Visually guided reaching: bilateral posterior parietal lesions cause a switch from fast visuomotor to slow cognitive control

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Abstract

The visually guided reaching of two patients with bilateral optic ataxia was explored in two experiments. In Experiment 1 simple delayed pointing was compared with immediate pointing. In the immediate pointing task both variable and constant errors increased with target eccentricity. In contrast to the performance of control subjects and contrary to their own beliefs, the patients both showed improved accuracy in the delay condition. This improvement was manifest as a reduction in both pointing variability and in the constant angular error towards the point of fixation. Both angular errors and their improvement with the delay were proportional to target eccentricity. Experiment 2 used a task in which the target was pre-viewed 5 s prior to its re-exposure for pointing (‘delayed real pointing’). On some trials a conflict was introduced between the present and previous visual information by changing the target’s location during the delay. In contrast to control subjects, who ignored the pre-viewed location and aimed directly at the current target, both patients with optic ataxia initiated their movements towards the previously viewed target location. Evidently they relied on off-line information in preference to on-line visual information. In addition, the patients often failed to detect the changes in target location. One of the patients sometimes even guessed incorrectly that the target had changed its location, and her movement trajectory was then more affected by her false belief than by the target’s actual location. These findings confirm that posterior parietal lesions severely disrupt direct visuomotor transformations, and suggest that the residual performance is mediated indirectly by expectations or beliefs about target position.

Keywords: Optic ataxia; Pointing; Dorsal stream; Ventral stream; Delayed response

1. Introduction

Patients with lesions of the posterior parietal cortex may exhibit a characteristic difficulty when they attempt to reach or grasp visual objects (Balint, 1909; De Renzi, 1989; Holmes, 1918). Balint (1909) suggested that this deficit was due to a visuomotor disconnection which he called ‘optic ataxia’, whereas Holmes (1918) proposed that these same deficits instead result from an impairment in spatial perception which he called ‘visual disorientation’. A series of single-case and group studies (Jeannerod, 1986; Jeannerod, Decety, & Michel, 1994; Perenin & Vighetto, 1988; Vighetto & Perenin, 1981) have confirmed Balint’s view of optic ataxia as a visuomotor deficit, by showing that it can be observed without a concomitant perceptual disorder. Although verbal discrimination of dot position was impaired in

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some of the patients tested in the group studies, these subtle deficits in visual space perception were poorly correlated with the misreaching errors. Similarly, as first discovered by Marc Jeannerod and his colleagues, patients with optic ataxia show an exaggerated and poorly scaled grip aperture during reaching for objects, despite being able to correctly match the object’s dimension with their finger grasp when not required to perform the reaching movement (Jakobson et al., 1991; Jeannerod, 1986, 1988, 1997; Jeannerod et al., 1994, 1995; Milner et al., 1991; Perenin & Vighetto, 1988).

At the same time and in the same research group, Perenin and Vighetto (1988) looked for the focus for optic ataxia in a group of patients with unilateral lesions by superimposing CT scans, and discovered that it was located in the superior parietal lobule, within and dorsal to the intraparietal sulcus, sparing the human inferior parietal lobule. Armed with this precise anatomical correlate, theorists have used optic ataxia as a key argument in recent functional interpretations of the two main cortical pathways of the visual system, the dorsal (occipito-parietal) and ventral (occipito-temporal) streams (Goodele & Milner, 1992; Jeannerod, 1994, 1997; Jeannerod & Rossetti, 1993; Milner & Goodele, 1995; Rossetti, 1996; Rossetti & Pisella, 2002). In sharp contrast to patients with optic ataxia, patients with a bilateral lesion of the lateral occipito-temporal cortex are impaired in shape perception (visual form agnosia) but remain able to reach for and grasp the very objects they cannot describe (Goodele, Milner, Jakobson, & Carey, 1991; James, Culham, Humphreyl, Milner, & Goodele, 2003; Lé et al., 2002). This reciprocal pattern of impairment in optic ataxia and visual form agnosia forms a functional double dissociation (Rossetti & Revonsuo, 2000), leading to the interpretation of the two visual streams as a dorsal pathway for direct visuomotor control and a ventral pathway for visual perception.

However, although these neuropsychological studies show that the two visual streams work differently in isolation, both streams must ultimately have an important influence on behaviour, in order for them to have evolved in the first place (Goodele & Milner, 1994; Milner & Goodele, 1995; Rossetti & Pisella, 2002; Rossetti, Vighetto, & Pisella, 2003). This point is borne out by other neuropsychological data, which provide good evidence for a ventral stream route to action (Goodele, Jakobson, & Keillor, 1994; Milner & Dijkerman, 2001; Rossetti & Pisella, 2002; Rossetti, Pisella, & Pelisson, 2000). More recently, research on the cortical connectivity throughout the visual-to-motor network shows that the primary motor area (M1) receives only indirect projections from both visual streams (Pisella & Rossetti, 2000; Rossetti & Pisella, 2002; Rossetti et al., 2000). However there are several functional differences between the two streams (review: Rossetti & Pisella, 2002).

One of the main differences between the dorsal and ventral routes from vision to action lies within each temporal dimension (Bullier, 2001; Nowak & Bullier, 1997). Neuropsychological data have revealed differential effects of delay on the visuomotor performances of patients with a lesion of the dorsal or ventral stream. The agnostic patient D.F. can reach and grasp objects that she cannot describe, but loses this preserved motor ability when her action is delayed by as little as 2 s (Goodale et al., 1994; Milner, Dijkerman, & Carey, 1999a).

