"Impaired predictive and reactive control of precision grip in chronic stroke patients"

Dispa, Delphine ; Thonnard, Jean-Louis ; Bleyenheuft, Yannick

Abstract

Skilled hand movements require a precise coordination between the grip force and the load force. To coordinate those forces, we rely on both a predictive and a reactive control. On the basis of specific impairments observed previously in children with hemiplegic cerebral palsy, we aimed to assess the predictive or/and reactive nature of hand deficits in stroke patients. This case–control study was carried out with eight stroke patients and eight control participants. The load of a handheld object was rapidly increased by dropping a mass attached to the object. We tested predictive and reactive aspects of the movement in the same task as the drop was triggered either unexpectedly by the examiner (reactive condition) or by the patient himself (predictive condition). Deficits observed in the paretic hand were similar to those highlighted previously in children with hemiplegic cerebral palsy. Under predictive conditions, temporal deficits were observed after impact. Under reactive cond...

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Skilled hand movements require a precise coordination between the grip force and the load force. To coordinate those forces, we rely on both a predictive and a reactive control. On the basis of specific impairments observed previously in children with hemiplegic cerebral palsy, we aimed to assess the predictive or/and reactive nature of hand deficits in stroke patients. This case–control study was carried out with eight stroke patients and eight control participants. The load of a handheld object was rapidly increased by dropping a mass attached to the object. We tested predictive and reactive aspects of the movement in the same task as the drop was triggered either unexpectedly by the examiner (reactive condition) or by the patient himself (predictive condition). Deficits observed in the nonparetic hand were similar to those highlighted previously in children with hemiplegic cerebral palsy. Under predictive conditions, temporal deficits were observed after impact. Under reactive conditions, the reflex latency was slightly increased in the paretic hand. The nonparetic hand showed similar results to controls. The predictive mechanism is present but altered in the paretic hand. These alterations suggest an inability to anticipate the consequences of dynamic perturbations in the paretic hand only.


Las destrezas manuales requieren coordinación precisa entre la fuerza de prensión y la fuerza de carga. Para coordinar dichas fuerzas se utiliza tanto un control predictivo como un control reactivo. Partiendo de las discapacidades específicas previamente observadas en niños con parálisis cerebral hemipléjica, el objetivo de este estudio fue evaluar la naturaleza predictiva y/o reactiva de las limitaciones manuales de pacientes con ictus. En este estudio de control de casos participaron ocho pacientes con ictus y ocho participantes de control. Se llevó a cabo un aumento rápido de la carga de un objeto portátil al añadir peso a dicho objeto. Durante la misma prueba, se analizaron los aspectos predictivos y reactivos del movimiento conforme el peso aumentaba bien de forma inesperada por parte del examinador (condición reactiva) o bien por parte del paciente (condición predictiva). Las limitaciones observadas en la mano parética fueron similares a las mencionadas previamente en niños con parálisis cerebral hemipléjica. Bajo condiciones predictivas, las limitaciones temporales fueron observadas después del impacto. Bajo condiciones reactivas, la latencia refleja aumentó ligeramente en la mano parética. La mano no parética presentó resultados similares a los del grupo control. El mecanismo predictivo se mantuvo presente pero alterado en la mano parética. Dichas alteraciones sugieren la incapacidad de anticipar las consecuencias de las perturbaciones dinámicas en la mano parética solamente.

Les mouvements habiles de la main nécessitent une coordination précise entre la force de préhension et la force de charge. Pour coordonner ces forces, nous devons appliquer un contrôle à la fois prédictif et réactif. Sur la base des troubles spécifiques observés précédemment chez les enfants atteints de paralysie cérébrale hemiplégique, nous avons cherché à évaluer le caractère prédictif et/ou réactif des déficiences de la main chez les patients victimes d'AVC. Cette étude de cas-témoins a été réalisée avec huit patients victimes d'AVC et huit participants de contrôle. La charge d’un objet tenu à la main a été augmentée rapidement en laissant tomber une masse attachée à l’objet. Nous avons testé les aspects prédictifs et réactifs du mouvement au cours de la même
tâche, la chute étant déclenchée soit de manière inattendue par l'examineur (condition réactive) ou par le patient lui-même (condition prédictive). Les déficiences observées dans la main paretique étaient similaires à celles précédemment mises en évidence chez les enfants atteints de paralysie cérébrale hémplégique. Dans des conditions prédictives, des déficiences temporelles étaient observées après l'impact. Dans des conditions réactives, la latence du réflexe présentait une légère augmentation pour la main paretique. La main non paretique présentait des résultats similaires à ceux des contrôle. Le mécanisme prédictif est présent mais modifié dans la main paretique. Ces altérations suggèrent une incapacité à anticiper les conséquences des perturbations dynamiques dans la main paretique uniquement. International Journal of Rehabilitation Research

