

Biomechanic Characteristics of Patients With Spastic and Dystonic Hypertonia in Cerebral Palsy

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ABSTRACT. Lebedowska MK, Gaebler-Spira D, Burns RS, Fisk JR. Biomechanic characteristics of patients with spastic and dystonic hypertonia in cerebral palsy. *Arch Phys Med Rehabil* 2004;85:875-80.

Objective: To determine what biomechanic characteristics of knee joint motion and walking show potential to quantitatively differentiate spasticity and dystonia in cerebral palsy (CP).

Design: Descriptive measurement study.

Setting: University hospital.

Participants: Seventeen pediatric and adult patients with CP.

Interventions: Not applicable.

Main Outcome Measures: We measured the resistance of the knee joint at different velocities and positions, maximum muscle activation during external motion, amplitude of knee tendon reflexes, maximum isometric flexion and extension torques, velocity of walking, and knee kinematics during the gait cycle. Patients were classified into 2 groups (dystonia or spasticity) if at least 2 of 3 physicians agreed that a prominent component of dystonia was present.

Results: Patients with dystonia had a greater degree of cocontraction and an increased resistance to external motion at slow velocities. The tendon reflexes were almost normal in patients with dystonia, whereas they were increased in patients with spasticity. Muscle strength was more impaired in patients with dystonia, probably as a result of greater muscle cocontraction. They also walked slower, with smaller knee ranges of motion, during the stance phase of walking.

Conclusions: The measurement of resistance and of muscle activation during passive motion and tendon reflexes shows potential to differentiate dystonia from spasticity in CP patients with a mixed form of hypertonia. More studies are needed to confirm these results.

Key Words: Cerebral palsy; Dystonia; Muscle hypertonia; Muscle spasticity; Rehabilitation.

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CEREBRAL PALSY (CP) IS A chronic motor disorder in childhood resulting from abnormal development of, or early injury to, the motor system of the brain; it involves various motor nuclei (motor cortex, basal ganglia, thalamus, cerebellum) and/or their connections (cerebral white matter, brainstem, spinal cord) within the central nervous system.¹ Impairment of voluntary movement related to hypertonia is common among patients with CP. In most children with CP, the hypertonia is of a mixed type, with components of both spasticity and dystonia; this is referred to as mixed hypertonia.¹ From a clinical standpoint, it is important to distinguish these 2 forms of hypertonia because the treatment of one differs from that of the other.¹ It is also important to develop objective measurement tools to assess spasticity and dystonia in the context of CP to evaluate the effectiveness of treatment interventions.

Spasticity is defined as hypertonia with a velocity-dependent increase in resistance and/or a rapid rise in resistance about a velocity or joint position threshold.¹ The most important feature of spasticity is the hyperexcitability of spinal motoneurons of complex origin that has the effect of increasing the activation of antagonist muscles during imposed limb movements because of abnormal stretch reflexes. The stretch reflexes in spasticity are under supraspinal as well as spinal influences, primarily from the motor cortex. The possible underlying mechanisms of spasticity were discussed elsewhere.² An estimated 75% of patients with CP have spasticity.³

Dystonia is defined as a syndrome of sustained muscle contraction that frequently causes twisting and repetitive movements or abnormal postures; it is often increased during voluntary movement.⁴ Dystonic movements are characterized by the cocontraction of antagonist muscles and overflow of activity into extraneous muscles resulting in an abnormal pattern of muscle activation during voluntary movement or the maintenance of posture.⁵ Evidence suggests that dystonia originates in the basal ganglia and involves deficient inhibition at the level of the motor cortex and probably also at the levels of the brainstem and spinal cord.⁵ If dystonia is present at rest, it may produce an abnormal posture and contribute to joint stiffness.¹ Dystonia might also produce hypertonia if muscle contraction begins before the start of externally imposed passive joint movement.¹ Dystonia associated with CP is classified as secondary dystonia, along with other disorders involving an insult to the brain.⁴ The anatomic localization of the lesions leading to dystonia in CP has not yet been identified.

Spasticity and dystonia may occur in the same limb in CP; distinguishing the 2 clinically is difficult and requires assessment of the velocity-dependent, action-induced, and posture-responsive components of muscle tone. Adding to the difficulty, both spasticity and dystonia can vary in presence and severity over time and can be influenced by one's emotional state, level of consciousness, and external stimuli (eg, temperature, sound). The quantification of hypertonia and the differentiation of spasticity and dystonia in children with CP are important to evaluate the effectiveness and to optimize the treatment outcome. A quantitative and objective measure of muscle tone is needed to achieve these goals.

