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Title: Reconsidering the role of attention in optic ataxia: A case report of optic ataxia in visual neglect and extinction

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Highlights

- We assessed optic ataxia in a right-hemisphere stroke patient with left visual neglect and extinction.
- Peripheral misreaching was observed in the contralesional field for both hands, as reported in optic ataxia.
- Results challenge the claims of a dissociation between attentional and visuomotor deficits in visual neglect and optic ataxia.

Abstract

Optic ataxia is a rare condition following dorsal visual stream lesions characterized by peripheral misreaching. This 'pure' visuomotor condition is claimed to be dissociable from attentional disorders, such as visual neglect and extinction. However, the relationship between optic ataxia, neglect and extinction is still controversial and recently it has even been suggested these conditions have a common attentional cause. In this single-case report, we investigated 67-year-old female patient E.B. with left visual neglect and extinction following right temporo-parietal and frontal strokes. Unlike most neglect patients, E.B. did not present left hemiparesis or left homonymous hemianopia. This unusual presentation allowed us to study, for the first time, the impact of left visual attentional disorders on reaching with both arms in both free and peripheral vision conditions. Specifically, we assessed whether patient E.B. would present the classic peripheral misreaching deficits reported in patients with optic ataxia. We found that patient E.B.'s reaching accuracy was significantly impaired, when compared to a gender and age-matched control sample (N=11), only in peripheral vision and only for targets presented in her neglected field, regardless of the hand used. Her performance was comparable to controls in free vision for both arms. This demonstrates that a patient with visual neglect and extinction also presents an ataxic field, a deficit typically described as optic ataxia. Our findings suggest that, at least in peripheral vision, attention and visuomotor deficits

- 43 may be related which challenges long-standing claims of a dissociation between attentional
- 44 disorders and optic ataxia.
- 45 **Keywords:** optic ataxia; attention; visual neglect; extinction; peripheral vision

46 1. Introduction

47 Misreaching to peripheral (but not central) visual targets is the hallmark of optic ataxia. This disorder
48 was initially described in a patient with Balint's syndrome and is generally considered a 'pure' deficit
49 of visuomotor control which cannot be explained by pure motor or visual deficits (Balint 1909,
50 translated by Harvey, 1995). As detailed in Perenin & Vighetto's (1988) seminal observations, optic
51 ataxic patients can show impaired peripheral reaching to only the contralesional visual field (i.e.,
52 field effect) and/or with the contralesional hand (i.e., hand effect), with more recent motion-tracking
53 studies replicating these field/hand effects (e.g., Revol et al., 2003; Jackson et al., 2005; Blangero et
54 al., 2007; 2008; 2011 Khan et al., 2005; Medina et al., 2020).

55 Classically, optic ataxia impairments of visuomotor control are often claimed to be
56 dissociable from disorders of visual attention (Perenin, 1997; also see Coulthard et al., 2007;
57 Himmelbach et al., 2007; Rossit & Harvey, 2012; Pisella et al. 2021), including visual neglect (i.e.,
58 attentional biases toward ipsilesional side of space; Bartolomeo, 2007) and visual extinction (i.e.,
59 difficulty disengaging from ipsilesional stimuli when both fields are simultaneously stimulated; De
60 Renzi, 1982). From a behavioural perspective, we and others have repeatedly shown that, despite
61 their difficulty re/-orienting attention to their contralesional side, visual neglect/extinction patients
62 show (unlike optic ataxia patients) no reaching impairments for actions directed to contralesional
63 space (e.g., Chieffi et al., 1993; Konczak & Karnath, 1998; McIntosh et al., 2002; Himmelbach &
64 Karnath, 2003; Rossit et al., 2009a; 2009b; 2011a; 2011b; Rossit, McIntosh, et al., 2012; McIntosh et
65 al., 2004; Milner & McIntosh, 2004). Likewise, neuroanatomical evidence shows that lesions typically
66 associated with attentional disorders and optic ataxia are distinct: optic ataxia is associated with
67 damage to the superior parietal lobule, intraparietal sulcus (e.g., Pisella et al., 2009) and occipito-
68 parietal junction (Karnath & Perenin, 2005) whereas more inferior locations are affected in visual
69 neglect (e.g., inferior parietal lobule; Mort et al., 2003; superior temporal gyrus; Karnath et al., 2001;
70 2004; 2011) or extinction (temporo-parietal junction; Karnath et al., 2003; Ticini et al., 2010).

Nevertheless, it remains unclear if there is a functional relationship between attentional biases and optic ataxia (particularly regarding the ‘field effect’). Classic descriptions of Balint’s syndrome describe optic ataxia, neglect and simultagnosia (i.e., a narrowing of attentional space to single objects) as a triad of closely related symptoms (Balint 1909, translated by Harvey, 1995; Pisella et al., 2009) and, further, Pisella et al. (2021) recently proposed that field effects in optic ataxia may be accounted for by a pathological “compression of visual space” (i.e., a disorder of visual attention; see also Pisella et al., 2007; 2009). In fact, this would explain why optic ataxia patients also show subtle impairments in detection of visual targets in the contralesional peripheral field (McIntosh et al., 2011; also see Striemer et al., 2009) or deficits in visual change detection for target location, size or orientation (Pisella et al., 2009; Medina et al., 2020). Such accounts that posit weaker distinctions between these attentional disorders and optic ataxia would also be commensurate with the fact that many prior optic ataxia cases have been described as either recovering from, or exhibiting symptoms that, resemble neglect (case 4, Hecaen & Ajuriaguerra, 1954; patient C.F., Khan et al., 2009; also see Striemer et al., 2007; 2008; 2009; cases 6 & 8, Perenin & Vighetto, 1998; patient M.H., Kitadono & Humphreys, 2007; Snow et al., 2013), difficulties processing objects away from the focus of visual attention like visual extinction/simultagnosia (patient I.G., Pisella et al., 2000; McIntosh et al., 2011; Bartolo et al., 2018; Vialatte et al., 2021; patient A.T., Michel & Henaff, 2004; Gaveau et al., 2008), or their combination (patient C.F., Khan et al., 2009; patient V.K.; Jakobson et al., 1991; patient O.K.N; Pierrot-Deseilligny et al., 1986; also see Rondot et al., 1977). Indeed, links between attention and visuomotor functions are the premise of several theoretical models of attention (e.g., premotor theory of attention or theory of event coding; e.g., Rizzolatti et al., 1987; Hommel et al., 2001; Smith & Schenk, 2012).

Taken together, there appears little consensus about whether attentional and visuomotor impairments are functionally linked in neurological populations. This is largely due to too few patients being tested, with too much divergence in methods. Since almost all patients with visual neglect syndrome present concomitant contralesional hemiparesis and hemianopia (Zebhauser et

al., 2019), investigations of reaching with these patients have exclusively examined ipsilesional reaching (typically right-hand only) to targets viewed in free vision. However, reaching accuracy could be impaired in neglect patients for the contralesional (rather than ipsilesional) left arm, which is chiefly controlled by the contralateral lesioned right hemisphere in most patients (for a similar argument see Harvey & Rossit, 2012). If found, this pattern of performance would resemble the increase in reaching errors seen in optic ataxia for the contralesional hand (e.g., Revol et al., 2003; Cavina-Pratesi, 2013). Further, because misreaching in optic ataxia is confined to reaches for targets viewed in peripheral vision (e.g., Jackson et al., 2009), it is also vital to examine the ability of neglect patients when reaching to targets in peripheral vision (rather than just in free vision conditions which has been the case so far). Thus, arguably, the only way to explore the link between attentional disorders and optic ataxia hand/field effects, is to examine both kinds of patients under the same experimental conditions: while it is well-established that patients with optic ataxia often pass basic clinical screening for attentional disorders (cancellation or confrontation tasks for neglect and extinction, respectively; e.g., Perenin & Vighetto, 1988; Meichtry et al., 2018), we need to test for optic ataxia hand and field effects in visual neglect/extinction by examining reaching with both hands, and under peripheral vision conditions, across visual fields.