Introduction

The manipulation of small objects between the thumb and the index finger requires a precise coordination between the grip force (GF) and the tangential load force (LF) (Johansson and Westling, 1984, 1988). This coordination of forces relies on two types of control mechanisms: a predictive control that allows healthy individuals to anticipate movement on the basis of sensorimotor memory and a reactive control that enables correction of the movement through feedback. An internal model in the central nervous system has been suggested to account for the predictive mechanisms (Wolpert and Ghahramani, 2000). Stroke patients usually do not present normal skilled hand movements. They typically show excessive GF and large perturbations in movement timing (Hermsdörfer et al., 2003; Nowak et al., 2003; Takahashi and Reinkensmeyer, 2003; McDonnell et al., 2006; Raghavan et al., 2006). However, it is not clear whether their impairments in fine prehension are linked to deficits in predictive or reactive control of the movement. The excess of GF has generally been associated with perturbations in sensory feedback and the disordered timing of the movement is currently attributed to deficits in internal models (Nowak et al., 2003; Takahashi and Reinkensmeyer, 2003). However, perturbations in the timing of the movement could also be because of altered sensory feedback transmission that could prevent updating of the sensorimotor memory. Therefore, it would be interesting to determine the relative contributions of predictive and reactive mechanisms to deficits in precision grip using paradigms in which both can be tested separately.

Such a paradigm has been used for stroke patients in the context of anticipatory postural adjustments of the arm (Bennis et al., 1996), but never to assess the subtle coordination of forces required to carry out precision grip tasks. In children with hemiplegic cerebral palsy, both predictive and reactive control have been studied previously, showing impairments in the delays related to both predictive and reactive control (Bleyenheuft and Thonnard, 2010a). In this study, the same paradigm, using the brisk loading of a handheld object (under predictable or unpredictable conditions), will be used to investigate the predictive or/and reactive nature of hand deficits in the paretic and nonparetic hands of chronic stroke patients.

Participants and methods

This study was authorized by the Ethical Committee of the Université catholique de Louvain, Faculty of Medicine in Brussels, Belgium. Eight stroke patients (three women, mean age: 54.5±11.0) with no cognitive deficits (> 26/30 on the Mini-Mental State Examination) and eight-matched controls (54.5±10.5) were assessed. Hemiplegia level was categorized using the Stroke Impairment Assessment Scale. A brief description of the patients is provided (Table 1).

Apparatus

A cylindrical object (80 mm diameter, 220 g) with two parallel force–torque sensors was used. Each sensor provided values of GF and LF, calculated from the three force components \(F_x\), \(F_y\), and \(F_z\). 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. The horizontal (x) and vertical (y) centers of pressure were also measured. The object was placed on an open table (Fig. 1) and a steel mass (100 g) was attached to the object by a Kevlar string. The additional mass could be placed on an electromagnet located a few centimeters above its lowest position, making it possible to lift the object without any influence of the additional mass.

Procedure and experimental protocol

Participants sat next to a table providing support to their forearm. They were presented with an object and instructed to grasp it and hold it in a standard position.
Three different conditions (predictive, unexpected blank, and reactive condition) were tested.

For predictive conditions, participants held a button switch in their free hand, which they pressed in response to an auditory signal. This instantly turned off the magnetic field, which caused the mass to drop (4 cm), followed by a sudden increase in LF (impact).

Blank conditions were similar to the predictive conditions at the beginning, but the release mechanism was unexpectedly blocked, meaning that no drop occurred.

Under reactive conditions, the drop of the mass applied to the handheld object was both sudden and unpredictable because the release mechanism was triggered by the examiner.

The dominant hand of control participants and both hands of stroke patients were tested systematically, beginning with the paretic hand of stroke patients. The use of only one hand in the healthy participants was justified by the absence of difference between both hands of controls in a previous study (Bleyenheuft and Thonnard, 2010a). Each participant performed 35 consecutive trials for each hand according to the following sequence: 15 predictive trials, five blank trials, five predictive trials, and 10 reactive trials. The consecutive presentation of the trials in each block allowed us to study the evolution of the motor response within each condition (stimulation predicted, no stimulation, stimulation not predicted). The participants were unaware that a transition between blank and impact trials would occur. As a consequence, trials 1, 16, and 21 were considered catch trials.

