Predictive and Reactive Control of Precision Grip in Children With Congenital Hemiplegia

Yannick Bleyenheuft and Jean-Louis Thonnard

Neurorehabil Neural Repair 2010; 24; 318 originally published online Dec 3, 2009;
DOI: 10.1177/1545968309353327

Neurorehabilitation and
Neural Repair
24(4) 318–327

DOI: 10.1177/1545968309353327
http://nnr.sagepub.com
SAGE

Predictive and Reactive Control of Precision Grip in Children With Congenital Hemiplegia

Yannick Bleyenheuft, PhD[1] and Jean-Louis Thonnard, PhD[1]

Abstract

Background and Objectives. Grasping an object between the thumb and index finger requires precise coordination between grip force (GF) and tangential load force (LF), which is impaired in children with congenital hemiplegia (CH). This study aimed to determine the respective contributions of predictive and reactive control in the impaired precision grip of 12 children with CH between 10 and 16 years of age when compared with age- and gender-matched controls. *Methods*. The load of a handheld object was increased rapidly by generating an impact through the drop of a mass attached to the object. The drop was triggered by the participant (predictive conditions) or unexpectedly by the examiner (reactive conditions). In both conditions, participants aimed to prevent the object from falling. Both hands of children with CH and controls were tested. *Results*. During our task, no differences in the GF levels were observed between paretic, nonparetic, and control hands. Under predictive conditions, the temporal variables related to the GF were preserved before impact in children with CH but altered after impact. Under reactive conditions, the reactive delays were longer in the paretic hand. Predictive and reactive control were preserved on the nonparetic hand. *Conclusions*. Deficits were observed in both predictive and reactive control for the paretic hand. The predictive control exists but is altered after the impact, suggesting an inability to anticipate the consequences of a dynamic perturbation. The authors suggest that the abilities of the nonparetic side could be used in neurorehabilitation to improve motor control of the paretic side.

Keywords

cerebral palsy, precision grip, predictive control, reactive control, hemiparesis

Introduction

Children with cerebral palsy (CP) who have congenital hemiplegia (CH) usually do not acquire normal skilled hand movements.[1-7] Their precision grip, necessary to pick up small objects between the thumb and index finger (eg, pencil, strawberry), is affected. In precision grip tasks that require precise coordination between the normal grip force (GF) and the load force (LF), which is tangential to grip surfaces, children with CH usually show a pattern of movement reminiscent of that observed in normal younger children with immature prehension.[1] In healthy adults, a good synchronization of GF and LF forces incorporates a predictive control, allowing anticipation of the movement on the basis of sensorimotor memory, and a reactive control, allowing correction of the movement through feedback mechanisms. Predictive control allows one to correctly target forces to lift a milk carton, for instance. Reactive control modulates the forces to stabilize the milk carton in the air. The particularities of GF and LF traces, present in both children with CP and young children, have been frequently interpreted as deficits of predictive programming.[1] It is not clear, however, if the impairments in fine prehension are linked to deficits in either predictive or reactive control because it was also hypothesized that these children were unable to adapt the GF according to cutaneous feedback.

Our protocol allowed us to test both the predictive and reactive aspects of precision grip control in the same task because the load of a handheld object was increased abruptly by the subject (predictive conditions) or unexpectedly by the examiner (reactive conditions). Such paradigms have been investigated in adults[8-11] and in healthy children[12] (Bleyenheuft and Thonnard, unpublished data, 2009). In adults, examiner-triggered impact evoked a reactive GF increase,

[1]School of Medicine, Université Catholique de Louvain, Brussels, Belgium

Corresponding Author:
Jean-Louis Thonnard, Université catholique de Louvain, Unité de Réadaptation et de Médecine Physique, Avenue Mounier 53, 1200 Bruxelles, Belgium
Email:Jean-louis.thonnard@uclouvain.be

described as an automatic long latency response (long-loop reflex).[8] In self-initiated load increases, the anticipation consists of a systematic increase in GF prior to impact, which is scaled to the impact properties.[8] A second increase in GF is often observed after the impact, leading to a maximum GF that occurs later than the LF maximum.[3,8,13-15] This GF increase after the impact is of a predictive nature in healthy young adults.[9] Under predictive conditions, healthy young children showed longer delays to increase forces after impact and longer delays to reach maximum GF (Bleyenheuft and Thonnard, unpublished data). In the present study, we aimed to identify the predictive or reactive nature of precision grip deficits in children with CH. This is of interest in neurorehabilitation because strategies of predictive control are likely to be improved through rehabilitation, whereas deficits in purely reactive conditions (long-loop reflex latencies[8]) present few chances for rehabilitation. On the basis of their abilities in precision grip tasks, we hypothesized that children with CH would present characteristics similar to those of younger children.

In addition, the study of predictive and reactive control was used to refine the use of corticospinal (CS) tract dysgenesis[16] as a predictive tool of hand deficits in children with CH.[7,17] We correlated CS tract dysgenesis with variables under both predictive and reactive conditions, aiming to determine if this measurement was able to anticipate deficits from impaired predictive control or from a disrupted long-loop reflex (reactive control).

The objectives of this study were thus to investigate (1) whether the impairments in the precision grip of children with CH were a result of deficits in their ability to anticipate movements and/or to impairments in reactive control and (2) if the CS tract dysgenesis measurement was able to anticipate deficits from an impaired predictive or reactive control.

Methods

This study was authorized by the Ethical Committee of the Université Catholique de Louvain, Faculty of Medicine, Belgium. The parents of the children provided written informed consent.