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The basic physical characteristic of muscle tone is the resistance of a joint to external motion. Clinically, it can be evaluated with the 4- or 5-point Ashworth Scale.^{6,7} Another method is with the Tardieu scale,⁸ in which a limb is moved at different velocities and the position and velocity of the "catch" are used as descriptors of abnormal tone. Clinical scales depend on the examiner's sense of force, especially in the case of children when the same angular displacement requires forces scaled by the examiner according to third power of patient's height, and therefore the scales lack objectivity. In general, the scales are based on ordinal scores and do not differentiate the basic types of hypertonia. The Fahn-Marsden scale⁹ and the pediatric Barry-Albright Dystonia Scale¹⁰ are ordinal clinical rating scales that quantify dystonia.

The application of complex torque devices to quantify the characteristic points of the torque-angle ($Tq-\alpha$) function, including the resistance of a joint to external motion, is limited in the clinical setting. In addition, the resulting biomechanical measures of resistance have a limited ability to discriminate patients from able-bodied subjects.¹¹⁻¹⁴ We showed¹⁴ that a portable handheld force transducer coupled with an electrogoniometer and a surface electromyograph can be used to quantify the biomechanical and bioelectric characteristics of resistance of the antagonist muscles at the knee joint in patients with CP. We found several different patterns of antagonist muscle activation associated with hypertonia in children with CP. The most common patterns were velocity-dependent activation of antagonist muscles, in which the resistance and the maximum electromyographic activation of antagonistic muscles increased with the velocity of passive motion (type I), and position-dependent activation, in which the resistance and the maximum electromyographic activation of antagonistic muscles increased rapidly when a certain position (threshold) was reached (type II). These 2 patterns of abnormal muscle activation were attributed to hypertonia of different origins. Less frequently, activation was evoked by passive motion but was neither velocity nor position dependent or showed inconsistent characteristics of both (type III).¹⁴ Considering that dystonia can be triggered by passive and/or active motion, it might be involved in type III hypertonia.

Our purpose in this study was to identify biomechanical measures at the level of the knee joint and of walking that have the potential to differentiate spasticity and dystonia. Patients were classified into 2 groups: those with and those without clinical signs of dystonia. Ambulatory patients were selected to allow study of the effects of hypertonia on walking. We compared various biomechanical measures of muscle tone and movement in the 2 groups: the resistance and activation of muscles acting at the knee during passive knee flexion and extension, the patellar knee tendon jerks, the velocity of walking, knee position at initial stance, and the range of motion (ROM) during the stance and swing phases of walking.

METHODS

Classification of Patients With CP

Three physicians (an orthopedic surgeon who evaluated the patients clinically, a neurologist, a pediatric physiatrist) independently classified the patients, based on their observations of a video of the patients walking in the laboratory space. No formal rating scale was used. They assessed changes in limb posture during standing and walking. Each clinician rated the subjects while blinded to the criteria used by the other physicians.

Measurement of Resistance at the Knee Joint

A handheld force transducer^a coupled with an electrogoniometer^{b,14} was used to passively flex or extend the knee. Torque was measured as a function of the knee angle ($Tq-\alpha$) over the entire ROM. The motion of the knee, pull force, and electromyographic activity of muscles acting at the knee joint were collected at 3 different velocities: slow (.2 radian/s), moderate (2 radian/s), and fast (5 radian/s). Analog signals from different transducers (motion, force) and the electromyographic signals were transferred to a personal computer and collected with Axoscope system software^c by using a 333-Hz sampling rate. Further analysis was performed with custom-written software based on Matlab.^d The $Tq-\alpha$ function was determined for each velocity of motion, and the slope of the $Tq-\alpha$ function at the initial position (resistance; $RESIST=dTq/d\alpha$) and the maximum activation of rectus femoris (REC) and hamstring (HAM) muscles during each pull were calculated. A simple analysis of the surface electromyographic signal was done to determine the highest level of muscle activation (maximal potential change in microvolts) during each pull. The resistance was normalized by the fifth power of body height.¹³ The direction of motion was designated by the subscripts *flex* for flexion and *ext* for extension. To determine the resistance and the maximum activation of the antagonistic muscles during slow motion (equivalent to the Ashworth test), the data at velocities lower than 1.5 radian/s were pooled. To determine the degree of velocity (v) dependency of the resistance, the best fit of the data (d_i) to the equation $d_i = a + bv^2$ was calculated. The relationship between the velocity (v) of passive motion and the resistance, and maximum activation of the rectus femoris and hamstring muscles was defined as coefficient b of the equation and these variables were designated as $RESIST_{flex,ext}$ INC (increment), $REC_{flex,ext}$ INC, and $HAM_{flex,ext}$ INC. The initial (neutral) knee position (α_{in}) was also calculated. Full knee extension was considered as 0 angular, which increased with knee flexion.

