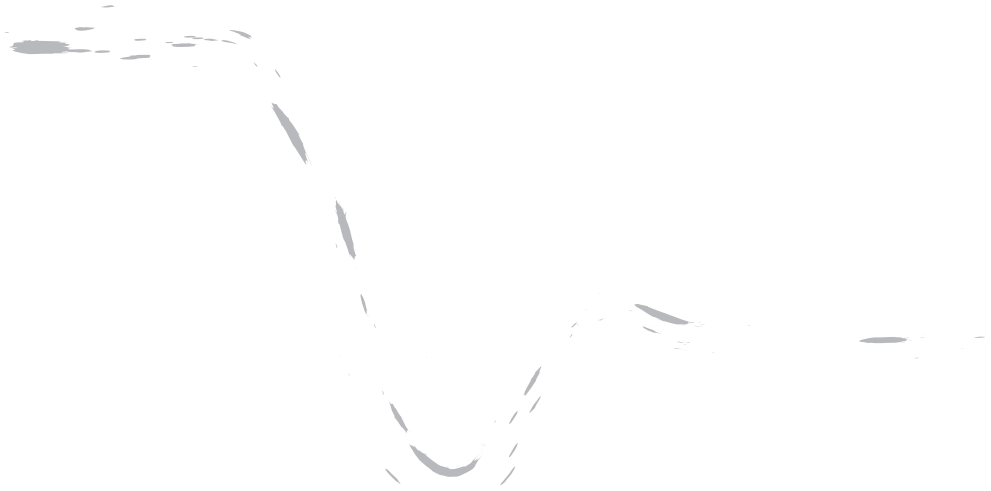
Chapter 3

Evaluation of the catch in spasticity assessment in children with cerebral palsy



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3.1 Abstract

Assessment of spasticity is important in children with spastic paresis, being the most common movement disorder in cerebral palsy (CP). Spasticity is clinically assessed by fast passive muscle stretch to detect a catch, subjectively encountered as a sudden resistance in the motion. The objective of this study is to evaluate whether the catch in clinical spasticity assessment in CP is the consequence of a sudden velocity-dependent increase in muscle activity, resulting from hyper excitability of the stretch reflex in spasticity. Twenty children with CP participated in the study (age 5-14 years; body mass 35±14 kg (mean ± standard deviation); body height 139±19 cm). Spasticity assessment tests (SPAT: using slow and fast passive stretch) were performed in the medial hamstrings, soleus and medial gastrocnemius muscles of the children by two experienced examiners. Surface electromyography (EMG) was recorded and joint motion was simultaneously measured using two sensors of an inertial and magnetic measurement system (IMMS). The encounter of a catch by the examiner was compared to the presence of a sudden increase in muscle activity ('burst'). The Average Rectified Value (ARV) of the EMG signal was calculated for each test. The study shows a sudden increase in muscle activity in fast passive stretch, followed by a catch (hamstrings 100%, soleus 95%, gastrocnemius 84%). The ARV in slow passive stretch was significantly lower. In conclusion, the results confirm that in children with CP an increase in muscle activity is primarily responsible for a catch in fast passive muscle stretch.

3.2 Introduction

Cerebral palsy (CP), an upper motor neurone syndrome, is the most prominent cause of motor disability in childhood [1]. The main group of motor disorders in CP is the spastic paresis [2], and assessment of spasticity is therefore important in children with CP. Spasticity is one of the excess features of the upper motor neurone syndrome and arises from disruption of certain descending pathways involved in motor control. One of the commonly used definitions of spasticity is described by Lance (1980): *"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 reflex, as one component of the upper motor neurone syndrome"* [3]. The stretch reflex could be evoked by a sudden increase in muscle length, resulting in a contraction of that muscle [4]. In spasticity, the stretch reflex is hyperactive [3,5] and evoked by a lower velocity of stretch of the muscle (i.e. the velocity threshold) as compared to normal muscles [6].

Spasticity could limit the performance of motor tasks [7]. Many therapies focus on its reduction with treatments like baclofen, botulinum toxin A and selective dorsal rhizotomy [8]. Identifying spasticity is relevant assisting clinical decision-making and evaluating treatment. Several methods were developed and used to assess spasticity. They can be classified into clinical, biomechanical, and neurophysiologic methods [9].