We assessed 12 children with CH (10 boys and 2 girls) between the ages of 10 and 16 years (Table 1) and 12 age- and gender-matched controls. The ability to meet the academic requirements of healthy peers was a selection criterion and assessed the absence of cognitive deficits. Severity of hemiplegia was categorized according to the Gross Motor Function Classification[18] as level 1 or 2. This classification is used to roughly categorize the motor function in children with CP. It presents 5 levels that reflect differences in motor function that are meaningful in the daily lives of children with CP. Distinctions between the levels are primarily based on sitting and walking abilities. Levels 1 and 2 mean that the child is able to walk independently.[18]

Apparatus

Fingertip GF and LF were measured with a handheld object (220 g mass) equipped with 2 parallel vertical grip surfaces of smooth brass (40 mm diameter, 30 mm apart, Figure 1). Two 3-dimensional force–torque sensors were used to measure the GF and LF values, calculated on the basis of the 3 orthogonal force components (F_x, F_y, F_z) applied to the grip surfaces. The F_x, F_y, and F_z sensing ranges were ±40, ±40, and ±120 N, with resolutions of 0.002, 0.002, and 0.006 N, respectively. LF, tangential to the grip surface, was the result of the vectorial sum of F_x and F_y, and the force normal to the grip surface (GF) was given by F_z.

The horizontal (x) and vertical (y) centers of pressure were also measured. The object was placed on an open table (Figure 1). A steel mass (100 g) attached to the object via a Kevlar string was placed on an electromagnet. This additional mass was loaded to the object when the magnetic field was turned off by a button switch.

Procedure and Experimental Protocol

First, the children washed their hands. Each child comfortably sat next to the table so as to provide support to the forearm. While keeping their forearm on the support, the child was required to grasp, with a precision grip, the object given by the examiner and maintain it in a standard position (keep a reference mark on the string in front of a reference mark on the table).

In the predictive trials, each child held a button switch in his or her free hand that, when pushed in response to an auditory signal, turned off the magnetic field. This led to the drop of the mass (4 cm) and a consequent sudden LF increase (impact). In the reactive trials, the examiner triggered the drop of the object, thereby creating a sudden and unpredictable impact.

Both hands were systematically tested in controls and children with CH, starting with the paretic hand for the latter group. Each child performed a sequence of 20 repeated trials under predictive conditions, followed by 10 under reactive conditions with each hand; 4 maneuvers preceded and followed the experiment to measure the coefficient of friction (CF) between the skin and the object. The children gripped the handheld object so that it was stationary and then slowly released the thumb and index finger until the object slipped. The CF was measured as $0.5 \times (LF/GF)$ at the onset of the slip, which was detected by the vertical component of the center of pressure. The 4 friction measurements taken before the experiment were not different from the 4 friction measurements performed afterward (paired

Table 1. Topography, Etiology, Scores on GMFC, and Corticospinal Asymmetry

Age (years)/ Gender	Topography	Etiology (MRI)	GMFC (Level)	CTsym (%)
10.1/M	L hemiplegia	R encephalomalacy in superficial sylvian area, R cerebral peduncle atrophy	I	36
10.5/M	L hemiplegia	R widespread micropolygyria (frontal lobe, insula, and part of temporal lobe); ventricular, thalamic, and peduncular asymmetry (L > R)	2	27
10.9/M	R hemiplegia	L subcortical malacical lesions (ovale centrum, periventricular white matter, caudate nucleus), L thalamic and peduncular atrophy	I	60
11.1/M	R hemiplegia	L periventricular leucomalacy (white matter, caudate nucleus), L moderate ventricular widening, relative thalamic atrophy	I	71
11.6/M	L hemiplegia	Widespread macrocystic leucomalacy, R parietal and frontal lobe, L discrete parietal lesion	I	53
11.9/F	L hemiplegia	R sylvian artery stroke with microcystic and macrocystic gliosis in the ovale centrum (white matter atrophy), R ventricular widening, R caudate nucleus lesion	2	61
12.6/F	L hemiplegia	Very discrete periventricular leucomalacy	I	88
13.3/M	R hemiplegia	Bilateral periventricular leucomalacy, with L predominance, thalamic asymmetry (R > L)	I	69
13.9/M	R hemiplegia	Large L macrocystic gliosis in the left hemisphere (anterior temporal pole, middle and superior temporal gyrus, inferior and middle frontal gyrus, insula, parietal lobe, part of occipital lobe, lenticular nucleus, and caudate nucleus body major part of thalamus); large L peduncular, corticospinal, and bulbar atrophy, L ventricular widening	I	21
15.1/M	L hemiplegia	Drained hydrocephaly, bilateral periventricular leucomalacy in posterior regions, predominant in R ovale centrum, corpus callosum atrophy	2	67
15.6/M	L hemiplegia	Large R macrocystic lesion in deep and superficial sylvian territory; hemispheric, thalamic, and peduncular atrophy; discrete L cerebellar atrophy	2	26
15.9/M	L hemiplegia	R large macrocystic gliosis in ovale centrum associated with R peduncular and corpus callosum atrophy consistent with vascular prenatal cerebral lesion; L discrete internal frontal closed schizencephaly	I	33

Abbreviations: M, male; F, female; R, right; L, left; GMFC, Gross Motor Function Classification; CTsym, corticospinal symmetry index.

t test, $P = .935$). The 8 measurements were therefore averaged to provide a single, reliable CF measurement.

A few minutes before starting the experiment, the maximal voluntary contraction (MVC) developed between the thumb and index finger was also measured by the instrumented object.