The coefficient of friction (CF) was measured through eight lift-and-drop maneuvers, which preceded and directly followed the experiment (Bleyenheuft and Thonnard, 2010a).

### Data acquisition and analysis

The signals from the force sensors were digitized on-line at 1000 Hz using a 12-bit6071E analog-to-digital converter in a PXI chassis (NI, Austin, Texas, USA). After analog-to-digital conversion, the GF and GFrate signals were further low-pass filtered with a fourth-order, zero phase-lag Butterworth filter with a cutoff frequency of 25 Hz.

The impact phase, defined as the period including the impact time and the modulation of GF preceding and following the impact, was analyzed using the following temporal variables (Fig. 2):
Anticipatory delay – the delay between the onset of GF and the impact.

Delay postimpact – the delay between the impact and the increase in GF after the impact.

Delay to GFmax – the delay between the impact and the GFmax.

In addition, dynamic variables were investigated during the impact phase: GF at impact, and GFmax and GFratermax before and after impact (Fig. 2). An average GF was also calculated in each trace during the stable phase defined visually before the impact on the LF trace.

Traces from a control and a stroke patient. Examples of GF, GFrater, and LF traces recorded under predictive and reactive conditions from a control and a stroke patient. In each trace, vertical dotted lines represent the time points used to calculate the different delays. a is the anticipatory delay, b is the delay after impact, and c is the delay to GFmax. The short vertical bar under the GF traces represents the moment the participant pressed the button switch. The auditory cue is not represented here as it arises previously. In the predictive condition, stroke patient trace presents a small slip, inducing a difference of 2 mm between the center of pressure before and after impact. This slip is shown in this example by a slight decrease in GF and a negative GFrater at the moment of the impact. However, as no drop of the object was observed, this was not considered as a failure in the task. GF, grip force; LF, load force.
The estimate of the impact occurrence ($t_0$) in blank trials was computed by calculating an average delay between switch and impact for each participant on all impact trials. In blank trials, the average delay for each participant (~200 ms) was added to the moment the participant pressed the switch, providing an estimate of impact occurrence.

For each trial, GFratemax, GFmax, and the impact (LFmax) were detected as the absolute maxima during the impact phase.

The number of slips was counted in both the predictive and the reactive trials. A slip was identified during the impact phase when the displacement of the vertical component of the center of pressure ($y$) was higher than 5 mm.

**Statistical analysis**

Previous studies (Bleyenheuft and Thonnard, 2010a, 2010b) showed that one trial was sufficient to obtain stable values for all variables studied. Therefore, in subsequent analysis, mean values excluded the first trial of each sequence.

ANOVA (or the Kruskal–Wallis test for nonparametric conditions) was performed to compare the three groups of data (paretic and nonparetic hands of stroke patients and dominant hand of control participants) in each condition (predictive and reactive). A Tukey pairwise multiple-comparison procedure, including an automatic $P$-value correction, determined which treatments were significantly different.

A repeated-measure ANOVA on ranks was performed on the first 25 trials of all participants to detect trial-to-trial differences as well as changes because of blank trials in the sequence. This analysis was carried out separately for data from each condition. Post-hoc analysis was carried out using Tukey tests.

**Results**

**Predictive and reactive conditions**

Figure 2 shows typical traces from trials involving a control participant (a) and trials in which the paretic hand of a stroke patient (b) was tested under predictive and reactive conditions.

As described previously (Bleyenheuft et al., 2009), under predictive conditions, control participants showed an increase in GF that preceded the impact and a second GF increase (that led to maximum GF) after the impact. Under reactive conditions, the GF of the control participant was stable before the impact. A rapid GF increase that led to GFmax was induced by the impact.

In stroke patients, under predictive conditions, the paretic hand showed an anticipatory delay that was similar to those observed in control participants, but the GFratemax was significantly lower than that of control participants. The postimpact increase in GF that leads to GFmax is also present in the paretic hand, but both the onset and the maximum occur after a longer delay than in controls. As expected, under reactive conditions, stroke patients presented a rapid GF increase that followed the impact. In the paretic hand, this elevation in GF had a slightly later onset but reached GFmax within a similar time to controls.

In the nonparetic hand, the variables measured were similar to those observed in the controls (Table 2).

