis (GM). Subjects were instructed to walk at self-selected walking speed. The EMG signals were band-pass filtered (20-400 Hz) and the root mean square amplitude was calculated in ten 10 % periods during stance and normalized to its maximal. The degree of spasticity was evaluated with the clinical Tardieu Test with the knee flexed in 90 degrees. Both limbs in bilateral CP, most affected limb in unilateral CP and left limb in TD were used. The limbs from the CP participants were classified upon clinical score from Modified Tardieu Scale: 1) no catch, 2) catch.

Results: Legs of children with CP with and without catch had significantly more activation in GM and SOL during 10-20 % of stance compared to TD ($p<0.05$). CP with catch had statistically non-significant more GM and SOL activation in that period than CP without catch ($p=0.335$, $p=0.338$). Both CP groups had higher ankle angular velocities during 10-20 % than TD children ($p<0.05$).

Conclusion: While the GM and SOL of TD are hardly active during 10-20 % of stance, those muscles and especially the SOL were more active in children with CP. Although not statistically significant, this seems to be more present in children with a clinically detected catch. This indicates that it is possible to observe signs of spasticity in stance phase during walking. Ankle angular velocities were higher during the Tardieu Test than during stance. This may point to a discrepancy between gait and the Tardieu Test, in relation to how a spastic reflex is elicited during the passive test, and in normal walking.

Sammendrag

Bakgrunn: Spastisitet er en av de vanligste nevrologiske forstyrrelsene som kan føre til gangavvik hos barn med cerebral parese (CP). Det hemmer funksjon og reduserer normal muskelforlengning og frivillig aktivering under gange. Dette testes vanligvis klinisk ved å passivt strekke muskelen, og klassifisere den følte motstanden i vevet, uten at relasjonen til gange er godt kjent. Under midt-stand fasen i gangsyklusen blir leggmusklene strukket, og muligens resultere i en spastisk reaksjon.

Mål: Målet er å se på relasjonen mellom muskelaktivitet under gange og i en passiv klinisk test for spastisitet hos barn med spastisk cerebral parese.

Metode: Tolv barn med CP, 7-16 år og 7 funksjonsfriske barn, 7-15 år deltok i en 3D ganganalyse med overflate EMG fra m. soleus (SOL) og m. gastrocnemius medialis (GM). Subjektene ble instruert til å gå med selvvalgt ganghastighet. EMG signaler ble filtrert med et båndpassfilter (20-400 Hz) og effektverdi amplituden ble kalkulert i ti 10 % perioder i standfasen normalisert til maksverdi. Graden av spastisitet ble vurdert ved den kliniske Tardieu Testen med kneet i 90º fleksjon. Begge bein i bilateral CP, mest affiserte i unilateral CP og venstre bein i funksjonsfriske ble brukt. Bein fra CP-subjektene ble delt inn på bakgrunn av Modifiserte Tardieu Skalaen: 1) no catch 2) catch

Resultat: Bein fra CP barn med og uten catch hadde en signifikant høyere muskelaktivering under 10-20 % av standfasen enn funksjonsfriske i GM og SOL ($p<0.05$). CP med catch hadde en statistisk ikke-signifikant høyere aktivering i GM og SOL enn CP uten catch i samme perioden ($p=0.335$, $p=0.338$). Begge gruppene med CP hadde høyere ankelangulære hastigheter under standfasen enn funksjonsfriske ($p<0.05$)

Konklusjon: Under 10-20 % standfasen var det lite aktivitet i GM og SOL hos funksjonsfriske, men høy aktivitet, spesielt i SOL hos barn med CP. Selv om det ikke var statistisk signifikant, virker den til å være mer aktiv hos barn med en klinisk påvist catch. Dette tyder på at det er mulig å oppdage tegn til spastisitet i løpet av standfasen under gange. Høyere angulære hastigheter i ankel under Tardieu-test enn under gange kan tyde på at den spastiske reaksjonen skjer med ulik hastighet i de to ulike situasjonene. Derfor kan denne relasjonen muligens trekkes i tvil.

Acknowledgement

First, I would like to thank my supervisor Siri Merete Brændvik for much appreciated guidance in this thesis and for insightful discussions. I would also like to thank my co-supervisor Karin Roeleveld for writing a matlab script for data analysis and for valuable guidance, co-supervisor Tobias Goihl for technical support and guidance and last, but not least, Jussi Nurminen for providing a script to analyze Tardieu Test data.

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Finally, I would like to thank Søren Kierkegaard for telling me that I will regret it either way

Contents

1. Introduction

Cerebral palsy (CP) is an umbrella term for a group of motor deficiencies affecting movement and posture resulting from a non-progressive lesion in the developing child's brain (1). The injury can occur before, during or after birth (2). The primary brain lesion strains the developing child's brain and secondary neuromuscular deficits occur, laying the foundation of gait and movement problems in CP (3). CP is also the most common motor disability in childhood affecting approximately 2-3 per 1000 live-births in the Western world (4, 5). Its heterogeneous nature gives it a wide clinical spectrum with many different ways for pathologies to develop (6). Although the primary brain lesion is static, secondary musculoskeletal impairments develops over time (1). This comes as a result of abnormal muscle tone and loss of voluntary muscle action which leads to reduced joint range of motion and increasing joint stiffness (7). Over time these impairments leads to deterioration in gait and reduction in muscle strength in adolescence and young adulthood in those diagnosed with a spastic-type CP (8).

Motor disorders are the key feature of CP with its deviance from a normal child development (1). Level of abnormality or aberration from normal development depends on which type of CP is diagnosed. The most common type of CP is a spastic-type, it affects approximately 80-90 % of those diagnosed with CP in European populations, either hemiparesis, one side more affected than the other, or diplegia where both sides are affected (9). Other main types of CP are ataxic and dyskinetic CP. These types involve a smaller percentage of the population with CP and have other key characteristics than a spastic-type (3, 9). One way to classify children with CP is with The Gross Motor Function Classification System (GMFS) (10). This classification seeks to enhance function rather than limitations. GMFCS groups children with CP into five based upon motor function in the lower body. Because of the ambulant function, from those who must be transported in a wheelchair, to the once having an ambulant function, walking ability will be reserved to the lower-level groups. In children with an ambulant function, or milder variants of CP, gait is affected, and abnormalities are commonly seen (2, 3).

Gait in children with CP deviates from typically developing children (TD) by an unfavorable use of muscles during gait. One characteristic that contributes to this problem is a diminished neural drive from the central nervous system, leading to alteration in skeletal muscle growth and neuromuscular activation (11, 12). Functional alterations in muscle strength and reflexive and passive resistance reflects these changes in muscle development (11).

Walking consists of two primary phases: stance and swing, defined as a gait cycle (Figure 1). One gait cycle - or stride, starts when one foot hits the ground and ends when same foot hits the ground again. Stance lasts for 60 % while swing 40 % of a normal gait cycle. Further on, stance can be subdivided into initial contact, mid-stance and terminal stance (13, p.42-46). During these phases it is expected that the joints go through different velocities and angles that will affect a spastic muscle in various ways accordingly. What is usually seen then is out of phase, continuous, premature or delayed use of calf muscles during walking (14). In the transition from ankle plantar-grade to dorsiflexion during the stance phase, the calf muscles are being stretched. During this transition, increased activity in spastic muscles is suggested to occur (15, 16). Spasticity is also predicted to occur when there is knee extension, since both m. soleus (SOL) and m. gastrocnemius medialis (GM) will be stretched during this period. Spasticity can also be triggered in swing (17), but this study will be focusing on mid-stance during the stance phase.