Data Acquisition and Analysis

For each trial, a stable phase was visually determined thanks to the stability in the LF trace, providing reference values during the waiting period that preceded the impact phase. The mean GF and mean safety margin (SM) were calculated during this stable phase. The SM to prevent slipping is the difference between the GF used and the minimal GF necessary to prevent slipping.[19] This SM was calculated according to the following equation:

$$\text{SM} = \text{GF} - \text{GF at slip}, \quad (1)$$

where GF at slip = 0.5 × (LF/CF).

During the impact phase, the following dynamic variables were investigated: GF and SM at impact and GF_{max} and the maximum GF rate before and after impact. The temporal variables were defined as follows (see Figure 2):

1. the anticipatory delay was defined as the duration between the onset of the GF and the impact;

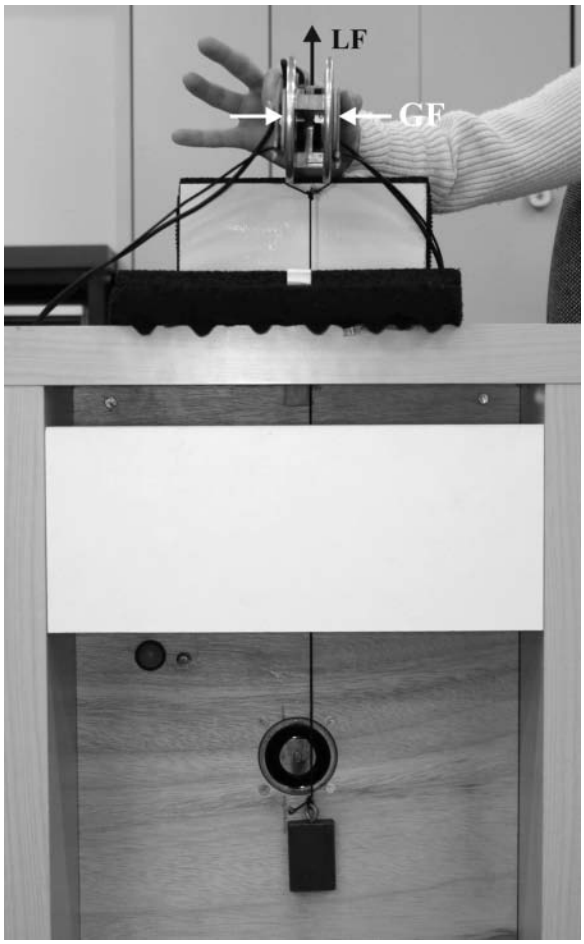

Figure 1. The handheld object used to measure the different forces during the precision grip task with the additional load suspended via a Kevlar string. The grip force (GF) normal to the contact surfaces is indicated by white arrows, and the tangential load force (LF) is indicated by a black arrow

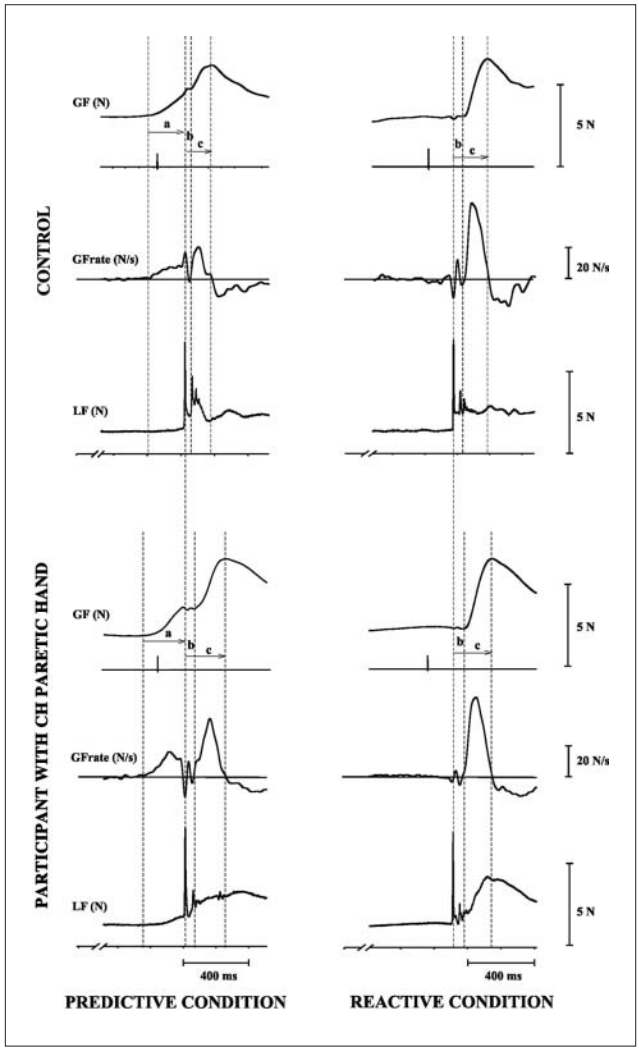

Figure 2. Examples of GF, GF rate, and LF traces recorded under predictive and reactive conditions from a control and a participant with CH. In each trace, the vertical dotted lines represent time references to calculate the different delays: A, the anticipatory delay; B, the postimpact delay; and C, the delay to GF_{max}. The short vertical bar indicates the moment when the participant pressed the button switch

2. the postimpact delay was defined as the duration between the impact and the increase in GF after the impact; and
3. the delay to GF_{max} was defined as the duration between the impact and GF_{max}.

The onset of GF increase before and after impact was determined by visual inspection of GF and by considering d(GF)/dt.