Conversely, a patient with optic ataxia (A.T.) performed imprecise reaching (Milner, Paulignan, Dijkerman, Michel, & Jeannerod, 1999b) and grasping (Milner et al., 2001) movements when instructed to respond immediately to objects, but she (paradoxically) improved when a delay was introduced between the stimulus presentation and the pointing or grasping response. This finding of improved reaching after a delay has recently been replicated by Revol et al. (2003) in an optic ataxic patient with a unilateral lesion (O.K.). Furthermore, Pisella et al. (1999, 2000) showed that although fast online visuomotor guidance is disrupted in bilateral optic ataxia (see also Gréa et al., 2002), slow intentional visuomotor guidance is preserved. Pisella et al. (2000) concluded that the superior parietal region processes mainly fast automatic visuomotor transformations. In its absence, alternative systems for visuomotor transformation, which do not involve the dorsal stream, have to be recruited (Milner & Dijkerman, 2001; Milner, Dijkerman, McIntosh, Pisella, & Rossetti, 2003; Rossetti & Pisella, 2002, 2003).

Behavioural differences between immediate and delayed actions have also been identified in normal subjects. For example, Rossetti et al. (unpublished; see Rossetti, 1998) had normal subjects point with various delays to visual targets flashed on a monitor. First, the global variability, as assessed by the area of the confidence ellipse fitting the movement endpoints, continuously increased with the delay. Second, the evolution of the orientation of the main axis of the confidence ellipses fitted for each target followed a two slope function: it tended to be aligned with movement direction in the absence of a delay but then rapidly increased at the 500 ms delay. Between the 500 ms and the 8 s delay, a plateau was reached, with ellipse orientation tending to be aligned with the target array, that is, orthogonal to movement direction. Third, the orientation of the constant error vector in space also followed a similar two-slope trend (see Rossetti et al., 2000: Fig. 2; Rossetti & Pisella, 2002: Fig. 4.11). These results indicate that a different type of sensorimotor process is at work in the immediate and in the delayed condition. A short-lived egocentric representation of the target location seems to be used to guide immediate actions. However, an allocentric coding of the visual target seems to participate in the delayed action, which is affected by the global spatial context of the experiment. Bridgeman, Kirch & Sperling (1981) and Bridgeman, Peery & Anand (1997) reached a similar conclusion when studying pointing to targets that were illusorily displaced. While the immediate pointing performance was more veridical than the cognitive description of the target location, the pattern of pointing responses became influenced by the illusion when a delay was introduced. Similar findings were made by Gentilucci, Chieffi, Daprati, Saetti & Toni (1996) in a study of the effect of the Müller–Lyer illusion on pointing accuracy.
These results converge towards the idea that when action is delayed and the object has disappeared, the positional and geometrical parameters of the object that are used by the action system can only be accessed from a sustained cognitive representation. This type of representation apparently relies on a different reference frame from that employed by the immediate action system (e.g. Jeannerod, 1997; Milner et al., 2003). Furthermore, the neuropsychological data suggest that the dorsal stream builds a very short-lived sensorimotor representation of the target that is available for immediate actions only (review: Rossetti, 1998).

The present study was designed to extend these findings along several lines. First, we re-analysed data from two optic ataxia patients previously tested on an immediate and a delayed pointing task (Milner et al., 1999a, 1999b; 2003; Rossetti & Pisella, 2003), to examine how the effects of delayed responding are modulated by target eccentricity. Secondly, we sought evidence for the preferential use of delayed ('off-line') visual information using a paradigm in which information about target location was available both on-line and in short-term memory (cf. Milner et al., 2001; Rossetti & Pisella, 2003). The specific task used in this experiment was one of 'delayed real pointing', a paradigm which allowed us to introduce a conflict between the two sources of information on test trials by switching the target location during the delay.

2. Materials and methods

2.1. Case reports

Two patients with bilateral optic ataxia were involved in the study. They were both selected through the Neurology units of the Lyon community hospital and gave their informed consent according to the French law (4 March 2002) on patients’ rights.

A.T.: At the age of 32 this woman suffered an eclamptic attack which provoked bilateral haemorrhagic softening in the territory of the parieto-occipital arteries. Structural MRI revealed bilateral parietal damage (Fig. 1A). For the initial 2 weeks, A.T. presented with a severe visual deficit resembling cortical blindness. At the time of the current testing, A.T. continued to show the symptoms of Bálint’s syndrome, including visual disorientation, simultanagnosia, and severe optic ataxia for targets in her peripheral visual field. On the other hand, she showed no clinical indications of occipito-temporal damage (e.g. alexia, object agnosia, achromatopsia, or prosopagnosia). Previous reports have described aspects of A.T.’s visuomotor behaviour (Jeannerod et al., 1994; Milner et al., 1999b), including initial analyses of the data presented here (Milner et al., 1999b, 2003). At the time of the experiments reported here A.T. was aged 44 (Experiment 1) and 46 years (Experiment 2).

I.G.: At the age of 29, this woman suffered an ischaemic stroke related to acute vasospastic angiopathy in the posterior cerebral arteries. She presented with a severe headache, followed by dysarthria and bilateral blindness that lasted for 3 days. After this episode, she initially complained of being unable to see more than one item at the same time and to evaluate distances when she attempted to grasp an object or to walk on uneven ground. Visual fields showed a partial right inferior homonymous quadrantanopia with temporal crescent sparing. Pattern-driven visual ERPs were normal. Structural MRI revealed fairly symmetrical damage to the posterior parietal and upper and lateral occipital cortico-subcortical regions bilaterally (Fig. 1B). I.G. showed no neglect on conventional testing but had bilateral optic ataxia, which was most severe when using her right hand to point to targets in her right visual field. When reaching towards objects, her hand posture was often inappropriate in terms of grip aperture and orientation, and she usually corrected her grip only through tactile feedback after she had contacted the object. Her actions to targets presented in foveal vision, however, were generally accurate. A more detailed description of I.G.’s medical history is available elsewhere (Pisella et al., 2000). Previous reports have described I.G.’s pointing and grasping performance (Gréa et al., 2002; Pisella et al., 2000; Rossetti & Pisella, 2002), as well as her ability to grasp in immediate versus delayed conditions (Milner et al., 2001, 2003). I.G. was tested in the present experiments at 2 years post-stroke, at the age of 31.

