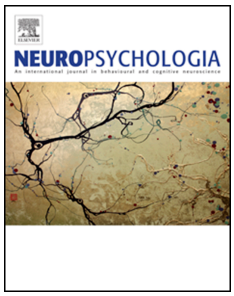

Peripheral and bimanual reaching in a stroke survivor with left visual neglect and extinction

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PII: S0028-3932(24)00116-7

DOI: https://doi.org/10.1016/j.neuropsychologia.2024.108901

Reference: NSY 108901

To appear in: *Neuropsychologia*

Received Date: 31 July 2023

Revised Date: 23 April 2024

Accepted Date: 1 May 2024

Please cite this article as: Knights, E., McIntosh, R.D., Ford, C., Buckingham, G., Rossit, S., Peripheral and bimanual reaching in a stroke survivor with left visual neglect and extinction, *Neuropsychologia*, https://doi.org/10.1016/j.neuropsychologia.2024.108901.

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Abstract

Whether attentional deficits are accompanied by visuomotor impairments following posterior parietal lesions has been debated for quite some time. This single-case study investigated reaching in a stroke survivor (E.B.) with left visual neglect and visual extinction following right temporo-parietal-frontal strokes. Unlike most neglect patients, E.B. did not present left hemiparesis, homonymous hemianopia nor showed evidence of motor neglect or extinction allowing us to examine, for the first time, if lateralised attentional deficits co-occur with deficits in peripheral and bimanual reaching. First, we found a classic optic ataxia field effect: E.B.'s accuracy was impaired when reaching to peripheral targets in her neglected left visual field (regardless of the hand used). Second, we found a larger bimanual cost for movement time in E.B. than controls when both hands reached to incongruent locations. E.B.'s visuomotor profile is similar the one of patients with optic ataxia showing that attentional deficits are accompanied by visuomotor deficits in the affected field**.**

Keywords: visual neglect; visual extinction; peripheral vision; bimanual; optic ataxia

Introduction

Within the influential perception and action model put forward by Milner & Goodale (1995), optic ataxia is seen as the characteristic deficit of the 'vision for action' dorsal visual stream (from occipital to the posterior parietal cortex; PPC). Optic ataxia is characterised by difficulty reaching toward and grasping objects presented in peripheral vision, whereas actions in free vision are generally accurate (Garcin et al., 1967; Perenin & Vighetto, 1988). More specifically, patients with unilateral optic ataxia often make large spatial errors when reaching or pointing at targets presented in the visual field contralateral to the lesion ('field effect'; Perenin & Vighetto, 1988) and/or when using their contralesional hand ('hand effect'; Perenin & Vighetto, 1988). The centre of lesion overlap is in the PPC, close to the parieto-occipital sulcus: at the junction between the inferior parietal lobule, superior parietal lobule and superior occipital cortex, extending medially to the precuneus (Karnath & Perinin, 2005; Pisella et al., 2009; see Fig. 3 in Harvey & Rossit, 2012).

Perenin and Vighetto's (1988) landmark case series of unilateral optic ataxia influentially characterised it as a "pure" visuomotor disorder, but this idea has more recently been questioned. Indeed, following Pisella et al.'s (2000) finding that patients with optic ataxia are impaired in perceptual location change detection in peripheral vision, various studies have demonstrated perceptual and attentional deficits in optic ataxia (for detailed reviews see Pisella et al., 2007, 2008, 2009, 2013, 2021). Using the classic Posner paradigm (Posner et al., 1984), Striemer at al. (2007) found that two right-hemisphere lesioned patients with left optic ataxia were slower to detect targets in their left ataxic visual field, regardless of whether a central predictive cue was valid, invalid or uninformative, consistent with deficits in orienting and reorienting of attention. Striemer et al. (2007) therefore suggested that optic ataxia is accompanied by a decrease in the salience of stimuli in the ataxic field. Similarly, it has been reported that patients with optic ataxia are impaired in perceptual letter discrimination (Pisella et al., 2007; Blangero et al., 2010) and present visuospatial deficits in their ataxic field (Khan et al., 2005; Gaveau et al., 2008). Together

these results challenge the established assumption that optic ataxia is a “pure” visuomotor deficit (Milner & Goodale, 1995; 2006; Perenin & Vighetto, 1988).

Indeed, in recent years, it has been further argued that some degree of attentional impairments (having perceptual consequences) may always accompany optic ataxia and should be understood as part of the same functional deficit (Pisella et al., 2007, 2008, 2009; 2021; Aguilar-Ro et al., 2021; McIntosh et al., 2011; but see Striemer et al., 2009). Regarding the perception and action model, perceptual deficits in optic ataxia suggest that the dorsal stream plays a role in visual perception via visuo-spatial/attentional processing (Pisella et al., 2009). Indeed, Pisella and colleagues (2013) go one step further and suggest that the ‘*core processing of the PPC appears to be attention and to only consequently affect “vision for action*”’ (page 319). This view seems more in line with the view that the dorsal visual stream processes location, rather than vision for action *per se* (Holmes, 1918; Ratcliff, 1991; Ratcliff & Davies-Jones, 1972; Ungerleider & Mishkin, 1982).

A related debate of whether attentional deficits are accompanied by visuomotor impairments, has also been ongoing for several decades regarding patients with visual neglect. Visual neglect is classically defined as an inability to detect, attend, or respond to visual stimuli located on the contralesional side of space. Milner & Goodale (1995, 2006) postulate that the high-level representational system that is damaged in neglect is more closely linked to the ventral stream of processing than to the dorsal (see also Milner, 1995, Milner & Harvey, 2006). Patients with visual neglect, by definition, show laterally asymmetrical patterns of behaviour in paper-and-pencil tasks including cancellation, line bisection or drawing. However, studies investigating direct visuomotor tasks, such as prehension or reaching, have produced inconsistent results (for reviews see Himmelbach et al., 2007; Coulthard, Parton & Husain, 2006, 2007; Harvey & Rossit, 2012; Ogourtsova, Archambault & Lamontagne, 2015).

Goodale et al. (1990) investigated visually guided pointing in right hemisphere damaged patients who had recovered from neglect. Patients were asked to point either

midway between two targets (bisection) or directly to a single target in free viewing conditions with visual feedback available. All patients made large rightward directional errors at the outset of the reach; however, they were more poorly corrected in the bisection task, so that the terminal errors were much larger than those seen in pointing. Harvey, Milner, and Roberts (1994) tested patients with right- or left-hemisphere stroke on similar tasks but included a condition without visual feedback (i.e., 'open-loop'). They reported rightward curvature in the reach trajectory and larger terminal errors for right hemisphere damaged patients in open-loop conditions, suggesting 'sub-clinical' optic ataxia as a possible cause (p. 349). Jackson et al. (2000) similarly reported rightward curved trajectories in the pointing movements (in free vision) of recovered left neglect patients.

However, several later studies with larger samples of neglect patients, failed to confirm trajectory biases or reduced accuracy in reaching movements to the neglected field, in free vision, and with or without visual feedback during movement (e.g., Perenin, 1997; Karnath, Dick & Konczak, 1997; Himmelbach & Karnath, 2003; Rossit et al., 2009a,b,c; Rossit et al., 2011a,b). Moreover, although movement duration may be extended in right brain damaged neglect patients, it is not differentially longer for targets on the contralesional side of space and is no longer than the movement durations of patients without neglect (e.g., Konczak & Karnath; 1998; Konczak et al, 1999; Himmelbach & Karnath, 2003; Karnath et al., 1997). The conclusion reached by a recent systematic review is that despite their severe contralesional visuospatial deficits, neglect patients can reach accurately to targets presented on both sides of space (Ogourtsova, Archambault & Lamontagne, 2015).

A popular interpretation of spared visuomotor control in neglect is that it dissociates from optic ataxia, as neglect patients do not show the consistent misreaching observed in optic ataxia (Perenin, 1997; Himmelbach et al., 2007; Harvey & Milner, 2006; Harvey & Rossit, 2012). This is often linked to the fact that neglect lesions often miss the superior and medial PPC regions of the dorsal visual stream, tending to be more inferior and lateral. Furthermore, it has been suggested that there is functional dissociation within the PPC (e.g.,

Himmelbach et al., 2007; Perenin, 1997; Perenin & Vighetto, 1988): brain regions damaged in optic ataxia are thought to be involved in short-lived, unconscious visuomotor computations, whereas the regions damaged in neglect patients seem to be involved in the more enduring and conscious representations underlying spatial cognition and complex spatially oriented behaviour. Consequently, the perceptual biases in neglect should not affect visuomotor performance (e.g., McIntosh et al., 2004; Milner, 1995, Milner & Harvey, 2006; Milner & McIntosh, 2005; Perenin, 1997).

