PERGAMON

Neuropsychologia 40 (2002) 16–27

NEUROPSYCHOLOGIA

www.elsevier.com/locate/neuropsychologia

Fast adjustments of ongoing movements in hemiparetic cerebral palsy

Edwin Van Thiel [a,*], Ruud G.J. Meulenbroek [a], Jeroen B.J. Smeets [b], Wouter Hulstijn [a]

[a] *Nijmegen Institute for Cognition and Information, University of Nijmegen, P.O. Box 9104, 6500 HE Nijmegen, The Netherlands*

[b] *Vakgroep Fysiologie, Erasmus Universiteit Rotterdam, P.O. Box 1738, 3000 DR Rotterdam, The Netherlands*

Received 8 November 2000; received in revised form 20 April 2001; accepted 20 April 2001

Abstract

The present study focuses on the ability of participants with spastic hemiparesis caused by cerebral palsy to adjust an ongoing movement. Typical symptoms associated with the disorder would lead one to expect that people with spastic hemiparesis would be unable to adjust their movements quickly and proportionally to a sudden change in the environment with their spastic arm. The results of the present experiment, however, prove otherwise. Eight hemiparetic adolescents with cerebral palsy and eight healthy control participants were asked to quickly hit a target projected onto a fronto-parallel screen. The target either remained stationary or started to move immediately after hand movement onset. Participants needed to adapt the ongoing movement to hit moving targets. The task was performed with the spastic and non-spastic arm by the hemiparetic participants and with the dominant arm by the healthy participants. Kinematic analyses showed that although the spastic arm of the hemiparetic participants displayed a significant increase in spatial variability which led to more errors, they were capable of successfully adapting their movement in a qualitative manner. The latency of the response to the change in target position was longer for the hemiparetic participants compared to the healthy control participants, but only 25 ms. Surprisingly, no between arm latency difference was found in the hemiparetic participants. Given the commonly observed movement deficits of the spastic arm, these results show that participants with spastic hemiparesis displayed a remarkable ability in adjusting movements quickly.

Keywords: Spasticity; Hemiparesis; Fast aiming; Kinematics

1. Introduction

Spastic paralysis can be described as a severe motor disturbance following lesions to the brain. Several posture- and movement-related symptoms characterize the disorder among which a velocity dependent increase in stretch reflex responses with exaggerated tendon jerks is the most prominent feature [16]. This hyperexcitability of the stretch reflex, which also leads to an increase in muscle tone [15], has been attributed to deficits in several neural mechanisms of which some will be discussed below. The increase in muscle tone has also been attributed to lasting physiological changes in muscle tissue (for reviews see Refs. [8,15]). Obviously, these changes in the motor system have serious consequences for the control of arm movements. Kinematic characteristics generally observed included prolonged reaction times, prolonged movement times, lowered peak velocities, dysfluency and increased spatial errors (misses in reaching) both in adult onset hemiparesis [17,23,31] and in hemiparesis caused by cerebral palsy [26,27,32].

In spasticity a disturbed modulation of motor neuron pool activity is thought to result in increased excitability of the motor neurons. Various evidence suggest that a loss of control over presynaptic inhibition of the motor neuron pools gives rise to the observed symptoms in spasticity [28]. This feature is not only present in adult onset hemiparesis but also in hemiparetic cerebral palsy [13]. The increases in motor neuron excitability not only give rise to increases in stretch reflexes in response to passive movements but may also disrupt the execution of active movements. The stretch reflex

* Corresponding author. Tel.: +31-24-3612618; fax: +31-24-3616066.

E-mail address: e.vanthiel@nici.kun.nl (E. Van Thiel).

0028-3932/01/$ - see front matter
PII: S0028-3932(01)00075-6

mechanism is considered to be senso-motoric in nature and thus plays an essential role in controlling movements [12]. Such an intimate relationship between proprioception and efferent control may well be a useful theoretical framework to describe spastic paresis as a motor disorder (cf. Ref. [18]).

Given these findings it is clear that something goes wrong during movement execution and therefore the need to study movement *execution* in hemiparesis, has become increasingly apparent. However, the effects of manipulations during movements to gain additional insight into how people with hemiparesis adjust ongoing movements have never been studied before. In our view this type of manipulation may particularly shed more light on the specific movement capabilities and loss of movement control in spastic hemiparesis.

Before specifying our expectations on the (dis)ability of hemiparetic participants to adjust an ongoing movement with their spastic arm, we first turn to experiments in which healthy participants performed such tasks. Healthy participants are very well capable of adjusting an ongoing movement appropriately to sudden changes in the environment. Studies have shown that humans can respond to changes in target position without being able to see their hand and that responses of the hand to changes in the target position may even remain unnoticed to the participants [6,14,19,22]. Furthermore, adjustments of arm movements also occur very quickly. Brenner and Smeets [5] showed that in a task in which participants had to hit targets that suddenly jumped to a different position or started to move, it took approximately 110 ms before a reaction became apparent in the hand movement. Many other studies have been conducted to determine the time it takes to respond to changes in target position, all of them with comparable results [22,25]. These results show that healthy arm movements can be adjusted adequately to changes in the environment, even during hitting movements which generally are performed within 300 ms. In the present experiment, we wanted to answer the question whether spastic arm movements can also be adjusted: (1) appropriately to changes in the environment *during* the movement and (2) whether this can be done as fast as healthy arm movements.

To answer these questions we used a simplified version of the experimental setup employed in the study of Brenner and Smeets [5]. We asked hemiparetic and healthy control participants to perform hitting movements towards circular targets that were projected onto a large projection screen (1×1 m). Targets always appeared stationary but in 80% of the trials they would start to move on hand movement onset. In these cases participants needed to adjust their ongoing movement in order to hit the target. When targets started to move they did so either to the left or to the right at one of two possible velocities. Although changes are not the exception in this design, there was maximum uncertainty about which of the conditions was going to be presented because the conditions were presented at random and each condition appeared an equal number of times. Moreover, we used perturbations in opposite directions, so that the average position of the perturbed targets was exactly at the position of the unperturbed target. So the best anticipation to a perturbation was to move to the unperturbed target.