Table 2 summarizes the mean values of the variables in stroke patients and control participants. Under predictive conditions, there were significant differences between the paretic hand of stroke patients and control participants in the defined primary variables. First, the postimpact delay was significantly longer in the paretic hand of stroke patients. Second, the delay to GFmax was more prolonged and showed greater variability (coefficient of variation: 851±215%, mean±SD) in the paretic hands of stroke patients than in either controls (36±11%) or the nonparetic hands of patients (29±23%). This indicated an inconsistent (less regular) temporal adjustment in reaching the GFmax under predictive conditions (Kruskal–Wallis, $H = 14.2$, 2 d.f., $P < 0.001$). In addition, before the time of impact, the GFratemax was significantly reduced in the paretic hand of stroke patients. Post-hoc analysis showed that the nonparetic hand did not present significant differences with the dominant hand of controls.

Under reactive conditions, the postimpact delay was significantly longer in the paretic hand of stroke patients than in controls. This delay was also more variable in the paretic hand (coefficient of variation: 36±19%) than in controls (21±12%). Neither the delay to GFmax (Table 2) nor the variability (Kruskal–Wallis, $H = 2$, 2 d.f., $P = 0.369$) were significantly increased in the paretic hand of stroke patients compared with controls.

The CF of stroke patients were not significantly different from those of controls (ANOVARM, $P = 0.925$). The number of trials during which a slip occurred was significantly higher in the paretic hand of stroke patients (11.5±11% of the trials) when compared with control values (4±4.3%), but only under predictive conditions (Kruskal–Wallis, $H = 7.74$, 2 d.f., $P = 0.021$). Post-hoc analysis showed that the percentage of slips on the nonparetic hand (5.5±6.7%) did not differ from control values.

**The use of blank trials**

**Controls**

As shown in Fig. 3a, the mean GFmax was significantly lower during all blank trials, except on the first (ANOVARM, $F = 7.8$, 24 d.f., $P < 0.001$). This first blank
trial (trial 16) was not significantly different from the preceding impact trials (Tukey test; $P > 0.05$). The second blank trial (17) was significantly different from trials 2 to 7. The third blank trial (18) was significantly different from trials 2 to 15. The fourth and fifth blank trials (trials 19 and 20) were significantly different from trials 2 to 15 and 21 to 25 ($P < 0.05$).

The delay between the impact and GF\textsubscript{max} was not significantly different between impact trials (160±29 ms) and blank trials (145±34 ms). The very first trial tended to present a longer delay (287±158 ms; Friedman analysis, $\chi^2 = 34.3$, 24 d.f., $P = 0.079$).

**Paretic hand**

Surprisingly, the mean GF\textsubscript{max} of paretic hands (Fig. 3b) was not significantly lower during blank trials (ANOVA, $F = 1.2$, 24 d.f., $P = 0.279$). Because of the large intraparticipant and interparticipant variability, there was no significant difference in the delay to GF\textsubscript{max} in impact trials (278±116 ms) compared with blank trials (~49±365 ms). During blank trials, there were typically shorter delays to GF\textsubscript{max} or even negative delays to GF\textsubscript{max} (GF\textsubscript{max} occurred before the expected impact).

**Nonparetic hand**

The results obtained from the nonparetic hand of patients were similar to those of controls. For example, the mean GF\textsubscript{max} (Fig. 3c) was significantly lower during all blank trials (ANOVA, $F = 2.9$, 24 d.f., $P < 0.001$), except on the first two trials (trials 16 and 17). These initial blank trials were not significantly different from the preceding impact trials (Tukey test; $P > 0.05$). Trials 18–20 were significantly different from other impact trials ($P < 0.05$). As in controls, the delay between the impact and GF\textsubscript{max} was not significantly different for impact trials (186±58 ms) compared with blank trials (175±101 ms; Friedman analysis, $\chi^2 = 34.3$, 24 d.f., $P = 0.501$). The very first impact trial did not present a longer delay.

**Discussion**

In this study, we investigated whether impaired precision grip of stroke patients resulted from deficits in the ability to anticipate movements and/or to perturbations in reactive loops. Significant perturbations of predictive regulation were observed for the delay to GF\textsubscript{max}, the postimpact delay. In reactive control, deficits were limited to the postimpact delay. Under predictive conditions, the GF\textsubscript{rate,max} was also altered and more slips were observed in the paretic hand of stroke patients. In addition, the nonparetic hand showed performances similar to controls under both predictive and reactive conditions.

