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A hand and a field effect in on-line motor control in unilateral optic ataxia

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ABSTRACT

Patients with bilateral optic ataxia fail to show rapid perturbation-induced corrections during manual aiming movements. Based on this, it has been proposed that this pathology results from a disruption of processes of on-line motor control in the posterior parietal cortex (PPC). Here, we show that on-line motor control performance in a patient with unilateral optic ataxia is similar to that of pointing towards stationary targets in peripheral vision, showing the same combination of hand and field effects. We also show that in the patient, manual correction towards his ataxic field was possible only when a preceding saccade (100 msec earlier) rapidly provides foveal information about the new target location. In control subjects, manual correction was often, but not necessarily preceded by a saccade. These results allow us to put forward a model of visuo-manual transformation, which involves updating of the reach plan based on the target–eye error, and rely upon two dissociated spatial representations (of the hand and of the target, respectively) within the PPC.

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Optic Ataxia (OA) was initially described as a visuo-motor coordination deficit, comprising a component of Bálint's syndrome (Bálint, 1909). Garcin et al. (1967) isolated this deficit, showing that it may appear independent of the other symptoms and without any primary visual, motor, or proprioceptive deficits. OA usually follows lesions of the superior lobule of the posterior parietal cortex (PPC, Perenin, 1997) or of the parieto-occipital junction (POJ, Karnath and Perenin, 2005). Patients with unilateral lesions show large inaccuracies when asked

to reach to an object presented in their contralesional/ataxic visual field ("field effect"), and also when they use their contralesional/ataxic hand ("hand effect"). These reaching inaccuracies are greatest when the two effects are combined, that is, when the contralesional hand is used to reach within the contralesional visual field (Perenin and Vighetto, 1988).

In general, reaching in central vision is spared (Perenin and Vighetto, 1988). However, findings from the single-case study of Pisella et al. (2000) (see Rossetti and Pisella, 2003, for

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a second patient) revealed that OA is also associated with a dramatic visuo-motor deficit in central vision, consisting in a lack of automatic corrections when the target position was unexpectedly modified at the onset of the reaching movement. Indeed, in such “target jump” experimental conditions, a fast “automatic pilot” allows healthy subjects to adjust their reaching trajectory, thus correcting the predefined motor plan according to the new target location (Goodale et al., 1986; PÉlisson et al., 1986; Pisella et al., 2000; Day and Lyon, 2000; Gréa et al., 2002; Cressman et al., 2006). The complex computations required to modify the ongoing action might be expected to increase the movement duration required to reach the target. However, in healthy subjects, these movement corrections can be implemented on-line (Goodale et al., 1986; PÉlisson et al., 1986; Pisella et al., 2000; Gréa et al., 2002). In contrast, patients with bilateral lesions of the PPC demonstrated slow intentional corrections, which drastically increased movement time (Pisella et al., 2000; Gréa et al., 2002; Rossetti and Pisella, 2003). Furthermore, while movement corrections in normal subjects are made in a smooth and automatic manner, gradually updating the movement trajectory, patients with bilateral OA display a trajectory composed of two distinct movements (Gréa et al., 2002). The first movement ends at the initial position of the target. These delayed movement corrections are also observed in the behaviour of OA patients when asked by a clinician to reach and grasp a pen in peripheral vision (Vighetto, 1980).

It is important to stress that the target jump paradigm was designed to study an ecological process; the planning of visually guided movements is always suboptimal, and the accuracy of reaching movements is especially degraded when the target is seen in peripheral vision. Small target jumps increase the intrinsic error of motor plans (Prablanc et al., 1979). Automatic correction mechanisms are constantly required for the performance of accurate visually guided reaching movements (Goodale et al., 1986; PÉlisson et al., 1986), and therefore require a fast comparison of target and hand locations. Many studies have demonstrated the involvement of the PPC in automatic corrections (Desmurget et al., 1999; Pisella et al., 2000; Gréa et al., 2002; Rossetti and Pisella, 2003). But what exactly is the role of the PPC in performing these target-hand comparisons, and at which stage(s) of the movement? Is it the processing of the target location and/or the processing of hand location? Until now, deficits in visuo-manual control have mostly been studied in patients with bilateral lesions of the PPC. One way to address these questions is to test whether the lack of automatic corrections in unilateral OA depends on the direction of the target jump and/or on the hand used. In the present study, we therefore investigated the performance of a patient with unilateral optic ataxia in a target jump pointing task, using both his contralesional and ipsilesional hands in both visual fields (contra and ipsilesional), in order to dissociate between deficits of on-line manual control linked to the direction of the target jump (“field effect”) and to the use of the ataxic hand (“hand effect”). In addition, we recorded the eye movements of patient CF and of a group of control subjects during this target jump pointing task. We investigated whether saccades are systematically elicited when the jump brings the target to peripheral vision, and whether there is a functional link between the visual

capture and the manual capture of the target (cf. Fisk and Goodale, 1985; Neggers and Bekkering, 2001), that is, between the occurrence of saccade and pointing corrections.

