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A general deficit of the 'automatic pilot' with posterior parietal cortex lesions?

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Abstract

Lesions of the parieto-occipital junction (POJ) in humans cause gross deviations of reaching movements and impaired grip formation if the targets are located in the subjects' peripheral visual field. Movements to central targets are typically less impaired. This disorder has been termed "optic ataxia". It has been suggested that a general deficit of online corrections under central as well as peripheral viewing conditions might be sufficient to explain this discrepancy. According to this hypothesis, patients with optic ataxia should demonstrate an impaired online correction of grip aperture under central viewing conditions if the target object changes its size during the grasping movement. We investigated this prediction in a patient with optic ataxia (I.G.) in a virtual visuo-haptic grasping task. We imposed an isolated need for online corrections of the hand aperture independently of positional changes of the target object. While we found some general inaccuracies of her grasping movements, the patient did not show a specific impairment of online adjustment of grip aperture. On the contrary, I.G. smoothly adjusted her grip aperture comparable to healthy subjects. A general deficit of fast movement correction affecting targets in peripheral as well as central visual fields thus does not appear to account for the overt visuomotor deficits in optic ataxia. Rather, it seems more likely that an anatomical dissociation between visuomotor pathways related to actions in the central and in the peripheral visual field underlies the dissociation of visuomotor performance depending on the retinotopic target position in optic ataxia.

Keywords: Grasping; Visuomotor; Optic ataxia; Dorsal stream; Prehension

1. Introduction

Lesions involving the parieto-occipital junction (POJ) on the lateral and medial aspect of the human brain typically lead to a remarkable disruption of visuomotor coordination called 'optic ataxia' (Karnath & Perenin, 2005). These patients reveal large deviations of pointing and reaching movements and an inappropriate grip scaling to visual targets and objects. The deficit is typically restricted to targets in the peripheral visual field whereas movements to foveated targets are comparable to the performance of healthy subjects (Bálint, 1909; Himmelbach & Karnath, 2005; Holmes, 1918; Jackson, Newport, Mort, & Husain, 2005; Jakobson, Archibald, Carey, & Goodale, 1991; Jeannerod, 1986; Karnath & Perenin, 2005; Khan et al., 2005; Khan, Pisella, Rossetti, Vighetto, & Crawford, 2005; Milner et al., 2001; Milner, Paulignan, Dijkerman, Michel, & Jeannerod, 1999; Perenin & Vighetto, 1988; Pierrot-Deseilligny, Gray, & Brunet, 1986; Revol et al., 2003).

The typically observed dissociation between the peripheral and central visual field has long been neglected and optic ataxia has been regarded as a general visuomotor disorder of goal-directed movements. This interpretation of optic ataxia was also improperly perceived in the common understanding of the 'Two visual systems hypothesis' put forward by Milner and Goodale (1995). Many studies referring to this hypothesis disregarded the already well-known behavioral difference between movements to foveated and movements to peripheral targets by claiming that the dorsal pathway represents an indispensable structure for

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all visuomotor actions. Just recently, the dissociation between central and peripheral targets has been rediscovered and a distinguished publication by Pisella et al. (2000) offered a new theoretical approach to this phenomenon. The authors revealed an inability to perform fast corrective movements if a visual target changed its position after movement onset in a patient with extensive bilateral lesions of the posterior parietal cortex PPC and the superior occipital cortex (I.G.). In the same patient this deficit was also observed during reaching movements by the same group (Gréa et al., 2002). Resuming their findings, the authors suggested the existence of an 'automatic pilot' for the hand which unconsciously adjusts ongoing movements in flight and seems to be damaged due to the bilateral occipito-parietal lesions in the reported case. In accordance with their findings and conclusions Desmurget et al. (1999) already reported a disruption of online adjustments in healthy subjects upon transcranial magnetic stimulation (TMS) of the PPC. While stimulating the left PPC they found a pronounced effect on pointing movements of the right but not of the left hand. Interestingly, this lateralization mimicked the corresponding observation of a hand-effect in patients with optic ataxia after unilateral lesions of the left parieto-occipital cortex (Himmelbach & Karnath, 2005; Khan et al., 2005; Perenin & Vighetto, 1988; Rondot, de Recondo, & Dumas, 1977).