In this single-case report we describe a rare stroke survivor with left visual neglect and extinction who, despite extensive right fronto-parietal lesions, retained full function of both arms and did not present a complete left homonymous hemianopia. This unusual presentation allowed us to test, for the first time, a patient with neglect and extinction on the standard optic ataxia task of reaching with both arms to targets presented in free and peripheral vision. Case-control statistics were used to compare her visuomotor accuracy (and movement timing) to that of healthy age- and gender-matched controls.

2. Materials and Methods

2.1 Participants

2.1.1. Patient E.B.

Patient E.B. was a 67-year-old right-handed retired woman who previously worked in a graduate position. She suffered two middle cerebral artery infarcts in the right hemisphere in her sixties. A computed tomography scan indicated lesions encompassing right frontal and parieto-temporal areas (Figure 1A). Specifically, the right parietal lesion comprised the superior parietal lobule which 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 which extended inferiorly into the orbitofrontal/cingulate gyri and insula. Eleven months post-stroke, patient 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 patient E.B.) provided informed consent prior to participation and were reimbursed for their time.

Patient E.B. was independent, well oriented, fully mobile and had spared motor speed, praxis, memory (working and episodic) and language abilities. She reported slight numbness in the left thumb and index finger, fatigue on left upper limb but was able to move both arms. As seen in Figure 1B, computerised perimetry visual field testing (e.g., Rossit et al., 2009a) detected a left lower quadrantanopia, where E.B. failed to detect stimuli presented 12° below fixation, but only when further than 16° in the horizontal axis. She also showed a left visual extinction in a computerised confrontation test (Figure 1C; <90% detection rates for unilateral left / right and catch stimulus presentations, compared to 8.3% for bilateral presentations; Rossit et al., 2009). Evidence of extinction was not observed in tactile or auditory domains during physical confrontation (100% detection rates). Finally, she also presented left visual neglect in several tasks (Figure 1D): E.B. was

145 impaired on several cancellation tasks (star cancellation: total score / cut-off = 50 / 51; left score =
146 23; Wilson, Cockburn & Halligan, 1987; Balloons test: index / cut-off = 37.5% / 45%; Edgeworth,
147 Robertson & McMillan, 1998), showed a significant rightward bias during line bisection (average
148 error / cut-off = 14mm / 6mm; Rossit et al., 2009a; Figure 1D) and omitted key left sided details
149 during figure copy (Figure 1C; Wilson, Cockburn & Halligan, 1987). Patient E.B. showed no visible
150 signs of hemiparesis (could move her arm freely) and had no proprioceptive difficulties (100%
151 accuracy using the contralesional limb to point to her own body parts on command with eyes closed;
152 Blangero et al., 2007).

Figure 1. Patient E.B.'s case history. **(A) Lesion map.** Patient E.B.'s area of damage was manually demarcated using MRIcron (Rorden, Karnath & Bonilha, 2007; <http://www.nitrc.org/projects/mricron>) 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, Bonilha, Fridriksson, Bender, & Karnath, 2012; <http://www.nitrc.org/projects/clinicaltbx>) with SPM12 (<https://www.fil.ion.ucl.ac.uk/spm>).

160 The lesion is shown in cyan overlaid on a high resolution scan provided within clinical toolbox (left;
161 MNI z coordinates of the transverse sections are given) and on a 3D rendered brain created with
162 MRICron (right). **(B) Visual field assessment.** A computerised perimetry test revealed left lower
163 quadrantanopia at 16° left and 12° below fixation. **(C) Visual extinction assessment.** Unilateral left
164 extinction was observed (failure to report left targets specifically during bilateral stimulation). **(D)**
165 **Visual neglect assessment.** On the day of the optic ataxia assessment, left visual neglect was
166 detected in several cancellation assessments (upper; circles show omitted targets), line bisection
167 and figure copying (lower).

2.1.2. Control Group

Fourteen neurologically intact healthy volunteers participated in the study. Three participants were excluded from data analysis due to reporting a glaucoma diagnosis, corrupted eye-tracking data or failing to respond to peripheral targets. Thus, the final control group used in analysis included 11 age and gender-matched participants (mean age = 65.9, SD = 4.7, range = 59 - 75). This group were all self-reported as right-handed (Oldfield, 1971) with no history of motor, psychiatric or neurological disorders and as having normal or corrected-to-normal visual acuity.

2.2. Free vs. Peripheral Reaching Task

2.2.1. Stimuli & Apparatus

Participants sat in an adjustable chair in front of our purpose-built grey table with their head stabilised in a chin rest aligned with the table midline (Figure 2A). A start box positioned 18.5cm (11°) in front of the participant was aligned with the participant's sagittal midline. Stimuli were circles (0.6cm²; ~0.5°) produced by Light-Emitting Diodes (LEDs) embedded in the table. Targets were red, located 40cm (26°) ahead of the participant in a horizontal line corresponding to distances of 0°, ± 11°, ± 17° & ± 28° with respect to the body's sagittal axis (e.g., Rossit et al., 2018; Figure 2A). The fixation was green, located 10cm (11°) ahead of the central target. Therefore, most targets (except the most eccentric) were clearly positioned above E.B.'s lower left quadrant scotoma, and during setup, patient E.B. was able to easily detect all targets under peripheral vision, including the most eccentric. The room was slightly darkened so that the targets were only visible when illuminated, but the participant's hands were visible 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. Participants rested their head in a chinrest and wore a head-mounted binocular eye-tracking system (Sensomotoric Instruments; 29Hz)

throughout the experiment to ensure 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 controlled by an in-house program written in Matlab (MathWorks Inc. 2013) with Psychtoolbox 3 (Brainard, 1995).

2.2.2. Procedure

A free vision and a peripheral vision condition were completed, with each hand separately, within independent blocks (Figure 2B) which were carried out in the following fixed order (to allow direct comparison between patient E.B. and the control group): right hand free vision, left hand free vision, right hand peripheral vision and 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 upper). Peripheral vision conditions involved performing the same action whilst maintaining fixation on the central light (Figure 2B lower) such that targets appeared in the periphery and participants were not allowed to move their eyes.

Target onset was triggered by the participant's button press (black circle in Figure 2). For all conditions, the fixation was continuously illuminated, and participants were instructed to act quickly and accurately. Conditions included 70 trials (i.e., 10 trials per seven target locations in a randomised order) where a single trial appeared and remained illuminated until the end of a trial (2s). Central fixation was monitored online and verbal feedback about fixation was provided to participants during peripheral vision conditions. To mitigate the issue of excluding trials from analysis due to errors (e.g., fixation errors or reaching errors), additional short blocks were completed immediately after a given block (i.e., only if participants committed errors and time permitted; for trial distributions see section: Materials and Methods - Data Processing).

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), one

217 by one, allowing the participants to adjust their terminal fingertip position until they believed the
218 target was perfectly occluded.

Free vs. Peripheral Reaching Task: Optic Ataxia Assessment

Figure 2. Free vs. Peripheral reaching task. **(A) Apparatus and stimuli.** 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 (-28° , -17° and -11°), central (0°) and right visual fields ($+11^{\circ}$, $+17^{\circ}$, $+28^{\circ}$) as well as for the central target and fixation. (Rossit et al., 2018). **(B) Reaching conditions.** Example trials for free vision and peripheral conditions (upper vs. lower panels). The dashed lines represent eye gaze. The four conditions, combining each vision condition with each hand, were performed in separate blocks.