1. Method

1.1. Subjects

CF is a right-handed male who suffered from a watershed posterior infarct (Fig. 1A), resulting in distributed and asymmetrical bilateral lesions of the parieto-occipital region (Brodmann’s areas 18, 19, 7, 5, and 2) with a minute extension into the semiovale centres and a parieto-frontal disconnection from intrahemispheric fibre lesions. At the time of testing, aged 27, CF showed optic ataxia predominantly with the left (contralesional) hand in the left (contralesional) visual field, thought to be the consequence of damage to Brodmann’s area 7 in the right hemisphere (larger than in the left hemisphere). He had no ataxia for targets in central vision, no purely motor, somatosensory, or visual deficits, and no sign of neglect. His left parietal lesion had no clinical expression in terms of right-sided sensori-motor deficits.

Five right-handed and age-matched (mean \pm SD = 27 \pm 4 years) control subjects performed the same pointing experiment as patient CF.

1.2. Materials

Subjects sat in front of a custom experimental device, comprising a high speed CRT screen (frequency: 160 Hz), a 21-inch touch screen using surface acoustic wave technology (Intellitouch™, ELO TouchSystems), coupled with a high spatial and temporal frequency stimulus presentation device (Visual Stimulus Generator™, ViSaGe, Cambridge Research Systems, Rochester, UK). A High Speed Video Eyetracker™, attached to a head and chin rest (Cambridge Research Systems, Rochester, UK), registered eye movements with an infra-red camera (sampling frequency: 250 Hz; resolution: 0.05°). The eyetracker, the ViSaGe, and the touch screen were synchronised by a custom software interface developed in our laboratory. The subjects’ eyes were at a distance of 37 cm from the screen.

1.3. Procedure

We used an experimental procedure similar to the “location-go” condition of Pisella et al. (2000, 1998). Subjects were asked to place their left or right index finger on a starting point at the bottom of the screen (Fig. 1B). After a random delay (800–1500 msec), one of the two possible target locations (at $\pm 5^\circ$ of visual angle with respect to the subject’s midline) was illuminated close to the top of the screen (27 cm from the starting position). Subjects were asked to fixate this target, and to reach and touch it with their index finger as fast and as accurately as possible. In 20% of the trials, the initial target turned off at the onset of the pointing movement (the release of the finger from the touch screen) and was immediately replaced by a target at the other location (target jump). Target jumps of 10° were produced in two directions: rightward for initial targets presented on the left (jumping into the right peripheral