The finding of a damaged 'automatic pilot' bears the potential to explain the dissociation between movements to peripheral and foveated targets in patients with optic ataxia if we agree on the assumption that movements to peripheral targets are less accurate from the very beginning and thus rely more on in flight corrections than movements to foveated targets (Glover, 2003; Rossetti, Pisella, & Vighetto, 2003). Indeed, in normal subjects movements to peripheral targets are generally less precise than trajectories to foveated targets (e.g. Bock, 1986, 1993; Henriques & Crawford, 2000; Henriques, Klier, Smith, Lowy, & Crawford, 1998; Khan, Lawrence, Franks, & Buckolz, 2004; Postma, Sterken, de Vries, & de Haan, 2000; Prablanc, Echallier, Komilis, & Jeannerod, 1979). However, whereas some studies simply reported a directionally unspecific increase of the absolute pointing error (Postma et al., 2000; Prablanc et al., 1979), most of these revealed a horizontal overshoot, i.e. a bias away from the fixation position (Bock, 1986, 1993; Henriques et al., 1998; Henriques & Crawford, 2000; Khan et al., 2004). Thus, the directional bias of movements to peripheral targets in healthy humans seems to be contrary to the pathological reaching bias towards the point of fixation in patients with optic ataxia (Carey, Coleman, & Della, 1997; Jackson et al., 2005; Ratcliff & Davies-Jones, 1972). Moreover, deficits of online adjustments in a patient with optic ataxia so far have only been demonstrated for pointing and reaching movements (i.e. proximal movement components) (Gréa et al., 2002; Pisella et al., 2000). However, we know that patients with optic ataxia typically demonstrate impairments of grip formation and hand orientation (i.e. distal movement components) as well (Jakobson et al., 1991; Jeannerod, 1986; Jeannerod, Decety, & Michel, 1994; Milner et al., 2001; Milner, Dijkerman, McIntosh, Rossetti, & Pisella, 2003; Perenin & Vighetto, 1988). This is especially true for patient I.G., the only patient who has been investigated in those two studies which led to the 'automatic pilot hypothesis' (Gréa et al., 2002; Pisella et al., 2000). The question thus arises whether these distal movement control deficits correspond with the 'automatic pilot hypothesis' and have to be attributed to deficiencies in online adjustment of hand formation?

In their investigation of grasping movements in I.G. Gréa et al. (2002) observed the initiation of a new grip formation concurrently to the execution of the corrective reaching movement. However, this new grip formation could be attributed to a complete re-start of the reaching movement aiming towards a new object position. Thus, it is unclear whether an observation of two peak apertures in perturbed trials in this experiment represents a specific deficit of the online control of finger formation. To decide whether or not such a defect is present in patients with optic ataxia after POJ lesions, an investigation of a change in object size without concomitant changes of the object's position is required. To investigate this question we employed a visuo-haptic virtual reality environment. The central idea of our experiment was to impose an isolated need for online corrections in the formation of grip aperture independently of positional changes of the target object. While object size could change, the targets remained in central vision throughout the ongoing grasping movement.

2. Methods

We investigated the 34-year-old right-handed female patient I.G. with bilateral parieto-occipital lesions due to ischemic strokes about 5 years before the present experiment (Fig. 1A). This patient suffers from chronic optic ataxia and has been investigated in numerous studies in recent years (e.g. Khan, Pisella et al., 2005; Milner et al., 2003; Pisella et al., 2000, 2004; Schindler et al., 2004). Her ataxic behavior thus is well documented in a number of different tasks, including pointing and reaching to changing target positions. For detailed clinical information please refer to Pisella et al. (2000). The patient's ataxic disorder was verified right before the present experiment: I.G. demonstrated gross misreaching when grasping with her right hand for a cylindrical object at random locations in her peripheral visual field. In contrast, her movements were accurate when she was allowed to fixate the object. For comparison, we recorded four healthy control subjects (two females, two males; age range 31–32 years) without any history of neurological or psychiatric disorders. All control subjects were right-handed and had normal or corrected to normal vision. The participants gave their informed consent to participate in the study which has been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

All participants performed a grasping task using the index finger and thumb of their right hand. The subjects' fingers were connected to two PHANToM^{TM} devices providing a translational force feedback to each finger individually (Fig. 1B). Each PHANToM^{TM} had 6 degrees of freedom and the subjects were able to move their fingers and hand essentially unrestricted performing a precision grip in front of them. The visual stimuli were stereoscopically rendered using OpenGL and an SGI Octane II workstation. They were presented using an upside down mounted monitor above a horizontal mirror. The participants looked through shutter glasses (Crystal Eyes^{TM}, Stereographics, Inc.) at the mirror which provided them with different stereo views for the left and right eye resulting in a 3D presentation. All stimuli were perceived as being located at an approximate distance of 50 cm from the viewer. The finger positions were represented by two yellow spheres (finger spheres) with a diameter of 16 mm (approximately the size of a real finger tip). Virtual discs with an initial diameter of 36 or 44 mm and a thickness of 15 mm served as target objects. When the finger spheres touched the virtual objects, a synchronous force feedback was applied to the real fingers by the two PHANToMs^{TM} thereby generating a 3D visuo-haptic simulation of a true object. Head movements were restricted by a chin rest, but the subjects were free to move their eyes throughout the whole measurement.