Our first question is concerned with the efficiency of hemiparetic participants in adjusting an ongoing movement of their spastic arm. Given the above-mentioned finding that participants with hemiparesis primarily show difficulties with movement execution, perturbations during movements might be particularly difficult for them to respond to. They might even be incapable of making a spatially appropriate response to a sudden change in target position. This indeed is plausible when we bear in mind the stereotypical synergies generally associated with spastic hemiparesis [4,7]. Due to these stereotypical synergies they could be expected to make a standard directional response. We therefore also varied the velocity of the target across trials to determine whether hemiparetic participants would be capable of making a spatially more appropriate response rather than just a standard directional response. Namely, the amount of directional change of their movement needs to be proportional to the target velocity in order to hit the target accurately. We did not expect hemiparetic participants to be capable of making such an efficient and appropriate change of their ongoing (spastic) movements. We rather expected them to use a stereotypical directional response that would ‘get the job done’ on most occasions. Additionally, we expected several generally observed kinematic characteristics of spastic arm movements to be present in the current experiment. These included, besides prolonged reaction times to the initial target appearance and prolonged movement times, increased movement variability and an increased number of misses.

Our second question concerns the time it takes for visually specified information on *changes* in target behavior to become apparent in the hand displacement, irrespective of whether this reaction is efficient or not. The results of several studies have shown that response latencies are generally prolonged in spastic hemiparesis both in adult onset hemiparesis and in hemiparetic cerebral palsy [17,27,31,32]. Consequently, not only will the response latency to the initial appearance of the target most probably be prolonged, but the subsequent response to changes in the target position might also be prolonged.

Table 1
Participant information

Hemiparetic	Age (years)	Sex	Etiology	Other
1	16.1	M	RH/CP	–
2	17.5	M	RH/BT[a]	–
3	19.4	M	LH/CP	–
4	17.7	M	RH/CP	–
5	20.1	M	LH/CP	–
6	17.5	M	LH/CP	Epileptic/scoliosis
7	17.3	M	LH/CP	Mild scoliosis
8	15.8	M	RH/CP	Epileptic
	17.4 (mean)			
Control				
	18.8	All male	5 Right handed/3 left handed	

RH, right hemiparesis; LH, left hemiparesis; CP, cerebral palsy; BT, brain tumor. Note: all hemiparetic participants were classified as having mild to moderate spasticity. No clinical or IQ measurements were available.
[a] Tumor was removed within the first year after birth.

2. Method

2.1. Participants

Eight hemiparetic adolescents with cerebral palsy and eight healthy control participants took part in the experiment. At the time of the study, the hemiparetic participants were students from the Werkenrode Institute (Groesbeek, The Netherlands) where they followed an adapted educational program. The control participants were psychology students at the University of Nijmegen. All participants gave signed consent and were paid ten guilders for their participation. Additional participant information is given in Table 1. The hemiparetic participants were pre-selected on the basis of (1) having been diagnosed as having stable hemiparesis and (2) the ability to actively stretch their arm far enough to perform the experimental task under study. Furthermore, none of the hemiparetic participants displayed cognitive dysfunction and the only treatment they received was physical therapy to prevent painful and disabling contractures. No clinical or IQ measurements were available.

2.2. Experimental setup

The participant was seated on a rigid chair positioned approximately 50 cm in front of a projection screen (1×1 m), which was tilted to 30° with the vertical to facilitate the task execution. The participant held a rod in his/her left or right hand and was instructed to adopt a comfortable posture. This posture was such that when the participant was holding the rod, his or her elbow was flexed and the rod was located at approximately the height of the participant's head in a horizontal position (see Fig. 1). The rod was made from light wood and was covered with duct tape to realize a comfortable and secure grip. The rod was 21 cm long with a diameter of 2.5 cm and attached to its head was the pod of a badminton shuttlecock which enabled a firm but safe impact with the screen.

A 3D motion-tracking system (Optotrak 3020) was used for recording at a sampling rate of 300 Hz the positions of two Infra Red Emitting Diodes (IREDs) attached to the rod. The position of the tip of the rod was calculated in real-time by linear extrapolation from the positions of the two IREDs attached to the rod. The resolution of the device was better than 0.1 mm in all three dimensions as calculated from the variability of the distance between the two markers over the successive measurements ($n = 3456$) of a previous experiment. The recordings of the rod positions were stored on the hard disk of the PC that also provided feedback of the rod's position to the experimenter

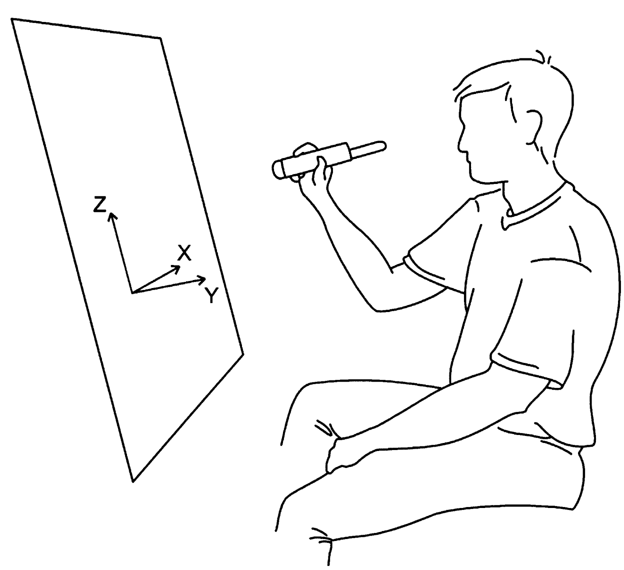

Fig. 1. The experimental setup.

between trials and regulated a second PC that guided the presentation of the stimuli. These stimuli were projected on the screen by means of a multimedia projector (M3™ MP8030; 60 Hz). Data collection and stimuli presentation were synchronized with a near constant delay of 20 ms.

2.3. Conditions and procedure

A yellow filled circle was used as a target (diameter 3 cm). The target appeared at the perpendicular projection of the tip of the rod on the projection screen. On hand movement onset the target could start to move (80% of the trials) with a constant velocity. Hand movement onset was defined as the moment at which the hand reached a velocity of 0.1 m/s. When the target started moving this could be with a velocity of 3 or 15 cm/s to either the left or to the right where each condition occurred in 20% of the trials. Therefore, in 20% of the trials the target remained stationary (velocity of 0 cm/s). These conditional values were derived from pilot experiments with hemiparetic participants. The target remained visible for the participant until the moment of impact when the moving hand occluded it. Each condition was presented 16 times resulting in a total of 80 trials per arm. Conditions were randomized within participants. The hemiparetic participants performed the task with both their spastic and non-spastic arms. The control participants performed the task with their dominant arm only. The spastic arm and the non-spastic arm were tested in separate blocks. The two groups (hemiparetic and healthy) were matched on sex and age and for the hemiparetic participants the order of blocks (spastic/non-spastic) was counterbalanced. Trials with incomplete data due to invisible IREDs during motion were repeated immediately. However, these occurred seldomly.