The number of slips were counted in both the predictive and reactive trials. A slip was identified during the impact phase when the difference between the highest and the lowest values of the *y* center of pressure (vertical axis) exceeded 5 mm.

Corticospinal Tract Dysgenesis

In a previous study, we showed a positive correlation between CS dysgenesis and upper limb impairments and disabilities in the same set of patients.[17] The more symmetric the tracts measured with diffusion tensor imaging, the better the scores of manual dexterity, digital dexterity, stereognosis, and manual ability. Briefly, a color-coded diffusion tensor imaging map[20] was generated, where the direction of fibers was determined by the direction tensor's main eigenvector, depicted in blue for the craniocaudal, green for the anteroposterior, and red for the transcallosal directions. At

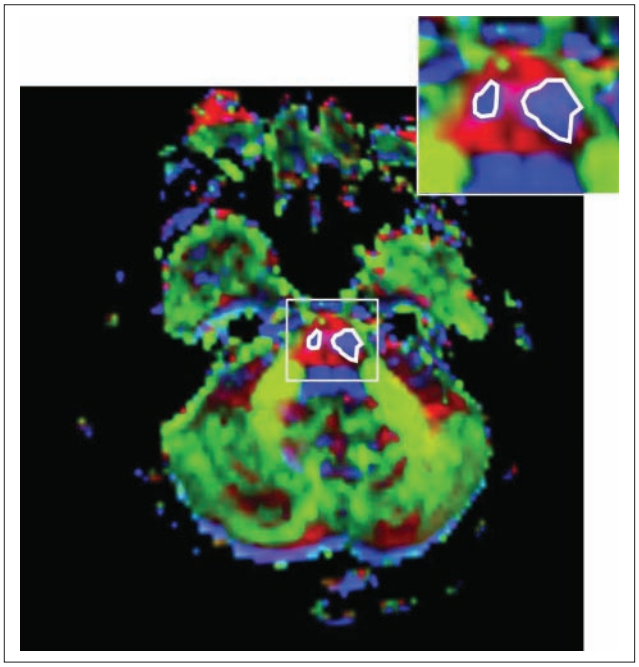

Figure 3. A diffusion tensor imaging slice at the level of the middle cerebellar peduncle in a child with CH. Transcallosal fibers are coded in red, anteroposterior in green, and craniocaudal in blue. The fibers of the CS tract are delineated in white

the level of the middle cerebellar peduncles, the CS tracts (in blue) were manually delineated,[21] and the area was calculated in pixels (Figure 3). A symmetry index between both sides was then calculated as: (contralateral area/ipsilateral area) × 100. The CS tract symmetry index of the 12 healthy children was 99% ± 9.3%.[17]

Statistics

Because it required only 1 trial for the control children and the nonparetic hand of the children with CH to attain stable values, we decided to perform the analysis on the last 19 and the last 9 trials for the predictive and reactive conditions, respectively.

A 1-way repeated ANOVA ($ANOVA_{RM}$ or Friedman analysis under nonparametric conditions) was conducted to compare the 4 groups of data—namely, the paretic and nonparetic hands of the children with CH and the dominant and nondominant hands of the controls. A Tukey test was used to determine the factors that were significantly different.

A 1-way ANOVA (or Kruskal-Wallis analysis under nonparametric conditions) was used to compare the number of slips between the 4 groups of data. A Tukey test was used to determine the factors that were significantly different.

Results

Figure 2 shows a typical trace of a control's hand (upper panel) and of the paretic hand of a child with CH (lower panel) under predictive (left panels) and reactive (right panels) conditions. Under predictive conditions, the control increased the GF before the impact, even before pressing the switch (short vertical bar). After the impact, a second increase in the GF was evident, leading to the maximum GF. In the trace of a child with CH, the GF of the paretic hand increased before the impact, showing a clear anticipation with delays similar to those of controls. After the impact, the second increase in GF was present, leading to the maximum GF. This increase began later than the increase observed in the controls, and a longer duration to reach GF_{max} was observed. The observations on the typical traces were confirmed by statistical analysis on the entire sample of children with CH and controls (Table 2). Although there were no significant differences between the dynamic variables of the children with CH and those of the controls, some temporal variables were different in children with CH.

Under predictive conditions, the postimpact delay was significantly longer in the paretic hands of children with CH when compared with the nonparetic hands and to both hands of the controls (Table 2). The delay to GF_{max} was also significantly longer in the paretic hand when compared with both hands of controls (Table 2). Interestingly, this delay was also far more variable in the paretic hands of children with CH (coefficient of variation: 66% ± 40%, mean ± SD) when compared with the hands of controls (28% ± 15%) and with the nonparetic hands of children with CH (31% ± 17%), showing an inconsistent (less regular) temporal adjustment in reaching the maximum of GF (Friedman analysis, $P = .016$).

Under reactive conditions, as observed in Figure 2, no increase was observed in the GF of the controls prior to the perturbation (right upper panel). The impact was followed by a rapid GF increase, which led to GF_{max}. In children with CH (Figure 2, right lower panel) as for control children, no increase was observed in the GF prior to the perturbation. The impact induced a rapid GF increase, which began later than the GF increase observed in controls. The GF_{max}, however, was reached with similar delays. Statistical analysis on the entire sample showed that the postimpact delay was significantly longer in the paretic hands of children with CH (Table 2); however, the delay to GF_{max} was not significantly different when compared with the hands of controls (Table 2). The variation in this delay was not significantly different between the 4 groups (paretic, nonparetic, and both hands of controls; $ANOVA_{RM}$: $P = .496$). As with predictive conditions, no differences in dynamic variables were observed between the children with CH and the controls under reactive conditions (Table 2).