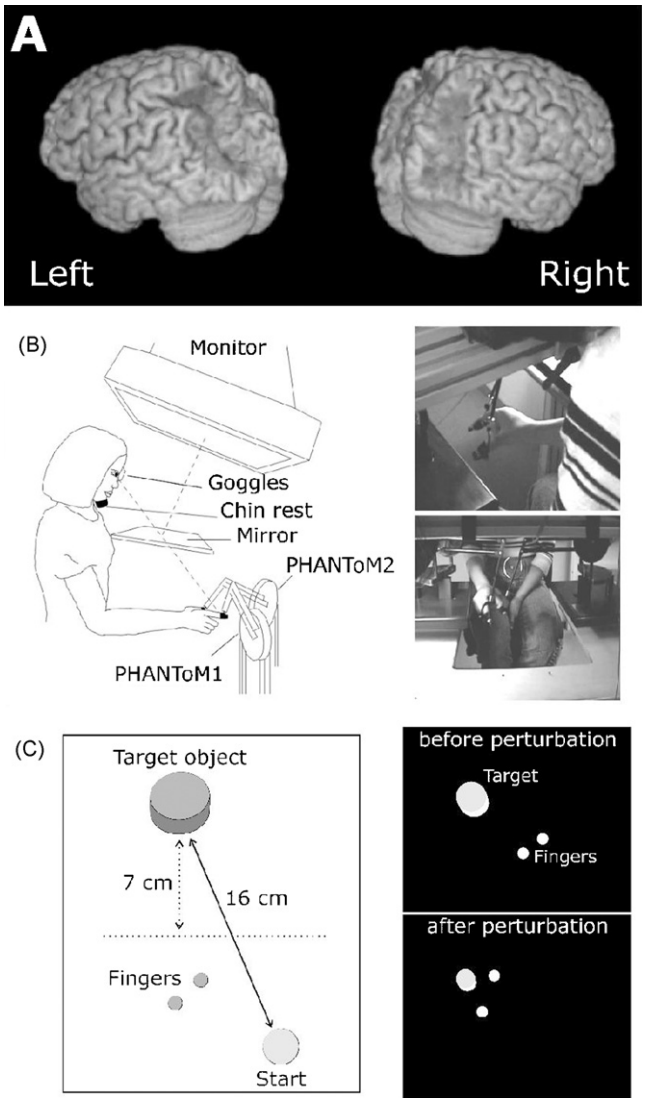

Fig. 1. (A) 3D view of the T1-weighted MRI-scan of I.G. taken at the time of the present experiment. (B) *Left*: Experimental setup, the subject sat in front of an upside down mounted monitor. Visual stimuli were seen by the subject via a horizontal mirror below the monitor through stereo goggles. Head position was restrained by a chin rest and haptic feedback was provided by two force-feedback devices (PHANToM^{TM}). The objects were perceived as being located below the mirror at a distance of about 50 cm from the subject's eyes. *Right*: The experimental setup as seen from behind and in front of the subject. The fingers were fixed to the PHANToM^{TM} devices which were placed on either side of the subject. (C) *Left*: A schematic drawing of the visual display: a start position was presented in the lower right quadrant until target presentation and reappeared after the fingers touched the object. The target disc was located 16 cm distant from the start position. The participants' fingers were represented by two spheres. A perturbation occurred in 50% of the trials, when the thumb crossed a virtual (here dotted) line 7 cm away from the disc's centre. This line was not visible in the actual display. *Right*: The visual display as seen by the subject during one trial of decreasing size perturbation. The two finger spheres moved toward the target which decreased in size when the thumb got closer than 7 cm to the target disc.