Between trials, real-time feedback of the position of the rod was provided to the experimenter for the following purpose. A trial started with the experimenter guiding the participant by means of verbal instructions to position his hand that held the rod within a predefined starting volume ($20 \times 10 \times 20$ cm^3, X, Y, Z, respectively). These instructions were based on the real-time position feedback (approximately 100 Hz) of the rod's location in the workspace. The center of this volume was at a distance of 40 cm from the screen. After holding the hand still within the starting volume for a randomly determined interval of 0.5, 0.6, 0.7 or 0.8 s, the target was projected on the screen.

The participant was instructed to hit the target quickly with the tip of the rod as soon as the target appeared on the screen. At the moment the rod hit the screen the target immediately changed color (green when hit; red when missed), and if the hit target was a moving target, it also stopped moving. At the position of the impact a yellow crosshair was shown to provide the participant with feedback on the place of impact. If the center of this crosshair lay outside of the target it was a miss. Furthermore, to give the participants additional feedback on their movement, the total movement time in units of 10 ms was shown on the screen. Feedback was provided to enable the participants to comply with the instruction.

Participants were allowed as many practice trials as they needed to get accustomed to the task and to find a comfortable posture that they were asked to maintain during the experiment. Also, the practice trials allowed the participant to find a balance between moving quickly while not missing the targets. This implied that each participant determined a suitable movement time that they used as guideline for their movement speed in the subsequent experiment.[1] The participants could pace the experiment themselves simply by pausing between trials prior to moving the hand towards the starting volume. On average, practice sessions took 25–35 trials (about 15 min including instructions) for the hemiparetic participants and 20–30 trials for the control participants.

2.4. Data analysis

We restricted our analyses of the movements to the XY plane since we were interested in whether position and velocity information of the targets influenced the lateral hand displacement. Only for segmentation purposes the tangential velocity-profile was derived from the 3D-displacement data. Reaction Time (RT) was defined as the interval between the moment of target presentation and the moment at which the hand started to move, i.e. the moment the hand reached a velocity of 0.1 m/s. The end of a movement was defined as the moment the tip of the rod hit the screen. Movement Time (MT) was defined as the time from hand movement onset to the moment of impact with the screen. Subsequently, the displacement data were filtered with a fourth-order Butterworth filter with a zero-phase lag and an effective cut-off frequency of 25 Hz.[2] Finally, we determined the time it took for the perturbation to start to influence the movement of the hand from the accel-

[1] The high occurrence of perturbations causes the participants to develop an anticipatory strategy. This, however, does not pose any problems for our research questions since the direction and magnitude of the response remains unpredictable. Also, the question of how fast participants with hemiparetic cerebral palsy can adjust their ongoing movement stays valid under this anticipatory strategy.

[2] Filtering was necessary in spite of the high accuracy of the motion-tracking device because of the amplification of the measurement errors caused by linearly extrapolating from the two markers to calculate the positions of the tip of the rod, and the double differentiation to obtain acceleration.

eration profile of the hand movement in the X-direction. The exact procedure to calculate this latency is given in Section 3 and is depicted in Fig. 7.

2.5. Statistical analysis

Our experimental design included the factors arm (spastic, non-spastic and control) and condition (0 cm/s and 3 and 15 cm/s to the left and right). Thus, two factors are nested under condition. These are target velocity (0, 3 and 15 cm/s) and direction (left and right). The dependent variables RT, MT, the mean end-positions of the rod on the screen, the percentage of misses, the Constant Error (CE), the Variable Error (VE) and the time until visual information became apparent in the hand displacement were evaluated statistically. The statistical procedures we used included t-tests (for comparisons between hemiparetic and control participants), paired t-tests and Repeated Measurement Analyses (for within participant comparisons). An alpha level of 0.05 was used for all statistical tests.

3. Results

We start by presenting the results on the analysis of the MTs and the percentages of misses. Subsequently, we give the results on the mean end-positions of the rod on the screen and present the results on the CEs and VEs produced by the participants. With these analyses we address the ability of hemiparetic participants to respond to the changes in target position during an ongoing movement in a spatially appropriate manner. Secondly, we present the results on the analysis of the RTs to the initial target appearance and the time it takes for visual information on target position change to become apparent in the hand movement.

Results are reported over all trials, thus including the movements that resulted in misses. Only those trials that were clearly executed contrary to the task requirements, i.e. had extreme RTs, MTs and those trials with movement direction reversals along the Y-axis were excluded from the statistical analysis. Out of the 1920 trials of the experiment 162 trails (8%) were excluded. Of these 162 trials 109 were trials performed with the spastic arm, 47 were performed with the non-spastic arm and six by the control participants.

3.1. Movement time

In the top panel of Fig. 2 the mean MTs are depicted as a function of arm and target velocity (thus data are pooled over direction). In this figure it can be seen that, as expected, the spastic arm moved significantly slower than the non-spastic arm and the control arm, $F(1,7) = 7.56$, $P < 0.05$; $T(1,14) = 4.10$, $P < 0.05$. Furthermore, the non-spastic arm was also significantly slower than the control arm, $T(1,14) = 5.00$, $P < 0.05$.

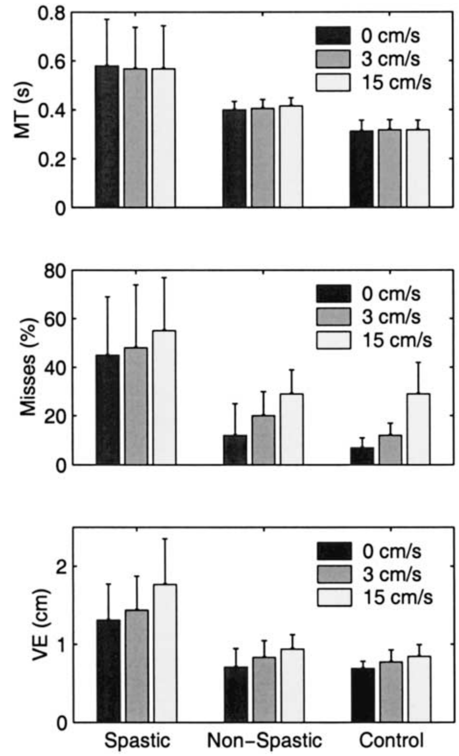

Fig. 2. Mean movement times (MTs, top panel), percentage of misses (middle panel) and variable error (VE, bottom panel) per arm and per target velocity. Error bars represent standard deviations over participants.