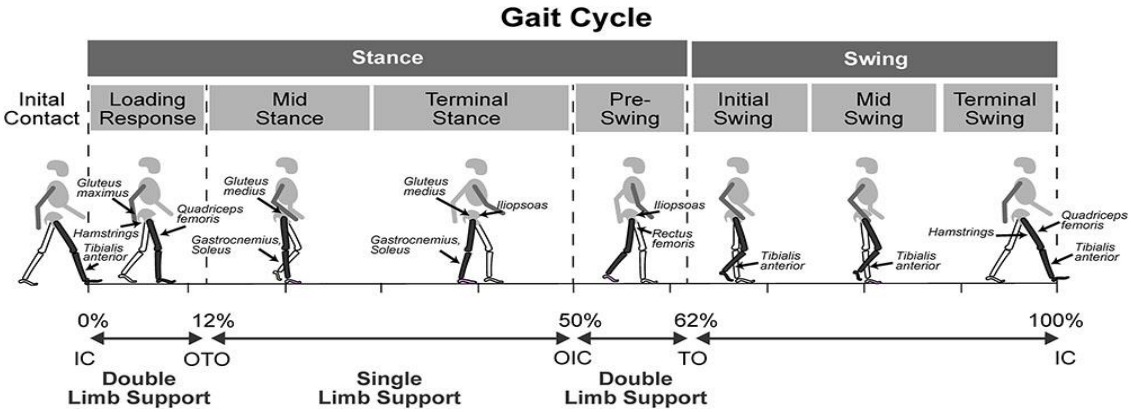


Figure 1: Illustration of a normal gait cycle with sub phases of stance and swing (3)

Spasticity is the primary neurological impairment in children with spastic-type CP. It is a common and complex phenomenon and a part of the upper motor neuron syndrome (UMNS) which can relate to both neural and non-neural components of the muscle (18). UMNS consists of positive features like spasticity and negative features like muscle weakness and loss of dexterity (19). One definition that seeks to embark on the main feature of spastic-type CP from UMNS, comes from Lance (20): ‘’*a motor disorder characterized by a velocity dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflexes, as one component of the upper motor neuron syndrome*’’.

Most common interventions used to treat spasticity consist of neurosurgery, selective dorsal rhizotomy or injections of botulinum toxin (BoNT), to try correct deviations in the pathological gait pattern, and improve active function (21, 22). Most treatments are used to improve gait and through reducing spasticity. Clinical scales assess the spasticity in each patient and contribute to the preferable treatment for the individual. Since the outcome of these scales are used when proposing treatment for this group of CP children, it is crucial to have a clinical tool that reflects function in everyday activities.

Definitional consensus lacks regarding Lance's (20) velocity-dependent definition of spasticity and between those who want a broader term. (18, 23, 24). Some definitions are more general by saying that it is a sensorimotor problem presenting itself as intermittent or sustained non-voluntary action of the muscles being used (25). For it to be conceptualized and accepted as a broad term it is necessary to try to look at what is meant by the definition and consider its relationship within a functional context (26).

Spasticity manifests itself by a tightening of the muscle and possibly surrounding tissue when it is rapidly being stretched (27). This results in deterioration of function, pain levels and long-term mobility outcomes for this patient group (28). Clinical measurements are the primary way of attaining knowledge of the role spasticity plays during walking, but its relation to spasticity during gait is not clear (29-32). One explanation could be the way spasticity is measured and interpreted is not aligned with the phenomenon itself. As well as in what way it actually contributes to gait difficulties in CP. One of the most used clinical tests to assess spasticity is the Tardieu Test (33). Assessment of spasticity in the lower limbs is performed in a supine position with the knee fully extended, evaluating GM, and the knee flexed to 90º, evaluating SOL. The muscles are tested passively in the shortest position to longest position at two different velocities, one below and one above the threshold velocity for triggering a spastic muscle reaction (34). The difference between the angle of arrest at both velocities is used to differentiate between spasticity and contracture in SOL and GM. Boyd and Graham (35) updated the original clinical scale from the Tardieu Test called Modified Tardieu Scale (MTS), and created two terms, R1 and R2, where a larger difference between these two angles indicating a more dynamical component. This test distinguishes between passive resistance in the tissue and spastic reflex activity by showing to smaller and larger differences in angle measurements during the test.

The aim of this study is to see if there is a relation between muscle activity during walking and a passive test for spasticity, in children with spastic cerebral palsy. Then following research questions will be addressed: How does a Tardieu Test reflect how spasticity may manifest itself during mid-stance? And are there differences between CP children assigned with or without a clinical catch?

2. Methods

2.1. Participants

The study participants consisted of 12 children and adolescents (from now on called children) with CP, 9 boys and 3 girls, [mean age (range): 10,6 (7-16)] and 7 typically developing (TD) children, 4 boys and 3 girls, [10,9 (6-15)]. TD children were recruited by employees from St.Olavs University Hospital and the Norwegian University for Science and Technology in Trondheim, Norway. Eight participants from the CP group were taken from an ongoing randomized clinical trial (The WE-Study), which tests the effects of botulinum toxin A on walking for children with CP. Four participants from the CP group were taken from clinical examinations as a link in a regular follow-up at the hospital.

Included patients were chosen on a basis of no botulinum toxin A-injections six months prior to testing, as well as no prior surgery in the lower limbs during the last two years, Gross Motor Function Classification System (GMFCS) (10) levels I-II, a diagnose of spastic unilateral or bilateral CP and ability to receive verbal instructions. Typically developing children were recruited locally by staff and recruitment leaders of the WE-Study. The inclusion criteria for TD children were an absence of any neurological problems. Written consent was given to either guardian or parent, for use of their data in research. The study was approved by Regional Committee for Medical and Health Research Ethics (REK).

2.2. Equipment and procedure

Participants went through a testing protocol in three settings: 1) Anthropometrical measurements 2) Tardieu Test to evaluate passive spasticity and 3) 3D gait analysis, both tests included sEMG recordings. During walking at a self-selected walking speed, gait kinematics, spatiotemporal parameters and muscle activity data using 3D motion capture analysis were collected.

2.2.1. Equipment

Neuromuscular activation was recorded during both test settings bilaterally with bipolar surface electromyography (sEMG) from the m. gastrocnemius medialis (GM) and m. soleus (SOL), with a 9-channel sEMG system (Myon AG, Baar, Switzerland) with a sampling rate of 1000 Hz. Ambu BlueSensor N electrodes (Ambu AS, Ballerup, Denmark) were prepared and placed over the participants muscles according to SENIAM recommendations (36).

To capture the assessments, an 8-camera VICON system (Vicon-UK, Oxford, UK) was used to detect marker trajectories, set at a sampling rate of 200 Hz. Nexus software (Oxford Metrics, Oxford, UK) was used to define gait cycles, calculate spatial parameters, joint centers and to estimate joint angles.

2.2.2. Anthropometrics

The participants measured body weight (kilograms), height (centimeters) and leg length measurements (centimeters) to estimate joint center locations and segmental inertia parameters.

2.2.3. Tardieu Test

For the Tardieu Test, subjects were placed on a bench for measurement of leg length with a measuring tape. Markers were then placed on the lateral malleolus of ankle, the upper third of the second metatarsal bone and the fibula head to create two vectors, leg and foot, to calculate between them to reflect dorsiflexion and plantarflexion of the ankle. Thereafter, the subjects were first placed in 90º and then 180º angle in the knee to initiate the Tardieu Test. Two clinical examiners were responsible for the test procedure, where one performed the test on the participants and one measured angles pre-test and monitored video recordings during the test. Testing conditions for all participants were equal in terms of temperature, time of day and the order of testing.