Table 2. Mean Values of Dynamic and Temporal Variables[a]

Variables	Values				*P* Value	Post Hoc Analysis, *P* Value		
	CH Paretic, Mean (SD)	CH Nparetic, Mean (SD)	CTRL Ndom, Mean (SD)	CTRL Dom, Mean (SD)	$ANOVA_{RM}$ or Friedman	CH Paretic/CH Nparetic	CH Paretic/ CTRL Ndom	CH Paretic/ CTRL Dom
Predictive condition								
GF stable phase (N)	3.2 (1.45)	3.2 (0.83)	2.9 (0.86)	3.2 (1.06)	.862			
GF at impact (N)	6.4 (3.38)	4.9 (1.47)	5.12 (1.18)	5.3 (1.32)	.187			
GF_{max} (N)	8.9 (4.62)	7.5 (1.96)	7.3 (1.79)	7.97 (2.08)	.729			
GF rate maximum after impact (N/s)	31 (18.8)	38.3 (15.11)	35.0 (16.3)	43.1 (18.39)	.299			
GF rate maximum before impact (N/s)	29 (19.9)	15.7 (8.1)	19.8 (7.64)	19.1 (4.09)	.195			
D anticipation (ms)	334 (49)	341 (81.8)	344 (114.9)	336 (95.4)	.993			
D postimpact (ms)	57 (10)	47 (7.5)	44 (6.3)	37 (5.3)	<.001[b]	.015[b]	.002[b]	<.001[b]
D to GF_{max} (ms)	210 (77)	167 (31.8)	132 (17.6)	143.3 (15.9)	<.001[b]	.115	.001[b]	.005[b]
Reactive condition								
GF stable phase (N)	3.9 (1.42)	3.8 (1.19)	3.4 (1.02)	3.6 (1.23)	.572			
GF at impact (N)	6.5 (5.17)	4.1 (1.59)	3.4 (0.88)	3.5 (1.20)	.058			
GF_{max} (N)	10.1 (6.1)	7.7 (2.54)	6.9 (1.94)	6.5 (1.86)	.167			
GF rate maximum after impact (N/s)	76.9 (64.89)	60.8 (35.13)	47.3 (22.31)	45.1 (21.99)	.801			
D postimpact (ms)	67 (10.5)	57 (6.9)	57 (6.0)	59 (4.9)	.006[b]	.011[b]	.017[b]	.043[b]
D to GF_{max} (ms)	243 (60.4)	212 (25.2)	218 (46.1)	208 (32.1)	.253			

Abbreviations: CH, patients with congenital hemiplegic; SD, standard deviation; CTRL, controls; Dom, dominant hand; Ndom, nondominant hand; Nparetic, nonparetic hand; D, delay; GF, grip force.
[a]Results of Tukey tests (post hoc) are given as *P* values in $ANOVA_{RM}$
[b]Indicates significant difference.

Correlation With CS Tract in Children With CH

To further investigate whether the differences between children with CH and controls observed after impact were of a predictive or reactive nature, we correlated the delay postimpact and the delay to GF_{max} and their coefficient of variation with CS dysgenesis. The only significant correlation appeared under predictive conditions, where the coefficient of variation of the delay to GF_{max} in the paretic hand was negatively correlated with CS tract dysgenesis (Spearman correlation: $r = -0.902$, $P < .001$; Figure 4), indicating that the children who reached GF_{max} with the most consistent delays also exhibited less CS tract dysgenesis.

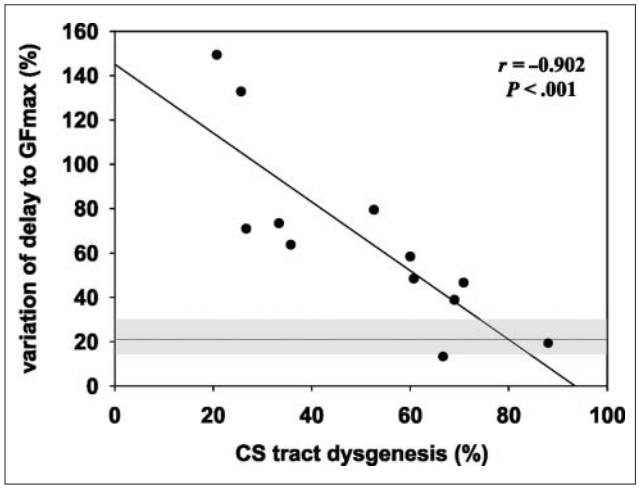

Figure 4. Correlation between the variability of delay to GF_{max} in the paretic hand and corticospinal (CS) tract dysgenesis as measured by diffusion tensor imaging. The gray area represents the 25th to 75th percentiles of the control values for the delay to GF_{max} variability. The CS tract symmetry index of the 12 healthy children was 99% ± 9.3% (mean ± SD)

Influence of Friction and MVC

The CF of the paretic hand of children with CH (0.65 ± 0.21) was lower than that of controls (1 ± 0.29; $ANOVA_{RM}$: $P = .006$), indicating that they had more slippery skin. With more slippery skin, the minimal GF necessary to prevent slipping is higher. Because the GF values during the stable phase and at impact were not different between the children with CH and controls (Table 2), the lower CF led to lower SMs (see Equation 1). Thus, the SMs were significantly lower in the paretic hands during the stable phase and at impact under both anticipation and reactive conditions (all $P < .001$). Moreover, the number of trials during which a "slip" occurred consequently to the impact was significantly higher in the paretic hands of children with CH under