The most commonly used test in clinical practice is the (modified) Ashworth Scale (AS) [10]. This test is based upon the assessment of resistance to passive stretch of a muscle group at one non-specified velocity, encountered by the examiner applying the movement [11,12]. The intra- and inter-rater reliability of the AS is questioned [13] and the lack of standardization in the velocity makes this method not completely consistent with Lance's definition of spasticity [11,14]. However, the (modified) Tardieu Scale (TS) [11,15-17] and the Spasticity Test (SPAT) [12,18] (a simplification of the TS) use at least two different velocities of passive muscle stretch. In these tests, a muscle (group) is first passively stretched at slow velocity to define the range of motion (ROM). The movement should be slow enough not to elicit any muscle activation. The TS describes the movement to be slower than the natural drop of the segment under gravity; in the SPAT the slow velocity movement should last more than three seconds to complete the ROM. A fast passive stretch is subsequently performed to evoke a stretch reflex and to detect the catch. In clinical assessment of spasticity, the catch is defined *as "a sudden appearance of increased muscle activity in response to a fast passive stretch, which leads to an abrupt stop or sudden increased resistance during the movement, at a certain angle before ROM is*

reached" [5,18]. In the TS, the fast velocity is described to be faster than the natural drop of the segment under gravity; in the SPAT the high velocity stretch should be performed in less than one second. The TS also describes a muscle stretch at a third velocity, i.e. under gravity. The catch is one of the phenomena in spasticity and has been suggested to be directly caused by the hyper excitability of the stretch reflex [5,11,18,19], and to be absent in slow passive stretch and in non-spastic muscles [6,19,20]. In the TS, modified TS and the SPAT intensity of muscle resistance is graded. Additionally, in the modified TS and the SPAT the joint angle of the catch (AOC) is measured, using goniometry after repositioning of the joint in the position where the catch first appeared. [11,12,16,18]

From a biomechanical perspective, the catch is defined as *"a transient increase in the force that opposes passive extension"* [21]. Biomechanical methods to assess spasticity therefore focus on resistance to movement in a joint to quantify the forces that are sensed by the examiner. Devices such as a hand-held dynamometer or isokinetic dynamometers to measure torque are used [21-25]. Neurophysiologic methods to assess spasticity often use surface electromyography (EMG) to record the responses of the muscle to passive movement [26]. These methods do not measure the catch as a resistance in the movement, clinically or biomechanically, but focus on the underlying mechanism of the stretch reflex, studying the EMG latencies and amplitudes. In several studies, the combination of biomechanical measures with concurrent EMG recordings to assess spasticity is a topic of interest [22,27-31].

Although in clinical practice the catch is used as a measure for assessing spasticity, some studies cast doubt upon the unique relationship between a catch, encountered as a sudden increase in resistance during the motion, and a sudden increase in muscle activity (the hyperactive stretch reflex) in fast passive muscle stretch [21,30,32]. Soft tissue structures may also produce a resistance that increases with muscle length and velocity of stretch [5,32,33], especially since these structures are altered in a spastic paresis [8,34-36]. However, most studies criticising the catch as a consequence of a sudden increase in muscle activity are based on the observation in stroke patients with use of the modified AS [21,30], which does not comply with the concept of spasticity described by Lance [11,14,37]. In 2005, the SPASM consortium (Support Programme for Assembly of database for Spasticity Measurement) redefined spasticity as *"disordered sensori-motor control, resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary activation of muscles"* [24,32]. This definition describes spasticity more generically compared to Lance's definition, including the entire range of signs and symptoms that are collectively described as the excess features of the upper motor

neurone syndrome, and not exclusively the hyper excitability of the stretch reflex. The TS (or TS-like scales such as the SPAT) is consistent with Lance's definition of spasticity, but publications regarding validity and reliability of the TS are scarce [37]. Whether the catch encountered in a TS-like clinical spasticity test is indeed the consequence of stretch reflex muscle activity, is therefore worthy of investigation.

The aim of this study was to evaluate whether the catch, encountered by the examiner during clinical spasticity assessment (as an abrupt stop or sudden increased resistance during the fast passive stretch movement, at a certain angle before the ROM is reached), is indeed the consequence of a sudden velocity-dependent increase in muscle activity, elicited by fast passive muscle stretch. We therefore evaluated: (i) the mean stretch velocities during slow and fast passive muscle stretch in children with spastic CP; (ii) the muscle activity during the slow passive stretch tests; (iii) the muscle activity, as well as the occurrence of a catch during the fast passive stretch tests; (iv) the time delay of muscle activity to appearance of a catch during the fast passive stretch tests. We hypothesized that no muscle activity is present during the slow passive stretch. We further hypothesized that when a catch is encountered by the examiner during fast passive stretch of a muscle, it is preceded by a sudden increase in muscle activity.

3.3 Methods

3.3.1 Subjects

Twenty children, between 5 and 14 years of age (10 girls, 10 boys, body weight 35±14 kg (mean ± standard deviation (SD)); body height 139±19 cm), diagnosed with spastic CP [1] (Gross Motor Function Classification System [38], range I-IV) participated in the study. Exclusion criteria are presented in Table 3.1. The children were recruited from a special school for children with physical disabilities. The Medical Ethics Committee of our hospital approved the study. Full written informed consent was obtained from all parents and from all children aged 12 years and older. The children were tested at school.