The participants rested their right hand at a starting position, indicated by a sphere on the right side of the display at a distance of 16 cm from the target position (Fig. 1C). Two thousand milliseconds after the fingers reached the starting position, the target disk was displayed and the participants initiated a grasping movement. The starting position disappeared at the time of target onset. The subjects were instructed to grasp the object firmly and transport it back to the starting position which reappeared when the fingers touched the virtual object. Each participant completed 240 trials. In 120 static trials the object's diameter remained unchanged throughout the movement. In the dynamic trials the object's diameter either suddenly increased to 52 mm ($n = 60$) or it decreased to 28 mm ($n = 60$). Please note that these changes took place in the visual as well as in the haptic dimension, thus no mismatch arose between the visual and haptic feedback in any condition. The sequence of trials was randomized and the perturbation occurred when the thumb got closer than 70 mm to the target's centre. Each participant performed a training session of at least 30 trials without perturbations. Kinematic data of the fingers' trajectories were recorded using the two PHANToM^{TM} devices. Movement onset was detected when thumb velocity exceeded 50 mm/s. The end of the movement was reached when either the thumb or the index finger were getting closer than 5 mm to the target disk. This criterion was chosen to avoid the influence of any haptic feedback on the analysis of online aperture scaling. Movement time (MT) was the time between the kinematic start and end of the grasping movement. The resultant velocity and acceleration of the hand's trajectory were calculated using the midpoint of the connecting line between the finger tips in each recorded frame. We further determined the onset time of the perturbation (PT) during the movement and the relative proportion of movement time spent after the perturbation (MTAP) when adjustments were expected to occur. In static trials without a perturbation this was simply the proportion of movement time after the thumb got closer than 70 mm to the target disk. To quantify the participants' ability to finally adjust their ongoing movement to a sudden perturbation, we calculated the grip aperture at a time point corresponding to 95% of movement time spend after the perturbation (AP95), thus very close to the final grip but lacking any haptic feedback. Individual aperture trajectories and the according velocity of the aperture change have been standardized to a number of 100 samples in the interval between PT and the kinematic end of the movement to calculate average aperture trajectories within each subject.

To avoid a bias due to few outliers, we rejected trials with extraordinary short/long MTAPs. These trials were identified by an MTAP more than 1.5 interquartile ranges below the first quartile or above the third quartile of the individual distribution in every subject. Thus, all following analyses were based on 212 trials in I.G. and 230–239 trials in the control subjects. In I.G. all but 4 of these outliers were caused by a very short MTAP when the index finger touched the target disk right after the thumb crossed the perturbation distance. This happened when (i) the aperture was quite large and (ii) the grip was oriented parallel to the movement direction. The results of I.G. were considered to be significantly different from the controls if they exceeded the 95% confidence interval (CI) of the control subjects.

3. Results

The qualitative inspection of the movement trajectories revealed differences between the investigated subjects. While three of the control subjects (C1, C2, C4) raised their fingers considerably above the 'ground', I.G. and C3 executed their movements primarily in the horizontal plane of the target object (Fig. 2). This behavior also explains the somewhat higher number of outliers in MTAP in I.G., since moving in the plane of the object increases the probability of a collision of the index finger with the object when the thumb just crossed the perturbation mark.

However, despite this obvious difference in the overall shape of movement trajectories, the optic ataxia patient was indistinguishable from controls with respect to most kinematic parameters. I.G.'s averaged reaction time (386 ms, S.D. 86) was not significantly different from the controls' mean value (343 ms, CI 257–428 ms). The same was true for MT in the respective experimental conditions (neutral: 567 ms, S.D. 79; controls' CI 539–776 ms; increasing size: 525 ms, S.D. 117; controls' CI 441–813 ms; decreasing size: 622 ms, S.D. 107; controls' CI