2.2. Experiment 1

Experiment 1 compared immediate pointing to visual targets with delayed pointing to remembered targets (‘delayed pantomimed pointing’). These investigations were conducted using different experimental set-ups for the two patients.

A.T.: This experiment was initially reported by Milner et al. (1999a, 1999b, but a more detailed analysis of the errors will be provided here. A.T. was required to point with her right hand to red target LEDs at seven locations, arranged in an arc of 55 cm radius around A.T.’s body, at eccentricities of −30°, −20°, −10°, 0°, 10°, 20° and 30° with respect to the body midline. Throughout each trial, A.T. maintained fixation on a green LED, 2.5 cm in front of the central target. All LEDs were embedded in black Plexiglas and were visible only when illuminated. A.T. performed four blocks of 28 trials. Within each block, she pointed to each target four times, with trial order pseudo-randomized. In the first and fourth blocks, the task was immediate pointing; in the second and third blocks the task was delayed pointing.

On immediate pointing trials, the target LED was illuminated and a tone sounded after 2 s, cueing A.T. to point to the target (the target remained visible until the end of the trial). On delayed pointing trials, the target was illuminated for 2 s only, and 5 s after its offset, a tone sounded, cueing A.T. to respond to the remembered location of the target LED. Responses were recorded by sampling the position of an infrared emitting marker, attached to the tip of the right index finger, at a rate of 200 Hz, using the Optotrak system (Northern Digital...
Fig. 1. Lesions of the patients. (A) A.T.’s cerebral MRI scans revealed bilateral parietal damage extending to the upper part of the occipital lobes and encroaching slightly into the medial part of the right premotor cortex. The calcarine area remained intact except for a part of the upper lip on the left side. (B) T2 MRI scans of I.G.’s brain. Reconstruction of the lesions indicated that they involved bilaterally mainly Brodmann’s areas 19, 18, 7, a limited part of area 39 and the intraparietal sulcus.

Inc.). One female aged 50 years was also tested as a control subject.

I.G.: Preliminary reports of this investigation have been provided elsewhere (Milner et al., 2003; Rossetti & Pisella, 2003). I.G. positioned her right hand on a starting point 15 cm in front of the body midline. Throughout each peripheral target trial, she fixated a 5 mm diameter red disc, 28 cm in front of and 20 cm to the left of the start position. Four peripheral target locations were used, 6, 12, 18 and 24 cm to the right of fixation (corresponding approximately to visual eccentricities of 8°, 15°, 23° and 30°). Additionally, the fixation point was itself used as a target location. These trials were conducted with no experimental control over fixation but one of the investigators controlled visually that no saccade was made by the patients. In all trials the fixation was monitored visually by one of the investigators. I.G. performed four blocks of 25 trials. Within each block, she pointed to each target five times, with trial order pseudo-randomized. In the first and fourth blocks, the task was immediate pointing; in the second and third blocks the task was delayed pointing.

At the start of each trial, a sheet of cardboard was used to screen off the area of the table to the right of fixation. A 5 mm diameter black target disc was placed at one of the target locations on marks that were not visible to the patient. On immediate pointing trials, the screen was lifted to reveal the target area, and a tone sounded after 2 s, cueing I.G. to point to the target. On delayed pointing trials, the screen was lifted to reveal the target, and a first tone sounded after 2 s, whereupon the screen was replaced. The target disc was then removed, and after a further 5 s, a second tone sounded, and the screen was lifted again. I.G. was then required to point to the remembered location of the target disc. Responses were recorded by sampling the position of a magnetic marker, attached to the tip of the index finger, at a rate of 103 Hz, using Minibird (Ascension Technology Inc.). Three healthy female control subjects, aged 26, 36 and 29, were also tested.

2.3. Experiment 2

This experiment used the same set-up as was used for I.G. in Experiment 1, with four target locations to the right of the fixation point, and a cardboard screen used to conceal the target area during the delay period. Each subject performed an initial training schedule of 40 trials (10 to each peripheral target location, in pseudo-random order) of ‘delayed real pointing’. These trials were identical to the delayed condition described for I.G. in Experiment 1, except that the target was always replaced on the table prior to the end of delay period. In the practice trials, the target was always replaced at the same location as it had occupied prior to the delay, so that the target location prior to the delay (target 1) was congruent with the target location after the delay (target 2). (This is a ‘delayed real pointing’ paradigm, since the target is present at the act of pointing as well as before the delay.)

Following the training period, a block of 64 experimental trials was performed in a single session with trial order
pseudo-randomized. Forty-eight trials were ‘congruent’ trials, identical to the delayed real pointing trials of the training period; there were 12 congruent trials for each of the four target locations. The remaining 16 trials were ‘incongruent’, such that the target location prior to the delay (target 1) was different from the target location after the delay (target 2). On eight incongruent trials, target 1 was at the near location and target 2 was at the far location (near-to-far) and on eight incongruent trials the reverse arrangement was used (far-to-near). The same healthy female control subjects, aged 26, 29 and 36, were also tested. For all subjects, both patients and controls, responses were recorded by sampling the position of a magnetic marker, attached to the tip of the index finger, at a rate of 103 Hz (Mimibird, Ascension Technology Inc.).

