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# The Relationship Between Spasticity and Muscle Volume of the Knee Extensors in Children With Cerebral Palsy

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## Abstract

**Purpose—**The purpose of this study was to examine the relationship between spasticity and muscle volume in children with cerebral palsy (CP), using isokinetic dynamometry and magnetic resonance imaging.

**Methods—**A retrospective sample of 8 children with diplegic CP was analyzed. One set of 10 passive knee flexion movements was completed at a velocity of 180° per second with concurrent surface electromyography of the medial hamstrings (MH) and vastus lateralis (VL) to assess knee extensor spasticity. Magnetic resonance imaging was used to measure maximum cross-sectional area and muscle volume of the quadriceps femoris.

**Results—**The quadriceps femoris muscle volume was positively correlated with MH reflex activity, VL reflex activity, MH/VL co-contraction, and peak knee extensor passive torque ($P < .05$).

**Conclusion—**The present findings suggest that higher levels of knee extensor muscle spasticity are associated with greater quadriceps muscle volume in children with spastic diplegic CP.

### Keywords

*child*; *cerebral palsy/physiopathology*; *electromyography*; *magnetic resonance imaging/methods*; *motor activity*; *muscle/anatomy/histology*; *muscle contraction*; *muscle spasticity muscle weakness/physiopathology*; *quadriceps muscle/physiopathology*; *retrospective study*

## INTRODUCTION AND PURPOSE

Spasticity is a common impairment found in children with cerebral palsy (CP)[1] and may contribute to limitations in motor function.[2] It has been recently defined as "disordered sensorimotor control, resulting from an upper motor lesion, presenting as intermittent or



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sustained involuntary activation of muscles."[3(p5)] One method of measuring spasticity involves an isokinetic dynamometer to passively move a limb through a defined range of motion at a defined velocity while the peak resistive torque is measured during the movement.[4,5] An advantage to using isokinetic dynamometry to assess spasticity is that both the reflexive and nonreflexive components of spasticity can be quantified objectively by concurrent acquisition of electromyographic (EMG) and torque data.[6] Recent studies have revealed increased co-contraction and reflex activity during the assessment of spasticity in children with CP compared with peers with typical development, using such isokinetic dynamometry methods.[7,8]

Reduced muscle mass is another common impairment found in children with CP. Malaiya and colleagues[9] reported less muscle volume of the medial gastrocnemius in children with hemiplegic CP than in children with typical development, using ultrasound imaging techniques. Alternatively, Lampe et al[10] found diminished volumes of the muscles of the more affected lower extremity when compared with the less affected lower extremity in a sample of adolescents and young adults with spastic hemiplegia, using magnetic resonance imaging (MRI) measurement techniques. Johnson et al[11] discovered 51% less mid-thigh cross-sectional area (CSA) in individuals with quadriplegic CP than in a control group. Finally, Moreau and colleagues[12] recently reported decreased CSA of the rectus femoris and decreased muscle thickness of the rectus femoris and vastus lateralis (VL) when comparing children with spastic CP with children with typical development.

The relationship between muscle volume and spasticity, however, has not been well investigated in children with CP. In a study of children and adolescents with CP, Ohata et al[13] found no correlation between muscle thickness of the quadriceps femoris (QF) measured by ultrasonography and spasticity measured by the Modified Ashworth Scale (MAS). Interestingly, Gorgey and Dudley[14] reported a significant correlation between the MAS score and muscle CSA in adults with incomplete spinal cord injury and suggested spasticity may assist with maintaining muscle size after an injury. The work of Lofvenmark and colleagues[15] supported this hypothesis in individuals with spinal cord injury reporting that those with severe spasticity as measured by the MAS had greater muscle mass in the lower extremities than individuals with no or mild spasticity. Because of the subjective nature of rating spasticity using the MAS, the validity and reliability of the MAS as a quantitative measure of assessing spasticity have been questioned by multiple authors.[16–18] The characteristics of the MAS may contribute to the inconsistent findings of the relationship between spasticity and muscle mass to date.

The use of reliable methods of spasticity measurement such as isokinetic dynamometry and concurrent EMG recording could provide more clear insight into the relationships between muscle volume and spasticity. To our knowledge, the relationship between co-contraction and reflex activity measured during the assessment of spasticity and muscle volume has not been previously investigated in children with CP. The purpose of this study was to examine the relationships between spasticity, reflex activity, co-activation, and muscle volume in children with CP, using isokinetic dynamometry and MRI measurement techniques.

## METHODS

### Subjects

A university-affiliated institutional review board approved this investigation. Written informed consent from each child's parents along with written assent was obtained from each child prior to participation. Children met the following inclusion criteria: (1) a diagnosis of spastic diplegic CP; (2) between 7 and 14 years of age; (3) no documentation in the medical record of hip subluxation, hip dislocation, or significant scoliosis (curvature >

40°); (4) ability to follow 1-step commands and to attend to tasks associated with data collection; (5) 1 or more years postsurgery of the lower extremities; (6) 6 months or more post–botulinum toxin injection; (7) 10° or less of knee flexion contracture; and (8) absence of magnetically active or cardiac implants. A retrospective sample of 8 children with CP (mean age = 10.4 years, SD = 1.8, range 8.0–13.6; Gross Motor Function Classification System [GMFCS] level II = 2, GMFCS level III = 6) who had participated in separate protocols involving spasticity testing and thigh MRI was analyzed. Spasticity and MRI protocols were completed within a period of 1 week.