Table 3. Mean Percentage of Maximum Voluntary Contraction[a]

Variables	Values				*P* Value	Post Hocs Analysis		
	CH Paretic, Mean (SD)	CH Nparetic, Mean (SD)	CTRL Ndom, Mean (SD)	CTRL Dom, Mean (SD)	$ANOVA_{RM}$ or Friedman	CH Paretic/ CH Nparetic	CH Paretic/ CTRL Ndom	CH Paretic/ CTRL Dom
Predictive condition (%)								
GF stable phase	27.4 (15.4)	10 (4.1)	10 (4.1)	11 (4.5)	.004[b]	S[b]	S[b]	S[b]
GF at impact	48 (29.7)	16 (8.4)	17 (6.8)	19 (6.8)	<.001[b]	S[b]	S[b]	S[b]
GF_{max}	65 (32.2)	24 (11.1)	26 (10.9)	27 (9.8)	<.001[b]	S[b]	S[b]	S[b]
Reactive condition (%)								
GF stable phase	32 (18.1)	12 (6.0)	11 (4.6)	13 (5.3)	<.001[b]	S[b]	S[b]	S[b]
GF at impact	47 (33.3)	13 (6.3)	11 (4.6)	13 (5.4)	<.001[b]	S[b]	S[b]	S[b]
GF_{max}	71 (30.9)	25 (13.7)	22 (9.7)	26 (10.3)	<.001[b]	S[b]	S[b]	S[b]

Abbreviations: CH, patients with congenital hemiplegic; SD, standard deviation; CTRL, controls; Dom, dominant hand; Ndom, nondominant hand; Nparetic, nonparetic hand; GF, grip force.

[a] Results of Tukey tests (post hoc) are given as *P* values in $ANOVA_{RM}$ and by letters (S = significant; NS = nonsignificant) for Friedman analysis.

[b] Indicates significant difference.

predictive ($P = .022$) and reactive conditions ($P = .003$) as compared with controls.

The MVC exerted on the handheld object was significantly smaller in the paretic hand (15 ± 6.7 N) than in the nonparetic hand (36 ± 15.7 N) and both control hands (31 ± 9 N; all $P < .012$). Because the GF values of the children with CH during the stable phase and at impact were not different from those of controls, the percentage of MVC used to perform the task was significantly higher in the paretic hands of children with CH (Table 3).

Discussion

Under predictive conditions, the postimpact delay was longer in the paretic hand of children with CH compared with the nonparetic hand and with both hands of controls. In a sample of 75 healthy children aged between 6 and 14 years, we showed that when asked to perform a self-imposed impact, young healthy children had a longer postimpact delay compared with older children (Bleyenheuft and Thonnard, unpublished data). This was explained by a less accurate predictive control of the GF increase after impact. In the present study, the later GF increase exhibited by the paretic hand could be explained by planning of the movement that is similar to the immature planning of young children.

The main impairment of children with CH under predictive conditions resides in the delay to GF_{max}, which was also longer and more variable in the paretic hand. This could be explained by an inability to sustain the anticipated motor plan when a dynamic perturbation arises. Indeed, before the impact, no difference was observed between the paretic hand and the hands of controls. After the impact, important differences appeared in the GF trace. The hypothesis that the grip was correctly executed before impact and disrupted afterward could explain why children with CH were unable to adapt the GF to a changing LF in this protocol or even in grip-lift tasks[1-3] but were able to maintain the object under static conditions prior to impact. Because the increase in force after impact is intended to ensure grasp stability,[13] the deficits in motor planning after impact (the longer and more variable delay to GF_{max}) could be partly responsible for the many slips detected in the paretic hand. In the context of the processing of tactile input, these results are quite surprising. In fact, it has been clearly demonstrated that manipulating objects with small GF, as in the present study, requires a high level of sensorimotor integration.[22,23] Central impairments, such as in CP, are therefore likely to affect this sensorimotor integration,[2] which should lead to unadapted levels of force.[2,6] In our experiment, the absence of differences in dynamic variables suggests that the sensorimotor integration of children with CH allows the anticipation of a GF equivalent to that of controls. The temporal deficits observed under predictive conditions, however, suggest that the sensorimotor integration of afferent information from the paretic hand is not adequate to manage the precise timing required for a predictable change in force.

When externally and unpredictably generated, impacts classically induce an increase in GF.[3,8] This increase has been described as a reactive response to the impact and has been called an automatic "long-latency" response.[8] In our

experiment, the postimpact delay was the only significantly disrupted variable under reactive conditions. This postimpact delay, which is purely reactive under these conditions, was significantly longer in the paretic hand of children with CH. This longer delay could be a result of the slower peripheral conduction velocities shown in children with CH for cutaneomuscular reflexes from the First Dorsal Interosseous (FDI).[24]

The findings of the present study allow us to make the following suggestions in relation to the rehabilitation of patients with CH. In this study, we have described deficits in predictive and reactive control in the precision grip of patients with CH. Deficits under reactive conditions were because of a disrupted automatic long-latency response, for which a rehabilitation strategy may not exist. Indeed, although the timing of these automatic responses can be shortened by increasing the preimpact force,[15] patients—already using a high percentage of MVC—are unlikely to develop higher static force in everyday life (too demanding). It would, therefore, be more interesting to focus on the rehabilitation of predictive control. In fact, as brisk load changes are present in everyday life (dog pulling on a leash, heavy bag ripping), it would be interesting to include predictive response to load changes in the rehabilitation programs. Moreover, because we have shown here that this predictive control was unimpaired in the nonparetic hand, training the nonparetic hand first, followed by training the paretic hand, could result in improvement of the predictive control of the movement. Indeed, grip-lift tasks with an alternative use of both hands showed that sensory information from the nonparetic hand could be used to anticipate and scale a following lift with the paretic hand.[5]

