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An Examination of Spasticity of the Lower Extremity among Young People with Cerebral Palsy

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An Examination of Spasticity of the Lower Extremity among Young People with Cerebral Palsy

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ABSTRACT

Background: Spasticity is the most common symptom among individuals with Cerebral Palsy (CP). Spasticity is often presented as stiff limbs often resulting in pain. Currently, clinicians are limited in diagnosing spasticity using observational tools. The purpose of this study was compare spasticity at various functional levels using dynamometry. **Methods:** Participants included nine adolescents (12-19) with CP and nine adolescents (age-matched) without CP. The participants participated in passive stretches delivered by the Biodex System 4 Pro Dynamometer at four different speeds (90, 120, 150, and 180 deg/s), Measurements of the quadriceps will be collected before and during passive knee extension stretches with the isokinetic dynamometer using angular displacement, torque, and angular velocity. **Results:** This study showed significant differences of resistive torque values (peak and mean) between controls and individuals with high CP. A significant difference of resistive torque values (peak and mean) was also shown between left and right lower extremities. A significant difference between different velocities was not found comparing mean resistive torque values but was found comparing peak resistive torque values at 180°/s to all of the other velocities. **Conclusions:** Dynamometry is able to detect muscle contractions that resist gravity by detecting the amount of torque present. A higher negative resistive torque value represents little to no muscle contraction present while a lower negative resistive torque value represents high muscle contraction present that was resisting gravity. Future research is needed to examine underlying mechanisms with the joint use of dynamometry and surface Electromyography (sEMG).

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TABLE OF CONTENTS

Chapter I:

INTRODUCTION

Cerebral Palsy (CP) is a disorder caused by fetal or infant brain damage affecting 3 to 4 in every 1,000 children in the United States (Boyle et al., 2011; Rosenbaum et al., 2007; Van Naarden Braun et al., 2016). The disorder is manifested in a collection of motor impairments including dystonia, rigidity, chorea, and spasticity (Sanger, 2015). Dystonia is characterized by muscles that twist, perform repetitive movements, and perform prolonged contractions (Fahn, Marsde, & Calne, 1987). Additionally, individuals can experience hypertonia, where muscles are improperly activated or hyperreflexia where muscles reflexes are exaggerated (Bar-On et al., 2015; Mugge et al., 2012). Rigidity is defined as continuous resistance and stiffness of "throughout the full range of motion" of a passive stretch (Levitt, 2009; National Institute of Neurological Disorders and Stroke, 2013)((NINDS), 2013). Spasticity affects approximately 80% of individuals with CP and is a painful stiffness or paralysis at a joint due to increased muscle tightness as a result of passive stretching (National Institute of Neurological Disorders and Stroke, 2013; Sanger, 2015). During such periods of prolonged muscle contraction in the affected limb, oxygen deprivation and ischemia cause the release of pain neurotransmitters (Ivanhoe, 2010). Spasticity-related pain significantly affects the daily living skills and functionality of an individual and is the cause of the most pain in individuals with CP affecting 75% of adolescent participants in a systematic review ("Cerebral Palsy," 2016; Novak, Hines, Goldsmith, & Barclay, 2012); The inability to use limbs in the lower extremity due to spasticity and pain limit ambulation (Esquenazi, 2010). Ambulation when inhibited by spasticity is unsafe, unnatural, and awkward. When postures are

altered by spasticity during gait, they are inefficient, unstable, and painful, resulting in a limited walking pace, maximum distance, and weight bearing capability (Esquenazi, 2010).