2.4. Data analysis

For Experiment 1, the raw data were filtered using a dual-pass second-order Butterworth filter with a cut-off frequency of 10 Hz. Movement onset was defined as the frame in which the tangential velocity first exceeded 50 mm/s, and movement offset was defined as the frame at which the velocity fell back below this threshold. The 2D coordinates of the marker in the plane of the table surface in the final frame of movement defined the end point of the reach.

The end-point of each individual reach was used to calculate several accuracy parameters (cf. Rossetti & Pisella, 2002: Fig. 4.8). First, constant errors were measured in amplitude (µ or RHO) and angular direction (α or ALPHA) relative to the ideal reach that would hit the target. Amplitude errors (µ) were negative when movement distance was shorter than the ideal reach, and angular errors (α) were signed negative when the reach was closer to the fixation point than the target. Variable errors were assessed by 95% confidence ellipses of the scatter of finger end positions (e.g. Rossetti & Regnier, 1995; Rossetti, Gaunet, & Thinus-Blanc, 1996). The area of the ellipse provided an estimate of global pointing variability. Although absolute error (D), the unsigned mean distance between the final pointing positions and the target, is a compound of both constant and variable error, it was also used in the present study in order to allow comparison with previous reports (e.g. Milner et al., 1999a, 1999b).

For Experiment 2, data analysis proceeded as for Experiment 1, except that angular error was the sole dependent variable. This was chosen because the perturbation of target location was a perturbation of lateral location which had a large influence upon the angular location of the target, but only a very slight influence upon the required reach amplitude.

2.5. Statistical analysis

For Experiment 1, the main analysis tested the effect of the delay for each subject with a factorial analysis of variance including the two within-subjects factors, delay and target eccentricity. Because only one ellipse-area value was available for each target position, such a factorial analysis could not be performed for this parameter. A second analysis specifically tested the effect of target eccentricity. For each patient linear regression analyses were computed on the mean error parameters obtained at each target eccentricity. This was applied to the immediate and the delayed condition as well as to the difference between these two conditions (delayed-immediate).

In the case of A.T., the symmetrical targets were assigned identical absolute eccentricity. For Experiment 2, only the angular errors (ALPHA) were analysed.

3. Results

3.1. Experiment 1

3.1.1. Subjective reports

As can be seen in Figs. 2 and 3, the typical medially biased error pattern and high pointing variability found in the two patients was reduced in the delay condition. Yet patient I.G. and all of her three controls commented that the delayed task was more difficult than the immediate task (unfortunately A.T. and her controls were not asked). This is in agreement with the subjective reports made by the unilateral optic ataxia patient O.K. in a similar comparison of immediate and delayed pointing (Revol et al., 2003).

3.1.2. A.T.’s errors

Errors tended to increase with target eccentricity and to decrease when the delay was introduced (Figs. 2 and 4). A significant negative effect of delay was obtained for the absolute error $D$ [F(1,98) = 22.0; $P < 0.001$] and amplitude error RHO $[F(1,98) = 8.3; P < 0.001]$. A significant effect of eccentricity was observed for the absolute error $D$, the angular error ALPHA, and the amplitude error RHO $[F(6,98) > 6.5; P < 0.0001]$. In addition a significant interaction between delay and eccentricity was obtained for $D$ $[F(6,98) = 2.8; P < 0.02]$ and ALPHA $[F(6,98) = 11.1; P < 0.0001]$. In contrast, no improvement of performance with delay was found in the control subject. The absolute error $D$ was significantly increased with the delay $[F(1,98) = 26.3; P < 0.0001]$ but no target effect and no interaction was observed. For ALPHA and RHO a significant delay × target interaction was obtained $[F(6,98) = 3.8; P < 0.005]$ but no clear pattern of error emerged from the data (see Fig. 4).

3.1.3. I.G.’s errors

For I.G. a significant effect of target eccentricity was obtained for $D$, ALPHA and RHO $[F(4,70) > 4.5; P < 0.0001]$. A significant delay main effect was found only for ALPHA $[F(1,70) = 33.7; P < 0.0001]$. In addition, a significant delay × target interaction was obtained for ALPHA and $D$ $[F(4,70) > 2.8; P < 0.05]$. The effect of delay on I.G.’s pointing parameters varied with target eccentricity: the pattern of error reduction was observed only for the most eccentric tar-
Fig. 2. Spatial plots of pointing performance of A.T. (right) and a control subject (left) in immediate (upper row) and delayed (lower row) conditions. Confidence ellipses (95%) were fitted to the pointing scatter for each target. In the immediate condition the typical pattern of error can be observed for A.T.: a large increase in pointing variability is associated with a pointing bias toward the midline. While the delay induced the expected increase in pointing variability in the control subject, both pointing variability and systematic bias were reduced in A.T., although depth errors were not affected.

gets (see Fig. 5). In contrast the three healthy control subjects did not improve their performance with the delay. Two of the healthy subjects actually exhibited a significant deleterious main effect of delay on $D \left[ F(1,70) > 10.8; P < 0.005 \right]$ (the last subject’s pattern was close to significance: $P < 0.07$), two on ALPHA $\left[ F(1,70) > 6.0; P < 0.05 \right]$, and one on RHO $\left[ F(1,70) = 8.5; P < 0.005 \right]$. A significant effect of target eccentricity was noted for the three healthy controls for at least one of the error parameters $\left[ F(4,70) > 2.6; P < 0.05 \right]$ and a significant delay $\times$ eccentricity interaction was obtained in one subject for all three error parameters $\left[ F(4,70) > 3.2; P < 0.05 \right]$, and in one subject for $D$ and RHO $\left[ F(4,70) > 2.6; P < 0.05 \right]$.