Resting anatomical position of the ankle was measured with a goniometer with 90º determined as zero, deviations from 90º were calculated to the nearest 5º (Figure 2). Then passive resistance was measured in a supine position for the most affected leg in unilateral CP, both in bilateral CP and left leg in TD.

After this measurement was completed a clinician started the Tardieu Test by placing a firm grip on the participant’s ankle and went through two velocities of movement: one very slow

movement from resting position to max plantarflexion (V1) and one movement as fast as possible through the range of motion (V3). One trial was conducted per movement in each leg. They were told to relax as best as they could when the test was performed. When sufficiently relaxed the examiner went through with the test. These procedures were done in the same way for both the 90º and the 180º knee angle positions.

Based on the test CP children were given a 5-item clinical score ranging from 0-4 called Modified Tardieu Scale (37), where 0 = no muscle reaction, 1 = mild resistance, 2 = clear catch, 3 = fatigueable clonus and 4 = unfatigueable clonus. The scale gives the clinician a subjective indication of spasticity. The clinical scoring from the Tardieu Test were gathered from either a patient's journals or from researchers connected with the gait lab at St.Olavs Hospital.

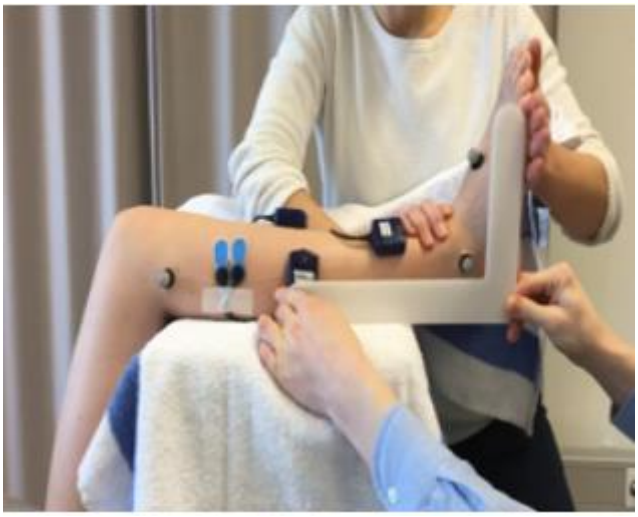


Figure 2: Screenshot from the starting position in the Tardieu Test 90º angle

2.2.4. 3D gait analysis

In the 3D gait analysis a lower-limb Plug-in-Gait model was used as the framework for the marker set-up placement (38). Reflexive markers were put on anatomical landmarks on the lower limbs of the participants

Two clinical examiners were responsible for the test procedure. One was responsible for instructions given during gait analysis, and the other responsible for the video recordings. The children were instructed to walk barefooted at a self-selected walking speed with the following instruction from a clinical examiner: ''Walk as you normally do'' along a 12-m

walkway. They were asked to continue back and forth in the lane until enough trials were captured according to the clinical examiner monitoring the recordings.

Three arbitrary trials with good quality in terms of kinematics and EMG data were selected from gait analysis, via Nexus. Both trials from the Tardieu Test were selected along with sEMG recordings.

2.3. Data analysis

The raw sEMG signal from both settings were filtered with a 4-th order Butterworth band-pass filter ranging from 20 to 400 Hz. Raw sEMG signals from these trials were visually inspected using Myon ProEMG (Myon AG, Baar, Switzerland) to exclude subjects with low quality EMG signals. The EMG root mean square (RMS) was used to smooth the signal for further analysis. Window width was set at 0.5 seconds for the 3D gait analysis at and 0.8 seconds for the Tardieu Test.

2.3.1. Tardieu Test

Data from both Tardieu 90º and Tardieu 180º was first processed in Nexus, then EMG and ankle kinematics were exported from Nexus to a Python (Python Software Foundation, Wilmington, DE, USA) script. The script allowed a selection of which leg to analyze from a Nexus software where a trial has been selected from either Tardieu Test 90º or Tardieu Test 180º angle. A window automatically generated a box with max EMG RMS from SOL and GM and maximal ankle angular velocity from each participant (Fig.3). Max ankle angular velocities were chosen to compare velocities in the Tardieu Test to angle angular velocities during walking. Max EMG RMS from SOL and GM were extracted from V3. In the same window R1 and R2 from V1 and V3 were subjectively marked out. R2 is the maximal dorsiflexion reached at the end of V1 and R1 is the angle at where the velocity-dependent muscle reaction occurred during V3. Spasticity angle was calculated by taking R2 minus R1 for children with CP using Boyd and Graham's MTS (35). These two angles gave an estimation of the velocity-dependent factor of spasticity along with joint ROM limitations.

Based upon the clinical scoring from the ordinal MTS, catch/no catch was defined with Tardieu Test 90º. Some overlapping occurred within the tests with 8 subjects defined as having a catch in both tests. Further on, CP children with a score from 0-1 were statistically coded as 1, and 2-3 coded as 2. TD children were given a score of -1 and coded as 0. Three groups were then generated on these terms: 1) CP with catch, 2) CP without catch and 3) TD.

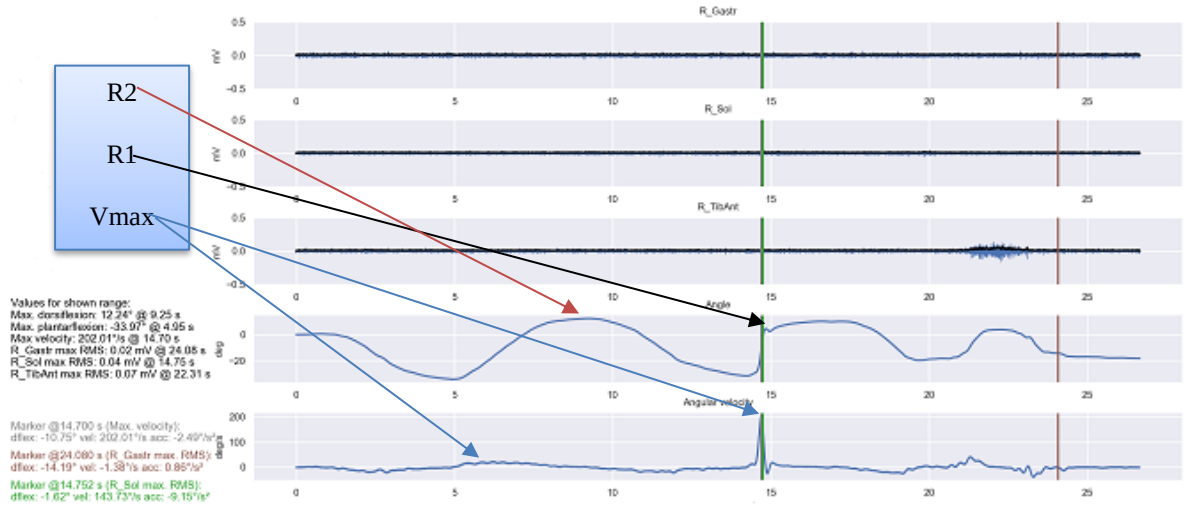


Figure 3: Screenshot of the output variables from a Tardieu Test 90º angle in a CP subject showing EMG (mV), angles (deg) and angular velocity (deg/s). R2 shown with a black line, R1 with a red line and Vmax with a blue line

2.3.2. 3D gait analysis

Nexus was used to process data and inspect gait cycle parameters from the 3D gait analysis. Two files were the generated per subject per trial resulting in one file for EMG data and one file for ankle and knee angles and angular velocities during this period. Kinematic and EMG data from Nexus was converted to a format (. ASCII) that could be read into Matlab (R2017a, MathWorks, Inc., Natick, MA, USA). These files were imported into a script in Matlab to analyze data into a coherent and visual material. Three arbitrary trials from the stance phase of the most affected leg in unilateral CP, both legs from bilateral CP and the left leg from TD were used. Data was then normalized to 0-100 % of the stance phase.

