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Constraint therapy versus intensive training: Implications for motor control and brain plasticity after stroke

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Constraint therapy versus intensive training: Implications for motor control and brain plasticity after stroke

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Many studies have demonstrated that constraint induced movement therapy (CIMT) improves upper limb motor impairment following stroke. This rehabilitation method combines constraint of the less-affected upper limb with intensive training of the paretic limb. The aim of the present study was to evaluate, in a single case study, the respective effects of each of these two therapeutic interventions. The patient selected was a 32-year-old right-handed woman. Three and a half years prior to inclusion, she suffered a left capsular infarct responsible for a right hemiparesis. Several assessments were carried out before and after constraint therapy and then after intensive training. Each assessment included measures of hand function as well as a three-dimensional (3D) analysis of prehension. Results showed a significant improvement of motor performance after the constraint period and an additional amelioration after the intensive training period. Kinematic analysis showed that the transport...
phase of movement (movement time and velocity peaks) was improved after the constraint period, whereas the grasping phase (maximum grip aperture) was modified after intensive training. These data could reflect a specific effect of treatment on each phase of the prehension task, or a more general proximal-to-distal gradient of recovery. Although firm conclusions are not warranted on the basis of this single case study, we confirm the utility of 3D motion analysis to evaluate objectively the effectiveness of a therapeutic intervention. We also discuss the implications of our findings for understanding processes of motor control reorganisation.

**Keywords:** Hemiplegia; Stroke; Constraint induced therapy; Cerebral plasticity; 3D kinematic analysis.

### INTRODUCTION

Each year, 130 000 new strokes are diagnosed in France (Bejot et al., 2007). The incidence of motor impairment after stroke is approximately 55–75% with severe negative repercussions on activities of daily living (ADL) and on quality of life, especially when the dominant limb is affected (Nakayama, Jørgensen, Raaschou, & Olsen, 1994). Among existing rehabilitation methods to facilitate upper limb recovery, constraint induced movement therapy (CIMT) has been extensively studied in recent years.

In humans, there is a high level of evidence supporting a therapeutic effect of this rehabilitation method. Several randomised controlled studies have shown lasting improvements in motor impairments and in ADL following CIMT compared with conventional rehabilitation (for a review see Bonaiuti, Rebasti, & Sioli, 2007). This was recently demonstrated in a large randomised controlled trial in which 222 patients were included (Wolf et al., 2006). In this study, as in most other studies evaluating this method, the protocol combined constraint of the less affected limb for 90% of waking hours with intensive rehabilitation of the paretic upper limb 6 hours per day over a period of 2 weeks (2 × 5 days). Although several variations of this protocol have been used with differences in duration of the constraint or in the duration of the intensive training periods (Page, Sisto, Levine, & McGrath, 2004; Sterr et al., 2002), no study has evaluated the respective individual effects of the constraint and the intensive training. The type of intensive training, irrespective of constraint, has been shown to play a role in functional recovery and argues for the importance of including functional motor activities in the rehabilitation programme (Lin, Wu, Liu, Chen, & Hsu, 2009).

From a theoretical point of view, CIMT is based on two principles (Taub, Uswatte, & Elbert, 2002): (i) To overcome learned non-use of the paretic limb by constraining movement of the less affected limb; and (ii) to facilitate motor recovery through intensive training of the paretic limb. Intracortical
microstimulation mapping in primates has shown that motor skill training can alter primary motor cortex organisation, with expansion or contraction of representations of different upper limb joints depending on the task trained (Nudo, Milliken, Jenkins, & Merzenich, 1996). Therefore, the two components of CIMT could involve different mechanisms, which could in turn have specific effects on motor recovery. The aim of this study was to determine the respective effects of the constraint therapy and of the intensive training.

METHOD

We performed a single case study with repeated evaluations after each rehabilitation period: (i) constraint of the less affected limb and (ii) intensive training of the paretic limb, respectively (see Figure 1).

Case description

We used the same inclusion criteria as those employed by Wolf et al. (2006). In order to ensure that motor performance was stable before beginning the study, two further inclusion criteria were added: (i) the delay after stroke had to be more than one year; and (ii) motor performance had to be stable on two pre-test sessions 15 days apart using the upper limb section of the Fugl-Meyer Scale.