Importantly, however, to the best of our knowledge, the studies investigating reaching in neglect have only tested reaching with the ipsilesional hand (typically the right) to targets viewed in free vision, conditions in which optic ataxia patients are unimpaired too. This gap in the literature may be unsurprising, given that many neglect patients have hemiparesis and/or homonymous hemianopia, which prevent the assessment of pointing with the contralesional hand and/or pointing to targets in the contralesional peripheral visual field, where optic ataxia could be revealed. The assumed dissociation between neglect and optic ataxia thus requires testing in neglect patients who can move both arms and do not show full visual field cuts.

Moreover, while the link between attention and action is well-studied in the context of unimanual movements (for review see Baldauf & Deubel, 2010), there is far less research examining how the attentional systems behave when both hands are moving simultaneously (Buckingham & Carey, 2015). This is surprising given that many everyday activities (such as dressing or eating) are bimanual. Studies investigating bimanual tasks in patients with optic ataxia have reported contradictory results. Perenin & Vighetto (1988) found that most of their 10 optic ataxia patients showed impairments in bimanual reaching to targets in the contralesional peripheral field. Similarly, Jackson and colleagues (2004) report that while optic ataxic patient JJ with bilateral parietal lesions was able to accurately perform unimanual movements with either limb, he was significantly less accurate in bimanual reach-to-grasp actions to peripheral targets. In contrast, more recently, Litovzky et al. (2019) found

that a posterior cortical atrophy patient with optic ataxia (MDK) performed significantly better in bimanual than unimanual peripheral reaching tasks. While MDK reached accurately to foveated targets, he undershot eccentric peripheral targets in both unimanual and bimanual conditions, with considerably smaller errors in bimanual than in unimanual conditions.

Bimanual actions have also been rarely examined in patients with visual neglect and/or visual extinction. Patients with neglect often have concomitant visual extinction whereby a contralesional stimulus fails to reach awareness when in simultaneous competition with an ipsilesional stimulus (Driver & Vuilleumier, 2001; Vossel et al., 2011; Vallar et al., 1994). The distinction between visual neglect and visual extinction is often blurry where both conditions can be seen as a gradual expression of an ipsilesional lateralised attentional bias (e.g., Pisella et al., 2013) with some considering extinction as a mild form of neglect (e.g., Di Pellegrino et al., 1998). However, they are usually operationalised as distinct clinical entities. Visual extinction is elicited under conditions of brief bimanual competition, for instance during confrontation testing by wiggling one or two fingers in front of the patient; whereas visual neglect refers to a lack of attention to contralesional stimuli even when presented for unlimited time, and in free viewing conditions, for instance in a target cancellation task. Neglect and extinction, as so defined, may dissociate across patients, suggesting (partly) separate neural mechanisms (Meister et al., 2006; Karnath, Himmelbach & Küker, 2003; Salvato et al., 2021).

However, the anatomical basis for such a dissociation has been debated, with lesion-symptom mapping studies associating both disorders with lesions in the right PPC and temporal-parietal junction (e.g., Ticini et al., 2010; Vossel et al., 2011; Chechlacz et al., 2013; Karnath, Himmelbach, & Küker, 2003). The right temporal parietal junction (TPJ) has been suggested to play a critical role in both extinction (e.g., Tinici et al., 2010; Meister et al., 2006) and neglect (Corbetta & Shulman, 2002), as part of the ventral frontoparietal attention network (Corbetta, Patel, & Shulman, 2008; Corbetta & Shulman, 2002). This right hemisphere dominant ventral attention network is thought to be involved in stimulus-driven

control responding when behaviourally relevant stimuli are detected, working as a "*circuit breaker*" for a dorsal frontoparietal attention network re-directing attention to salient events. The dorsal attention network, which includes the intraparietal sulcus and superior parietal lobule, is hypothesized to generate, and maintain endogenous signals based on current goals and pre-existing information about likely contingencies, and to send top-down signals that bias the processing of appropriate stimulus features and locations in sensory cortex (for review see Corbetta, Patel, & Shulman, 2008; Corbetta & Shulman, 2002). Alternatevely, visual extinction could, unlike neglect, correspond to the impairment of a symmetrical attentional network given that extinction-like behaviour can be produced by Transcranial Magnetic Stimulation (TMS) to either the left or right hemisphere in healthy participants (Pascual-Leone et al. 1994). Its been suggested that the right predominance and the involvement of the TPJ in causing visual extinction could simply be the result of indirect dysfunction of the dorsal attentional network in case of damage to the ventral attentional network (Corbetta et al., 2005). Thus, there is not yet consensus about the anatomical basis between extinction versus neglect.

In contrast to the sensory-attentional imbalance usually implied by extinction, *motor* extinction refers to a deficit of motor production that either worsens disproportionately, or only becomes apparent, when the patient performs bimanual actions (Punt & Riddoch, 2006; Coulthard et al., 2006). Motor extinction is conceptually related to motor neglect, being characterized by an underuse of the contralesional limb which cannot be explained by primary motor or sensory deficits (Laplane & Degos, 1983). Interestingly, patients with motor neglect (who underuse the contralesional limb) are impaired when using their contralesional limb during bimanual tasks but spared when performing contralesional limb movements unimanually (Punt et al., 2005, 2013; also see the Balint's syndrome patient described in Edwards & Humphreys, 2002). However, whether contralesional motor impairments during bimanual action reflect *attentional* or *intentional* deficits remains debatable. According to the attentional account, patients may initiate bilateral movements but only be aware of the

sensory consequences of moving the ipsilesional limb leading to execution deficits with their contralesional limb (Driver & Vuilleumier, 2001). Alternatively, the intentional account argues that motor extinction/neglect is caused by a failure to form a movement plan (Heilman et al., 2003): in motor neglect this would manifest itself as a failure to move the contralesional limb even unimanually, whereas in motor extinction an intention failure would only manifest itself during bimanual tasks. Thus, if assessing target-directed actions in the context of neglect, it is therefore of interest not only to characterise unimanual reaching, but also to study the consequences of demands for bimanual simultaneous movements.

Here we report a case study where we had the opportunity to investigate the reaching performance of a stroke survivor (E.B.) with left visual neglect and extinction, who retained full function in both upper limbs and did not have a left homonymous hemianopia. Our first goal was to contribute evidence bearing on the question of whether neglect and extinction are associated with deficits characteristic of optic ataxia, particularly misreaching in the contralesional peripheral visual field. We used peripheral visual presentation of unimanual reaching targets, with central fixation, as is standard in the assessment and experimental study of optic ataxia (e.g. Blangero et al, 2010; Borchers et al., 2013; Milner et al., 1999; Perenin & Vighetto, 1988).

We also tested bimanual reaching only in free vision as we wanted to examine the impact of planning and executing reaches to competing target locations. Bimanual reaching was assessed in free vision, to minimise the influence of deficits of peripheral reaching (assessed in the unimanual condition). We included congruent trials, in which the two hands reach to mirror-symmetric target locations, and incongruent trials, in which the target locations are asymmetric. On congruent trials, participants may exploit a preference to produce mirror-symmetric movements, focusing attention to one of the target locations. The lack of symmetry for incongruent targets increases the complexity of spatial representations and the attentional demands of monitoring movements to these positions. This allowed us to examine, for the first time, if visual neglect and extinction are accompanied by differential

difficulty in executing simultaneous target directed movements on both sides of space. The present study is exploratory and preliminary, intended to inform future larger studies.

Materials and Methods

Participants

Patient E.B.

Patient E.B. was a 67-year-old right-handed retired woman who previously worked as a research interviewer. A year before participating in this study she suffered two middle cerebral artery infarcts in the right hemisphere within the same month. A computed tomography scan indicated lesions involving the right frontal and parieto-temporal areas (Figure 1A). The right parietal lesion affected the superior parietal lobule and extended inferiorly toward the intraparietal sulcus and inferior parietal lobule. This lesion continued into the temporal lobe, involving the superior, middle, and posterior temporal gyri. The right frontal lesion was located around the inferior frontal gyrus and middle frontal gyrus and extended inferiorly into the orbitofrontal/cingulate gyri and insula. Eleven months post-stroke, E.B. volunteered to take part in the present study at the Neuropsychology lab (University of East Anglia). Ethical approval was granted by the University of East Anglia ethics committee and all participants (including E.B.) provided informed consent prior to participation and were reimbursed for their time.

