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# Can Spasticity and Dystonia Be Independently Measured in Cerebral Palsy?

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**Selecting and evaluating appropriate treatments for children with cerebral palsy has been challenging. One difficulty is in the ability to quantify the presence and importance of coexisting motor signs. This study presents quantitative measures developed to assess spasticity and dystonia. Children diagnosed with extrapyramidal or spastic cerebral palsy and matched control children were studied. Spasticity was measured as the slope of the force-velocity relationship from a test where we measured the forces required to passively extend the elbow at different velocities. Dystonia was assessed by measuring "overflow" movements of arm during active movement of the other arm. Measures of dystonia and spasticity did not correlate with one another, but did correlate with their respective clinical measurement tools, the Modified Ashworth scale and the Barry-Albright Dystonia scale. Most children had a combination of both spasticity and dystonia, despite diagnosis. Our measures also related to different aspects of reaching: children with increased dystonia made more curved paths, and children with increased spasticity hit higher peak velocities. These measurements allow us to distinguish between different motor disorders and the degree to which each contributes to reaching performance. Use of quantitative measures should improve selection and evaluation of treatments for childhood motor disorders.** 

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## Introduction

Cerebral palsy represents a heterogeneous group of disorders caused by nonprogressive disturbances of the developing brain, leading to dysfunction of movement and postural development [1-4]. Other impairments (i.e. in sensation, cognition, communication, perception, behavioral, seizure disorder) often accompany the motor dysfunction [1]. The motor disturbances associated with cerebral palsy can range from mild to severe, and may dramatically impair a child's functional abilities. Children with cerebral palsy frequently have mixed motor disorders (e.g., spasticity, athetosis, ataxia, and weakness), and each likely impairs their functional movement in a different way. Despite the coexisting motor disorders, children with cerebral palsy often fall into one of two classifications: "spastic" or "extrapyramidal" cerebral palsy.

Children with "spastic" cerebral palsy characteristically present with spasticity, weakness, and loss of manual dexterity due to abnormalities in descending motor pathways and motor cortex. Spasticity is defined as increasing resistance to increasing speed of stretch relative to the direction of joint movement or a rapid rise in resistance above a speed or joint position threshold [5]. Spasticity has been widely measured by clinical rating scales (Ashworth scale) [6] and instrumented measures (torque-velocity relationships and velocity-electromyography relationships) [7,8]. Studies of the degree of spasticity in the lower extremities have not correlated well with aspects of gait function [9].

Dystonia, rigidity, and athetosis are primary neurologic findings of "extrapyramidal" cerebral palsy, presumably a result of abnormalities in basal ganglia-cortical circuits. Dystonia is defined as sustained or intermittent muscle contractions causing twisting and slow repetitive move-

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ments or abnormal postures [5]. It can manifest as overflow of activity to muscles that are normally silent during a voluntary movement (e.g., other muscles in that limb or other limbs), or involuntary activation of muscles at rest [10]. Dystonia is often measured clinically in children with cerebral palsy using the Barry-Albright rating scale [11]. As with spasticity, the relatively few published studies describe physiologic measures of dystonia, though some have reported relationships with voluntary movements and walking [12,13].

Quantitative measures of movement are currently available but have not been widely used. A recent study was the first to describe biomechanical measures that varied in children who were clinically described as having predominantly spasticity vs dystonia [13]. Children with dystonia had greater co-contraction, normal tendon reflexes, and reduced muscle strength all at the knee joint. They also walked with reduced range of knee motion compared with children with spasticity. Thus, preliminary evidence suggests biomechanical measures may be used to distinguish between these types of hypertonicity.