Our patient was a 32-year-old right-handed woman. In 2004, she sustained a right hemiplegia following a lacunar infarct of the posterior limb of the left internal capsule. Following stroke, the patient received conventional rehabilitation as an in-patient on a neurological rehabilitation ward. After discharge home, she received twice weekly physiotherapy, which mostly consisted of passive mobilisation of the upper and lower limbs. She was unable to resume her previous work as a care assistant.

She was assessed 3.5 years after stroke. The clinical examination found right-sided hemiparesis, which was purely motor and consistent with an upper motor neuron lesion with brisk, diffuse osteotendinous reflexes. Her right upper limb was weak and finger movements were slow and lacked selectivity. Flexion-extension movements of wrist and fingers were possible but their active amplitude was limited to approximately 20°. There was moderate spasticity of the shoulder internal rotators with a score of 1 on the modified

Figure 1. Experimental design.
Ashworth scale (MAS); elbow flexors (MAS = 1), elbow extensors (MAS = 1+), wrist flexors (MAS = 1+), pronators (MAS = 1+) and finger flexors (MAS = 1). There was no sensory deficit. Passive amplitudes were normal. Only gross-type prehension movements were possible. She was unable to carry out certain bimanual tasks or tasks requiring force. Most ADL could be performed accurately but slowly. However, she remained dependant for grooming (to style her hair), feeding (to cut meat), bathing (to wash her back), dressing (to put on her bra and to lace up shoes), and showering (anti-slip mat). The patient could write, but only with her left (non-dominant) hand. The patient was able to walk independently without any technical aids, but she had to use a banister for stairs. Her Functional Independence Measure (FIM) score was 114/126.

Experimental design

At baseline, the stability of motor performance was assessed by two pre-tests performed 2 weeks apart (S0). The first therapy period involved constraint of the less-affected arm. Three post-tests were run immediately after therapy, and 1 month after and 2 months after therapy (S1). The second therapy period was intensive training of the paretic limb, initiated after stabilisation of motor performance on the Fugl-Meyer Scale. Then, 3 post-tests were performed immediately after, 1 month after and 2 months after therapy (S2) (see Figure 1). Each evaluation consisted of a clinical assessment and a 3D motion analysis of a grasping task.

Treatment programme

For the first rehabilitation period, we arbitrarily chose to begin with the constraint therapy. During the constraint period, which lasted 2 weeks (5 days per week), the patient was instructed to wear a restrictive sling and a mitt on the less affected upper limb during 90% of waking hours.

The second rehabilitation period was intensive training. A 6 hour daily rehabilitation programme was arranged over 2 weeks (5 days per week). This consisted of physiotherapy, occupational therapy and adapted physical activities. Activities of daily living such as bathing and dressing were supervised and included in the rehabilitation time. Each activity was supervised by the relevant therapist or nurse for 30 minutes followed by 30 minutes of unsupervised training. During these two rehabilitation periods, the patient was an in-patient on the neurological rehabilitation ward.

Outcome measures

At each assessment, motor impairment was measured with the upper-limb section of the Fugl-Meyer Scale (Fugl-Meyer, Jääskö, Leyman, Olsson, &
Steglind, 1975); grip strength with a dynamometer (average of three trials) (Mathiowetz et al., 1985a); and manual dexterity with the Box and Block Test (Mathiowetz, Volland, Kashman, & Weber, 1985b).

At the end of each rehabilitation period, the patient’s self-assessment of her progress was estimated using a 10 point visual analogue scale (0 = no improvement, 10 = best conceivable progress).

A three-dimensional analysis of prehension was also performed at each assessment. This complementary evaluation was implemented in order to gather objective and reliable measurements which could be compared before and after the two therapeutic interventions. Moreover, these data on the temporal and spatial characteristics of upper-limb movements provided the opportunity to make inferences about possible motor control reorganisation (Boisson, 2004). In normal subjects, kinematic analysis has shown that a grasping movement involves a reaching phase ensuring the transport of the arm to the object and a grasping phase corresponding to the adjustment of finger grip aperture (Jeannerod, 1986). This observation, together with lesion studies on monkeys and humans suggest the existence of two parallel visuomotor channels governing these two components of prehension. We used a Vicon® system comprising six infrared cameras to record the 3D motion of six passive markers during a functional hand task. The patient sat at a table and the task consisted of grasping a 6 cm diameter glass, positioned at 20° to either side of the median line and 40 cm from the starting position (hand in mid-pronation, thumb and index finger touching). The instruction given to the patient was simply to take the glass and to bring it to her mouth as naturally as possible. This task was chosen because it is a functional task, which has already been used by our team for the evaluation of spontaneous recovery of prehension in hemiplegic patients (Luaute´ et al., 2002). Reflective markers were positioned on the nails of the thumb and index finger, internal radial styloid, the lateral epicondyle and the two acromio-clavicular joints. A red diode was switched on to signal the start of the trial. A green arrow lit up to indicate the glass to be grasped (left or right). One condition consisted of 22 movements directed in a random order either to the left or the right glass. The beginning and end of each movement was determined by sensors placed under the starting position and under each glass.

