

ABSTRACT: The motions of lower-limb extension, adduction, and internal rotation are frequently coupled in persons with cerebral palsy (CP) and are commonly referred to as an extension synergy. However, the underlying joint moments that give rise to these coupled motions are not well understood. We hypothesized that maximal voluntary exertions in a direction of one component of a synergy (e.g., hip extension) would result in the concurrent presence of other components of the synergy in subjects with CP but not in control subjects. To test this hypothesis, we measured three-dimensional moments about the hip and knee as nine subjects with spastic CP and six control subjects performed maximal isometric exertions of the hip and knee flexors and extensors. During maximal hip extension exertions, control subjects simultaneously generated a knee flexion moment, whereas CP subjects generated a knee extension moment ($P < 0.05$) and a larger hip internal rotation moment than did controls ($P < 0.05$). During maximal knee extension exertions, control subjects generated a hip flexion moment, whereas CP subjects generated a hip extension moment ($P < 0.05$). The patterns of joint moments generated by CP subjects are consistent with an extension synergy and may underlie the coupled motion patterns of the lower extremity in such persons.

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ABNORMAL COUPLING OF KNEE AND HIP MOMENTS DURING MAXIMAL EXERTIONS IN PERSONS WITH CEREBRAL PALSY

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The coordination of complex, purposeful movements requires a sufficient amount of independent control of joint trajectories. Walking, for example, involves asynchronous transitions between extension and flexion of the hip and knee. Infants initially lack the capacity to perform complex lower-limb tasks and tend to move their limbs in flexion or extension patterns^{20,25}; however, they acquire the ability to coordinate precise, fractionated movements as the nervous system matures.²¹

The motions of joints frequently remain coupled in flexion and extension patterns in persons with cerebral palsy (CP). Similarly, coupled flexion and extension motion patterns of the hip, knee, and ankle have been observed in adults with hemiplegia following stroke; historically, these coupled motions have been called “synergies.” Several investigators^{5,22,24,26,29} have described a lower-limb “extension synergy” consisting of combined extension, adduction, and internal rotation of the hip, extension of the knee, and plantarflexion and inversion of the ankle. A “flexion synergy” consists of the opposite motions: flexion, abduction, and external rotation of the hip, flexion of the knee, and dorsiflexion and eversion of the ankle. Synergies may be complete (i.e., all components observed) in some hemiparetic subjects and partial in others.^{22,24} In the lower limb, the extension synergy may be more dominant than the flexion synergy.²⁴ It is difficult to distinguish between the possible causes of coupled motions during complex tasks, such as walking, because the ob-

Abbreviations: A/D, analog-to-digital; CP, cerebral palsy; EMG, electromyogram

Key words: cerebral palsy; coupling; extension synergy; joint moments; maximal exertion

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jective of the nervous system is unclear¹ and the complexity of the dynamics is great.³¹ It is easier to study neural control during static activities where a clear goal can be presented to the nervous system and the links between electromyographic (EMG) patterns and joint moments are more easily interpreted. Indeed, several studies have provided new insights into intermuscle coordination in control subjects^{6,7,14,27} and subjects after stroke^{2,11,12,15,16} using well-controlled static tasks.

Although the concept of neural coupling between joint motions has played a role in the development of therapies for CP,⁸ there is little experimental evidence revealing the biomechanical factors underlying coupled joint motions in this population. Our study explores this issue. We hypothesized that maximal voluntary exertions in the direction of one component of a synergy (e.g., hip extension) would result in the concurrent presence of other components of the synergy in CP subjects but not in control subjects. We also hypothesized that the extension synergy would be more prevalent than the flexion synergy in the lower limbs of CP subjects. We tested these hypotheses by recording lower-extremity joint moments and EMG activity during maximal isometric exertions in prescribed directions. Our results provide quantitative evidence for the existence of the classically defined extension synergy in CP.