The characteristics of the $Tq-\alpha$ function were measured, including a position (CATCH) and torque (TQ_CATCH) when the torque sign changed from positive to negative and a position (R2) and torque (TQ_R2) when the torque sign changed from negative to positive. Torques were normalized by the fourth power of body height.¹⁵

Measurement of Maximum Voluntary Isometric Knee Flexion and Extension Torque

To measure the maximal voluntary extension and flexion torques of the muscles acting about the knee joint, patients sat on an examination table with their lower legs hanging over its edge and the knee flexed at 90°. A strain-gauge force transducer^a was attached at the level of the ankle joint in a neutral position. Patients were familiarized with the apparatus and were instructed on how to exert maximal voluntary flexion and extension torques before measurements were taken. They were instructed to exert the maximum force and to maintain it for 3 to 4 seconds. The maximal isometric torques in extension (MVC_{ext}) and in flexion (MVC_{flex}) were calculated as the product of the force and the perpendicular distance between the line of action of the force and the axis of rotation at the knee. The data were normalized by dividing torque by the fourth power of body height. The MVC parameters obtained in women were normalized to male values by multiplying by 1.07, and the results from female and male patients were pooled. The electromyographic signals were collected as previously described. To obtain the cocontraction ratios during voluntary flexion (COFLEX) and extension (COEXT), the

Table 1: The General Characteristics of the Limbs of Patients With Dystonia and Spasticity

	Spasticity				Dystonia				P*
	N	Median	Min	Max	N	Median	Min	Max	
α_0 (radian)	22	1.32	1.06	1.59	22	1.22	0.97	1.31	<.01
MVC _{flex} (N/m ³)	22	1.97	0.26	3.56	22	0.60	0.00	2.11	<.001
COFLEX (%)	22	25	5	100	22	55	20	100	<.002
MVC _{ext} (N/m ³)	22	6.52	2.6	13	22	3.9	15	5.12	<.001
COEXT (%)	22	20	10	60	22	52.50	20	80	<.001

Abbreviations: Max, maximum; Min, minimum; N, number of limbs.
*Probability from the Kruskal-Wallis test of medians.

mean electromyographic activity of the antagonist muscle was expressed as a percentage of the agonist's activity

Measurement of Patellar Tendon Reflexes

The measurements of patellar tendon reflexes were made with the patients sitting with their lower legs hanging over the edge of an examination table as previously described.² A strain-gauge force transducer^a was attached at the ankle joint of the limb to be studied. A few minutes of biofeedback training was used to teach the patients how to relax the limb. A standard reflex hammer was used to tap on the patellar tendon of the quadriceps muscle. Ten to 15 reflexes were evoked and recorded for further analysis. The mean biomechanic reflex response (R_{rel}) was normalized for body height and expressed as a percentage of voluntary extension torque in able-bodied subjects.¹⁵

Gait Evaluation

Standard gait analysis with the 5-camera system^e and 2 force platforms^f was used. Retroreflective markers were placed on selected body points to define body segments. Markers were attached to the skin surface at positions corresponding to the sacrum, anterior superior iliac spines, laterofemoral epicondyles, lateral malleoli, and third metatarsophalangeal joints. Lateral thigh and tibial markers were also used. Patients walked on the walkway several times at self-selected speeds. Electromyographic activity (tibialis anterior, gastrocnemius, rectus femoris, hamstrings, gluteus maximus, gluteus medius) was collected with standard surface electrodes.^g Kinetic and kinematic gait parameters were calculated with the Workstation.^e The following kinematic parameters in the sagittal plane were analyzed: the knee angular position at the beginning of the stance phase (FLEX_{in}), the minimum knee angle during stance phase (STANCE_{min}), and the maximum knee angle during the swing phase (SWING_{max}). Knee ROM during the stance (ROM_{stance}) and swing phases (ROM_{swing}) of walking were calculated, as well as the walking velocity.