3.3.2 Procedure

Unilateral spasticity assessment of the medial hamstrings, soleus and medial gastrocnemius muscles was performed using the Spasticity Test (SPAT) [12,18], three sessions for each muscle, separated by at least 15 minutes. The tests were performed in standardized postures according to the protocol of the SPAT [12,18]: slow passive stretch

Table 3.1. Exclusion criteria

Casting or botulinum toxin A injections within the previous 4 months
Orthopaedic surgery within the previous 12 months
Previous selective dorsi rhizotomy or intrathecal baclofen treatment
Maximum passive ankle dorsiflexion (knee extended) of 15° plantar flexion
Maximum passive hip extension in prone position of 15° flexion
Disturbed behaviour that would make it difficult for the child to understand the tests or to cooperate during the study
Inability to relax during the measurements

to determine the ROM, and fast passive stretch to detect the catch. Subject positioning and stretch procedures of the SPAT are described in Table 3.2. The hamstrings muscle was stretch through a knee extension motion in supine position, starting from a maximally flexed position. The soleus muscle was stretched through an ankle dorsiflexion motion in supine position with hip and knee 90° flexed, starting from maximal plantar flexion. The gastrocnemius muscle was stretched through an ankle dorsiflexion motion in supine position, starting from end ROM position of soleus dorsiflexion, following by a slow extension of the knee, and maximal plantar flexion of the ankle. The contralateral leg maintained in extension.

One examiner (a researcher, experienced in SPAT assessment) tested each muscle twice, and one examiner (an experienced paediatric physical therapist) tested each muscle once. In total 60 slow and 60 fast passive stretch tests per muscle were performed. Either the affected leg (hemiplegia), the most affected leg (asymmetric diplegia), or the right leg (symmetric diplegia) was tested. During the fast passive stretch, intensity of muscle resistance was graded by the examiners on the 4-point spasticity scale of the SPAT [12,18]: *0*, normal or increased muscle resistance over the whole ROM; *1*, increase in muscle resistance somewhere in the ROM without a catch; *2*, catch and release; *3*, catch blocking further movement.

To evaluate the muscle activity, surface EMG was recorded from two disposable surface electrodes (ARBO, H124SG) with a sample frequency of 1000 Hz, using costume made Labview-based software (National Instruments; DAR: Data Acquisition in Rehabilitation). The signals were full-wave rectified and bidirectional fourth-order low-pass filtered at 40 Hz (Butterworth). Electrode placement was based on the recommendations of the SENIAM protocol (Surface Electromyography for the Non-Invasive Assessment of Muscles) [39]. Before electrode placement, the skin was carefully cleaned with alcohol.

To measure joint angles (^{o}) and to evaluate the stretch velocity (^{o}/s), two lightweight sensors of an inertial and magnetic measurement system (IMMS) (MT9, Xsens Technologies, the Netherlands) tracked the motion of the proximal and distal body segments with a sample frequency of 100 Hz [12], using MT9 Software (Xsens Technologies, the Netherlands). Trials were excluded from analysis in case of (i) obvious movement artefacts in the EMG signals, (ii) obvious artefacts in the IMMS data, (iii) continuous muscle activity (i.e. no muscle relaxation) at the start of the movement.

3.3.3 Data analysis

For data analysis of the EMG signals and IMMS data, MATLAB Software (Version 7.2.0.232, R2006a, The Mathworks) was used. To quantify the stretch velocities during physical examination, the mean joint angular velocities (i.e. the mean velocity from start to end of the movement) of both the slow and fast passive muscle stretch were calculated by differentiation of the joint angle in the sagittal plane, using the IMMS data as described in Van den Noort et al. [12] (Chapter 2). The intensity of muscle resistance graded by the examiner during fast passive stretch was used to assign the trials of the fast passive stretch to either trials where a catch was observed (grade 2 and 3 of the SPAT scale, ROM > AOC) or to trials where no catch was encountered (grade 0 and 1). This dichotomous classification is used in all further analyses.

Muscle activity of the slow and fast passive muscle stretch of the three muscles was examined in two different ways. First, by evaluating muscle activity increase using the threshold of onset of EMG, defined at 3 SDs beyond the mean of the baseline activity of the muscle [40]. When the EMG signal did not cross the threshold or crossed the threshold for less than 20 milliseconds [40], the muscle was graded as having ‘no activity’. When an increase of EMG beyond the threshold was observed during the passive stretch preceding the catch which persisted for more than 20 milliseconds, the muscle activity was graded as a ‘burst’. Second, the muscle activity was examined by calculation of the Average Rectified Value of the EMG signal (ARV: the time windowed mean of the absolute value of the signal) [41] from start to end of the movement (i.e. end ROM in slow passive stretch, and the catch in fast passive stretch, measured with the IMMS). Since the ARV is confounded by the duration of the movement, which is different between slow and fast passive stretch, the peak value of the burst in muscle activity, when present, was taken into account as well. The appearance of the catch, subjectively encountered by the examiner, had to be made objectively measurable in the joint angle signal. It was determined as a biomechanical event, at the moment where the measured joint angular deceleration was

maximal. This determination of the catch is in accordance to the definition of the catch as a consequence of muscle activity (neurophysiologic), which causes a force opposite to the movement of the stretch (biomechanical: using Newton's law $F=m*a$, an increase in force in the opposite direction is the same as a deceleration of the motion), felt as a sudden resistance or abrupt stop in the movement (clinical). The angular acceleration/deceleration ($^{o}/s^{2}$) was obtained by double differentiation of the joint angle and bidirectional first-order low-pass filtered at 10 Hz (Butterworth) [12]. To study the catch as a consequence of muscle activity, the time delay between onset of muscle activity and appearance of the catch was calculated.