MATERIALS AND METHODS

Subjects. Six control subjects (3 women and 3 men; mean age, 28 years) without neurological or orthopedic impairments and nine subjects with CP (5 women and 4 men; mean age, 20 years) participated in this study (Table 1). Seven of the subjects with CP had a diagnosis of spastic hemiplegia, and two had a diagnosis of spastic diplegia. The CP sub-

jects exhibited no cognitive deficits, were able to cooperate with instructions, and had minimal sensory loss on routine examination. Prior to participation in the experiment, each subject was seen in a gait analysis laboratory to assess walking ability. All subjects who participated in the study were able to walk independently in the community, could feel light touch to the thigh, leg, and foot, exhibited good voluntary control over the lower limb, and could understand and follow all instructions given during the experiment. None of the CP subjects had undergone surgery within the past year, and none were using any antispasticity medication. The surgical histories of the CP subjects are summarized in Table 1. The experiments were conducted at the Rehabilitation Institute of Chicago. The Institutional Review Board of Northwestern University approved the experimental protocol, and each subject provided informed consent in accordance with institutional policy.

Experimental Arrangement. The experimental apparatus allowed the computation of isometric flexion/extension, abduction/adduction, and internal/external rotation moments about the hip and flexion/extension moments about the knee (Fig. 1). We tested the dominant limb of the control subjects, the impaired limb of the hemiplegic CP subjects, and the more impaired limb of the diplegic CP subjects. Subjects were seated in an adjustable chair and positioned so that their knee and hip angles were each fixed in 45° of flexion. To minimize pelvic rotation, two straps were placed across the pelvic region. A body harness was placed over the subject's shoulders and secured to the chair to keep the subject from sliding during maximal exertions. A fiberglass cast was placed around the ankle. The cast was then fixed

Table 1. Characteristics of CP and control subjects included in the study.

Subject	Gender	Age (yr)	Mass (kg)	Ht (cm)	Limb tested	Surgeries*
1	F	16	45.5	165	Left	TAL
2	F	17	50.0	142	Left	TAL
3	M	15	46.4	157	Left	No surgeries
4	M	24	68.2	173	Right	Rectus, hamstring, TAL
5 [†]	M	24	90.9	183	Left	Hamstring, TAL
6	F	22	48.6	165	Right	TAL, SPLATT
7	M	15	50.0	168	Right	No surgeries
8 [†]	F	34	53.6	155	Right	Adductor, hamstring, TAL
9	F	20	70.0	159	Right	No surgeries
CP Average		20.8 ± 6.2	58.2 ± 15.2	163 ± 11		
Control average		28.2 ± 4.0	74.4 ± 16.5	179 ± 9		

*TAL, Achilles tendon lengthening; rectus, rectus femoris transfer; hamstring, distal hamstring lengthening; adductor, adductor myotomy; SPLATT, split anterior tibialis tendon transfer.

[†]Subjects diagnosed with diplegia.

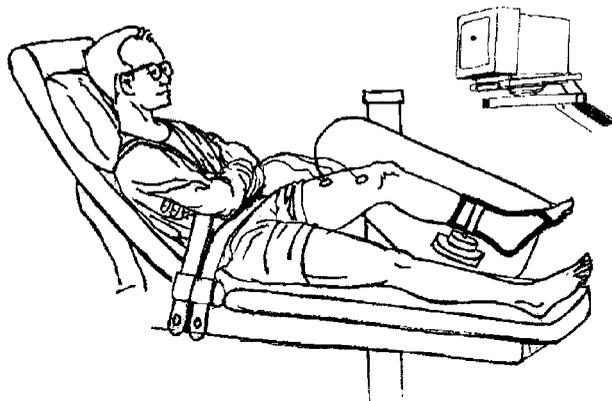


FIGURE 1. Experimental arrangement. Subjects were secured to an adjustable chair with their hip and knee flexed. A six degree-of-freedom load cell, fixed to the ankle, was used to measure joint moments about the hip and knee. The moment generated by the subjects in the prescribed direction was displayed to them on a computer monitor. The EMG activity of eight muscles was also monitored.

within a metal ring and attached to a six degree-of-freedom load cell (Assurance Technologies, Garner, North Carolina). Once positioned, the subject's limb segment lengths were measured to allow for real-time computation of joint moments.