The only clear pattern that emerged from these analyses was a general trend towards increasing errors with increasing target eccentricity.

For the variable error only the regression analyses could be performed.

### 3.1.4. The effect of eccentricity

Graphical presentation of the data clearly shows an effect of target eccentricity on the patients’ performance (Figs. 4 and 5). The effect of eccentricity was tested using regression analysis separately in the immediate condition and in the delayed condition. Then an additional regression analysis was performed on the difference between the performances obtained in the two conditions. The results of these regres-
Fig. 4. Pointing parameters (SD whiskers) plotted against target eccentricity: absolute error, angular error, amplitude error and global variability, for A.T. (left column) and a control subject (right column). A.T.’s errors are greatest in peripheral vision, especially for area (global variable error) and ALPHA (angular error), reflecting an attraction of pointing towards the midline. This target eccentricity effect is reduced in the delay condition: the improvement found after the delay increases as a function of target eccentricity. The same pattern is not observed in the control subject.
Fig. 5. Pointing parameters (SD whiskers) plotted against target eccentricity: ellipse area (global variability), amplitude, direction, absolute errors for I.G. (left) and one representative control (right). As for A.T., target eccentricity strongly affected the initial pattern of error and modulated the effect of the delay in I.G.

As for A.T., the reduction of the eccentricity effect after a delay was particularly clear for variable error (the area measure). In contrast, the control subject did not show any improvement of performance with the delay.
Experiment 1: linear regressions assessing the effect of target eccentricity (re-hand starting position) on constant and variable errors found for immediate and delayed pointing and for their difference (delayed–immediate results).

<table>
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<th>Variables</th>
<th>A.T. d.f. = 5</th>
<th>Control d.f. = 5</th>
<th>I.G. d.f. = 3</th>
<th>Control d.f. = 13</th>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>Surface</td>
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<td>(y = 7.3x + 42.4)</td>
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<td>(y = 0.1x - 1.1)</td>
<td>(y = 0.8x + 4.2)</td>
<td>(y = 14 + 0.1)</td>
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<td>(P &lt; 0.05)</td>
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<tr>
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<tr>
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<tr>
<td>(D)</td>
<td>(y = 2.0x + 8.9)</td>
<td>(y = -0.1x + 15.8)</td>
<td>(y = 1.6x + 19.9)</td>
<td>(y = 0.4x + 3.2)</td>
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<tr>
<td>(P &lt; 0.05)</td>
<td>(P &lt; 0.74)</td>
<td>(P &lt; 0.05)</td>
<td>(P &lt; 0.15)</td>
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</tr>
<tr>
<td>Delayed–immediate</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Surface</td>
<td>(y = 103.2x + 247.3)</td>
<td>(y = 14.2x + 322.0)</td>
<td>(y = 126.7x + 866.5)</td>
<td>(y = 3.1x + 25.7)</td>
</tr>
<tr>
<td>(P &lt; 0.22)</td>
<td>(P &lt; 0.34)</td>
<td>(P &lt; 0.05)</td>
<td>(P &lt; 0.14)</td>
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<tr>
<td>ALPHA</td>
<td>(y = 0.1x + 7.1)</td>
<td>(y = -0.2x + 2.8)</td>
<td>(y = 0.2x + 2.4)</td>
<td>(y = -0.1x + 0.07)</td>
</tr>
<tr>
<td>(P &lt; 0.57)</td>
<td>(P &lt; 0.14)</td>
<td>(P &lt; 0.05)</td>
<td>(P &lt; 0.07)</td>
<td></td>
</tr>
<tr>
<td>RHO</td>
<td>(y = -0.4x + 26.3)</td>
<td>(y = -0.9x + 15.0)</td>
<td>(y = 0.3x - 2.9)</td>
<td>(y = -0.3x + 0.9)</td>
</tr>
<tr>
<td>(P &lt; 0.12)</td>
<td>(P &lt; 0.05)</td>
<td>(P &lt; 0.07)</td>
<td>(P &lt; 0.08)</td>
<td></td>
</tr>
<tr>
<td>(D)</td>
<td>(y = -2.1x + 3.7)</td>
<td>(y = -0.3x + 6.9)</td>
<td>(y = 2.2x + 24.9)</td>
<td>(y = 0.2x + 1.3)</td>
</tr>
<tr>
<td>(P &lt; 0.05)</td>
<td>(P &lt; 0.29)</td>
<td>(P &lt; 0.08)</td>
<td>(P &lt; 0.31)</td>
<td></td>
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</table>

Regressions are shown for the two optic ataxic patients and their respective controls.
Fig. 6. Trajectories made by a control (A), and patients I.G. (B), and A.T. (C), in the incongruent trials (black) and the corresponding congruent trials (grey) of Experiment 2. The grey circle represents the fixation point. The control subject was not influenced by the change in target location during the delay period. If anything, they may exhibit a trend towards the side opposite to the initial target. In contrast, both A.T. and I.G. were strongly affected by the changes in target location. While they could reach fairly accurately in the congruent trials, their reaches on incongruent trials were initially directed to the target presented before the delay. Both patients were more affected by the change from the least to the most eccentric position than vice-versa. The main difference between the two patients was that I.G. could perform late corrections of her reaches and detect the target shift, while this was never the case in A.T.