Based on kinematic analysis of normal prehension movement (Jeannerod, 1986), we selected the most relevant parameters related both to the transport (movement time, velocity peak) and to the grasping phases (maximal grip aperture). Movement time (MT) in milliseconds (ms) is determined as the time from the start of the hand lift to the start of the glass lift. For this study, the number of velocity peaks was measured with a specific algorithm (see Figure 2). Maximal grip aperture (MGA) measures the maximum distance in millimetres (mm) between the thumb and the index finger during the course of the grasping phase. In addition, we measured the
latency, defined as the time between the start signal and the hand lift-off from the table (i.e., the time between the start signal and the beginning of the movement). This gives a temporal measure of the cognitive processes involved before movement execution: spatial coding of the glass position,

Figure 2. Velocity profiles of the prehension movement. This figure shows different velocity profiles across sessions and illustrates how the peaks are counted. The curves presented in this figure result from a single movement. A peak was counted when subsequent velocity values showed a sequence of increase-decrease. A grey mark on the x-axis time line indicates each time a peak was detected. Figure 2a illustrates a movement performed with the unaffected hand, recorded during one pre-test (C). Figures 2b to 2d illustrate movements performed with the paretic arm, recorded during one pre-test trial (S0) (Figure 2b), one post-constraint trial (S1) (Figure 2c) and one post-intensive training trial (S2) (Figure 2d). Contralateral condition is displayed in the left column and ipsilateral condition is displayed in the right column.
motor programming, and movement initiation. Finally, we analysed wrist, thumb and index trajectories in the horizontal plane to explore movement homogeneity and trajectory variability. The contralateral condition relates to the right hand – left glass movement (paretic hand) and to the left hand – right glass movement (less affected limb). The ipsilateral condition relates to the right hand – right glass movement (paretic hand) and to the left hand – left glass movement (less affected limb). Movements performed with the less affected limb served as a control condition (C).

In order to ensure reliable measurements, clinical tests were administered by a multi-disciplinary team composed of a medical doctor specialised in physical medicine and rehabilitation, a physiotherapist and an occupational therapist. The physiotherapist and the occupational therapist also participated in the training programme. The engineer in charge of 3D motion analysis was blind to the study hypothesis and as to whether the patient was performing pre- or post-tests. The patient was also blind to the study hypothesis.

Statistical analysis

For clinical measures (Fugl-Meyer, grip strength and Box and Block Test), there were not enough data to perform an ANOVA in order to evaluate the respective effects of constraint therapy and intensive training. Hence, changes in these scores across sessions were calculated as a percentage of the maximal possible score or normal score, namely 66 for the Fugl-Meyer Scale (Fugl-Meyer et al., 1975), 35.73 kg for the grip strength (Mathiowetz et al., 1985a) and 85 cubes per minute for the Box and Block Test (Mathiowetz et al., 1985b).

For each kinematic parameter, we carried out analysis of variance with one principle factor “session”: pre-tests with the unaffected hand (C), pre-tests with the paretic hand (S0), post-constraint – paretic hand (S1) and post-intensive training – paretic hand (S2). Post-hoc analysis using Scheffé’s test was conducted if the factor “session” was significant.

RESULTS

For the less affected upper-limb the ANOVA showed a significant effect of session only for latency in the contralateral condition, $F(2, 6) = 36.54; p < .001$. Scheffé post hoc tests showed that the decrease of latency is related to the constraint period (significant difference between S0/S1 and S0/S2 but no difference between S1/S2). No session effect was observed for the other parameters; hence, apart from latency, repetition of the task across sessions did not modify the less affected limb performance.
The evolution of motor performance across sessions for the paretic upper-limb is detailed in Table 1. All clinical scores were improved after constraint therapy and a further gain was observed after intensive training.