Clinical classifications have been useful in guiding treatments for children with specific motor disorders. However, mixed hypertonia, with components of spasticity and dystonia, is likely to be found in the vast majority of children with cerebral palsy [5]. Identifying the degree to which each motor component contributes to functional movement within an individual is challenging owing to limitations of clinical measurement tools that may be insensitive to small changes in motor performance, and may not specify the mechanism of improvement. With distinct pharmacologic interventions available for different neurologic findings, motor signs, and movements observed in children with cerebral palsy, quantitative measures of motor deficits could provide a more accurate means by which to assess treatments for cerebral palsy [14]. Reported here are relatively simple quantitative measures developed for assessing the motor disorders present in children with "extrapyramidal" and "spastic" cerebral palsy and the extent to which each was found to contribute to the impairments in voluntary arm movements.

## Methods

### *Subjects*

Thirteen children ages 7 to 17 years, diagnosed with cerebral palsy and subcategorized as spastic vs extrapyramidal (Table 1) were studied. For comparison, eight age-matched healthy control subjects were tested as well. The mean age of the cerebral palsy group was 12 and the control group was 10. No subjects had ever had surgery on the tested upper extremity. One subject (cerebral palsy 10) had Botox to the wrist flexors of the test arm 8 months before testing. Medical history was obtained from medical chart review or the family of each child, including history of: shunted hydrocephalus (Subjects 1 and 5), human immunodeficiency virus (Subject 2), in utero drug exposure (Subject 4), and toxoplasmosis/cytomegalovirus (Subject 12). Four 4- and 5-year-old children were initially recruited but were excluded from the study and the statistical analyses secondary to inability to attend to and complete the task. Parents or guardians gave informed consent for the children, and older children gave assent in accordance with the Institutional Review Board of the Johns Hopkins University School of Medicine.

### *Tasks*

Before testing, all subjects were rated on the Modified Ashworth scale at the elbow and wrist, and on the Barry-Albright Dystonia scale. Measurements were then made on the arm most affected in the cerebral palsy subjects. During testing, subjects were frequently given breaks to ensure that fatigue did not contribute to movement abnormalities.

**Table 1. Clinical demographics**

| Cerebral Palsy Subject | Age (yr) | Sex | Clinical Diagnosis | Gest. Age (wk) | MRI Results | BAD | Ash. | Medications |
|---|---|---|---|---|---|---|---|---|
| 1 | 17 | M | Spastic Di | 28 | Not available | 10 | 0 | None |
| 2 | 8 | F | Spastic Di | 40 | Not available | 12 | 1.5 | Baclofen |
| 3 | 9 | F | Spastic Di | 30 | Not available | 2 | 1 | None |
| 4 | 9 | M | Spastic Di | 32 | Mild-moderate PVL | 5 | 1 | Baclofen |
| 5 | 17 | F | Spastic Quad | 30 | Mild-moderate PVL | 14 | 2 | Baclofen |
| 6 | 17 | F | Spastic Di | 32 | Not available | 6 | 1.5 | Baclofen |
| 7 | 10 | M | Extrapyramidal | 40 | Schizencephaly/BG Injury | 4 | 0 | None |
| 8 | 13 | M | Spastic Di | 40 | Schizencephaly | 13 | 1.5 | None |
| 9 | 11 | M | Spastic Quad | 28 | Not available | 7 | 1 | None |
| 10 | 7 | F | Spastic Di | 26 | Moderate PVL | 18 | 2 | Artane |
| 11 | 12 | M | Spastic Di | 29 | Severe PVL/(R) porencephaly | 7 | 0 | None |
| 12 | 10 | M | Spastic Di | 27 | Not available | 22 | 2 | Valium Robinul |
| 13 | 16 | F | Spastic Quad | 26 | Not available | 27 | 2 | Valium |

Abbreviations:
Ash. = Modified Ashworth Score
BAD = Barry-Albright Dystonia scale
BG = Basal ganglia
Di = Diplegia
MRI = Magnetic resonance imaging
PVL = Periventricular leukomalacia
Quad = Quadriplegia
(R) = Right