The overarching goal of treatment for spasticity is to address deficiencies in capacity to promote motor independence ("Cerebral Palsy," 2016). However, a major limitation of current treatments is a lack of understanding of the underlying mechanisms and their contributions to spasticity. Current treatments such as botulinum toxin injections, oral medication, and stretching result in temporary improvement in stiffness and range of motion and also promotes muscle weakness (Rush & Kumbhare, 2015). Botulinum toxin inhibits the release of acetylcholine to reduce muscle activation and tone, while oral medications target the central nervous system or muscle to reduce muscle tone (Scott, 1980; Thibaut et al., 2013). A better understanding of spasticity would allow improvements in treatments options to be made so that the underlying mechanisms of spasticity are directly targeted instead of generic muscle tone. Limited evidence suggests spasticity can be diminished through continuously applied force at slow velocities; specifically, weight bearing exercises that work against gravity (Hughes & Howard, 2013; Rush & Kumbhare, 2015). Lengthening at slow velocity allows muscle cross-bridges go through the normal attachment cycle, while a high amount of force from weight bearing that occurs in muscle lengthening can be associated with the myosin heads rapidly re-attaching to actin. The greater the amount of cross-bridge attachments made, the greater torque produced, which may be instigated by neural control (Cochrane, 2010; Duchateau, 2004; Jones, Round, & de Hann, 2004). This study has potential to

better the understanding of the components of spasticity and how they differ among various levels of function.

Statement of the problem

Many individuals who are diagnosed with CP face challenges related to spasticity which can in turn result in decreased functionality and increased pain. These symptoms influence an individual's ability to participate in activities of daily living, which negatively impacts quality of life ("Cerebral Palsy," 2016). Current treatment options have shown improvement in pain and spasticity but the improvement is either temporary or results in muscle weakness (Rush & Kumbhare, 2015). The most common treatment involves the injection of Botulin toxin into an affected muscle. This treatment is easy to use and reduces muscle tension; however multiple treatments are required and when the toxin is injected it weakens the muscle (Esquenazi, 2010). Oral medications are less successful in treating spastic hypertonia and tend to show no significant differences in functional improvements of spasticity (Meythaler & Kowalski, 2010). When improvements in spasticity are made with these medications, they negatively affect other aspects of functionality such alertness and strength (Ivanhoe, 2010). Additionally, these medications that were developed for individuals with spinal cord related spasticity, have negative cognitive affects and are not recommended by many clinicians (Meythaler & Kowalski, 2010).

All of the risks negative affects associated with these treatment options are due to the broad nature of the treatments. The underlying mechanisms of spasticity include hypertonia, where muscles are improperly activated and/or hyperreflexia where muscle reflexes are exaggerated. These mechanisms are influenced by the functioning

capabilities of the spinal neurons and motor subsystems along with the supraspinal and suprasegmental mechanisms where the tendon compliance alters, muscle fibers change and affect the functionality of muscles, and the spinal reflexes that regulate the excitability of a muscle loses the ability to inhibit causing hyperexcitability in the muscles (Mukherjee & Chakravarty, 2010). Furthermore, the effects of supraspinal and spinal mechanisms result in a loss of normal functioning motor units which in turn causes a decline in motor neuron firing rate and muscle contraction efficiency (Thibaut et al., 2013). Current treatment options for spasticity could be improved by a better understanding of the components of spasticity and how they differ among various levels of function.

Statement of the Purpose

The purpose of this study is to examine the differences in spasticity at different speeds among individuals with varying levels of spasticity compared to healthy controls through a case study with multiple participants. Patten, Condliffe, Dairaghi, and Lum (2013) found that spasticity is impacted at the cellular level through an increase in motor neuron excitability and a decrease in presynaptic inhibition. To further examine the spastic responses to movement, each participant will engage in a passive stretching protocol, delivered at four criterion speeds (90, 120, 150, and 180°/s). Spasticity will be defined as hypertonic (i.e. improperly activated muscles) and hyperreflexive (exaggerated muscle reflexes) responses elicited during knee extension measured via a dynamometry. This study has the potential to provide additional insight into the underlying mechanisms of spasticity and to inform clinical practices in the fields of occupational and physical therapy.

Research Question

1. What are the differences in spasticity during passive knee extension among individuals with CP compared to healthy controls?

Significance of the Study

It is important to understand the differences in spasticity at different speeds in order to gain more insight into the underlying mechanisms of spasticity. The underlying mechanisms of hypertonia and hyperreflexia are complex and the contribution of each mechanism to spastic response to passive movement remains unknown. By developing a feasible and reliable method to examine the underlying mechanisms related to spasticity, conclusive results of this study may increase the knowledge of the underlying mechanisms of spasticity. Potential benefits of this study include providing increased knowledge for the potential of treatment for these symptoms instead of botulinum toxin (botox) injections and oral medication. The benefits of new treatments include better access to treatments due to reduced cost and presence of insurance coverage, and the avoidance of painful injections which may cause potential muscle damage.