Statistical Analysis

Patients were classified into either the spasticity group or the dystonia group if 2 of 3 physicians classified them into the same group. The distributions of data in the limbs of patients in both groups were checked with the Kolmogorov-Smirnov 1-sample statistic with Lilliefors test of normality and the Shapiro-Wilk W test. Most of the data were not normally distributed. The medians of the parameters obtained in the limbs of all patients were compared with the Kruskal-Wallis analysis of variance test. Statistical analysis was performed with Statistica,^h with the level of significance set at P less than .05. The reliability of clinical raters was computed with the Cronbach α for the results of the 3 physicians on all patients using SAS software.ⁱ

Participants

Seventeen patients (13 children, 4 adults) were studied, and a total of 28 limbs were evaluated. Each participant was informed regarding the experimental procedures, and they gave their written consent before participating in the testing session. All experiments were approved by the Springfield Committee for Research Involving Human Subjects.

RESULTS

In 3 of 17 patients, at least 2 of 3 the physicians diagnosed dystonia as a prominent component, and in the 14 remaining patients they diagnosed spasticity without dystonia. The reliability coefficient between the 3 physicians was .80 for the right side of the body and .81 for the left side.

The results of the comparison of different variables in the limbs of patients with spasticity and patients with dystonia are presented in tables 1, 2, 3, and 4. We found that the neutral position of the knee while patients were sitting at rest (under the force of gravity) was less extended ($P < .05$) in the limbs of patients with spasticity (range, 1.06–1.59 radian) than in the limbs of patients with dystonia (range, .97–1.31 radian).

The maximal isometric flexion torques were lower ($P < .001$) in the limbs of patients with dystonia (range, 0–2.1N/m³) than in the limbs of patients with spasticity (range, 0.26–3.56N/m³). The cocontraction of antagonist muscles during isometric flexion was greater ($P < .05$) in limbs with dystonia (range, 20%–100%) than with spasticity (range, 5%–100%). The maximal isometric extension torques were also lower ($P < .001$) in the limbs of patients with dystonia (range, 1.5–5.12N/m³) than in those with spasticity (range, 2.6–13N/m³). The cocontraction of the antagonist muscles during isometric extension was larger ($P < .05$) in limbs of patients with dystonia (range, 20%–80%) than in patients with spasticity (range, 10%–60%).

Knee tendon reflexes (table 2) were larger ($P < .05$) in the limbs of patients with spasticity (range, 2%–20% MVC) than in those with dystonia (range, 2%–6.6% MVC). During passive knee extension at slow velocities ($v < 1.5$ radian/s), the activity of the hamstring muscles was higher ($P < .001$) in the limbs of patients with dystonia (range, 6.4–317 μ V) than in patients with spasticity (range, 1–317 μ V), and the activity of the rectus femoris muscle was much higher ($P < .05$) in the limbs with dystonia (range, 9.4–285 μ V) than with spasticity (range, 2.3–126 μ V).

During slow passive flexion ($v < 1.5$ radian/s), the resistance at the beginning of motion was higher ($P < .05$) in limbs of patients with dystonia (range, 1.15–0.34N·m⁻⁴·radian⁻¹) than in those with spasticity (range, 0.42–15.5N·m⁻⁴·radian⁻¹). The activation of the rectus femoris muscle was higher ($P < .05$) in the limbs of patients with dystonia (range, 5.4–317 μ V) than in patients with spasticity (range, 2–130 μ V), and the activation of the hamstring muscles was also higher ($P < .001$) in the limbs

Table 2: The Reflex and Resistance Characteristics in the Limbs of Patients With Dystonia and Spasticity