3.3.4 Statistical analysis

Repeated measures analysis of variance (ANOVA) (three tests per subject per velocity) were performed (SPSS Version 15.0, Chicago, IL) to evaluate whether (i) the mean joint angular velocities, and (ii) the measured ARV values, were significantly different in slow versus fast passive muscle stretch. Statistical significance was accepted for a *P*-value less than 0.05. Before analyses, data were checked for normality, assumption of sphericity, and homogeneous variances.

The relative amount (%) of trials with EMG activity in slow passive stretch was calculated. Each condition in the fast passive stretch (no catch / no activity / catch / burst) was expressed with respect to the total included fast stretch trials. The amount of correct identified catches (with EMG burst) with respect to the total amount of catches was calculated. In addition, the sensitivity, specificity [42], and probability of error of the fast passive stretch tests were calculated for the spasticity scale (no catch/catch) versus the muscle activity scale (no activity/burst). The sensitivity expressed the fraction actual positives which are correctly identified (i.e. trials with catch and EMG burst with respect to all trials with EMG burst). The specificity expressed the fraction actual negatives which are correctly identified (i.e. trials without catch and EMG burst with respect to all trials without EMG burst). The probability of error expressed the fraction of incorrect identification. A specificity and sensitivity of 0.8 or higher and a probability error of 0.2 or lower were considered to be acceptable.

Table 3.2. The Spasticity Test

SPAT			
	Hamstrings	**Soleus**	**Gastrocnemius**
Joint	Knee	Ankle	Ankle
Segments			
Proximal	Thigh	Shank	Shank
Distal	Shank	Foot	Foot
Patient Position			
Supine/prone	Supine	Supine	Supine
Hip	90° flexion	90° flexion	Extension
Knee	Maximal flexion	90° flexion	Extension
Ankle	Not relevant	Maximal plantar flexion	Maximal plantar flexion
Hind foot	Not relevant	Neutral varus/valgus	Neutral varus/valgus
Contra lateral leg	Extension	Extension	Extension
Location IMMS sensors			
Proximal	Laterally along the long axis of the thigh	Laterally along the long axis of the shank	Laterally along the long axis of the shank
Distal	Laterally along the long axis of the shank	Laterally along the long axis of the foot sole	Laterally along the long axis of the foot sole
Passive stretch			
Joint motion	Knee extension	Ankle dorsi flexion	Ankle dorsi flexion
Angle of interest	(Popliteal) Knee angle	Ankle angle	Ankle angle
Slow stretch	Excursion of the maximum range of motion (ROM) in three or more seconds		
	Maximal ROM at moment at which the contralateral leg starts to move due to pelvic movement	Maximal ROM at maximal dorsi flexion	Maximal ROM at maximal dorsi flexion
Fast stretch	Excursion towards the maximum ROM within one second (as fast as possible)		
Spasticity scale			
0	Normal or increased muscle resistance over the whole ROM		
1	Increase in muscle resistance somewhere in the ROM without a catch		
2	Catch and release		
3	Catch blocking further movement		

IMMS = inertial and magnetic measurement system
ROM = range of motion

3.4 Results

Figure 3.1 presents a typical example of a slow (A-B) and fast passive muscle stretch (C-E) of the medial hamstrings muscle in one subject. The figure shows no muscle activity in the slow passive stretch and a (limited) ROM of the knee of 67^{o} (popliteal angle; full extension is 0^{o}). In the fast passive stretch a sudden increase in muscle activity is present, followed by a catch at 105^{o} (the AOC). The muscle activity is transient, it disappears after the stop of movement.

Tables 3.3 through 3.5 present the mean joint angular velocities and the ARVs of the EMG signals for both the slow and fast passive stretch tests of the medial hamstrings, soleus and medial gastrocnemius muscles. The numbers and percentages of trials ex- and included in the analysis are shown. Specificity (the relative number of true negatives), sensitivity (the relative number of true positives) and probability of error of the fast passive stretch tests are also presented in the tables.

Mean velocities (±SD) of the slow passive stretch of the hamstrings, soleus and gastrocnemius were 28±18, 9±5 and 5±3^{o}/s, respectively. The mean velocities (±SD) in the fast passive stretch were 259±69, 272±81, 245±81^{o}/s, respectively. The latter were significantly higher for all muscles.