EMG, angle and angular velocity data were divided into 10 bouts of 10 % in the stance phase to get the mean change from one period to the next, along with max EMG RMS from 0-100 and 10-20 % of stance (Figure 4). 10 10% bouts from 0-100 were used to see where in the EMG pattern there were changes from between CP and TD. 10-20 % of stance phase was where GM was stretched along with knee extension and dorsiflexion of the ankle. To look at possible spastic EMG activity in the calf muscles during 10-20 % stance GM and SOL were chosen. To compare angles and angular velocities to Tardieu Test, peak average angles and angular velocities values from three trials were extracted from 0-100 % of stance phase and 10-20 % of stance shown in Figure 5. This was done for the Tardieu Tests in periods where spasticity is suggested and for normalization purposes. Maximal EMG from period from 0-100 % in stance phase were used to normalize EMG data for comparison between groups.

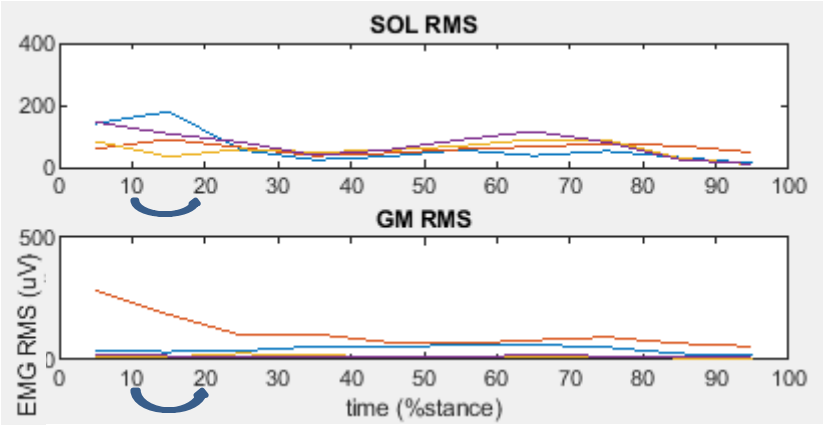


Figure 4. Average EMG RMS from SOL and GM from four legs in bouts of 10 % normalized to 0-100 % of stance. X-axis shows stance phase from 0-100 and y-axis shows EMG RMS in microvolt (uV). Colored lines are legs from three participants. Arrows point to the stance phase from 10-20 % with high SOL and GM EMG activity in CP children.

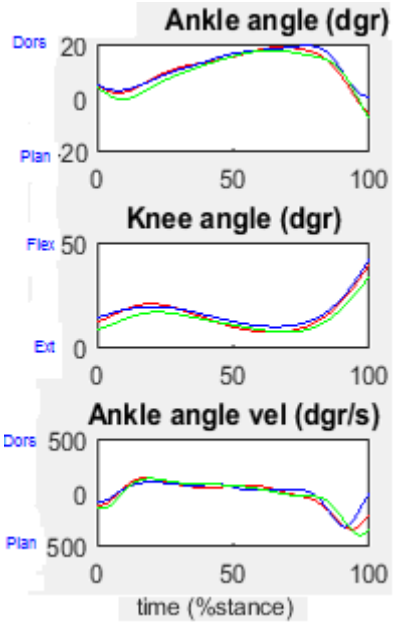


Figure 5: Screenshot of three trials (colored lines) from stance phase in one TD subject. From top to bottom: Ankle angle, knee angle and ankle angular velocities. X-axis show the stance phase from 0-100 % and y-axis show angle in degrees and angular velocities in degrees per second. Motion in dorsiflexion/knee flexion and plantarflexion/knee extension. 0 = fully extended knee.

2.4. Statistical analysis

The variables were first plotted into Excel 2016 version (Microsoft Inc., Redmond, WA, USA) then imported to other software for further analysis. IBM SPSS Statistics Pack version 25 (SPSS, Inc., Chicago, IL, USA) were used to analyze the different variables. To test for group differences in EMG activity, angles and angular velocities t-tests was used. Normal distribution of the data was checked with Shapiro-Wilk test and histograms with a normal distribution curve. Whether variables were normally distributed or not, an independent samples t-test or Mann-Whitney U-test respectively, were used. Those variables not normally

distributed were: Max RMS SOL Tardieu 180, Max RMS GM Tardieu 90 and 180 and SOL EMG RMS 10-20. Boxplots are presented as median for the variables included. SigmaPlot v.14 (Systat Software Inc, San Jose., CA, USA) were used make graphs of EMG activity of GM and SOL EMG during gait. All statistical analysis in SPSS was set at a two-tailed significance test with a significance level of $p < 0.05$. All measures of variability are expressed as mean/median ± standard deviation (SD).

3. Results

The results are presented in three parts. First background characteristics of participants in the three groups that are compared in this study. CP with catch (felt increased resistance to rapid stretch), CP no catch and TD children. EMG and kinematics during Tardieu Test will be presented first, then EMG and kinematics during gait.

Table 1: Background characteristics (mean/median ± with CI and p-value) for the two groups

Variable	CP	CP no catch	TD	Group difference p-value (CI)
N (boys/girls)	12 (9/3)		7 (4/3)	
Age (years)	10.6 ± 2.9		10.9 ± 3.02	p= 0.859 (-3.6-3.1)
Body weight (kg)	35.27 ± 11.7		40.2 ± 15.7	p= 0.744 (-15.7-11.4)
Height (cm)	143.4 ± 15.4		148.2 ± 23	p= 0.854 (-19.9-16.7)
CP (unilateral/bilateral)	10/2		n/a	
GMFCS (I/II)	10/2		n/a	
Affected limb (catch/no catch)	10/4		n/a	
Passive dorsiflexion 90º (deg)	13.6 ± 6.5	5.04 ± 9.4	29.4 ± 6.5	**, ***
Passive dorsiflexion 180º(deg)†	7.9 ± 2.3	-1.6 ± 7.6	26.9 ± 12.5	*,**,***
Normalized SOL EMG† 10-20 % of stance (%)	95 ± 31	56 ± 32	19 ± 12.6	**,***
Normalized GM EMG 10-20 % of stance (%)	63 ± 25.7	47 ± 30.8	17.29 ± 8.5	**,***

*Abbreviations: GMFCS = Gross Motor Function Classification System. CP: Cerebral palsy, Age, Body weight, height, passive dorsiflexion 90º and normalized GM EMG are presented as mean ± SD, catch|no catch from the Modified Tardieu Scale, Passive range of motion from Tardieu Test 90 and 180, *significant between CP catch and CP no catch, p<0.05, **significant between TD and CP with catch, p<0.05, ***significant between TD and CP no catch, p <0.05, †presented as median*

An independent t-test demonstrated no significant differences between the groups in terms of anthropometrics. There were ten children with unilateral CP and two with bilateral CP. These were split into affected limbs, so the total number of limbs were 14 when counting both limbs in bilateral CP as affected. All except for two were defined in GMFCS group I. Passive dorsiflexion in 90º angle and 180º angle were significantly lower in both CP groups compared

to TD, with the latter also showing a significantly lower passive dorsiflexion in CP children with catch compared to CP children without catch (Table 1).