## Measurements

The procedures to measure spasticity used in this investigation have been described elsewhere[8] and are summarized here. The right limb was tested, and 2 surface EMG electrodes were placed over the VL and the medial hamstring (MH). Electrical activity of the muscles was recorded using Delsys signal conditioning electrodes (Delsys Inc, Boston, Massachusetts) with a parallel bar arrangement (contact area 1 × 10 mm, 10-mm interelectrode distance), a gain of 1000 V/V, a common mode rejection ratio of greater than 80 dB at 60 Hz, a noise level of 1.5-μV RMS, and a band-pass filter of 20 to 450 Hz. A Bagnoli 4-channel EMG system (Chattex Corporation, Chattanooga, Tennessee) with a band-pass filter between 20 and 450 Hz and a gain of 1000 V/V provided additional processing of the EMG signal. The sample rate of EMG signals was 1.2 kHz.

Subjects were seated on the isokinetic dynamometer (National Instruments Corporation, Austin, Texas) in a position of 80° of hip flexion, 90° of knee flexion, and with the ankle unrestricted. The axis of the knee joint was visually aligned with the axis of the dynamometer. Baseline resting EMG data were collected while the subjects were not moving. One set of 3 continuous passive movements at a velocity of 5°/s was collected for gravity correction of the limb's weight. Data were collected using a personal computer and custom software written in Labview 5.1 (The Math Works Inc, Natick, Massachusetts) for offline data analysis. One set of 10 continuous passive movements from 25° of knee flexion to 90° of knee flexion was completed at a velocity of 180°/s, with a return speed of 5°/s to assess knee extensor spasticity. A custom Matlab program (The Math Works Inc) was used for postprocessing and analysis of data. The gravity corrected knee extensor peak resistive torque was calculated during the constant velocity portion of each movement. The peak torque was identified as the maximum passive resistive torque of the 10 movement repetitions.

EMG data were full-wave rectified and processed using a second-order Butterworth 10-Hz low-pass filter with phase correction to create a linear envelope. Electromyographic onset and offset were defined as muscle activity that was 3 SDs above baseline and occurred for a minimum of 50 milliseconds.[19] The percentage of the range of movement with EMG classified as reflexive or co-activation was quantified for the repetition exhibiting peak torque. Electromyographic activity of either the MH or VL during passive movement was defined as reflexive muscle activity. Simultaneous EMG activity of both the MH and VL during passive movement was defined as co-activation.

The MRI protocol used in this investigation has been described elsewhere[20] and is summarized here. Within 2 days of spasticity testing, a clinical 1.5-T magnet MRI (GE Medical Systems, Waukesha, Wisconsin) was used to measure the maximum muscle anatomic CSA and muscle volume of the QF. A standard thoracic coil was used to collect images of the right leg while the child was in the supine position. Sequential scans were used to acquire 3-dimensional data from the QF from proximal to distal, using a standard spoiled gradient-echo sequence. Coronal T1-weighted spin-echo localizing scans and transverse 3-dimensional spoiled gradient-echo images were acquired with an encoding

matrix of 256 × 256 × 28. The slice thickness was 7 mm, and chemically selective fat suppression was used to enhance visual definition between muscle groups for all scans.

The fat-free CSA of each imaged muscle for each slice was calculated using an interactive computer program and a correction algorithm.[21] Individual segments of each head of each muscle were analyzed and then summed to determine the CSA of each slice. The slice with the highest summed CSA of the component muscle heads was defined as the maximum CSA. Volume was calculated by multiplying by the number of slices and the slice thickness by the total CSA of the muscle.

Data analysis was completed using SPSS version 14.0. Data were analyzed for normality and found to be nonnormally distributed. Spearmen correlation coefficients were completed to determine the relationship between measurements of knee extensor muscle mass (maximum CSA, muscle volume, CSA normalized to patient weight, and muscle volume normalized to patient weight) and knee extensor spasticity (peak passive torque, reflex activity of the MH and VL, and co-contraction). The level for statistical significance was set at $\alpha < .05$.

## RESULTS

The correlation matrix examining relationships between measures of QF muscle mass and knee extensor spasticity are presented in the Table 1. Significant positive correlations were found between muscle volume and the percentage of the passive range of motion with MH activation, VL activation, and co-contraction, ($P < .05$). Also, peak knee extensor passive torque was positively correlated to muscle volume ($P < .05$).

Significant positive correlations were found between maximal CSA and the percentage of the passive range of motion with MH activation, VL activation, and cocontraction ($P < .05$). There was also a significant positive correlation between muscle volume normalized to weight and VL activation ($P < .05$).