In the slow passive stretch tests no sudden increase of muscle activity (burst) was seen in most of the included tests (Tables 3.3 through 3.5). The mean velocity of trials with a burst was significantly lower than the fast stretch velocity. Although a burst was present in those trials, the ARV and the peak value in EMG were significantly lower compared with fast stretch trials with a burst.

During fast passive stretch tests, the examiners encountered a catch in the majority of the included tests: hamstrings 79%, soleus 91% and gastrocnemius 98% (Tables 3.3 through 3.5). When a clear catch was observed during the joint movement, the hamstrings muscle always showed a sudden increase of muscle activity preceding the catch (100% correct identified catches). In 4% of the soleus and 16% of gastrocnemius trials, no distinct EMG burst preceding the catch was observed (soleus: 95% correct identified catches; gastrocnemius: 84% correct identified catches). The burst in muscle activity was transient in 85% of the cases, that is, the muscle activity disappeared after the catch, when the movement stopped. In 8% muscle activity continued after the catch and in 7% a clear clonus was observed. The time delay (Figure 3.1) between onset of EMG activity and the catch was 136±35 milliseconds for the hamstrings, 38±16 milliseconds for the soleus and 39±17 milliseconds for the gastrocnemius.

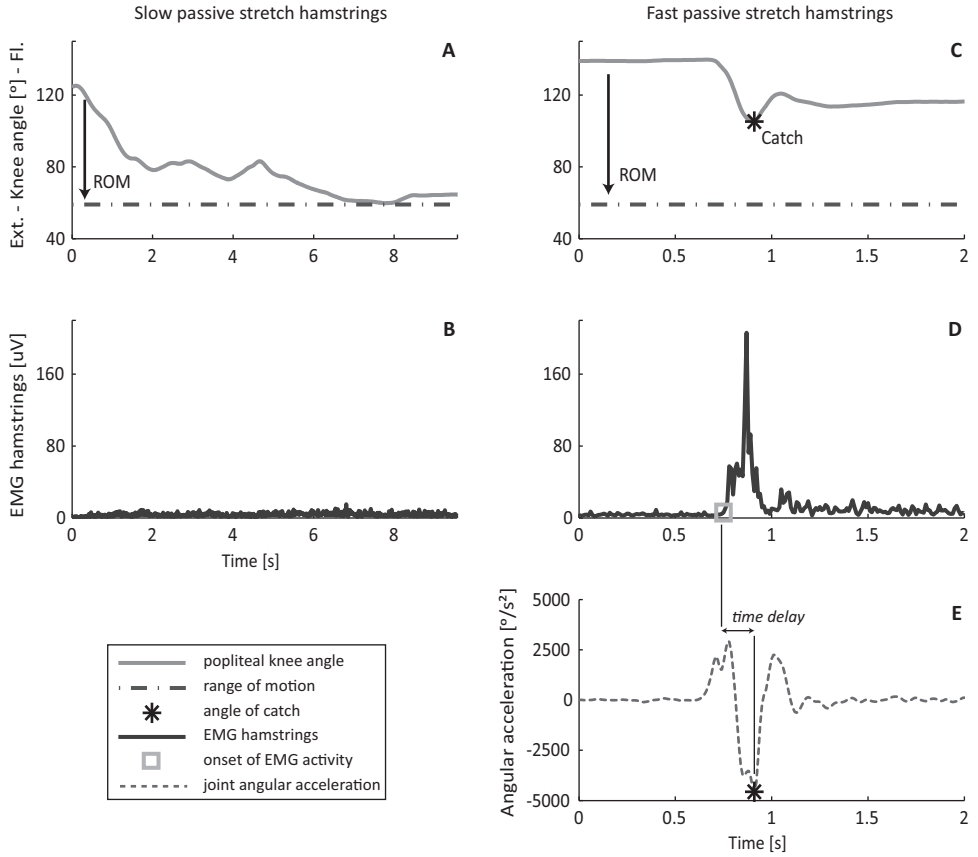

Figure 3.1. Slow and fast passive stretch of the medial hamstrings muscle. (A) Knee angle (solid line) in slow passive stretch of the medial hamstrings muscle, to determine the range of motion (dot-dashed line). (B) Muscle activity of the medial hamstrings during slow passive muscle stretch (C) Knee angle (solid line) in fast passive stretch of the medial hamstrings muscle. The catch (asterisk) appeared before the end range of motion (dot-dashed line) was reached. (D) Muscle activity of the medial hamstrings during fast passive muscle stretch. The onset (square) of the EMG burst was determined at three standard deviations beyond the baseline activity. (E) Joint angular acceleration ($^{o}/s^{2}$) during fast passive muscle stretch (dashed line). The appearance of the catch (asterisk) was determined at the moment of maximal joint angular deceleration. The time delay (arrow) between onset of EMG activity (square in D) and the appearance of the catch (asterisk) is shown.