Limitations

1. Differences in participants functional capabilities before testing
2. Small sample size

Delimitations

1. Study participants were from the same geographic background
2. Participants suffered from spasticity that affected functionality

Assumptions

1. Laboratory devices were accurate
2. Participants answered questions truthfully
3. All data was recorded correctly
4. All data was analyzed correctly
5. All participants had an accurate clinical diagnosis of CP as provided by a physician

Definition of Terms

Ataxia – According to ("Ataxia" 2016), Ataxia is "impaired muscle coordination".

Cerebral Palsy – According to ("Cerebral Palsy" 2016), the term Cerebral Palsy is a disorder cause by fetal or infant brain damage that affects the motor functioning of an individual.

Chorea – According to ("Chorea," 2016), Chorea is a condition where the body has sudden irregular and involuntary muscle contractions.

Dynamometer – A dynamometer is a device used to measure power, force, and torque.

Dystonia – According to Fahn et al. (1987), Dystonia is when a muscle twists, performs repetitive movements, and has unnatural postures due to prolonged contractions.

Hyperreflexia- According to Mugge et al. (2012), hyperreflexia is when muscle reflexes are exaggerated

Hypertonia- According to Bar-On et al. (2015), Hypertonia is where muscles are improperly activated

Rigidity - According to Levitt (2009), rigidity is continuous resistance and stiffness of "throughout the full range of motion" of a passive stretch.

Spasticity – According to Sanger (2015), The term spasticity means a painful velocity dependent stiffness or paralysis at a joint that develops increased muscle tightness (Sanger 2015).

Surface Electromyography – Surface Electromyography (EMG) is a device that measures the neuron signal transmission levels in muscles on the surface

Chapter II:

REVIEW OF LITERATURE

Cerebral Palsy

Cerebral Palsy (CP) is a neuromuscular disorder, occurring in 3-4 per 1,000 live births in the United States (Boyle et al., 2011; Rosenbaum et al., 2007; Van Naarden Braun et al., 2016). CP is classified as a group of developmental disorders that limit movement and effect posture and is associated with Limitations in areas such as cognition, perception, and communication (Rosenbaum, 2007). Symptoms of CP include: ataxia, Spasticity, weakness, toe-walking, "variations in muscle tone", shaking, motor skill development delays, difficulty with fine movements (National Institute of Neurological Disorders and Stroke, 2013).

While the exact mechanism of causation for CP is often unknown, the result is damage to the fetal or infant brain preventing typical development (Rosenbaum et al., 2007). This damage occurring before, during, or shortly after birth is often manifested in injury to white matter in the brain, irregular brain development, and/or a lack of oxygen to the brain ("Cerebral Palsy," 2016; National Institute of Neurological Disorders and Stroke, 2013) As white matter carries impulses between nerve cells and is responsible for the transmission of nerve signals, any damage to white matter causes delays and abnormalities in signal transmission (National Institute of Neurological Disorders and Stroke, 2013). Damage to the brain affects both the central and peripheral nervous system (CNS and PNS) as movement occurs through interactions between the both systems; therefore damage to any part of the systems can impair motor control and movement (Shepherd, 2014).

A recent study analyzed the activity levels of children with CP in comparison to individuals without a disability finding that children with CP did not participate as much in actions that required moderate to high levels of activity (Ryan, Forde, Hussey, & Gormley, 2015). The mortality rate for individuals with CP depends on their severity of their disability and their age, younger individuals and those with more severe impairments had lower life expectancy rates of living until thirty years old, where 99% of children with little impairment survived but only 78% of children with two impairments were able to live until this age (Haak, Lenski, Hidecker, Li, & Paneth, 2009). Once an individual survived to be twenty years old the average life expectancy to live to fifty was 86% (Hemming, Hutton, & Pharoah, 2006). In general, individuals with CP have a higher mortality rate than those without but as their age increases so does their life expectancy (Haak et al., 2009). Understanding the differences in spasticity at different speeds among individuals with varying levels of spasticity will potentially allow for better treatment options, which in turn could decrease mortality rates.