2.3. Data processing

Raw movement data was processed using a custom Matlab scripted interface. 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. After online and offline screening (see error descriptions below), modified single case t-tests (see section: Materials & Methods - Statistical Analysis) showed there were no significant differences between the number of trials analysed for patient E.B. and the mean of the control group for any of the four conditions (collapsed across target locations: all p 's > 0.37; mean total trials = 11). First, we excluded specific trials from further analysis with eye fixation errors [94 (2.8%) trials were excluded due to saccade detection during peripheral vision] and where no response was performed [35 (1.6%) trials]. Analysis of error frequencies showed that patient E.B. had difficulty with the peripheral vision condition because, relative to the control group, she committed significantly more eye fixation errors 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 also 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)]. We also excluded specific trials due to data acquisition errors (77 (2.3%); i.e., obscured infrared markers) or reaching errors (15 (0.4%); i.e., slow responses such that end velocity criterion was not exceeded and no end coordinate could be obtained).

In the remaining trials, we computed absolute reaching error [$\sqrt{(X \text{ error}^2 + Y \text{ error}^2)}$] for comparability with previous optic ataxia studies (Milner et al., 1999; Rossetti et al., 2005; Revol et al., 2003; Blangero et al., 2007; Cavina-Pratesi, et al., 2010; Dijkerman et al., 2006; Striemer et al., 2009; Hesse et al., 2014). Measures of X and Y error were calculated by subtracting end-point coordinates from target 'true' location (obtained from calibration recordings). For exploratory analyses, we also computed Reaction Time (RT) (i.e., movement onset - stimulus onset) and

Movement Time (MT) (i.e., movement onset - movement offset) to examine if patient E.B. presented movement timing deficits.

2.4. Statistical analysis

For each variable, analysis was performed for each side of space (e.g., collapsing left vs. right target positions) and for the central target position, separately¹. To compare patient E.B. against control performance we used the Bayesian Test of Deficit (BTD; Crawford, Garthwaite & Porter, 2010) for each condition and visual field separately. Additionally, to test whether patient E.B. demonstrated a larger dissociation between the free vs. periphery (i.e., vision comparison) and right vs. left hand (i.e., hand comparison) conditions compared to condition differences observed in the control sample, we used the Bayesian Standardised Difference Test (BSDTs; Crawford & Garthwaite, 2007; Crawford, Garthwaite & Porter, 2010). 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 ($p = 0.05$); given the low power of case-control comparisons, we did not apply corrections for multiple comparisons (McIntosh & Rittmo, 2021).

2.5. Data Availability

Processed and analysed kinematic data are available on the Open Science Framework (<https://osf.io/q8nj6/>), including links to the code for conducting the experiment and analysis.

3. Results

3.1. Reaching accuracy

As can be seen in Figure 3, in free vision E.B.'s reaching end-points were like controls' except when she undershot the most eccentric right target with her left hand in a few trials. This may reflect a

¹ We also repeated analyses when treating the seven targets independently, but, since results converged with our main side-collapsed analysis, for brevity these are reported on the Open Science Framework (see section: Data Availability).

274 biomechanical limitation for patient E.B. in reaching across the body (e.g., due to arm-span), as it
275 was not specific to peripheral vision (i.e., this occurred in free vision conditions also). In contrast, for
276 the peripheral vision conditions, patient E.B.'s reach endpoints were visibly more variable for targets
277 presented in her left visual field, regardless of the hand used (especially in the Y plane; Figure 3B).

Figure 3. Normalised endpoint coordinates. Each marker represents the endpoint coordinate in the X and Y planes for a single trial for patient E.B. (Figure 3A) and a representative control (Figure 3B). Marker shade denote reaches to targets presented at different eccentricities and a black cross represents the reference for each normalised true target location.

In line with the descriptive data of patient E.B.'s end-points (Figure 3), the case-control comparisons of absolute reaching error (Figure 4; see Table 1 for statistics) revealed a field effect resembling what is typically observed in optic ataxia patients (e.g., Perenin & Vighetto, 1988; Blangero et al., 2008): accuracy was impaired, relative to controls, when reaching to the contralesional field with either hand, specifically in the peripheral vision condition. First, as in Figure 4A, for the peripheral vision conditions (bottom row) with both the left and right hands, patient E.B.'s absolute reaching error in the left visual field was significantly impaired relative to controls. Second, the vision comparison analysis demonstrated that patient E.B. had a significant dissociation between peripheral and free vision specifically in the left visual field (Figure 4B) where, for both hands, her accuracy degraded more than that for controls during the peripheral vision condition. Additionally, patient E.B. demonstrated a significant deficit for reaches to the right visual field with the left hand in both the free and peripheral vision conditions (Figure 4A). This final effect is likely attributed to biomechanics, since patient E.B. was short and struggled with across-body reachability for the extreme target (see right-most target in Figure 3A), particularly since no dissociation was observed between the visual conditions for this right visual field (see Table 1). No other statistically significant deficits were observed (Table 1), including for the hand comparison analyses (Figure 4C).

Figure 4. Absolute reaching error results. **(A) Accuracy results across visual fields.** Patient E.B.'s field effect is shown for the left visual field in peripheral vision (lower row) with both hands (left and right plots). **(B) Comparison of vision conditions.** This field effect is supported by a significant dissociation between the visual conditions for the left visual field where, for both hands, patient E.B. is more inaccurate than controls during the peripheral (vs. free) vision condition. **(C) Comparison of hand conditions.** A significant effect was not observed between E.B. and controls when comparing between-hand accuracy differences. Across panels, performance is shown for patient E.B. (clear circles in Figures A and outlined circles in Figures B/C) and each individual control participant (dashed lines in Figures A and non-outlined circles in Figures B/C), with Crawford cut-off error bars

309 (95% confidence interval) for the BTDs based on the control group's performance. Asterisks
310 represent $p < 0.05$.

Table 1. End-point accuracy case-control statistical results. Estimates for effect sizes (BTD: z_{cc} /BSDT: 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 nine and eight degrees of freedom for BTD and BSDT respectively. The *Free Vision* and *Peripheral Vision* rows correspond to direct comparisons (BTD) of these conditions between the patient and controls (Figure 4A). The *Vision Comparison* and *Hand Comparison* rows correspond to comparisons of between-condition differences (BSDT) for patient and controls (i.e., comparing the difference of free vs. peripheral/left vs. right hand conditions for the patient and controls; Figure 4B/4C).