The sensitivity analysis showed a good sensitivity for the fast passive stretch of all the three muscles (>0.8). In more than 87% of trials with an EMG burst, a catch was encountered. In 11% of the fast passive stretch tests of the hamstrings and in 2% of the soleus and gastrocnemius tests, an EMG burst was observed, without the occurrence of a catch. The specificity analysis showed a good specificity for the hamstrings test, but not for the soleus and gastrocnemius test (Tables 3.3 through 3.5). No catch was encountered in the hamstrings tests, when no EMG burst was present. However, in all gastrocnemius trials without muscle activity, a catch was encountered, which is the main cause of incorrect identification, expressed by the probability of error (0.18). In the hamstrings test incorrect identification (probability of error, 0.11) is caused by false negatives (i.e. trials reported without catch but with muscle activity). The probability in the soleus test is 0.06.
The ARV of the EMG signals in the fast stretch tests with catch was significantly higher than the ARV in the slow stretch tests (on average 8.5 times higher; $P<0.001$). When a burst was present, the peak value was significantly higher in the fast passive stretch ($P<0.001$). When no catch was encountered, the ARV values of the fast passive stretch were significantly higher in the hamstrings muscle compared to the slow stretch (4.7 times higher; ARV, $P=0.003$; peak value EMG, $P=0.003$).

3.5 Discussion

3.5.1 Spasticity Test performance

Mean joint angular velocities were significantly higher in fast stretch compared with slow stretch. In order not to reach the stretch velocity threshold of the muscle, the slow passive stretch to determine the ROM had to be performed with a very slow velocity. The measured mean velocities of slow passive stretch ranged below the threshold velocity reported by Tuzson et al. (60^{o}/s) [31], except for only three trials of the hamstrings test. However, the examiners did not always comply to the SPAT instruction [18], and completed the full excursion in less than 3 seconds in several cases (i.e. in 23% of the hamstrings tests, 5% of the soleus tests and 10% of the gastrocnemius tests). This indicates that, especially in the presence of a large potential ROM (e.g. the knee), the instructions for the SPAT for slow stretch should be revised to avoid high stretch velocities. The measured fast stretch velocities were above the threshold velocities of 60^{o}/s [31] and all stretch movements lasted less than one second, in accordance with the SPAT instructions [18].

3.5.2 Electromyography responses in slow stretch

The results showed little or no muscle activity in slow passive stretch, partly confirming our hypothesis that no muscle activity would be present during the slow passive stretch. However, a burst was observed in 24% of the slow test trials. The low stretch velocities (Table 3.3 through 3.5) of those trials indicate that the muscle activity was not caused by unintended accidental higher stretch velocities, except in a few trials of the hamstrings.
Muscle activity during slow passive stretch could be caused by activity due to inability to relax during the performance of the test. Moreover, the activity may be caused by a very low threshold velocity of the spastic muscles in some cases.

Table 3.3. Outcome parameters of the slow and fast passive hamstrings stretch

Hamstrings				
		slow	**fast**	
			no catch	*catch*
Total tests				
Total number of tests	*N(%)*	60 (100)	13 (22)	47 (78)
Number of excluded tests	*N(%)*	8 (13)	2 (3)	5 (8)
Included tests				
All included tests	*N(%)*	52 (100)	11 (21)	42 (79)
	Velocity [°/s]	28±18	293±80 *	244±59 *
	ARV [uV]	3±2	14±8 *	30±16 *
Tests without EMG activity	*N(%)*	40 (77)	5 (10)	0 (0)
	Velocity [°/s]	29±18	297±70 *	NR
	ARV [uV]	3±1	8±4 *	NR
Tests with EMG burst	*N(%)*	12 (23)	6 (11)	42 (79)
	Velocity [°/s]	23±16	289±95 *	244±59 *
	ARV [uV]	5±3	19±6 *	30±16 *
Correct identified catch (%)			100%	
Sensitivity			0.88	
Specificity			1.00	
Probability of error			0.11	

** P<0.05 (fast vs. slow)*
Values mean ± standard deviation, or as otherwise indicated
N = amount of tests; EMG = electromyography; ARV = Average Rectified Value

Rabita et al. [29] reported reflex responses of the medial gastrocnemius at 10°/s in adult patients with spastic hypertonia while Levin et al. [6] described that velocity-dependent thresholds correlate with the severity of spasticity. In this case, the muscle activity could have been contributed to a stop in the movement, which is reported by the examiners as a limited ROM (e.g. due to a shortened muscle (contracture) [20] as is frequently seen in CP patients [8]). Still, the bursts that were observed in slow passive stretch showed significantly lower ARV values than ARV values in fast passive stretch (on average 6.5 times lower), as well as lower peak values.