Correlation With CS Tract Dysgenesis

Under predictive conditions, the variation in the delay to GF_{max} was correlated with CS dysgenesis. This suggests that the adjustment in the timing to reach GF_{max} is a central process mediated through the CS tract and that an alteration of the CS tract was paralleled by an inconsistent temporal adjustment of this delay. The concept of a central process that adjusts the GF_{max} timing is consistent with our recent data, which described the GF increase after impact as essentially predictive in self-triggered impacts.[9] In children with CH, the larger the lesion in the CS tract, the less consistent the temporal adjustment of this predictive response is after the impact. Under reactive conditions, no correlations were observed between CS tract dysgenesis and the GF increase after impact.

Influence of Coefficient of Friction

The children with CH showed a lower CF between the skin and the object, indicating that they had more slippery skin. With more slippery skin, the minimal GF necessary to prevent slipping is higher. Because the GF of children with CH was equivalent to that of controls, the lower CF led to lower SMs (see the Methods section). This suggests that children with CH were unable to adapt their force to produce a SM equal to that of controls. This may be explained by a lesser ability to develop force.[25] Because the MVC in the paretic hand was significantly lower than that of the hands of controls, the percentage of force used to generate a GF equivalent to that of the controls was much higher (Table 3). In normal adult subjects, contractions of the FDI are considered to be of high intensity when they reach 50% of the MVC.[26] For these high intensities, the achievement of a repetitive position task (supporting an inertial load) leads to an inability to pursue the task in short durations of approximately 40 s.[27] In our patients, the percentage of force used to reach GF_{max} values under both conditions was clearly larger than 50% of the MVC. Under predictive conditions, even the GF at impact reached this value. The children with CH, therefore, may have avoided using a higher GF because it would have led to an inability to pursue the task. Furthermore, even in static phases, the percentage of force used was above the threshold of 20%, which is the maximal value for a low contraction.[27] This lesser ability to develop force could result from many factors. First, the size and limb characteristics of children with CP are substantially smaller for their age, comparable with the values of younger children.[28] As muscle strength develops during growth,[29] which depends primarily on an increase in the diameter and length of muscle fibers,[30] it is probable that the less-developed anatomy of children with CH induces lower maximal forces. Furthermore, independent of age, it has been shown that CP itself generates considerable remodeling of muscles,[31,32] which could affect force development.

Along with the lesser ability to develop force, an alternative explanation for the inability of children with CH to develop SMs equal to controls could be a problem with integrating cutaneous feedbacks. As CF is the criterion used to regulate GF with LF,[33] the integration of frictional properties seems crucial to the performance of precision grip tasks. Children with CH are able to adequately scale their GF to different grip surfaces but only for successive lifts performed under predictable conditions.[3,4] This may be because of deficits in the storage of sensory information.[3] Tasks that require the alternate use of both hands, however, show that sensory information from the nonparetic hand can be used to anticipate and scale a subsequent lift with the paretic hand.[5] This suggests that it is not the storage but the sensorimotor integration that is mainly impaired in the paretic hand.[34] These lower SMs, whatever the underlying mechanism, contributed to the larger number of slips observed in the paretic hands of children with CH.

In conclusion, both predictive and reactive mechanisms are impaired in the paretic hand. Under predictive conditions, temporal alterations appeared after the impact. As this predictive ability to anticipate changes in force was unimpaired in the nonparetic hand, we suggest that alternate training of both

hands, starting with the nonparetic hand, may result in improving the control of the movement. It would be interesting to measure the predictive abilities of children with CH after specific rehabilitation sequences using both hands in an alternate fashion. Moreover, because the main deficits in predictive control are linked to impaired temporal adjustments, it would be interesting to test such rehabilitation sequences with different timing of changes in load.

Acknowledgments

The authors are grateful to Professor Christine Detrembleur for reading over a previous version of this manuscript.

Declaration of Conflicting Interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

This study was supported by a grant from Fonds de la Recherche Scientifique Médicale (FRSM, convention 3.4525.04).