The longer postimpact delay under both conditions as well as the lower GF\textsubscript{rate,max} of the paretic hand were likely linked to muscular modifications. It is well known that many patients with stroke show muscle weakness (Bohannon, 2007). This weakness could be linked to a loss of functioning motor units (Arasaki et al., 2006) or to a selective affectionation of the large motor units with a high threshold (Lukács et al., 2008). These muscular alterations are likely to affect the development of force as the recruitment of new motor units according to increasing size is one of the mechanisms used to increase one’s force output (Henneman and Olson, 1965; Henneman et al., 1965a, 1965b).

Muscular modifications are also most likely responsible for the longer postimpact delay under both conditions. This delay is either wholly (under predictive conditions) or partly (under predictive conditions) because of the latency of a stretch reflex induced by the impact. In view of the defined order with which motor units that

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**Table 2** Mean values of dynamic and temporal variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>Stroke paretic</th>
<th>Stroke nonparetic hand</th>
<th>CTRL</th>
<th>$P$-value ANOVA or Kruskal–Wallis</th>
<th>Post-hoc tests ($P$-value)</th>
</tr>
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<tr>
<td></td>
<td>GF\textsubscript{stable phase} (N)</td>
<td>11.2 (5.84)</td>
<td>10.7 (4.17)</td>
<td>8.3 (4.41)</td>
<td>0.225</td>
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<tr>
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<td>GF at impact (N)</td>
<td>12.8 (6.58)</td>
<td>15.7 (4.90)</td>
<td>15.4 (6.39)</td>
<td>0.575</td>
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<tr>
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<td>GF\textsubscript{max} (N)</td>
<td>16.1 (6.63)</td>
<td>22.2 (6.17)</td>
<td>21.7 (6.71)</td>
<td>0.140</td>
</tr>
<tr>
<td></td>
<td>GF\textsubscript{rate,max} before impact (N/s)</td>
<td>15 (9.6)</td>
<td>30 (23.3)</td>
<td>38 (16.5)</td>
<td>0.045$^*$</td>
</tr>
<tr>
<td></td>
<td>GF\textsubscript{rate,max} after impact (N/s)</td>
<td>69 (45.2)</td>
<td>118 (49.5)</td>
<td>108 (43.4)</td>
<td>0.106</td>
</tr>
<tr>
<td></td>
<td>D (ms)</td>
<td>359 (30)</td>
<td>329 (75)</td>
<td>376 (82)</td>
<td>0.185</td>
</tr>
<tr>
<td></td>
<td>D (ms)</td>
<td>71 (22)</td>
<td>51 (9)</td>
<td>43 (7)</td>
<td>0.003$^*$</td>
</tr>
<tr>
<td></td>
<td>D to GF\textsubscript{max} (ms)</td>
<td>283 (150)</td>
<td>186 (58)</td>
<td>160 (29)</td>
<td>0.001$^*$</td>
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<tr>
<td><strong>Predictive condition</strong></td>
<td>GF\textsubscript{stable phase} (N)</td>
<td>11.9 (5.82)</td>
<td>11.2 (4.78)</td>
<td>10.1 (5.41)</td>
<td>0.505</td>
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<tr>
<td></td>
<td>GF at impact (N)</td>
<td>12.1 (5.41)</td>
<td>11.5 (5.14)</td>
<td>10.6 (5.96)</td>
<td>0.862</td>
</tr>
<tr>
<td></td>
<td>GF\textsubscript{max} (N)</td>
<td>17.2 (7.22)</td>
<td>20.8 (7.61)</td>
<td>21.6 (5.94)</td>
<td>0.475</td>
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<tr>
<td></td>
<td>GF\textsubscript{rate,max} after impact (N/s)</td>
<td>85 (22.8)</td>
<td>117 (41.9)</td>
<td>144 (42.4)</td>
<td>0.073</td>
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<tr>
<td></td>
<td>D postimpact (ms)</td>
<td>71 (18)</td>
<td>57 (13)</td>
<td>52 (11)</td>
<td>0.028$^*$</td>
</tr>
<tr>
<td></td>
<td>D to GF\textsubscript{max} (ms)</td>
<td>266 (98)</td>
<td>243 (66)</td>
<td>230 (34)</td>
<td>0.596</td>
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<tr>
<td><strong>Reactive condition</strong></td>
<td>GF\textsubscript{stable phase} (N)</td>
<td>11.9 (5.82)</td>
<td>11.2 (4.78)</td>
<td>10.1 (5.41)</td>
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Results of Tukey tests (post hoc) were given as $P$-values in ANOVA and by letters (S, significant; NS, nonsignificant) for Kruskal–Wallis test. ANOVA, analysis of variance; CTRL, control participants; D, delay.