3.2. Experiment 2

This experiment was mainly aimed at testing the effect of incongruent preview information on the patients’ behaviour. As can be clearly seen in Fig. 6, the incongruent trials did not affect the normal subjects’ pointing. In contrast, the trajectories produced by the two patients for incongruent trials were severely affected (Fig. 6B,C). In addition to this surprising result, two other unexpected observations were made during this experiment. First, both patients were impaired at detecting the occurrence of incongruent trials. Second, having noticed the occurrence of incongruent trials, one of the patients (I.G.) began to behave abnormally on congruent trials as well.

3.2.1. Subjective reports

Normal controls were not affected by the occurrence of incongruent trials. They reacted normally to the final target presented during the trial and were perfectly aware of all target changes in location. Yet despite the large extent of these changes in target location (from about 10° to 40° in the peripheral visual field or vice-versa), A.T. was never aware of the changes. She never expressed any sign of surprise when the target location was altered during the delay period. When specifically asked at the end of the session, she unequivocally confirmed her unawareness of any changes in location. In contrast, I.G. became aware of the target changes within a few trials. Thereafter she was systematically asked on each trial to report whether she had noticed a change in target location. She correctly detected 12 out of the 16 incongruent trials (75%), but most of her answers appeared to be based on guesses rather than on fully confident experience. I.G. explicitly commented that she could not fully trust her perception. In accordance with this, she also produced 12 (25%) false positive reports for 48 congruent trials and occasionally performed striking anti-corrective trajectory changes in these trials (see Fig. 7).

3.2.2. Congruent trials

For the congruent trials, pointing accuracy of the two patients (Fig. 8) was not better than the delayed condition of Experiment 1 even though the target was now present. A.L-PHA was close to the normal range for the two patients, at least for the targets closest to the fixation point.

3.2.3. Incongruent trials

The trajectories obtained for the two types of incongruent trials are presented in Fig. 6. If anything, normal controls...
Fig. 7. Congruent trials of Experiment 2 for which I.G. wrongly believed there had been a target movement. The grey circle represents the fixation point. The target locations were targets 2, 3 and 4 in the left, right and middle graphs respectively. The ellipses of movement endpoints correspond to pointing responses performed towards the same target in delayed trials recorded in Experiment 1. As can be obviously seen for target 2 and target 4 (left and right graphs), I.G. occasionally initiated her reaches to the correct target location. But these movements could be re-oriented later in the trajectory, in such a way as to paradoxically drive the hand away from the target. In a few other trials (e.g., targets 2 and 3, left and middle graphs) she initiated reaches away from the target and did not perform late corrections.

followed trajectories slightly deviated away from the first target (Figs. 6 and 8). For A.T., trajectories performed in the near-to-far (A → B) incongruent trials were initially aimed at the near target and the trajectories performed in the far-to-near (B → A) trials were attracted towards the far target B. For I.G., the same trend as for A.T. was observed, but additional late corrections occurred, such that the final errors were greatly reduced. In both patients a greater deficit was found for the trials in which the target was displaced from the near to the far position than for the far-to-near trials.

The analysis presented in Table 2 tabulates the influence of the first target in the two patients throughout the unfolding of the reach trajectories. A series of two-way ANOVAs investigated the effects of target 1 (the pre-delay location) and target 2 (the post-delay location) as the two factors, at points along the trajectories located every 10 or 50 mm from the starting position along the depth axis in each patient separately. The results are clear-cut. The normal controls did not show a significant effect of target 1 at any stage of the pointing trajectory. Their trajectories were influenced only by the position of the second target, such that the trajectories obtained in congruent and incongruent trials could not be distinguished. In contrast, the trajectories of both patients were influenced by the location of the first target throughout the whole length of the reaches. There was initially no effect of target 2 at all in the two patients, and its influence only gradually became significant during the course of the reaches. In addition, the asymmetry found between the near-to-far and the far-to-near trials (Fig. 6) led to a significant target 1 by target 2 interaction for patient A.T.

The angular error was only slightly increased in the incongruent trials to the near target, but strongly increased for the far target, again reflecting an asymmetry between the near-to-far and the far-to-near trials (Fig. 8). In the near-to-far trials the negative values obtained for ALPHA reflect the attraction of the trajectory towards the first target. In the far-to-near trials, only A.T. showed an increased angular error indicating an attraction to the first, far target.

Fig. 8. Angular errors (in °) of the patients in Experiment 2. A.T.'s (hatched (congruent) and black (incongruent trials) bars) and I.G.'s (thin (congruent) and bold (incongruent trials) empty bars) angular errors are shown in the diagram. In the AB trials, i.e. in incongruent trials, A.T. showed an increased angular error indicating an attraction to the first target. By contrast, no significant errors are observed in the control subjects (e.g. CC).
Table 2

<table>
<thead>
<tr>
<th>Experiment 2: spatial analysis of the pointing trajectory for target location shift during the delay period</th>
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<tr>
<td>The table shows the influence of the first (T1) or the second target (T2) at different depth displacements during the reach. A strong contrast can be seen between the healthy subjects (influence of target 2 only) and the patients (strong influence of target 1). Level of significance is indicated by asterisks * for $p &lt; 0.05$, ** for $p &lt; 0.005$, and *** for $p &lt; 0.0005$. Shaded areas indicate non-significant outcomes.</td>
</tr>
</tbody>
</table>

4. Discussion

Five main results emerge from the present study. First, the analyses applied in Experiment 1 confirm previous reports of a paradoxical improvement of delayed pointing accuracy in the peripheral vision of optic ataxia patients. In other words, optic ataxia patients can perform better when they base their actions on past information rather than on present inputs. Second, our analyses show clearly that not only the immediate deficit but also the delay-related improvement was strongly dependent on retinal eccentricity. Third, Experiment 2 shows that in a ‘delayed real pointing’ task, our two patients with optic ataxia initiated their movements on the basis of the previewed target location, rather than on the actual visible stimulus, as shown by probe trials where the target was shifted during the delay. Evidently they relied on off-line information to remedy their impaired access to on-line visual information. Fourth, in parallel with this visuomotor deficit, the two patients often failed to notice the target displacements introduced on the probe trials. And fifth, one of the patients demonstrated that her actions were strongly contaminated by her beliefs about target location. We shall first discuss these findings in detail below and then consider their implications for the roles of the two visual streams in action control.