Spasticity of the arm tested was evaluated using the rigidity analyzer. The device has previously been used in measuring rigidity in subjects with Parkinson's disease [15]. Subjects once again sat in a chair with their back supported and arm in a neutral shoulder position with elbow flexed at 90 degrees and forearm supinated. The rigidity analyzer, a specialized pneumatic cuff with force sensor and gyro (Neurokinetics Inc., Alberta, Canada), was placed on the subject's forearm using the same arm tested for the kinematic tasks. Electromyography leads were placed on the subject's anterior deltoids, posterior deltoids, biceps, triceps, wrist flexors, and wrist extensors. Subjects were instructed to relax their arm and allow the investigator to move it without resistance or aid. During each trial, the investigator passively extended the subject's forearm (i.e., elbow joint) between the fully flexed and starting position three times while being paced by a metronome. This procedure was performed for two trials at slow (metronome setting 25 beats/minute), intermediate (100 beats/minute), and fast (175 beats/minute) speeds. Thus there were six cycles collected at each speed. The raw velocity and net force data (external force used to move the cuff minus force used to grip the cuff) output from the rigidity analyzer were collected. Electromyographic data were also collected to ensure that the movements were passive. When electromyographic recordings indicated significant tonic voluntary muscle activity, the trial was disregarded and not used in data analysis. Electromyographic recordings with reflex responses were included.

Dystonia and reaching performance were assessed using kinematic measures. Subjects sat in a chair with their backs supported, arms positioned with the shoulder neutral (vertical), elbow flexed to 90 degrees, and forearm in pronation (Fig 1). Four to five infrared emitting diodes were placed on the segments of the upper arm, forearm, and hand. A marker was also placed on the index finger tip. Rigid body calculations from the segment marker positions were used to determine the shoulder, elbow, wrist, and knuckle (second metacarpophalangeal) joint positions and angular rotations. The three-dimensional positions of all infrared emitting diodes were sampled at 100 Hz using an OPTOTRAK motion measurement system (Northern Digital, Waterloo, Ontario, Canada).

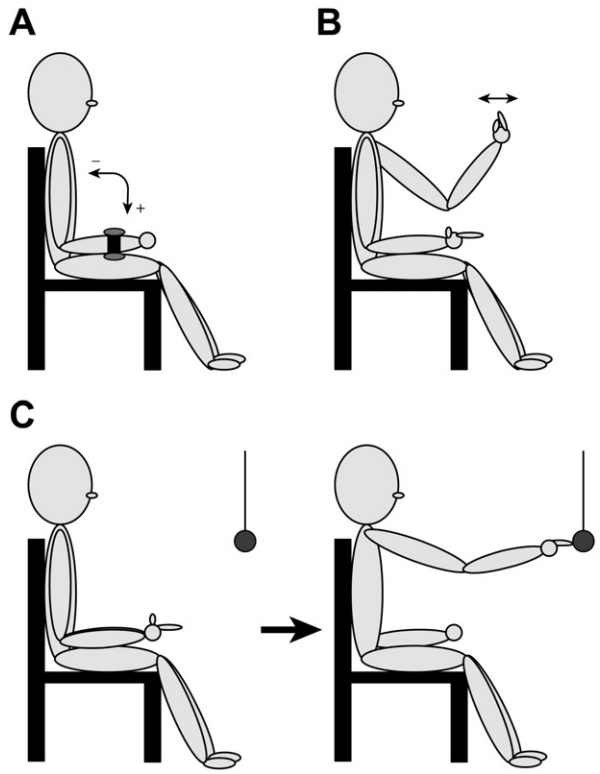


*Figure 1. Schematic of the tasks. (A) Schematic of a subject with rigidity analyzer cuff in place. The subject's arm was passively moved through extension and flexion. A positive force extended the arm, while negative force flexed the arm. (B) Schematic of tapping task. Data were recorded from the resting arm while the subject tapped the forefinger and thumb of the contralateral hand. (C) Schematic of reaching task. The subject began with the arm at rest (bottom left) and, on a "go" command, reached to touch and hold on the target with the forefinger (bottom right).*

Dystonia was quantified by measuring the amount of overflow movement in a resting arm that was caused by tapping the fingers of the contralateral arm. In the rest-tap task, subjects were instructed to tap the thumb and forefinger of their raised contralateral hand upon a "go" signal. At least three trials were collected.