During formal neuropsychological assessment completed a few months before participation, E.B. was independent, well oriented [The Birmingham Cognitive Screen (BCoS) orientation], fully mobile and had spared psychomotor speed (Delis-Kaplan Executive Function System Trail making test – motor speed), praxis (BCoS gesture production, BCoS imitation, BCoS Multi-step object use and BCoS figure copy), memory (Weschler Memory Scale) and language abilities. She reported slight numbness in the left thumb and index finger, and fatigue of the left upper limb but was able to move both limbs unimanually and bimanually.

At the time of the present study, as seen in Figure 1B, computerised perimetry visual field testing (e.g., Rossit et al., 2009a) detected a left lower scotoma, whereby E.B. failed to detect stimuli presented 12° below fixation, but only when further than 16° within the left visual field. She showed left visual extinction in a computerised confrontation test (Figure 1C; >90% accuracy for unilateral left / right and catch stimulus presentations, compared to 8.3% accuracy for bilateral presentations; Rossit et al., 2009). Evidence of extinction was not observed in tactile or auditory domains during physical confrontation (100% accuracy on all trials). Finally, she also presented with left visual neglect in several tasks (Figure 1D): E.B. was impaired on several cancellation tasks (star cancellation: total score / cut-off = 50 / 51; left score = 23; Wilson, Cockburn & Halligan, 1987; Balloons test: index / cut-off = 37.5% / 45%; Edgeworth, Robertson & McMillan, 1998), showed a significant rightward bias during line bisection (average error / cut-off = 14mm / 6mm; Rossit et al., 2009a; Figure 1D) and omitted key left sided details during figure copy (Figure 1C; Wilson, Cockburn & Halligan, 1987). Patient E.B. showed no visible signs of hemiparesis nor motor neglect/extinction (could move either limb freely) or had no primary proprioceptive difficulties (100% accuracy using the contralesional limb to point to her own body parts on command with eyes closed).

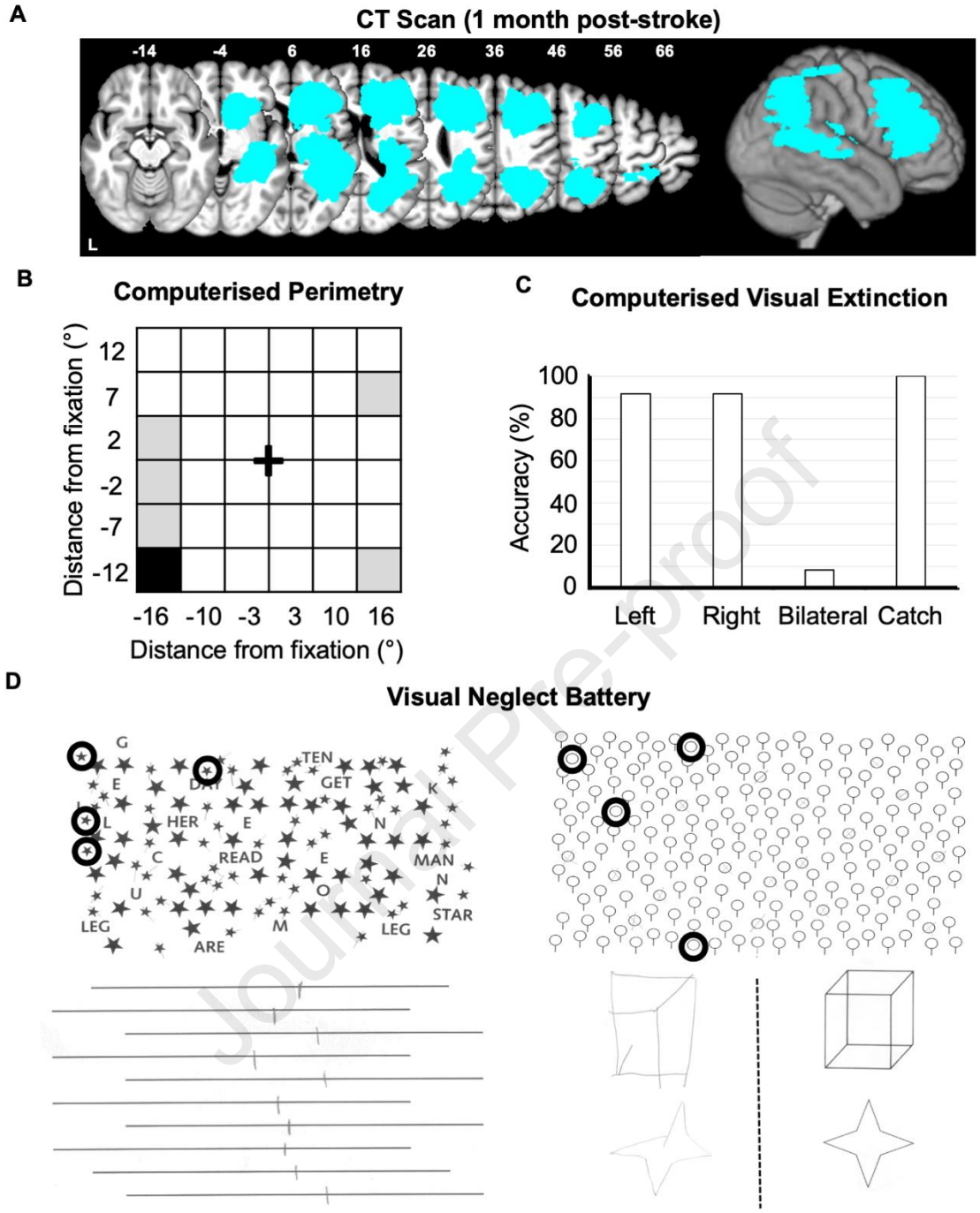

Figure 1. Patient E.B.'s case history. **(A) Lesion map**. Patient E.B.'s area of damage was manually demarcated using MRIcron (Rorden, et al., 2007) on each axial slice of the clinical CT scan to create a volume of interest (VOI). The original scan and VOI were normalized to standard Montreal Neurological Institute (MNI) space using the clinical toolbox (Rorden et al., 2012) with SPM12. The lesion is shown in cyan overlaid on a high-resolution scan

provided within the clinical toolbox (left; MNI z coordinates of the transverse sections are given) and on a 3D rendered brain created with MRIcron (right). **(B) Visual field assessment.** A computerised perimetry test revealed left lower scotoma at 16° left and 12° below fixation. **(C) Visual extinction assessment.** Unilateral left extinction was observed (failure to report left targets specifically during bilateral stimulation; all, except one, of the bilateral errors involved her reporting only the right-sided stimulus). **(D) Visual neglect assessment.** On the day of the optic ataxia assessment, left visual neglect was detected in several cancellation assessments (upper; circles show omitted targets), line bisection and figure copying (lower).

Control Group

Ten healthy volunteers, age- and gender-matched to E.B., participated in the study (mean age = 65.9, SD = 4.7, range = 59 - 75). This group are all right-handed (Oldfield, 1971), had no history of motor, psychiatric or neurological disorders and had normal or corrected-to-normal visual acuity according to our in-house questionnaire. They were not invited to take part if they reported any deficits such as cataracts, amblyopia, epilepsy, dementia, cognitive impairment, brain tumours, traumatic brain injury or stroke.

Stimuli & Apparatus

Participants sat in an adjustable chair in front of a purpose-built grey table with their head stabilised in a chin rest aligned with the table midline (Figure 2A). A start box was positioned 18.5cm in front of the participant's midline. Stimuli were circles (0.6cm^2; ~0.5°) produced by white Light-Emitting Diodes (LEDs) embedded in the table (see Procedure sections for locations). The room was slightly darkened, and targets were visible only when illuminated. Participants could see their reaching limb throughout the movement.

Pointing responses were recorded by sampling the 3D position of spherical infrared reflective markers that were attached to the tip of the right/left index finger at a rate of 179Hz, using a six-camera motion-tracking system (Qualisys; Sweden). Additional markers

were attached to each wrist, for measuring velocity and to define movement on/offset. To determine reaching accuracy, three calibration trials were collected and averaged after the experiment. These involved continuous illumination of each target (and the start position; see Figure 2), one by one, allowing the participants to adjust their terminal fingertip position until they believed the target was perfectly occluded.