4.1. The effect of delay

The analyses presented in Experiment 1 confirm that reaching accuracy was improved in the delayed condition in both of our patients. In addition, they provide confirmation that both patients had a reduced bias towards the point of fixation after the delay, in support of our earlier conclusions (Milner et al., 2003).

The results of Experiment 2 confirm and extend this superior ‘off-line’ visual processing in optic ataxia. Strikingly, it was found that both patients were strongly influenced by the pre-viewed target position, such that their reaches were initially directed towards the remembered, rather than to the present, target location. The control subjects showed no sign of such an effect. This shift of influence from the proximal visual stimulus to one which had been present a few seconds earlier indicates that the two patients could no longer engage the direct visuomotor mechanisms of the parietal dorsal stream even in an ostensibly immediate reaching task. Instead, their behaviour became dominated by an indirect cognitive route from the visual stimulus to the response. In other words, as we have suggested elsewhere (Milner et al., 2003; Rossetti & Pisella, 2002, 2003), the pointing behaviour of optic ataxic patients seems to be governed predominantly, if not entirely, by processing in more ventral areas (see discussion below) in both immediate and delayed conditions.

Our observations are consistent with the view that immediate and delayed reaching tasks activate distinct systems in normal subjects (Milner & Dijkerman, 2001; Milner et al., 2003; Pisella & Rossetti, 2000; Rossetti, 1998; Rossetti & Pisella, 2002; Rossetti et al., 2000). These systems have been
described slightly different by several authors. Bridgeman (1991) and Paillard (1987, 1991) initially contrasted a cognitive with a sensorimotor system for encoding spatial information. Then Goodale and Milner (1992); Milner and Goodale (1995) proposed to identify the two systems with more broadly conceived ‘visuomotor’ and ‘perceptual’ visual processing streams. Marc Jeannerod (Jeannerod, 1994, 1997; Jeannerod & Rossetti, 1993) characterized the distinction as one between separate ‘pragmatic’ and ‘semantic’ visual systems. All of these functional distinctions have been proposed to correspond to the anatomical distinction between the dorsal and ventral streams of the visual system (Ungerleider & Mishkin, 1982). As argued elsewhere (Rossetti & Pisella, 2002; Rossetti et al., 2000; Pisella & Rossetti, 2000), the two streams have different temporal properties of transfer of visual information, which would particularly suit the dorsal stream for governing rapid immediate reactions. In contrast, the ventral stream, in its role of providing perceptual analyses and providing off-line forms of visual guidance, has no need for such rapid transmission properties. Thus, patients with dorsal stream lesions (i.e. optic ataxia) are impaired for immediate visuomotor control, whereas patients with ventral stream damage or deafferentation (visual form agnosia or hemianopia) are mostly impaired on offline or delayed tasks. This clear dissociation has been interpreted as a persuasive argument for a double dissociation between visual agnosia and optic ataxia (see Milner et al., 2003; Rossetti et al., 2003). However, the visuomotor performance of the two types of patients has not yet been tested in all possible conditions, especially with respect to the distinction between central and peripheral vision. The issue of the perceptual deficit in peripheral vision will be discussed below.

4.2. The effect of eccentricity

Both the immediate deficit and the delay-related improvement were strongly dependent on retinal eccentricity. The fact that optic ataxia patients exhibit larger errors in more peripheral vision has been known for some years (e.g., Jeannerod, 1988; Perenin & Vighetto, 1983, 1988; Ratcliffe & Davies-Jones, 1972; Vighetto, 1980; Vighetto & Perenin, 1981), but their good performance in central vision is only rarely emphasized (Buxbaum & Coslett, 1998; Gréa et al., 2002; Rossetti et al., 2000). Specifically, patients with optic ataxia have been shown to exhibit a consistent pattern of results, such that their pointing errors in peripheral vision are proportional to retinal eccentricity and are biased towards the fovea (cf. Blangéro, 2003; Ota et al., 2002).

Interestingly, the patients especially benefited from delayed responding to the more peripheral targets. This contrast is particularly clear in I.G. (Fig. 5). The same result was also apparent in the unilateral optic ataxia patient O.K. in a previous study (Revot et al., 2003). The observation that the delay effect depends on target eccentricity points towards a specific role of the dorsal stream in the processing of peripheral visual information (Falchier, Barone, & Kennedy, 2002) as well as in the processing of on-line motor control (Gréa et al., 2002; Pisella et al., 2000). The data suggest that the impairment found in patients with optic ataxia is greatest in the peripheral visual field because of an increased need there for fast visuomotor processing, as suggested by Pisella et al. (2000).