Visual Field	BTD / BSDT		<i>p</i>	<i>z</i> cc / <i>z</i> Dcc	CI Low.	CI Upp.	Prop. (%)	CI Low.	CI Upp.
Left									
	Free Vision	Left Hand	.797	-0.1	-0.71	0.53	39.86	23.07	49.55
		Right Hand	.799	-0.08	-0.7	0.54	39.97	23.07	49.55
	Peripheral Vision	Left Hand	<.001	6.85	3.62	10	0.01	<.001	0.01
		Right Hand	<.001	7.29	3.96	10.57	<.001	<.001	<.001
	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
Central									
	Free Vision	Left Hand	.24	1.32	0.45	2.16	11.98	1.54	32.49
		Right Hand	.523	-0.69	-1.39	0.02	26.15	8.18	47.42
	Peripheral Vision	Left Hand	.53	-0.67	-1.34	0.03	26.48	9	47.63
		Right Hand	.741	0.27	-0.36	0.9	37.06	18.45	49.41
	Vision Comparison	Left Hand	.174	1.61	0.56	2.55	8.72	0.54	28.78
		Right Hand	.524	-0.7	-1.39	0.03	26.19	8.2	47.51
	Hand Comparison	Free Vision	.073	2.27	0.91	3.41	3.63	0.03	18.02
		Peripheral Vision	.544	-0.66	-1.34	0.06	27.2	9.03	47.84
Right									
	Free Vision	Left Hand	.01	3.45	1.76	5.1	0.48	<.001	3.9
		Right Hand	.515	0.7	<.001	1.39	25.74	8.18	47.31
	Peripheral Vision	Left Hand	<.001	5.71	3.04	8.38	0.02	<.001	0.12
		Right Hand	.117	1.82	0.77	2.83	5.85	0.23	21.94
	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

320 3.2. Movement Timing

321 RT analysis showed that patient E.B.'s left hand was slow in all conditions (Figure 5A/5B, Table 2).
322 However, her right hand was specifically slow during peripheral reaching (Figure 5A/5B; Table 2) and
323 this impairment was most pronounced for the left visual field according to additional BSDTs
324 comparing visual fields [left vs. centre: $p = 0.006$, $z_{Dcc} = 7.08$ (95% CI = 2.13, 11.08), proportion above
325 case (%) = 0.28 (95% CI = < .001, 1.67); left vs. right: $p < .001$, $z_{Dcc} = 8.48$ (95% CI = 3.35, 12.62),
326 proportion above case (%) = 0.04 (95% CI = < .001, 0.04)]. In addition, the hand comparison in free
327 vision, confirmed the general slowing of the left hand (Figure 5C upper).

Figure 5. Reaction time results. **(A) RT results across visual fields.** Patient E.B. showed a right hand slowing specific to the peripheral vision condition, and a general slowing of the left hand. **(B) Comparison of vision conditions.** Consistent with patient E.B.'s slowed right hand during peripheral vision, there were significant dissociations between vision conditions for all visual fields with additional visual field BSDTs showing that this effect was stronger for the left, than centre or right, visual field. **(C) Comparison of hand conditions.** The right hand was faster than the left throughout free vision. In peripheral vision, the left hand appeared to react faster than the right specifically for the left visual field.

337 Table 2. Reaction time results. For conventions see Table 1.

Visual Field	BTD / BSDT		<i>p</i>	zcc / zDcc	CI Low.	CI Upp.	Prop. (%)	CI Low.	CI Upp.
Left									
	Free Vision	Left Hand	0.001	5.01	2.68	7.38	0.05	<.001	0.37
		Right Hand	0.175	1.53	0.61	2.48	8.74	0.65	27.14
	Peripheral Vision	Left Hand	0.005	3.89	2.03	5.74	0.24	<.001	2.13
		Right Hand	0.001	5.26	2.78	7.73	0.04	<.001	0.27
	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									
	Free Vision	Left Hand	0.011	3.33	1.69	4.95	0.55	<.001	4.6
		Right Hand	0.496	0.73	0.02	1.42	24.79	7.77	46.73
	Peripheral Vision	Left Hand	0.102	1.91	0.83	2.94	5.12	0.16	20.34
		Right Hand	0.014	3.21	1.6	4.78	0.69	<.001	5.43
	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									
	Free Vision	Left Hand	0.01	3.44	1.72	5.14	0.49	<.001	4.28
		Right Hand	0.804	-0.04	-0.65	0.58	40.19	23.94	49.6
	Peripheral Vision	Left Hand	0.047	2.41	1.15	3.68	2.33	0.01	12.53
		Right Hand	0.168	1.57	0.62	2.5	8.41	0.63	26.92
	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

339 For MT, patient E.B. showed a general slowing of both hands during free, but not peripheral,
340 vision reaches (Figure 6A; Table 3). The specificity of these deficits to the free vision conditions were
341 particularly evident for the left hand since (1) the vision comparison showed a significant
342 dissociation between free and peripheral vision MTs (Figure 6B) and (2) the hand comparison
343 showed that the free vision slowing was more pronounced for the left than right hand when
344 reaching to the left visual field (Figure 6C; Table 3).

Figure 6. Movement time results. **(A) MT results across visual fields.** Patient E.B.'s general slowing for free vision is shown for both hands (upper left/right plots). **(B) Comparison of vision conditions.** This specific slowing for free vision is particularly evident for the left hand where there is a dissociation between vision conditions. **(C) Comparison of hand conditions.** A significant dissociation observed for the left visual field in free vision, where performance was worse for patient E.B.'s left, than right, hand.

352 Table 3. Movement time results. For conventions, see Table 1.

Visual Field	BTD / BSDT		<i>p</i>	zcc / zDcc	CI Low.	CI Upp.	Prop. (%)	CI Low.	CI Upp.
Left									
	Free Vision	Left Hand	<.001	7.38	4	10.97	<.001	<.001	<.001
		Right Hand	0.019	2.99	1.48	4.46	0.97	<.001	6.88
Peripheral Vision		Left Hand	0.415	0.89	0.14	1.62	20.77	5.3	43.4
		Right Hand	0.777	0.18	-0.45	0.81	38.87	20.7	49.56
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									
	Free Vision	Left Hand	0.002	4.47	2.39	6.6	0.11	<.001	0.83
		Right Hand	0.011	3.32	1.69	4.98	0.56	<.001	4.6
Peripheral Vision		Left Hand	0.548	0.65	-0.05	1.33	27.4	9.26	47.71
		Right Hand	0.486	0.76	0.02	1.46	24.29	7.26	46.6
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									
	Free Vision	Left Hand	0.001	5.38	2.83	7.89	0.04	<.001	0.23
		Right Hand	0.033	2.61	1.27	3.96	1.67	<.001	10.28
Peripheral Vision		Left Hand	0.797	-0.09	-0.71	0.53	39.86	23.26	49.58
		Right Hand	0.766	0.21	-0.42	0.83	38.31	20.18	49.49
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

4. Discussion

To investigate whether visual neglect is associated with misreaching deficits in peripheral vision, we examined the performance of a rare visual neglect and extinction patient during the classic optic ataxia paradigm (i.e., free vs. peripheral reaching). Patient E.B. demonstrated the classic optic ataxia field effect: her reaching accuracy was impaired for the contralesional peripheral visual field with both hands but spared in free vision (e.g., Perenin & Vighetto, 1988; Revol et al., 2003). Clearly, neurological patients can demonstrate symptoms of optic ataxia without concurrent clinically diagnosable attentional problems (e.g., Ratcliff & Davies-Jones, 1972; Perenin & Vighetto, 1998; Revol et al., 2003; Karnath & Perenin, 2005), however by testing and finding the reverse (i.e., peripheral reaching impairments in a patient with impaired attention) our results raise questions about whether either type of patient (optic ataxia, neglect/extinction) are ever free from associated attentional and visuomotor deficits in their affected field (at least when carefully testing for all conditions).

First, our results demonstrate that free vision reaching is unimpaired even when neglect patients are asked to reach with the contralesional limb. This rules out an important concern that most (if not all) previous accounts of spared reaching accuracy in neglect syndrome were simply due to only testing performance of the patients' ipsilesional limb (i.e., because this is the limb controlled by the non-lesioned hemisphere; Harvey & Rossit, 2012). Second, in the peripheral vision condition, we demonstrated the optic ataxia field effect in a patient with attentional disorders. In fact, patient E.B.'s accuracy impairments (Figure 3A/3B) mirror those of unilateral optic ataxia patients who display larger immediate pointing errors to their contralesional visual field (e.g., Revol et al., 2003; Dijkerman et al., 2006; Himmelbach & Karnath, 2005; Khan et al., 2005; Striemer et al., 2009). Finding this field effect, alongside visual neglect and extinction, challenges many prior claims, including our own, that the visual neglect syndrome and optic ataxia represent a double dissociation (e.g., Perenin & Vighetto, 1988; Perenin, 1997; Milner, 2004; Coulthard et al., 2006; Himmelbach et

al., 2007; Rossit & Harvey, 2012). Instead, our findings are consistent with a growing body of evidence showing that patients with optic ataxia also have difficulties allocating attention to their ataxic field like typically described in the neglect syndrome (for reviews, see Rossetti et al., 2000; 2017; Pisella et al., 2009; 2021), including evidence that these optic ataxia patients (as well as patient E.B.) show attentional disengagement impairments, akin to visual extinction (Michel & Henaff, 2004; Striemer et al., 2007).