	Spasticity				Dystonia				P*
	N	Median	Min	Max	N	Median	Min	Max	
R _{rel} (% MVC _{ext})	22	7.20	2	20	6	3.70	2	6.60	<.002
RESIST _{ext} (N·m ⁻⁴ ·radian ⁻¹)									
v<1.5 radian/s	22	1.05	0.44	3	16	1.01	0.5	13.7	.52
HAM _{ext} (μV) v<1.5 radian/s	22	13.2	1.24	317	6	132.5	6.4	317	<.001
REC _{ext} (μV) v<1.5 radian/s	22	9	2.33	126	16	27.7	9.5	284	<.002
RESIST _{ext} INC (N·s ² ·m ⁻⁴ ·radian ⁻³)	22	0.32	0.12	7.67	6	0.56	0.07	3.37	.21
HAM _{ext} INC (μV·s ² ·radian ⁻²)	22	1.66	-0.06	9.6	6	-3	-29	9.4	.08
REC _{ext} INC (μV·s ² ·radian ⁻²)	22	0.4	-0.46	6.07	6	-0.95	-31	0.73	<.005
RESIST _{flex} (N·m ⁻⁴ ·radian ⁻¹)									
v<1.5 radian/s	21	0.98	0.42	15.5	20	1.45	0.84	32.43	<.01
HAM _{flex} (μV) v<1.5 radian/s	21	11	2.63	57.2	20	61	9.8	317	<.001
REC _{flex} (μV) v<1.5 radian/s	21	13.1	2	129.9	6	28.6	5.4	317	<.01
RESIST _{flex} INC (N·s ² ·m ⁻⁴ ·radian ⁻³)	21	0.8	0.28	6	6	1.15	0.34	3.4	.48
HAM _{flex} INC (μV·s ² ·radian ⁻²)	21	0.05	0.3	6	6	-2.18	-32.42	36.99	.92
REC _{flex} INC (μV·s ² ·radian ⁻²)	21	0.9	-1.78	3.54	6	1.93	-1.2	6.7	.41

*Probability from the Kruskal-Wallis test of medians.

of patients with dystonia (range, 9.8–317 μV) than in the limbs of patients with spasticity (range, 2.6–57 μV).

Analysis of the characteristic points of the Tq-α function (table 3) found that, during passive knee extension, the torque of the catch was larger ($P<.05$) in the limbs of patients with spasticity (range, 0.7–4.6 N/m³) than in the limbs of patients with dystonia (range, 0.99–2.3 N/m³). During passive knee flexion, the position of the first catch was smaller ($P<.001$) in the limbs of patients with dystonia range, (.92–1.75 radian) than in patients with spasticity (range, 1–2 radian), and the position of the second catch was also smaller ($P<.001$) (range, .92–2 radian vs range, 1.08–2.39 radian).

When we compared gait parameters, we found that the minimum angle of the knee position in the stance phase of walking was smaller ($P<.05$) in patients with spasticity (range, -10° to 40°) than in patients with dystonia (range, 0°–35°) (table 4). Knee ROM in the stance phase of walking was smaller ($P<.001$) in patients with dystonia (range, 0°–20°) than in patients with spasticity (range, 0°–45°). The velocity of self-paced walking was lower ($P<.001$) in patients with dystonia (range, .13–.50 m/s) than with spasticity (range, 0.38–1.23 m/s).

Other differences were not statistically significant ($P<.05$).

DISCUSSION

We found that the neutral position of the knee (table 2), when patients were sitting and in a relaxed state, was more extended in the limbs of patients with dystonia (range, 55°–75°) than in limbs of patients with spasticity (range, 60°–91°), which suggests an increased resistance of the knee muscles affecting posture. No electromyographic activation was shown before any motion started, which suggests that the increased tone of the knee muscles at relaxation was below the threshold of detection. This finding is consistent with the view that, in dystonia, the limb at rest tends to move toward the extremes of joint angles.¹

The maximum flexion and extension torques were reduced to a greater extent in the limbs of patients with dystonia than in patients with spasticity. When the median values were compared with normal values,¹⁵ the limbs of patients with spasticity exhibited 46% of normal strength in extension and 34% of normal strength in flexion, whereas those of patients with dystonia exhibited 27.6% of normal strength in extension and 10.3% of normal strength flexion. The results are consistent with previous reports^{2,16–22} that patients with spasticity are substantially weaker than able-bodied subjects on these tasks.