For the reaching task, a 4-cm ball target was suspended from a wire in front of the subject at shoulder height and 90% of arm's available reach. Subjects were instructed to "reach and touch the ball and hold" upon a "go" signal. Five trials were collected.

## *Data Analysis*

Spasticity was defined as the force-velocity relationship at the elbow joint. For each cycle of movement, the peak elbow angular velocity and the average force over 100 ms after that time point were determined. Intuitively, one would expect that a spastic muscle would require more force to be passively stretched at higher velocities, whereas a normal muscle would not. We chose the peak angular velocity time point for measurement because this reflects the fastest speed in a cycle, and because it reduces the influence of arm inertia since angular acceleration is zero at that time. We also normalized the force measure to arm mass in order to be able to make comparisons between subjects of different size. Force values were then plotted against peak angular velocity for each elbow extension movement of all six cycles at the three speeds. Using MatLab, a best-fit line was plotted and the slope of the line, the "extension slope," was reported. Trials in which electromyographic data indicated signs of voluntary active movement by the subject were excluded from the analysis.

Kinematic measures were used to quantify dystonia and also to assess reaching performance. Before any calculations, marker position data were smoothed using a second-order Butterworth filter at 10 Hz. Angular displacement data were calculated for all joints from marker position data. Angular and linear displacement data were numerically differentiated to calculate velocity and acceleration. Dystonia was assessed in the following manner: during rest-tap trials, the maximum excursion of all joint rotations of the resting arm was calculated; this is effectively a kinematic measure of overflow. The sum of the excursions was taken as the index of dystonia. Joint rotations included in the analysis were shoulder, elbow, and wrist flexion-extension excursions (i.e., maximum flexion minus maximum extension), shoulder abduction-adduction, shoulder internal-external rotation, and wrist pronation-supination.

For reaching performance, the "start" of the movement was the time and position at which the wrist linear velocity exceeded 5% of its peak. The "end" of the first portion of the movement was the time and position at which the wrist linear velocity reached its first steady minimum. The "movement" phase of the reach was defined as the time from the start to the end of the reach. The "hold" phase of the reach was defined as the time from the end of the reach to the end of the trial. Measurements of interest included (1) peak velocity, (2) wrist path ratio (curvature), (3) end point error (overshoot or undershoot), and (4) hold distance. The wrist path ratio was the ratio of the length that the wrist actually traveled to a straight line between the start and end positions. End point error was the distance between the tip of the index finger and the target at the end of the first phase of movement. Hold distance was the distance traveled by the wrist during the hold phase and was used to represent how well the subject held a steady position.

## *Statistical Analysis*

Student *t* tests were used to compare control and cerebral palsy groups on all measures except for the clinical rating scales, which were not administered to the control subjects. Pearson correlations were used to determine relationships between the spasticity measure of extension

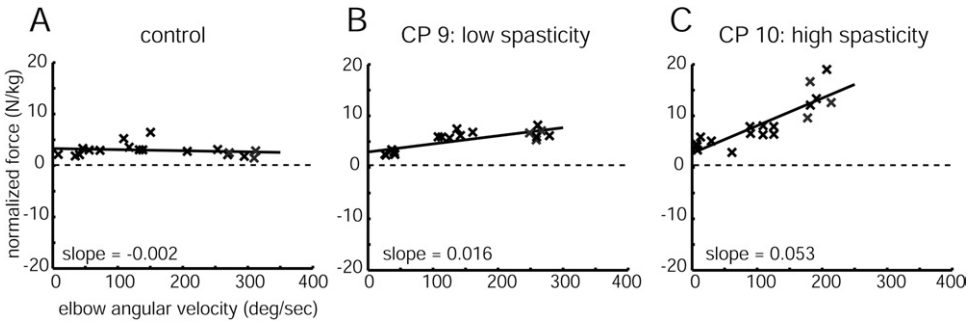


*Figure 2. Spasticity measure (normalized force vs peak velocity). The slope of the best-fit line was the measure of spasticity. (A) A representative control subject. (B) A cerebral palsy subject with low spasticity. (C). A cerebral palsy subject with high spasticity.*