Model AMI 1719-003 Ag-AgCl surface electrodes (Medtronic, Minneapolis, Minnesota) were used to monitor the EMG activities of eight muscles of the lower extremity: gluteus maximus, gluteus medius, adductor magnus, rectus femoris, vastus lateralis, vastus medialis, semitendinosus, and biceps femoris long head. Electrodes were placed over the muscles in accordance with the protocol described by Perotto.²³ The EMG signals were preamplified with a gain of 1000. The signals were then amplified with a gain of 10–50 to utilize the ± 10 -V range of the data acquisition equipment. All moment and EMG data were sampled at 1000 Hz using a 12-bit analog-to-digital (A/D) board.

Experimental Protocol. Subjects were asked to generate maximal voluntary exertions in four primary task directions: hip flexion, hip extension, knee flexion, and knee extension. The order in which the tasks were presented to each of the subjects was randomized to eliminate bias that could arise due to practice or fatigue. In all tasks, a measure of the moment developed in the primary task direction, termed “the primary moment,” was displayed on a computer monitor located directly in front of the subject (Fig. 1). A cursor on the monitor moved upward in proportion to the primary moment generated by the subject. No feedback was given for any moments other than the primary moment. Subjects

were allowed several minutes to practice each task before data collection to find the strategy that enabled them to generate the largest primary moment. The number of practice trials was not strictly controlled, but subjects were allowed to practice until they believed that they could consistently produce the primary moment. The control subjects typically performed fewer practice trials than did the CP subjects. Each trial began with the subject in a relaxed position and consisted of a 4-s ramp up to a maximal effort. Subjects repeated three to six trials for each task, with a 1-min rest period between trials. The task was completed when subjects performed three maximal trials with less than 10% variation in the magnitude of the primary moment.

Data Analysis. At each time step (1-ms intervals) of a trial, an inverse static analysis of the lower extremity was used to compute the hip and knee joint moments from the forces and moments measured by the load cell. The primary moment was defined as the maximal moment in the primary task direction generated over any 300-ms window during the trial (Fig. 2). That is, all possible 300-ms windows over the 4-s test interval were examined, and the window that yielded the maximal average moment in the primary task direction was used. The associated moments about the other degrees of freedom were determined by averaging the associated moment curves over the same 300-ms window. Similarly, muscle activities were quantified by the mean, rectified EMG signals over the same window. A 300-ms window was used to eliminate high-frequency fluctuations in the moments and EMGs without excessively smoothing the data. Moment and EMG data for individual subjects were normalized to allow for intersubject comparisons. For each task direction, moments were normalized by the maximal primary moment produced by the subject in that direction. The EMG data for each muscle were normalized by the maximal EMG recorded for that muscle over all trials.

Student's *t*-tests were used to identify associated moments that differed significantly from zero. Repeated measures analysis of variance (ANOVA) was used to test our hypothesis that maximal voluntary activation of one component of a synergy would result in the concurrent presence of other components of the synergy in subjects with CP but not in control subjects. For each primary task direction, we tested for significant differences between the control subjects and CP subjects in the normalized associated moments and normalized EMG activities. Three repeated trials were included for each subject in the analysis. No statistically significant trial effects were