Spasticity

Spastic CP is the most common type of CP affecting 70-80% of all CP cases (Thanda, Soe, & Thaingi, 2016). Spastic quadriplegia, the most severe case of spastic CP, is characterized by stiffness in all four extremities (National Institute of Neurological Disorders and Stroke, 2013). Damage to the CNS affects the motor impairments that characterize CP one of which is Spasticity. Damage to central neuron pathways above T12 vertebrae induces muscle weakness, contracture, and muscle over activity associated with spasticity (Mcguire, 2016). Spasticity results from affected motor neurons that result in a velocity dependent stretch reflex that is 'hyper-excited' and is characterized by

stiffness, cramping", or spasms (Brashear & Elovic, 2015; Elovic et al., 2016; Hughes & Howard, 2013; Rush & Kumbhare, 2015). These cramps and spasms occur due to the muscles affected by damage to the CNS. Stretching various speeds result in a spastic 'catch' during a particular range of motion due to muscle reflexes being exaggerated (hyperreflexia) or improperly activated (hypertonia) (Bar-On et al., 2015; Levitt, 2009; Mugge et al., 2012). Limbs affected by spastic CP can be held in abnormal postures due to the inability of weakened muscle groups to overcome the contracture of the spastic muscles (Levitt, 2009). For example, hyperactive tendons found in the lower extremity are described to have "scissor-like" movements because they cannot obtain a normal postural alignment (Levitt, 2009; National Institute of Neurological Disorders and Stroke, 2013). Contractures of spastic are influenced by the functional capabilities of the spinal neurons and motor subsystems along with the Supraspinal and suprasegmental mechanisms where the tendon compliance and muscle fiber properties are altered and the spinal reflexes that regulate the excitability of a muscle lose the ability to inhibit hyperexcitability (Mukherjee & Chakravarty, 2010). Furthermore, the contributions of supraspinal and spinal mechanisms result in a loss of normal functioning motor units which in turn causes a decline in motor neuron firing rate and muscle contraction efficiency (Thibaut et al., 2013).

How spasticity affects quality of life

If increased tone associated with spasticity is not treated, muscle contraction can worsen to a point where non affected muscle groups cannot overcome the strength of the spastic muscles and voluntary movement can be lost (Levitt, 2009). Additionally, movement's impaired by spasticity are also found to be very painful (Rush & Kumbhare,

2015). The complications associated with spasticity such as muscle contraction, abnormal postures, and tissue damage from abnormal posture also result in increased pain (Sheean, 2009). Muscle contractions cause painful spasms in the active muscles along with surrounding soft tissue and joint; abnormal postures cause pain through stiffness, joint subluxation, and improper weight bearing on limbs; tissue damage causes painful skin breakdown and ulcers (Sheean, 2009).

Increased spasticity, increased pain, and decreased functionality all influence an individual's ability to participate in activities of daily living (Goh, Thompson, Huang, & Schafer, 2006). The inability to participate in activities of daily living such as walking, reaching, grasping, and sitting can result in a decreased quality of life (Levitt, 2009); Ivanhoe 2011). According to the World Health Organization (1997), Quality of life is an "individuals perception of their position in life in the context of the culture and value systems" and "a broad ranging concept affected in a complex way by the person's physical health, psychological state, level of independence, social relationships, personal beliefs and their relationship to salient features of their environment" (p. 1). These activities require selective control of muscles to perform specific movements, and spasticity can interfere with their coordination. Spasticity affects movement by making them jerky, fragmented, and difficult to maintain and makes any fine motor movement very difficult (Levitt, 2009). In the lower extremity, spasticity affects an individual's ability to walk and sit, with severe cases of spasticity hindering mobility completely (Ivanhoe, 2010). Immobility can lead to further complications including difficulties toileting and performing other tasks required for self-care. A study conducted by Jaspers et al. (2013) found a fairly correlated inverse relationship in self-reported levels of

spasticity and quality of life with a correlation coefficient of 0.46 in children with lower extremity impairments.