slope, the dystonia measure of joint excursion during contralateral tapping, the clinical rating scales for spasticity and dystonia (Ashworth and Barry-Albright), and the measured abnormalities in the reaching tasks (peak velocity, wrist path ratio, end point error, and hold distance). A Spearman rank order correlation was used to determine if the gestation at birth or age at testing correlated with our dystonia or spasticity measures. Bonferroni corrections were performed to account for multiple tests.

## Results

### *Clinical Scales*

All cerebral palsy subjects were assessed using the Ashworth and Barry-Albright scales (Table 1). There was a significant correlation between the Ashworth and Barry-Albright scores (r = 0.68, $P = 0.01$). This finding suggests that these scales do not necessarily isolate measures of spasticity and dystonia, or that the severity of spasticity and dystonia tend to co-vary in individuals with cerebral palsy.

### *Spasticity*

To measure spasticity in each subject, the slope of the best-fit line in the force vs velocity plot, or the "extension slope," was determined. Figure 2 presents representative graphs for a control subject, a cerebral palsy subject with low spasticity (lesser extension slope), and a cerebral palsy subject with high spasticity (greater extension slope). Control subject slope values were significantly smaller (i.e., less spasticity) than cerebral palsy subjects (mean ± S.D.: 0.0001 ± 0.0028 for controls vs 0.0291 ± 0.0067 for the cerebral palsy group, $P < 0.005$). Cerebral palsy group slope values correlated with the Ashworth scale, the standard measure of spasticity (r = 0.56, $P < 0.05$) but not with the Barry-Albright scale, the standard measure of dystonia (r = 0.44, $P = 0.14$). The slopes did not correlate with gestation at birth (Spearman R 0.15, $P = 0.60$). However, they manifested a trend towards correlating with age at testing, with older children revealing lower levels of spasticity (Spearman R −0.53, $P = 0.06$).

### *Dystonia*

Dystonia was measured as the total excursion of all joints' movement during the rest-tap paradigm. Figure 3 presents a plot of joint angles changing over time for a control individual and two subjects with cerebral palsy. The control individual had no dystonia, thus no overflow is observed and the rest arm remains still. One child with cerebral palsy produced a similar pattern, whereas the other exhibited dystonia as indicated by the overflow. The total joint excursion, our measure of dystonia, indicated only a strong trend towards being greater in the cerebral palsy group than the control group owing to large vari-

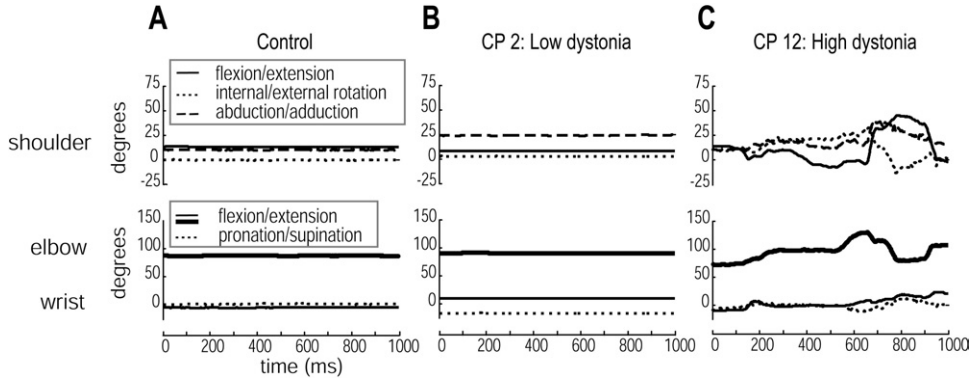


*Figure 3. Dystonia kinematics. Displacement of the shoulder, elbow, and wrist joints over time during the rest-tap paradigm. (A) A representative control subject. (B) A cerebral palsy subject with low dystonia. (C) A cerebral palsy subject with high dystonia. Note that in all plots, positive angles are when the joint is in flexion, external rotation, abduction, or supination.*