*Significant difference.
innervate different types of fibers are recruited (Calancie and Bawa, 1984), the selective affection of large, high-threshold motor units (Lukács et al., 2008) could provide an explanation for the increased lag in the postimpact delay under both conditions. However, the longer and more variable delay to reach \( \text{GF}_{\text{max}} \) cannot be related to these muscular modifications. A purely muscular phenomenon should have induced the same perturbations under both predictive and reactive conditions and no changes in the delay to \( \text{GF}_{\text{max}} \) could be
identified under purely reactive conditions. Therefore, this delay is likely to be impaired under predictive conditions because of higher-order perturbations. It has been shown recently in self-triggered impulsive loading tasks that the increase in GF arising after impact is intrinsically of a predictive nature (Bleyenheuft et al., 2009). This last part of the trace would be planned in advance: a moderate GF at impact would be used to dampen the collision and an increase in force would be developed afterwards to stabilize the object. The different time taken to reach $GF_{\text{max}}$ in the paretic hand is thus evidence of an important perturbation of predictive control in the paretic hand of stroke patients. This is further supported by the variability in the temporal adjustment of this delay, which indicates either an inability to reproduce a motor plan or an inability to form it in the first place. The disordered nature of this delay was probably responsible for the larger number of slips observed in the paretic hand of stroke patients under predictive conditions. Interestingly, slips were no more prevalent in the paretic hand under reactive conditions. The hypothesis of a high-order motor planning deficit under predictive conditions is further supported by the lack of decrease in $GF_{\text{max}}$ for blank trials completed with the paretic hand. On the paretic hand, patients were not able to regulate the amplitude of $GF_{\text{max}}$ to the different conditions. This deficit in GF regulation and in the timing to reach the maximum (D to $GF_{\text{max}}$) strongly suggests impairments in predictive control. Altogether, these results, acquired in chronic patients with cortical and subcortical lesions, are consistent with high-order motor planning deficits in skilled hand movements, probably because of deficits in the implementation of internal models. This is consistent with previous studies in stroke patients. In acute stroke patients performing point-to-point movement with handheld objects, deficits were observed in prediction of the inertial load profile (Nowak et al., 2003). Identical perturbations were shown by patients with cortical and subcortical lesions, suggesting that an internal model responsible for the precise regulation of forces was perturbed. Although internal models are believed to be formed in the cerebellum (Wolpert et al., 1998; Wolpert and Flanagan, 2001), the authors suggested that cortical and subcortical structures could be involved in the subsequent processing of motor commands. The ability to learn anticipation has also been studied in chronic patients by applying forces to the patient’s arm while they tried to reach a target (Takahashi and Reinkensmeyer, 2003). It was also concluded that implementation of internal models is impaired in patients with cortical and subcortical lesions, although an incomplete ability to form and use internal models remains. It is of great interest that the high-order motor planning deficits observed in the paretic hand of stroke patients with subcortical problem can be corrected by transferring information from the unaffected hand – at least in right hemiparesis (Raghavan et al., 2006).

This last study is of particular interest as we show here that the management of a rapid increase in forces is preserved by the nonparetic hand of stroke patients. The nonparetic hand is thus likely to be used to form a correct internal model with the relevant information being used for the benefit of the paretic hand. This argues in favor of an alternate use of both hands in rehabilitation programs starting with the nonparetic hand to implement a correct planning of movement in the paretic hand. Interestingly, this is reinforced by the consistency of these results with previous results obtained in the same task for children with hemiplegic cerebral palsy (Bleyenheuft and Thonnard, 2010a). The potential use of the nonparetic hand to form correct internal models is further supported by the results obtained when performing blank trials. Similar to healthy control participants, the nonparetic hand of stroke patients showed both an ability to adapt the amplitude of $GF_{\text{max}}$ as a function of previous trials and also constancy in the delay to reach $GF_{\text{max}}$, which indicated the predictive nature of this late GF increment (Bleyenheuft et al., 2009). In contrast, such predictive planning could not be observed in the paretic hand, as proven by the lack of adaption of $GF_{\text{max}}$ to previous trials and the high variability of the delay to reach $GF_{\text{max}}$ under both impact and blank conditions.

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Conflicts of interest
There are no conflicts of interest.

References


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</tr>
</tbody>
</table>