Participants rested their head in a chinrest and wore a head-mounted binocular eye-tracking system (Sensomotoric Instruments; 29Hz) throughout Experiment 1 to ensure central fixation was maintained during the peripheral vision conditions. Eye gaze was monitored online (iView, Version 3.6) and checked offline (BeGaze, Version 3.6). The experimental sequence was synchronised to the motion-capture system and controlled by a custom program written in Matlab (MathWorks Inc. 2013) with Psychtoolbox 3 (Brainard, 1995).

Procedure

Experiment 1: Peripheral Reaching

The first experiment involved reaching during a 'free vision' and a 'peripheral vision' condition with each hand separately during independent blocks (Figure 2B) adapted from Milner et al.'s (1999) paradigm used with optic ataxia patients (see also Rossit et al., 2018). To allow direct comparison between patient E.B. and the control group, a fixed block order was used: right hand free vision, left hand free vision, right hand peripheral vision, left-hand peripheral vision. Free vision conditions involved reaching and pointing to targets with the index finger when participants could move their eyes (Figure 2B; Figure 3A). Peripheral vision conditions involved performing the same action whilst maintaining fixation on the central light (Figure 2B; Figure 3A).

Red targets were located 40cm (26°) ahead of the participant in a horizontal line corresponding to the visual angles of 0°, ± 11°, ± 17° & ± 28° (e.g., Rossit et al., 2018; Figure 2A). The fixation was green, located 10cm (11°) straight ahead of the central target.

Therefore, targets were positioned outside E.B.'s lower left lower scotoma and visible to her (she was able to verbally detect all peripheral targets). The fixation target was illuminated continuously, and participants were required to fixate throughout each trial. Stimulus onset was under the participant's control, being shown when the participant pressed the start button. Participants were required to reach quickly and accurately toward the target when it was illuminated. The target remained illuminated for 2 seconds in each trial. In each block, there were 70 trials (i.e., 10 trials per seven target locations in a randomised order). Central fixation was monitored online and verbal feedback about fixation was provided to participants during peripheral vision conditions.

Experiment 2: Bimanual Reaching

The second experiment, adapted from Jackson et al. (2000) bimanual pointing study, was completed after a short break following Experiment 1. Reaching performance was measured across four separate blocks in the following fixed order: unimanual right hand, unimanual left hand, bimanual congruent and bimanual incongruent. For unimanual trials, a single target appeared, ipsilateral to the responding hand, and at a 'near' or 'far' distance (Figure 2C; Figure 3B). For bimanual trials, a left and right target was presented simultaneously. For the congruent bimanual condition, the distances of the two targets were matched, so that mirror-symmetrical reaching movements of the two hands were required. For the incongruent bimanual condition, they were mismatched so that the two hands were required to reach to different distances (Figure 2C; Figure 3B).

A single trial involved resting the index finger on the start position, pointing to the target (participants were instructed to act 'quickly and accurately') and returning to the start position. The starting position was constantly illuminated, and targets were displayed for 3 seconds. There were two starting positions one for the left hand in the left visual field and one for the right hand in the right visual field, matched between unimanual and bimanual trials. There was an intertrial interval of 2 seconds and an auditory beep co-occurred with

target onset. Twenty (+10 practice) trials were completed for each block with 10 trials occurring for each of the two possible targets in a randomised order.

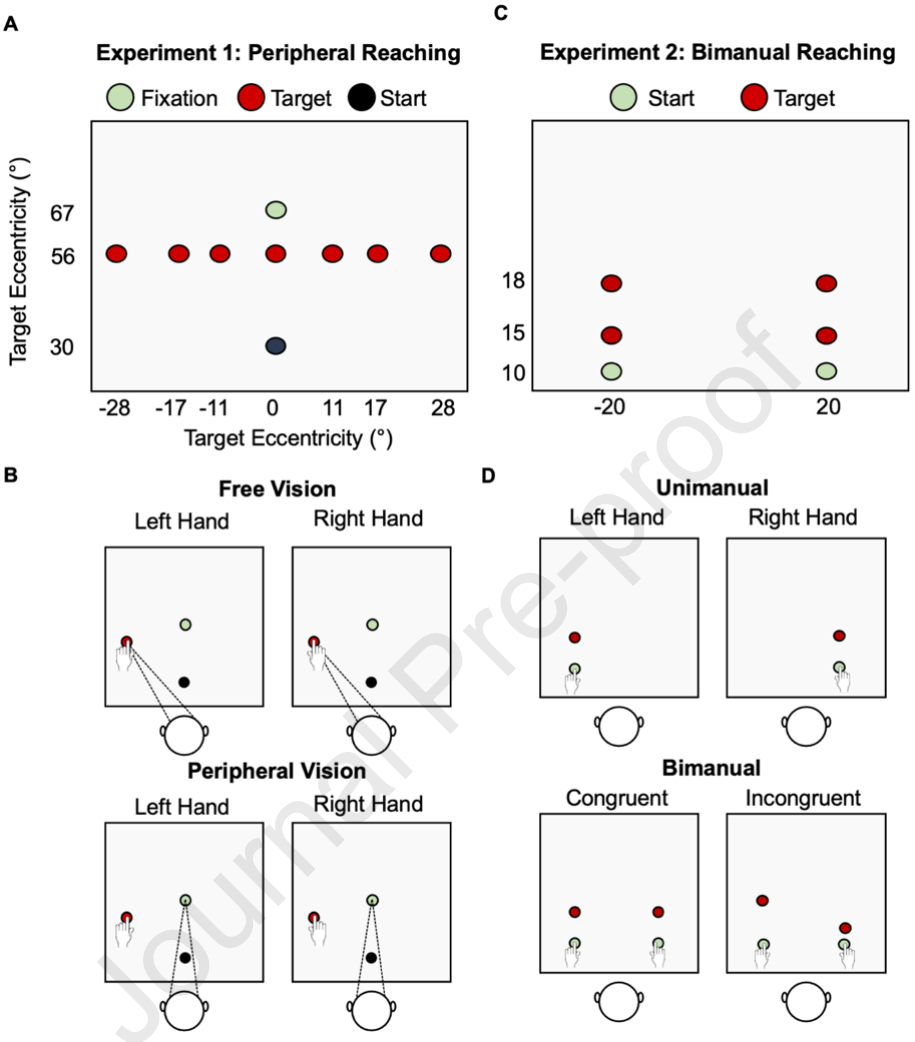

Figure 2. Apparatus & Stimuli. (A) Experiment 1 setup. Overhead depiction of the purpose-built table in which LEDs were embedded to represent the seven targets (red circles) at varying eccentricities in the left, central and right visual fields as well as for the central target and fixation. (Rossit et al., 2018). **(B) Experiment 1 reaching conditions.** Example trials for the left target are represented for the free vision and peripheral conditions (upper vs. lower panels). During the study, targets appeared on both left and right visual fields. The dashed lines represent eye gaze. **(C) Experiment 2 setup.** Red LED targets were located at a 'near' or 'far' distance from the hand starting position (green LED). **(D)**

Experiment 2 reaching conditions. Example trials are represented for the unimanual and bimanual conditions (upper vs. lower panels).