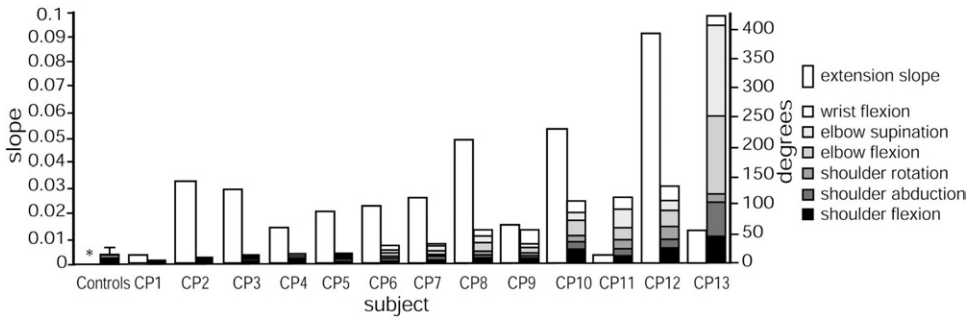


*Figure 4. Individual cerebral palsy subject data for spasticity and dystonia measures. Spasticity, as measured by the extension slope, is plotted as a single open bar on the left for each subject (left scale). Dystonia, as measured by the sum of joint excursion about the shoulder, elbow, and wrist joints, is represented by stacked bars (right scale). Control mean and standard deviations are presented for comparison. Severity of spasticity and dystonia do not correlate for individual subjects. *Mean and standard deviation of spasticity for the control group is plotted here, but is so low as to be indistinguishable from the x-axis.*

ability in the cerebral palsy group (12.9 ± 4.1 degrees for control subjects vs 68.7 ± 27.6 degrees for the cerebral palsy group, $P = 0.07$). Figure 4 indicates that some children had large joint excursions, whereas others did not. Our measure of dystonia correlated with the Barry-Albright Dystonia scale ($r = 0.75$, $P < 0.005$) but not with the Ashworth scale ($r = 0.36$, $P = 0.22$).

Figure 4 presents bar plots representing our measure of spasticity and dystonia for each cerebral palsy subject, and a mean of control values for comparison. Note that children with high dystonia did not necessarily have high spasticity (e.g., cerebral palsy 11) and vice versa (e.g., cerebral palsy 8). Thus our dystonia measurement did not correlate with our spasticity measurement ($r = 0.03$, $P = 0.924$), suggesting independent mechanisms for the phenomena that we measured. Also of interest is that gestation at birth exhibited a trend towards correlating with our dystonia measure, with children born later manifesting less dystonia (Spearman R −0.53, $P = 0.06$). Age at testing did not correlate with dystonia (Spearman R 0.017, $P = 0.96$).

### *Reaching*

Figure 5 illustrates the wrist paths in the sagittal plane for reaching movements made by a control and example subjects with cerebral palsy with high dystonia, high spasticity, or both. Note that the subjects with dystonia and dystonia plus spasticity reached with a more curved path than the control and subject with spasticity. Overall, the peak velocity was higher for control children (673 ± 103 mm/s vs 457 ± 41, $P = 0.04$) and controls moved in a straighter path (1.11 ± 0.05 vs 1.35 ± 0.09, $P = 0.04$). Both groups stopped short of the target, though endpoint error trended towards a smaller magnitude for controls (−38.0 ± 11.4 vs –58.7 ± 10.0, $P = 0.07$). The hold distance was not statistically different between groups (controls 182 ± 57 vs cerebral palsy 228 ± 42, $P = 0.53$).