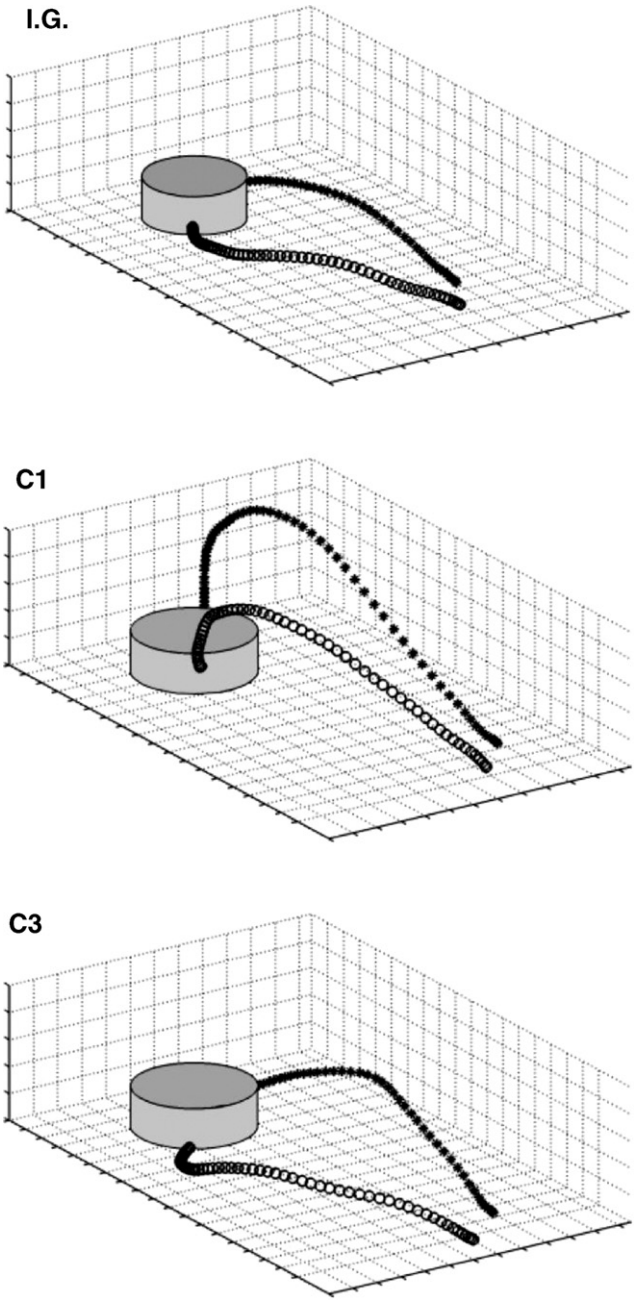

Fig. 2. Exemplary 3D trajectories of thumb (circles) and index finger (asterisks) of I.G. and two controls.

619–854 ms). MT was analyzed separately, as the different final target sizes in the dynamic trials might have had an impact on the overall MT while RT should not be influenced by the unknown upcoming experimental condition and final target size. The MTAP in I.G. was different from the controls' behavior in the static trials only (I.G.: mean 43.3%, S.D. 7.5; controls' CI 47.0–63.6%). In contrast, there was no significant difference in the dynamic conditions (increase I.G.: 37.7%, S.D. 8.3; controls' CI 46.6–71.6%; decrease I.G.: 48.6%, S.D. 7.6; controls' CI 40.2–58.9%).

The control subjects slowed down their movements after passing the perturbation distance. This behavior is reflected in a positive difference after the subtraction of the groups' mean hand velocity during the time interval after the perturbation from the mean velocity before the perturbation (increase: 140 mm/s CI 100–181 mm/s; decrease: 177 mm/s, CI 148–207 mm/s). The same pattern was observed in the static trials (neutral: 155 mm/s, CI 135–175 mm/s). Thus, this behavior does not seem to be a reaction to the perturbation but simply a pronounced deceleration in the later part of the movement. The analysis of the patient's data disclosed a decreased difference between the first and second part of the movement in all conditions. Although I.G. was also slowing down after crossing the perturbation distance, the respective mean differences exceeded the controls' confidence intervals (neutral: 92 mm/s, S.D. 36; increase: 60 mm/s, S.D. 50; decrease: 111 mm/s, S.D. 32). This discrepancy was not caused by a diminished overall mean velocity. The averaged hand velocity of the whole movement path of I.G. (neutral: 225 mm/s, 23 S.D.; increase: 239 mm/s, 32 S.D.; decrease: 209 mm/s, S.D. 31) lay well within the respective confidence intervals of the controls (neutral: 242 mm/s, CI 194–290 mm/s; increase: 248 mm/s, CI 190–307 mm/s; decrease: 213 mm/s, 161–265 mm/s).

I.G. was different from the controls in that she was less successful in grasping the object firmly. In comparison to controls, she had to perform a second approach to lift the object more often. However, although she was generally worse than controls there was no difference of the frequency of second attempts between static (I.G.: 15.2%; controls' CI 0.2–7.4%) and dynamic trials (I.G.: 16%; controls CI 0.02–10.3%). Her errors thus seem to reflect a general ataxic behavior compared to healthy subjects but not a specific deficit of movement adjustment related to target changes.