Current treatment options

Current treatment options such as medication and some therapies result in improvements in pain and spasticity, but improvements are either temporary or promote muscle weakness (Rush & Kumbhare, 2015). The most common treatment involves the injection of botulin toxin into an affected muscle. Botulin toxin (botox) comes from Clostridium Botulinum and made of seven types (A-G) whereby an injectable toxin inhibits the release of acetylcholine at the neuromuscular junction, reducing the contraction of the muscle (Pavone et al., 2016). Reducing muscle contraction provides the potential for a greater range of motion and greater capacity for voluntary movement (Esquenazi, 2010). This treatment is easy to use, directly targets specific muscles, and does not require the use of anesthetics, however it does cause some pain and discomfort (Koman et al. (2001). According to Montastruc et al. (2016), adverse drug reactions to botox injections include "dysphagia, muscular weakness, pneumonia, botulism, or death", where a significant association between botox and death exists for children with CP. Botox injections require multiple treatments, on average 3.7 treatments per year and cost £153.21 per 500-unit vial, which weaken the targeted muscle (British Medical Association and Royal Pharmaceutical Society of Great Britain, 2007; Royal College of Physicians, British Society of Rehabilitation Medicine, Chartered Society of Physiotherapy, & Association of Chartered Physiotherapists Interested in Neurology, 2009). A constant weakening of the muscle multiple times results in almost a "paralytic effect" where the muscle loses functionality (Esquenazi, 2010). Patients who suffer from

spasticity suffer from increased tone and progressing weakness in the affected muscles. When botulin toxin was injected into these muscles pain and tone was reduced, but the limited functional capabilities that the individuals possessed was also reduced because the muscle tone was no longer present (Orsini et al., 2015).

Meythaler and Kowalski (2010) recently examined differences between oral medication trials used to treat spinal and cerebral causes of spasticity. In general, oral medications were less successful in treating spastic hypertonia and tended to show no significant differences in functional improvements of spasticity. When improvements in spasticity are made with these medications, they often negatively affect other aspects of functionality such alertness and strength (Ivanhoe, 2010). Due to the many negative affects of some current treatment options, other modalities and therapies to reduce spasticity are urgently needed.

Chapter III:

METHODOLOGY

The purpose of this study was to examine underlying mechanisms of spasticity in Cerebral Palsy (CP). Chapter 2 addressed the need for further examination of the possible benefits of hypertonia and hyperreflexia on spasticity individuals with CP.

Participants

This study had 18 participants who participated in a passive stretch protocol at four different velocities using the Biodex system 4 Pro dynamometer. The participants included nine adolescents, five female and 4 male (mean age: 15.2; standard deviation: 2.1), with CP and nine age-matched youth or adults (mean age: 15.3; standard deviation: 2.5) without CP or any other neurological disorders were recruited from the local southeast Georgia area. Confirmation of diagnosis was done through a review of medical records. Participants met the most recent definition of CP by Rosenbaum et al. (2007) and were within Levels I through V on the Gross Motor Function Classification System (GMFCS). According to the GMFCS, Level I represents individuals with motor impairments whose "functional limitations are less than what is often associated with Cerebral Palsy" and includes children "diagnosed as having 'minimal brain dysfunction'" and Level V represents individuals with severe functional limitations and severe brain dysfunction. Participants with CP were further broken down into two groups, individuals with low CP (GMFCS I-III) and high CP (GMFCS IV & V). Participants were selected based on the following criteria: a) between the ages of 12 and 19 years old, b) within levels I through V of the Gross Motor Classification System, and c) cleared by a medical

professional for physical activity. The diagnosis of CP will be accepted from the student's individualized education program (IEP) document.

Protection of Human Subjects

Approval was obtained from Georgia Southern University's IRB in spring of 2016. Participation in the exercise program was voluntary and informed content was received from both the participant and the participant's guardian. Each family was compensated $50 each time they come to campus for data collection. Paying families $50 for each data collection session increased participation dramatically, reduced attrition, and compensated them for their time and transportation. Many potential participants were of low socioeconomic status or lived in remote, rural areas in Bulloch County, making transportation difficult. An incentive of $50 covered all potential costs and promoted attendance to data collection. Participants were informed of their ability to discontinue the stretching intervention and of the steps taken to protect their confidentiality.