The MTs did not differ significantly as a function of direction for both the spastic arm and the non-spastic arm. In contrast, the control arm moved slightly faster to the right than to the left. Mean MTs were 311 and 323 ms, respectively, $F(1,7) = 11.12$, $P < 0.05$. MT did not vary as a function of target velocity for any arm. Test statistics were $F(2,7) = 0.81$, $P > 0.05$; $F(2,7) = 2.33$, $P > 0.05$; $F(2,7) = 3.131$, $P > 0.05$, for the spastic, non-spastic and control arm, respectively.

3.2. Misses

The spastic arm of the hemiparetic participants not only moved slower, but also produced significantly more misses (Fig. 2, middle panel). For the spastic arm of the hemiparetic participants 50% of the trials were misses. For the non-spastic hemiparetic arm and the control arm the percentages of misses were 22 and 18%, respectively. The statistic for the spastic versus non-spastic arm comparison was $F(1,7) = 13.56$, $P < 0.05$. For the comparisons between the spastic and control arm and non-spastic and control arm the statistics were $F(1,7) = 15.60$, $P < 0.05$ and $F(1,7) = 1.16$, $P > 0.05$, respectively.

As can be seen in the middle panel of Fig. 2 the percentage of misses generally increased as a function of target velocity for all arms. Test statistics were $F(2,7) = 3.70$, $P > 0.05$; $F(2,7) = 15.235$, $P < 0.05$; $F(2,7) = 19.01$, $P < 0.05$, for the spastic, non-spastic and control arm, respectively. Furthermore, this increase in misses is not different across arms since there were no significant interactions as a function of target velocity with arm.

Although there were no significant differences in the number of misses as a function of direction for the spastic and the control arm, the non-spastic arm produced significantly more misses when hitting to the right; 18 versus 27% misses for movements to the left and to the right, respectively, $F(1,7) = 7.45$, $P < 0.05$.

3.3. Mean end-positions of the rod on the screen

In the panels of Fig. 3 all trajectories are shown of the movements of a hemiparetic participant who performed the task with his spastic arm. It can be seen that the mean end-position of the trajectories towards targets moving with 15 cm/s to the left (top panel; solid lines) is located to the left of the mean end-position of trajectories towards targets that moved at 3 cm/s to the left (bottom panel; solid lines). The reverse is true for trajectories towards targets moving at 15 cm/s to the right. The mean end-position of these trajectories is located to the right of the mean end-position of trajectories towards target that moved at 3 cm/s to the right and targets that remained stationary. Actually, there is an order of mean end-positions from the left to the right as a function of condition. This order is present in all arms of all participants and demonstrates that all participants were not only capable of adjusting an ongoing movement in response to a change in target position but that they also differentiated between target velocities. This finding is illustrated in Fig. 4 where it can be seen that lines never cross, reflecting the consistent order in the mean end-positions as a function of condition. Also, it can be seen that there is a strong left–right symmetry in the position where the screen, on average, was hit. Actually, the mean end-positions of the movements did not vary significantly as a function of direction for any arm, $F(1,7) = 0.01$, $P > 0.05$; $F(1,7) = 4.62$, $P > 0.05$; $F(1,7) = 1.30$, $P > 0.05$, for the spastic, non-spastic and control arm, respectively.

3.4. Constant error

All participants made a substantial CE (an overshoot) when trying to hit targets moving at 3 cm/s. Contrarily, movements towards targets moving at 0 and 15 cm/s did not produce such a CE. Only those participants that performed the task fastest produced a small undershoot when hitting targets moving at 15 cm/s.

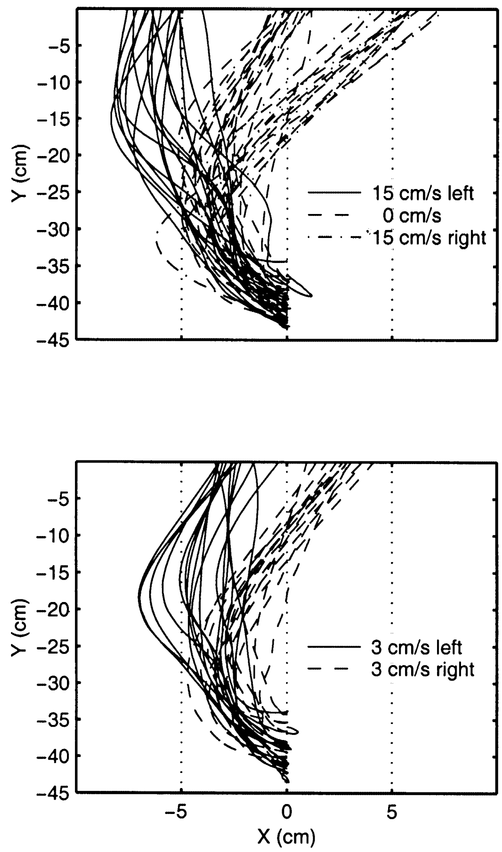

Fig. 3. All trajectories of a hemiparetic participant (participant 3) performed with his spastic arm. The top panel shows, from the left to the right, the trajectories made towards targets moving at 15 cm/s to the left (solid lines), 0 cm/s (dashed lines) and 15 cm/s to the right (solid lines). The bottom panel displays the trajectories of movements made towards targets moving at 3 cm/s to the left (solid lines) and 3 cm/s to the right (dashed lines). Note the different scales on the X- and Y-axes.

However, given the size of the target (3 cm) relative to the observed CEs, participants still hit the targets successfully. But is this behavior in correspondence with our expectations?

We expected that the mean end-positions of the rod on the screen would be proportional to the target velocity for the control participants and for the non-spastic arm of the hemiplegic participants. We did not expect this to be true for the spastic arm of the hemiparetic participants. In Fig. 5 the observed mean values per arm are plotted against the target velocities. If participants responded proportionally to the target velocities the lines should resemble a straight line. This, of course, is only true when participants do not vary their MT as a function of target velocity as was the case in the present experiment. However, the lines resemble an S-shape.