Finally, we analyzed the grip aperture in all subjects. A first inspection of the averaged standardized aperture trajectories after the perturbation revealed a smooth adjustment of I.G.'s hand shape after the perturbation (Fig. 3, left column). The average velocity of her grip aperture change showed no instantaneous deflection in the dynamic conditions indicating a continuous incorporation of the new object size into her movements. Her behavior was not distinguishable from the controls' performance. An exemplary control subject is shown in the right column of Fig. 3. For statistical analysis, we compared the mean grip aperture at 95% MTAP (AP95) in all subjects. First, we compared AP95 between neutral trials with an object size of either 36 or 44 mm by means of a Mann–Whitney U-test in I.G. This comparison revealed a highly significant difference between trials directed towards the two different (unchanged) object sizes ($U = 389$, $p < 0.001$; Fig. 4). We performed the same comparison between trials directed towards the two different object sizes in the dynamic conditions. If I.G. would not have been able to adjust her grip aperture to the increased or decreased object size after the perturbation, we should have observed the same significant difference between trials with different initial sizes in the dynamic conditions. This was not the case. In fact, we found similar values of AP95 with the same final size (increase: 52 mm; decrease: 28 mm) independently of the different initial sizes at the beginning of the trial (increase: $U = 159$, $p = 0.124$; decrease: $U = 297$, $p = 0.082$; Fig. 4). All control subjects showed the same pattern with significant differences