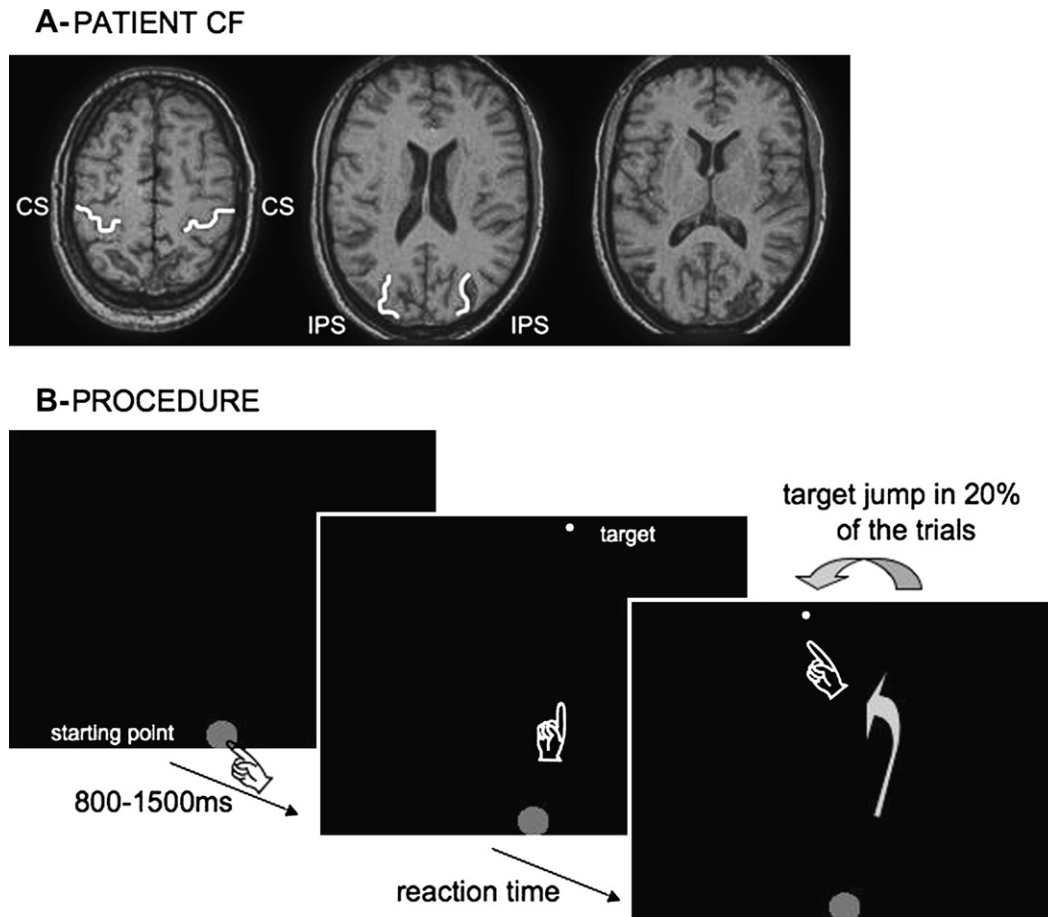


Fig. 1 – (A) Patient CF's MRI showing asymmetrical bilateral lesions of parieto-occipital regions (Brodmann's areas 18, 19, 7, 5, and 2). (B) Description of the experimental procedure. When the subject had placed his index finger at the starting position for a random time between 800 msec and 1500 msec, the target appeared at one of the two possible locations (right or left). The subject had to reach and point to the target as fast and as accurately as possible. In 20% of the trials, the target was displaced towards the other possible target position (from right to left on the figure) at movement onset.

visual field) and leftward for initial targets presented on the right (into the left peripheral visual field). In response to the target jumps, subjects were required to correct their movement in order to point to the new target location. In the remaining 80% of trials, the target remained stationary.

The subjects performed a total of 400 pointing trials. In order to elicit fast movements and automatic pointing corrections, an auditory signal warned the subjects if their movement duration exceeded 300 msec in a first session (200 trials), and 250 msec in a second session (200 trials). Depending on the block of trials, pointing movements were performed with either the left or the right hand, following an ABBA blocked design. The direction of the target jump was varied pseudo-randomly within each block and pointing session.

Movement durations were computed from the touch screen data as the difference between the time of finger release from the starting point (at the bottom of the screen) and the time of the finger touching the screen. The touch screen also provided the horizontal x-coordinate of the endpoints, which allowed us to categorise the movements post hoc as corrected or uncorrected when a target jump occurred. If the endpoint was outside the spatial range of the endpoints of movements

made towards the stationary targets, it was considered to be a corrected movement. The percentage of manual corrections (corrected movements) made towards the new target location was then computed for each condition of pointing hand and direction of target jump. Targets were already foveated when pointing movements were initiated. In the case of target jumps, the horizontal displacement (10°) of the targets could produce saccadic eye movements. The percentage of saccade occurrence towards the new target location as well as its amplitude and its time of occurrence relative to hand movement onset (i.e., time of target jump) and offset were also computed.

1.4. Statistical analyses

Statistical analyses were conducted separately for patient CF and for the control group. Non-parametric Chi2 (χ^2) analyses were performed on the occurrence of manual corrections and saccades. Factorial ANOVAs were performed on the spatial (amplitude) and temporal (latency and time interval between saccade onset and pointing offset) characteristics of the saccades taking into account two factors: the direction

of target jump (leftward or rightward) and manual correction (corrected or uncorrected pointing movements).

2. Results

2.1. Manual corrections

The patient exhibited significantly different manual correction patterns depending on the pointing hand and on the target jump direction (Fig. 2, top left corner). Statistical χ^2 analyses showed that there was a clear effect of both the hand used to point and the visual field to which the target jump occurred. The patient produced significantly fewer manual corrections when the target jumped towards his left (ataxic) visual field (relative to target jumps towards the right “healthy” field), regardless of whether the movement was performed with the ataxic ($\chi^2 = 4.7, p < .05$) or with the healthy hand ($\chi^2 = 11, p < .001$). He also produced fewer manual corrections when the movement was performed with the left (ataxic) hand, when the target jumped towards both the ataxic ($\chi^2 = 4.7, p < .05$) and the healthy field ($\chi^2 = 11, p < .001$). These hand and field effects were combined, in that the worst performance was observed in the condition where the target jumped towards the ataxic field and the pointing movement was performed with the ataxic hand (only 16.6% of movements were corrected in this condition). The best performance

was observed when the patient used his healthy hand and the target jumped towards his healthy visual field (77.1% corrected movements) and intermediate performance (39%) was observed in the two crossed conditions.