Table 3.4. Outcome parameters of the slow and fast passive soleus stretch

Soleus				
		slow	**fast**	
			no catch	*catch*
Total tests				
Total number of tests	*N(%)*	60 (100)	7 (12)	53 (88)
Number of excluded tests	*N(%)*	22 (37)	3 (5)	12 (20)
Included tests				
All included tests	*N(%)*	38 (100)	4 (9)	41 (91)
	Velocity [°/s]	9±5	316±90 *	268±80 *
	ARV [uV]	3±2	10±10	36±24 *
Tests without EMG activity	*N(%)*	34 (89)	3 (7)	2 (4)
	Velocity [°/s]	9±6	317±127	278±161
	ARV [uV]	3±2	5±2	6±0
Tests with EMG burst	*N(%)*	4 (11)	1 (2)	39 (87)
	Velocity [°/s]	6±1	314	264±81 *
	ARV [uV]	5±1	25	37±23 *
Correct identified catch (%)			95%	
Sensitivity			0.98	
Specificity			0.60	
Probability of error			0.06	

** P<0.05 (fast vs. slow)*
Values mean ± standard deviation, or as otherwise indicated
N = amount of tests; EMG = electromyography; ARV = Average Rectified Value

In some cases (hamstrings 8 trials; gastrocnemius 5 trials) the muscle activity continued after the movement stopped. This might indicate the presence of a position-dependent stretch reflex, rather than velocity dependent, as been presented by Powers et al. [43] and Malholtra et al. [30], although it could also be caused by the inability to relax.

3.5.3 Fast stretch without catch

A sudden increase in muscle activity was observed in some cases of fast passive stretch while no catch was encountered by the examiner. For the hamstrings, it was found that the ARV and peak values in those trials were significantly higher than the ARV and peak values in the slow passive stretch.

Table 3.5. Outcome parameters of the slow and fast passive gastrocnemius stretch

Gastrocnemius				
		slow	**fast**	
			no catch	*catch*
Total tests				
Total number of tests	*N(%)*	60 (100)	2 (3)	58 (97)
Number of excluded tests	*N(%)*	29 (48)	1 (2)	14 (23)
Included tests				
All included tests	*N(%)*	31 (100)	1 (2)	44 (98)
	Velocity [°/s]	5±3	153	247±81 *
	ARV [uV]	5±4	12	27±20 *
Tests without EMG activity	*N(%)*	19 (61)	0 (0)	7 (16)
	Velocity [°/s]	5±3		208±75 *
	ARV [uV]	3±1		7±5 *
Tests with EMG burst	*N(%)*	12 (39)	1 (2)	37 (82)
	Velocity [°/s]	6±3	153	254±83 *
	ARV [uV]	8±4	12	31±19 *
Correct identified catch (%)			84%	
Sensitivity			0.97	
Specificity			0.00	
Probability of error			0.18	

** P<0.05 (fast vs. slow)*
Values mean ± standard deviation, or as otherwise indicated
N = amount of tests; EMG = electromyography; ARV = Average Rectified Value

Further analyses of those fast passive stretch trials revealed that the muscle activity appeared at the end of the joint motion when the joint angle had already reached the end of the ROM, defined by the slow passive stretch. We concluded no muscle activity was observed in those slow passive stretch trials, showing the ROM was not limited due to muscle activity. Otherwise, this could have explained that no difference was found between ROM and the joint angle in fast passive stretch. The ROM was sometimes limited due to shortened muscles (contractures), which could interfere with the muscle activity and affect the perception of resistance of the examiner. In order to investigate muscle activity in non-spastic muscles, we suggest performing fast passive muscle stretch in healthy subjects and to evaluate whether such muscle activity at the end ROM might be physiological.

3.5.4 Fast stretch with catch

In the vast majority of the fast stretch trials with a clinically observed catch, a sudden increase in EMG activity preceding the catch was observed (hamstrings 100%, soleus 95%, gastrocnemius 84%). In general, this burst in muscle activity disappeared when the movement stopped (as can be seen in Figure 3.1). This confirms that muscle activity, due to a velocity-dependent hyperactive stretch reflex, is primarily responsible for a catch in the fast passive stretch. The clinical score corresponds to the measured EMG activity as shown by the high sensitivity and low probability of error: more than 87% of tests with muscle activity were correctly identified with a catch and more than 82% of all the tests were correctly identified with or without a catch. This result contrasts with that of Malhotra et al. [30], who found no association between muscle activation patterns and resistance to passive movement. However, Malhotra et al. used the modified AS and measured the wrist joint in a stroke population, which may explain the difference with our result. Patrick and Ada [20] concluded that, in contrast to the TS, the AS is confounded by contracture, due to one non-standardized velocity of stretch. Pandyan et al. [14] described the AS can be used as a measure of resistance, but not spasticity. In addition, spasticity in another muscle group in a different pathology (i.e. stroke) in adults may not simply be generalized to spasticity in children with CP, according to a difference in motor distribution and motor type and a difference in development of muscle contractures (from spasticity) with age in children with motor disorders [44,45].

In some cases (8%), muscle activity continued after the catch, which might indicate the presence of a position-dependent stretch reflex [30]. In 7% a clear clonus was present after the passive stretch.