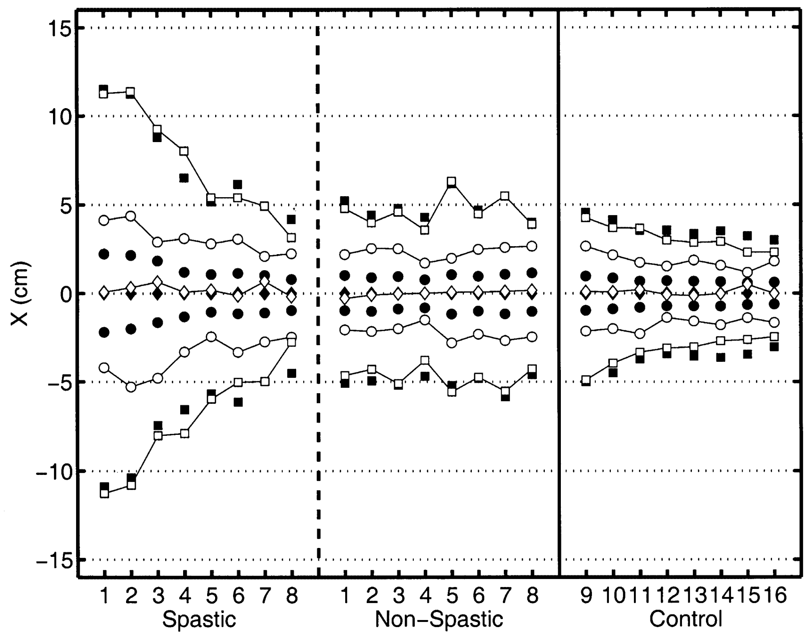

Fig. 4. Mean end-positions of the hand (white symbols) per condition, per arm, and per participant. The black symbols represent the corresponding mean end-positions of the targets. Squares represent the conditional values of 15 cm/s, circles represent target velocities of 3 cm/s and diamonds represent the stationary targets. The numbers along the bottom axis represent the spastic participants (1–8) and the control participants (9–16). To facilitate comparisons, lines connect symbols representing the same condition. Also, the order of the spastic arms as well as the order for the control arms was determined by the mean end-position of the trajectories towards targets moving at 15 cm/s to the left (from small to large). Therefore, the spastic arm labeled 1 had the smallest value for the mean end-position of movements made towards the target moving at 15 cm/s to the left (negative y-axis represents movements to the left). The non-spastic arms have not been sorted thereby keeping the correspondence between arms, i.e. the spastic and non-spastic arm, of the hemiparetic participants.

To evaluate the observed mean values against the hypothetical straight line we specified the specific statistical contrast corresponding to the factor condition $[-15\ \ -3\ \ 0\ \ 3\ \ 15]$ and evaluated how well the observed means fitted this contrast. If indeed the observed means follow an S-shape rather than a linear course then a cubic description actually should fit better. We therefore compared a linear and a cubic contrast. For all arms both contrasts provided a good description for the data but the cubical contrast consistently produced higher F statistics than the linear contrast. This indicates that indeed the data can be better described by an S-shape than by a linear trend. Therefore, the CEs observed are pronounced, thereby falsifying our expectations on the proportionality of the responses as a function of Condition. The test statistics are given in Table 2.

3.5. Variable error

As a measure of VE we calculated per participant and per target velocity the standard deviation of the distance between the final rod- and target-position. Mean VEs per arm and target velocity are depicted in the bottom panel of Fig. 2. The VE for the spastic arm was significantly larger when compared with the non-spastic arm and the control arm, $F(1,7) = 28.40$, $P < 0.05$ and $F(1,14) = 18.33$, $P < 0.05$, respectively. The VE of the non-spastic arm was not significantly different from the VE of the control arm $F(1,14) = 0.47$, $P > 0.05$. For all arms the VE increased as a function of target velocity but not always significantly. This effect was significantly stronger for the spastic arm when compared to the control arm. Actually, for the control arm this effect just failed to reach significance. For the spastic, non-spastic and control arm the statistics were $F(2,7) = 10.24$, $P < 0.05$; $F(2,7) = 10.20$, $P < 0.05$; $F(2,7) = 3.35$, $P > 0.05$, respectively.

3.6. Reaction times

The response of the spastic arm to the initial appearance of the target was significantly delayed when compared to the non-spastic and the control arm, $F(1,7) = 17.73$, $P < 0.05$; $T(1,14) = 3.51$, $P < 0.05$, but the non-spastic arm did not react significantly different from the control arm, $T(1,14) = 0.84$, $P > 0.05$. Mean RTs for the spastic, non-spastic and control arm were 426, 338, and 318 ms, respectively (top panel Fig. 6).

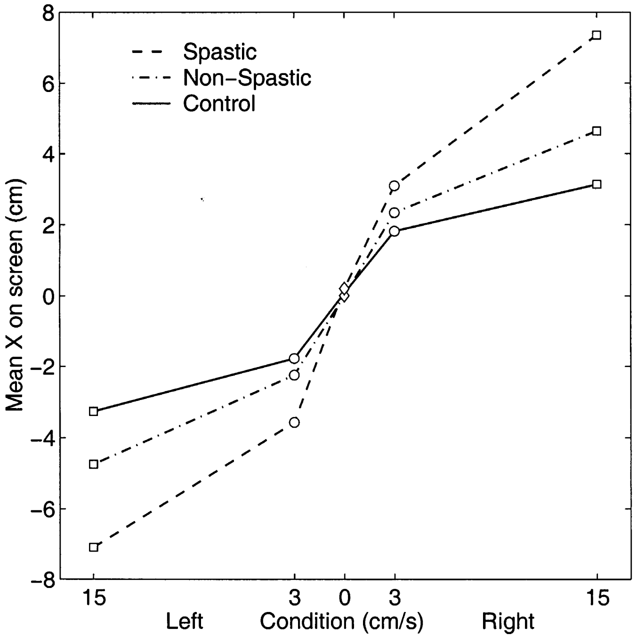

Fig. 5. The mean end-positions on the screen (presented per arm and condition) are not perfectly proportional to the target velocity. Symbols are used as in Fig. 4.

3.7. Time until visual information becomes apparent in the arm displacement

The procedure to calculate the time until visual information became apparent in the arm displacement is shown in Fig. 7. A mean time of 101 ms for the control participants corresponded well with earlier findings [5; 110 ms]. The mean values were 126 ms for the spastic and 118 ms for the non-spastic arm. The mean values per participant are shown in the bottom panel of Fig. 6 grouped by arm. As expected the spastic and control arm differed significantly but this difference was only 25 ms, $T(1,14) = 3.12$, $P < 0.05$. The non-spastic arm also differed significantly from the control arm, $T(1,14) = 2.48$, $P < 0.05$. Most importantly, however, the spastic and non-spastic arm did not differ significantly from one another $F(1,7) = 1.25$, $P > 0.05$.