In addition, motor corrections involving a large increase of movement time with respect to stationary trials cannot be considered as “on-line” but rather as “slow corrections” (Pisella et al., 2000). When the target jumped towards his left (ataxic) field, only 54% of the corrected movements produced by patient CF were in the range of the movement times of the stationary trials (<400 msec). On the other side, when the target jumped to his right field, 86% of the corrected movements did not involve an increase of movement time, indicating that the patient mainly displayed “on-line” corrections in his ipsilesional visual field.

In contrast, the control subjects (Fig. 2, bottom left corner) did not show any difference in the percentage of manual corrections between conditions (61.6% on an average, all $\chi^2 < 1.4, p > .05$); they exhibited no differences in manual corrections for either hand used or for either target jump direction. Control subjects typically provided corrected movements with duration within the range of the movement times of the stationary trials.

2.2. Saccade occurrences

The control subjects did not show any differences in the percentage of saccade occurrences for either the hand used

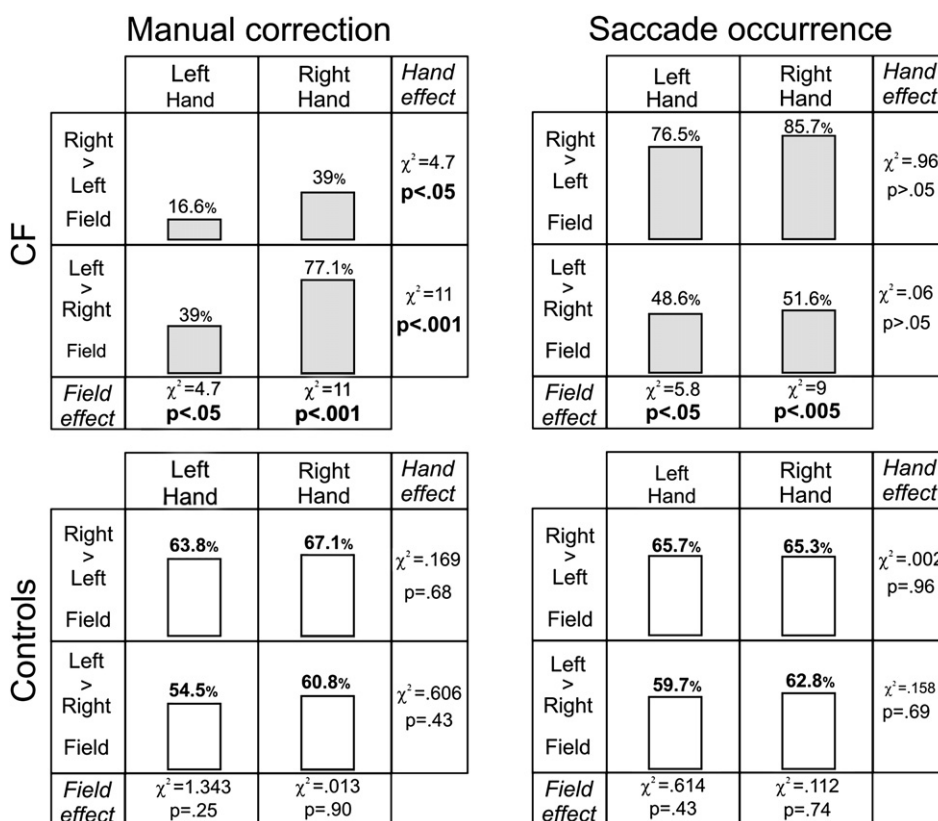


Fig. 2 – Percentages of trials with manual correction (left panel) and saccade occurrence (right panel) for the OA patient (CF) and for the control group. From the combination of trials according to the pointing hand (left or right) and the direction of the target jump (towards the left or towards the right field), χ^2 analyses were performed to test the hand effect and the field effect (see Section 1.4).

or the target jump direction (63.4% of saccade occurrence on an average, all $\chi^2 < 1$; Fig. 2, bottom right corner).

Patient CF produced significantly more saccades when the targets jumped towards his left (ataxic) visual field than towards his right visual field with both hands (right hand $\chi^2 = 9, p < .01$; left hand $\chi^2 = 5.8, p < .05$). There was no effect of the hand used on the occurrence of saccades for either target jump direction (rightward: $\chi^2 = .06, p > .05$; leftward: $\chi^2 = .96, p > .05$). The pattern of saccade occurrence thus did not show a hand effect and shows instead an “inverse” field effect (Fig. 2, top right corner): when the target jumped towards the ataxic field, more saccades were elicited and they had shorter reaction times than when the target jumped towards the right visual field (210 msec vs 270 msec, $F(1,69) = 22.97, p < .001$), without being correlated with more manual corrections.