## DISCUSSION

Our results suggest that quadriceps muscle volume is greater in children with diplegic CP who have higher levels of spasticity as measured by both passive torque and EMG measures of spasticity. While Lieber et al[22] summarized the literature regarding structural changes in spastic muscle and noted that changes in muscle fiber size, fiber type distribution, stiffness, and extracellular matrix contributed to spasticity, this study is the first to report a relationship between muscle mass and spasticity.

Our results conflict with the report of Ohata et al,[13] who found no relationship between spasticity and muscle thickness. However, differences in the methodology used to assess spasticity (MAS vs isokinetic dynamometry) and muscle size (ultrasonography vs MRI) could account for these conflicting results. Specifically, the use of isokinetic dynamometry to measure spasticity differs from the MAS in a number of ways. First of all, the MAS employs an ordinal scale that may be less sensitive than isokinetic dynamometry that measures spasticity on a ratio scale. Also, the position of the child being assessed is more standardized with isokinetic dynamometry through the use of seat belts and other equipment. Finally, the speed of passive limb movement is standardized using an isokinetic dynamometer whereas the speed of passive movement using the MAS is dependent on the assessor. An additional methodological issue that may have affected our results is the use of the percentage of the range of motion with EMG activity as our measurement of EMG response. The use of a 3-SD response from baseline as the measurement was decided to maximize the reliability of the determination of EMG onset,[19] but the magnitude of the

EMG above 3 SDs was not calculated, which may have provide additional information regarding the reflexivity of the muscle. Further research investigating different methodologies for the assessment of spasticity is warranted.

The direction of a possible causal relationship between muscle spasticity and muscle volume is unknown because of the correlational design of this study. Although our previous research suggests that passive muscle stiffness contributes more to spasticity in children with CP than increased reflex activity,[8] we hypothesize that the muscles of children who have increased reflex muscle activity associated with their spasticity may have increased muscle volume due to the reflexive muscle contractions decreasing the effect of disuse atrophy. Our finding of a relationship between MH activity and QF muscle mass is likely due to MH activity occurring in conjunction with VL activity as co-contraction.[7] A startle response to the movement of the limb by the dynamometer may have also caused this cocontraction, [7] but we are unable to differentiate a startle response from co-contraction due to the retrospective nature of this study. It is unknown whether the increase in muscle size positively affects function and muscle strength, especially since the investigations of the relationships between spasticity, strength, and function have reported conflicting results in children with CP.[2,23,24]

An alternative hypothesis for our results is that children with increased muscle size show increased spasticity because their larger muscles would be stiffer during passive movements. Fewer significant relationships were detected using normalized measures of muscle mass, which suggests that relative muscle size may be less of a factor with spasticity whereas growth and aging may contribute to greater spasticity. Although our previous work has shown a positive relationship between age and knee flexor spasticity, no relationship was found between age and knee extensor spasticity.[25] Further research is needed to determine the contribution of aging to the relationship between spasticity and muscle mass.

The clinical significance of our finding of a positive relationship between spasticity and muscle volume in children with CP is unknown. There is an emerging literature that has investigated the possible beneficial effect of spasticity in people with spinal cord injury. Gorgey and colleagues[26] recently reported that muscle spasticity in people with chronic complete spinal cord injury was associated with improved body fat composition as measured by fat-free mass and indirectly improved metabolic profiles with regard to lipid profile and glucose homeostasis. Also, Bennegard and Karlsson[27] found that spastic lower extremities demonstrated increased glucose uptake compared with control subjects and concluded that spasticity may be protective with regard to diabetes. An important consideration for children with CP is that they may be at risk for diabetes due to increased adipose tissue infiltration of skeletal muscle[11] and greater inactivity[28] than children with typical development. Additional research is clearly needed to investigate the possible positive effects of spasticity on metabolism and body composition in children with CP.

The results of this study should be interpreted cautiously because of the small sample size used in the investigation. It is unknown whether the results of this study would generalize to other muscle groups and age ranges in children with spastic CP. One limitation of this study is that our data analysis was limited to the QF and did not examine the knee flexor muscle group. Also, children with different levels of functional mobility may or may not show a similar relationship between spasticity and muscle size. Additional research is needed to determine whether our finding of a relationship between spasticity and muscle volume is generalizable to children with different age ranges, levels of functional mobility, and muscle groups.

## CONCLUSION

Our results suggest that higher levels of knee extensor muscle spasticity are associated with greater quadriceps muscle volume in children with spastic diplegic CP. The clinical significance of this finding is unknown, and additional research is required to investigate the possible positive effects of spasticity on metabolism and body composition in children with CP.

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## TABLE 1

Correlation Matrix for Measures of Quadriceps Femoris Muscle Mass and Knee Extensor Spasticity

| | Peak Knee Extensor Passive Torque | EMG Percentage | | |
|---|---|---|---|---|
| | | Medial Hamstrings | Vastus Lateralis | Co-contraction |
| Maximal cross-sectional area | 0.619 | 0.732[a] | 0.735[a] | 0.786[a] |
| Volume | 0.738[a] | 0.845[a] | 0.831[a] | 0.896[a] |
| Cross-sectional area/weight | 0.071 | 0.282 | 0.374 | 0.270 |
| Volume/weight | 0.405 | 0.374 | 0.735[a] | 0.516 |

[a] $P < .05$.