The crucial role played by retinal eccentricity in the generation of the optic ataxia deficit has strong theoretical implications. Given that patients with optic ataxia produce normal visually guided actions in central vision, then two distinct functional systems must be invoked to account for the dissociation in behaviour found between central and peripheral vision (Milner et al., 2003; Rossetti et al., 2003). Interestingly, recent neuroanatomical tracing has suggested that dorsal stream areas are preferentially fed with afferents from the peripheral retina, whereas ventral stream areas receive afferents mainly from the fovea (Falchier et al., 2002). This result would be coherent with the proposal that optic ataxia is caused by a disruption of the on-line system guiding the arm in peripheral vision (Gréa et al., 2002; Pisella et al., 2000).

4.3. Off-line versus on-line visual information processing

The main outcome of Experiment 2 was that both patients relied on remembered information when initiating their actions, even when the reaching target was present for them to use. Two distinct systems have to be invoked to account for these results. In both the congruent and the incongruent trials of Experiment 2 two types of representation must have been simultaneously activated, either in a congruent or in an incongruent way. Clearly the off-line system predominated over the on-line visuomotor processing system at the outset of the movement. If a single visuomotor system were responsible for mediating both immediate and delayed pointing, then it would follow that this system would have to be reset when the object has been moved during the delay, and hence a degraded immediate response should be observed rather than a response based on past information.

4.4. Perceptual deficit

An unexpected outcome of Experiment 2 was that both of our patients with optic ataxia often failed to notice the large difference between the object position before and after the delay on incongruent test trials. This observation contrasts with most descriptions of optic ataxia, for which normal visual perception is usually reported. It should be emphasized that I.G. never presented as having a Béclnt syndrome, so that this deficit in perceiving changes does not require that other aspects of the Béclnt syndrome are associated with the optic ataxia. At least one previous observation may be related to the present finding. In a recent report, Michel & Hénaux (2004) described a number of perceptual tests performed with A.T. It was concluded that she exhibited a reduced attentional field, as already explicitly proposed by Béclnt (1909) in his pioneer-
While most of these tasks were performed in central vision, the strongest impairment was found in a task where she had to track visual targets moving among visual distractors (Intriligator and Cavanagh, 2001). This task, like the present one, involves keeping track of changes in stimulus location in the peripheral visual field. Taken together with the unexpected present finding, this result suggests that optic ataxia patients may exhibit not only visuomotor but also visuo-perceptual defects in their peripheral visual field.

4.5. The role of belief

One of the patients demonstrated that her actions were strongly affected by her beliefs, in conditions where she was not confident about whether a particular trial in Experiment 2 was congruent or incongruent. This became apparent when I.G. began to make ‘false positive’ guesses that stimulus displacements were made even on some congruent trials. Her behaviour on these trials departed both from normal (direct pointing to the present target location) and from her own earlier behaviour (correcting her initial tendency to point towards the pre-viewed target location). On some of these trials, I.G. produced late movement reorienting which could drive the hand away from the actual target (see Fig. 7). On other trials, I.G. made movements straight to a very different location than the actual target (Fig. 7). Altogether, these peculiar reaches show that I.G. placed little reliance on current visual information, not only at the outset but also even late during the execution of some of her reaches, using her beliefs rather than her current visual input to guide her movements. These bizarre behaviours suggest an extraordinary dominance of belief over bottom-up influences on action (see Rossetti & Pisella, 2003). This dominance fits well with the previous finding that I.G.’s intentional control of action has been left intact by her lesion, which instead mainly disrupts more automatic visuomotor guidance (Gréa et al., 2002; Pisella et al., 2000).

4.6. The indirect visuomotor system

The present findings confirm that visuospatial processing independent of the posterior parietal cortex participates in visuomotor functions (cf. Milner & Dijkerman, 2001; Milner et al., 2001, 2003; Rossetti & Pisella, 2002, 2003). The most probable candidate structures for mediating this ability are the inferior parietal lobule and the ventral stream of the temporal lobe. Arguments for the ventral stream have been reviewed elsewhere (Milner & Dijkerman 2001; Milner et al., 2003; Rossetti, 1998; Rossetti et al., 2000). In support of the inferior parietal lobule (IPL), it has been shown that patients with a focal lesion there are impaired at motor programming (e.g. Mattingley et al., 1998). Furthermore, Darling, Rizzo, and Butler (2001) found clear deficits in a delayed reaching task in patients with IPL lesions. Complementary brain activation studies have sometimes observed activations in the supramarginal gyrus and inferior parietal lobule during pointing by normal subjects (e.g. Lacquaniti et al., 1997). Given that the visual form agnosia patient D.F. can perform the fast, automatic reaches that are impaired in optic ataxia but remains unable to make accurate delayed motor responses (Milner et al., 1999a), we suggest that the visual input to this IPL system may come from the ventral stream. If this is correct, then D.F. would not be able to use her relatively intact IPL due to its being visually deafferented bilaterally (James et al., 2003). In agreement with these interpretations, a preliminary functional MRI study of patient I.G. has reported increased neural activity in temporal lobe areas specifically during memory-guided movements (Himmelbach, Clavagnier, Perenin, & Karnath, 2003). In contrast, immediate pointing was associated with activations in superior parietal areas surrounding the bilateral lesions in the patient.

5. Conclusions

Patients with optic ataxia are impaired for immediate visuomotor processing, but improve when required to delay before responding. This paradoxical improvement suggests that a slower, less direct system circumventing the lesioned dorsal stream becomes implicated in the control of action when sufficient time is available. These results are compatible with the idea that in normal subjects the dorsal and the ventral streams are respectively responsible for on-line and off-line visuomotor control. The important question now is how the two different systems are integrated during normal visuomotor behaviour (Goodale & Milner, 2004; Milner & Dijkerman, 2001; Pisella & Rossetti, 2000; Rossetti & Pisella, 2002; Rossetti & Revonsuo, 2000).

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