Table 2
The F statistics and R^2 per arm for the linear and cubic contrasts. All P values <0.05

	Linear $F(1,7)$	Cubic $F(1,7)$	R^2 (linear)	R^2 (cubic)
Spastic	68.17	208.30	0.986	0.993
Non-spastic	35.87	75.64	0.973	0.987
Control	157.20	188.57	0.994	0.995

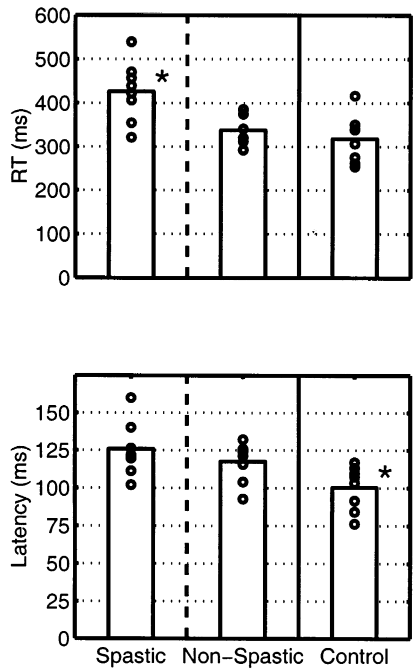

Fig. 6. Top panel: reaction time to the initial target appearance (ms). Bottom panel: latency (ms), i.e. time until visual information on target displacement becomes apparent in the lateral hand acceleration. RTs and latencies are given for each participant (open circles). Bars represent the mean per arm. Bars with an asterisk deviate significantly from the other bars. Therefore, in the top panel the non-spastic arm and control arm do not differ significantly. Contrarily, in the bottom panel the spastic and the non-spastic arms do not differ significantly.

4. Discussion

In the present experiment we examined the ability of participants with spastic hemiparesis to respond adaptively to sudden changes in the environment. Because of the typical symptoms associated with spastic hemiparesis [4,7,8,15] one might expect hemiparetic participants to be incapable of responding in a spatially adaptive manner with their spastic arm as compared to their non-spastic arm. One might also expect them to be incapable of responding as quickly as healthy control participants to a change in target position.

More specifically, the direction and the extent of responses to a change in target position were not expected to be functionally tuned to the target position and velocity. The results, however, clearly show that all participants, both hemiparetic and control were capable of adjusting their movements differentially to the target characteristics. This is illustrated by the finding that the mean end-positions of the rod on the screen varied as a function of the target velocities, not only for the control group but also for the hemiparetic participants performing the task even with their spastic arm.

Strikingly, all participants displayed the same movement strategy with which they produced a systematic overshoot when hitting targets moving at 3 cm/s. No such overshoot was found when hitting targets moving at 15 cm/s. We expected only hemiparetic participants to be incapable of responding proportionally to the various target velocities with their spastic arm. This, however, was not the case. All participants responded in the same non-proportional manner to the targets moving at 3 cm/s. It appears that the participants either anticipated the worst and therefore always initiated a movement towards a moving target as if it was moving at 15 cm/s or their initial response was one towards a target of average velocity (7.5 cm/s). Although this strategic behavior in itself may be an interesting topic of investigation, the finding that hemiparetic participants displayed the same behavior compared to healthy control participants is important for the present study. This, namely, shows that mild to moderate hemiparetic participants are very well capable of adapting their movements in the same qualitative manner as healthy control participants even with their spastic arm.

Although spastic movements were well adapted in accordance with changes in target position, the movements themselves were performed poorly. First, the spastic arm moved significantly slower compared to healthy control participants but nevertheless produced much more misses. The non-spastic arm was also slower than the control arm but it did not produce more errors. A similar pattern of results is visible in the related measurements on the variable error. The variable error was significantly larger in the spastic arm when compared to the non-spastic and control arms. Furthermore, an increase in variable error as a function of target velocity was observed and this increase was significantly larger for the spastic arm. The results

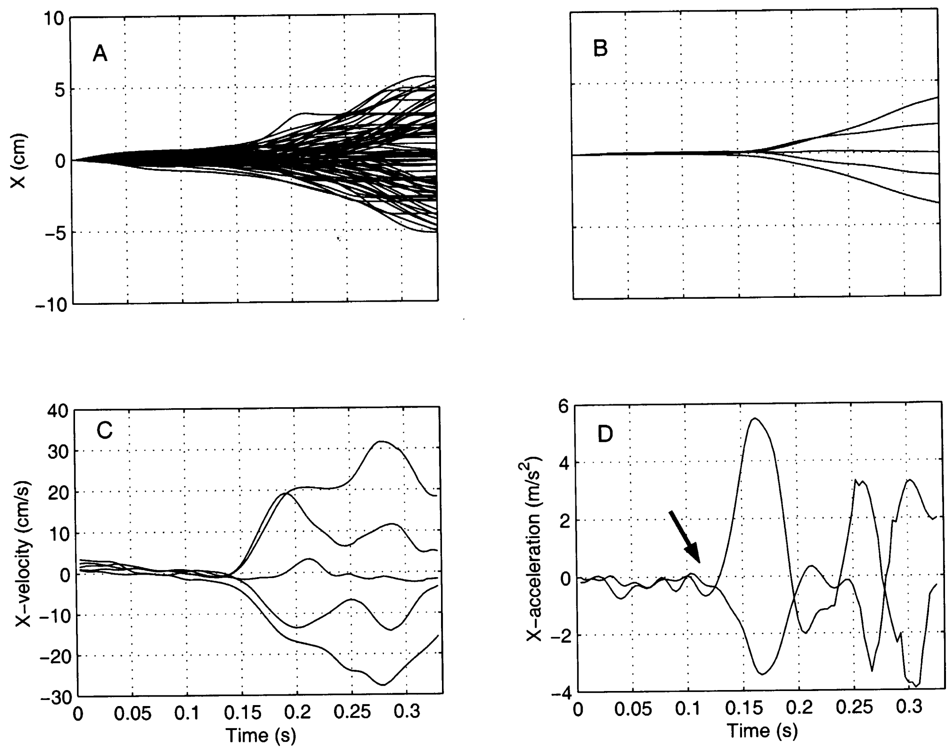

Fig. 7. Stepwise overview of the analysis applied to calculate the time until visual information starts to influence lateral acceleration of the hand for one participant. First, we synchronized all trials with respect to the moment at which the target started to move and subsequently took from that moment on the next 100 samples, i.e. 333 ms, of each trajectory. If a movement ended earlier, the trajectory was extrapolated with its last value to a total duration of 100 samples. This procedure is depicted in A. Next, we calculated the mean lateral trajectory per condition; see B. These means may be distorted somewhat by the artificial extrapolation of trajectories with a shorter duration than 333 ms after target motion onset, but this does not affect our calculation. This is so because we expected that it would take about 110 ms [5] for visual information to become apparent in the arm and movements were never performed that fast. From the resulting mean trajectories velocity-profiles were calculated which are depicted in C. Finally, from the velocity-profiles for movements towards the targets that started moving in the same direction the means were calculated and from these means a mean acceleration was derived. This resulted in the acceleration profiles depicted in D. We took the last moment in time that these profiles crossed before diverging as the moment at which visual information became apparent in the lateral displacement (indicated by the arrow in D). This procedure circumvented artificial effects of absolute and relative thresholds and is possible because of the left–right symmetry in the conditions.