Table 3: The Tardieu Scale Characteristics in the Limbs of Patients With Dystonia and Spasticity

	Spasticity				Dystonia				P*
	N	Median	Min	Max	N	Median	Min	Max	
CATCH_EXT (radian)	82	1.00	0.67	1.41	10	1.00	0.75	1.08	.26
TQ_CATCH_EXT (N/m ³)	82	-1.98	-4.60	-0.70	10	-1.40	-2.30	-0.99	.02
R2_EXT (radian)	82	0.56	0.19	1.05	10	0.47	0.27	0.70	.26
TQ_R2_EXT (N/m ³)	82	-1.39	-3.24	-0.32	10	-1.07	-2.23	-0.82	.25
CATCH_FLEX (radian)	120	1.57	1.00	2.00	30	1.42	0.92	1.75	<.001
TQ_CATCH_FLEX (N/m ³)	119	1.11	0.38	3.71	30	1.17	0.22	2.19	.70
R2_FLEX (radian)	119	1.93	1.08	2.39	30	1.65	0.92	2.00	<.001
TQ_R2_FLEX (N/m ³)	119	0.83	0.32	3.33	30	1.02	0.04	2.46	.08

Abbreviation: N, number of limbs.

*Probability from the Kruskal-Wallis test of medians.

Table 4: The Gait Characteristics in the Limbs of Patients With Dystonia and Spasticity

	Spasticity				Dystonia				P*
	N	Median	Min	Max	N	Median	Min	Max	
FLEX _{in} (deg)	22	23	0	40	6	35	5	55	.120
STANCE _{min} (deg)	22	10	-10	40	6	25	0	35	.020
SWING _{max} (deg)	22	48	5	70	6	46.5	5	75	.750
ROM _{stance} (deg)	22	20	0	45	6	9	0	20	<.001
ROM _{swing} (deg)	22	7	-3	30	6	8.50	0	23	.500
Velocity (m/s)	23	0.95	0.38	1.23	6	0.37	0.13	0.50	<.001

Abbreviation: N, number of limbs.

*Probability from the Kruskal-Wallis test of medians.

The presence of dystonia appears to impair strength to an even greater degree.

The fact that cocontraction during isometric tasks was more severe in the limbs of patients with dystonia than with spasticity (table 1) suggests that the decrease in strength in the limbs with dystonia is related to supraspinal cocontraction, which is characteristic of dystonia, and that this might be more of a contributing factor than cocontraction of spastic origin. This finding concurs with the reported simultaneous cocontraction of agonists and antagonists in voluntary movements in patients with dystonia.¹

The values found for the knee tendon reflexes at relaxation (table 2) in the limbs of patients with spasticity (range, 2%–20% MVC) were considerably increased and were similar to those previously reported in patients with spasticity of different origins (range, 1%–30% MVC), whereas the values in the limbs of patients with dystonia (range, 2%–6.6% MVC) were slightly elevated compared with the values reported in able-bodied subjects (range, .35%–3.4% MVC).¹⁵ The finding that the knee tendon reflexes were only slightly increased compared with normal values confirms previous studies²³ showing that monosynaptic reflexes are within the normal range in patients with dystonia. Figure 1 shows that the velocity-dependent activation of the rectus femoris muscle (REC_{flex} INC) is proportional to the monosynaptic reflexes of the rectus femoris

muscle in relaxation in patients with spasticity but not in patients with dystonia.

Passive motion at slow velocities ($v < 1.5$ radian/s) is equivalent to the velocities used during the clinical application of the Ashworth Scale. We found that the resistance at the knee joint during slow movements (table 2) was higher in the limbs of patients with dystonia than in the limbs of patients with spasticity and that it doubled in magnitude (median values) during passive flexion. The fact that, during both passive flexion and extension of the knee, activation of the antagonist and agonist muscles was greater in the limbs of patients with dystonia suggests that slow limb movements are more impaired in those patients than in patients with spasticity. When we considered the velocity dependency of resistance and of muscle activation (table 2), we found that the dependency of resistance and antagonist muscle activation were greater in the limbs of patients with spasticity than with dystonia; however, the differences were not statistically significant, probably because of the variable patterns of muscle activation that occur in spasticity. Our findings are consistent with the clinical findings that, in dystonia, the increased resistance to externally imposed joint movement is present at very low speeds of movement and does not exhibit a speed or angle threshold and/or velocity dependence.¹

The finding that the catch position in knee flexion occurs earlier in limbs of patients with dystonia suggests that the threshold for the onset of the stretch reflexes²⁴⁻²⁷ is not the major factor that determines catch. Additional studies are required for an understanding of the impact of cocontraction on joint dynamics at different velocities of stretch.