2.3. Eye-hand coordination

In the control group, the saccade occurrence appeared to be predictive of a manual correction but not necessary for it (Fig. 3, right panel). Indeed, 93.5% of the corrected pointing movements were accompanied by a preceding saccade (with no effect of target jump direction). However, 6.5% of the manual corrections were performed without any saccades and, conversely, 15.6% of the pointing movements were uncorrected despite a saccade occurrence.

For patient CF, we found that saccade occurrence was predictive of a manual correction only for the right visual field (when the target jumped from the left to the right), but not for the left (ataxic) visual field. When the target jumped towards the right (healthy) visual field, 72.5% of corrected trials were preceded by a saccade compared to only 10.7% of uncorrected trials. In contrast, when the target jumped to the left (ataxic) visual field, both corrected and uncorrected movements were mostly preceded by a saccade (95% and 73.5% respectively, Fig. 3, left panel). In conclusion, for patient CF, the percentage of saccade occurrence was not predictive of

the percentage of manual correction when the target jumped into his ataxic visual field.

In order to understand whether the lack of manual correction when the target jumped towards the ataxic field resulted rather from the temporal or the spatial characteristics of the saccades in these conditions, we performed a factorial ANOVA on the saccadic parameters (saccade amplitude and the interval between the beginning of the saccade and the end of the hand movement) with the direction of target jump and the presence or absence of manual correction as factors. The hand was not included as a factor because no effects of the hand used on the saccade occurrence either in the patient or in the controls were observed.

The amplitudes of the leftward and rightward saccades were comparable in patient CF (no main effect of the target jump direction, $F(1,83) = .0002, p = .99$). Saccades were larger when the pointing movement was corrected (main effect of manual correction, $F(1,83) = 8.6, p < .005$). However, there was no interaction effect ($F(1,83) = .08, p > .05$), meaning that saccade amplitude cannot explain the effect of field on the performance of manual corrections in patient CF.

We calculated the interval between the time of saccade onset and the time of endpoint for trials. Note that since the target was presented in central vision no saccade precedes the pointing movement onset. A saccade may occur in reaction to the target jump (which was synchronised on pointing movement onset). This interval is therefore an indicator of how long the system deals with central vision of the target, when a saccade was performed during pointing movement execution. We tested this interval for differences with and without manual corrections, and for rightward and leftward target jumps. As can be seen in Fig. 4, the controls performed independent of the target jump direction. However, they showed a main effect on the production of manual correction ($F(1,188) = 5.42, p < .05$): manual corrections occurred with a longer interval (difference of 20 msec on an average) for corrected compared to uncorrected pointing movements (primarily due to an increase of pointing movement time).

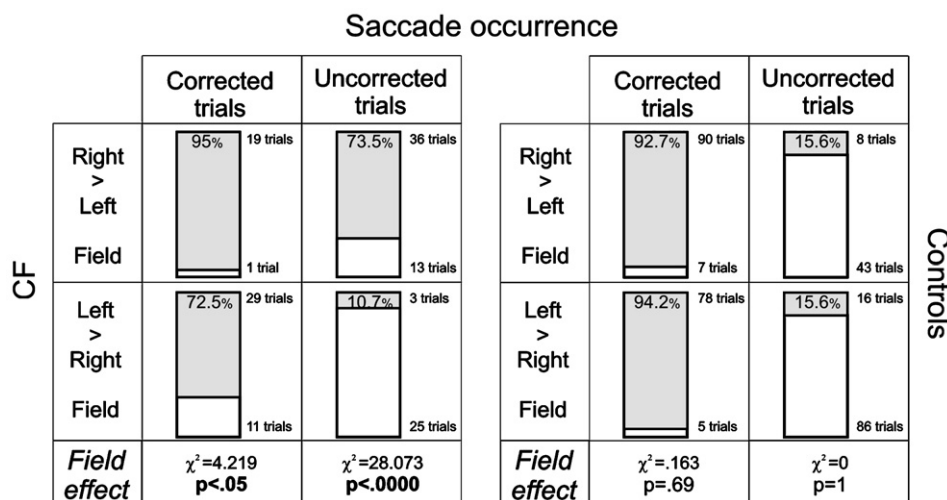


Fig. 3 – Percentages of trials with (grey) and without (white) saccade occurrences for the OA patient (CF) and for the control group according to the presence of manual corrections (corrected or uncorrected trials), and to the direction of the target jump (towards the left or towards the right field). χ^2 Analyses were performed to test the field effect.