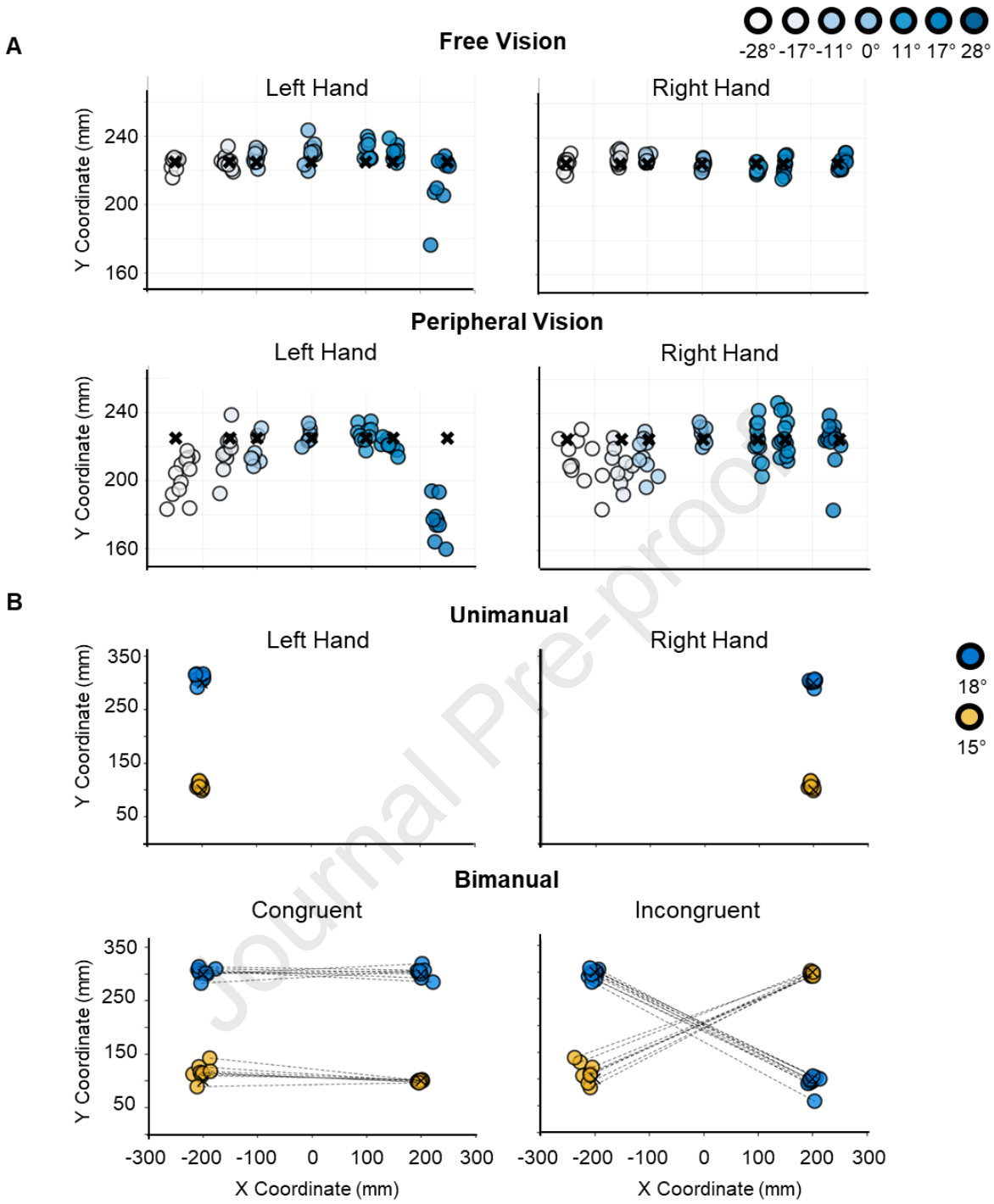

Figure 3. Endpoint coordinates for Experiment 1 (A) and Experiment 2 (B). Each marker represents the endpoint coordinate in the X and Y planes for a single trial for patient E.B. Marker shade denotes reaches to targets presented at separate locations (horizontal and vertical eccentricities in Experiments 1 & 2, respectively) and a black cross represents the reference for each target location. Dashed lines join the left- and right-hand responses completed on the same bimanual trials.

Data processing

The raw positional data was processed using a custom MATLAB scripted interface. First, timeseries were filtered by a dual-pass second-order Butterworth filter (cut-off frequency = 10Hz) before defining movement on/offsets using a velocity-based criterion of 50mm/s.

Trials were excluded from Experiment 1 when there were fixation errors in the peripheral vision condition (2.8% trials), or when no reaching response was made (1.6% trials). We also excluded trials if the reaching response did not finish during the recording period (Experiment 1 = 0.4% trials; Experiment 2 = 10.7% trials) or if infrared markers were obscured during the movement (Experiment 1 = 2.3% trials; Experiment 2 = 10% trials).

For analysis of Experiment 1, we computed absolute reaching error [$\sqrt{}$ (X error^2 + Y error^2)] for comparability with previous optic ataxia studies (e.g., Milner et al., 1999; Revol et al., 2003; Blangero et al., 2007; Dijkerman et al., 2006; Striemer et al., 2009). For analysis of Experiment 2, we calculated the bimanual cost of reaction time (RT) and movement time (MT; Punt et al., 2005; Buckingham et al., 2010) by subtracting the appropriate unimanual score from that of the bimanual action. For consistency across experiments, we also analysed the remaining variables (i.e., RT and MT in Experiment 1 and absolute reaching error in Experiment 2).

Statistical analysis

For Experiment 1, we used the Bayesian Standardised Difference Tests (BSDTs; Crawford & Garthwaite, 2007; Crawford, Garthwaite & Porter, 2010) to compare E.B.'s accuracy to controls in free vs. peripheral reaches (vision-comparison) and left vs. right hand reaches (hand-comparison). In Experiment 2, we used a Bayesian Test of Deficit (BTD; Crawford, Garthwaite & Porter, 2010) to compare E.B.'s bimanual cost to controls for each hand (i.e., left vs. right) and bimanual condition (i.e., congruent vs. incongruent), per variable. All statistical tests were implemented using the *singcar* package (version 0.1.3; Rittmo &

McIntosh, 2020) in R (version 3.6.1). Two-tailed results are reported, based on a standard alpha level to determine statistical significance (p = 0.05).

It has recently been emphasised that the power of case-control comparisons is severely limited, relative to more standard group-based tests (McIntosh & Rittmo, 2021). Given the size of the control group for this study (n = 10), and a conventional alpha level of .05 (one-tailed), the power to detect a deficit size of 2SD would be around 56% only. This entails that the risk of Type II error (failing to detect a true deficit) is around nine times as high as the risk of Type I error (falsely identifying a deficit). Power to detect a true deficit would be further reduced by adjusting the significance criterion for multiple comparisons, and so we chose to run all statistical comparisons with an unadjusted significance threshold of .05. This should be borne in mind when evaluating the p values reported in Table 1, but note that this analytical choice was not critical to our main findings of theoretical significance (pattern of impairment for Absolute Reaching Error is unaffected).

Data Availability

Processed and analysed kinematic data are available on the Open Science Framework (https://osf.io/q8nj6/) along with analysis code hosted on GitHub (https://github.com/ethanknights/neglect).

Results

Analysis of fixation and trial error frequencies showed that patient E.B. had difficulty with the peripheral vision condition in Experiment 1. She committed significantly more eye fixation errors than controls during the left hand condition (p = 0.007, z_{cc} = 3.65; 95% Confidence Interval (CI) = 1.87 - 5.41; patient E.B. = 14 trials, mean control group = 2.9; SD = 3.04) and made significantly more ‘no responses’ during peripheral vision conditions with both hands (left hand: p = 0.033, z_{cc} = 6.64; 95% CI = 1.28 - 3.97; patient E.B. = 4 trials, mean control group = 0.7; SD = 1.25; right hand: p < 0.001, z_{cc} = 12.38; 95% CI = 6.74 - 18.01; patient E.B. = 10 trials, mean control group = 1.34; SD = 0.7). In Experiment 2, there were no

significant differences between the number of 'no responses' made by patient and controls (all p's > 0.72).

Experiment 1: Peripheral Reaching

The case-control comparisons of absolute reaching error (Figure 4A; Table 1) revealed a field effect in E.B. akin to what is typically observed in optic ataxia (e.g., Perenin & Vighetto, 1988; Blangero et al., 2008). Patient E.B. demonstrated a significant dissociation between peripheral vs. free vision specifically in her left visual field regardless of hand used: her accuracy degraded more than that of controls in left peripheral vision for both hands. No other statistically significant deficits were observed for absolute reaching error, including for the hand comparison analyses.

For RT (Figure 4B; Table 1), E.B. displayed a general slowing to initiate reaching with her right hand during peripheral reaching across the visual field. This impairment was most pronounced for the left visual field, according to a set of additional BSDTs that compared RT performance of the right hand across visual fields [left vs. centre fields: p = 0.006, zDcc = 7.08 (95% CI = 2.13, 11.08), proportion above case (%) = 0.28 (95% CI = < .001, 1.67); left vs. right fields: p < .001, zDcc = 8.48 (95% CI = 3.35, 12.62), proportion above case (%) = 0.04 (95% CI = < .001, 0.04)]. In addition, the hand comparisons demonstrated that E.B. was generally slower with the left, relative to right, hand in free vision across visual fields (Figure 4B).