suggest that the principle of a speed-accuracy trade-off is in effect in the hemiparetic participants, especially in the spastic arm. Participants chose to move slower probably to suppress the variability of their movements. This choice may very well be an active, i.e. strategic one, since in a previous study [32] we showed that under the instruction to move as fast as possible no significant differences in MT existed between arms of hemiparetic participants, whereas the variability of the spastic arm was significantly increased. Likewise, in the current experiment it seems that the between arm difference in hemiparetic participants is most clearly observed in our measurements of variability. This variability in turn leads to increased levels of task failure. Indeed, increased levels of variability are highly characteristic for this disorder [18,32]. Still, the finding that hemiparetic participants produced the same qualitative behavior with their spastic arm shows in our opinion a striking unexpected adaptiveness of movements in participants with spastic hemiparesis.

Our second expectation related to the time until a response becomes apparent in the lateral hand acceleration in reaction to the change in target position. Because we found longer reaction times for the spastic arm compared to the non-spastic and healthy control arms, one could expect the spastic arm to respond later than the other two to changes in target position. In fact, our results partly confirm this expectation. The change in the lateral hand acceleration of the spastic arms became apparent at a significantly later moment in time when compared to the control arms. However, this delay in responding was only 25 ms. Moreover, whereas there was a large between arm difference for the hemiparetic participants in the reaction times to the initial target appearance, strikingly, this between arms difference is absent in the response to the change in the target position.

The delay in the response to the appearance of the target can be interpreted according to the speed-accuracy trade-off mentioned above. Responding later offers the hemiparetic participants simply more time to prepare the movement accurately with their spastic arm. On the other hand, the task prompted the participant to not only to move quickly but also to react quickly since the total MT was to be minimized. Therefore, it might well be that physiological differences between the spastic and non-spastic arm cause the spastic arm to respond slower. Spastic arm movements are characterized by muscular weakness [9] caused by an inadequate recruitment of motor neurons [29]. Therefore, the difference in responding to the appearance of the target might be explained by difficulties with *initiating* a movement with a spastic arm.

Consequently, when the arm is already moving, initiation problems already have been solved and therefore no differences in response times to changes in the target position between arms should follow from it. Although this reasoning explains the absence of a between arm difference in hemiparetic participants, the fact remains that their responses to the change in target position are delayed significantly, albeit only 25 ms. It might be argued that this delay is also strategic. We doubt, however, that the fast adjustments in the present experiment are under volitional control and therefore susceptible to strategic interference. More probable is that initiating a change of movement is also (slightly) impaired. The finding that this is the case for both arms then becomes particularly interesting. A speculative but tentative explanation may be offered in terms of a disturbance of the modulation of presynaptic inhibition [12,28]. Perhaps a disturbance of the modulation also implies a delay in modulation. It is known that the 'healthy' side in hemiparetic patients also shows motor disturbances [1]. Even pathological stretch reflexes have been observed on the 'good' side of hemiparetic patients [30]. Thus, it is not surprising that also the non-spastic arm displayed a prolonged latency in response to changes in target position.

Alternatively, the finding that these latencies are very short in healthy participants [5,22,25] may well indicate that only sub-cortical processing is involved (see also Ref. [20] for the putative role of the propriospinal pathway). This may very well be the case since it is known that healthy participants can respond to changes in the environment without even being aware of it [6,14,19,22] and on the mere basis of internal feedback-loops only [2].

In this context it is informative to know that the hemiparetic participants in our experiment suffered from cerebral palsy, a condition primarily associated with upper motor neuron damage [24]. Upper motor neuron damage only leaves sub-cortical processes, possibly responsible for the earliest responses, intact. Then it might be reasoned that no difference in latency between the arms of the hemiparetic participants should have occurred. As such, only volitional movements that require cortical mediation may be disrupted but more reflexive reactions to sudden changes in the environment may have remained unaffected. We, however, do not know if the hemiparetic participants in our experiment indeed only suffered from upper motor neuron damage since they are students rather than patients and therefore elaborate diagnostic information was not available. This reasoning however might explain the absence of a between arm difference for the hemiparetic participants when responding to changes in target position during movement. Moreover, combined with the existence of pathological stretch reflexes in both arms, the results become explainable.

Recently, however, studies have indicated that the posterior parietal cortex (PPC) may be actively involved in fast adjustments during arm movements

[10,21]. If this indeed is the case then our reasoning given above becomes invalid. Suppose that the PPC is responsible for the fast adjustments of arm movements to changing target positions. Since cerebral palsy is associated with upper motor neuron damage, it might be that the PPC has been damaged as well but, if so, only unilaterally in hemiparetic cerebral palsy. Then, it might be reasoned that deficits in responses to changes in target position should only be observed in the impaired side, which was not the case in the present study. Although this reasoning seems valid in its own right, we must add the notion that during the many years that the damaged motor system had time to adjust, ipsilateral branching of presynaptic axons may have occurred [3,11]. Then, fast adjustments of the spastic arm (and also non-spastic arm) may be controlled by the ipsilateral and undamaged side. This might also explain the absence of a between arm difference in fast adjustments, although it does not explain the small but significant delay.

Although the hemiparetic participants were capable of responding adaptively within the current setup, an interesting question that remains is if these participants will also be capable of responding correctly to very unexpected perturbations. In the current setup, participants most likely anticipated a perturbed trial on every trial since in 80% of the cases the target would start to move after hand movement onset. Further study of this aspect is needed to arrive at conclusive statements in this respect.