The present finding of optic ataxia in a patient with attentional problems could be interpreted as evidence against the existence of ‘pure’ optic ataxia/neglect/extinction (e.g., if previous attentional patients were able to perform this careful assessment of peripheral/contralateral reaching, would they similarly show optic ataxia?). Importantly, patient E.B.’s neglect and optic ataxia impairments may be causally related to lesions of her dorsal visual stream (Milner & Goodale, 1995; 2006) given that her lesion extended into both inferior and superior parietal lobules (Figure 1A) which is a point that awaits verification from further neuropsychological studies or Transcranial Magnetic Stimulation (TMS). An important contribution of our results, therefore, is in providing evidence that measuring only ipsilesional arm movements may be the most practical approach for overcoming hemiparesis-related barriers in future optic ataxia assessments. That is, whilst future investigations can confirm whether a visual neglect syndrome patient without dorsal visual stream lesions is free from field effects (i.e., showing that field effects result from attentional impairments, rather than dorsal visual stream lesions per se), it is very unlikely such a patient would be able to perform reaches with their contralateral limb (unlike our rare patient E.B.). Therefore, by demonstrating that patient E.B.’s reaching errors occurred for both hands (Figure 3, Figure 4), our results suggest that testing only the ipsilesional limb in peripheral vision is sufficient to uncover visuomotor deficits in patients with attentional disorders.

In either case, appealing to an account that functionally links attentional and visuomotor disorders (rather than a strict visuomotor account) better describes two important aspects of patient

E.B.'s peripheral reaching behaviour. First, patient E.B. clearly showed a field effect on the same side as her attentional deficits. Arguably, future testing could examine whether the lack of a hand effect may be related to the presence of visual feedback in the current experiment, since hand effects are worsened by open-loop reaching conditions (Blangero et al., 2007). Nevertheless, the current results do fit with well with patient E.B.'s lesion because hand effects are more common in left-brain damaged patients whereas her strokes were in the right hemisphere (Perenin & Vighetto, 1988).

Second, aside from her general slowing (Figure 5; 6), particularly for peripheral vision conditions (also seen in optic ataxia patient M.H.'s RTs; Rice et al., 2008), patient E.B. was especially slow to initiate right hand reaches to the contralesional side (relative to the central/ipsilesional targets; Figure 4A/4B). This effect is expected given documentation of directional hypokinesia in neglect (e.g., Mijovic et al., 1991; Mattingley et al., 1992; Husain et al., 2000) and, crucially, this visuomotor deficit has long been contemplated as a cause of the pervasive attentional biases in the neglect syndrome (Heilman & Valenstein, 1979; Heilman et al., 1985; Mattingley et al., 1994). Further, such slowing resembles findings that optic ataxia patients' slow contralesional pointing reactions are correlated with their detection times (Striemer et al., 2009; McIntosh et al., 2011) as well as that during online correction, patients' pointing and saccadic (i.e., an overt measure of attention) behaviour share 'serial' characteristics (Gaveau et al., 2008; Pisella et al., 2007). Beyond that, a restrictive visuomotor-only account that appeals to patient E.B.'s right hemisphere posterior parietal lesion, cannot explain why this slowing occurs for the ipsilesional hand (i.e., this account predicts a slowing of the contralesional hand, and not necessarily for the contralesional field only). Nevertheless, drawing on the limb cueing/activation hypothesis (i.e., that the act itself of using the contralesional limb reduces neglect (Halligan et al., 1991) and extinction severity (Robertson & North, 1993)) enables an intuitive attentional account of why the contralesional field slowing only occurred for the ipsilesional limb.

Like our patient, most (perhaps all) seminal cases (e.g., in Holmes, 1918; Hecaen & De Ajuriaguerra, 1954; Rondot et al., 1977; Perenin & Vighetto, 1988) presenting with optic ataxia have also showed signs of attentional (and other) disturbances. Yet the direct functional link between these symptoms and visuomotor deficits are frequently disregarded because this account cannot explain the specific presentation in which misreaching occurs with one hand (excluding a visual origin) to one field (excluding a proprioceptive/motor origin). Crucially, as a result, later optic ataxia studies may have tended to downplay the concurrency of visuomotor and attentional symptoms despite the possibility that the latter causes the less specific presentation of a field effect (e.g., by a compression of space; Pisella et al., 2021); this report provides evidence for reconsidering the role of attention deficits in optic ataxia.

As for total movement time, there was no comparable contralesional deficit as seen for RT, though patient E.B.'s MT slowing that occurred for both sides of space was found to be restricted to free vision conditions (Figure 6A/6B). This is unlike the previous reports of optic ataxia patients who also show rather distinct presentations: bilateral patient M.H. moved faster to right- and left-sided targets in free and peripheral vision, respectively (Rice et al., 2008), whereas bilateral patient C.F. was slow to the right side (though that study did not include a free vision condition; Striemer et al., 2009). Results for patient E.B. uniquely resembles a speed-accuracy trade-off, which we suspect might be caused by the patient disproportionately relying on visual feedback for free vision conditions (relative to healthy controls). Critically, a speed-accuracy explanation cannot account for the peripheral misreaching field effects because, if peripheral misreaching is simply caused by committing faster movements during peripheral vision, then reaching accuracy would be impaired across visual fields (rather than manifesting as a field effect).

To summarise, we found that a rare visual neglect patient who, despite their right-hemisphere stroke, was still able to undergo an optic ataxia assessment and displayed a field effect characteristic of optic ataxia. Contrary to popular claims that attentional and visuomotor

453 impairments are dissociable, our case report provides evidence in line with (at least some) functional
454 similarity between attentional and visuomotor field impairments.

Credit author statement

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

Declaration of interest

None.

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469 References

- 470 Bálint, R. (translated by Harvey, M.) (1995 [1909]). Psychic paralysis of gaze optic ataxia and spatial
471 disorder of attention. *Cogn Neuropsychol* 12, 265–281.
- 472 Bartolomeo, P. (2007). Visual neglect. *Current opinion in neurology*, 20(4), 381-386.
- 473 Behrmann, M., & Meegan, D. V. (1998). Visuomotor processing in unilateral neglect. *Consciousness*
474 and *Cognition*, 7(3), 381-409.
- 475 Blangero, A., Gaveau, V., Luaute, J., Rode, G., Salemme, R., Guinard, M., ... & Pisella, L. (2008). A
476 hand and a field effect in on-line motor control in unilateral optic ataxia. *Cortex*, 44(5), 560-
477 568.
- 478 Blangero, A., Khan, A. Z., Salemme, R., Deubel, H., Schneider, W. X., Rode, G., ... & Pisella, L. (2010).
479 Pre-saccadic perceptual facilitation can occur without covert orienting of attention. *Cortex*,
480 46(9), 1132-1137.
- 481 Meichtry, J. R., Cazzoli, D., Chaves, S., von Arx, S., Pflugshaupt, T., Kalla, R., ... & Müri, R. M. (2018).
482 Pure optic ataxia and visual hemiagnosia—extending the dual visual hypothesis. *Journal of*
483 *neuropsychology*, 12(2), 271-290.
- 484 Blangero, A., Khan, A., Rode, G., Rossetti, Y., & Pisella, L. (2011). Dissociation between intentional
485 and automatic remapping: Different levels of inter-hemispheric transfer. *Vision research*,
486 51(8), 932-939.
- 487 Blangero, A., Ota, H., Delporte, L., Revol, P., Vindras, P., Rode, G., ... & Pisella, L. (2007). Optic ataxia
488 is not only ‘optic’: impaired spatial integration of proprioceptive information. *Neuroimage*,
489 36, T61-T68.
- 490 Borchers, S., Müller, L., Synofzik, M., & Himmelbach, M. (2013). Guidelines and quality measures for
491 the diagnosis of optic ataxia. *Frontiers in human neuroscience*, 7, 324.