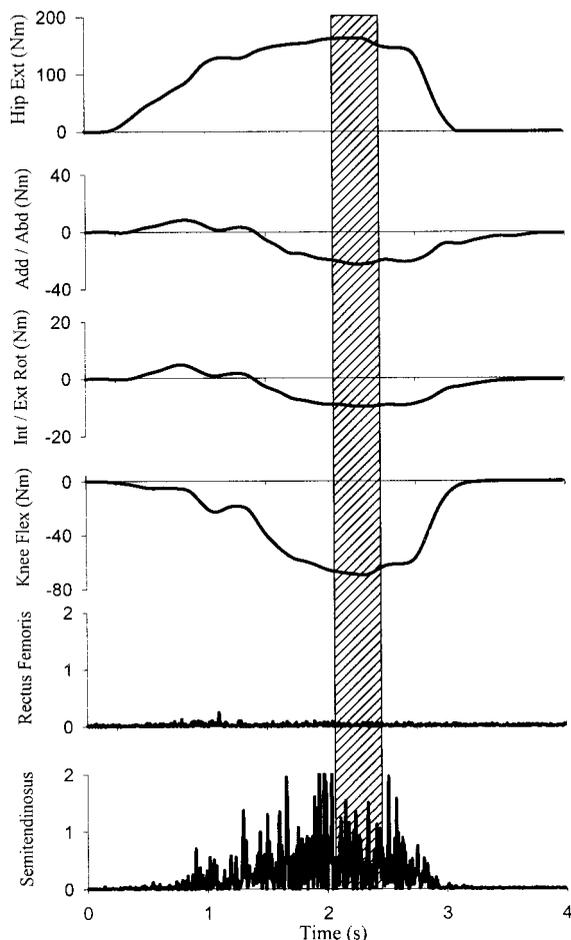


FIGURE 2. Sample time history of the joint moments (in Nm) and normalized EMGs during a maximal voluntary hip extension effort. In this task, hip extension (top panel) is considered to be the “primary moment” and the other moments are termed “associated moments.” The maximal primary moment during any 300 ms window was found (vertical shaded region). The associated moments and mean rectified EMG signals were quantified over this same window. In this control subject, a maximal exertion in hip extension resulted in the co-generation of associated moments in hip abduction, external rotation and knee flexion.

found, so only group (control versus CP) effects are reported. All statistical analyses were completed with Systat (SPSS, Inc., Chicago, Illinois). A significance level of $P = 0.05$ was used for all comparisons.

RESULTS

Hip Extension. When generating a maximal voluntary hip extension moment, control subjects simultaneously generated an associated knee flexion moment (Fig. 3A). In contrast, when generating a maximal hip extension moment, CP subjects generated an associated knee extension moment ($P < 0.005$) and a significantly larger hip internal rotation moment ($P < 0.05$) than did controls. The differ-

ences demonstrate the tendency for the CP subjects to generate moments that are consistent with an extension synergy. Significantly larger EMG activities of the rectus femoris, vastus lateralis, and vastus medialis muscles in CP subjects contributed to the knee extension moment generated during maximal voluntary hip extension.

Knee Extension. When generating a maximal knee extension moment, control subjects simultaneously generated hip flexion, abduction, and internal rotation moments (Fig. 3B). The CP subjects demonstrated an opposite strategy by generating a hip extension moment during this task ($P < 0.005$). The CP subjects also generated a significantly smaller hip abduction moment than did control subjects ($P < 0.05$). The EMG activities of the semitendinosus and biceps femoris muscles were larger ($P < 0.005$) in the CP subjects during maximal knee extension efforts, whereas activity of the rectus femoris was lower in the CP group ($P < 0.05$). Given that the hamstring muscles generate a hip extension moment and the rectus femoris generates a hip flexion moment, these activation differences likely contributed to the hip extension moment generated by the CP group during knee extension efforts.

Hip Flexion. During maximal hip flexion exertions, control subjects did not generate associated moments about any other degrees of freedom (Fig. 4). Similarly, none of the mean associated moments generated by the CP subjects were significantly different from zero. The CP subjects had larger activities in the semitendinosus ($P < 0.05$) and biceps femoris ($P < 0.005$) muscles during hip flexion exertions.

Knee Flexion. The control and CP groups generated hip extension, adduction, and internal rotation moments during maximal knee flexion exertions (Fig. 4B). None of the associated moments differed significantly between the CP and control groups. Gluteus medius EMG activity was higher in the CP group ($P < 0.05$), but EMG activities were similar between groups for all other muscles examined.

Isometric Strength. The CP subjects exhibited significant ($P < 0.05$) weakness of the hip extensors, hip adductors, knee flexors, and knee extensors compared with control subjects (Table 2). The greatest deficits were observed in the knee flexion and extension tasks, where CP subjects possessed only 51 and 43% of the normalized strength measured in the control group, respectively.