3.1. Tardieu Test

3.1.2. EMG during Tardieu Test

CP children with catch had more activity in SOL during Tardieu Test 90 than both TD and CP without catch with a max of 0.12 mV compared to 0.06 and 0.07 in TD and CP without catch (Fig.7). More variation in EMG activity in CP children with catch than CP without catch. No other results in GM or SOL were significantly different between the groups in either tests. CP children with catch had a significant higher mean difference of 0.06 mV in SOL activity from Tardieu 90 in CP children with catch compared to TD (Mann-Whitney U test, p=0.049). A Mann-Whitney U test showed a mean 0.08 mV higher GM EMG activation in TD children compared to CP without catch (p=0.013) during Tardieu 180 and in CP children compared to CP without catch (p=0.005).

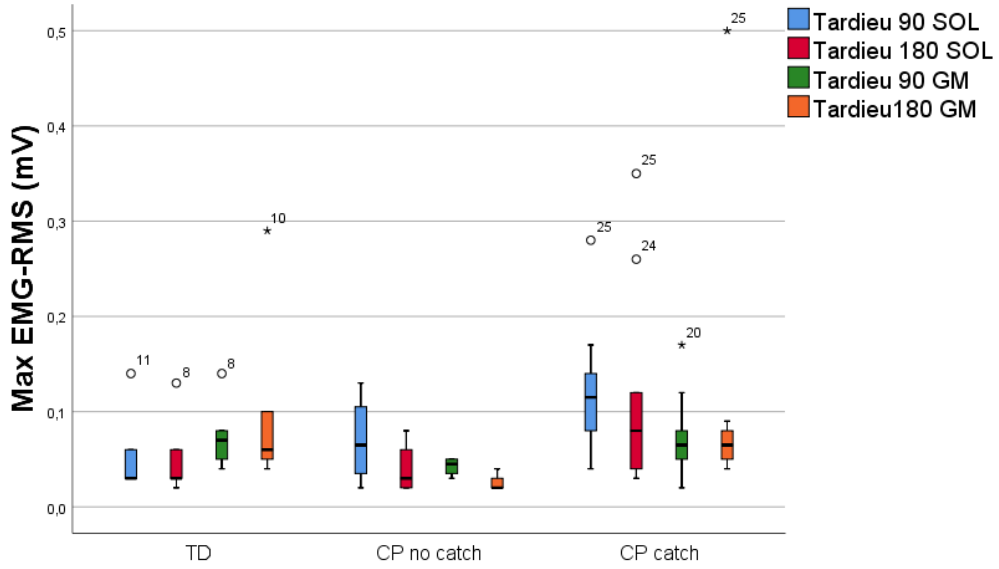


***Figure 7:** Max EMG from m. soleus (SOL) and medial. gastrocnemius (GM) during V3 in Tardieu 90 and 180 in TD and CP with and without catch. Illustrated in a boxplot with a median (dashed line), 25-75 % percentiles with range from min to max (T-bars) excluding outliers (circles) and extreme outliers (stars).*

3.1.2. Kinematics from Tardieu Test

The muscle reaction during the fast movement occurred earlier in the CP group with catch, than in the CP group without a catch in both tests, and slightly earlier in Tardieu 90 (Fig. 8). CP children with a catch had a larger spasticity angle in both tests than CP children who were not categorized with a clinical catch. Both spasticity angles measured from Tardieu 180 (p=0.046) and Tardieu 90 (p=0.002) were significantly different between the CP groups.

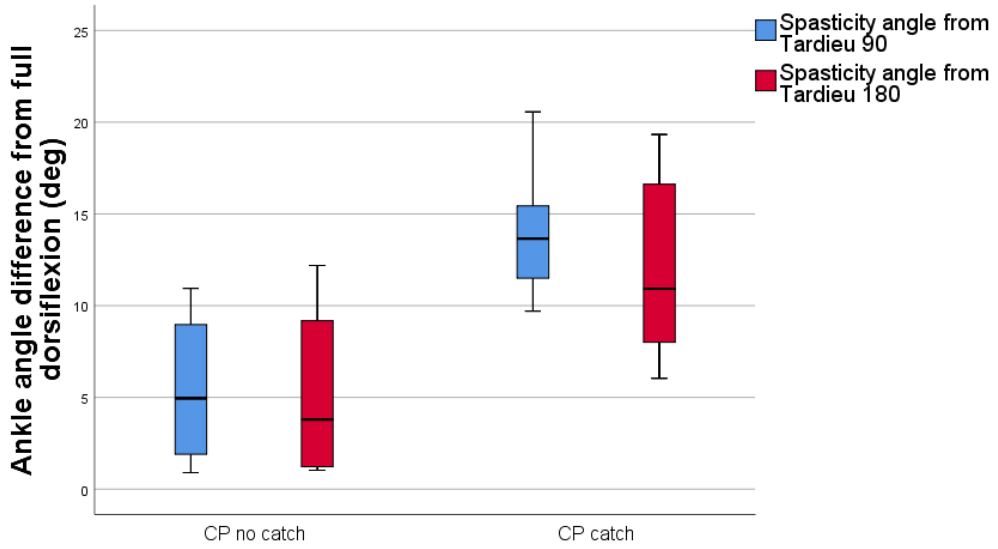


Figure 8: Spasticity angle from Tardieu 90 and Tardieu 180 in CP with and without catch shown in a boxplot with a median (dashed line), 25-75 % percentiles with range from min to max (T-bars).

More variation was observed in both CP groups than in TD. TD children had higher max ankle angular velocities in V3 than both CP groups, but not significant. CP children with catch had slightly higher angular velocities on average than CP without catch in V1 from both Tardieu Tests and in V3 from Tardieu 90. An independent t-test showed that CP children with catch had a significantly higher max ankle angular velocities in V1 during Tardieu Test 90 when compared with TD (p=0.03).