492 Borchers, S., Müller, L., Synofzik, M., & Himmelbach, M. (2013). Guidelines and quality measures for
 493 the diagnosis of optic ataxia. *Frontiers in human neuroscience*, 7, 324.

494 Buxbaum, L. J., & Coslett, H. B. (1997). Subtypes of optic ataxia: Reframing the disconnection
 495 account. *Neurocase*, 3(3), 159-166.

496 Chechlacz, M., Rotshtein, P., Hansen, P. C., Deb, S., Riddoch, M. J., & Humphreys, G. W. (2013). The
 497 central role of the temporo-parietal junction and the superior longitudinal fasciculus in
 498 supporting multi-item competition: evidence from lesion-symptom mapping of extinction.
 499 *Cortex*, 49(2), 487-506.

500 Chieffi, S., Gentilucci, M., Allport, A., Sasso, E., & Rizzolatti, G. (1993). Study of selective reaching and
 501 grasping in a patient with unilateral parietal lesion: dissociated effects of residual spatial
 502 neglect. *Brain*, 116(5), 1119-1137.

503 Coulthard, E., Parton, A., & Husain, M. (2006). Action control in visual neglect. *Neuropsychologia*,
 504 44(13), 2717-2733.

505 Crawford, J. R., & Garthwaite, P. H. (2007). Comparison of a single case to a control or normative
 506 sample in neuropsychology: Development of a Bayesian approach. *Cognitive*
 507 *Neuropsychology*, 24(4), 343-372.

508 Dafotakis, M., Grefkes, C., Wang, L., Fink, G. R., & Nowak, D. A. (2008). The effects of 1 Hz rTMS over
 509 the hand area of M1 on movement kinematics of the ipsilateral hand. *Journal of Neural*
 510 *Transmission*, 115(9), 1269-1274.

511 De Renzi, E. (1982). *Disorders of Space Exploration and Cognition*. Chichester: John Wiley

512 Delis, D., Kaplan, E., & Kramer, J. (2001). *The Delis-Kaplan Executive Function System*. San Antonio,
 513 TX: Psychological Corporation.

514 Dijkerman, H. C., McIntosh, R. D., Anema, H. A., De Haan, E. H. F., Kappelle, L. J., & Milner, A. D.
 515 (2006). Reaching errors in optic ataxia are linked to eye position rather than head or body
 516 position. *Neuropsychologia*, 44(13), 2766-2773.

517 Doricchi, F., de Schotten, M. T., Tomaiuolo, F., & Bartolomeo, P. (2008). White matter (dis)
 518 connections and gray matter (dys) functions in visual neglect: gaining insights into the brain
 519 networks of spatial awareness. *cortex*, 44(8), 983-995.

520 Ferber, S., & Karnath, H. O. (1999). Parietal and occipital lobe contributions to perception of straight
 521 ahead orientation. *Journal of Neurology, Neurosurgery & Psychiatry*, 67(5), 572-578.

522 Gallivan, J. P., McLean, D. A., Flanagan, J. R., & Culham, J. C. (2013). Where one hand meets the
 523 other: limb-specific and action-dependent movement plans decoded from preparatory
 524 signals in single human frontoparietal brain areas. *Journal of Neuroscience*, 33(5), 1991-
 525 2008.

526 Gaveau, V., Pélisson, D., Blangero, A., Urquizar, C., Prablanc, C., Vighetto, A., & Pisella, L. (2008).
 527 Saccade control and eye–hand coordination in optic ataxia. *Neuropsychologia*, 46(2), 475-
 528 486.

529 Goodale, M. A., Milner, A. D., Jakobson, L. S., & Carey, D. P. (1990). Kinematic analysis of limb
 530 movements in neuropsychological research: Subtle deficits and recovery of function.
 531 *Canadian Journal of Psychology/Revue canadienne de psychologie*, 44(2), 180.

532 Granek, J. A., Pisella, L., Blangero, A., Rossetti, Y., & Sergio, L. E. (2012). The role of the caudal
 533 superior parietal lobule in updating hand location in peripheral vision: further evidence from
 534 optic ataxia. *PLoS One*, 7(10), e46619.

535 Gréa, H., Pisella, L., Rossetti, Y., Desmurget, M., Tilikete, C., Grafton, S., ... & Vighetto, A. (2002). A
 536 lesion of the posterior parietal cortex disrupts on-line adjustments during aiming
 537 movements. *Neuropsychologia*, 40(13), 2471-2480.

538 Rorden, C., Karnath, H. O., & Bonilha, L. (2007). Improving lesion-symptom mapping. *Journal of*
539 *cognitive neuroscience*, 19(7), 1081-1088.

540 Rorden, C., Bonilha, L., Fridriksson, J., Bender, B., & Karnath, H. O. (2012). Age-specific CT and MRI
541 templates for spatial normalization. *Neuroimage*, 61(4), 957-965.

542 Halligan, P. W., Manning, L., & Marshall, J. C. (1991). Hemispheric activation vs spatio-motor cueing
543 in visual neglect: a case study. *Neuropsychologia*, 29(2), 165-176.

544 Harvey, M., Jackson, S. R., Newport, R., Krämer, T., Morris, D. L., & Dow, L. (2001). Is grasping
545 impaired in hemispatial neglect?. *Behavioural Neurology*, 13(1, 2), 17-28.

546 Harvey, M., Milner, A. D., & Roberts, R. C. (1994). Spatial bias in visually-guided reaching and
547 bisection following right cerebral stroke. *Cortex*, 30(2), 343-350.

548 Harvey, M., Milner, A. D., & Roberts, R. C. (1995). An investigation of hemispatial neglect using the
549 landmark task. *Brain and cognition*, 27(1), 59-78.

550 Hillis, A. E. (2006). Neurobiology of unilateral spatial neglect. *The neuroscientist*, 12(2), 153-163.

551 Himmelbach, M., & Karnath, H. O. (2003). Goal-directed hand movements are not affected by the
552 biased space representation in spatial neglect. *Journal of Cognitive Neuroscience*, 15(7), 972-
553 980.

554 Hommel, B., Müsseler, J., Aschersleben, G., & Prinz, W. (2001). The theory of event coding (TEC): A
555 framework for perception and action planning. *Behavioral and brain sciences*, 24(5), 849-
556 878.

557 Humphreys, G., Bickerton, W. L., Samson, D., & Riddoch M. J. (2012). BCoS cognitive screen.
558 Psychology Press, Hove.

559 Husain, M., & Nachev, P. (2007). Space and the parietal cortex. *Trends in cognitive sciences*, 11(1),
560 30-36.

561 Husain, M., Mattingley, J. B., Rorden, C., Kennard, C., & Driver, J. (2000). Distinguishing sensory and
562 motor biases in parietal and frontal neglect. *Brain*, 123(8), 1643-1659.