References

1. Eliasson AC, Gordon AM, Forssberg H. Basic co-ordination of manipulative forces of children with cerebral palsy. *Dev Med Child Neurol*. 1991;33:661-670.
2. Eliasson AC, Gordon AM, Forssberg H. Impaired anticipatory control of isometric forces during grasping by children with cerebral palsy. *Dev Med Child Neurol*. 1992;34: 216-225.
3. Eliasson AC, Gordon AM, Forssberg H. Tactile control of isometric fingertip forces during grasping in children with cerebral palsy. *Dev Med Child Neurol*. 1995;37:72-84.
4. Gordon AM, Duff SV. Fingertip forces during object manipulation in children with hemiplegic cerebral palsy. I: anticipatory scaling. *Dev Med Child Neurol*. 1999;41:166-175.
5. Gordon AM, Charles J, Duff SV. Fingertip forces during object manipulation in children with hemiplegic cerebral palsy. II: bilateral coordination. *Dev Med Child Neurol*. 1999;41: 176-185.
6. Forssberg H, Eliasson AC, Redon-Zouitenn C, Mercuri E, Dubowitz L. Impaired grip-lift synergy in children with unilateral brain lesions. *Brain*. 1999;122(pt 6):1157-1168.
7. Duque J, Thonnard JL, Vandermeeren Y, Sébire G, Cosnard G, Olivier E. Correlation between impaired dexterity and corticospinal tract dysgenesis in congenital hemiplegia. *Brain*. 2003;126(pt 3):732-747.
8. Johansson RS, Westling G. Programmed and triggered actions to rapid load changes during precision grip. *Exp Brain Res*. 1988;71:72-86.
9. Bleyenheuft Y, Lefèvre P, Thonnard JL. Predictive mechanisms control grip force after impact in self-triggered perturbations *J Mot Behav.* 2009;41(5):411-7.
10. Turrell YN, Li FX, Wing AM. Grip force dynamics in the approach to a collision. *Exp Brain Res*. 1999;128:86-91.
11. Witney AG, Goodbody SJ, Wolpert DM. Predictive motor learning of temporal delays. *J Neurophysiol*. 1999;82:2039-2048.
12. Eliasson AC, Forssberg H, Ikuta K, Apel L, Westling G, Johansson R. Development of human precision grip. V. Anticipatory and triggered grip actions during sudden loading. *Exp Brain Res*. 1995;106:425-433.
13. Delevoye-Turrell YN, Li FX, Wing AM. Efficiency of grip force adjustments for impulsive loading during imposed and actively produced collisions. *Q J Exp Psychol A.* 2003;56: 1113-1128.
14. Nowak DA, Hermsdorfer J. Predictive and reactive control of grasping forces: on the role of the basal ganglia and sensory feedback. *Exp Brain Res.* 2006;173:650-660.
15. Serrien DJ, Kaluzny P, Wicki U, Wiesendanger M. Grip force adjustments induced by predictable load perturbations during a manipulative task. *Exp Brain Res.* 1999;124:100-106.
16. Porter R, Lemon R. *Corticospinal Function and Voluntary Movement*. Oxford, UK: Clarendon; 1993.
17. Bleyenheuft Y, Grandin CB, Cosnard G, Olivier E, Thonnard JL. Corticospinal dysgenesis and upper-limb deficits in congenital hemiplegia: a diffusion tensor imaging study. *Pediatrics.* 2007;120:e1502-e1511.
18. Palisano R, Rosenbaum P, Walter S, Russell D, Wood E, Galuppi B. Gross motor function classification system for cerebral palsy. *Dev Med Child Neurol*. 1997;39:214-223.
19. Westling G, Johansson RS. Factors influencing the force control during precision grip. *Exp Brain Res.* 1984;53:277-284.
20. Pajevic S, Pierpaoli C. Color schemes to represent the orientation of anisotropic tissues from diffusion tensor data: application to white matter fiber tract mapping in the human brain. *Magn Reson Med.* 1999;42:526-540.
21. Virta A, Barnett A, Pierpaoli C. Visualizing and characterizing white matter fiber structure and architecture in the human pyramidal tract using diffusion tensor MRI. *Magn Reson Imag.* 1999;17:1121-1133.
22. Johansson RS. Sensory control of dexterous manipulation in humans. In: Wing AM, Haggard P, Flanagan JR, eds. *Hand and Brain: The Neurophysiology and Psychology of Hand Movements*. San Diego, CA: Academic Press; 1996:381-414.
23. Ehrsson HH, Fagergren A, Forssberg H. Differential frontoparietal activation depending on force used in a precision grip task: an fMRI study. *J Neurophysiol.* 2001;85:2613-2623.
24. Evans AL, Harrison LM, Stephens JA. Cutaneomuscular reflexes recorded from the first dorsal interosseous muscle of children with cerebral palsy. *Dev Med Child Neurol*. 1991;33:541-551.
25. Smits-Engelsman BC, Rameckers EA, Duysens J. Muscle force generation and force control of finger movements in children with spastic hemiplegia during isometric tasks. *Dev Med Child Neurol*. 2005;47:337-342.
26. De Luca CJ, LeFever RS, McCue MP, Xenakis AP. Behaviour of human motor units in different muscles during linearly varying contractions. *J Physiol*. 1982;329:113-128.
27. Maluf KS, Shinohara M, Stephenson JL, Enoka RM. Muscle activation and time to task failure differ with load type and

contraction intensity for a human hand muscle. *Exp Brain Res.* 2005;167:165-177.
28. Kong CK, Tse PW, Lee WY. Bone age and linear skeletal growth of children with cerebral palsy. *Dev Med Child Neurol*. 1999;41:758-765.
29. Beenakker EA, van der Hoeven JH, Fock JM, Maurits NM. Reference values of maximum isometric muscle force obtained in 270 children aged 4–16 years by hand-held dynamometry. *Neuromuscul Disord*. 2001;11:441-446.
30. Aherne W, Ayyar DR, Clarke PA, Walton JN. Muscle fibre size in normal infants, children and adolescents: an autopsy study. *J Neurol Sci*. 1971;14:171-182.
31. Fridén J, Lieber RL. Spastic muscle cells are shorter and stiffer than normal cells. *Muscle Nerve*. 2003;27:157-164.
32. Ito J, Araki A, Tanaka H, Tasaki T, Cho K, Yamazaki R. Muscle histopathology in spastic cerebral palsy. *Brain Dev*. 1996;18:299-303.
33. Cadoret G, Smith AM. Friction, not texture, dictates grip forces used during object manipulation. *J Neurophysiol.* 1996;75: 1963-1969.
34. Gordon AM, Charles J, Steenbergen B. Fingertip force planning during grasp is disrupted by impaired sensorimotor integration in children with hemiplegic cerebral palsy. *Pediatr Res*. 2006;60:587-591.