In the literature, the electromechanical delay (EMD) has been described, which is the time between onset of EMG activity and onset of biomechanical force [46,47]. This time is required to initiate the contractile processes in the muscle and remove the slack from the musculotendinous system before a force is effective on the bones. It has been reported that in CP the EMD is shortened [46]. In our study, the time delay in fast passive stretch is determined between the onset of muscle activity and the catch, i.e. until maximal deceleration (Figure 3.1). We did not determine the time until onset of deceleration, which equals the onset of force ($F=m*a$) used for the EMD, but this is obviously shorter than the time delay we measured. The hamstrings' time delay was remarkably long (136±35 milliseconds) compared to the average EMD of 50±30 milliseconds reported in the literature [46,48], although EMDs have also been reported up to 121 milliseconds [46]. This long time delay may be explained by the higher mass of the shank and foot: compared to the ankle movement in the soleus and gastrocnemius tests, more muscle force is required to oppose the movement due to the inertia of both the shank and the foot. The time delays of the soleus and gastrocnemius are of similar length as the EMD of the soleus muscle reported by Winter et al. [47], indicating that the actual EMD of those muscles in the children with CP is shorter compared to the healthy subjects of Winter et al.

In the hamstrings tests the catch encountered by the examiner was always preceded by muscle activity, confirming the hyperactive stretch reflex is primarily responsible for the catch. However, the specificity analysis showed a poor specificity for the soleus and gastrocnemius tests: despite the lack of muscle activity during the fast passive stretch, the examiners encountered a catch (5% soleus, 17% gastrocnemius). Biomechanical changes in the muscles can affect the perception of resistance. Besides the active response of the muscle, resistance in joint motion could be the result of a biomechanical response from altered passive tissue, as was also suggested by the SPASM consortium [32]. The passive tissue around a joint, including tendons, subcutaneous tissue, fascia, ligaments, joint capsule, cartilage and muscles, shows a viscoelastic behaviour. This means it is position dependent (i.e. elasticity or stiffness) and also velocity dependent (i.e. viscosity or damping). In spasticity, the musculotendinous structure may be altered in fibre type, fibre distribution, fibre length, proliferation of extracellular matrix material and increased stiffness of spastic muscle cell and tissue [35]. Increase in collagen in spastic muscles of children with CP may play a role in the viscoelastic response [49]. Price et al. [45] found a significant difference in viscoelastic properties of passive tissue of children with spastic CP

compare to healthy children. In contrast, Nordez et al. [50] reported velocity-dependent viscosity only plays a minor role in the passive mechanical behaviour of the joint complex.
Whether resistance in joint motion results from passive tissues and/or from active response of the muscle due to hyperactive stretch reflexes could be investigated using system identification techniques such as proposed by De Vlugt et al. [51] and Alibiglou et al. [52]. Lehmann et al. [53] proposed a method to measure the path length in a Nyquist diagram (elastic stiffness versus viscous stiffness) as a measure of the gain of the reflex response in spasticity. It would be interesting to use these techniques in conjunction to a spasticity test as this would enable proper estimation of the contribution of viscoelastic and reflex components to encountered resistance during motion and to explain a clinically measured catch.
Several studies reported the relation of EMG amplitude and stretch velocity: amplitudes of EMG increased with velocity of stretch [4,26,54,55] and were higher in spastic CP patients with respect to normally developing subjects [45,46,56]. This illustrates that the stretch velocity is the most important factor that influences the outcome of clinical spasticity tests. Future research should explore velocity thresholds for each muscle, to quantify the minimal and maximal stretch velocity at which the slow and fast passive stretch have to be performed. When an IMMS, including online feedback, is used to measure the motion, feedback of the stretch velocity can be given to the examiner to standardize the physical examination even further, and the joint angle of catch can be objectively determined [12].

3.5.5 Limitations of the study

The measurements in this study were performed by two examiners, one testing each muscle twice, and one testing each muscle once. Although the examiners were both experienced physical therapists, differences in stretch velocity could occur and influence the muscle response. Intra- and inter-rater reliability of the SPAT was not taken into account in this study, but should be investigated in the future.
Mainly because of movement (cable) artefacts in the EMG data, many trials had to be excluded from analyses (slow stretch: hamstrings 13%, soleus 37%, gastrocnemius 48%; fast stretch: hamstrings 12%, soleus and gastrocnemius 25%). No bias was expected from excluding these trials, because the stretch velocity of the excluded trials was not significantly different from the included trials. In the majority of the excluded trials of the fast stretch a catch was encountered.
The validation of the specificity analysis in this study is questionable, since the data were not equally distributed: EMG activity was observed in the majority of the tests, causing a

low amount of trials used in the specificity analysis. Including more data would increase the statistical power of the analysis.

3.6 Conclusion

During slow passive stretch of soleus, gastrocnemius and hamstrings muscle in children with cerebral palsy, little or no muscle activity was observed compared to fast passive stretch of these muscles. During fast passive stretch, the appearance of a catch was preceded by a burst in muscle activity in the majority of cases. In some cases, the response by biomechanical properties of the muscle and joint can mimic a catch. This indicates muscle activity is primarily responsible for the catch.

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