The slower (2.5-fold) self-paced walking of patients with dystonia (median values were compared; table 4) appeared to be related to the greater reduction in strength in their limbs (fig 2). The smaller (25°) knee ROM in the stance phase of walking in patients with dystonia was in part accounted for by the knee hyperextension (genum recurvatum) in some limbs of patients with spasticity.

The finding that spasticity was present in 82.4% of the limbs of patients with CP and that dystonia was present in 17.6% of the limbs is similar to the reported frequency of these signs in patients with CP.³ This suggests that to obtain data on dystonia in CP, a study of at least 100 children will be required. The different patterns of abnormal tone in patients with CP¹⁴ would also have to be considered in determining the sample size. The results of this study are limited by the relatively small number of patients with dystonia and individual limbs studied.

The agreement regarding the clinical diagnosis (spasticity vs a prominent component of dystonia) among the 3 physicians was relatively high (0.8). The physicians, who were experienced clinicians, were asked to classify the patients based only

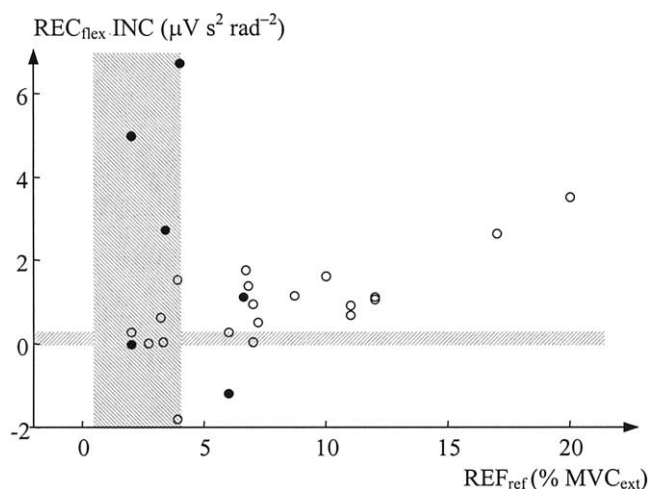


Fig 1. The strength of velocity-dependent activation of the rectus femoris muscle (REC_{flex} INC) depends on the amplitude of tendon jerks in patients with spasticity (○), but not in patients with dystonia (●). The ranges of the normal values marked with the shaded area.

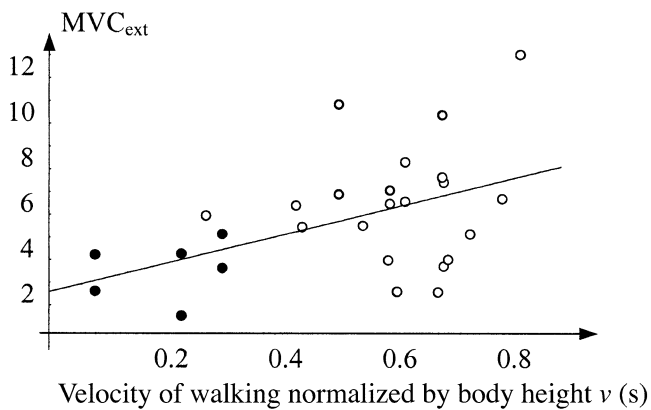


Fig 2. Patients with dystonia (●) walked slower (v) than patients with spasticity (○), as a result of decreased strength (MVC_{ext}). NOTE. $MVC_{ext}=2.58+6.16v$; $r=.54$.

on the presence or absence of a prominent component of dystonia. The classification of a patient as spastic or dystonic, based on the agreement of at least 2 of 3 physicians, represented a practical approach but also had its limitations.

CONCLUSIONS

Despite the small number of limbs studied in patients with signs of dystonia and the fact that we limited the study to a patient population of ambulatory patients, it appears that certain measures of resistance show potential to differentiate hypertonia of spastic and dystonic origin. The independence of the muscle activation (electromyographic) on the velocity of motion and amount of stretch and monosynaptic reflexes within the normal range appear to differentiate patients with a prominent component of dystonia from patients with spasticity. To assess the discriminating value of these measures in CP will require a larger study of selected patients with pure and mixed forms of hypertonia.

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