The 3D motion analysis also revealed modification of gesture execution after therapy (Table 1, Figures 2 and 3). ANOVA showed a significant effect of session for all kinematic parameters analysed, $F(3, 8) > 7.99; p < .01$.

**Paretic limb versus less affected limb.** Scheffe post hoc tests showed, for all kinematic parameters except MGA (contralateral condition only), a significant difference between pre-tests performed with the paretic arm as compared to the less affected arm (significant difference between C and S0, $p < .05$). The difference was still significant between C and S1 for MT and number of velocity peaks (ipsilateral condition) and remained significant between C and S2 only for MT (ipsilateral condition). For all other parameters, no statistical difference was observed, either between C and S1 or between C and S2. Hence, except for MT in the ipsilateral condition, performance of both limbs was almost comparable at the end of the treatment programme.

**Pre-test versus post-tests with the paretic limb.** Scheffe post hoc tests showed that kinematic parameters were differently affected by constraint therapy and intensive training.

1. Decrease of latency (contralateral condition) and increase of MGA (ipsilateral condition) were observed after the combination of constraint and intensive training (significant difference between S0 and S2, $p < .01$, but no significant difference either between S0/S1 or between S1/S2).
2. A reduction in latency (ipsilateral condition), MT (both conditions) and the decrease of number of velocity peaks (both conditions) was observed after the constraint period (significant difference between S0/S1 and S0/S2, $p < .01$, but no difference between S1/S2).
3. An increase in MGA (contralateral condition) was observed after the intensive training period (significant difference between S0/S2 and S1/S2, $p < .05$, but no difference between S0/S1).

Qualitatively, wrist, thumb and index trajectories showed a clear improvement of smoothness after both constraint and intensive training. A decrease in variability was observed mainly after intensive training in the contralateral condition (see Figure 4).

After the constraint, the patient rated her progress with a score of 8/10, while this score was 7/10 following intensive training. The patient did not report any pain during either rehabilitation period. Spasticity was modified neither after constraint nor after intensive training.
Table 1  
Evolution of motor performance across sessions

<table>
<thead>
<tr>
<th></th>
<th>C</th>
<th>S0</th>
<th>S1</th>
<th>S2</th>
<th>S0/S1</th>
<th>S1/S2</th>
<th>S0/S2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Scheffé’s test</td>
<td>Mean (SD)</td>
<td>Scheffé’s test</td>
<td>Mean (SD)</td>
<td>Scheffé’s test</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C/S0</td>
<td></td>
<td>S0/S1</td>
<td></td>
<td>S1/S2</td>
</tr>
<tr>
<td>Fugl-Meyer (maximum = 66)</td>
<td>NR</td>
<td>28.0 (0.0)</td>
<td>NA</td>
<td>47.3 (3.5)</td>
<td>NA</td>
<td>54.3 (1.2)</td>
<td>NA</td>
</tr>
<tr>
<td>Box and block test (cubes/min)</td>
<td>NR</td>
<td>26.5 (5.0)</td>
<td>NA</td>
<td>41.3 (5.1)</td>
<td>NA</td>
<td>48.0 (1.0)</td>
<td>NA</td>
</tr>
<tr>
<td>Grip strength (kg)</td>
<td>NR</td>
<td>9.1 (1.3)</td>
<td>NA</td>
<td>16.6 (0.2)</td>
<td>NA</td>
<td>18.0 (0.0)</td>
<td>NA</td>
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<td></td>
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<tr>
<td><strong>Contralateral condition</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Latency (ms)</td>
<td>624.4 (47.9)</td>
<td>880.6 (80.3)</td>
<td>&lt;.01</td>
<td>743.3 (49.1)</td>
<td>n.s.</td>
<td>579.0 (73.7)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Movement time (ms)</td>
<td>970.0 (51.7)</td>
<td>2585.7 (459.1)</td>
<td>&lt;.01</td>
<td>1500.0 (140.3)</td>
<td>&lt;.01</td>
<td>1350.3 (93.7)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Number of velocity peaks</td>
<td>1.0 (0.0)</td>
<td>11.1 (3.4)</td>
<td>&lt;.01</td>
<td>3.7 (0.6)</td>
<td>&lt;.01</td>
<td>2.7 (0.6)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Maximum grip aperture (mm)</td>
<td>110.4 (4.0)</td>
<td>102.2 (3.8)</td>
<td>n.s.</td>
<td>103.8 (4.9)</td>
<td>n.s.</td>
<td>115.3 (1.4)</td>
<td>&lt;.05</td>
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<td></td>
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<tr>
<td><strong>Ipsilateral condition</strong></td>
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<td></td>
</tr>
<tr>
<td>Latency (ms)</td>
<td>508.0 (68.9)</td>
<td>840.5 (77.8)</td>
<td>&lt;.01</td>
<td>556.0 (81.4)</td>
<td>&lt;.01</td>
<td>464.6 (51.0)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Movement time (ms)</td>
<td>982.8 (62.1)</td>
<td>2206.1 (184.9)</td>
<td>&lt;.01</td>
<td>1512.4 (12.2)</td>
<td>&lt;.01</td>
<td>1413.4 (51.7)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Number of velocity peaks</td>
<td>1.0 (0.0)</td>
<td>8.2 (0.9)</td>
<td>&lt;.01</td>
<td>4.1 (0.4)</td>
<td>&lt;.01</td>
<td>2.7 (0.9)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Maximum grip aperture (mm)</td>
<td>112.3 (3.2)</td>
<td>98.9 (6.3)</td>
<td>&lt;.05</td>
<td>107.1 (2.2)</td>
<td>n.s.</td>
<td>114.6 (0.3)</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