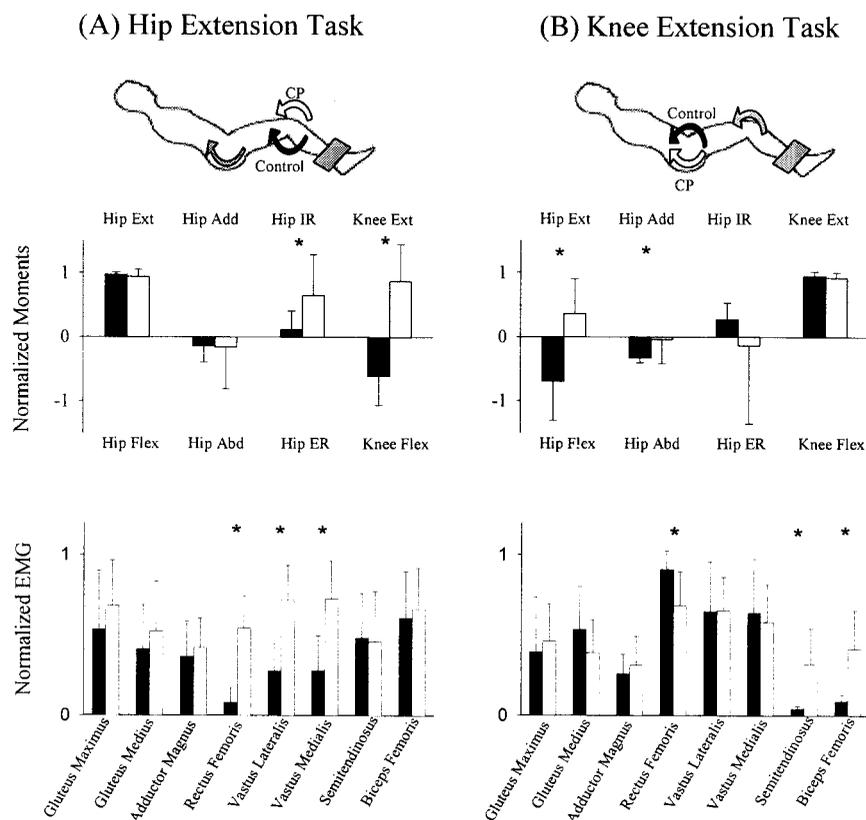


FIGURE 3. Normalized mean (\pm SD) moments and muscle activities for control (solid bars) and CP (open bars) subjects during maximal voluntary hip extension (A) and knee extension (B). Significant differences between groups are denoted (* $P < 0.05$). The CP subjects, on average, generated a hip extension moment (Hip Ext) during maximal knee extension exertions and knee extension moments (Knee Ext) during maximal hip extension exertions, a pattern opposite that of control subjects and consistent with the extension synergy. Also shown are the associated moments generated in hip flexion (Hip Flex), hip adduction (Hip Add), hip abduction (Hip Abd), hip internal rotation (Hip IR), hip external rotation (Hip ER), and knee flexion (Knee Flex) during these tasks.

DISCUSSION

We hypothesized that maximal voluntary exertions in the direction of one component of the classically defined extension synergy would result in the concurrent presence of other components of the synergy in CP subjects but not in control subjects. To test this hypothesis, we measured the moments generated about the knee and hip during maximal voluntary isometric exertions. During these tasks, the CP subjects demonstrated evidence of some elements of the extension synergy. During hip extension efforts, CP subjects demonstrated abnormal co-generation of knee extension and hip internal rotation moments. Also, during knee extension exertions, CP subjects generated a hip extension moment and had a tendency toward hip adduction. The EMG data suggest that the joint moment couplings were related to a greater degree of coactivation of the knee and hip extensors in CP subjects. These data represent quantitative evidence of the coupling of the joint moments that could potentially give rise to synergistic motion patterns in persons with CP.

The control subjects demonstrated couplings of hip and knee moments during extension tasks that were consistent with mechanical demands of the task. For example, when asked to generate a maximal hip extension moment, control subjects pushed posteriorly on the load cell placed just above the ankle (Fig. 1) and co-generated a knee flexion moment (Fig. 3). By adopting this strategy, control subjects were able to produce a resultant force vector at the load cell that had a large extension moment arm about the hip. In order to co-generate hip extension and knee extension moments (as the CP subjects did), one must generate a resultant force vector that is directed between the hip and knee. This limits the size of the extension moment arm of the resultant force about the hip, and thus requires a larger resultant force to produce a hip extension moment of comparable magnitude to that of the control subjects.