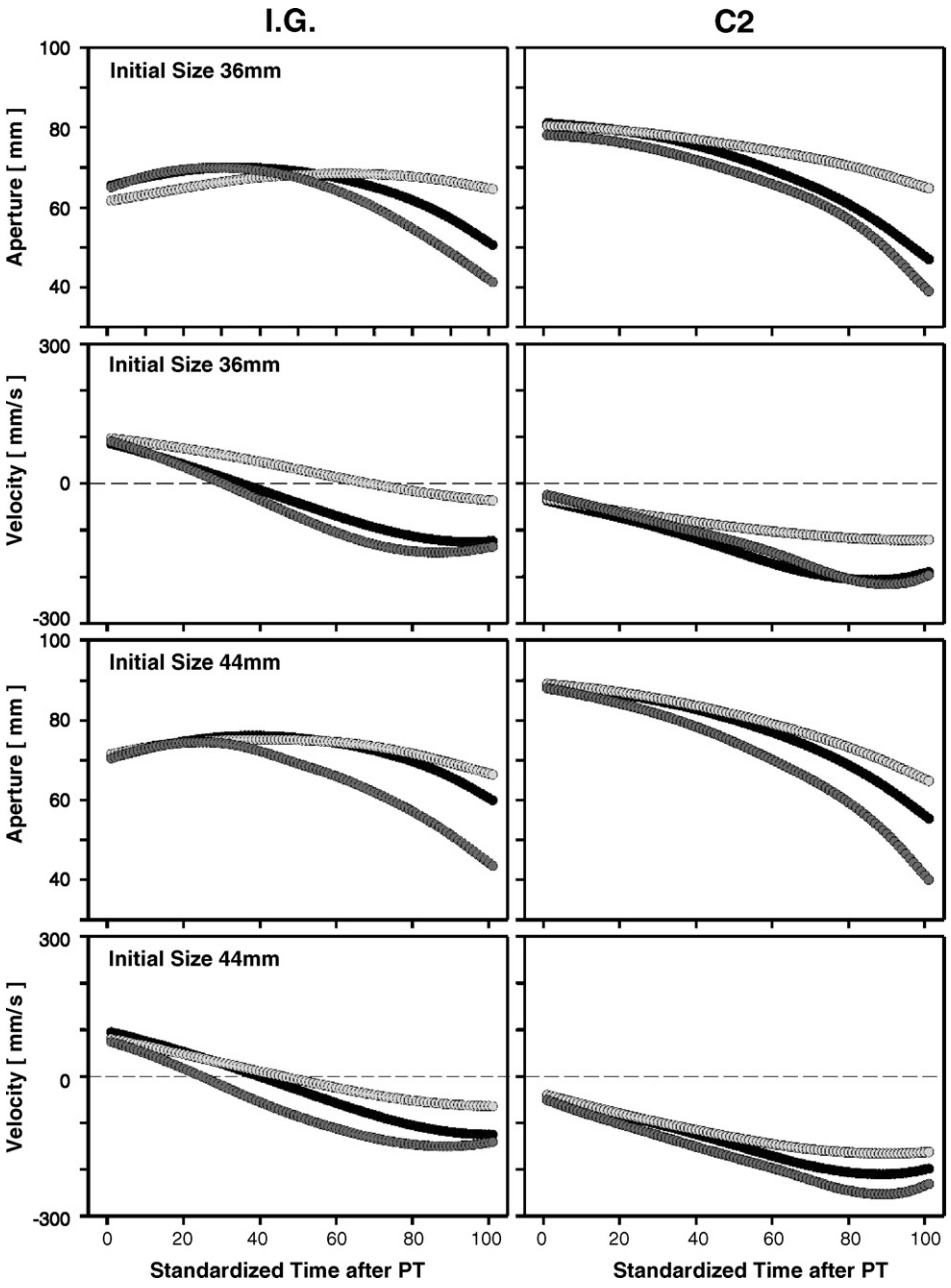

Fig. 3. Mean aperture and velocity of aperture change of I.G. (left column) and one exemplary control subject (right column) plotted against standardized movement time between the onset of the perturbation (PT) and movement end. Trials with different object sizes at the beginning (initial size 36 or 44 mm) have been plotted separately because of differences in the general aperture scaling. Black: neutral, dark grey: decrease, light grey: increase.

between trials with different initial (and final) sizes in the static condition ($U = 112$–297, $p < 0.001$ in all control subjects) and insignificant findings with the same comparison in the dynamic conditions (increase: $U = 316$–386, $p = 0.155$–0.985; decrease: $U = 339.5$–375, $p = 0.102$–0.282).

4. Discussion

The aim of our study was to investigate whether or not a deficit of online correction of grip formation exists in optic ataxia when grasping movements are performed under central viewing conditions without changes of the object's position. It was interesting to examine this question particularly in patient I.G. since deficient online adjustment of proximal movement components has been documented previously in this patient (Gréa et al., 2002; Pisella et al., 2000) as well as deficits of grip scaling to objects under peripheral viewing conditions (Milner et al., 2001). Thus, considering the explicit assumptions of the online correction hypothesis (Glover, 2003; Rossetti et al., 2003), we expected to find impaired online adjustments of distal hand aperture upon sudden changes of the target object's size.

We found general inaccuracies of grasping movements, expressed as an increased frequency of insecure grips in static *and* dynamic conditions as well as a reduced slowing of the trans-

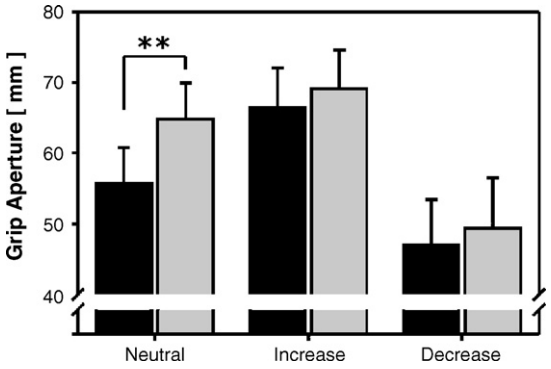

Fig. 4. Average grip aperture of the patient I.G. at 95% of MTAP with standard deviation. Trials with an initial object size of 36 mm are depicted in black, trials with an initial size of 44 mm are depicted in grey. Note that in the neutral trials the initial and final object size were the same. In contrast, in condition 'increase' all trials ended with a final object size of 52 mm, while in condition 'decrease' all trials ended with a final object size of 28 mm. $^{**}p < 0.001$ Mann–Whitney *U*-test.

port phase in the later part of the movements in all experimental conditions. However, no specific deficits of online adjustment of grip aperture upon in flight changes of the target size were observed. I.G.'s average grip aperture development and the late grip size just before touching the object after changes of the targets' diameter were indistinguishable from controls. Thus, the previously observed deficit of online movement correction in this patient (Gréa et al., 2002; Pisella et al., 2000) seems to be restricted to her pointing and reaching movements.

Grip aperture adjustments without changes of the object's spatial position have been investigated also in healthy subjects (Bock & Jüngling, 1999). Presenting luminous virtual objects Bock and Jüngling (1999) observed a latency of the corrective movement after a change of target size similar to the reaction time upon the initial target presentation. Their findings extended previous reports about online grip adjustments which were confounded by simultaneous changes of the target's position (Paulignan, Jeannerod, MacKenzie, & Marteniuk, 1991). Just recently, these reports have been complemented by two studies investigating the consequences of TMS application for the execution of online adjustments in grasping movements (Glover, Miall, & Rushworth, 2005; Tunik, Frey, & Grafton, 2005). Both groups applied TMS over the anterior IPS subsequently to movement onset and found a reduced adjustment of hand aperture to the new object size which has changed at movement onset. Interestingly, applying TMS so early during movement execution led to a decline of adjustments for changes from small to large objects only (Glover et al., 2005). In contrast, the adjustment to a change from large to small objects was impaired only if TMS was applied after 50% MT. This finding corresponds to a later grip size correction upon changes from a large to a small object and the earlier occurrence of corrective movements in the reverse change in the report by Paulignan et al. (1991). Tunik et al. (2005) investigated size increases only. Their finding of a significant impact of TMS at movement onset confirmed the results of Glover et al. (2005).