Instrumentation

Biodex System 4 Pro dynamometer. The Biodex system 4 Pro dynamometer is a "computerized robotic dynamometer" used to collect neuromuscular data, "helps patients build strength, endurance and coordination," and aid in Spasticity management through "objective quantification at specific contraction"(Biodex Medical Systems, 2017). In previous studies the Biodex has been proven to be reliable in assessing "peak torque" and changes in muscle contractions (Feiring, Ellenbecker, & Derschei, 1990; Hartmann, Knols, Murer, & de Bruin, 2009). The device was used to conduct a passive stretching intervention with repetitions of extension and flexion within a comfortable preset range

of motion following the protocol established by (Moreau, Li, Geaghan, & Damiano, 2009).

Spastic responses to passive stretching was measured in the same way as Moreau et al. (2009) where, spasticity was operationalized as the peak resistive torque (RT) during the isokinetic portion of the passive range. Velocity-dependent hypertonia and hyperreflexia responses were tested by operating the dynamometer in passive mode. Each trial had two phases: i) passive knee extension at criterion speed; ii) passive return to knee flexion. Range of motion (ROM) was preset at a comfortable range for each participant and each participant went through a passive ROM at a constant speed for five repetitions. During all movement phases, participants will be instructed to relax as the limb was moved through the full range of knee motion by the dynamometer. Torque, position, and acceleration data were collected before and during passive knee extension stretches. Passive stretches were delivered at four criterion speeds (i.e., 90, 120, 150 and 180°/s). Passive range of motion set limitations on the motion throughout the testing process.

Procedures. After institutional review board (IRB) approval, participants with CP attended 2 sessions and each age match control attended one session. A total of 1.5 hours was spent for each treatment session. During all movement phases, participants were instructed to relax as the limb was moved through the full range of elbow and knee motion by the dynamometer. Repeated extension and flexion of the knee was done at an individual-based set range of motion. Passive stretching was completed using dynamometry to elicit spastic responses. Participants were stretched at four criterion speeds (i.e., 90, 120, 150 and 180°/s). Each trial had two phases: i) passive knee extension

at criterion speed; ii) passive return to knee flexion. Range of motion (ROM) was preset at a comfortable range for each participant and each participant went through a passive ROM at a constant speed for five repetitions.

Data Collection. Data Collection will be held at the Human Performance and Biomechanics Laboratory at Georgia Southern University. The individuals who will administer the passive stretching intervention underwent extensive training with the equipment and exercises to be executed.

Passive knee extension at four criterion speeds (i.e., 90, 120, 150, and 180°/s). was delivered using a dynamometer to observe potential spastic responses.

For each test session, participants were seated in the dynamometer chair with the back angled at 85°, the trunk stabilized using waist and trunk straps, and the feet supported using the leg rest. The testing leg was positioned with the knee in 0° abduction, 0° flexion with the lateral epicondyle of the femur aligned with the dynamometer rotational axis. Passive knee extensions covered the participant's full anatomical available range of motion. The anatomical position was determined using a handheld goniometer and reported in degrees of knee flexion (i.e., full extension = 0°). Anatomical angles were used to report subject-specific joint angles for possible the onset of spastic activity.

Velocity-dependent spastic responses were be tested by operating the dynamometer in passive mode. Each trial had two phases: i) passive knee extension at criterion speed; ii) passive return to knee flexion. During all movement phases, participants were instructed to relax as the limb was moved through the full range of knee

motion by the dynamometer. Torque, position, and acceleration data was collected before and during passive knee extension stretches.

Data Analysis. A mixed model repeated measures analysis of variance was performed to examine differences in resistive torque during passive knee extension at the four different speeds among young people with CP in GMFCS I-III, GMFCS VI-V, and healthy controls. It was hypothesized that participants with lower function would exhibit greater levels of resistive torque. Statistical analysis was performed using SAS© software, Version 9.3 (Cary, NC, USA). Alpha level was set at .05.