For each parameter studied (lines), motor performances (average and standard deviations) are displayed across sessions (columns): pre-tests with the unaffected upper limb (C); pre-tests with the paretic limb (S0); constraint-therapy post-tests with the paretic limb (S1) and intensive training post-tests with the paretic limb (S2). For kinematic parameters recorded during the contralateral and the ipsilateral movements, results of Scheffé’s tests are inserted in separate columns; comparison between control and pre-tests (C/S0); between pre-tests and constraint-therapy post-tests (S0/S1); between constraint-therapy post-tests and intensive training post-tests (S1/S2); and between pre-tests and intensive training post-tests (S0/S2).

Abbreviations: SD = standard deviation; NR = not recorded; NA = not applicable; n.s. = non significant; cubes/min = cubes per minute; kg = kilograms; ms = milliseconds; mm = millimetres.
Figure 3. Movement performance across sessions. Figure 3a shows clinical scores. For each variable, the average score was calculated across sessions as the percentage of the maximal possible score or normal score (see the Statistical analysis section). Figures 3b to 3d show kinematic parameters. The average score across session is shown for Latency (Figure 3b), Movement Time (Figure 3c), Number of velocity peaks (Figure 3d), Maximum Grip Aperture (Figure 3e). Results of the contralateral condition are displayed in the left column and results of the ipsilateral condition are displayed in the right column. MGA = maximum grip aperture; MT = movement time. *significant difference at $p < .05$ (Scheffé’s tests).
Figure 4. Wrist, thumb and index trajectories in the horizontal plane. This figure shows average and standard deviation trajectories. Wrist trajectory appears in light grey (green online), index finger in dark grey (red online), and thumb in black (blue online). Each trajectory represents the mean of the 11 trials of a given condition (contralateral condition in the right column and ipsilateral condition in the left column). The trajectory variability was evidenced by width. The thinner the trajectory, the most reproducible the movement was. Figure 4a illustrates a movement performed with the unaffected limb during one pre-test (C). Figures 4b to 4d illustrate movements performed with the paretic arm, recorded during one pre-test condition (S0) (Figure 4b), one post-constraint condition (S1) (Figure 4c) and one post-intensive training condition (S2) (Figure 4d).
DISCUSSION

The results of this case study demonstrate a significant and dramatic improvement of upper-limb motor performance after a period of constraint therapy and an additional beneficial effect after intensive training. These results are remarkable given the delay of 3.5 years after stroke and confirm the usefulness of both components of CIMT in selected hemiparetic patients even at a chronic stage when motor function has reached a plateau.