For MT (Figure 4C; Table 1), E.B. was faster in peripheral, than free, vision when using the left hand to reach, in both visual fields. The hand comparison analysis additionally showed that MT for the left visual field specifically was faster for the left, than right, hand.

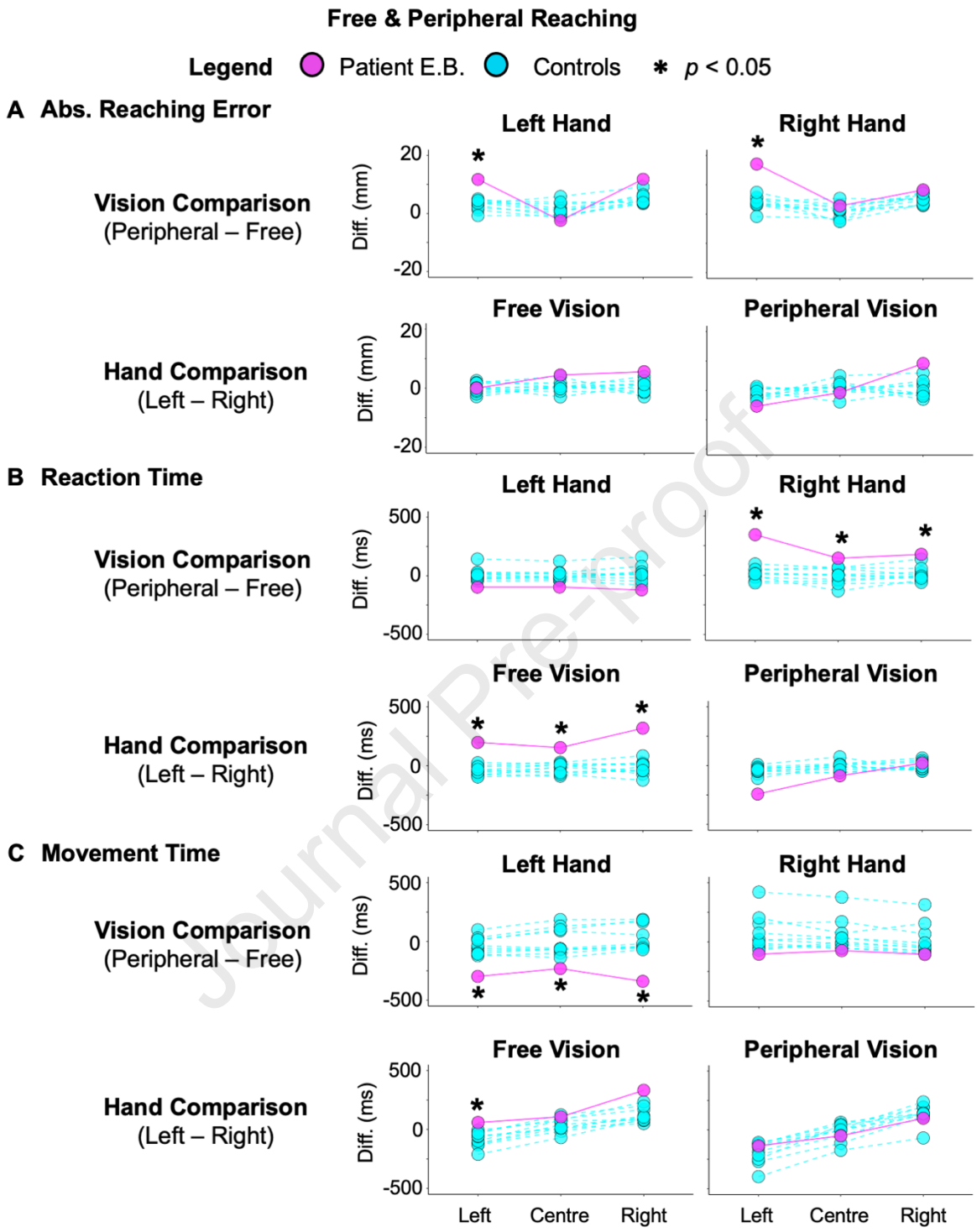

Figure 4. **Free and Peripheral reaching mean differences in Experiment 1** for controls and E.B on absolute reaching error (A), reaction time (B) and movement time (C). Greater values reflect larger absolute reaching errors (i.e., poorer reaching accuracy) or larger reaction/movement times (i.e., slower reaching). Results are shown for the difference (diff.)

when subtracting performance during the vision conditions (vision comparison) and hand conditions (hand comparison).

Table 1. Case-control statistics for each condition (cond.) for experiment 1.

Cond.	BSDT		*p*	zDcc	CI Low.	CI Upp.	Prop. (%)	CI Low.	CI Upp.
Abs. Reaching Error									
Left									
	Vision Comparison	**Left Hand**	**.003**	**-5.22**	**-8.27**	**-2.23**	**0.15**	**<.001**	**1.3**
		Right Hand	**.001**	**-6.09**	**-9.61**	**-2.57**	**0.07**	**<.001**	**0.51**
	Hand Comparison	Free Vision	.803	-0.02	-0.63	0.62	40.15	23.87	49.59
		Peripheral Vision	.291	-0.41	-4.48	3.59	14.53	<.001	47.18
Centre									
	Vision Comparison	Left Hand	.174	1.61	.56	2.55	8.72	0.54	28.78
		Right Hand	.524	-.7	-1.39	.03	26.19	8.2	47.51
	Hand Comparison	Free Vision	.073	2.27	.91	3.41	3.63	.03	18.02
		Peripheral Vision	.544	-.66	-1.34	.06	27.2	9.03	47.84
Right									
	Vision Comparison	Left Hand	.151	-2.58	-5.63	0.49	7.54	<.001	43.78
		Right Hand	.311	-1.25	-2.33	-0.08	15.55	1	44.08
	Hand Comparison	Free Vision	.115	2.11	0.56	3.74	5.74	0.01	28.79
		Peripheral Vision	.056	3.12	0.72	5.73	2.81	<.001	23.58
Reaction Time									
Left									
	Vision Comparison	Left Hand	0.269	1.58	-1.47	4.51	13.46	<.001	47.31
		Right Hand	**0.002**	**-7.26**	**-10.93**	**-2.75**	**0.08**	**<.001**	**0.3**
	Hand Comparison	**Free Vision**	**0.001**	**8.73**	**3.53**	**12.89**	**0.03**	**<.001**	**0.02**
		Peripheral Vision	0.064	-4.13	-7.5	-0.03	3.19	<.001	36.6
Centre									
	Vision Comparison	Left Hand	0.128	2.38	0.12	4.4	6.42	<.001	39.78
		Right Hand	**0.01**	**-4.32**	**-6.59**	**-1.62**	**0.52**	**<.001**	**5.27**
	Hand Comparison	**Free Vision**	**<.001**	**9.66**	**4.16**	**13.99**	**<.001**	**<.001**	**<.001**
		Peripheral Vision	0.102	-2.59	-4.63	-0.3	5.08	<.001	35.7
Right									
	Vision Comparison	Left Hand	0.295	1.43	-0.67	3.45	14.77	0.03	47.07
		Right Hand	**0.024**	**-3.21**	**-4.76**	**-1.32**	**1.21**	**<.001**	**9.29**
	Hand Comparison	**Free Vision**	**0.001**	**6.8**	**2.99**	**9.91**	**0.03**	**<.001**	**0.14**
		Peripheral Vision	0.08	2.7	0.52	4.48	4.02	<.001	29.66
Movement Time									
Left									
	Vision Comparison	**Left Hand**	**0.001**	**8.25**	**3.25**	**12.61**	**0.03**	**<.001**	**0.06**
		Right Hand	0.079	2.31	0.79	3.83	3.93	0.01	21.46

	Hand Comparison	**Free Vision**	**0.042**	**4.26**	**0.64**	**7.96**	**2.11**	**<.001**	**25.32**
		Peripheral Vision	0.317	1.19	0.21	2.01	15.85	2.21	41.26
Centre	Vision Comparison	**Left Hand**	**0.027**	**3.36**	**1.19**	**5.57**	**1.36**	**<.001**	**11.8**
		Right Hand	0.09	2.35	0.64	4.04	4.48	<.001	26.16
	Hand Comparison	Free Vision	0.295	1.41	-1.17	3.9	14.77	<.001	47.58
		Peripheral Vision	0.735	-0.2	-0.96	0.59	36.76	16.5	49.39
Right	Vision Comparison	**Left Hand**	**0.005**	**4.64**	**1.94**	**7.31**	**0.27**	**<.001**	**2.63**
		Right Hand	0.126	1.94	0.58	3.27	6.3	0.05	28.01
	Hand Comparison	Free Vision	0.098	2.89	0.12	5.68	4.89	<.001	37.77
		Peripheral Vision	0.663	-0.46	-1.07	0.23	33.17	14.18	49.04

Estimates for effect sizes (z_{Dcc}) and the proportion (Prop.) of the control population (%) obtaining a lower score than the case, are followed by 95% Confidence Intervals (CI). Bold rows show significant findings, based on eight degrees of freedom BSDT.