563 Jackson, S. R., Newport, R., Husain, M., Harvey, M., & Hindle, J. V. (2000). Reaching movements may
564 reveal the distorted topography of spatial representations after neglect. *Neuropsychologia*,
565 38(4), 500-507.

566 Jackson, S. R., Newport, R., Mort, D., & Husain, M. (2005). Where the eye looks, the hand follows:
567 Limb-dependent magnetic misreaching in optic ataxia. *Current Biology*, 15(1), 42-46.

568 Jeannerod, M., Decety, J., & Michel, F. (1994). Impairment of grasping movements following a
569 bilateral posterior parietal lesion. *Neuropsychologia*, 32(4), 369-380.

570 K.M. Heilman, E. Valenstein, R.T. Watson The neglect syndrome J.A.M. Frederiks (Ed.), *Handbook of*
571 *clinical neurology*, Vol. 1. Clinical neuropsychology, Elsevier, Amsterdam (1985), pp. 153-183

572 Karnath, H. O., Dick, H., & Konczak, J. (1997). Kinematics of goal-directed arm movements in neglect:
573 control of hand in space. *Neuropsychologia*, 35(4), 435-444.

574 Karnath, H. O., Dick, H., & Konczak, J. (1997). Kinematics of goal-directed arm movements in neglect:
575 control of hand in space. *Neuropsychologia*, 35(4), 435-444.

576 Karnath, H. O., Himmelbach, M., & Küker, W. (2003). The cortical substrate of visual extinction.
577 *Neuroreport*, 14(3), 437-442.

578 Khan, A. Z., Blangero, A., Rossetti, Y., Salemme, R., Luauté, J., Deubel, H., ... & Pisella, L. (2009).
579 Parietal damage dissociates saccade planning from presaccadic perceptual facilitation.
580 *Cerebral Cortex*, 19(2), 383-387.

581 Khan, A. Z., Pisella, L., Vighetto, A., Cotton, F., Luaute, J., Boisson, D., ... & Rossetti, Y. (2005). Optic
582 ataxia errors depend on remapped, not viewed, target location. *Nature neuroscience*, 8(4),
583 418-420.

584 Khan, A. Z., Prost-Lefebvre, M., Salemme, R., Blohm, G., Rossetti, Y., Tilikete, C., & Pisella, L. (2016).
585 The attentional fields of visual search in simultanagnosia and healthy individuals: how object
586 and space attention interact. *Cerebral Cortex*, 26(3), 1242-1254.

587 Konczak, J., & Karnath, H. O. (1998). Kinematics of goal-directed arm movements in neglect: control
588 of hand velocity. *Brain and Cognition*, 37(3), 387-404.

589 Marotta, J. J., McKeeff, T. J., & Behrmann, M. (2003). Hemispatial neglect: its effects on visual
590 perception and visually guided grasping. *Neuropsychologia*, 41(9), 1262-1271.

591 Martin, J. A., Karnath, H. O., & Himmelbach, M. (2015). Revisiting the cortical system for peripheral
592 reaching at the parieto-occipital junction. *Cortex*, 64, 363-379.

593 Mattingley, J. B., Bradshaw, J. L., & Phillips, J. G. (1992). Impairments of movement initiation and
594 execution in unilateral neglect: directional hypokinesia and bradykinesia. *Brain*, 115(6),
595 1849-1874.

596 Mattingley, J. B., Bradshaw, J. L., & Phillips, J. G. (1992). Impairments of movement initiation and
597 execution in unilateral neglect. Directional hypokinesia and bradykinesia. *Brain: a Journal of*
598 *Neurology*, 115, 1849-1874.

599 McIntosh, R. D., McClements, K. I., Dijkerman, H. C., Birchall, D., & Milner, A. D. (2004). Preserved
600 obstacle avoidance during reaching in patients with left visual neglect. *Neuropsychologia*,
601 42(8), 1107-1117.

602 McIntosh, R. D., McClements, K. I., Dijkerman, H. C., Birchall, D., & Milner, A. D. (2004). Preserved
603 obstacle avoidance during reaching in patients with left visual neglect. *Neuropsychologia*,
604 42(8), 1107-1117.

605 McIntosh, R. D., Mulroue, A., Blangero, A., Pisella, L., & Rossetti, Y. (2011). Correlated deficits of
606 perception and action in optic ataxia. *Neuropsychologia*, 49(1), 131-137.

607 McIntosh, R. D., Pritchard, C. L., Dijkerman, H. C., Milner, A. D., & Roberts, R. C. (2001). Prehension
608 and perception of size in left visual neglect. *Behavioural Neurology*, 13(1, 2), 3-15.

609 Medina, J., Jax, S. A., & Coslett, H. B. (2020). Impairments in action and perception after right
610 intraparietal damage. *Cortex*, 122, 288-299.

611 Michel, F., & Henaff, M. A. (2004). Seeing without the occipito-parietal cortex: Simultagnosia as a
612 shrinkage of the attentional visual field. *Behavioural neurology*, 15(1, 2), 3-13.

613 Mijović, D. (1991). Mechanisms of Visual Spatial Neglect. Absence of Directional Hypokinesia in
614 Spatial Exploration. *Brain: a journal of neurology*, 114, 1575-1593.

615 Milner, A. D. (1995). Cerebral correlates of visual awareness. *Neuropsychologia*, 33(9), 1117-1130.

616 Milner, A. D., & McIntosh, R. D. (2004). Reaching between obstacles in spatial neglect and visual
617 extinction. *Progress in Brain Research*, 144, 213-226.

618 Milner, A. D., & McIntosh, R. D. (2005). The neurological basis of visual neglect. *Current opinion in*
619 *neurology*, 18(6), 748-753.

620 Milner, A. D., Dijkerman, H. C., McIntosh, R. D., Rossetti, Y., & Pisella, L. (2003). Delayed reaching and
621 grasping in patients with optic ataxia. *Progress in brain research*, 142, 225-242.

622 Ogourtsova, T., Archambault, P., & Lamontagne, A. (2015). Impact of post-stroke unilateral spatial
623 neglect on goal-directed arm movements: systematic literature review. *Topics in stroke*
624 *rehabilitation*, 22(6), 397-428.

625 Perenin, M. T. (1997). Optic ataxia and unilateral neglect: clinical evidence for dissociable spatial
626 functions in posterior parietal cortex. *EXPERIMENTAL BRAIN RESEARCH SERIES*, 25, 289-308.

627 Perenin, M. T., & Vighetto, A. (1983). Optic ataxia: A specific disorder in visuomotor coordination. In
628 *Spatially oriented behavior* (pp. 305-326). Springer, New York, NY.

629 Perenin, M.-T. Optic ataxia and unilateral neglect: Clinical evidence for dissociable spatial functions
 630 in posterior parietal cortex. In *Parietal Lobe Contributions to Orientation in 3D space*, P.
 631 Thier and H.-O. Karnath (Editors), pp 289–308. Springer-Verlag, Heidelberg, 1997.

632 Pierrot-Deseilligny, C. H., Gray, F., & Brunet, P. (1986). Infarcts of both inferior parietal lobules with
 633 impairment of visually guided eye movements, peripheral visual inattention and optic ataxia.
 634 *Brain*, 109(1), 81-97.

635 Pisella L, Striemer C, Blangero A et al. (2007). Perceptual deficits in optic ataxia? In: Book:
 636 sensorimotor foundations of higher cognition: attention and performance XXII, Chapter: 3,
 637 Publisher, Oxford University Press, pp. 47–72.