We found no evidence of a flexion synergy during tasks that involved the generation of maximal hip and knee flexion moments. These results are

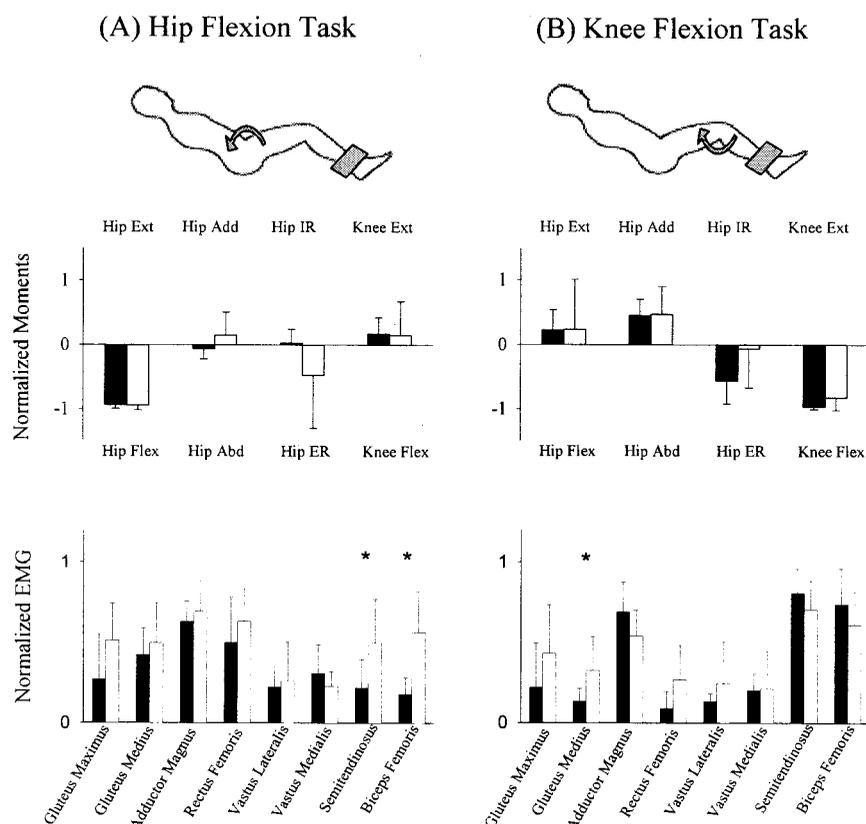


FIGURE 4. Normalized mean (± 1 SD) associated moments and muscle activities for controls (solid bars) and CP (open bars) subjects during maximal voluntary hip flexion (**A**) and knee flexion (**B**). There were no significant differences in the associated moments between controls and CP subjects during either of these tasks. Significant differences between the muscle activity levels of controls and CP subjects are denoted (* $P < 0.05$). (Abbreviations for the associated moments are given in Fig. 3.)

consistent with clinical observations of subjects with hemiplegia following stroke, which suggest that the extension synergy is more dominant than the flexion synergy in the lower limb.²⁴ However, the limited number of subjects precludes us from clarifying the prevalence of the flexion synergy in CP subjects. The CP subjects were weaker than the control subjects, with the greatest deficits found at the knee. Although these results differ from those of Wiley and Damiano,³⁰ who found the greatest deficit in hip

extension, the magnitude of knee strength deficits are within the range reported in other studies. Strength in subjects with CP as a percentage of control values has ranged from 42 to 69% for the knee extensors and 45 to 77% for the knee flexors.^{10,13,18,30} A number of mechanisms could contribute to strength deficits in persons with CP, including decreased neural drive, muscle atrophy, changes in muscle mechanical properties, and impaired coordination.³

Task	Control group (n = 6)	CP group (n = 9)	Percent of control (current study)	Percent of control (Wiley and Damiano, 1998)
Hip flexion	1.54 \pm 0.29	1.17 \pm 0.59	76	73 [†]
Hip extension	2.33 \pm 0.72	1.37 \pm 0.76 [†]	59 [†]	47 [†]
Hip abduction	1.03 \pm 0.41	0.70 \pm 0.61	68	69 [†]
Hip adduction	0.87 \pm 0.37	0.54 \pm 0.19 [†]	62 [†]	72 [†]
Knee flexion	0.87 \pm 0.30	0.44 \pm 0.45 [†]	51 [†]	77 [†]
Knee extension	0.95 \pm 0.21	0.41 \pm 0.28 [†]	43 [†]	69 [†]

*Data are mean (\pm SD) maximum isometric moments normalized to body mass (expressed in N.m.kg⁻¹).