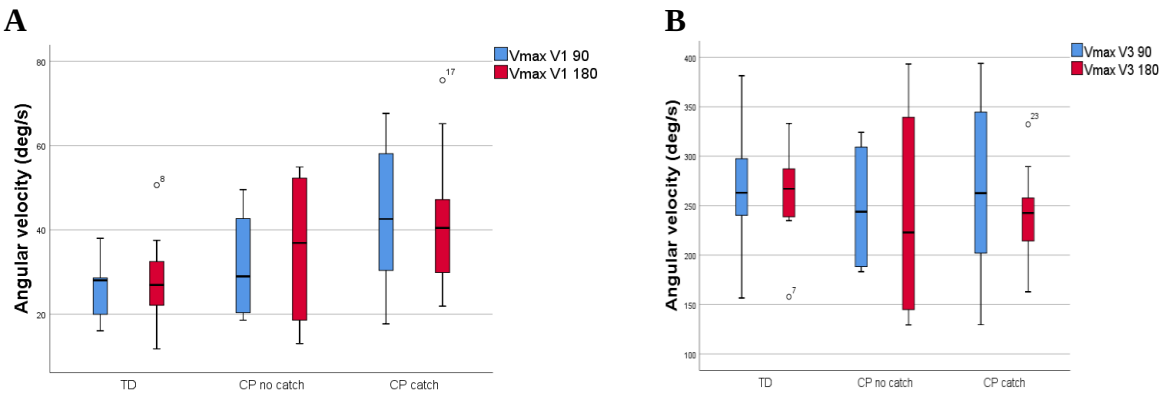


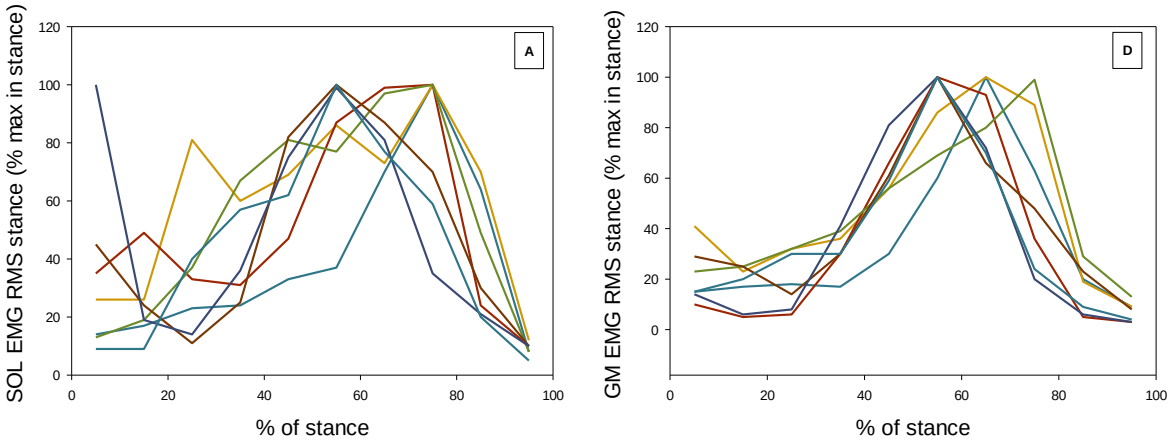
Figure 9: Max ankle angular velocities from V1(A) and V3 (B) in Tardieu Test 90 and Tardieu Test 180 for TD and CP with and without catch shown in a boxplot with a median (dashed line), 25-75 % percentiles with range from min to max (T-bars) excluding outliers (circles).

3.2. Gait

3.2.1. EMG during 3D gait analysis

Figure 11 illustrates average SOL and GM EMG RMS activity in 10 % bouts from the stance phase normalized to max in stance from both CP with catch (Figure 11B and E) and CP without catch (Figure 11C and F) along with TD (Figure 11A and D). There are recognizable SOL and GM EMG activity in CP with catch at the point of 20 % of stance (Figure 11B and E). In the CP group with no catch there were not as recognizable as in CP with catch (Figure 11C and F). TD children showed no particular EMG activity in the same phase when compared to both CP groups (Figure 11A and D)

CP children with catch had a significantly higher activity in GM (on average 46%, p=0.001) and SOL (on average 56 %, p=0.006) during 10-20 % of stance phase when compared with children with TD (table 1). CP children with catch had a non-significantly higher average EMG activation in GM (p=0.335) and SOL (p=0.338) than CP children without catch. CP children without catch had a significantly higher EMG activation in SOL (mean difference of 38 %, p=0.023) and in GM (mean difference of 30 %, p=0.035) compared to TD.



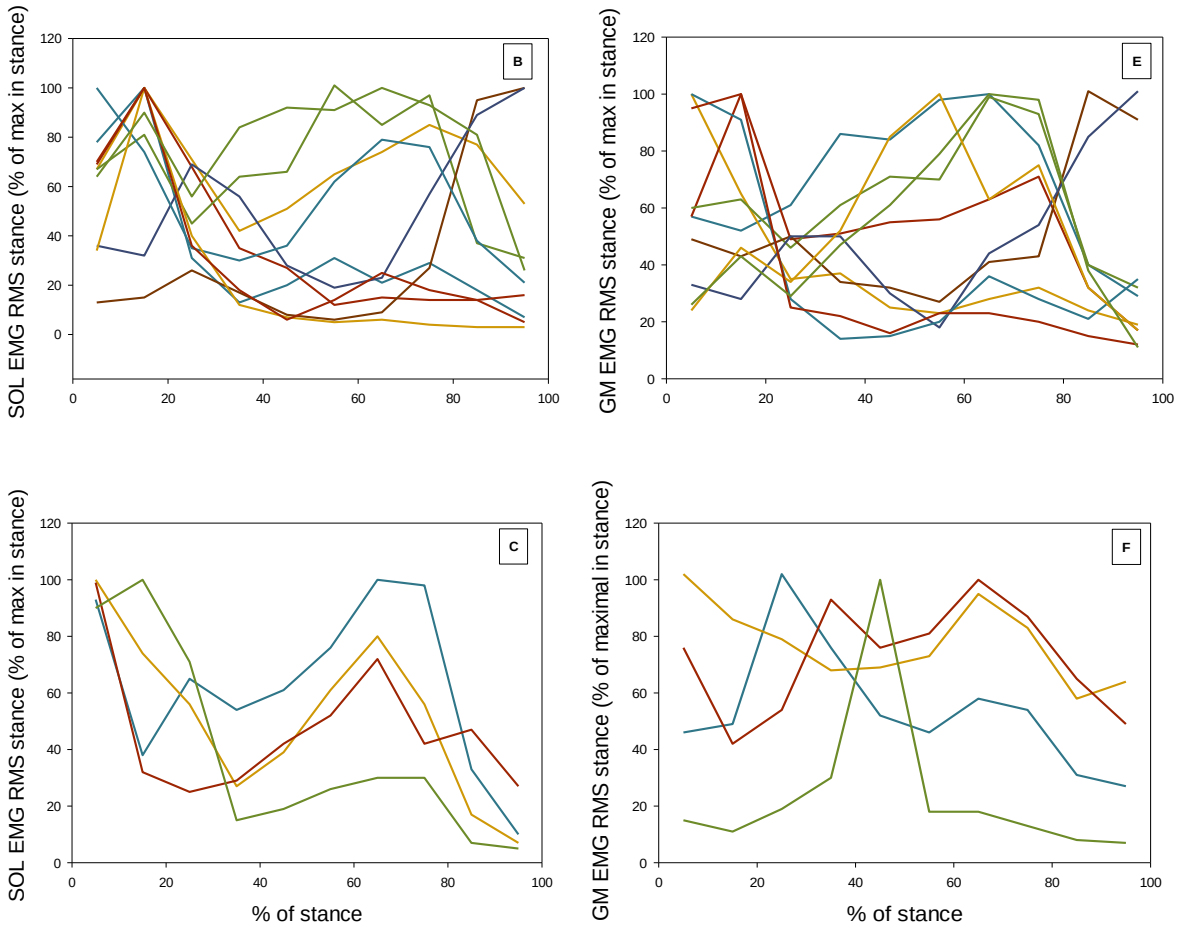


Figure 11: Average m. soleus (SOL) and m. medial gastrocnemius (GM) EMG RMS change in 10 % periods from 0-100 % of stance phase normalized to max EMG during stance in TD (A and D), CP with catch (B and E) and CP without catch (C and F). The colored lines represent individual subjects related to the specific group in each subfigure (A-F).

3.2.2. Kinematics from 3D gait analysis

More variation was found in maximal dorsiflexion in children with CP during both phases when compared to TD (Figure 12A). Higher maximal ankle angular velocities were found during 0-100 % of stance in both CP groups and in 10-20 % of stance in CP children with catch compared to TD (Fig 12B). An independent t-test showed that CP children without catch ($p=0.047$) and CP children with catch ($p=0.023$) had a significant higher max ankle angular velocity in the period from 0-100 % stance, when compared to TD. CP children with catch had a significantly larger dorsiflexion peak angle in the period from 10-20 % of stance compared to CP children without catch ($p=0.020$). CP children with catch also had a significantly higher ankle angular velocities than TD in the periode from 10-20 % of stance ($p=0.045$).