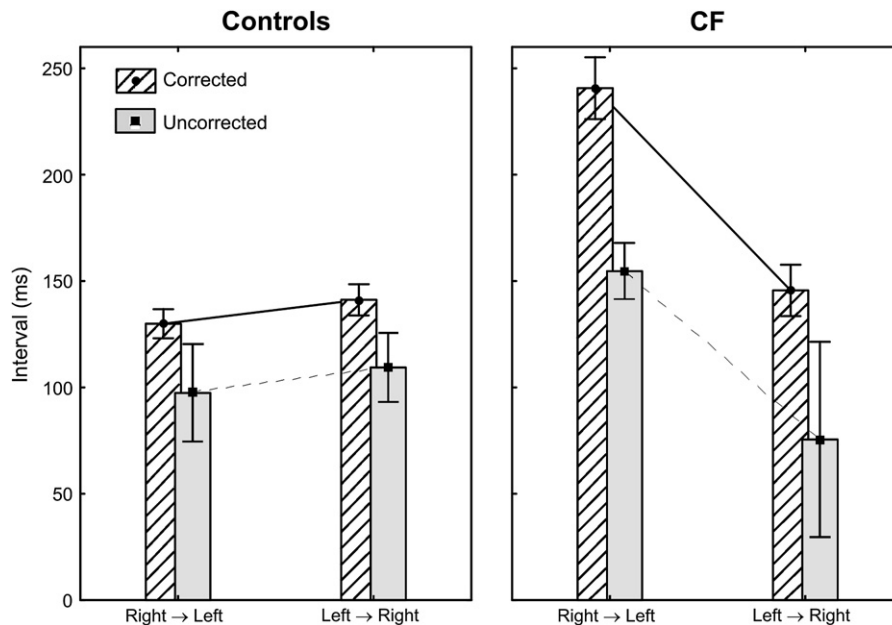


Fig. 4 – Mean and standard error of the time interval between the beginning of the saccade and the end of the pointing movement reflecting the duration of central vision of the new target location available during pointing execution. This interval is displayed for the OA patient (CF) and for the control group for the two directions of target jump and for whether a manual correction was produced (stripes) or not (plain grey). Patient CF showed a main effect of the presence of a manual correction and a main effect of the direction of the target jump (more time was needed when the target jumped into the left, ataxic field).

This main effect of manual correction was also present for patient CF ($F(1,71) = 6.68, p < .05$), but was accompanied by a significant main effect of the target jump direction ($F(1,71) = 8.32, p < .005$) and with no interaction effect ($F(1,71) = .07, p > .05$). Indeed, within the same time interval in which control subjects corrected their movements (about 150 msec), patient CF could correct his pointing movements to the right but not to the left (ataxic) visual field. When the target jumped towards his left (ataxic) visual field, patient CF was able to produce a few manual corrections ($n = 20$, corresponding to 27.8% of manual corrections, see Figs. 2 and 3) but only if a saccade was produced within a time interval of 100 msec, longer that is, only when possibility of a saccade was produced with short latency to provide possibility of foveal updating of the target location.

3. Discussion

Previous studies have demonstrated the lack of an automatic pilot for the hand in bilateral optic ataxia and have therefore put forward the specific involvement of the PPC in on-line motor control (Pisella et al., 2000; Milner et al., 2003, 2001; Gréa et al., 2002; Rossetti et al., 2003, 2005; Glover, 2003; Tunik et al., 2005; Himmelbach et al., 2006). The first main result of the present study is the confirmation of deficits in the automatic pilot for the hand consecutive to lesion of the PPC in a patient with unilateral optic ataxia. Pointing movements were corrected less often when the target jumped from central vision towards the ataxic visual field (field effect, 39%) and when the patient pointed with his ataxic hand (hand

effect, 39%). The worst performance was observed in the condition combining these two effects (16.6%). When the patient had to correct movements made with his healthy hand towards his healthy field, 77.1% of his pointing movements were corrected, which demonstrated even better performance than the average performance of the control group (60.8%). His performance of on-line visuo-manual control thus demonstrates the same combination of a hand and a field effect as his performance of pointing towards stationary targets in peripheral vision.

The second main result of this study is the different eye-hand coordination pattern that the patient displayed, depending upon the direction of the target jump. More saccades were executed when the target jumped towards his (left) ataxic field, which do not necessarily allow the patient to correct his pointing movements as well as when the target jumped towards the healthy visual field, despite saccades of similar latency in the two conditions. Manual corrections were observed in response to leftward target jumps in CF, but only in the rare trials in which a saccade preceded the end of the pointing movement of 250 msec. This time interval necessary for manual correction towards his ataxic field was 100 msec longer than the interval required for manual correction in patient CF when the target jumped towards his healthy visual field and for the control subjects. This abnormal delay was observed when a saccade occurred early, thus rapidly providing acute (foveal) information about the new target location that was missing in his ataxic visual field (see model of Fig. 5).