In conclusion, although hemiparetic participants respond later to changes in target position than the control subjects, the magnitude of this effect was surprisingly small. The hemiparetic participants did produce significantly more errors with their spastic arms and also moved significantly slower. Still they displayed the ability to respond adaptively to the changes in the target position and even responded differentially to the various target velocity conditions. Moreover, the spastic arm did not react slower than the non-spastic arm. Given the typical symptoms associated with the disorder this demonstrates remarkable movement flexibility with the spastic arm. Such demonstration contributes, in our view, to the search for the exact nature of the control problems in spastic hemiparesis.

Acknowledgements

The Netherlands Organization for Scientific Research is gratefully acknowledged. This research was conducted while Edwin Van Thiel was supported by a grant of the Foundation for Behavioral and Educational Sciences of this organization (575-23-003). We thank Chris Bouwhuisen for his technical assistance in setting up the experiment.

References

[1] Baldiserra F, Cavallari P, Tesio L. Coordination of cyclic coupled movements of hand and foot in normal subjects and on the healthy side of hemiparetic patients. In: Swinnen S, Heuer H, Massion J, Casaer J, editors. Inter-limb coordination: neural, dynamical and cognitive constraints. San Diego: Academic Press, 1994:229–42.

[2] Bard C, Turrell Y, Fleury M, Teasdale N, Lamarre Y, Martin O. Deafferentation and pointing with visual double-step perturbations. Experimental Brain Research 1999;125:410–6.

[3] Benecke R, Meyer BU, Freund HJ. Reorganisation of descending motor pathways in patients after hemispherectomy and severe hemispheric lesions demonstrated by magnetic brain stimulation. Experimental Brain Research 1991;83:419–26.

[4] Bobath B. Adult hemiplegia evaluation and treatment. London: Butterworth and Heinemann, 1978.

[5] Brenner E, Smeets JBJ. Fast responses of the human hand to changes in target position. Journal of Motor Behavior 1997;29:297–310.

[6] Bridgeman B, Lewis S, Heit G, Nagle M. Relation between cognitive and motor-oriented systems of visual position perception. Journal of Experimental Psychology: Human Perception and Performance 1979;5:692–700.

[7] Brunnstrom S. Movement therapy in hemiplegia. New York: Harper and Row, 1970.

[8] Carr L, Shepherd R, Ada L. Spasticity: research findings and implications for intervention. Physiotherapy 1995;81:421–9.

[9] Colebatch JG, Gandevia SC. The distribution of muscular weakness in upper motor neuron lesions affecting the arm. Brain 1989;112:749–63.

[10] Desmurget M, Epstein CM, Turner RS, Prablanc C, Alexander GE, Grafton ST. Role of the posterior parietal cortex in updating reaching movements to a visual target. Nature Neuroscience 1999;2:563–7.

[11] Farmer SF, Harrison LM, Ingram DA, Stephens JA. Plasticity of central motor pathways in children with hemiplegic cerebral palsy. Neurology 1991;41:1505–10.

[12] Feldman AG, Levin MF. The origin and use of positional frames of reference in motor control. Behavioral and Brain Sciences 1995;18:723–806.

[13] Filloux FM. Neuropathophysiology of movement disorders in cerebral palsy. Journal of Child Neurology 1996;11:S5–S12.

[14] Goodale MA, Pelisson D, Prablanc C. Large adjustments in visually guided reaching do not depend on vision of the hand or perception of target displacement. Nature 1986;320:748–50.

[15] Katz RT, Rymer WZ. Spastic hypertonia: mechanisms and measurement. Archives of Physical Medicine and Rehabilitation 1989;70:144–55.

[16] Lance J. Symposium synopsis. In: Feldman R, Young R, Koella W, editors. Spasticity disordered motor control. Chicago: Yearbook Medical, 1980:485–94.

[17] Levin MF. Interjoint coordination during pointing movements is disrupted in spastic hemiparesis. Brain 1996;119:281–93.

[18] Mon Williams M, Tresilian JR, Wann JP. Perceiving limb position in normal and abnormal control: an equilibrium point perspective. Human Movement Science 1999;18:397–419.

[19] Pelisson D, Prablanc C, Goodale MA, Jeannerod M. Visual control of reaching movements without vision of the limb. II. Evidence of fast unconscious processes correcting the trajectory of the hand to the final position of a double-step stimulus. Experimental Brain Research 1986;62:303–11.

[20] Pierrot DE. Transmission of the cortical command for human voluntary movement through cervical propriospinal premotoneurons. Progress in Neurobiology 1996;48:489–517.

[21] Pisella L, Grea H, Tilikete C, Vighetto A, Desmurget M, Rode G, Boisson D, Rossetti Y. An 'automatic pilot' for the hand in human posterior parietal cortex: toward reinterpreting optic ataxia. Nature Neuroscience 2000;3:729–36.

[22] Prablanc C, Martin O. Automatic control during hand reaching at undetected two-dimensional target displacements. Journal of Neurophysiology 1992;67:455–69.

[23] Roby-Brami A, Fuchs S, Mokhtari M, Bussel B. Reaching and grasping strategies in hemiparetic patients. Motor Control 1997;1:72–91.

[24] Shumway-Cook A, Woollacott M. Motor control: theory and practical applications. Baltimore: Williams and Wilkins, 1995.

[25] Soechting JF, Lacquaniti F. Modification of trajectory of a pointing movement in response to a change in target location. Journal of Neurophysiology 1983;49:548–64.

[26] Steenbergen B, Hulstijn W, Lemmens IH, Meulenbroek RG. The timing of prehensile movements in subjects with cerebral palsy. Developmental Medicine and Child Neurology 1998;40:108–14.

[27] Steenbergen B, Van Thiel E, Hulstijn W, Meulenbroek RG. The coordination of reaching and grasping in spastic hemiparesis is characterized by segmentation. Human Movement Science 2000;19:75–105.

[28] Stein RB. Presynaptic inhibition in humans. Progress in Neurobiology 1995;47:533–44.

[29] Tang A, Rymer WZ. Abnormal force–EMG relations in paretic limbs of hemiparetic human subjects. Journal of Neurology, Neurosurgery and Psychiatry 1981;44:690–8.

[30] Thilmann AF, Fellows SJ, Garms E. Pathological stretch reflexes on the 'good' side of hemiparetic patients. Journal of Neurology, Neurosurgery and Psychiatry 1990;53:208–14.

[31] Trombly CA. Deficits of reaching in subjects with left hemiparesis: a pilot study. The American Journal of Occupational Therapy 1992;46:887–97.

[32] Van Thiel E, Meulenbroek RG, Hulstijn W, Steenbergen B. Kinematics of fast hemiparetic aiming movements towards stationary and moving targets. Experimental Brain Research 2000;132:230–42.