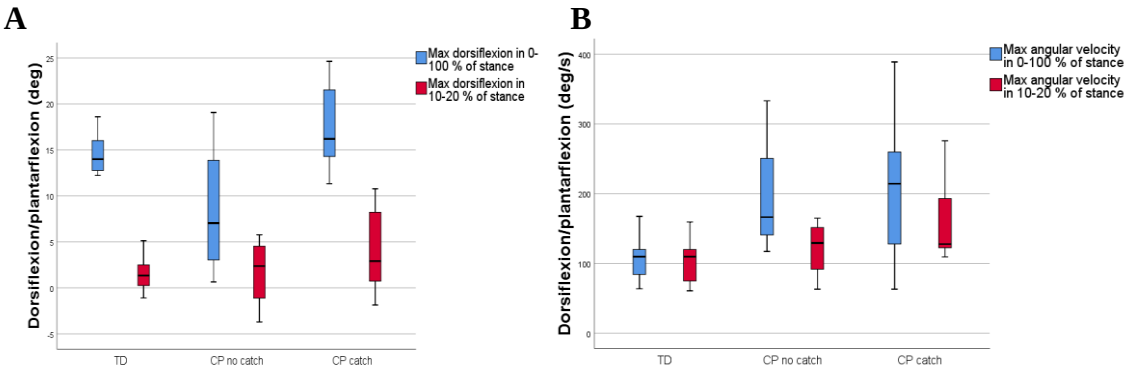


Figure 12: A) Maximal dorsiflexion and B) Maximal ankle angular velocities from 0-100 % and 10-20 % of the stance phase in TD and CP with and without catch. Shown in a boxplot with a median (dashed line), 25-75 % percentiles with range from min to max (T-bars) excluding outliers (circles).

CP children had more knee extension than TD during this period. All groups had about 5 degrees dorsiflexion in the period from 10-20 % of stance (Fig.13A). TD had a significant larger change in knee angle (p=0.007, figure 13A) and a significant mean difference of 97,2º/s in ankle angular velocity (p=0.003) compared to CP with no catch (Fig.13B). TD had a higher ankle angular velocity while CP overall had lower angular velocity in both knee and ankle in the period from 10-20 % of stance (Fig.13B). TD also had a significant higher mean difference of 99º/s (p=0.03) compared to CP with catch. CP children without catch had a higher, but non-significantly, mean difference in knee angular velocity (p=0.319) compared to CP with catch.

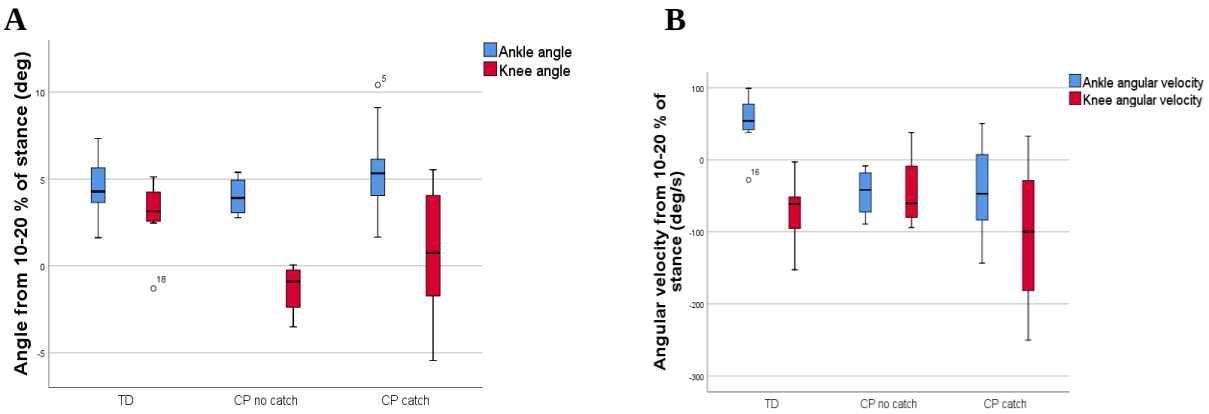


Figure 13.: Average knee and ankle angles (A) and angular velocities (B) from 10-20 % of stance for TD and CP with and without catch shown in a boxplot with a median (dashed line), 25-75 % percentiles with range from min to max (T-bars) excluding outliers (circles).

4. Discussion

The overall purpose of this study was to see if signs of spasticity in the EMG pattern of SOL and GM were present during walking, based upon a clinical scale, assigned to give an indication of spasticity. Furthermore, I wanted to look at joint angles and angular velocities when considering muscle activity during walking and in the passive Tardieu Test. This has been done to compare typically developing peers and reflect a ‘’normal’’ condition. Groups were then compared, and differences were seen. The main findings are that CP children assigned with a catch have a higher EMG activation in SOL and GM during 10-20 % of stance, compared to TD and higher activation in both muscles compared to CP without catch. They also have a slightly higher max EMG RMS activation in SOL, during Tardieu 90 and higher EMG RMS activation in GM during Tardieu 180 than CP children without catch. Overall CP children had higher angular velocities in V1 and lower angular velocities in V3 than TD children. During walking, CP with catch, had a lower max knee angular velocity than CP without catch. During stance both CP groups had a higher ankle angular velocity than TD. CP children also had higher ankle angular velocities during 10-20 % of stance than TD. During 10-20 % of stance, TD had a significant larger knee flexion angle compared to CP with no catch and CP children had more knee extension. Knee flexion angle was non-significantly larger in TD than CP with catch. The CP groups were significant from each other in terms of ankle dorsiflexion in 10-20 % of stance, where CP group with catch had a larger peak dorsiflexion angle than CP without catch.

The discussion will further be divided into smaller units based upon the aim of the study. First, I will discuss EMG activation during walking and from the Tardieu Test, then I will discuss the comparisons between angles and angular velocities during both settings. Next, I will discuss with the strengths and limitations of the study and finally propose possible further research.

4.1. EMG during walking and Tardieu Test

This study presented a higher EMG activation in SOL and GM from both CP with catch and without catch when compared to TD children in a phase of stance where spasticity is suggested. In line with earlier research (14), a heightened EMG activity in the calf muscles during gait, was found in the period from 10-20 % of the stance phase. SOL had high EMG activation in both settings for CP with catch. Since the clinical scoring was based upon Tardieu 90, this may not surprising, and could point to Tardieu 90 being accurate in measuring spasticity in SOL. SOL was also the muscle that seemed most affected in CP with

catch, compared to CP children without catch, although non-significant. Eight CP participants were also classified with a catch in Tardieu 180, and maybe the most affected muscle would have been GM instead, but since it was the most pronounced in Tardieu 90, assumptions can be made to say this that muscle is overall more affected. Mostly CP children showed a premature EMG pattern. They had about 5 degrees of dorsiflexion in the period from 10-20 % as well as more knee extension than TD, possibly indicating a more normal dorsiflexion. Therefore, a stretch happening in GM, pointing to more muscle activity in CP with a catch. With many hemiplegic and all but two in GMFCS level I, they are highly functional in a CP perspective. There seems to be a trend towards high activation in EMG during Tardieu resembling high EMG activation during walking in a phase where pre-stablished walking mechanics were suggesting spasticity. Previous studies that have measured this phenomenon have also found increased activity during stance (30, 39-41) and in swing (17). Although the study showed a heightened muscle activity in the EMG pattern of SOL, we cannot know whether this really is spasticity or not (29). Dietz and coworkers (29) found that spasticity may not be as deliberating to active function as assumed. Spasticity can vary according to conditions and in static or dynamic situations. This could explain some of the differences in EMG activation during V3 and during 10-20 % of the stance phase, in children with CP.

CP children with catch had a non-significantly heightened activation in both SOL and GM during 10-20 % of stance compared to CP children with no catch. Differences were found even though the groups were small. EMG activation under Tardieu Test did not show the same distinctive differences as under gait, between CP children and TD children. This could have something to do with the position they were in when muscle activity was measured. Since spasticity can behave differently in different positions.