Chapter IV:

RESULTS

Multiple statistically significant interactions were observed by groups at different velocities (see Table 1 and 2). There was a significant difference ($p<.05$) of mean (*M)* resistive torque between the control group and the GMFCS IV & V group. No significant difference in mean (*M)* resistive torque was found throughout the change in velocity. There was a significant difference between Mean (*M)* resistive torque left (-18.8 N-m) and right (-17.5 N-m) where $p<.0001$. There was a significant difference ($p<.05$) of overall peak resistive torque for all speeds between the control group (-34.3 N-m) and the GMFCS IV & V group (-17.1 N-m) and no significant difference was found comparing the control and GMFCS IV & V groups to the GMFCS I-III group (-27.4 N-m). There was a significant difference between peak resistive torque left (-26.8 N-m) and right (-25.6 N-m) where $p=.0059$. There was not a significant difference of overall peak resistive torque for groups among velocities of 90°/s^a (-26.8 N-m), 120°/s^a (-27.1 N-m), and 150°/s^a (-26.7 N-m); a significant difference was found comparing velocities 90°/s, 120°/s, and 150°/s to the velocity of 180°/s (-24.5 N-m) where $p<.05$.

Table 1

Interactions of Resistive Torque Observed by Groups at Different Velocities

	Group		
Velocity (°/s)	Control *M* (SE)	GMFCS I-III *M* (SE)	GMFCS IV & V *M* (SE)
90	-19.3[a] (1.30)	-15.3[ab] (1.57)	-11.3[b] (2.22)
120	-22.8[a] (1.30)	-18.2[ab] (1.57)	-13.0[b] (2.22)
150	-23.5[a] (1.30)	-20.5[ab] (1.57)	-14.2[b] (2.22)
180	-24.9[a] (1.30)	-20.9[ab] (1.57)	-14.2[b] (2.22)

Note. Groups with the same symbol (a, ab, b) in a row/column are not significantly different.

Table 2.

Interactions of Overall Resistive Torque Observed by Groups

Group		
Control *Peak* (SE)	GMFCS I-III *Peak* (SE)	GMFCS IV & V *Peak* (SE)
-34.3[a] (2.14)	-27.4[ab] (2.61)	-17.1[b] (3.69)

Note. Groups with the same symbol (a, ab, b) in a row/column are not significantly different.

Chapter V:

DISCUSSION

Results indicated that the Biodex System 4 Pro dynamometer was able to detect differences in spastic muscle contractions that resist gravity by using the knee attachment to detect the amount of torque (N-m) present, where a higher negative resistive torque value represents little to no muscle contraction present while a lower negative resistive torque value represents high muscle contraction present that was resisting gravity. Resistive torque during passive knee extension increases as functionality levels of the participants decreases due to the fact that a decrease functionality corresponds with a higher active spastic muscle contraction resisting gravity. Theses findings are similar to the findings of Damiano, Quinlivan, Owen, Shaffrey, and Abel (2001) that quantified quadriceps and hamstrings resistive torque values of individuals with CP at velocities of 30, 60 and 120 °/s finding a correlation of higher resistance torque values with participants ranked lower on the GMFMS. However, in this study only the quadriceps were analyzed and higher velocity values were used and mean resistive torque values were also analyzed. According to the literature, stretching various speeds result in a spastic 'catch' during a particular range of motion due to muscle reflexes being exaggerated (hyperreflexia) or improperly activated (hypertonia) (Bar-On et al., 2015; Levitt, 2009; Mugge et al., 2012). In this study, a significant difference in resistive torque values only occurred during a velocity of 180°/s when analyzing overall peak torque values. Further research into is needed to analyze the underlying mechanisms of CP using both dynamometry and sEMG. More research in understanding the underlying mechanisms would potentially help develop a better treatment option.

Hypertonia and hyperreflexia are two of the underlying mechanisms that affect and mediate the stretch reflex observed in spastic responses. Hypertonia is a symptom caused by several components both neural and non-neural where both components contribute to the resistance to passive motion; the non-neural component include stiffness and viscosity while the neural component includes stimulation of the Hoffman and tendon stretch reflexes (Bar-On et al., 2015; de Gooijer-van de Groep et al., 2013; Harlaar, Becher, Snijders, & Lankhorst, 2000; Meyer, McCulloch, & Lieber, 2011). According to Smith, Lee, Ward, Chambers, and Lieber (2011), non-neural components contribute to increased stiffness due to higher hamstring extracellular matrix collagen levels which start taking affect during early childhood (Barber, Barrett, & Lichtwark, 2011; Willerslev-Olsen, Lorentzen, Sinkjær, & Nielsen, 2013). The neural component is affected by subject specific factors including activation patterns, biomechanical properties, muscle specific responses, and the joint position before a stretch (Bar-On et al., 2015; Musampa, Mathieu, & Levin, 2007).