638 Pisella, L., André, V., Gavault, E., Le Flem, A., Luc-Pupat, E., Glissoux, C., ... & Gonzalez-Monge, S.
 639 (2013). A test revealing the slow acquisition and the dorsal stream substrate of visuo-spatial
 640 perception. *Neuropsychologia*, 51(1), 106-113.

641 Pisella, L., Grea, H., Tilikete, C., Vighetto, A., Desmurget, M., Rode, G., ... & Rossetti, Y. (2000). An
 642 ‘automatic pilot’ for the hand in human posterior parietal cortex: toward reinterpreting optic
 643 ataxia. *Nature neuroscience*, 3(7), 729-736.

644 Pisella, L., Sergio, L., Blangero, A., Torchin, H., Vighetto, A., & Rossetti, Y. (2009). Optic ataxia and the
 645 function of the dorsal stream: contributions to perception and action. *Neuropsychologia*,
 646 47(14), 3033-3044.

647 Pisella, L., Vialatte, A., Khan, A. Z., & Rossetti, Y. (2021). Bálint syndrome. In *Handbook of Clinical*
 648 *Neurology* (Vol. 178, pp. 233-255). Elsevier.

649 Ratcliff, G., & Davies-Jones, G. A. (1972). Defective visual localization in focal brain wounds. *Brain*,
 650 95(1), 49-60.

651 Revol, P., Rossetti, Y., Vighetto, A., Rode, G., Boisson, D., & Pisella, L. (2003). Pointing errors in
 652 immediate and delayed conditions in unilateral optic ataxia. *Spatial vision*, 16(3-4), 347-364.

653 Rittmo, J. O., & McIntosh, R. D. (2020). singcar: Comparing Single Cases to Small Samples. R package
654 version 0.1.3. <https://CRAN.Rproject.org/package=singcar>

655 Rizzolatti, G., Riggio, L., Dascola, I., & Umiltà, C. (1987). Reorienting attention across the horizontal
656 and vertical meridians: evidence in favor of a premotor theory of attention.
657 *Neuropsychologia*, 25(1), 31-40.

658 Robertson, I. H., & North, N. (1993). Active and passive activation of left limbs: influence on visual
659 and sensory neglect. *Neuropsychologia*, 31(3), 293-300.

660 Rondot, P., De Recondo, J., & Dumas, J. L. (1977). Visuomotor ataxia. *Brain: a journal of neurology*,
661 100(2), 355-376.

662 Rossetti, Y., Pisella, L., & Pélisson, D. (2000). New insights on eye blindness and hand sight: Temporal
663 constraints of visuo-motor networks. *Visual Cognition*, 7(6), 785-809.

664 Rossetti, Y., Pisella, L., & Vighetto, A. (2003). Optic ataxia revisited. *Experimental Brain Research*,
665 153(2), 171-179.

666 Rossit, S., Fraser, J. A., Teasell, R., Malhotra, P. A., & Goodale, M. A. (2011b). Impaired delayed but
667 preserved immediate grasping in a neglect patient with parieto-occipital lesions.
668 *Neuropsychologia*, 49(9), 2498-2504.

669 Rossit, S., Malhotra, P., Muir, K., Reeves, I., Duncan, G., & Harvey, M. (2011a). The role of right
670 temporal lobe structures in off-line action: evidence from lesion-behavior mapping in stroke
671 patients. *Cerebral Cortex*, 21(12), 2751-2761.

672 Rossit, S., Malhotra, P., Muir, K., Reeves, I., Duncan, G., Birschel, P., & Harvey, M. (2009c). The neural
673 basis of visuomotor deficits in hemispatial neglect. *Neuropsychologia*, 47(10), 2149-2153.

674 Rossit, S., Malhotra, P., Muir, K., Reeves, I., Duncan, G., Livingstone, K., ... & Harvey, M. (2009b). No
675 neglect-specific deficits in reaching tasks. *Cerebral Cortex*, 19(11), 2616-2624.

676 Rossit, S., McIntosh, R. D., Malhotra, P., Butler, S. H., Muir, K., & Harvey, M. (2012). Attention in
 677 action: Evidence from on-line corrections in left visual neglect. *Neuropsychologia*, 50(6),
 678 1124-1135.

679 Rossit, S., Muir, K., Reeves, I., Duncan, G., Birschel, P., & Harvey, M. (2009a). Immediate and delayed
 680 reaching in hemispatial neglect. *Neuropsychologia*, 47(6), 1563-1572.

681 Schenk, T., & Hesse, C. (2018). Do we have distinct systems for immediate and delayed actions? A
 682 selective review on the role of visual memory in action. *Cortex*, 98, 228-248.

683 Snow, J. C., Miranda, R. R., & Humphreys, G. W. (2013). Impaired visual sensitivity within the
 684 ipsilesional hemifield following parietal lobe damage. *Cortex*, 49(1), 158-171.

685 Striener, C., Blangero, A., Rossetti, Y., Boisson, D., Rode, G., Vighetto, A., ... & Danckert, J. (2007).
 686 Deficits in peripheral visual attention in patients with optic ataxia. *Neuroreport*, 18(11),
 687 1171-1175.

688 Striener, C., Blangero, A., Rossetti, Y., Boisson, D., Rode, G., Salemme, R., ... & Danckert, J. (2008).
 689 Bilateral parietal lesions disrupt the beneficial effects of prism adaptation: evidence from a
 690 patient with optic ataxia. *Experimental brain research*, 187(2), 295-302.

691 Striener, C., Locklin, J., Blangero, A., Rossetti, Y., Pisella, L., & Danckert, J. (2009). Attention for
 692 action?: Examining the link between attention and visuomotor control deficits in a patient
 693 with optic ataxia. *Neuropsychologia*, 47(6), 1491-1499.

694 Ticini, L. F., de Haan, B., Klose, U., Nägele, T., & Karnath, H. O. (2010). The role of temporo-parietal
 695 cortex in subcortical visual extinction. *Journal of Cognitive Neuroscience*, 22(9), 2141-2150.

696 Vialatte, A., Yeshurun, Y., Khan, A. Z., Rosenholtz, R., & Pisella, L. (2021). Superior parietal lobule: a
 697 role in relative localization of multiple different elements. *Cerebral Cortex*, 31(1), 658-671.

698 Vossel, S., Eschenbeck, P., Weiss, P. H., & Fink, G. R. (2010). The neural basis of perceptual bias and
699 response bias in the Landmark task. *Neuropsychologia*, 48(13), 3949-3954.

700 Warrington, E. K. (1997). The graded naming test: a restandardisation. *Neuropsychological*
701 *Rehabilitation*, 7(2), 143-146.

702 Warrington, E. K., & James, M. (1991). The Visual Object and Space Perception Battery. Bury St.
703 Edmunds (UK): Thames Valley Test Company.

704 Wechsler, D. (2001). Wechsler Test of Adult Reading (WTAR). San Antonio, TX: The Psychological
705 Corporation

706 Wechsler, D. (2008). Wechsler adult intelligence scale—Fourth Edition (WAIS—IV). San Antonio, TX:
707 NCS Pearson, 22(498), 1.

708 Wechsler, D. (2009). Wechsler Memory Scale—Fourth Edition. San Antonio, TX: Pearson

709 Wilson, B.A., Alderman, N., Burgess, P.W., Emslie, H., & Evans, J.J. (1996). Behavioural assessment of
710 the dysexecutive syndrome. St Edmunds, UK: Thames Valley Test Company.

711 Zebhauser, P. T., Vernet, M., Unterburger, E., & Brem, A. K. (2019). Visuospatial neglect-a theory-
712 informed overview of current and emerging strategies and a systematic review on the
713 therapeutic use of non-invasive brain stimulation. *Neuropsychology review*, 29(4), 397-420.