[†]Significant difference ($P < 0.05$) between control and CP groups.

It is not clear whether the differences in coordination between CP and control subjects observed in this study during maximal exertions would be present during submaximal exertions. Previous studies have shown that synergies that are not present at submaximal levels tend to emerge under conditions of extreme effort.^{17,28}

Several limitations of this study should be considered. First, subjects were tested in a reclined, seated position rather than in a more extended weight-bearing position. It is possible that muscular coordination patterns would differ if added demands, such as maintaining balance, were placed on the subject or if different sensory information, such as altered input from vestibular or cutaneous afferents, were available.⁴ Second, no attempt was made to quantify neural coupling that might exist between the muscles of right and left limbs. We recorded moments and EMG patterns from a single limb and thus cannot address questions related to interlimb coupling. Third, the subjects examined in this study were functioning at a high level. All were able to ambulate in the community and complete all the tasks during the experiment. Thus, conclusions cannot be drawn about the presence or absence of coupling between the hip and knee in subjects with a greater degree of neurological impairment.

To compare data between subjects, we normalized joint moments and muscle activities to maximal levels recorded for each individual subject. Because these tasks involved voluntary exertions, it is possible that these maximal measures do not reflect the full capacities of the subjects. Our testing procedure, however, was designed to produce repeatable strength measures. For each primary task direction, subjects repeated the task until they generated primary moments that did not vary more than 10% across three trials. It is noteworthy that the most important group differences in associated moments involved exertions in opposite directions; thus, those results are independent of the normalization procedure. For example, the observation that control subjects generated a knee flexion moment, whereas CP subjects generated a knee extension moment, during maximal hip extension efforts is independent of the technique used to normalize moments.

Abnormal muscle activity in subjects with CP frequently is attributed to spasticity.⁹ It is important to note that the abnormal patterns of muscle activation observed in this study likely cannot be attributed to abnormalities of the monosynaptic stretch reflex,¹⁹ because the subjects were tested under isometric conditions that did not stretch the muscle.

Newborn children typically exhibit stepping-type movements with synchronous flexion or extension of the lower-limb joints.²⁰ As infants mature, these patterns of mass activation disappear as supraspinal inputs integrate with spinal control centers. These inputs help to fractionate the activation patterns, allowing for more specific control of individual muscles or muscle groups. Our results show that individuals with CP continue to exhibit an extension pattern, suggesting that the supraspinal inputs may fail to properly integrate with spinal control systems. These results indicate that abnormal neural control, consistent with the classically defined extension synergy,²⁴ may contribute to stereotypical movement abnormalities in CP.

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REFERENCES

1. Bernstein N. The coordination and regulation of movements. New York: Pergamon Press; 1967. 196 p.
2. Bourbonnais D, Vanden Noven S, Carey KM, Rymer WZ. Abnormal spatial patterns of elbow muscle activation in hemiparetic human subjects. *Brain* 1989;112:85-102.
3. Bourbonnais D, Vanden Noven S. Weakness in patients with hemiparesis. *Am J Occup Ther* 1989;43:313-319.
4. Brown DA, Kautz SA, Dairaghi CA. Muscle activity patterns altered during pedaling at different body orientations. *J Biomech* 1996;29:1349-1356.
5. Brunstrom S. Associated reactions of the upper extremity in adult patients with hemiplegia: an approach to training. *Phys Ther Rev* 1956;36:225-236.
6. Buchanan TS, Almada DP, Lewis JL, Rymer WZ. Characteristics of synergic relations during isometric contractions of human elbow muscles. *J Neurophysiol* 1986;56:1225-1241.
7. Buchanan TS, Roval GP, Rymer WZ. Strategies for muscle activation during isometric torque generation at the human elbow. *J Neurophysiol* 1989;62:1201-1212.
8. Carr JH, Shepherd RB. Neurological rehabilitation: optimizing motor function. Oxford: Butterworth Heinemann; 1998.
9. Crothers B, Paine R. The natural history of cerebral palsy. In: Mitchell R, editor. *Classics in developmental medicine*, vol. 2. Philadelphia: Lippincott; 1959.
10. Damiano DL, Vaughn CL, Abel MF. Muscle response to heavy resistance exercise in children with spastic cerebral palsy. *Dev Med Child Neurol* 1995;37:731-739.
11. Dewald JP, Pope PS, Given JD, Buchanan TS, Rymer WZ. Abnormal muscle coactivation patterns during isometric torque generation at the elbow and shoulder in hemiparetic subjects. *Brain* 1995;118:495-510.
12. Dewald JP, Beer RF. Abnormal joint torque patterns in the paretic upper limb of subjects with hemiparesis. *Muscle Nerve* 2001;24:273-283.
13. Engsberg JR, Ross SA, Hollander KW, Park TS. Hip spasticity and strength in children with diplegia cerebral palsy. *J Appl Biomech* 2000;16:221-233.
14. Filiatrault J, Bourbonnais D, Gauthier J, Gravel D, Arsenuit AB. A method for quantification of directional patterns of muscle activation at the lower limb. *J Electromyogr Kinesiol* 1992;2:81-90.