What are the consequences of our results for the understanding of visuo-motor processing within the PPC?

Medendorp et al. (2005) have shown that both the hand and the target locations are integrated within the PPC. The pattern of manual corrections shown by patient CF allows us to address how and where the comparison of target and hand locations necessary for on-line motor control takes place. More specifically, we may ask which type of information is used, and whether this comparison is processed by the PPC using single or multiple spatial representation(s). Optic ataxia has been proposed to result from a parieto-frontal disconnection within the damaged cortical hemisphere or a parieto-cerebellar disconnection (see e.g., Battaglia-Mayer and Caminiti, 2002; Glickstein, 2000, respectively, for reviews). However, these models that propose an impaired connection between an area processing target location and another area processing hand location cannot account for the combination of the hand and the field effects in unilateral optic ataxia. The idea of damage to a single spatial map in which both the target location and the hand location are represented (Buneo et al., 2002) also does not fit with these additive effects on the pointing performance of unilateral OA patient. Alternatively, the processing of the target location in the contralesional visual field and the processing of the location of the contralesional hand could be impaired in two different spatial representations. The hand location may subsequently be transformed into the visual map in order for it to be compared with the target location or vice versa (Blangero et al., 2005). The model of a hand effect and a field effect relying on damage to two dissociated spatial representations of the hand and the target

locations, respectively (Fig. 5), is reinforced by the observation of a patient with an isolated hand effect (Binkofski and Fink, 2005; Pisella et al., 2006).

A recent imaging study (Prado et al., 2005) demonstrated the involvement of two distinct anatomical modules during reaching in normal subjects: (1) an “accessory” module located at the POJ and specifically activated when reaching movements are performed in peripheral vision, and (2) a “main” reach-related module located more anteriorly (medial intraparietal sulcus: mIPS) and activated for reaching in central and in peripheral vision. The model illustrated in Fig. 5 also fits with these data. We can speculate that the area that processes peripheral target locations (mentioned in Fig. 5) corresponds to the accessory POJ module proposed by Prado et al. (2005). This POJ module may be involved not only when the reach is directed towards peripheral targets but also when the reach, initially directed towards a central target, has to be corrected because the target jumps towards the peripheral visual field. The area processing the hand location (mentioned in Fig. 5) may correspond to the main anterior module as suggested by Prado et al. (2005). We propose that the POJ module codes target location in oculo-centric coordinates and that the resulting visuo-spatial signal is used directly to ‘update’ the manual reach plans which would be elaborated based on eye fixation in the main anterior module. The observation in OA patients of pointing errors depending on the target location in an oculo-centric reference frame (Khan et al., 2005; Dijkerman et al., 2006; for review: Pisella