Both TMS studies carried out in healthy subjects found online adjustments of grasping not completely abolished, but only delayed or reduced (Glover et al., 2005; Tunik et al., 2005). In contrast, Desmurget et al. (1999) reported a "disruption" of any fast corrections of pointing movements upon the application of inhibitory TMS in four out of five healthy subjects. In agreement with the latter results, the findings in patient I.G. documented in preceding reports argue for an abolishment of online corrections in pointing and reaching movements (Gréa et al., 2002; Pisella et al., 2000). In contrast, we found no evidence for a specific impairment of in flight adjustments of distal grip formation in this patient. Thus, a disturbance of the normal function of the lateral PPC either by a permanent damage due to stroke lesion (I.G.) or a 'virtual lesion' by application of TMS seem to have less impact on distal movement components than on proximal components.

But why were there no impairments at all in patient I.G. if TMS application over the regions which are damaged in the patient led to at least some effects in online correction in healthy subjects? Remote effects of TMS in structures of the brain which are connected with but are quite distant from the TMS application site have been demonstrated (Bestmann, Baudewig, Siebner, Rothwell, & Frahm, 2003, 2004). The effect of TMS application over the lateral PPC on pointing and reaching adjustments might be mediated by a direct interference with the processing of the proximal movement component (direction, amplitude) at this site. Contrary, the smaller effect of TMS application on hand aperture adjustments might be mediated by an indirect interference with information processing in connected areas subserving the distal movement component in grasping. Neuroanatomical distinctions between reaching and grasping pathways have been suggested based on experimental findings in humans and monkeys (Jeannerod, Arbib, Rizzolatti, & Sakata, 1995). Thus, it is possible that although the lesion in patient I.G. encloses the stimulation site of TMS interference studies of pointing (Desmurget et al., 1999) and grasping (Glover et al., 2005; Tunik et al., 2005) in healthy subjects, distant areas mediating the delay of grip aperture adjustment might be less affected by her lesions. Furthermore, we must keep in mind potential changes in the functional organization of the patient's brain. Several years have gone since she suffered the stroke. It is possible that functional reorganization might have improved her online capabilities for distal movement components, while her capabilities with respect to the control of the proximal movement component might have profited less from these changes.

Finally, a decisive difference between the previous observation of inaccurate grasping of peripheral objects (Milner et al., 2001) and the present report of unimpaired grip aperture adjustments must be mentioned. Whereas the former investigation used real objects, we employed a virtual reality setup that provided the subjects with congruent visual and haptic feedback. Might this difference explain the discrepant findings? A recent investigation of kinematic differences in healthy subjects and hemiparetic stroke patients revealed only slight differences between movement control under real physical conditions on the one hand and a visual virtual environment lacking any haptic feedback on the other (Viau, Feldman, McFadyen, & Levin, 2004). Since our present experiment included valid

haptic feedback concurrent with the 3D presentation of the targets, we would even expect less differences to a real situation. Nevertheless, our setup certainly does not equate to a real environment and thus, general movement impairments which have been observed in I.G. in the present investigation might be attributed to the specific environment used here.

In conclusion, the present findings suggest that a general deficit of fast movement correction affecting targets in peripheral as well as central visual fields (Glover, 2003; Rossetti et al., 2003) does not seem to account for the overt visuomotor deficits in optic ataxia. Previously observed deficits in grip scaling with respect to peripherally presented objects in I.G. (Milner et al., 2001) cannot be explained by a generally deficient automatic pilot including grip formation. While structures within those parts of the parieto-occipital cortex which are permanently damaged in I.G. are necessary for the in flight control of proximal movement components, they seem to be less important for controlling distal movement components. Rather, it seems more likely that an anatomical dissociation between visuomotor pathways related to actions in the central and in the peripheral visual field underlies the dissociation of visuomotor performance depending on the retinotopic position of the target object in patients with optic ataxia. Supporting evidence for this conclusion has recently been obtained in a functional imaging experiment in healthy subjects (Prado et al., 2005). The results suggested a specific parieto-frontal pathway for peripheral pointing movements that exceeded the network involved in pointing movements to foveated targets. The authors found an activation of the POJ and the dorsal premotor cortex bilaterally for pointing movements to peripheral targets only. This finding in healthy subjects corresponds remarkably well to the typical lesion site causing optic ataxia (Karnath & Perenin, 2005).

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