In the cerebral palsy group, some of these reaching features exhibited a relationship to our spasticity or dystonia measures (Table 2). We did not have a priori hypotheses regarding these correlations, so the statistics reported are corrected for multiple comparisons and only two correlations reached significance. The spasticity measure correlated significantly with peak velocity ($r = 0.71$, $P = 0.006$); children with greater spasticity hit higher peak velocities. The dystonia measure correlated significantly with the curvature of the path ($r = 0.70$, $P = 0.008$); children with higher dystonia made more curved reach paths.

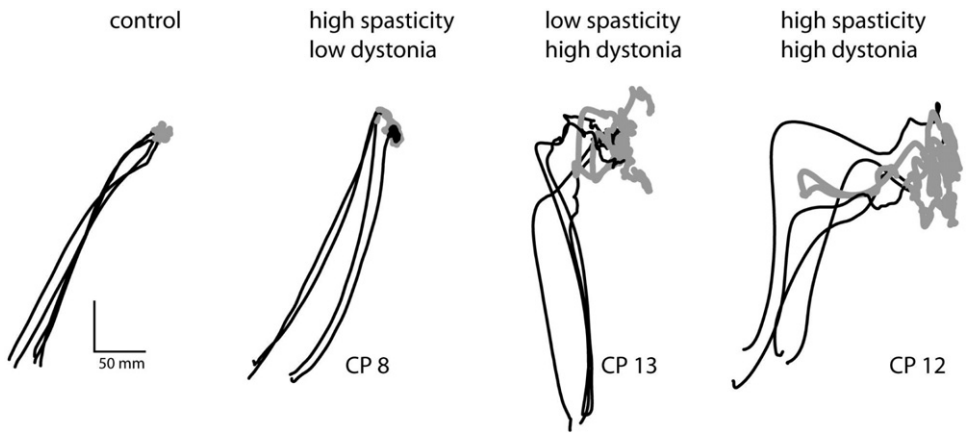


*Figure 5. Representative paths of sagittal plane wrist motion during the movement (thin line) and hold (thick gray line) phases of the reaching task. A control and subjects with different mixtures of spasticity and dystonia are illustrated.*

**Table 2. Reaching feature correlations**

| Measurement | Correlation With Extension Slope (Spasticity) | | Correlation With Rest-tap Total Movement (Dystonia) | |
|---|---|---|---|---|
| | R Coefficient | *P* Value | R Coefficient | *P* Value |
| Peak velocity | **0.71** | **0.006** | 0.35 | 0.238 |
| Reach path ratio (curvature) | 0.24 | 0.430 | **0.70** | **0.008** |
| Hold distance | 0.59 | 0.032 | 0.39 | 0.193 |
| Endpoint error | −0.01 | 0.975 | −0.65 | 0.016 |

Note: Applying the Bonferroni correction, a *P* value <0.0125 is used to indicate statistical significance. Boldfaced data indicate values are statistically significant after applying the Bonferroni correction for $P < 0.0125$.

## Discussion

This study has demonstrated that spasticity and dystonia can be measured in children with cerebral palsy. Our measures correlate with their respective clinical rating scales (Modified Ashworth and Barry-Albright Dystonia scales), suggesting that we are capturing clinically significant elements of these motor signs. Importantly, our measures are not correlated with each other, suggesting some independence of the findings being measured. These tests do not require the child to perform difficult tasks: allowing the clinician to passively move her arms to measure spasticity and sitting at rest while tapping her fingers to measure dystonia. This feature enables children of many ages to be tested.