Hyperreflexia occurs when a muscle's stretch reflex is exaggerated (Mugge et al., 2012). The hyperexcitability of the stretch reflex can be attributed to the central mechanisms of reflex control; injury or disease can affect spinal inhibition, plasticity, motor neuron excitability, and an increase in tonic stretch reflexes with exaggerated tendon jerks (Ashby, Mailis, & Hunter, 1987; Bandaru, Liu, Waxman, & Tan, 2015; Bennett, Li, Harvey, & Gorassini, 2001; Boulenguez & Vinay, 2009; Hultborn & Nielsen, 2007; Hunanyan, Petrosyan, Alessi, & Arvanian, 2013; Lance, 1980; Nielsen, Crone, & Hultborn, 2007). Dendritic spines have the ability to alter sensory information and are responsible for efficient signal transmission (Bourne & M., 2007; Calabrese,

Wilson, & Halpain, 2006; Tan, Choi, Waxman, & Hains, 2009). According to Yates, Charlesworth, Reese, Skinner, and Garcia-Rill (2008), exercise before the onset of hyperreflexia and continuous exercise after the onset can help prevent hyperreflexia, in addition, excerise only initiated after the onset can help the animal return to normalized reflexes but requires a longer duration of treatment. Due to the possibility of preventing and recovering from hypertonia more studies are needed to further understand the mechanisms of spasticity and their contributions to spastic response in hopes of developing better treatment options.

One of the difficulties in treating spasticity comes from the difficulties in understanding the differences between its underlying mechanisms. Recent studies have developed algorithms and models to help differentiate between spastic and healthy muscle (Bar-On et al., 2015). These studies have tried to distinguish between non-neural and neural components but have led to contradictions, inaccurate estimations, and had an inability to distinguish between the properties of muscle and tendon (Bar-On et al., 2015) This difficulty in this differentiation has been acknowledged in other studies (Lorentzen et al., 2010; Willerslev-Olsen et al., 2013). According to Geertsena et al. (2015), an future protocol would involve both biomechanical and electrophysiological methods with, the potential to allow for accuracy and distinguishability while limiting contradictions. Knowledge of the underlying mechanisms of spasticity will allow training modalities to be improved by developing treatments to target specific mechanisms that are found to affect the individual; targeting these mechanisms will allow for a potentially more effective treatment option. Currently, there is no feasible and reliable method to examine these underlying mechanisms and their contributions. A more precise method to

measure these mechanisms and their contributions to spasticity could lead to the development of more targeted and effective treatments.

Limitations

Although this study provides useful information for future research, it does have some limitations. There are limitations with a small samples size being used and all the participants being from the same geographical area, so the data collected may not adequately represent all individuals with CP. Another limitation is in the variability in participants' functional capabilities before testing, individuals with greater functional capabilities potentially have the ability to move their extremities through greater ranges of motion. In addition, during testing there was a limitation with participants exhibiting spastic movements prior to initiation of testing due to stretching required for body positioning. However, the primary goal of this study was to examine the differences in spasticity at different speeds among individuals with varying levels of spasticity, so more knowledge can be gained rather than to represent all adolescents with CP.

Implications

In summary, this study implicates that dynamometry can be used to detect differences in spastic muscle contractions by detecting the amount of torque (N-m) present. This information can potentially help differentiate between the underlying mechanisms of spasticity. Knowledge of the underlying mechanisms of spasticity will allow improvements in treatments to target specific mechanisms of spasticity; targeting these mechanisms will allow for a potentially more effective treatment option that will aid in the avoidance of painful injections that cause muscle damage, along with a

potential to reduce treatment cost and increase the presence of insurance coverage thus allowing for better access to treatment.

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