Until now, only one study (Wolf, Lecraw, Barton, & Jann, 1989) had evaluated the effects of constraint therapy alone. It showed improvements in motor ability in 25 stroke and traumatic brain damaged patients more than one year after insult. A significant improvement was demonstrated immediately after the constraint on eight motor tasks and the results were maintained at 12 months with significant improvements in 20 of the 21 tasks. Sterr et al. (2002) showed that the beneficial effect of intensive training, associated with constraint therapy, is related to the duration of the daily training programme. This study compared a 3 hour daily intensive training programme with a 6 hour daily intensive training programme. Fifteen hemiplegic patients (13 stroke and 2 brain injury) included at least one year post-lesion were evaluated repeatedly before and after the rehabilitation using two tests – the Wolf Motor Function Test (WMFT) and the Motor Activity Log (MAL). Significant improvements were found in both groups but these improvements were greater in the group who received intensive training 6 hours per day. Our data showed a combined effect of constraint therapy and intensive training; however, a simple comparison of both components of the CIMT in this single case study is difficult given a necessary order effect and a possible ceiling effect due to the arbitrary choice to perform constraint therapy before intensive training.

Analysis of spatio-temporal kinematic parameters gives an indication of the mechanisms, which may contribute to motor recovery. Two non-exclusive hypotheses can be proposed to account for the differential effects observed after each therapeutic intervention: (i) a specific effect of treatment and/or (ii) a proximal-to-distal gradient of recovery. A future study could investigate these two hypotheses by reversing the order of both interventions. In the context of the present study, we discuss these results with respect to the existing literature.

Modulation of the transport phase by constraint therapy. It is interesting to note that constraint therapy improved kinematic parameters related to the transport component of movement (MT, number of velocity peaks) of the paretic hand. These results suggest that the remediation of learned non-use through constraint therapy modulates preferentially brain regions involved in the transport component of movement via proximal upper-limb motricity.
The existence of two anatomically segregated parieto-frontal circuits responsible for the control of the transport and the grasping component of arm movement is still debated. However, lesion studies in monkeys (Brinkman & Kuypers, 1973) and neuropsychological patient studies (Colebatch & Gandevia, 1989) suggest that the control of upper-limb proximal motricity relies on more bilateral descending motor pathways than distal motricity. Inter-hemispheric inhibition between motor cortices for the control of upper-limb motricity has been demonstrated in healthy subjects and seems to play a crucial role in motor recovery after stroke (Murase, Duque, Mazzocchio, & Cohen, 2004). Reversal of learned non-use through constraint therapy could be mediated by the modulation of the interhemispheric balance. This hypothesis could be tested in future work.

**Modulation of the prehension phase by intensive training.** In the contra-lateral condition, intensive training improved only the MGA, a parameter classically attributed to the grasping component of movement. In the ipsilateral condition, MGA increased after both therapeutic interventions but the improvement reached statistical significance only after intensive training. This result corroborates previous work in monkeys showing an increased representation of the fingers in the primary motor area after intensive training (Nudo et al., 1996). Thus grasp improvement after intensive training could be the result of an extension of finger representation.

Of note, spasticity of the upperlimb did not increase after intensive training. This result provides further evidence that repetitive training is not systematically associated with increased spasticity and can stimulate positively brain plasticity (Sterr & Freivogel, 2004).

**Proximal to distal gradient of recovery.** Alternatively our finding might reflect a proximal-to-distal gradient of functional recovery over time, which has been observed during spontaneous recovery (Rode et al., 2001). The combined effect of constraint and intensive training on MGA observed in the ipsilateral condition may well be explained by this hypothesis.

**Trajectory smoothness and variability.** It is interesting to note that trajectory smoothness was improved after constraint, whereas variability was mainly reduced after intensive training. This result, albeit qualitative, could suggest that mechanisms modulated by the constraint therapy are also involved in the control of trajectory smoothness, whereas mechanisms modulated by intensive training could play a role in trajectory variability.

**Effect of treatment on latency.** There was a significant reduction of latency, as has been reported previously after CIMT (Wu, Chen, Tang, Lin, & Huang, 2007a; Wu, Lin, Chen, Chen, & Hong, 2007b). This may reflect
a non-specific change in motor cognitive processes involved before movement execution, as has been previously suggested.

CONCLUSION

The results of this case study demonstrate a cumulative beneficial effect of constraint and intensive training on motor performance. Kinematic analyses suggest that reversal of learned non-use through constraint influences the transport phase of movement, whereas intensive training influences the grasping phase. Alternatively a proximal-to-distal sequence of functional motor recovery could also explain these results independently from the order of the therapeutic interventions. This study further emphasises the utility of 3D motion analysis to follow-up dynamic changes which reflect motor plasticity.

REFERENCES