The slope of the force-velocity relationship at the elbow was used here as a measurement of spasticity. These slopes were close to zero in control subjects (who should not have any spasticity) and higher in cerebral palsy subjects. This measure also yielded a moderate but significant correlation with the modified Ashworth scale. There are many other studies that have used well-established methods to measure spasticity [8,16,17]. For example, use of isokinetic devices to stretch a group of muscles ensures that the imposed movements are of constant and consistent speed. Our measure was not intended as a substitute for these methods, but as an alternative that has the advantage of being easily portable and quickly used in a clinical setting. Our measure also parallels the type of examination that clinicians use to make subjective ratings.

Few studies have quantified any aspect of dystonia [12,13,18]. In the present study, a straightforward kinematic measure of overflow was used as the indicator of dystonia, which was the sum of arm joint motion during attempted rest while tapping the fingers of the contralateral arm. We chose to use a provoking stimulus (finger tapping) because clinicians find that it is often required to elicit dystonia. Using our measure, dystonia was close to zero in control subjects and varied in the cerebral palsy subjects. It correlated well with the Barry-Albright Dystonia scale in the cerebral palsy subjects. In most subjects with dystonia, a slowly changing posture was observed rather than a fixed posture. We judged this to be consistent with clinical definitions of dystonia [5], but acknowledge that faster movements could represent other types of involuntary movements such as chorea. That being said, none of the subjects manifested clinical signs of chorea during examination or testing. We also acknowledge that our kinematic measure of dystonia would not be sensitive enough to detect muscle activations that are subthreshold for eliciting a movement or changing a posture. We chose not to use changes in muscle activation magnitude as the dystonia measure because of the inherent variability in electromyography and the difficulties in quantifying magnitude. We felt that our measure was more consistent with the standard clinical examination and rating scales for dystonia.

Our spasticity and dystonia measures were correlated with specific features of reaching in the children with cerebral palsy that were studied. This finding is perhaps surprising, particularly because only two of the many possible deficits that could affect reaching performance were measured. Other deficits such as weakness, poor selective muscle activation, impaired sensation, ataxia, and apraxia were not considered. This study found that children with more severe dystonia tended to reach in a more curved path than those with low dystonia, and that people with higher spasticity tended to reach faster than those with low spasticity. If these impairments are truly giving rise to these qualities of reaching, then one might expect that treatments to alter spasticity or dystonia might also change related aspects of reaching performance.

Though the majority of the subjects (12 of 13) had a diagnosis of spastic cerebral palsy, many had elements of both spasticity and dystonia comprising their movement disorders. These mixed movement disorders often go unrecognized, and therefore untreated. Brunstrom et al. [18] reported on a 16-year-old female with spastic quadriplegic cerebral palsy who manifested marked motor function benefits from levodopa, a drug most often used for extrapyramidal dysfunction. This case report emphasized the importance of recognizing and treating all aspects of motor dysfunction in cerebral palsy. If a child with previously diagnosed "spastic" cerebral palsy has elements of dystonia, the child may benefit from drugs

such as levodopa. Similarly, if a child previously diagnosed with "extrapyramidal" cerebral palsy manifests some spasticity, he or she may benefit from medications targeting this spasticity. The measures reported in this study indicate that, while most of the cerebral palsy subjects had a dominant movement disorder, many of them were found to have aspects of both spastic and extrapyramidal cerebral palsy. These children, who had previously been diagnosed by only one movement dysfunction, can now be recognized as having mixed hypertonia and treated appropriately.

Another important advantage to using quantitative data for assessing these motor signs is sensitivity. Clinical scales like the Barry-Albright or Ashworth scales can provide a categorical measurement of dysfunction, though the spasticity and dystonia measures reported here are more sensitive measures of the movement disorders. We expect that these measures could be monitored more closely than a clinical score, as they are more sensitive to small changes. Moreover, these quantitative measures can be used in research studies to identify appropriate study populations and to determine the efficacy of medical treatments and therapeutic techniques. Future directions of this research include reliability trials, treatment research, and clinical use. We expect to use these quantitative measures to assess the efficacy of medications and therapies.

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