15. Filiatrault J, Bourbonnais D, Gauthier J, Gravel D, Arsenault AB. Directional patterns of muscle activity at the lower limb in subjects with hemiparesis and in healthy subjects: a comparative study. *J Electromyogr Kinesiol* 1992;2:91-102.
16. Gauthier J, Bourbonnais D, Filiatrault J, Gravel D, Arsenault AB. Characterization of contralateral torques during static hip efforts in healthy subjects and subjects with hemiparesis. *Brain* 1992;115:1193-1207.
17. Hellenbrandt FA, Waterland JC. Expansion of motor patterning under exercise stress. *Am J Phys Med* 1962;41:56-66.
18. Horvat M. Effects of a progressive resistance training program on an individual with spastic cerebral palsy. *Am Correct Ther J* 1987;41:7-11.
19. Katz RT, Rymer WZ. Spastic hypertonia: mechanisms and measurement. *Arch Phys Med Rehabil* 1989;70:144-155.
20. Leonard CT, Hirschfeld H, Forsberg H. The development of independent walking in children with cerebral palsy. *Dev Med Child Neurol* 1991;33:567-577.
21. Leonard CT. The neurophysiology of human locomotion. In: Craik RL, Oatis CA, editors. *Gait analysis: theory and application*. St. Louis: Mosby; 1995. p. 46-64.
22. Michels E. Synergies in hemiplegia. *Clin Manag* 1982;1:9-16.
23. Perotto AO. *Anatomical guide for the electromyographer: the limbs and trunk*, 3rd ed. Springfield: Charles C. Thomas; 1994. 309 p.
24. Sawner K, LaVigne J. *Brunnstrom's movement therapy in hemiplegia: a neurophysiological approach*, 2nd ed. Philadelphia: JB Lippincott; 1992. 276 p.
25. Scherzer AL, Tscharnuter I. *Early diagnosis and therapy in cerebral palsy*, 2nd ed. New York: Marcel Dekker; 1990. 351 p.
26. Sutherland DH, Davids JR. Common gait abnormalities of the knee in cerebral palsy. *Clin Orthop* 1993;288:139-147.
27. Vasavada AN, Delp SL. Influence of muscle morphometry and moment arms on the moment-generating capacity of human neck muscles. *Spine* 1998;23:412-422.
28. Waterland JC, Munson N. Involuntary patterning evoked by exercise stress. *Phys Ther* 1964;44:91-97.
29. Waters RL, Frazier J, Garland DE, Jordan C, Perry J. Electromyographic gait analysis before and after operative treatment for hemiplegic equine and equinovarus deformity. *J Bone Joint Surg* 1982;64A:284-288.
30. Wiley ME, Damiano DL. Lower-extremity strength profiles in spastic cerebral palsy. *Dev Med Child Neurol* 1998;40:100-107.
31. Zajac FE, Gordon ME. Determining muscle's force and action in multi-articular movement. *Exerc Sport Sci Rev* 1989;17:187-230.