Experiment 2: Bimanual Reaching

In terms of RT, E.B. demonstrated no specific increased bimanual cost when compared to controls, showing that her bimanual reaching initiation was similar in timing to that of controls despite her visual neglect and extinction (Figure 5; Table 2). However, the bimanual cost for MT was significantly higher for E.B. than controls: she took longer to complete bimanual incongruent reaches than controls with both hands (Figure 5; Table 2). In terms of absolute reaching error E.B. demonstrated no specific increased bimanual cost when compared to controls (Figure 5; Table 2).

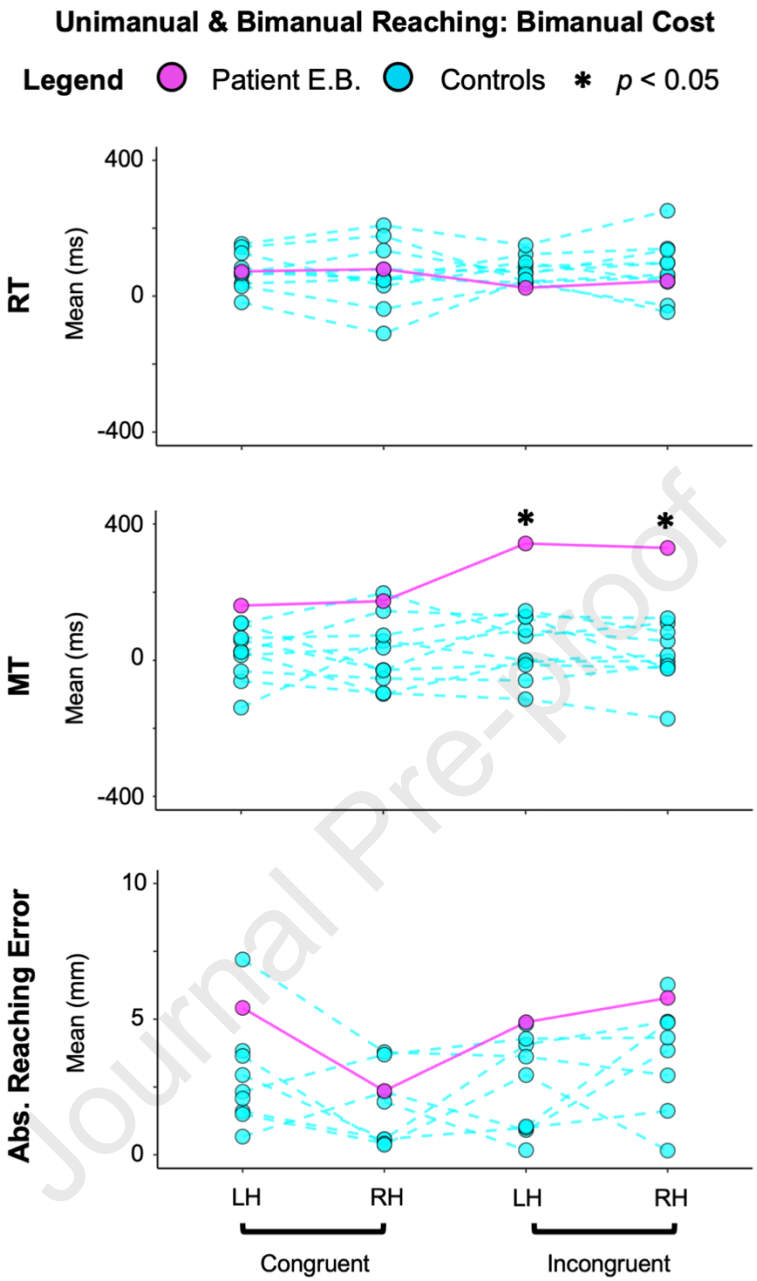

Figure 5. Bimanual cost means for controls and E.B. for absolute reaching error, reaction time and movement time. *RH = right hand; LH = left hand.*

Table 2. Bimanual cost statistics.

Condition			*p*	zcc	CI Low.	CI Upp.	Prop. (%)	CI Low.	CI Upp.
RT									
	Cong.	Left	0.8	-0.06	-0.68	0.58	39.99	23.49	49.59
		Right	0.778	0.17	-0.46	0.79	38.92	21.42	49.51
	Incong.	Left	0.231	-1.35	-2.2	-0.46	11.54	1.41	32.14
		Right	0.67	-0.42	-1.06	0.24	33.49	14.42	49.17
MT									
	Cong.	Left	0.113	1.85	0.78	2.88	5.66	0.2	21.79
		Right	0.176	1.54	0.59	2.46	8.8	0.7	27.6
	Incong.	**Left**	**0.009**	**3.47**	**1.79**	**5.13**	**0.46**	**<.001**	**3.71**
		Right	**0.007**	**3.67**	**1.88**	**5.43**	**0.34**	**<.001**	**2.99**
Absolute Reaching Error									
	Cong.	Left	0.215	1.4	0.5	2.27	10.76	1.16	30.88
		Right	0.526	0.69	-0.03	1.35	26.31	8.78	47.65
	Incong.	Left	0.608	0.53	-0.15	1.17	30.41	12.05	48.73
		Right	0.282	1.2	0.35	2.01	14.11	2.23	36.11

Bold rows show significant findings, based on nine degrees of freedom for each BDT. *Cong. = Bimanual Congruent; Incong. = Bimanual Incongruent.* For remaining conventions, see Table 1.

Discussion

In the present exploratory study, we tested unimanual free versus peripheral reaching and unimanual versus bimanual reaching (in free vision) in an unusual patient who presented visual neglect and extinction, with no hemiparesis or homonymous hemianopia. This allowed us to examine, for the first time, if a patient with lateralised attentional deficits, such as neglect and extinction, is impaired in peripheral and bimanual reaching tasks, providing data to inform future studies in a larger sample of patients.

Impairments in peripheral reaching

We first investigated whether her visual neglect and extinction was accompanied by misreaching in peripheral vision by using the classic optic ataxia paradigm (i.e., free vs. peripheral reaching; e.g. Blangero et al, 2010; Borchers et al., 2013; Milner et al., 1999;

Perenin & Vighetto, 1988). E.B. showed obvious signs of an optic ataxia field effect (Figure 4A): her reaching accuracy was significantly more impaired than controls in the contralesional peripheral visual field regardless of the hand used (e.g., Perenin & Vighetto, 1988; Revol et al., 2003). Clearly, neurological patients can demonstrate symptoms of optic ataxia without concurrent clinical neglect or extinction (e.g., Ratcliff & Davies-Jones, 1972; Perenin & Vighetto, 1998; Revol et al., 2003; Karnath & Perenin, 2005). However, experimentally measuring attentional performance using computerised tasks can reveal additional lateralised attentional deficits in optic ataxia (e.g., Revol et al. 2003; Pisella et al., 2007; Blangero et al., 2010). In the present case, we show that the reverse may also be true: testing reaching in peripheral vision may reveal additional lateralised visuomotor impairments associated with neglect and extinction.