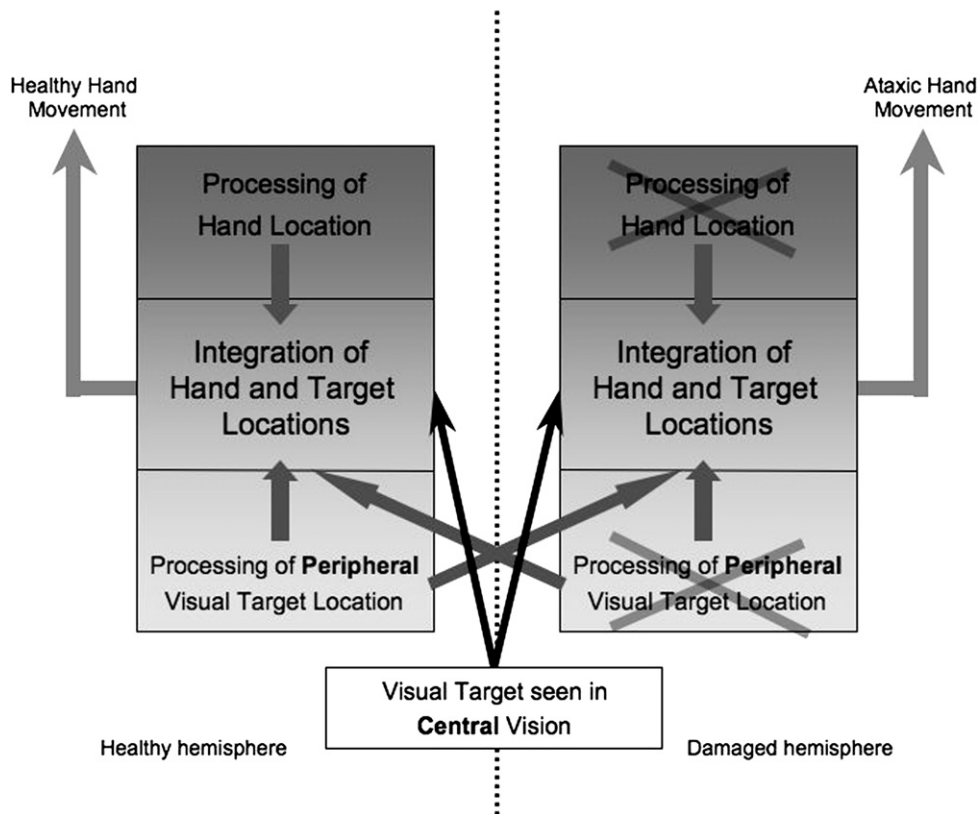


Fig. 5 – Model of visuo-manual transformation. The boxes correspond to the dissociated functional modules. The arrows illustrate transfer of information. The crosses indicate the putative modules damaged by the lesions resulting in optic ataxia, such as those sustained by patients like CF.

et al., 2007) and the reports of parietal patients systematically pointing towards eye fixation (“magnetic misreaching”: Carey et al., 1997; Jackson et al., 2005) are compatible with such a model of visuo-manual transformation. Interestingly, this visuo-manual transformation, involving an update of the reach plan based on the target–eye error, would also be particularly relevant for fast visuo-motor control.

The saccade data did not show a clear relationship with manual correction neither in patient CF nor in the control group. The data of controls show that no coupling between saccade and pointing correction is needed. This result may be surprising as it has been proposed that there is a functional link between the visual capture and the manual capture of the target (Neggens and Bekkering, 2001). But if this functional link existed, manual corrections would necessarily have been accompanied by the occurrence of a saccade in controls. In fact, even with a target jump of 10°, the “perifoveal” vision is usually able to provide sufficient information about the new target location. In patient CF, the coupling becomes obligatory for manual corrections to occur in the contralesional visual field suggesting that it reflects compensatory eye–hand coordination. The more frequent occurrence of a saccade when the target jumps towards the ataxic field would be a consequence of a difficulty in integrating the target jump (i.e., in fast updating target location in peripheral vision) in patient CF, analogous to his lack of fast manual correction. In this condition, only the early foveal capture of the new target location seems to allow the patient to guide manual correction, possibly without relying on the POJ module which deals with target location in peripheral vision (see pathways illustrated with black arrows in Fig. 5).

This same pathway would also be used for reaching stationary targets in central vision. In this case, the model predicts pointing errors if the ataxic hand is used (Fig. 5). However, previous studies have shown that pointing errors are not typically observed in central vision in OA patients (Perenin and Vighetto, 1988). To understand this apparent contradiction between OA behaviour and the model, it is important to note that clinical testing of OA is usually performed in conditions allowing visual feedback of the hand location prior to and during movement execution. Blangero et al. (2007) have shown that visual feedback of the hand improves the accuracy of pointing to peripheral targets by a specific reduction of the hand effect (with respect to the condition of pointing to peripheral visual targets in the dark). If visual feedback of hand location reduces the hand effect, then it is logical that no errors are produced by OA patients when reaching central targets in the light. Since vision of the hand appears to compensate for the errors due to the hand effect, the question arises as to which type of information may be used by the parietal module which processes hand location, and is damaged in OA? Hand location information can be provided both by vision and by proprioception (Rossetti et al., 1995, 1994), and both prior to movement and during movement execution (also by forward models: Desmurget et al., 1998). The visuo-visual comparison between target and hand locations is considered to be too slow to be involved in on-line motor corrections (Goodale et al., 1986; Pélisson et al., 1986) and is probably preserved in OA patients (Pisella et al., 2000; Gréa et al., 2002; Rossetti and Pisella, 2003). The lack of on-line motor

corrections in OA would then suggest a specific disruption of the visuo-proprioceptive comparison of target and hand locations (Vindras, 1998) or an impaired forward modelling of the hand trajectory used for on-line comparison to visual target location (Desmurget and Grafton, 2000). These two propositions are compatible since the precision of forward models depends on the knowledge about initial static hand location to which proprioception contributes. On this point, Blangero et al. (2007) have demonstrated, in OA patients, a dramatic mislocalisation of their ataxic hand in extrapersonal space when hand position information is provided only by proprioception. This high-level deficit was revealed, in the absence of a primary proprioceptive deficit, by a task of proprioceptive pointing (performed with the healthy hand in a dark room) towards the finger of the ataxic hand positioned passively at different locations on a fronto-parallel plane. The parietal module processing hand location may thus be able to transform proprioceptive information into visual (eye-centred) information to be compared on-line with visual target location (Blangero et al., 2007), possibly by forward modelling.

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