Some studies have found that spasticity measured during passive conditions may not be as causative to gait problems (30, 31). Since spasticity may be a solution to a problem the original brain injury presents to a neurological impaired population caution should be drawn as to what effect spasticity has on gait. The abnormal muscle patterns could be a solution instead of a problem.

Muscle activation in Tardieu contrasted with muscle activation in the period from 10-20 % of stance. CP children and TD were not very different from each other in Tardieu while they were significantly different from each other. In stance CP children with catch had higher activation in both SOL and GM than TD. The spastic response could be different between

static and dynamic situations. There were considerably higher ankle angular velocities in both Tardieu Tests contra 10-20 % of stance. Overall there were not possible to observe distinctive groups differences in EMG from the Tardieu Test, as it was in the period from 10-20 % of stance. Some differences were seen in SOL and GM between TD and CP children, possibly pointing to a difference in activation during the test.

4.2. Kinematics from Tardieu Test and 3D gait analysis

Overall the CP children had higher peak angular velocities in the period from 10-20 % of the stance phase and lower average angular velocities during 10-20 % of stance phase. During max EMG RMS there were more variation in the TD group than in CP groups and little differences between the groups. Could this be because the tests procedure was bad for some subjects so that the peak happened at different time stamp than others. CP children had on average higher ankle angular velocities during stance and lower velocities during Tardieu Test compared to TD. Overall these velocities were not similar in both settings. They were higher in Tardieu Test than during gait. This points to a discrepancy between the two settings and the passive test seems not to reflect angular velocities during stance in CP children, especially in CP with catch. Pre-established walking biomechanics (15, 16) referred to around 70º/s in the ankle would elicit and spastic muscle reaction during gait, but from the Tardieu Test in this study there were peak joint angular velocities at 200º/s and above. The same was seen in children with CP during 10-20 % of stance phase, with values close to 200º/s. Max angular velocity from Tardieu Test seems on average not that different.

Maximal dorsiflexion during 10-20 % of stance were quite similar in the three groups, but CP children had a larger range than TD. CP children with catch had a small but larger significant dorsiflexion peak during 10-20 % of stance than CP without catch. The same is observed from the spasticity angle calculation where they had a larger dorsiflexion angle difference. This may reflect some functional utility of the Tardieu Test in a way that it is expected spastic muscle reactions in both settings. More average knee extension in CP children in the period from 10-20 % of stance than TD, suggests that there is stretch in GM and SOL. This is in line with higher SOL and EMG activation during the same phase.

4.3. Strengths and limitations

Strengths of this study was a control group of typically developing children and a passive test for spasticity in line with Lance's (20) definition of spasticity. sEMG recordings along with the test and during gait to assess neurophysiology of the participants.

Included subject were mostly on the lowest scale of GMFCS and had a relatively high functioning walking ability. This could have led to a selection of the best possible children regarding function and may not be deducible to CP children with a less ambulant function.

As with all tests involving subjects set in a ‘’artificial’’ environment, it wants to replicate the real world. Clinical utility is crucial to offer be able to offer inventions in CP. In a gait analysis, participants are asked to walk at their preferred speed. Some may be able to accurately reflect their walking pattern in day-to-day situations, but that is a tough task for anybody. Therefore, imperfectness must be outlined when interpretations are to be drawn. These tests have the potential to give patients and parents direct advice when clinicians interpret the findings. So as means not waste time for the patients these clinical scales need to reflect walking. One especially reason some say that spasticity may not be such a deliberant impairment during walking is that we perhaps may not where to pinpoint where and how it manifests itself. Another is how spastic muscle reactions gets elicited during the passive test. Time of day, prior stretching as to where in the test the spastic reaction occurs.

A weakness is that the study has not use a muscle lengthening velocity model that has been highlighted as a better model to assess spasticity during resting conditions (42). But the lack of a model available and the level of skill needed to handle it, joint angles and joint velocities were used instead. I have also used an unpublished script for analyzing Tardieu Test data, which is not perfect and could have led to wrong interpretations, concerning the way variables and numbers drawn out.

The chosen CP group with catch were selected from the Tardieu Test in 90º and not Tardieu 180º. This was arbitrary since it was 8 participants overlapping between the tests. When interpreting results, I could have been overstating the effect on both EMG and kinematics in an individual not assign with a catch or vice versa.

Using clinical scales as a measure for spasticity suffers from subjective and imprecise interpretations in a diagnostic language and spectral scope. These instruments are the tipping point of the understanding leading to treatment of patients with spastic cerebral palsy. Therefore, these scales need improvement considering how decisive they are in the evaluation process. Given the large variability in V3 angular velocities it is hard make predictions at which velocity a spastic muscle really occurs.

Clinical scales that would accurately describe the phenomenon that is measured is ideal, since this tool is least expensive and applicable in daily clinical practice, compared to biomechanical and neurophysiological measurements. But that is not reality today, they need improvement to reflect joint angles and joint velocities during a functional activity. Reliability and validity of these scales differ in comparison to what type of neurological population is in question (43). Caution must be drawn when citing studies relating to another spastic etiology, even though the condition is universal.

Modified Ashworth Scale (MAS) has been the most common clinical test to assess the level spasticity in the scientific literature. Banky and colleagues (26) found that most of the studies linking spasticity to a functional activity used this test and only one used the Tardieu Scale. This is despite the most applied definition by Lance (20), links up better with Tardieu Scale than MAS. And it has been acknowledged that MTS is a better measure for spasticity and not confounded in its scoring system regarding contracture, the way the definition concerning the velocity-dependent factor of UMNS, is what is sought to interpret (44, 45). Definitional power seems to be more important than objective reality. The Modified Tardieu Scale (MTS) has an advantage over MAS because it separates contracture from spasticity. And the two velocities make it a more suitable scale and it is also based directly upon Lance's definition of spasticity (33, 35). Thus far neither scales can identify the functional severity of spasticity (43).

A small number of subjects without catch in comparison to those with catch may present a problem when considering statistical power. I could also just have captured more of the heterogeneity of CP by differentiating between these two. Though the groups have shown differences in in both EMG and kinematics, the number of participants in the CP group, is no small to draw any firm conclusions from. I have only looked at a fragment of the clinical picture within a small spectrum of children with CP.

4.4. Further research

It is important to get a definition on what spasticity means in a functional context, that all can agree upon. Especially considering clinical practice. Banky and colleagues (26) mentions that improving measurements methods which deals with these concerns is essential for validation of clinical scales. Instrumented tests are a preferred tool for many labs, but they are hard to implement in a normal clinical setting since they demand special education and are expensive.

Therefore, improvement of these scales seems like a less expensive initiative in order provide specific and sensitive measurements to children with CP.

In line with Banky and coworkers (26), a further development of the spasticity angle could be engaging. Exploring and possibly relate the angle of arrest from V3, to a specific phase in the gait cycle during walking.

4.5. Conclusion

The accuracy of MTS scoring system seems to be sufficient at differentiating between those with spasticity and those with contractures. The results from Tardieu matched well with the spasticity angle measurements but had showed higher angular velocities than during gait. This points to a discrepancy in angular velocities at the elicitation of a spastic muscle reaction.

CP children with catch had higher SOL and GM EMG activation in the period from 10-20 % of stance, than both TD and CP children without catch. This suggests that it may be possible to observe signs of spasticity during the stance phase in walking.

Spasticity or voluntary activation is hard to distinguish based upon EMG from the Tardieu Test. Most movements in daily life is not performed ‘’as fast as possible’’ as during V3. The nature of the movement may not reflect active movement during walking.

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