Finding an optic ataxic left field effect where reaching errors occur to the contralesional field (e.g., Revol et al., 2003; Dijkerman et al., 2006; Himmelbach & Karnath, 2005; Khan et al., 2005; Striemer et al., 2009) alongside the presence of left visual neglect and extinction is consistent with a growing body of evidence that patients with optic ataxia also present difficulties allocating attention to their ataxic field (for reviews, see Rossetti et al., 2000, 2003; Pisella et al., 2007; 2009; 2021). The question then is, whether the attentional and visuomotor deficits in both optic ataxia and neglect share the same functional basis? In this vein, Pisella et al. (2007) suggested that the perceptual ability of patients with optic ataxia is impaired in peripheral vision due to impairment of visuo-spatial processing and/or spatial attention, but that this attentional deficit is different to the one causing neglect. They argue that optic ataxia patients systematically fail under conditions in which the location of gaze is dissociated from the location of the visual stimulus of perceptual or motor interest. Pisella et al. (2021), further suggest that these deficits result from a core mislocalization deficit, in accordance with their previous proposal that the core deficit of Bálint's syndrome is attentional (Pisella et al., 2009, 2013, 2017). Because covert attention improves spatial resolution in the visual periphery (Yeshurun and Carrasco, 1998), a deficit

of covert attention would increase spatial uncertainty and impair both visual object identification and visuomotor accuracy. In line with this view, it has been shown that the perceptual and motor deficits significantly correlate in patients with optic ataxia (McIntosh et al., 2011). These neuropsychological cases include optic ataxia patients that present attentional disengagement impairments, akin to visual extinction (Michel & Henaff, 2004; Striemer et al., 2007). In a complementary way, the symptoms of neglect and extinction in patient E.B. are accompanied by visuomotor deficits akin to optic ataxia. The present study establishes a precedent for being able to test optic ataxia in a patient with neglect, and our results indicate that optic ataxia may be identified in neglect patients by measuring peripheral reaching with the ipsilesional limb alone (since E.B. was impaired with both hands), at least in patients without complete contralesional hemianopia. This point is valuable because it would allow testing for optic ataxia in a larger sample of patients, including in patients with contralesional hemiparesis, as only the right hand would need to be tested.

Nevertheless, it is important to acknowledge that patient E.B.'s optic ataxia may be caused by her dorsal visual stream lesions (Milner & Goodale, 1995; 2006) since her stroke extended to the superior parietal lobule (Figure 1A). That is, her behaviour could represent a case of optic ataxia co-occurring with neglect due to concomitant lesions to the inferior and superior PPC, rather than implying a direct functional association. Therefore, future research is needed to examine more fully the relation between attention and visuomotor problems in patients with neglect and extinction. Lesion-symptom mapping studies and/or functional neuroimaging in patients with optic ataxia and neglect will help address this limitation of our study.

Another limitation of this case study is that, while we found E.B. was spared in a clinical test of proprioception ability, we were unable to run an experimental motion-tracking task of proprioceptive pointing (Blangero et al., 2007), due to time restrictions on testing. It would be interesting to include this condition in future studies in patients with neglect and

extinction. In a similar vein, future studies should examine ipsilateral peripheral reaching with and without visual feedback, since both hand and field effects are exacerbated in open loop (e.g., Blangero et al., 2007). Nevertheless, E.B.'s presentation of a left field effect is consistent with her right hemisphere stroke; hand effects are more commonly observed in optic ataxia patients following lesions to the opposite (left) hemisphere (Perenin & Vighetto, 1988). In fact, the absence of a hand effect in E.B. could suggest that only the visuomotor deficits in the contralesional visual field ('field effects') in optic ataxia are related to attention.

Appealing to a theoretical account that functionally links attentional and visuomotor disorders (rather than a strict visuomotor account where these disorders are strongly dissociated) better accounts for another aspect of E.B.'s peripheral reaching behaviour. Specifically, aside from her general RT slowing with the right hand for peripheral vision reaches (Figure 4B; also seen in optic ataxia patient M.H.'s RTs; Rice et al., 2008), we found that E.B. was especially slow to initiate these reaches to the contralesional side (relative to the central/ipsilesional targets; Figure 4B). The opposite explanation, where E.B.'s slow RT is simply due to damage to key visuomotor control nodes (e.g., due to her right hemisphere posterior parietal lesion) cannot account for why this slowing occurred only for the ipsilesional hand (i.e., a right posterior parietal lesion would predict a slowing of the *contralesional* hand and would not lead to the observed exacerbation of this effect for the contralesional field). In fact, it would be interesting to investigate timing differences between free vs. peripheral version in patients with optic ataxia as most studies to date have focused on accuracy alone.

Impairments in incongruent bimanual reaching

Having established that E.B. was impaired in peripheral reaching we then investigated her bimanual performance in congruent vs. incongruent conditions in free vision. This revealed that despite her visual extinction (inability to perceptually report bilateral stimuli) in our computerised extinction test, E.B. was able to initiate both congruent and incongruent bimanual actions at the same speed and accuracy as controls, thus confirming she does not

have motor neglect. However, once initiated, her bimanual movement execution was markedly slow for both hands when reaching to spatially incongruent targets simultaneously. Importantly, the fact that she only presented deficits in incongruent trials suggests that her bimanual deficit in movement time cannot be caused by motor extinction since if this was the case, deficits would have also been observed in congruent bimanual trials. Instead, our hypothesis is that E.B. slowed down in the incongruent condition to allow her to switch her attention between the competing movements of the two hands to maintain accuracy. Thus, her attentional limitations may have impaired her performance on this task, but this was measurable in terms of movement duration for both hands, rather than visible as elevated spatial error. This suggests that her attentional deficits significantly increased bimanual interference even for her ipsilesional hand. These findings may represent the first causal evidence that damage to the right posterior parietal cortex impairs processing of motor conflict during bimanual incongruent movements, in line with previous neuroimaging findings in healthy participants (e.g., Wenderoth et al., 2004; 2005; Diedrichsen et al., 2006). Of course, the lack of accuracy deficits in bimanual tasks in E.B. could also be explained by the task being performed with free vision so it would be important to compare bimanual reaching in both free and peripheral vision in future studies.

Considering her specific reaching deficits in peripheral and bimanual incongruent conditions it could argued that E.B. shows visuomotor deficits only in tasks that required visual attention or eye movements to be decoupled from hand movements. As vision could only be directed to one target at a time, the bimanual incongruent task created a challenge as to where to direct vision as the movement unfolded with each limb and target competing for visual resources (Sardar et al., 2023). In a similar vein, the peripheral reaching task requires attention to be directed at fixation while reaching is performed to a peripheral/ off-fixation location. On the other hand, while her peripheral reaching deficits in the left visual field follows her left lateralised attentional deficit, the bimanual deficits are present on both sides of space suggesting a different underlying mechanism. In any case, it is interesting to

note that patients with optic ataxia, similarly to E.B., also present deficits in bimanual actions suggesting that there is a larger overlap between the behaviour of patients with neglect and optic ataxia than previously assumed (Perenin & Vighetto, 1988; Perenin, 1997; Harvey & Rossit, 2012). In the future, it would be important to test bimanual incongruent actions also within the same hemifield so that visual field and hand effects can be dissociated.

Conclusion

Overall, we report a patient with clinical neglect and extinction who also presents a clear optic ataxic 'field effect' and temporal deficits in bimanual reaching to incongruent locations. We hope that these findings lay the foundations for future enquiry investigating whether optic ataxia is common in patients with neglect and hemiparesis by testing peripheral reaching with the ipsilesional limb alone. Theoretically our data is consistent with a fundamental role of PPC in attention rather than "pure" visuomotor control, in line with a large body of work (e.g., Pisella et al., 2007). Thus, both neglect and optic ataxia may be associated with attentional and visuomotor deficits in the affected visual field when eye position and target location are dissociated. Attentional limitations may also cause problems in simultaneously monitoring bimanual movements made bilaterally. Future research is needed to investigate the occurrence of visuomotor impairments in larger group of patients with neglect and/or extinction, and to further characterise lateralised attention in patients with field-dependent optic ataxia.

Credit author statement

Ethan Knights: Methodology, Software, Formal Analysis, Investigation, Data Curation, Writing - Original Draft, Visualization, Project administration. **Robert McIntosh:** Methodology, Software, Formal Analysis, Investigation, Writing - Review & Editing. **Catherine Ford:** Resources, Writing - Review & Editing. **Gavin Buckingham:** Conceptualization, Methodology, Writing - Review & Editing. **Stéphanie Rossit:** Conceptualization, Methodology, Software, Formal Analysis, Investigation, Writing – Original

draft; Writing - Review & Editing, Supervision, Project administration, Resources, Funding acquisition.

Declaration of interest

None.

Acknowledgements

We would like to thank patient E.B. for her patience and kind participation as well as all control participants. Additionally, we thank Annie Warman, Dr. Helen Morse, and Dr. David P. Carey and the other reviewer for their constructive comments on an earlier draft of this manuscript.

Funding

This work was funded by the University of East Anglia.

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